Live Long in Scotland – the relative contribution of medicine and standards of living to Scotland's falling mortality rates

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Declaration

I, Catriona Macdonald, hereby certify that this thesis

a) has been composed by myself, and

b) that the work contained herein is my own, excepting in those areas where the help of others has been acknowledged

Date 25/5/06

Abstract

Between 1950 and 1999 the Scottish death rate, standardised to the 1950 population, declined from 13.1 per 1000 to 7.8 for men and from 12 per 1000 to 6.3 for women. The main aim of this thesis has been to establish the key influences on this decline. Thomas McKeown examined the decline of mortality in an earlier time period (up to the 1970s) and concluded that: falls in deaths from infectious diseases were responsible for the majority of the decline in mortality rate; and the main influence on their decline was standards of living, in particular diet. This thesis has updated McKeown's study for the latter half of the 20th century by establishing which causes of death contributed the most to the fall in mortality in Scotland, and the main influences on their decline.

The causes of death which contributed most to the decline in mortality rates 1950-1999 were established by digitising GRO(S) mortality records and calculating 'potential lives saved'. This method allows an estimate to be made of the number of deaths which would be expected to occur in 1999, taking into account changing age structure, if the death rates from 1950 still applied. Tuberculosis (TB), stomach cancer (SC), ischemic heart disease (IHD) and stroke were selected as case studies; these accounted for over 70% of the decline of male and female mortality.

The trajectory of decline in these causes of death was then considered in detail and set in social and medical context. The role of medicine was defined broadly, including not only treatments, but also changes in the environment or in population behaviour which benefited health and could be proven to have been inspired by medicine. This differs from McKeown's approach which had a more clinical focus.

The thesis has reached an alternative conclusion from that of Thomas McKeown. It is argued that improving standards of living were responsible for some of the decline in SC and haemorrhagic stroke mortality, and the pre-1950 decline of TB; however, the main influence on the decline of TB, IHD and ischemic stroke mortality post-1950 was medicine. Using the wider definition of the influence of medicine adopted in this study, medicine is considered to account for the majority of the decline in the identified causes of mortality. Medicine has, at last, delivered important contributions to the life expectancy of the Scottish population.

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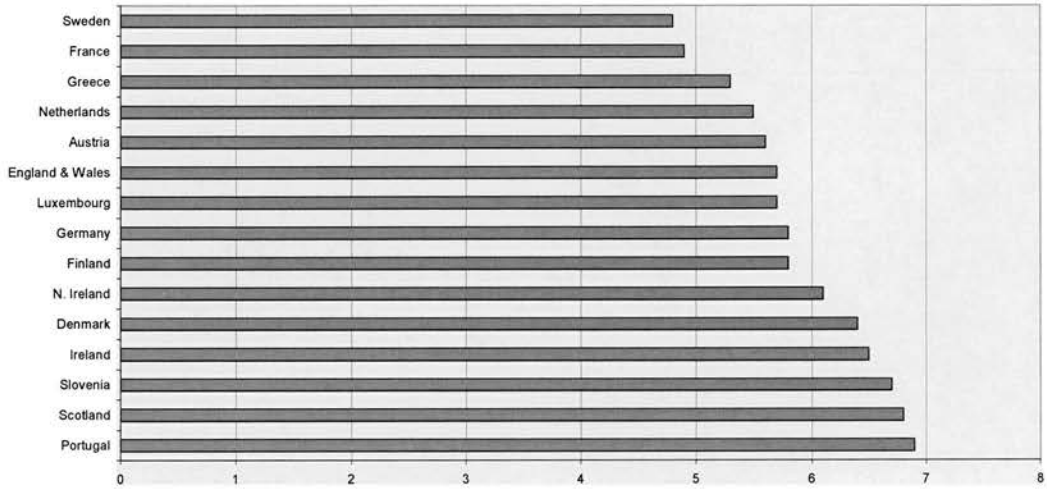
1. Introduction

At the end of the 20th century Scotland was still trying to shed its reputation as the 'sick man' of Europe. This imagery is reinforced by the fact that Scotland has the highest death rates in Western Europe for both ischemic heart disease (IHD) and malignant neoplasms (Scottish Executive, 2001). Only Portugal has a higher all-cause death rate than Scotland (see figure 1.1).

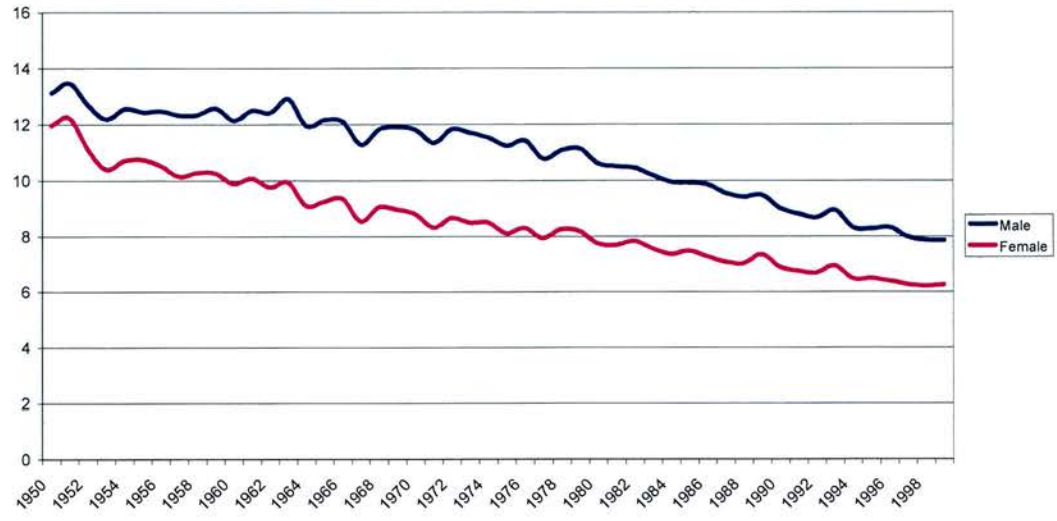
Figure 1.1. 1996 age standardised all-cause death rates, per 1000 population, selected Western European countries



Source: Adapted from Scottish Executive, 2001.

Comparing Scotland's mortality statistics relative to those of its near neighbours provides a negative impression of the health of the nation. However, this bleak picture disguises the decline in Scotland's mortality during the second half of the 20th century from 13 per 1000 among males, and 12 per 1000 among females, in 1950, to 8 and 6 respectively in 1999 (see figure 1.2). These declining death rates have led to an increase in life expectancy of 9.4 years for males and 10.2 years for females since 1950.

Figure 1.2. All cause death rates, standardised to the 1950 population, per 1000, 1950-99



Source: Original analysis of data acquired from the General Registrar's Office for Scotland.

It is this *decline* in mortality with which this thesis concerned. The main aim is to establish the contribution of standards of living and medicine to the mortality decline, focusing on the years 1950-1999. This time period was chosen for a number of reasons. The start date of 1950 was selected as this covers a time when the NHS was in existence and many of the modern innovations in medicine first became available. In addition the selection of the dates was influenced by International Classification of Disease (ICD) coding changes. An end date of 1999 was selected as in 2000 ICD 10 was introduced. This involved significant changes to the way in which death data were coded and would have led to complications in tracing trends in mortality by cause. The implications of coding change are discussed in depth in section 4.4.

It should be emphasised that this thesis is interested in the influence of standards of living and medicine on *mortality* decline. Factors influencing morbidity will not be covered. This is because mortality provides a more robust measure of population health, with good quality statistics available on deaths in Scotland by cause over the time period covered. The study of morbidity would require the use of a wide range of statistics on health and quality of life which would be unmanageable for this thesis.

The study of the influences on mortality decline is not new and a large body of literature exists on the subject. This will be considered in chapter two which will outline the main theories on mortality decline, including early 20th century theory which attributed much of the decline to medicine, the work of Thomas McKeown who challenged these theories, and the findings of late 20th century commentators who again placed medicine at the centre of the mortality decline. In addition, the current direction of Scottish health policy, and any influence which these theories may have had on policy, will be discussed. The findings and problems encountered in these studies will be considered in relation to the aims of this thesis. These will be incorporated into the thesis methodology, which will be outlined in chapter three.

In order to consider why mortality declined it is first necessary to establish which causes of death contributed most to the mortality decline. The process of creating a shortlist of causes of death will be discussed in chapters four and five. These include a description of the source of the death data (the Registrar General's Death Returns) and the problems which are involved when using this source. The process of sampling, and the calculations and criteria employed to create a shortlist of causes of death, will be described.

From the Registrar General's Death Returns, four causes of death will be selected as case studies. The relative contribution of standards of living and medicine to the decline of each case study disease will be discussed in chapters six, seven and eight. The approach taken to examining each disease will vary slightly depending on the timing of each disease's mortality decline and the factors which are found to influence their decline.

In the conclusion (chapter nine) the problems which have been encountered by this thesis on the study of mortality decline post-1950 will be outlined. The relative contribution of medicine and standards of living to the overall decline in mortality between 1950-1999 will then be discussed. Finally, the implications of the findings of this thesis for future mortality decline will be considered.

2. Literature review

2.1. Introduction

The main aim of this thesis is to establish the relative contribution of medicine and standards of living to the decline of mortality in Scotland since 1950. A body of literature has developed on this subject since the growth of modern medicine in the early 19th century. The conclusions reached in the literature have not remained constant, with both medicine and standards of living being afforded the more significant role in the mortality decline during different historical periods. These findings are important as they may influence the direction of policy aimed at health improvement. By establishing the main factors influencing the decline of mortality in Scotland since 1950, the appropriateness of current policies towards health improvement can be assessed. This chapter will review the literature dealing with the main theories of the decline in mortality, providing a historical and methodological context for the thesis.

Much of the literature covering mortality decline has not dealt specifically with Scotland. However, most of the studies have considered mortality decline in countries which experienced similar mortality trends, such as England and Wales, and it is reasonable to start with the assumption that the issues addressed in the literature apply to Scotland too. The first theories which will be reviewed are those which prevailed before the 1970s when the general belief, amongst medical professionals, was that medicine *was* the main determinant of the decline of mortality. Next, the work of Thomas McKeown will be considered. McKeown was one of the first commentators to challenge the role attributed to medicine in the modern decline of mortality. His work will be considered in depth as it is the forerunner of this thesis. His findings and methodology have come under scrutiny and the literature dealing with this will also be considered. The long term impact of McKeown's work will then be assessed. Those commentators who consider that medicine has had a more proactive role will also be reviewed. In addition, current Scottish health policy will be discussed.

2.2. Pre-1970s theories on the rise of population and the role of medicine

Population growth accelerated in Britain from the 18th century. Prior to the publication of Wrigley and Schofield's (1989) research on parish registers, it was assumed that the factor driving this increase was declining mortality rates (Buer, 1926). The decline of mortality since the 18th century was ascribed to two sets of influences. The first was the absence of famine following the introduction of new farming methods during the agricultural revolution (Buer, 1926). Famine had always been associated with excess mortality, not only as the direct result of starvation, but also because of the diseases which accompanied it. The absence of famine meant that population growth was no longer curtailed by a 'mortality crisis'. The other factor believed to underlie the long term growth in population was medical advance. The role which medicine was thought to play in mortality decline will be outlined briefly.

The belief in both the preventive and curative powers of medicine dates from the revolution which occurred in medicine after the 17th century. Until the renaissance, 'Western' medical theory and treatment had not advanced since the time of Galen in the second century, after which the care of the sick had fallen into the hands of the church. However, from the 17th century Western medicine began to develop along more scientific lines, based upon observation and characterised by advances such as Versalius's work on anatomy and Harvey's study of the movement of the blood (Buer, 1926). Increasing knowledge of the body's mechanisms led to the belief, amongst both doctors and the public, that medicine was responsible for the reduction in disease by repairing the body. These views are described in Buer's 1926 study of the decline in mortality, which saw medicine as a central force. Buer highlighted the role which medicine played in the prevention of disease throughout the 18th century, citing examples such as the introduction of inoculation for smallpox. Medicine was also linked with environmental improvements which were believed to have led to the reduction of malaria, typhus, scurvy and the plague.

In the 19th century medicine was credited with more direct interventions in both the prevention and cure of disease. This conviction was reinforced by a number of high profile medical discoveries. Pasteur's research on germs was seen as opening the door to disease prevention by allowing the development of vaccination. The 19th century witnessed the rise of hospitals, encouraged by the growth in medical knowledge. Discoveries, such as Semmelwies's demonstration of the infectious nature of puerperal fever, led to an increase in the belief that medicine offered the potential of cure. The role of the hospital expanded

following Lister's work on antiseptic surgery which meant that operations no longer carried the very high death rate with which they had previously been associated (Haggard, 1929). Medical knowledge advanced into the 20th century, with the introduction of the first effective drug treatments against a number of conditions. This began with the treatment of syphilis and subsequently the development of the sulphonamides in the 1930s, leading to the introduction of antibiotics.

These high profile medical advances led to the belief that the main determinant of health was medical treatment. The strength of this conviction is illustrated in the description by a doctor in 1929 of what he thought would happen to a city such as London, and then the rest of the country, if medicine were suddenly to disappear.

> *"...its civilisation would go back not merely 50 years it would go back 500 years, if indeed the demoralisation and panic at first produced did not destroy the city entirely... The pestilences would return, epidemics would sweep across the country and within a decade the greater part of the population would be wiped out".* (Haggard, 1929: 381).

2.3. Anti medical opinion – Ivan Illich

By the mid 20th century the general opinion, amongst the medical profession, was that medicine had been responsible for the decline of mortality. However, this was not a view held by everyone. One of the harshest critics of the medical profession was Ivan Illich. Illich did not write exclusively about medicine; rather, he was concerned with the corruption of institutions. In his 1975 book *Medical Nemesis* he outlined his case against medicine. Illich saw medicine as an essentially sinister institution and offered three main reasons for this conclusion.

1. That medicine does more harm than good.
2. That the demand for clinical services increases with their availability.
3. That medical treatments reduce the individual's capacity to care for himself and to face suffering and death.

Rather than turning to the medical profession in times of ill health, Illich instead recommended self treatment. This was a recommendation he followed himself when he was diagnosed with cancer in 1983, after which he refused all treatment by doctors until his death in 2002 (Bunker, 2003).

2.4. Thomas McKeown

Thomas McKeown (1911-1988) entered the debate on the determinants of mortality in the 1960s. His central contention was that the medical profession, and in particular medical treatment, was not the main influence on the decline in mortality. He argued that people equated the growth of medical knowledge with the ability to cure disease. McKeown acknowledged major breakthroughs in the study of the body and disease but believed that these had led to the faulty analogy of the body as a machine: when a part broke down, medicine could repair it. This, he argued, led to physicians focusing on the diagnosis and treatment of disease based around acute hospital services, rather than the investigation of disease origins and prevention. His criticism of medicine was not as radical as that of Illich and he distanced himself from Illich's anti-medical views. Instead, McKeown wanted to see the development of a new type of health service. This service was to incorporate curative medicine, but also disease prevention and the promotion of the care and comfort of patients who, he argued, were neglected under the existing system. In order to illustrate that the health services in the mid to late 20th century were too focused upon treatment, McKeown revised the accepted version of the mortality decline (previously described in section 2.2) and attempted to demonstrate that medical intervention had little to do with the long term decline in mortality; rather, he pointed to the decline in disease incidence. He used these finding to justify his model of a new role for medicine in the late 20th century. The 'McKeown thesis' will be considered in detail, together with the criticisms which have been levelled at this work.

2.4.1. Thomas McKeown – background

McKeown first began to doubt the value of hospital based medicine when he was a student at Oxford before World War Two. He felt that medicine was focused on the diagnosis of disease in the patient and the prescription of the relevant treatment, with little attention being paid to the efficacy of the treatment once the condition had been diagnosed. This led him to question whether medicine was "*making anyone any wiser or any better, and [he] soon came to the conclusion that they were not*" (McKeown, 1979: xii). Whilst medicine centred on diagnosis and treatment, McKeown saw little evidence of interest in the origins of disease; he wanted to know *why* a patient had become ill in the first place. He believed that the main role of medicine was not to treat patients once ill but to identify the reasons why illness had occurred and attempt to prevent the disease from developing in the first instance. After his appointment to the post of Professor of Social Medicine at Birmingham McKeown pursued this aim. First, he wrote a series of articles dealing with the determinants of the mortality

decline, then he wrote a book, *The Role of Medicine* (1979), in which he used his reassessment of the role of medicine in the past to map out a new role for medicine in the future.

2.4.2. The McKeown thesis

McKeown set out to re-examine the influences on the increase in population since the 18th century. He agreed with the views of earlier commentators that the main influence on the increase in population was the decline in mortality, which, he stated, began in around 1770. McKeown considered the influences on mortality between 1770 and 1970. The methodology he employed fell into two main areas: first, the establishment of the causes of death which contributed most to the decline in mortality; and, second, the extent to which their decline was due to advances in medicine or improving standards of living. This methodology and his main findings are discussed below.

2.4.2.1. Calculation of the causes of death which contributed most to the decline in mortality

The source used by McKeown was the Registrar General's Death Returns for England and Wales, which are available for the period following 1838. McKeown referred to "*impressive indirect evidence*" (McKeown, 1979: 29) that mortality had been declining before this date but provides no references to this evidence. McKeown briefly considered the reliability of the death data over such a long time period, and argued that the effect of misdiagnosis and registration errors could be reduced by dealing with the infections within broad categories based on disease origin. Grouping the infections was intended to reduce problems caused by differential diagnosis amongst diseases which were transmitted in the same way, for example typhoid and cholera. McKeown used death rates to measure mortality trends. He standardised these to the 1901 population to take into account changing population structure. From these he attributed 74.4% of the total decline in mortality between 1838-1854 and 1971 to infectious diseases, and 25.6% to the decline of non-infective causes. Therefore McKeown focused his study on 'the infections'.

In *The Role of Medicine* (1979) McKeown did not set out his methodology for calculating the relative contribution of specific infections to the decline in mortality. Instead his approach is implied within the discussion of his findings. The consequence of this is that it is difficult to establish how he achieved his results. Although a detailed discussion of his calculations is not provided in the text it is possible to deduce what process he followed. To

calculate the contribution of an individual disease to the decline in total mortality McKeown first calculated the difference in the all-cause death rate between 1848-1854 and 1971. This produced a figure for the total decline in mortality. This figure is not provided in the text but the decline in the all-cause death rate is illustrated in a graph (McKeown, 1979: 31). McKeown repeated the process of calculating the difference in the death rates of the main infections over the period between 1848-1854 and 1971. By relating these figures to the difference between the all-cause death rate at two time periods, he estimated the contribution which each disease made to the decline in total mortality. The results of this process for airborne infections are shown in table 2.1.

Table 2.1. Standardised death-rates (per million) from airborne diseases in England and Wales and percentage reduction in all-cause mortality by cause

	1848-1854	1971	% of reduction from all causes attributable to each disease
Tuberculosis	2901	13	17.5
Bronchitis, pneumonia, influenza	2239	603	9.9
Whooping cough	423	1	2.6
Measles	342	0	2.1
Scarlet fever & diphtheria	1016	0	6.2
Smallpox	263	0	1.6
Infections of the ear, pharynx, larynx	75	2	0.4
Total airborne	7259	619	40.3

Source: McKeown (1979) p.34.

McKeown grouped the infections according to how they were transmitted and calculated the percentage of the total decline in mortality due to each group (see table 2.2). Of particular significance was tuberculosis, to which McKeown attributed 17.5% of the decline in total mortality.

Table 2.2. McKeown's disease groupings and percentage contribution to the decline in mortality between 1848-54 and 1971

Disease group	Percentage of total mortality decline attributable to disease group
Airborne disease. For example; tuberculosis, whooping cough, measles, bronchitis, scarlet fever, diphtheria and smallpox	40
Water and food borne diseases. For example; cholera, typhoid, diarrhoea and dysentery.	21
Other diseases due to micro-organisms. For example; typhus, syphilis, puerperal fever.	13

Source: McKeown (1979) p.33.

2.4.2.2. Relative contribution of medicine and standards of living to the decline in mortality

Having established the percentage of the total decline in mortality due to each of the above disease groups, McKeown then considered what factors could have influenced their decline. He did this by considering the timing of mortality decline of specific causes against developments in disease virulence, medical treatments, exposure and standards of living.

1. Change in the character of the disease

In this category McKeown considered whether a reduction in the virulence of disease may have contributed to the decline in mortality from any of his disease groupings. In the case of the water and food borne infections and the other diseases categories, he concluded that there was no change in the virulence of disease. The only disease for which he claimed declining virulence may have had an impact was scarlet fever. Overall, he concluded that declining disease virulence would have had very little effect on the mortality decline.

2. Immunisation and therapy

This category referred to the prevention or cure of a disease by a medical intervention, involving either the administration of vaccine or the treatment of the condition once disease was established. This was the area which, prior to McKeown, had been seen by commentators as making the greatest contribution to the mortality decline. McKeown assessed the effectiveness of medical interventions by comparing the date of introduction of vaccinations and therapies with the timing of the mortality decline. He also considered what percentage of the overall decline in mortality from specific diseases occurred before or after their introduction. An example of this is shown in table 2.3 which displays the proportion of the overall decline of the airborne infections, between 1848-1854 and 1971, which occurred after the introduction of effective medical interventions. McKeown calculated that, between 1901–1971, 56% of the overall decline in the infections occurred *before* the 1930s which was when sulphonamides, the first effective treatments against a variety of infections, became available (McKeown *et al*, 1975). Therefore, although he acknowledged the effectiveness of these treatments, he devalued their contribution to the long term reduction in mortality arguing that the diseases were already in decline when they were introduced, and would presumably have continued to decline in their absence. McKeown also claimed that vaccination did not play a large role in the prevention of the infections, as much of the decline in incidence had occurred before they became available. He therefore refuted the previously central role which Pasteur's work on germ theory and the development of vaccine was thought to occupy. In the case of other infectious diseases, McKeown did acknowledge the contribution of Semmelweis's discoveries on puerperal fever on the reduction of maternal deaths and also the treatments developed in the first decades of the 20th century against syphilis. Nevertheless, McKeown concluded that, over the entire time period, immunisation and therapy contributed little to the total decline in mortality.

Table 2.3. Airborne diseases: fall of mortality since the introduction of specific measure of prophylaxis or treatment, 1848-54 to 1971

Cause	*Fall in standardised death rate per million, 1848-54 - 1971*	*Fall as a percentage of fall of all causes*	*Year when specific measures became available*	*Fall by 1971 after the introduction of specific measures*	*Proportion of total fall after the introduction of specific measures*
Tuberculosis (respiratory)	2888	17.5	1947	409	0.14
Bronchitis, pneumonia, influenza	1636	9.9	1938	531	0.32
Whooping cough	422	2.6	1938	43	0.10
Measles	342	2.1	1935	50	0.15
Scarlet fever	1016	6.2	1935	15	0.30
Diphtheria			1894	263	
Smallpox	263	1.6	Before 1848	263	1.00
Infections of the ear, pharynx, larynx	73	0.4	1935	65	0.89
All airborne infections	6640	40.3		1668	0.25

Source: McKeown (1979) p.51.

3. Reduction of exposure to infections

The consequence of reduced exposure to infection is the decline in disease incidence. The main influence on reduced exposure during the time period covered by McKeown was the introduction of public health services. In respect of airborne infections McKeown claimed that it is "*virtually impossible to prevent transmission of airborne infections*" (McKeown *et al*, 1975: 55). McKeown also claimed that, since tuberculosis had been in decline before the introduction of registration in 1838, the work of preventative medicine could have had little effect as there were no measures in place at that time. The only disease group which McKeown claimed the public health services were effective in reducing were the water and food borne diseases, for which a "*substantial reduction of mortality is likely to be achieved by prevention of exposure*" (McKeown, 1979: 56). McKeown placed the start of the decline of the water and food borne group in the 1870s and linked this to improvements in conditions brought about by the sanitary authorities at that time. Overall McKeown

attributed one sixth of the total decline of mortality to the water and food borne diseases and the work of the sanitary authorities.

4. Improving standards of living and nutrition.

McKeown next turned his attention to rising standards of living. He employed the logic of Sherlock Holmes: "*when we have eliminated the impossible, whatever remains, however improbable, must be the truth*" (McKeown and Record, 1963: 94). To McKeown, this logic suggested that since the first three scenarios were not responsible for the decline in mortality, then the last must be true. He argued that rising real wages throughout the 19th century had led to improvements in diet, and that this strengthened resistance to disease. Tuberculosis was of central importance to McKeown's argument. McKeown had isolated tuberculosis as making the single largest contribution to the decline in mortality. McKeown had already established that the disease had not declined in virulence and that effective medical treatment was not available until 1947. He also claimed that public health measures would not have been effective against it as it was an airborne infection. The fact that McKeown placed the start of the decline of tuberculosis before 1838 is also significant as McKeown claims that the only one of his four groups of influences which was improving at that time was diet. Therefore, McKeown claimed the most significant influence on the mortality decline had been diet.

2.4.3. McKeown's conclusions

McKeown's findings regarding influences on the mortality decline between 1838-54 and 1971 are very different from those outlined in section 2.2. Previously medical treatments and vaccination had been seen as essential to the decline of the infections. McKeown suggested that, taken as a whole, the majority of the decline in the infections had occurred before effective treatments or vaccines were available. Although McKeown did acknowledge the work of the sanitary authorities in the prevention of water and food borne infections, he placed improving diet and standards of living at the centre of his explanation of mortality decline. It is this evidence of the past ineffectiveness of medicine which McKeown used to justify his call for a different role for medicine in the late 20th century (see later section 2.4.6).

The McKeown thesis has attracted criticism; this will be considered to determine the strengths and weaknesses of his appraisal of the role of medicine, and to highlight any methodological problems which may be of relevance to this thesis.

2.4.4. The 'McKeown thesis' revisited

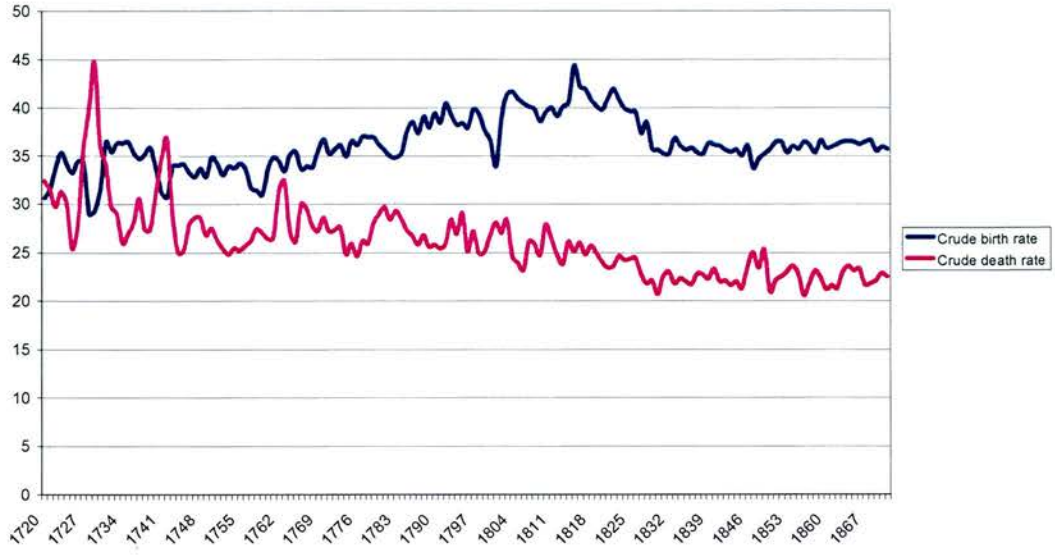
In most cases McKeown's critics agree with his main point, that clinical medicine contributed little to the overall decline, but question aspects of his methodology and his treatment of the role of the public health services. One of McKeown's most prominent critics is Szreter (1988) who has highlighted three areas of concern.

1. McKeown's findings are based on misinterpreted data

McKeown believed, as did earlier researchers, that the main influence on the increase in population since the 18th century had been the decline in mortality. McKeown had presumed that fertility levels had always been at a maximum meaning that there was no potential for population growth from higher fertility. By placing the start of the mortality decline in 1770, McKeown was able to claim that medicine had not played a role in the decline as there were virtually no effective medical or public health measures available at that time; this strengthened his argument for the importance of diet.

Evidence uncovered since the publication of *The Role of Medicine* (1979) has shown that McKeown was wrong in his assumption that declining mortality was the main factor driving the pre-1838 population increase. Official national statistics for mortality for England and Wales are available from 1838, the start of national registration (this was introduced in 1854 in Scotland). Prior to this the only source of information were Parish Registers. Wrigley and Schofield (1989) have modelled the information from these registers on births, burials and marriages to reconstruct levels of fertility and mortality between 1541 and 1870. The results over the period 1720 and 1870 are displayed in figure 2.1.

Figure 2.1. Crude birth and death rates, per 1000, 1720-1870 (England and Wales)



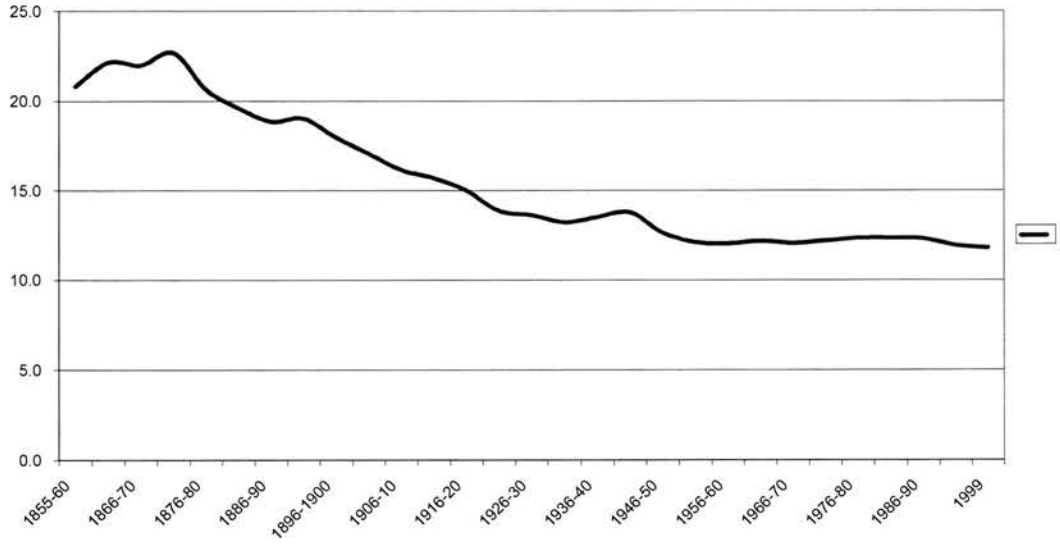
Source: Adapted from Wrigley and Schofield (1989).

Wrigley and Schofield's study showed that up until the 18th century population remained relatively stable and growth was held by a series of Malthusian checks. The first of these checks was preventative: population remained in line with resources by limiting fertility. This took the form of later marriage during times of shortage. If this measure proved ineffective then a 'positive check' occurred. This took the form of a mortality crisis, such as famine or an epidemic which drastically reduced the population. The last examples of 'crisis mortality' are shown in the early 18th century in figure 2.1. This situation began to change in the 18th century as the improved farming methods of the agricultural revolution led to an increase in resources (food), leading to a gentle increase in the birth rate up until the mid 18th century. The birth rate then increased at an accelerated rate to reach a peak in 1816, after which there was a slight fall, before the rate increased steadily until the 1870s.

In the case of mortality, Wrigley and Schofield (1989) identified a rate which stayed relatively high until the second half of the nineteenth century. The absence of famine and epidemics led to a reduction in crisis mortality; however, this was replaced by endemic disease, as a greater proportion of the population crowded into towns, which suffered from excess mortality compared to rural areas. Although a slight decline in mortality was seen in the late 18th and early 19th centuries, mortality then remained stable until the 1870s.

This reinterpretation of the pattern of population increase revealed a different situation to that assumed by McKeown and earlier researchers. The increase in fertility seen during and after the agricultural and industrial revolutions, rather than declining mortality, was the main determinant of the rise of population. Mortality rates did benefit from the absence of 'crises' of earlier centuries; however, any significant decline in the death rate was prevented by the deterioration of the conditions in the towns. Wrigley and Schofield did not detect any secular downwards trend in mortality until the latter part of the nineteenth century. These findings are in line with General Registrar's Office for Scotland (GROS) total mortality data for the nineteenth century which do not date the start of the Scottish mortality decline until the 1870s (see figure 2.2.) .

Figure 2.2. Scottish death rates per 1000, 1861-2000



Source: Adapted from GROS data.

In addition to Wrigley and Schofield's revision of the pattern of decline of total mortality, Szreter (1988) also questioned the accuracy of McKeown's findings regarding tuberculosis. McKeown placed tuberculosis at the centre of his argument in favour of diet, arguing that, as tuberculosis had been declining before 1838, improving diet was the only factor which could have influenced its decline. Szreter, however, claims that the slight reduction of tuberculosis witnessed after the introduction of registration was only a short term discrepancy and that the long term trend of reduction was only established in the second half of the nineteenth

century. If tuberculosis and other causes of deaths began to decline at this later date, the contribution of other factors (which McKeown rejected, based on his belief that mortality was declining before they were introduced) will now have to be considered, in particular the role played by public health.

2. McKeown's definition of medical influences on mortality

Most commentators on the McKeown thesis agree with McKeown's assessment of the contribution of immunisation and therapy: the majority of the decline of infections occurred before effective measures became available. It is McKeown's definition of public health and preventative medicine which has received the most criticism. McKeown's assessment of public health included only the work of the sanitary authorities, which he credited with the decline of the water and food borne infections after the 1870s. However, McKeown failed to consider the vast array of other measures introduced by local and national government which were aimed at improving health. Perenoud (1991) has drawn attention to the work of national government as far back as the 17th century in bringing crisis mortality from plague under control through the use of measures such as quarantine. Most researchers have focused their attention on the 19th and 20th centuries and have highlighted the work of the medical profession in reducing mortality through lobbying for reform, direct action and their work as researchers and statisticians (Woods, 1991). Szreter (1988) has stressed the role played by both medical and sanitary reformers at the local level to improve the environment through the regulation of housing, factories, food, drainage and sewage. In addition, Morel (1991) has emphasised the contribution of the medical profession in the education of the public in areas such as infant hygiene and feeding practices. McKeown attributed the reduction in infant mortality to improvements in diet. However, if the decline was linked to hygienic measures, a connection could be made between infant mortality and the diffusion of the findings of Pasteur.

Thus, given the later date of the mortality decline established by Wrigley and Schofield, all the measures outlined above could potentially have influenced the decline of mortality in the late 19th and 20th centuries by improving the environment and so reducing the population's exposure to infection.

3. McKeown's use of exclusion to prove his conclusions regarding standards of living and diet

As we have seen, McKeown reached his conclusion, that improving standards of living and diet were the most important influences on the mortality decline until the 1970s, by a process of elimination. Szreter (1988) has claimed that this involved "*the sceptical devaluing of other factors, including medical intervention, rather than because of convincing evidence in its [diet's] favour*" (p.10). Szreter is referring to the order in which McKeown examined each of the four sets of influences which he isolated as having potentially reduced mortality. Szreter claims that by considering last the role played by standards of living and diet, after excluding all other influences, McKeown required a lower standard of evidence to prove his case than he had in the earlier scenarios.

2.4.5. Implications of the review of the 'McKeown thesis'

The main criticism of the McKeown thesis centres on methodological issues, in particular his examination of death data, and his method of measuring the relative contribution of various influences on mortality decline. Although the McKeown thesis has received close scrutiny, most researchers have accepted his findings regarding the role played by medical therapy and immunisation. The main area of concern has been his treatment of the public health services and their role in the prevention of disease.

However, McKeown's main aim in examining the mortality decline was to justify his plan for the future role of medicine. In this McKeown wished to emphasise the importance of disease prevention caused by improving standards of living, to call for changes in the priorities of Britain's health services, away from the focus on acute services towards disease prevention. McKeown's model for disease prevention is discussed below.

2.4.6. McKeown's concept of the role of medicine in the late 20th century

McKeown did not view the majority of disease as inevitable. Rather, he saw disease as the result of environmental influences working on genetic material. If disease is determined by environment, he argued, then mortality from the disease can be reduced by controlling aspects of the environment. McKeown divided the residual disease problems of Britain into four groups depending on the origins of the disease, and then considered what measures could be taken to prevent their development. These will be discussed below. Finally McKeown's view on the future role of the clinical medical services will be considered.

2.4.6.1. Role of medicine in disease prevention

1. Relatively intractable diseases

This is the one disease group which McKeown identified as not being influenced by environment. Relatively intractable diseases are those which are determined before birth and include a wide range of congenital conditions. In the case of mild disorders or those which only manifest themselves in old age, McKeown advised that no action be taken. In case of serious congenital abnormalities, such as anecephalus, McKeown stated that the only way in which mortality could be reduced was by preventing birth. To achieve this he recommended the use of diagnostics to identify affected pregnancies and the use of selective abortion (McKeown, 1979: 167-168).

2. Preventable, associated with poverty.

Included within this category are the diseases of absolute want, caused by complete deficiency of resources. These include causes of death such as starvation. McKeown stated that these types of disease are no longer present in industrialised countries, and are now associated with developing nations (McKeown, 1979: 168-169).

3. Preventable, associated with affluence.

McKeown claimed that the majority of the residual disease problems in industrialised nations are associated with affluence. Interestingly, many of the diseases which McKeown included under this heading are now seen in the Western world as diseases of relative poverty. However, under McKeown's definition these include all diseases that are associated with modern society, including: lung cancer, ischemic heart disease, and most other forms of cancer. McKeown argued that most of the potential for mortality reduction comes from the prevention of these causes. He recommended three ways in which medicine could approach their prevention (McKeown, 1979: 169-171).

- *Nutritional*

McKeown identified a deficit of food as being one of the main determinants of past mortality, with malnourished individuals having a lower resistance to infections. In the later half of the 20th century, when mortality was dominated by the degenerative diseases, McKeown again identified diet as a major determinant of health and mortality. However, in this case it was excess food consumption and poorly balanced diets, rather than actual want of food, which McKeown considered to be the main problem. The prevention of these diet-

related illnesses lies in the modification of population diet towards healthier options. McKeown viewed the role of the medicine as two-fold: first, the identification of unhealthy aspects of diet; and, second, advice to government, which McKeown argued should lead to food subsidies, rather than seeing diet modification as an area of individual responsibility.

- *Environmental*

McKeown classed environmental medicine as the work of the sanitary authorities. However, McKeown recommended a far-reaching role for medicine in the future through the control of environmental hazards which affect health and mortality. These included influencing policy towards housing, atmospheric pollution, traffic and working conditions.

- *Behavioural*

McKeown identified personal behaviour as one of the main determinants of ill-health and mortality. When McKeown published *The Role of Medicine* in 1979, the question of whether it was appropriate to attempt to change personal behaviour was novel. It was argued that this was an unreasonable intrusion on the rights of the individual, whilst the feasibility of bringing about widespread behaviour change was questioned. McKeown wanted to see medicine actively involved in educating the public of the dangers of smoking and a sedentary lifestyle. In addition, McKeown advocated an imaginative approach to behaviour modification, which not only included public health messages but also the creation "*of an environment which encourages people to do what is good for them and to avoid what is bad*" (McKeown, 1979: 125).

4. Preventable, not known to be related with poverty or affluence

The final group of diseases which McKeown considered were those which are theoretically preventable and not known to be influenced by either poverty or wealth, examples of which include the common cold, influenza and many psychiatric conditions. In many of these cases the original cause of the disease had not been identified at the time at which McKeown was writing; he was therefore unable to suggest a strategy for their prevention. However, he foresaw a point in the future when this should be possible (McKeown, 1979: 171-174).

2.4.6.2. Role of clinical medicine

Although McKeown questioned the superior position afforded to personal medicine, and in particular the focus of attention and resources on the acute hospital sector, his criticism was not as extreme as that of Illich, who viewed medicine as actually harmful. Instead, McKeown recognised the role which medicine could play in the treatment of those already ill, and acknowledged the increasing effectiveness of medical treatment, although he wanted to see a growth in the formal appraisal of both treatments and services. However, McKeown still expected clinical medicine post-1971 to make only a relatively small contribution to the prevention of sickness and death (McKeown, 1979: 179). In McKeown's model of medicine in the future the earlier roles would be reversed, with clinical medicine taking the subordinate position to the role of medicine in disease prevention by encouraging population behavioural change and environmental improvement.

2.5. McKeown's impact on late 20th century views on the role of medicine

The long term impact of the McKeown thesis is echoed in the way the medical profession have modified their views on the effectiveness of medical interventions in reducing mortality. Whilst this is an important topic within medicine today, it is possible that many commentators are unaware that it was McKeown who began this re-assessment of the role of medicine. Prior to the publication of the McKeown thesis, the focus of medicine was cure-based and centred on institutional treatment. This aspect of medicine remains. However, understanding of how to improve health, and who is responsible for health improvement, has broadened. Disease prevention is now seen as an important part of health improvement and mortality reduction. These changing views are reflected in the statement of the Chief Medical Officer for England and Wales regarding the role of medicine in the 1990s: "*to improve health, health care and quality of life of the individual and population by health promotion, prevention of illness, treatment and care, and the effective use of resources, all within the context of a team approach*" (Calman, 1994: 1140). This statement emphasises the importance which the medical profession now attach to disease prevention.

The changing perceptions of their role amongst the medical profession is reflected in the findings of the 1994 meeting of doctors' leaders (Morrison and Smith, 1994). This was the first time since 1961 that the leaders of the BMA, GMC, Royal Colleges and representatives of the health departments had met. Their aim was to discuss the core values of medicine and to plan for the future role of medicine. At the centre of these discussions were two main

points. First, they perceived a shift in power in medical care towards patients (or consumers) who are prepared to challenge the authority of the doctor and who want a greater role in clinical decision making. Second, they perceived shifting boundaries of health and illness. Here the influence of the McKeown thesis can be detected. The results of these talks produced a vision for the future of medicine which strongly resembled that outlined by McKeown in the 1970s. The doctors stressed that health is not simply determined by medical treatments, but by the interaction of social, economic and psychological factors. The responsibility for tackling these problems was not purely medical, but rather required a combined approach involving medicine, government and other agencies.

This thesis argues that McKeown's research began a shift in opinions which led to questions being asked about the best way in which to improve health. Prior to the publication of the McKeown thesis the predominant view was that medicine, in the form of treatments to cure disease, and vaccinations to prevent disease, had been the main influence on the decline of mortality. This was reflected in the growth of a medical profession which focused on the cure of disease, rather than investigating disease origins to prevent the development of disease. The McKeown thesis changed this by arguing not only that mortality had declined because fewer people were becoming ill, but that medical treatments had little to do with this reduction. Instead, environment, in the form of improving standards of living and diet, had been the main force driving improving health and declining mortality.

McKeown's vision for the future role of medicine, and the influence which his findings have had on the perceived value of medicine, have been challenged by a number of studies which have examined the influences on mortality *post-1950*. These studies are discussed in the following section.

2.6. Post-McKeown commentators on mortality decline

Since the publication of the McKeown thesis a number of commentators have emerged who challenge not only McKeown's appraisal of the past influences on mortality, but also his recommendations for the future role of medicine. In particular these critics argue that McKeown's assessment of the past influence of clinical medicine is unfair, as many medical interventions were not available when he was working. As such, they maintain that the value of clinical medicine should be based on a time when effective measures *were* available. A number of studies which have dealt with the determinants of declining mortality post-1950 are discussed below.

2.6.1. John Bunker

Bunker has reviewed the influences on mortality decline since 1950 (Bunker, 1995, Bunker 2001). He placed his research in the context of the McKeown thesis, and stated that he wished to address the questions which have emerged amongst the medical profession following the publication of McKeown's *The Role of Medicine* (1979) and Illich's *Medical Nemesis* (1975). Bunker has argued that these works led the medical profession to believe that clinical medicine only has a small effect on health, an opinion reflected in Morrison and Smith's 1994 article. Instead Bunker argued that these views are based on the erroneous belief that clinical medicine contributed little to mortality decline post-1950. He has claimed that, as the McKeown thesis ends in 1971, McKeown did not consider the explosion of medical treatments which emerged since that time. In response Bunker reviewed the influences on mortality between 1950 and 1989 in America, a time period when many medical innovations first became available. Bunker's methodology falls into two main areas: the identification of the causes of death which have declined most since 1950 and the role which medicine played in their decline.

Bunker measured the decline in mortality by calculating life expectancy. First, he calculated the gain in life expectancy between 1950-1989 using data from the US National Centre for Health Statistics. From their life tables he established that the all-cause age adjusted death rate had fallen from 840.5 per 100000 in 1950, to 523 per 100000 in 1989, leading to an increase in life expectancy of 7.1 years. Bunker then used the decline in cause-specific death rates to calculate the contribution which individual diseases made to the increase in life expectancy. He found that 'diseases of the heart' had contributed the most to the increase in life expectancy between 1950-1989, as the death rate had declined from 307 per 100000 to 156 per 100000, contributing 3.88 years to the total increase in life expectancy.

When considering what factors might have influenced cause-specific gains in life expectancy, Bunker only looked at the influence of clinical medicine in the prevention or treatment of disease. He included only those influences in which there was an interaction between a medical professional and a patient, such as screening programmes in the case of prevention, and therapies in the case of treatment. Bunker claimed that it is not possible to accurately measure the role which social, psychological or economic factors may have on mortality, due to the absence of measurable data. To measure the contribution of clinical medicine, Bunker employed data from clinical trials and meta-analyses to establish the effectiveness of interventions. He then identified the population 'at risk' and estimated the

percentage of this population who received the intervention. From this he estimated the gain in months or years of life expectancy which may be attributed to specific interventions. A sample of his findings are shown in table 2.4.

Table 2.4. Sample of gains in life expectancy attributed by Bunker to preventive and curative medical service in the USA, 1950-1989

Preventative/ curative service	Gains in life expectancy distributed across the US population
Screening for hypertension	1.5 – 2 months
Immunisation for diphtheria	10 months
Immunisation for smallpox	3 – 6 months
Treatment for IHD	1.2 years
Treatment for hypertension	3.5 – 4 months
Treatment for appendicitis	4 months
Treatment for diabetes	6 months

Source: Adapted from Bunker (1995).

Having considered the contribution of medicine to the increase in life expectancy Bunker concluded that 3.5 to 4 years of this gain could be attributed to curative services and 1.5 years to clinical prevention (Bunker, 1995). Therefore, Bunker in total credited medicine with between 5 and 5.5 years of the overall gain in life expectancy. Bunker did briefly consider the role played by standards of living, in particular the influence which this may have on class differentials in mortality. However, he stressed that the wide differentials seen between classes 1 (professional) and 5 (unskilled labourer) are extremes, and that in the middle classes differences in life expectancy are small. In his investigation of the post-1950 mortality decline, Bunker thus reached the opposite conclusion from McKeown. This, in turn led him to make different recommendations for the future direction of health services. Bunker argued that funding for clinical medicine should continue as "*improvements in public health [are] now largely complete, medical care is now the major determinant of life expectancy, its impact substantially greater than that of social environment or lifestyle*" (Bunker, 2001: 1262).

2.6.2. Johan Mackenbach

Mackenbach (1988, 1996) also considered the influences on mortality decline, and placed his study within the context of the McKeown thesis. He agreed with McKeown that environmental change was responsible for a large proportion of the mortality decline during certain historical periods. However, he stressed that, because this was the most important influence in the past, it did not mean it was the most important influence post-1950. Furthermore, Mackenbach agreed with Bunker's assessment that the McKeown thesis led to the belief amongst the medical and scientific community that medicine has little impact on health problems. In order to produce a more up to date estimation of the role of medicine Mackenbach reviewed the mortality decline in the Netherlands between1950/54 and 1980/84.

Like Bunker, Mackenbach measured the relative importance of specific diseases to the decline in mortality by using gains in life expectancy. In addition, he only considered the role which medicine played in their decline. Mackenbach's method of assessing the contribution of medicine differs from that used by Bunker. Instead of performing calculations based on effectiveness data from clinical trials, Mackenbach compiled a list of causes of death for which it could be proven that death could be avoided by adequate preventative or therapeutic interventions. He then attributed the decline in these causes of death to medicine; these findings are shown in table 2.5. Mackenbach acknowledged that it was possible not all of the decline in mortality from these conditions could be attributed to medicine, but argued that any over estimation was compensated for by the absence of treatments for ischemic heart disease from his analysis. Therefore, Mackenbach accredited a gain of 2.96 years in male life expectancy, and 3.95 year gain in female life expectancy, to medical interventions.

Table 2.5. The effects of mortality reductions for conditions that have become amenable to medical interventions on average life expectancy at birth (in years) in the Netherlands, 1950/54 – 1980/84.

	Male	Female
Infectious diseases	0.94	1.13
Certain surgical conditions	0.36	0.25
Maternal & perinatal conditions	0.72	0.63
Hypertensive and cerebrovasuclar disease	0.43	1.32
Other amenable conditions	0.58	0.80
All amenable conditions	2.96	3.95

Source: Adapted from Mackenbach (1996) p.1212.

2.6.3. Capewell *et al*

There has been no study which has looked at the factors influencing the decline in all-cause mortality since 1950 specifically in Scotland. However, Capewell *et al* (1999) considered the influences on the decline of coronary or ischemic heart disease in Scotland. Their study examined the relative contribution of medical treatment and risk factor reduction to coronary heart disease in Scotland between 1975 and 1994. As coronary heart disease has been one of the leading causes of death in Scotland during the second half of the 20th century (Capewell *et al*, 1999), the methodology and findings from the study will be considered. As with the studies which have considered the influence on the decline of all-cause mortality, Capewell *et al*'s study can also be divided into two main sections: first the calculation of the decline in mortality, then the analysis of the factors influencing this decline.

To measure the extent to which coronary heart disease mortality declined in Scotland between 1975 and 1994, Capewell *et al* calculated the number of deaths which would have occurred in 1994 if conditions had remained the same as they were in 1975. To do this they calculated the age and sex specific coronary heart disease death rates for 1975 then applied them to age and sex specific population data for 1994. This produced the number of deaths which would be anticipated in 1994, if the death rate remained unchanged. The actual number of deaths which occurred in 1994 were then subtracted from this to give the number of deaths avoided, or potential lives saved in 1994 compared with 1975. Capewell *et al*

(1999) calculated that there were 6747 fewer coronary heart disease deaths than expected in 1994 compared with 1975.

The second part of Capewell *et al*'s (1999) study estimated the relative contribution of medical treatment and risk factor reduction to potential lives saved. In order to approximate the contribution of medical treatments Capewell *et al* used a variety of different sources. These were: information on treatment effectiveness from clinical trials and meta-analyses; data on patient numbers; and treatment uptake. Capewell *et al* (1999) provide details of the calculations they employed and these are shown in box 2.1. The first calculation provides an estimate of the number of deaths which were avoided due to the use of a specific treatment. Capewell *et al* attributed 2722 deaths avoided or lives saved in 1994 to medical treatment. Capewell *et al* then estimated the number of deaths avoided due to the decline in coronary heart disease risk factors. The equation for this calculation is shown in box 2.1. The data required for this were the number of deaths which occurred amongst risk factor groups in 1975 and the percentage decline in the prevalence of the risk factor between 1975 and 1994. These data were acquired from population surveys. In addition Capewell *et al* used a calculated standardised Beta coefficient, interpreted as the percentage reduction in mortality which would be expected for every percentage reduction in the prevalence of the risk factor. This calculation was undertaken for each of the main coronary heart disease risk factors, and Capewell *et al* estimated that 4025 death were avoided, or lives saved, in 1994 compared with 1975.

In total Capewell *et al* (1999) estimated that 40% of the decline in coronary heart disease between 1975 and 1994 was the result of medical treatments for the disease, and that 51% of the decline was the result of the reduction in the main disease risk factors. The shortfall of 9% was attributed to unquantifiable risk factors.

Box 2.1. Capewell *et al*'s calculations

Calculation for deaths prevented due specific medical treatment

X=pta

Where X is the number of deaths prevented, p is the number of patients, t is treatment uptake and a is absolute mortality reduction.

Calculation for death prevented by risk factor reduction

X=drb

Where X is the number of deaths prevented, d is the number of deaths in that group in 1975, r is the risk factor decline and b is the Beta coefficient.

Source: Capewell *et al* (1999).

2.6.4. Post-1950 study conclusions

All three of the authors discussed above came to the opposite conclusion to McKeown: medicine had made a significant contribution to mortality decline. In Bunker's (1995, 2001) and Mackenbach's (1988, 1996) studies on the decline in all-cause mortality, estimates of the role of medicine in increasing life expectancy ranged from 2.96 years to 5.5 years. In both these studies medicine was defined as clinical prevention or treatment; that is, a personal interaction between a medical professional and a patient. Capewell *et al*'s (1999) study of heart disease used a different method to measure the decline in mortality; however, this study also credited a large share of the decline in mortality from heart disease to medical treatments. In addition 51% of the decline in heart disease mortality was attributed to 'risk factor reduction', but no information was provided to indicate if this was the result of medical or non-medical factors.

2.7. Scottish health policy at the start of the 21st century

This chapter has considered the main theories on mortality decline. These fall into two main camps: McKeown who advocated disease prevention by dealing with disease origins, and the pro-medical commentators who argue that clinical medicine is a major contributor to mortality decline. In this section the current direction of health policy in Scotland will be considered to establish whether policy is influenced by McKeown or his critics.

At the start of the 21st century the Scottish Executive defined the main aim of their health policy as one which "*shifts the emphasis away from ill health to [a policy] which focuses much more on prevention and health improvement*" (Scottish Executive, 2003: 12). Thus, disease prevention was made a priority. The blueprint for achieving this was set out in the White Paper *Improving Health in Scotland* (Scottish Office, 1999). In this White Paper echoes of the McKeown thesis can be detected in areas such as disease prevention through environmental and behavioural improvements. The main aim of the White Paper was the prevention of disease, to be achieved by the identification and amelioration of the root causes of ill health. That is, instead of attacking the causes of individual disease, the overall causes of disease were to be tackled. The main causes of ill health were identified as smoking, diet and lack of exercise, and these were linked to poverty and unemployment. A "*sustained attack on inequality, social exclusion and poverty*" (Scottish Office, 1999: 1) was made the overarching aim of the White Paper. Under this plan the medical profession was not considered solely responsible for the improvement of health (or the reduction in mortality). Instead a multi-agency approach, involving national and local government, private, voluntary and community agencies, and individuals was advocated. The plan was divided into three parts which will be discussed below.

Life Circumstances

Poor life circumstances, in the form of poverty and inequality, were identified as a major underlying cause of ill health, by creating an unhealthy environment. The aim of the White Paper was to improve the circumstances in which people live and encourage lifestyle change. Instead of the delivery of medical treatment to improve health, policies such as Working Families Tax Credit, fuel assistance and improved pensions were explicitly identified as measures to improve health. In this way the White Paper mirrors the McKeown thesis's recommendation to create an environment that makes it easier for people to behave in a way that is good for them.

Lifestyles

Under the heading of 'lifestyles' the White Paper emphasised the role of behavioural change in the prevention of disease by reducing the levels of five main risk factors: smoking, poor diet, low levels of physical activity, excessive alcohol consumption and drug misuse. Several initiatives were outlined to achieve this aim, including the use of health education to inform the public and specific strategies designed to reduce unhealthy behaviours. In addition, government regulation and legislation were identified as tools to reduce smoking prevalence. The most notable impact of this has been the Scottish Executive decision to ban smoking in public places from March 2006.

Health Topics

The 'health topics' section of the White Paper dealt with causes of death which had been identified as of specific concern in Scotland: heart disease and cancer. This involved a mixture of approaches to both prevent and treat these conditions. In the case of cancer, the focus was on the development of screening programmes such as that for colo-rectal cancer. For heart disease, the White Paper outlined a combination of NHS treatments aimed to improve the survival of those already ill and a number of community based projects aimed at the prevention of heart disease. These initiatives are discussed further in *Coronary heart disease and stroke: strategy for Scotland* (2002).

Although by the late 1990s policies had been developed which were aimed at disease prevention, the traditional role of medicine in the care and treatment of the sick still remained at the centre of the Scottish health service. In considering the role for the NHS the Scottish Executive identified as priorities: therapeutic and behavioural interventions for those with heart disease, the effective management of high blood pressure, chronic disease management systems for diabetes and asthma, and cancer screening (Scottish Executive, 2003). Furthermore plans for continued investment in established medical areas, such as hospital building were provided (Scottish Executive, 2002).

Thus, although the 'traditional' medical areas remained significant, disease prevention became a priority, by the end of the 20th century. The Scottish direction was mirrored in policies outlined for England and Wales in the 2004 White Paper *Choosing Health.* In all these documents the influence of the McKeown thesis on the importance of disease prevention, by dealing with the origins of disease, is evident.

These policies for health improvement raise a number of issues regarding the role of medicine. When McKeown (1979) considered the influences on mortality prior to 1970, he classified medicine as immunisation and therapy, as well as the work of the sanitary authorities. This definition was challenged by Szreter (1988), who argued that the influence of medicine extended further into the realm of organised human agency, and he identified a wide range of initiatives by the public health authorities under this heading. The policies identified under the 1999 White Paper as for health improvement can be seen as public health measures, but they extend beyond any previous definition of the medical role. Measures such as Working Family Tax Credit and fuel assistance would traditionally have been regarded as linked to standards of living. That is, in the context of the debate over the influence of medicine versus standards of living on mortality, level of income available to a family would typically have been classed as part of the 'standards of living' component. This raises the question of whether initiatives such as these, when explicitly identified in health policy documents, should be considered as medical measures. These issues are discussed further in section 3.3.

Thus, the influence of McKeown's ideas, if not directly cited, can be detected within Scottish health policy documents, with disease prevention having become central to policy aimed to improve health and reduce mortality. However, although disease prevention has come to occupy an increasingly central role in Scottish health policy rhetoric, the reality of spending reveals a different picture, with only 2% of NHS expenditure in 2003/4 being spent on health promotion (ISD, 2004). The vast majority (98%) of the NHS budget was spent on clinical services, in line with the recommendations of Bunker and Mackenbach.

2.8. Conclusions

The literature on mortality decline covers a wide timeframe, and as yet there is no consensus on what would produce the best long term reduction in mortality in Scotland. The majority of commentators agree with McKeown's main point, that medical treatments and care contributed little to the overall decline in mortality between 1838 and 1970. Their criticism is directed towards his methodology and in particular his treatment of the work of public health.

McKeown's ideas have had a lasting impact and can be recognised in the changing role of the medical profession. This has seen a shift since the 1970s from hospital focused, curative services (although these do remain an essential element of health services in Scotland)

towards a health service which is more concerned with the prevention of disease. Concern for disease prevention has seen the development of wide ranging initiatives which are designed to change individuals' behaviour, not only through health education, but by introducing polices designed to tackle underlying causes of ill health such as poverty. In introducing these policies to improve health, the medical profession becomes part of a wide range of bodies, including government and the private and voluntary sector, whose aim is to prevent disease and mortality by improving health.

This model for reducing mortality via prevention has been challenged by the findings of Bunker and Mackenbach and by the results of Capewell *et al*'s (1999) study on heart disease. Their main argument is that it is wrong to base a modern approach to mortality decline on the pre-1970 findings of McKeown, when few effective medical treatments were available. They contend that when only the period post-1950 is considered, medicine, in the form of treatment to cure or prevent disease, has been the most significant influence on mortality decline. However, these studies adopted a one sided approach, looking only at the role which medicine may have played; they do not address the more problematic area of the influence of improving standards of living. As such, they may have overlooked an influential factor in the post-1950 mortality decline.

2.9. Aims and objectives

The aim of this thesis is to establish what have been the main influences on mortality in Scotland since 1950. By establishing what were the most important influences on mortality in the recent past, this will allow recommendations to be made of what will produce the greatest reduction on mortality in the future. If medicine in the form of treatment has been more effective than the McKeown thesis suggests, then continued investment in medical care may be recommended. Alternatively, if improving standards of living were most significant, policies aimed at improving life circumstances may yet yield the greatest results. Although this thesis will look at the long term decline of causes of death to provide context, only the factors influencing post-1950 decline will be measured. This should produce the most accurate picture of what have been the most effective factors influencing the decline of mortality in recent times. The following chapter will describe the framework which has been devised to achieve this.

3. Methodology

3.1. Introduction

This chapter will outline the methodological approach which has been developed for this thesis. The methodology has drawn on the methods of the mortality studies which were discussed in chapter 2. This thesis falls into two main areas. First, the calculation of the causes of death which have contributed the most to the decline of mortality in Scotland between 1950 and 1999. These causes of death will act as case studies and the relative contribution of medicine or standards of living to their decline will be estimated. The development of the methods for achieving these aims will therefore be discussed. The framework for analysing the influences on the decline of diseases selected as case studies will then be described. In addition, the definitions employed by this thesis of 'medicine' and 'standards of living' will be outlined.

3.2. Methodology for manipulating death data

3.2.1. Calculation of death rates

The source of the data on deaths in Scotland are the Registrar General's Death Returns for Scotland. These data were acquired from the General Registrar's Office for Scotland (GROS). The process of sampling and digitising these data is described in section 4.7. In order to generate a list of the causes of death which contributed the most to the Scottish mortality decline, the first task was the generation of death rates. In order to generate accurate death rates it was decided to standardise the death rates to the 1950 population. Unstandardised, the death rates for Scotland appear to show a morality rate which has undergone very little decline, especially amongst the female population. This does not portray a true picture but is rather an artefact of the ageing population. The method adopted by this thesis was direct standardisation, which takes into account the ageing of the population.

In order to standardise death rates, a number of different sources of information are utilised: these are population by age band and year, deaths by age and year, and the percentage of the population in each age band by year. The process for calculating standardised death rates used in this thesis is shown in box 3.1.

Box 3.1. Steps involved in the calculation of standardised death rates

1. Calculation of unstandardised death rates per 1000.

X=(d/p)1000

Where X is the unstandardised death rate, d is the number of deaths in each age group and p is the population in each age group.

2. Calculations to generate standardised death rates per 1000.

X=(dp)summed

Where X is the standardised death rate, d is the unstandardised death rate by age group in 1999 and p is the percentage of the population by age group in 1950.

3.3.2. Calculation of the relative contribution of selected causes of death to the overall decline in mortality

Previous researchers on mortality decline have used a number of different methods to measure the relative contribution of different causes of death to mortality decline. McKeown calculated the contribution of different causes to the decline in mortality between 1838-1854 and 1971 using death rates which were standardised to the 1901 population. Both Bunker and Mackenbach measured improvements in mortality in terms of gains made to life expectancy. Finally Capewell *et al* (1999) used a method which estimated the number of deaths which were avoided, or the potential number of lives which were saved, comparing the final year of their study with the base year. Each of these methods is a valid way to measure the decline in mortality. This thesis has adopted an expanded version of the method employed by Capewell *et al.*

Capewell *et al* used the calculation of potential lives saved to estimate the number of deaths from heart disease which had been avoided in 1994, compared to 1975. This process is described in section 2.7.3. It required the calculation of the death rates in 1975 for each 5 year age group. These rates were then applied to the population within each age band in 1994, to give the number of deaths which would have been expected in 1994 if the death rate had remained the same. The actual number of heart disease deaths for 1994 were then subtracted, to give the number of potential lives saved. For this thesis this calculation will be undertaken for all-cause mortality and for the causes of death which are selected as case

studies. This will provide the total number of potential lives saved in 1999 compared with 1950, as well as the number of potential lives saved attributable to each case study disease. The percentage contribution of each case study disease to overall potential lives saved will then be calculated. This process is described in more detail in section 5.2.

The decision to use the calculation of potential lives saved as opposed to another method, such as gains in life expectancy was based on a number of factors. These include the fact that this thesis is interested in different causes of death, and comparing cause specific death rates over time provides a more meaningful and easily graspable concept than trying to isolate the contribution of death from different causes to a global life expectancy figure. In addition, the use of life expectancy 'from birth' can lead to an over representation of deaths in younger age groups.

3.3. Methodology for the analysis of the contribution of medicine and standards of living to the decline of mortality from case study causes of death

Two main approaches were used in the mortality studies described in chapter two to measure the relative contribution of medicine and standards of living to mortality decline. The pros and cons of these methods were considered in the development of an approach for this thesis. These are discussed below.

Both Bunker's (1995, 2001) and Capewell *et al*'s (1999) method of measuring the contribution of medicine to mortality decline was based upon a formula for statistical estimation. This involved using data from meta-analyses and clinical trials to acquire figures for the effectiveness of specific treatments in individuals receiving them. Calculations were then performed using data on population at risk and the percentage of that population in receipt of that treatment (this process is described in greater depth in sections 2.6.1 and 2.6.3). This provided an estimate of the overall benefit which a treatment may have at population level. Capewell *et al* (1999) also used equations to calculate a numerical value for the impact of risk factor reduction (see section 2.6.3). For example, in the case of smoking, data were available on the decline of smoking prevalence between 1975 and 1994, whilst studies indicated the reduction in risk of death from ischemic heart disease associated with smoking cessation, allowing an estimate to be made of the benefit which this risk factor reduction had on population level mortality.

Both the approaches described have the benefit of providing relatively robust results which allow quantification of the contribution of specific medical measures and risk factors reductions to population level mortality. The use of some of the data sources employed in these calculations have received criticism, in particular the use of treatment effectiveness data from clinical trials to estimate the benefit of a treatment in routine practice. The administering of treatments in clinical trials requires special circumstances, with high levels of surveillance on patient compliance. This is not the case in routine practice and may lead to an over estimate of the benefit of the treatment at population level (Hart 2001). With reference to this thesis the approaches of Bunker and Capewell *et al* are also problematic for a number of other reasons. In order to perform these calculations, good quality data are required from a number of different sources. Although these were available for Capewell *et al* (1999) in their study of heart disease, this is unlikely to be the case for all the case study diseases covered by this thesis. Effectiveness data on treatment from clinical trials are only available for those treatments which became available in the latter quarter in the 20th century, making it impossible to calculate the benefit of treatments which may have become available in the earlier part of the century.

Another problem with the approaches used by Bunker and Capewell *et al* is that they do not provide a method for measuring the impact of improving standards of living. Capewell *et al*'s (1999) study does quantify the effect of risk factor reduction, but they do not offer an explanation as to why the risk factor may have declined, failing to ask whether it was due to medicine or standards of living. In addition, there is no means of quantifying improving standards of living which is valid across different causes of death. Standards of living encompass a number of different elements, such as access to a good quality diet and adequate housing, and different aspects of standards of living will affect risk differently according to the cause of death being examined. Therefore, a more flexible approach is required when considering the effect of standards of living which takes into account all the different factors which may influence it. In this regard McKeown's methodology for considering the impact of improving standards of living may be more appropriate. McKeown examined the decline of the infections within their historical context, and considered what happened to different facets of standards of living relevant to each disease under investigation. For example, in the case of tuberculosis he focused on changes in dietary trends, whilst for water borne infections he considered access to clean water. McKeown has, however, been criticised for failing to provide adequate evidence of

improving standards of living. In addition, McKeown's approach to measuring the impact of medicine to mortality decline has also been criticised (see section 2.4.4).

Taken individually, none of the methodologies used in existing studies of mortality decline is appropriate in its entirety for the purpose of this thesis, that is to estimate the relative contribution of both medicine and standards of living to mortality decline. Therefore, an alternative method has been devised which incorporates the most appropriate elements of the methods discussed above and is intended to avoid their pitfalls.

This thesis will adopt a case study approach to the measurement of medicine and standards of living to mortality decline. This differs from the method employed by both Bunker and Mackenbach, who first identified effective medical treatments, then calculated the impact which these would have had on life expectancy. The use of a case study approach is comparable to that taken by McKeown. He first identified the main infectious disease groups which had declined, then investigated what caused their decline. One drawback of the use of case studies is that influences on the total decline on mortality cannot be measured, as by selecting case study diseases this means than certain causes which also contributed to the decline in mortality will be excluded from the analysis. However, the case studies which will be selected for this thesis are the causes of death which contributed the most to the overall decline in mortality, therefore this will allow the majority of the decline to be covered. In addition, the selection of case studies will allow a detailed examination of *all* influences on the decline of that cause, not just one set of influences such as medical treatments.

For each case study disease the first aspect to be considered is the main reason why it declined. There are two ways in which mortality can be reduced: by an increase in the number of people who recover from a disease once they are ill (increasing survival), and by a reduction in the number of people who become ill in the first place (declining incidence). Within this context the relative contribution of medicine and standards of living will be considered. In the case of medicine, a mixture of methods and sources will be utilised. First, the pattern of mortality decline will be placed within its historical context to identify when the disease first began to decline. Then, through a review of the medical literature, any treatments or preventive measures which were introduced will be considered to determine if these coincided with the start of the mortality decline. Where available, data on effectiveness will be sought from clinical trials and meta-analysis. Measuring the

contribution of standards of living to the decline of mortality is more problematic. This is largely due to problems in achieving a satisfactory operational definition of 'standards of living'. In order to address this, for each case study disease the main risk factors will be established from the literature. Once these are known, it will then be possible to state what factors would have influenced their prevalence, facilitating a discussion of whether they may have influenced the decline of mortality. A number of different sources will be used to measure the different areas of standards of living, including data from the National Food Survey, Social Trends and the secondary literature. One way in which this thesis will measure standards of living is through the use of socio-economic class, to provide an indicator of differing standards of living between groups, and across time. However, this thesis is aware that socio-economic group is not only defined by standard of living, but is influenced by other factors including cultural values and local physical environment. To help the researcher, and the reader, a framework for each case study disease has been adopted.

3.4. Framework for the case studies

The framework within which each case study disease will be considered is shown below. The headings of each sub-section are displayed with a brief explanation of the purpose of each. This is not a rigid framework and will be adapted so that it is appropriate to each disease being considered.

Introduction

This section will introduce the chapter and also offer a justification for why the disease being reviewed has been chosen as a case study.

Background

A description of the disease will be provided, including; age groups primarily effected, clinical description, signs and symptoms and disease risk factors.

Mortality trends

In this section the reliability of the original death data will be considered. Following this, standardised death rates for the disease will be examined. The purpose of this is to isolate when the case study disease first began to decline. If the disease began to decline prior to 1950, or if the post-1950 pattern of mortality suggests the continuation of a trend established prior to that date, the earlier pattern of mortality will also will considered. This will involve

the digitising of earlier years from the Registrar General's Death Returns where appropriate. The date at which the mortality decline began is significant as this will dictate the way in which the analysis of each case study is structured. For each case study disease the main division in the framework will be the relative contribution of increasing survival or declining incidence to the overall decline in mortality. However, the way in which the analysis is divided will depend on the date of the start of the morality decline. For example if a disease had undergone a period of increase before a pattern of decline was established, then the framework will be subdivided to consider whether mortality in the earlier period of increase was influenced by reduced survival or increasing incidence. Consideration of the earlier influences on mortality will allow the later decline of the disease to be placed in its historical context. Therefore, in some instances a case study disease chapter may be subdivided into two or more historical periods, and within these the relative contribution of survival and incidence to the increase, or decrease in mortality will be considered.

Increasing survival

For each case study the extent to which there was an increase in survival amongst those with the disease will be established. A number of different sources will be used for this depending on the disease being considered. For example in the case of cancers, survival data are available from NHS Scotland. Where reliable statistics on survival are not available, an estimate will be based on the secondary literature. Once this has been achieved the relative contribution of medicine and standards of living to increasing (or decreasing) survival rates will be considered using the methods described in section 3.3.

Disease incidence

The extent to which mortality from a disease has been influenced by a reduction in the number of people who have become ill will be established. Again, a number of different sources will be employed to achieve this depending on the disease being examined. These include incidence data derived from NHS Scotland, notification data in the case of infections and the secondary literature. The relative contribution of medicine and standards of living to disease incidence will be considered using the methods discussed in section 3.3.

Conclusion

Each case study disease chapter will have its own conclusion offering an assessment of the relative contribution of medicine and standards of living to the overall decline in mortality from that disease.

3.5. Thesis definitions of medicine and standards of living

One of the main issues to emerge out of the review of existing literature on mortality decline is the importance of differentiating reliably between what constitutes a medical influence or a consequence of improving standards of living. The way in which these concepts are defined can significantly alter the findings of a study in favour of one influence or the other. This is reflected in the criticism which was directed at the definition of medical influences in the McKeown thesis. McKeown (1979) defined medical influences rigidly as immunisation and therapy; the only non-clinical medical influence which he identified was the work of the sanitary authorities. Szreter (1988) argued that by failing to consider the wider role which medicine may have played through the work of the public health authorities much of the influence of medicine on mortality decline was excluded from McKeown's analysis. Although McKeown argued that the main influence on the mortality decline up to 1971 was improving standards of living, and in particular diet, he did not provide a comprehensive definition of what is included under the heading 'standards of living'; instead this became a residual category. In addition he failed to produce adequate evidence that standards of living were improving. In the case of Bunker (1995, 2001) and Mackenbach (1988, 1996), neither attempted to define or measure standards of living. Bunker's definition of medicine was quite rigid, including clinical prevention and clinical treatment. Capewell *et al* (1999) provided examples of all the treatments which encompassed medical treatments for IHD. However, in their analysis of risk factor reduction they did not provide an explanation of the relative contribution of medicine and standards of living to their decline.

None of the above 'definitions' (or lack of definitions) of medicine and standards of living are appropriate for use in this thesis. In many cases the definitions are either too narrow, meaning that many influences are excluded from a category, or they are too vague, such as McKeown's standards of living category. In order to avoid these criticism this thesis has devised a three category system for distinguishing between the influences on mortality. The use of three categories instead of two, should allow the wider influences of medicine to be accurately identified. These categories are described below (see box 3.2). If an influence

falls within group 1 or 2 it will be defined as a 'medical' influence and if it falls into group 3 it will be defined as 'standards of living' influence.

Box 3.2. Definitions of influences on mortality decline

Group 1. Medical - interventions conceived and delivered by health care professionals This group encompasses all events where a health care professional delivers a service or product for the use of an individual patient. This group could be compared to McKeown's medical measures group. Inclusion within this group does not necessarily denote an interaction with a doctor but rather includes all interactions with health care providers at an individual level which may help reduce mortality (e.g. a dietician). Examples include surgery, therapy, immunisation and vaccination; maternal and infant health services.
Group 2. Medically influenced - interventions demonstrably informed by medical knowledge, but which may be conceived and/or delivered by other agencies This group is designed to include those interventions which have been influenced by medicine but do not involve the interaction of a health care professional with an individual; rather, they are delivered at a population or group level. It is intended that this group should cover those areas which McKeown has been accused of ignoring – that is, public health and health education. The criterion for inclusion in this group is that there is evidence to show that improving health was a priority when specific interventions were introduced. Examples include: health education work utilising medical knowledge; advice about the dangers of smoking; road safety regulation; heating allowances for the elderly.
Group 3. Standards of living - developments which have a secondary impact on health, although they were created outside the medical sphere, and did not prioritise health This includes all developments which have had a positive impact on health, but for which there is no evidence that improving health was a consideration in their delivery or inception. This category can be seen as roughly comparable to McKeown's standards of living. Examples which may appear in this group include improvements in diet brought about by rising real wages or the greater availability of healthy food produce; improvements in housing conditions; improvements in working conditions; higher levels of education; reduction in family size.

3.6. Conclusion

This chapter has outlined the approach which this thesis will take to the two main aims of this thesis: the identification of the diseases which contributed the most to the decline of mortality in Scotland between 1950 and 1999, and the relative contribution of medicine and standards of living to the decline of these diseases. The next two chapters will deal with the first of these aims. These will describe the problems associated with the Registrar General's Death Returns and the process by which they were digitised. In addition, the selection process for the case study diseases will be outlined. Subsequent chapters will consider individually the influences on the decline of these diseases.

4. Identifying causes of death contributing to the decline of mortality – data issues

4.1. Introduction

If the relative contribution of medicine and standards of living to the decline of mortality in Scotland is to be considered, the causes of death which contributed the most to the decline must first be isolated. This chapter will outline the process involved in generating this list of causes of death, including the data source employed and problems inherent to these data. The method of sampling used to create an original shortlist of causes of death will be discussed. This list will then be refined to create a list of six causes of death. The contribution of each of these causes to the decline in mortality will then be considered in chapter five.

4.2. Data acquisition

The source used by this thesis to measure the mortality decline is the same as that employed by McKeown, namely the Registrar General's Death Returns. These were available from the General Registrar's Office for Scotland (GROS) in paper format. The years 1950-1999 were acquired initially and this was expanded back to 1911 as the range of the study increased. This source includes every death registered in Scotland tabulated by: year, cause, sex and age. A sample page is displayed in figure 4.1. Each cause of death is coded to a specific number, known as a *rubic*, and deaths are grouped into sections, known as chapters, according to organ systems (see box 4.1). In addition to the death returns, population data for Scotland dating back to 1911 were also acquired from the GROS. These were available in a digital format and were broken down by sex and age group for each year.

4.3. Problems relating to data source

The death returns for Scotland represent a unique and invaluable source to any researcher investigating mortality trends in Scotland. However, there are a number of problems which must be addressed when using this data. Firstly, for the majority of the time period covered by this thesis, the death returns were only available in paper format. This meant that any cause of death which this study wished to investigate had first to be identified and traced through time using the paper records, before the appropriate figures could be entered manually into an Excel spreadsheet. Given the volume of material contained in the death returns, entering *all* the data for every cause of death, for every year, was impossible to

complete within the constraints of a PhD study. Therefore, a process of sampling was devised to isolate a shortlist of causes of death which could then be digitised in their entirety.

Secondly, before digitisation could be attempted, a number of other areas of concern relating to the reliability of the data had first to be addressed. The first of these was the implications of cause of death coding change for the continuity of cause of death categories and the second was the accuracy of death certification data. These areas of concern will be discussed and any implications which they may have on this thesis will be considered.

Figure 4.1. Sample page from Scottish death returns.

Table 6.4 Deaths, by sex, age and cause, Scotland, 1998

NOTE: Where a cause of death is omitted there were no events registered

ICD Nos.	Cause of death		All ages	Age group 0	1-4	5-	10-	15-	20-	25-	30-	35-	40-	45-	50-	55-	60-	65-	70-	75-	80-	85+
	All causes	**All**	**59,164**	**320**	**63**	**62**	**58**	**139**	**268**	**334**	**388**	**543**	**785**	**1,125**	**1,777**	**2,433**	**3,811**	**5,777**	**7,924**	**9,612**	**8,886**	**14,809**
		Males	**28,132**	**183**	**37**	**34**	**39**	**134**	**200**	**247**	**277**	**355**	**488**	**676**	**1,120**	**1,492**	**2,336**	**3,363**	**4,383**	**4,828**	**3,757**	**4,183**
		Females	**31,032**	**137**	**26**	**28**	**19**	**55**	**68**	**87**	**111**	**188**	**297**	**449**	**657**	**941**	**1,475**	**2,414**	**3,541**	**4,784**	**5,129**	**10,626**
001–139	**I. INFECTIOUS AND PARASITIC**	**M**	**248**	**3**	**6**	**1**	**1**	**1**	**-**	**1**	**9**	**13**	**11**	**4**	**14**	**17**	**14**	**26**	**27**	**31**	**30**	**39**
		F	**236**	**1**	**7**	**2**	**1**	**4**	**2**	**4**	**4**	**5**	**7**	**5**	**4**	**5**	**17**	**15**	**27**	**31**	**34**	**83**
001–009	**Intestinal infectious diseases**	**M**	**12**	-	-	-	-	-	-	-	-	-	-	-	-	-	**2**	**4**	**1**	**1**	**2**	**2**
		F	**22**	**1**	-	-	-	**1**	-	-	-	-	-	-	-	**1**	-	**3**	**1**	**4**	**3**	**8**
003	Other salmonella infections	M	5	-	-	-	-	-	-	-	-	-	-	-	-	-	1	3	-	1	-	-
		F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
008	Intestinal infections due to other organisms	M	5	-	-	-	-	-	-	-	-	-	-	-	-	-	1	1	-	-	2	1
		F	18	-	-	-	-	-	-	-	-	-	-	-	-	-	-	3	1	4	3	7
009	Ill-defined intestinal infections	M	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	1
		F	4	1	-	-	-	1	-	-	-	-	-	-	-	1	-	-	-	-	-	1
010–018	**Tuberculosis**	**M**	**27**	-	-	-	-	-	-	-	-	1	**1**	-	**2**	**2**	**1**	**4**	**3**	**5**	**5**	**3**
		F	**19**	-	-	-	-	-	-	-	-	-	**1**	-	-	-	**1**	**3**	**4**	**6**	**2**	**2**
011	Pulmonary tuberculosis	M	21	-	-	-	-	-	-	-	-	1	-	-	1	2	1	3	3	5	2	3
		F	14	-	-	-	-	-	-	-	-	-	1	-	-	-	-	3	2	4	2	2
012	Other respiratory tuberculosis	M	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-
		F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
013	Tuberculosis of meninges and central nervous system	M	1	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-
		F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
014	Tuberculosis of intestines, peritoneum and mesenteric glands	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
		F	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	2	-	-
015	Tuberculosis of bones and joints	M	2	-	-	-	-	-	-	-	-	-	-	-	1	-	-	1	-	-	-	-
		F	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-
018	Miliary tuberculosis	M	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	-
		F	1	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-
020–027	**Zoonotic bacterial diseases**	**M**	**2**	-	-	-	-	-	-	-	-	-	-	-	-	**1**	-	-	-	**1**	-	-
		F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
027	Other zoonotic bacterial diseases	M	2	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	1	-	-
		F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
030–041	**Other bacterial diseases**	**M**	**133**	**2**	**5**	**-**	**1**	**1**	**-**	**-**	**2**	**-**	**3**	**1**	**4**	**10**	**7**	**12**	**17**	**20**	**18**	**29**
		F	**150**	**-**	**5**	**1**	**1**	**3**	**2**	**2**	**-**	**2**	**3**	**2**	**3**	**3**	**11**	**8**	**16**	**19**	**23**	**45**
031	Diseases due to other mycobacteria	M	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
		F	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1	-	-
036	Meningococcal infection	M	8	-	6	-	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-
		F	12	-	3	-	1	3	2	-	-	-	-	-	-	-	-	-	2	1	-	-
038	Septicaemia	M	116	2	-	-	-	-	-	-	2	-	3	1	4	10	7	10	16	19	17	25
		F	129	-	2	1	-	-	-	2	-	2	2	2	1	3	11	7	13	16	23	44
040	Other bacterial diseases	M	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	1
		F	1	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-
041	Bacterial infection in conditions classified elsewhere and of unspecified site	M	6	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	-	1	1	2
		F	6	-	1	-	-	-	-	-	-	-	-	-	2	-	-	-	1	1	-	1
042–044	**HIV infection**	**M**	**25**	-	-	-	-	-	-	**1**	**5**	**9**	**4**	**1**	**3**	**1**	**1**	-	-	-	-	-
		F	**7**	-	-	-	-	-	-	**2**	**2**	**3**	-	-	-	-	-	-	-	-	-	-
042	Human immunodeficiency virus infection with specified conditions	M	22	-	-	-	-	-	-	1	5	7	4	-	3	1	1	-	-	-	-	-
		F	6	-	-	-	-	-	-	1	2	3	-	-	-	-	-	-	-	-	-	-
044	Other human immunodeficiency virus infection	M	3	-	-	-	-	-	-	-	-	2	-	1	-	-	-	-	-	-	-	-
		F	1	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-
045–049	**Poliomyelitis and other non-arthropod-borne viral diseases of central nervous system**	**M**	**6**	-	-	-	-	-	-	-	-	**2**	-	-	**1**	**1**	-	-	-	-	**1**	**1**
		F	**3**	-	-	-	-	-	-	-	-	-	-	-	-	-	**1**	-	**1**	**1**	-	-
046	Slow virus infection of central nervous system	M	4	-	-	-	-	-	-	-	-	2	-	-	1	1	-	-	-	-	-	-
		F	2	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1	-	-	-
047	Meningitis due to enterovirus	M	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	1
		F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
049	Other non-arthropod-borne viral diseases of central nervous system	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
		F	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-
050–057	**Viral diseases accompanied by exanthem**	**M**	**6**	**1**	-	**1**	-	-	-	-	**1**	-	-	-	**1**	-	**1**	-	-	**1**	-	-
		F	**9**	-	-	**1**	-	-	-	-	**1**	-	-	-	-	-	**1**	-	**1**	-	**1**	**4**
052	Chickenpox	M	2	-	-	1	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-
		F	1	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
053	Herpes zoster	M	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-
		F	5	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	1	3
054	Herpes simplex	M	3	1	-	-	-	-	-	-	-	-	-	-	1	-	1	-	-	-	-	-
		F	3	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1	-	-	1
070–079	**Other diseases due to viruses and chlamydiae**	**M**	**10**	-	-	-	-	-	-	-	**1**	-	**1**	**2**	**2**	-	**1**	**1**	-	-	**2**	-
		F	**16**	-	**1**	-	-	-	-	-	**1**	-	**3**	**2**	-	**1**	**2**	-	**2**	-	**1**	**3**
070	Viral hepatitis	M	10	-	-	-	-	-	-	-	1	-	1	2	2	-	1	1	-	-	2	-
		F	12	-	-	-	-	-	-	-	1	-	3	2	-	1	2	-	2	-	1	-

Box 4.1. International Classification of Disease chapter headings

I.	Infective and parasitic
II.	Neoplasms
III.	Endocrine system, metabolic and nutritional diseases and immunity diseases
IV.	Diseases of the blood and blood-forming organs
V.	Mental disorders
VI.	Diseases of the nervous system and sense organs
VII.	Diseases of the circulatory system
VIII.	Diseases of the respiratory system
IX.	Diseases of the digestive system
X.	Diseases of the genito-urinary system
XI.	Complications of pregnancy, child-birth and the puerperium
XII.	Diseases of the skin and cellular tissue
XIII.	Diseases of the musculoskeletal system and connective tissue
XIV.	Congenital anomalies
XV.	Certain conditions originating in the perinatal period
XVI.	Symptoms, signs and ill-defined conditions
EXVII.	Supplementary classification of external causes of injury and poisoning

4.4. Death registration and the International Classification of Causes of Death (ICD)

4.4.1. Background

Forms of death registration have existed in the UK for centuries, starting with registration during times of crisis, such as plague years. From the mid 18th century Parish Registers began to record deaths (Alter and Carmichael, 1999). The registration of deaths by cause developed from a system devised by William Farr, who classified deaths under headings which included: epidemic, endemic and contagious diseases, constitutional diseases, diseases of the different organ systems and deaths from external causes. It was according to this model that deaths were coded when the compulsory registration of deaths in Scotland was introduced under the Registration of Births, Marriages and Deaths Act 1855 (Hardy, 1994). In the interests of international comparability, ICD, which is based on Farr's original

classification system, was developed at the end of the 19th century. Scotland adopted this system during its second revision in 1911. Under this system every death must be certified by a doctor and a copy of this certificate sent to the GROS where it is coded and compiled in the death returns which are then published annually. As the use of ICD developed, the World Health Organisation (WHO) regulated the system of coding and published guidelines under which specific causes of death on death certificates are translated into ICD codes (WHO, 1967, WHO, 1977).

4.4.2. Problems with ICD coding

One of the main problems presented to this study is the revisions of ICD codes which take place approximately every decade (see box 4.2). The purpose of this is to take into account medical advances in disease identification and aetiology, and changes in medical terminology. Since the death returns were acquired in paper format any cause of death which this study wished to investigate had to be traced through time and across coding changes, before being digitised (see appendix 1). Any unobserved change in the code relating to a specific cause can lead to an inaccurate recording of deaths from that cause. In addition, any alteration in the instruction as to how to code a cause of death from a death certificate to an ICD code can lead to a miscalculation in the number of deaths attributed to a certain cause over time, as deaths are assigned to a variety of different causes.

Box 4.2. Revisions of International Classification of Causes of Death

1911-1920	2nd revision
1921-1930	3rd revision
1931-1940	4th revision
1941-1949	5th revision
1950-1957	6th revision
1958-1967	7th revision
1968-1978	8th revision
1979-1999	9th revision

For most causes of death the introduction of a new version of ICD has little impact on the number of deaths attributed to that cause. However, in a few instances the effect can be large. This can be illustrated by the case of cerebrovascular disease (stroke). In 1967, under ICD 7, Chapter 6 of the death returns, Diseases of the Nervous System and Sense organs

accounted for 4377 male deaths and 5933 female deaths (or 14% of male and 20% of female total mortality). By 1968 the number of deaths attributed to Chapter 6 of the death returns had decreased to 330 male deaths and 346 female deaths (or roughly 1% of all male and female mortality). This was not caused by a dramatic decline in real deaths, but was rather an artefact of the introduction of ICD 8 under which codes for cerebrovascular disease had been changed. As the understanding of stroke increased, it had become apparent that the disease no longer belonged in the category for Diseases of the Nervous System, where it had occupied codes 330-334, but was instead a circulatory disease. Therefore, since ICD 8 it has been represented by codes 430-438 under Chapter 7, Diseases of the Circulatory System.

The effect of ICD coding change on cerebrovascular disease is one of the most well known and obvious examples. However, the problem for this study arises when trends in cause of death are being traced through time and coding change has had a smaller effect, especially when the code for a disease remains the same but the instructions to coders may have changed. This means that deaths which may previously have been allocated to that code are now allocated elsewhere, or vice versa. This can lead to a false decline in a cause of death being observed, when in fact the decline has been caused by deaths being recorded elsewhere. This was the case with chronic obstructive pulmonary disease and will be discussed in section 5.3.2.

4.4.3. Solutions to ICD revisions

The effect of changes in ICD can be reduced by referring to the Registrar General's Annual Reports for the years when a new version has been introduced. These reports contain 'transference tables' where deaths are coded according to both the old and new versions of ICD, indicating any discrepancy which may have occurred in the number of deaths coded to a certain cause. In addition, any change in the ICD code relating to a cause of death is also detailed. These tables were consulted for all the causes of death considered in this thesis, thus minimising any influence which coding change might have had on the accuracy of results.

4.5. Death certification

The second factor which may influence the accuracy of the death data for specific causes is death certification. The death returns are based on the cause of death recorded by doctors on death certificates. Therefore any inaccuracy in these affects the integrity of the death returns. Concerns regarding the accuracy of information contained within death certificates

have been raised over several decades. These were stated in the Brodrick Report (1971) which reviewed legislation concerning the certification, registration and disposal of the dead. These issues were revisited by a working party set up in 1980 to review death certification (Joint Report of the Royal College of Physicians and the Royal College of Pathologists, 1982). Criticism of certification has fallen into two main areas.

4.5.1. Inaccurate completion of death certificates

It is the legal duty of the doctor who attends the patient during their last illness to complete a death certificate. In Scotland the death certificate is known as Form 11, a copy of which is shown in figure 4.2. Detailed instructions are available, in line with WHO recommendations, to advise doctors on the proper procedure for completing the form. For the purposes of ICD coding the 'underlying' cause is the cause which is listed in the death returns, and this should appear in the last line of Part 1 of the death certificate, while the doctor can include unrelated but contributory causes in Part 2. The 'underlying' cause of death is defined as:

> *"a. the disease or injury which initiated the train of morbid events leading directly to death, or*
> *b. the circumstances of the accident or violence which produced the injury"* (GROS, 2000).

Figure 4.2. Medical certificate of cause of death

Figure A2.1: Extract from Scottish medical certificate of cause of death

Cause of death

I hereby certify to the best of my knowledge and belief, the cause if death was as stated below:

Approximate interval between onset and death

			Years	Months	Days
I Disease or condition directly leading to death*	(a) ..... *due to (or as a consequence of)*				
Antecedent causes Morbid conditions, if any, giving rise to the above cause, stating the underlying condition last	(b) ..... *due to (or as a consequence of)*				
	(c) ..... *due to (or as a consequence of)*				
	(d) ..... *due to (or as a consequence of)*				
II Other significant conditions contributing to the death, but not related to the disease or condition causing it	.....				

* This does not mean mode of dying, such as heart failure or respiratory failure; it means the disease, injury or complication that caused death.

The Joint Report of the Royal Colleges (1982) identified problems with the way in which this procedure was carried out. These include only listing the mode of death, for example heart failure, rather than the underlying cause. The use of abbreviations and lack of detailed information on the forms were also criticised. These inaccuracies can lead to the wrong cause of death being coded. The Joint Report attributed the poor completion of death certificates to a general lack of understanding amongst the medical profession of the importance of certification, and the way in which the cause listed on the death certificate is allocated an ICD code. The large proportion of death certificates completed by junior doctors reflected the low priority which was attached to this task. Resistance to diagnosing unnatural causes, such as alcoholism or suicide, which would require the intervention of the Procurator Fiscal (Scotland's independent public prosecution and deaths investigation service) was also identified.

4.5.2. Failure to diagnose correct cause of death

Incorrect diagnosis was identified as a significant problem in death certificate accuracy (Cameron and McGoogan, 1981a, Busuttil *et al*, 1981). In the majority of cases, the cause which is listed on the death certificate is determined by clinical signs before death. However, the only way in which a diagnosis can be confirmed is by an autopsy. The certifying doctor can indicate on the death certificate if an autopsy will be carried out. In such cases the GROS sends out a form for amendment of cause of death (SM2) which allows the results of the autopsy to be included on the final death certificate, and as such influences the ICD code assigned to the deceased. However, the extent to which this procedure is carried out has been questioned (Busuttil *et al*, 1981). In addition, an autopsy is only carried out in about 25% - 35% of hospital deaths, with a lower figure for those dying at home (Cameron and McGoogan, 1981a, Sington and Cottrell, 2002).

The extent to which misdiagnosis of the 'underlying' cause of death on the death certificate occurs has been considered by a number of studies which have compared autopsy findings with the cause originally recorded on the death certificate. These studies investigated hospital deaths and inaccuracy was found as a result of both under- and over-diagnosis of conditions. In their study of autopsies in South Lothian, Cameron and McGoogan (1981a) found that 22% of cases had been wrongly diagnosed, while Busuttil (1981) examined autopsy findings in North Lothian and found that 20% of cases had been assigned the wrong underlying cause. These studies found that misdiagnosis increased with age, as the elderly are more likely to be suffering from several conditions at the time of death making the

isolation of the 'underlying' cause more difficult. In addition, levels of accuracy were found to vary widely amongst different causes. The conclusion which can be drawn from these studies is that roughly 20% of all deaths recorded in the death returns have been assigned to the wrong ICD code.

4.5.3. Recommendations of the Joint Report of the Royal Colleges and their influence on death certificate accuracy

The Royal Colleges issued a number of recommendations in their 1982 report which were aimed at improving the accuracy of certification. These focused on educating the medical profession to increase understanding of the value of death data, and the need for accuracy when recording cause of death. Recommendations included the following:

a. that the instruction of the proper procedure for the completion of death certificates should become part of undergraduate education in all medical schools,
b. that recently qualified doctors receive training in accurate certification of cause of death,
c. that postgraduate instruction be provided for hospital doctors and General Practitioners on the role and value of accurate death certification
(Joint Report of the Royal College of Physicians and the Royal College of Pathologists, 1982).

The extent to which the recommendations of the Royal Colleges have improved accuracy since the 1980s is uncertain. A review of death certification in 1996 found that many of the earlier problems identified by the Royal Colleges still existed, and that most of the Royal College's recommendations had not been carried out (Maudsley and Williams, 1996). These findings were confirmed by a 2002 study which looked at the quality of death certification at a large teaching hospital and found that 45% of certificates contained faults. These included the use of terms like old age, the provision of inadequate information, illogical sequence or the use of abbreviations (Swift and West, 2002). Not all of these faults would have led to the assignment of an incorrect code but they do indicate the continuation of improper death certification.

4.6. Implications of ICD coding change and death certificate accuracy on the reliability of death data

The literature concerning the reliability of death data indicates that they are a source which should be treated with caution. However, this is the only source which provides details of every death in Scotland dating back to 1855 with an associated cause of death. The problems associated with this source cannot be entirely avoided and it is important to consider their likely impact on results. In respect of the revisions of ICD codes and the effect which this has on tracing causes of death through time, consultation of the transference tables contained in the Annual Reports should reduce any inaccuracy resulting from changes in coding and practice. The inaccuracy rate of approximately 20% resulting from wrong diagnosis on a death certificate is more problematic. One possible way in which the influence of wrong diagnosis can be reduced is by considering the extent to which this may have occurred in each of the causes of death which this thesis has selected as case studies. The reliability of death certification will be considered in each of the case study chapters under the heading 'mortality trends'.

4.7. Cause of death sampling

It has already been noted that the death returns were only available in a paper format and that the digitising of all the returns between 1950 and 1999 was not practical. Therefore, a system of sampling was devised which took into account the problems with the data which have been outlined above. A number of stages were involved in the sampling process, the first of which was the isolation of the causes of death which contributed the most to total mortality. This allowed a basic analysis to be performed to indicate which of these causes had declined over the time period.

4.7.1. Sampling procedure

The first step in the sampling process was to isolate those causes of death which contributed the most to mortality in 1950. This was done by reviewing all causes of death in 1950 and selecting those which either accounted for 1% or more of total male or female mortality, or which discussion during supervision had indicated may be of particular interest (indicated by a star in table 4.1). This generated a list of 17 male and 17 female causes which accounted for 70.2% of all male, and 70.5% of all female mortality (table 4.1). The total number of deaths for each of these causes was then identified in six sample years (1950, 1958, 1968,1979,1988, 1999) and entered into a spreadsheet (Microsoft Excel). The relevant ICD

codes for these causes over time were acquired from the transference tables in the Registrar General's Annual Reports and are available in appendix 1.

Table 4.1. Percentage of total deaths attributable to selected causes in 1950

Cause of death	Male	Female
Accidental Falls	0.8	1.2
Breast Cancer		2.6
Cerebrovascular Disease	11.3	16
Chronic Obstructive Pulmonary Disease	4.2	2.3
Colon Cancer	1.9	2.4
Conditions originating in the perinatal period (infancy)	3.3	2.3
Congenital Anomalies	1.0	0.9
Diabetes Mellitus	0.5	1.3
Hyperplasia of the Prostate	1.6	
Ischemic Heart Disease	27.7	27.5
Lung. Trachea and Bronchus Cancer	2.9	0.8
Ovarian Cancer		0.7
Pneumonia	3.5	3.2
Prostate Cancer	1.0	
Road Traffic Vehicle Accidents*	0.8	0.4
Senility	1.5	2.0
Stomach Cancer	3	2.8
Suicide*	0.5	0.3
Tuberculosis	4.7	4.1

Source: Original analysis of GROS data.

4.7.2. Selection process for sampled causes of death

Data for 17 causes of death, for men and women for every year, represented a considerable volume of data which could not be entered in its entirety into Excel. Therefore the list of 17 of causes of death was further refined to a shortlist of 6. In order to facilitate this, the death rates for each of the causes of death were graphed for the sample years, to indicate which of the causes had experienced a decline of mortality. The death rates for the 17 causes of death were not standardised to the 1950 population as this would have required digitising all deaths by age for these causes which would have considerably increased the duration of the sampling process. Based on these results each of the causes was allocated to a group. If they were allocated to the first two groups (1 and 2 below) they were excluded from further study, if they were included in the third group (group 3) they would receive further attention. The selection process is detailed below and the individual death rate graphs are contained in appendix 2.

1. *Causes of death for which the death rate increased between 1950 and 1999 – excluded from further investigation*

The causes of death which are included in this category are breast cancer, ovarian cancer, prostate cancer, diabetes mellitus, pneumonia, accidental falls and suicide. The justification for their exclusion at this stage was that the death rate for all of these causes increased, compared to the death rate in 1950. This study is only interested in causes of death which could have contributed to the overall decline in mortality. As the proportion of total deaths attributable to lung cancer was so large over the time period covered by this thesis further research into this cause of death was initially considered. All deaths from lung cancer between 1950 and 1999 were subsequently digitised. However, this cause of death was eventually excluded from further analysis as, although male deaths had seen a decline since the 1970s, the overall death rates in 1999 were still higher than those in 1950, meaning that it failed to contribute to the overall decline in mortality over the entire time period under study.

2. *Causes of death for which the death rate decreased between 1950 and 1999 – excluded from further investigation*

Included in this category are senility, colon cancer, hyperplasia of the prostate, congenital anomalies and conditions originating in the perinatal period. Although death rates from these causes declined they were nevertheless excluded for the following reasons.

- Senility

This category stems from those death certificates which describe the underlying cause of death as 'old age'. The death rate from this cause of death declined from 0.2 per 1000 males in 1950 to 0.01 in 1999, and from 0.3 per 1000 females to 0.06. The reality of this decline can be questioned, as a more likely explanation is not that fewer people died from this cause, but rather the this unscientific term became increasingly unacceptable as a cause to be entered on a death certificate (Charlton, 2002). It is probable that, the deaths which in earlier years would have been attributed to 'old age' were ascribed to specific causes in later years.

- Colon cancer.

Although the death rate from colon cancer did decline, the gradient of the decline was slight, with the death rate only falling from 0.26 per 1000 among males and 0.3 per 1000 among females in 1950, to 0.2 for both sexes in 1999. Colon cancer's contribution to the overall decline in mortality was insignificant.

- Hyperplasia of the prostate

Hyperplasia of the prostate refers to the swelling of the prostate which can cause bladder obstruction and poor urinary flow (Dawson and Whitfield, 1996). Its presence in men increases with age: 88% of men over the age of 80 have been found to have the condition at autopsy (Boyle, 1994). Although the condition is now regarded as relatively harmless, in 1950 it was responsible for a considerable proportion of all male mortality (1.6%). However, by 1999 only a fraction of male mortality was attributed to this cause. The decline in mortality from hyperplasia of the prostate is not unique to Scotland and has been the subject of a number of international studies (La Vecchia, 1995, Boyle *et al*, 1996, Levi *et al*, 2003). Each of these studies reached the same conclusion: that improved medical management and surgical intervention helped prevent the most severe complications which can accompany the disorder.

Given the large reduction in mortality from hyperplasia of the prostate, and existence of body of literature which has already investigated the possible factors underlying this decline, this cause was originally considered as a possible case study. However, although this cause may have been a significant contributor to the overall decline in male mortality, it would have had no effect on the decline of mortality amongst women. The decision was taken to

focus on causes of death from which both males and females would have benefited from any reduction in mortality.

- Congenital anomalies

These conditions were identified by McKeown as being 'relatively intractable', that is conditions determined before birth and not due to environment. Included under this heading are conditions such as neural tube disorders (NTDs), for example spina bifida and anencephalus. In the *Role of Medicine* (1979) McKeown considered what could possibly influence the decline of this group of disorders. He rejected factors such as improving standards of living; instead he saw a role for medicine in identifying cases during pregnancy, to prevent subsequent deaths by offering a termination. Between 1950 and 1999 the death rate from congenital anomalies declined significantly, from 0.13 per 1000 males and 0.11 females, to 0.03 for both sexes. The majority of this decline occurred from the 1970s onwards. A number of possible influences on this decline can be identified, including advances in pre-natal diagnostics, such as ultrasound, to allow selective termination. This was developed by Professor Iain Donald and his team at Glasgow University. Diagnosis during pregnancy was also aided by a number of tests, to identify affected pregnancies, which evolved through the 1970s, such as the Alfa Foetal Protein blood test and amniocentesis (Nicholson, 2003).

In addition to the reduction of congenital anomalies deaths via termination, medicine also introduced the use of folic acid to prevent neural tube disorders. The use of folic acid in the prevention of NTDs was first proposed in the 1960s. However, it was not until 1991 that final proof of the link was available. The MRC Vitamin Study Research Group (1991) found that women taking 4mg of folic acid periconceptually were afforded 75% protection against having a NTD affected baby. The role which this has had on the overall reduction of congenital anomalies mortality up until the late 1990s has, however, been questioned as many women were not aware of the correct time to take the supplement (Kadir *et al*, 1999).

Congenital anomalies were therefore another cause of death which were considered as a case study, especially given the apparent links between their decline and advances in medicine, in particular diagnosis during pregnancy. They were rejected at this point because they represented a diverse group of conditions, with several different aetiologies. As such, any investigation of the reasons underlying the decline of this disease group would have to consider a wide variety of possible influences and clear conclusions could be difficult.

- Conditions Originating in the Perinatal Period

In the earlier versions of ICD, this group of diseases was referred to as 'the conditions of infancy'. The death rate for this group of conditions declined significantly between 1950 and 1999 from 0.43 per 1000 males and 0.28 females, to 0.03 and 0.02 respectively. These conditions were not selected as a case study group for reasons similar to those described for congenital anomalies. The group contains a variety of different conditions, none of which individually accounted for 1% or more of total mortality, but which taken together represented a large number of deaths, especially in 1950. Included under this heading are conditions such as birth trauma, conditions relating to the mother and slow foetal growth. It is possible that all of these conditions may have declined due to the same factors, such as improvements in maternal and infant care. However the variety of conditions involved meant that it was possible that any analysis of the overall reasons for their decline could become too complex.

3. *Causes of death for which the death rate decreased between 1950 and 1999 – selected for further study*

Included in this category are tuberculosis (TB), stomach cancer, chronic obstructive pulmonary disease (COPD), cerebrovascular disease (stroke), ischemic heart disease (IHD) and road traffic vehicle accidents (RTVA). These causes of death accounted 52% of all male, and 53% of all female, mortality in 1950, and had been shown in a basic analysis of sample years to have declined over the period under review.

4.8. Data entry and death rates

All deaths, from the causes identified above as having experienced a decline in mortality, were isolated from the death returns for the years 1950 - 1999 and entered into Excel, disaggregated by five year age group and sex. Once this information was available in a digitised format, death rates standardised to the 1950 population were calculated according to the method described in section 3.2.1. In addition age specific death rates were calculated by sex for the age bands <25, 25 – 44, 45 – 64 and >=65.

4.9. Conclusion

The death returns contain almost 1000 causes of death, all of which had originally to be considered as possible contributors to the overall decline in mortality in Scotland between 1950 and 1999. After a process of refinement seven causes of death were isolated from the death returns. With the exception of lung cancer, all of these causes experienced a decline in their death rate between 1950 and 1999. For each of these causes of death, death rates standardised by year and by age group were generated. The following chapter will examine the extent to which each of these causes of death contributed to the overall decline in mortality. Three of these causes will be selected as case studies, and the relative contribution of medicine and standards of living to their decline will be considered.

5. Contribution of selected causes of death to the decline in overall mortality

5.1. Introduction

In the previous chapter, tuberculosis, stomach cancer, COPD, IHD, stroke and RTVA, were identified as causes of death which were responsible for a significant share of mortality in 1950 (52% of male and 53% of female mortality) and which had declined substantially by 1999. The number of deaths for each of these causes of death, by age and sex, were digitised for every year between 1950 and 1999. These data were then analysed to create death rates standardised to the 1950 population and age specific death rates. Based on this work this chapter has two main aims:

- to calculate the contribution of each of the six causes of death to the overall decline of mortality in Scotland between 1950 and 1999,
- to refine this original list to a shortlist of three causes of death which will be explored in depth (case studies).

5.2. Calculation of the relative contribution of selected causes of death to the overall decline in mortality, 1950 – 1999

5.2.1. The calculation of 'potential lives saved'

The method employed to measure the relative contribution of each of the causes of death selected to the overall decline in mortality is the calculation of potential lives saved. This calculation has been adapted from Capewell *et al*'s (1999) study of ischemic heart disease in Scotland. In this study Capewell *et al* calculated the number of IHD deaths which they anticipated would occur in 1994 if the IHD death rate had remained the same as it was in 1975. Once the actual number of IHD deaths was subtracted from the projected number of IHD deaths, the remaining figure was the number of deaths which had been 'prevented' between 1975 and 1994. This thesis has adapted the calculation, and considered the *total* number of deaths which would have been prevented in 1999, if the death rate had remained the same as it was in 1950, taking into account changing population structure. The numbers of deaths prevented, or potential lives saved, for each of the six short-listed causes were then calculated, and the relative contribution of each of these to all lives saved was determined.

5.2.2. All cause potential lives saved

Between 1950 and 1999, the number of deaths which occurred annually remained roughly constant, with a total of 32191 male deaths and 31805 female deaths in 1950, and 28605 male deaths and 31676 female deaths in 1999. This relatively constant level of mortality disguises the downward trend in mortality rates which occurred over this time period. This is because between 1950 and 1999 the population of Scotland aged considerably, meaning that if the death rate had not declined, many more deaths would have been anticipated in 1999. This saving of life is reflected in the calculation of all cause potential lives saved. In this calculation the death rate for 1950 is applied to the 1999 population. In order that changes in population structure are accounted for, this calculation is performed by five year age group, so that the population in 1999 is multiplied by the death rate for the age group in 1950 to give the number of deaths that would be expected in each age group in 1999. The actual number of deaths in each age group in 1999 is subtracted. Once the savings in each five year age group are summed this gives the actual number of deaths prevented. It was calculated that there were 16388 fewer male deaths, and 22645 fewer female deaths, in 1999 than would have been anticipated if conditions had remained the same as they were in 1950.

McKeown concluded that much of the earlier decline in total mortality was attributable to the reduction in mortality amongst the young. However, when the distribution of lives saved amongst age groups in 1999 is considered, a different picture emerges, with the majority of lives saved being amongst the over 65s (table 5.1). This difference in the age distribution of lives saved is reflected in the causes of death which were responsible for the decline of mortality. In McKeown's era the infections, which are associated with excess mortality amongst the young, were responsible for most of the decline in mortality. In this study the degenerative diseases were responsible for most of the mortality decline, and these are associated with mortality in mid to old age.

Table 5.1. Percentage of total potential lives saved by age group in 1999 compared with the 1950 death rate

	Male %	Female %
Under 25	10	7
25 – 44	5	6
45 – 64	28	14
65+	57	73

Source: Original analysis of data obtained from the GROS.

5.2.3. Potential lives saved by cause

The calculation of potential lives saved described in 5.2.2. was applied to each of the six short-listed causes of death in order to calculate the number of deaths which had been prevented in 1999 which were attributable to each of these causes. The contribution of each of these to overall saving was then calculated. Table 5.2. displays the percentage of the total saving in mortality which was due to each cause. The largest contributor to the decline in both male and female mortality was IHD, accounting for 38% of male, 49% of female deaths prevented in 1999 compared to 1950. Stroke was the next largest contributor to lives saved, followed by tuberculosis (which is the only cause of death which has survived from the original causes of death considered by McKeown). Stomach cancer was the only cancer for which mortality had declined significantly enough to be considered in this study. Finally, COPD and RTVA were responsible for 2% and 1% of all male deaths prevented in 1999. In the case of women RTVA did not result in an overall saving of life, and COPD produced a negative result as there were more deaths in 1999 compared to 1950.

Table 5.2. Contribution of selected causes of death to total potential lives saved in 1999 when compared with the 1950 death rate

	Male %	Female %
IHD	38	49
Stroke	18	20
Tuberculosis	9	5
Stomach cancer	5	4
COPD	2	-1
RTVA	1	0
Other Causes	27	23

Source: Original analysis of data obtained from the GROS.

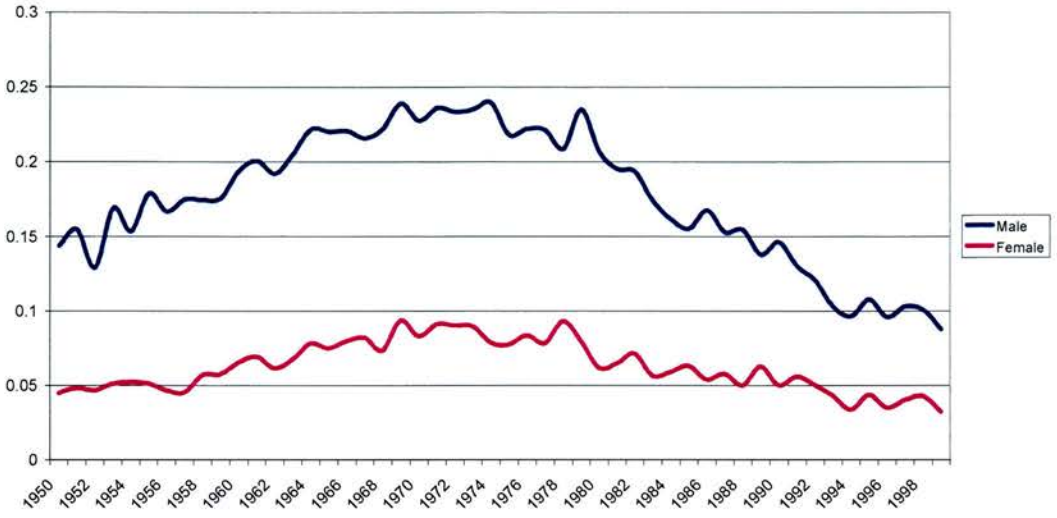
5.3. Selection of the case studies

The six causes of death which have been considered thus far were responsible for 73% of male lives saved and 77% of female lives saved between 1950 and 1999. However, within the limitations of a PhD it was initially decided that a detailed examination of the factors determining their decline could only be conducted for three of these causes of death, especially since the intention was to explore the influence of standards of living and medicine in a way which few other commentators had previously undertaken. Several different criteria were considered for selection of causes of death as case studies, and the justification for exclusion from the final selection is detailed below.

5.3.1. Road Traffic Vehicle Accidents (RTVA)

Included under the heading road traffic vehicle accidents (RTVA) are deaths which occur as a result of the collision of motor vehicles with one another, the collision of motor vehicles with a pedestrian, and deaths which occur when a motor vehicle loses control on the road. Throughout the time period 1950–1999 RTVA mortality was higher amongst males than females. Death rates for both sexes increased from 0.14 per 1000 males and 0.04 per 1000 females in 1950 until the 1970s (see figure 5.1). Throughout the 1970s death rates remained stable and high, peaking for males in 1979 at 0.24 per 1000 and 1978 for females at 0.09, after which the death rates for both sexes underwent a period of decline, with death rates reaching 0.09 per 1000 males and 0.03 per 1000 females in 1999.

Figure 5.1. Road traffic vehicle accidents death rates, per 1000, standardised to the 1950 population, 1950-99



Source: Original analysis of GROS data.

A number of factors originally made this cause of death attractive as a potential case study. One of these is the fact that this is not a disease, and as such the reason why death occurred is readily identifiable, that is violence caused by a car crash, as opposed to the numerous possible influences on a disease such as IHD. There are only two ways in which mortality from RTVA can be reduced: by a reduction in collisions or by the increasing survival of those who were involved in a collision. The factors influencing these determinants of survival might have proved worthy of further explanation.

Reductions in the number of collisions would require the investigation of the effectiveness of various government measures to improve safety on the road. These include the influence of changing road systems, such as the role which the introduction of dual carriageways and motorways would have had on reducing head-on collisions. Before the late 1960s no speed limit existed on the open road, therefore the effect of speed restrictions on collisions would have to be considered. Throughout the time period covered by this study, drink driving became increasing unacceptable, both under the law and socially; therefore this area would also require investigation.

A number of factors could possibly affect the survival of individuals once a crash has occurred, including developments in car design to lessen the impact of crashes. The

introduction of legislation for the compulsory wearing of seat belts may also have helped reduce the severity of injuries to those involved in car crashes. For those who had suffered serious injuries, improvements in ambulance services and emergency medical treatment could also have increased survival.

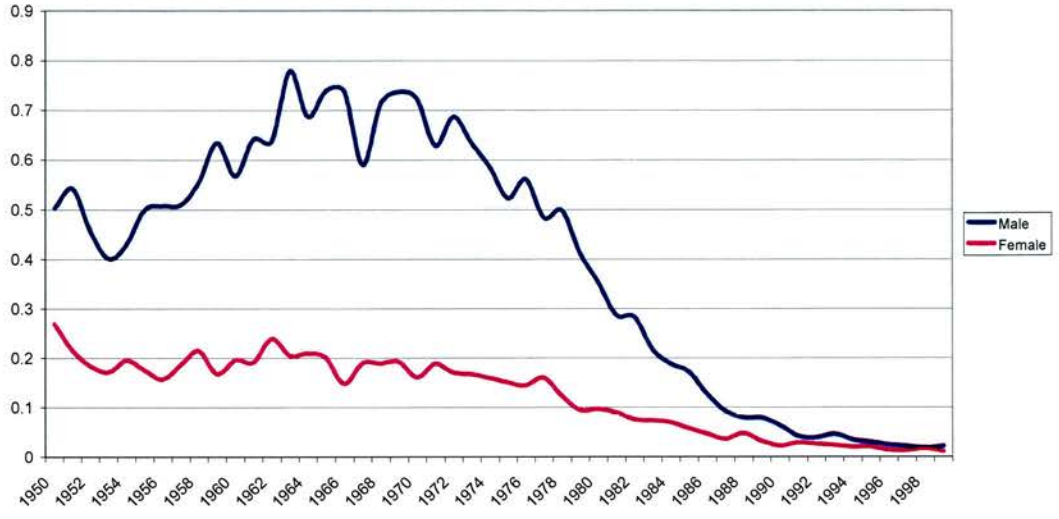
RTVA is also an interesting potential case study as the pattern of mortality witnessed between 1950 and 1999 can almost be seen as paradoxical. Throughout this time period the number of cars on Scotland's roads increased, with only 31% of the population having the regular use of a car in 1961 compared to 72% in 1999 (Social Trends 33, 2004). Given this increase in car usage it might be anticipated that deaths rates from RTVA would have continued to increase after the 1970s. Therefore any investigation of the factors influencing the decline in mortality would have to take into account this growth in car usage, when considering the post-1970s mortality decline. Another interesting feature of the influences on RTVA is that most of them are not readily identifiable as being the result of improvements in either medicine or standards of living. This means that any reduction in mortality which could be attributed to factors such as the reduction in the speed limit, legislation against drink driving or the introduction of seat belts would require consideration of the nature of their influence.

In spite of the argument outlined above for the inclusion of RTVA as a case study, this cause of death was not selected. The main reason was that it was responsible overall for only a small reduction in total mortality between 1950 and 1999. Although male mortality saw a considerable decline since rates peaked in the late 1970s, when the death rate from 1999 is compared with that of 1950, only 1% of the total reduction is attributable to RTVA. Amongst females the reduction was even less with the death rate only declining fractionally.

5.3.2. Chronic Obstructive Pulmonary Disease (COPD)

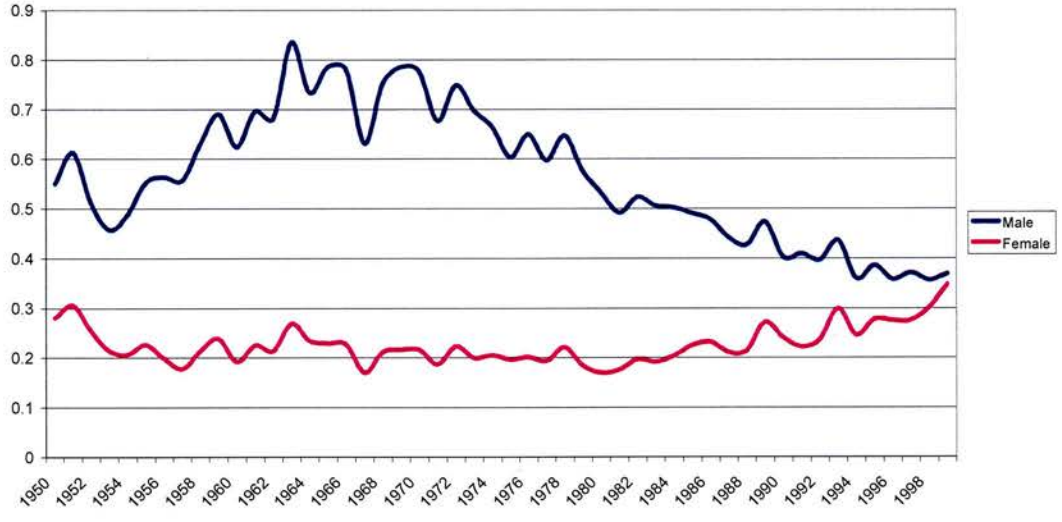
In 1950 the vast majority of what are now classed as chronic obstructive pulmonary disease deaths were coded in the death returns to chronic bronchitis. It was this cause of death which was originally considered as a case study. All deaths from chronic bronchitis were isolated from the death returns and entered into Excel by age and sex. The results of the analysis of these data are shown in figure 5.2, which reveals that the burden of mortality fell upon men, with the male death rate increasing until the mid 1960s, after which it remained stable before entering a period of sharp decline. The female death rate remained relatively constant until the mid 1970s before also entering a period of decline. This large reduction in the chronic bronchitis death rate originally meant that it was a likely candidate for selection as a case study. However, an initial survey of the literature, followed by a review of the Registrar General's transference tables revealed that this was not a true decline in mortality, but was instead an artefact of the introduction of new ICD codes and the revision of instructions issued to coders. Up until 1968 almost all chronic respiratory deaths were coded to chronic bronchitis. However, with the introduction of ICD 8 a new category entitled, 'other diseases of the respiratory system' (this was renamed COPD under ICD 9) was defined. It was to this new category that deaths previously assigned to chronic bronchitis were now being allocated. In light of this, a new dataset was created to include all deaths under the definition of chronic obstructive pulmonary disease, and the results of the revised analysis are shown in figure 5.3. The variations in the death rate emphasises the need for assessing reliability when tracing disease mortality trends.

Figure 5.2. Bronchitis death rates, per 1000, standardised to the 1950 population, 1950-99



Source: Original analysis of GROS data.

Figure 5.3. COPD death rates, per 1000, standardised to the 1950 population, 1950-99



Source: Original analysis of GROS data.

The inclusion of the COPD deaths with those from chronic bronchitis had little impact on the death rate until the late 1960s. From this time the male COPD death rate entered a period of

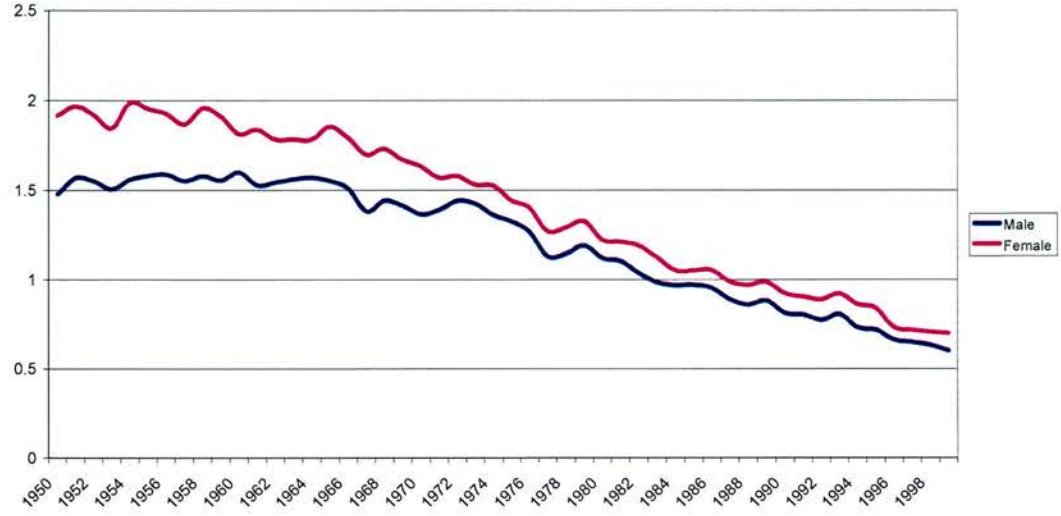
decline. However, the gradient of the decline is not as steep as that witnessed for chronic bronchitis alone (figure 5.2). Amongst women the revision of the dataset had a more profound effect, under the new classification of COPD the death rate in 1999 was slightly higher than it had been in 1950, at 0.34 per 1000 compared to 0.27 in 1950.

Following the modification of the dataset, COPD was considered as a potential case study. The definition COPD refers to an inflammatory lung condition, which can lead to airflow restriction. The main risk factors for COPD are smoking and air pollution. A link has also been established between childhood infections, such as measles, and susceptibility to COPD in later life (Mercer, 1986). Therefore any review of the factors influencing the decline of COPD would have to consider the prevalence of both smoking and air pollution and their determinants, as well as medical treatment. The association with childhood infections also provides a direct link to McKeown's findings for the earlier influences on mortality.

COPD is therefore an interesting disease as it is influenced by factors relating to personal behaviour (smoking) and to other non-personal factors (pollution). In addition, the differing patterns of mortality experienced by males and females would allow a consideration of the different risks to which the sexes were exposed over time. However, the effect of the revision of the dataset made COPD a less attractive potential case study. In the case of men, although the disease did decline from the early 1970s, its overall contribution to the mortality decline was small, and in the case of women the death rate increased to be slightly higher in 1999 than it was in 1950. Therefore COPD was excluded from further study at this stage.

5.3.3. Cerebrovascular disease (Stroke)

Figure 5.4. Stroke death rates, per 1000, standardised to the 1950 population, 1950-99



Source: Original analysis of GROS data.

When the process of choosing case studies for further study was initially developed, it was decided that only three diseases would be selected. The reason for this was to allow adequate time for each disease to be examined in depth. Additionally it was intended to select three diseases which not only contributed significantly to the overall share in mortality, but which were also subject to differing sets of influences. The reason for this was to allow a wider discussion of the overall influences on mortality decline. Stroke was therefore initially rejected as a case study in spite of the fact that it accounted for 18% of the decline in male, and 21% of the decline in female mortality. Stroke exclusion was based on the belief that many of its findings would overlap with those for IHD, leading to repetitiveness. After the review of the first three case study diseases the decision to exclude stroke was reversed for two reasons. The first was the large proportion of the mortality decline attributable to stroke. Through stroke's inclusion a far larger proportion of the overall decline in mortality could be reviewed. The second reason was that the findings of IHD were not as applicable to stroke as had been initially anticipated. Although there is a large degree of overlap in the findings of IHD and stroke, significant differences also emerged. Therefore, stroke has been included in this thesis as a case study disease within an extended chapter on cardiovascular diseases.

5.4. Causes of death selected as case studies

From the original short-list of six causes of death, road traffic vehicle accidents and chronic obstructive pulmonary disease were excluded. The main reason for their exclusion was the small contribution which they made to the overall reduction in mortality. These exclusions leave tuberculosis, stomach cancer, IHD and stroke as the causes of death which have been selected as case studies. Together these account for 70% of all male, and 78% of all female, potential lives saved in 1999 compared with 1950. A number of factors, including their overall contribution to total potential lives saved, influenced the selection of these causes of death and these will be discussed in more depth in the chapters dealing with each of these causes. In brief, tuberculosis was selected as it was the only infection which contributed a significant share to the overall decline in mortality, whilst also providing a direct link with the earlier work of McKeown. Stomach cancer is of interest as its downward trend contradicts that of most other cancers during the time period. In the case of IHD, this is the disease for which Scotland has an unenviable reputation for excess mortality and yet this was the largest contributor to the overall decline in mortality. The reasons for the selection of stroke have been given in section 5.3.3.

5.5. Conclusions

Using the calculation of potential lives saved, it has been estimated that tuberculosis, stomach cancer, COPD, IHD, stroke and RTVA were responsible for over 70% of the decline of male and female mortality. To allow an in-depth analysis to be carried out on the factors responsible for this decline in mortality, two of these case studies were excluded for a variety of reasons. This has left tuberculosis, stomach cancer, IHD and stroke as the causes of death which this thesis will consider in depth. Each of these causes of death will be considered in turn over the following three chapters to establish the relative contribution of medicine and standards of living to their mortality decline within the framework described in section 3.4.

6. Tuberculosis

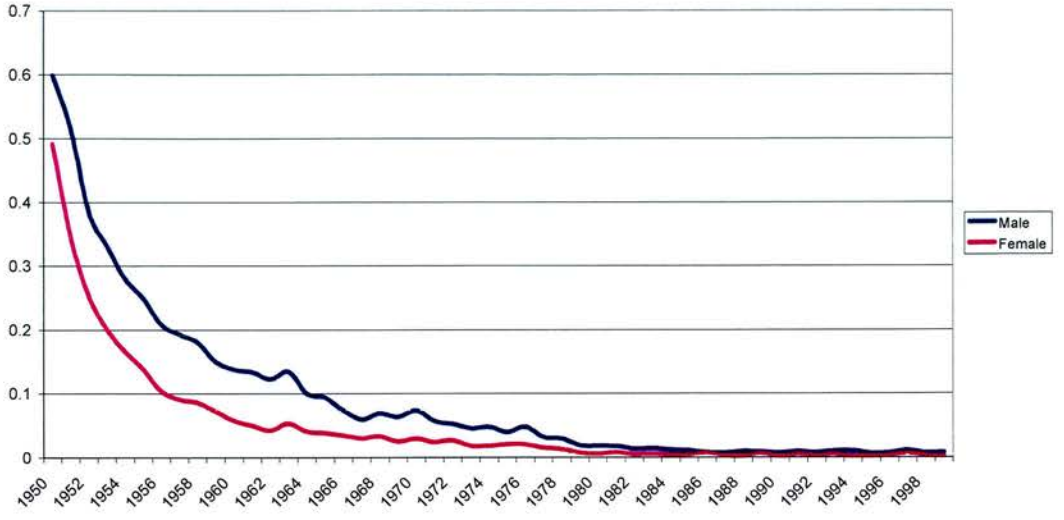
6.1. Introduction

The World Health Organisation estimates that there are nearly 2 billion people infected with tuberculosis world-wide, of whom 8.74 million will go on to develop clinical disease, with 2 million eventually dying (Kumaresan, 2002). Of these deaths, 98% occur in the developing world (Zumla and Grange, 1999). Within the developed world tuberculosis has only recently disappeared as a major killer, and remained the single largest cause of death in Scotland until the start of the 20th century.

This excessive tuberculosis mortality was rapidly eliminated over the time period covered by this thesis, as the share of mortality attributable to tuberculosis fell from over 12% of both male and female deaths at the start of the 20th century, to nearly 4% of male and 3% of female deaths in 1951. By 1999, tuberculosis's contribution to total mortality was negligible. In chapter five, tuberculosis was identified as the third largest contributor to the decline in overall mortality between 1950–99. The fall in mortality from 1950 occurred rapidly (see figure 6.1), with the majority of the decline occurring in the first 10 years. The gradient of this decline at the start of the graph indicates that this decline may have been long standing. Therefore this chapter will also consider influences on tuberculosis during the earlier part of the century.

Tuberculosis also makes an interesting case study as it is the only infectious disease which accounted for a significant share of the post-1950 mortality decline. In addition tuberculosis provides a link with the earlier research of McKeown (see chapter 2). McKeown placed the decline of tuberculosis at the centre of his explanation for the decline in mortality up to 1971, and attributed the majority of the mortality decline to improvements in diet. A review of factors influencing tuberculosis mortality pre-1950 will allow a debate of the relative contribution of diet to mortality decline in an era when effective medical treatment had become available.

Figure 6.1. Tuberculosis death rates, per 1000, standardised to the 1950 population, 1950-99



Source: Original analysis of records obtained from the GROS.

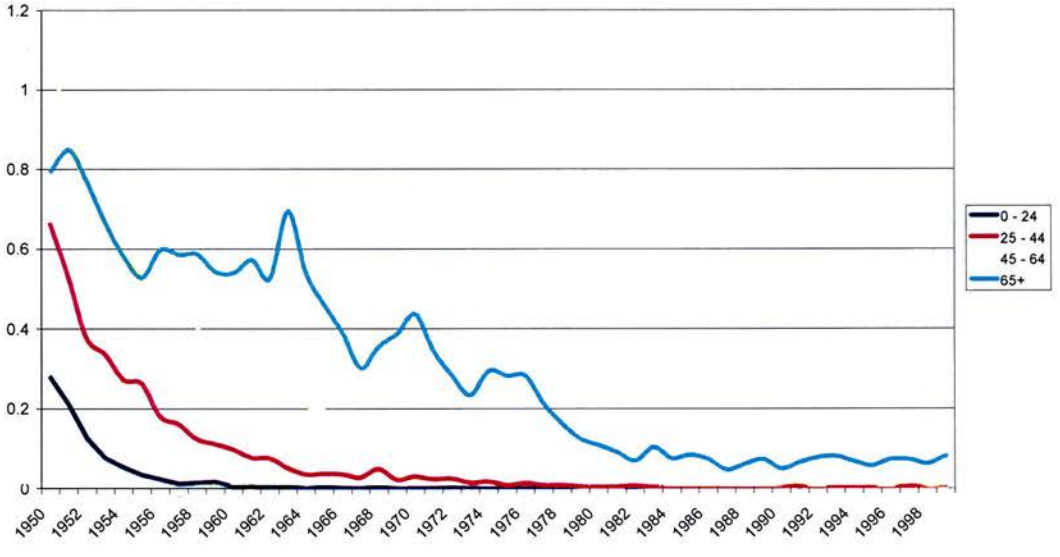
This chapter will consider what factors led to the decline of mortality from tuberculosis. In the following section background information on tuberculosis will be provided, including the age groups which the disease affects and a description of the disease. The pattern of mortality decline followed by tuberculosis will then be reviewed to isolate time periods when mortality was either increasing or decreasing. These timeframes will provide a framework within which the relative contribution of medicine and standards of living to the mortality decline will be discussed.

6.2. Background

6.2.1. Age groups affected

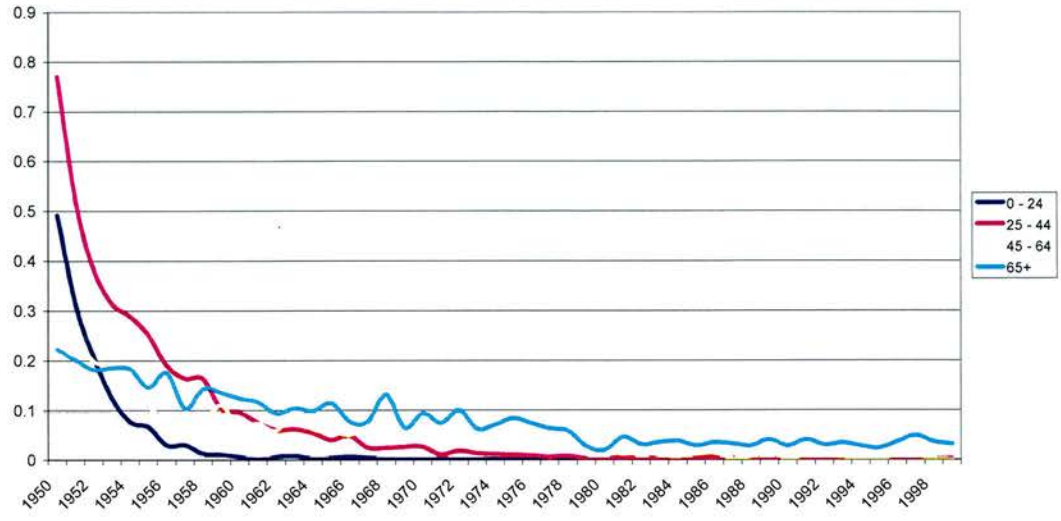
The tuberculosis death rates by age group are displayed in figures 6.2 and 6.3. In the case of males the death rate is highest in the age groups 45-64 and 65+. The death rate in the age groups 0-24 and 25-44 is relatively high in 1950 after which it enters a period of decline. The age distribution of the death rates for females is slightly different, as both the age groups 0-24 and 25-44 are higher than those in the older age groups during the 1950s, although the death rate was in decline for all age groups.

Figure 6.2. Male tuberculosis death rate, per 1000, by age group, 1950-99



Source: Original analysis of records obtained from the GROS.

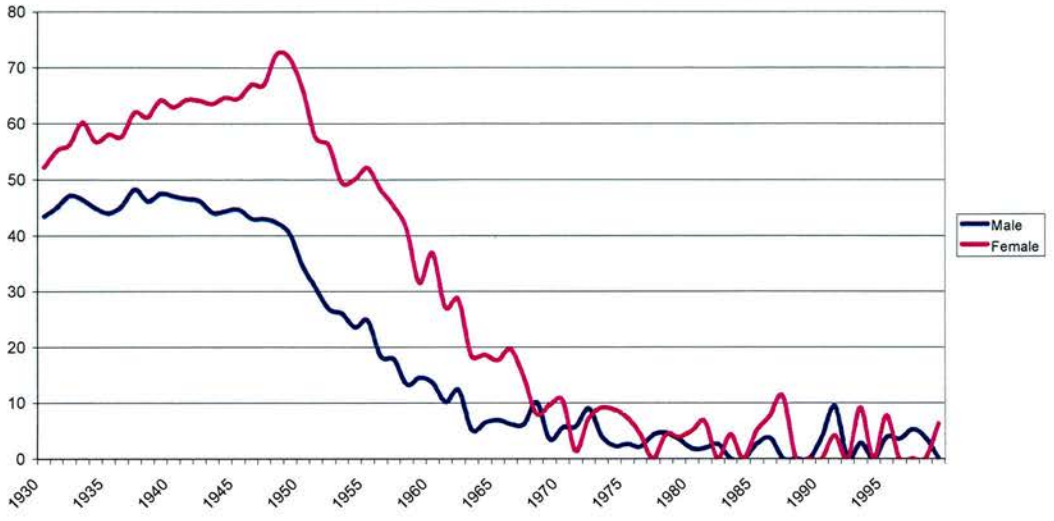
Figure 6.3. Female tuberculosis death rates, per 1000, by age group, 1950-99



Source: Original analysis of records obtained from the GROS.

The high mortality rates amongst younger age groups have also been identified elsewhere in the literature on tuberculosis (see Bryder, 1988, MacFarlane, 1990, Smith, 1988). This excess mortality amongst the young is reflected in the total share of tuberculosis mortality accounted for by the younger age groups, and the percentage of total mortality amongst the young which was attributable to tuberculosis. In figure 6.4 the percentage of total tuberculosis mortality accounted for by the age group 15-39 is displayed. This graph covers the period 1930-1999 (the process of expanding the tuberculosis dataset is described in section 6.3.2). The most striking feature of this graph is the large share of total mortality accounted for by the age group 15-39 from the 1930s until the early 1960s. This is of particular significance for females, with over 70% of all females dying of tuberculosis in 1948 being aged between 15-39. In addition, over 50% of all mortality amongst 15-39 year olds was caused by tuberculosis. Tuberculosis was therefore a disease which disproportionately affected the young in the early part of the time period covered by this thesis. This pattern was reversed by the late 1960s when the majority of deaths, for both sexes, occurred amongst the over 45s.

Figure 6.4. Percentage of tuberculosis mortality attributable to age group 15-39, 1930-99



Source: Original analysis of records obtained from the GROS.

6.2.2. Clinical description

Tuberculosis is an infectious disease, which can be caused by two agents – the mycobacterium bovis (M. Bovis) and the mycobacterium tuberculosis (M. TB). The M. Bovis is transmitted from cattle to humans via contaminated milk or animal produce. More common is the M. TB which is an airborne disease transmitted when an infected person coughs, spits or sneezes and someone nearby breathes in the tubercle bacillus sprayed in the air. Infection can lead to the development of tuberculosis in virtually any part of the body. Forms of tuberculosis include milliary tuberculosis, which occurs when infection enters the blood stream, and was invariably fatal before the advent of chemotherapy. Tuberculosis of the bones was associated with the consumption of infected milk and could lead to bones collapsing in advanced cases. By far the most common disease site was the lung, in the form of pulmonary tuberculosis which accounted for over 80% of total tuberculosis mortality (MacFarlane, 1990). This chapter will focus on this type of tuberculosis as it was the most common.

Pulmonary tuberculosis can develop after an individual inhales the tubercle bacillus from the air into the lungs. If disease develops, this causes cavities to appear in the lung, which can, if left untreated, lead to the destruction of the lung (British Thoracic Society, 2003). For the

individual affected, the disease can cause years of impaired health before premature death occurs. However, infection with the tubercle bacillus does not necessarily lead to clinical disease, with only about 10% of those infected going on to develop clinical tuberculosis (Davis and Grange, 2001).

6.2.3. Signs and symptoms

The diagnosis of pulmonary tuberculosis has historically been difficult. Before the introduction of effective medical treatments the disease normally followed a long course, with the individual affected becoming increasingly debilitated. During this time the patient could exhibit a wide range of symptoms, not all of which were present in every case. These include fever, fatigue, night sweats, difficulty breathing, haemoptysis (coughing blood) and weight loss (Bryder, 1988). Throughout the 20th century there were a number of medical advances in the diagnosis of tuberculosis. This began with the development in the first decade of the century of skin tests, which could tell if an individual had been exposed to the infection. Diagnosis of clinical disease was later made possible using x-rays and sputum tests (Bryder, 1988).

6.2.4. Risk factors

For an individual to die of tuberculosis they must pass through three stages in the development of the disease. Each of these stages has its own set of risk factors and these are outlined below.

1. Exposure to the tubercle bacillus

As tuberculosis is an infectious disease, it cannot occur unless an individual has first been exposed to the bacillus. Tuberculosis is an air-borne infection, therefore conditions which increase the likelihood of inhalation of the bacillus also increase risk. These include, both overcrowded living and working conditions, or poorly ventilated conditions (Hardy, 1993).

2. Development of clinical disease

It has been stated (section 6.2.2) that not everyone who is infected with the tubercle bacillus goes on to develop clinical tuberculosis. The vast majority of people who have been infected experience no ill effects. The reason why some individuals succumb to clinical disease once infected, whilst others do not, is still imperfectly understood. However, the most likely explanation is the condition of the individual's immune system. That is, anyone whose immune system is compromised is at increased risk of developing the disease. Factors

influencing this include pre-existing medical conditions and lack of adequate nutrition (Davis and Grange, 2001).

3. Death from clinical disease

Once an individual has developed clinical tuberculosis they may either recover from the disease or go on to die from the condition. In the absence of effective medical treatments the majority of sufferers die from the condition. Before the introduction of chemotherapy, it was believed that rest and proper feeding could increase the potential for survival; however, there is little evidence that this was successful (see section 6.4.1.1).

6.3. Mortality trends

6.3.1. Data reliability

The process of digitising tuberculosis from the Registrar General's Death Returns has been outlined in section 4.8. However, before the death rates generated from these data can be discussed, it is first necessary to consider briefly the accuracy and reliability of the original death data. Two main problems with data reliability have been highlighted by this thesis; inaccurate completion of the death certificate and failure to diagnose the correct cause of death.

Inaccurate completion of the death certificate in the case of tuberculosis has been identified in the literature. This is mainly in respect of the pre-1950 period. The principal reason for this was the stigma which was associated with the diagnosis of tuberculosis. This stigma was a result of the poverty which was associated with the disease. There were two reasons for this: firstly, the poor were initially more at risk of the disease, and secondly sufferers could drift into poverty as the result of the long course followed by the disease which often meant they were no longer able to work. Many also associated the disease with vice. This is illustrated by the views of a member of the National Association for the Prevention of Tuberculosis, quoted in Bryder (1988), who stated in 1912 that, "*...sexual vice is one horror; alcoholic habit is another; and the two are seldom found apart from the tubercle... tubercle attacks failures. It attacks the dispossessed, the alcoholic, the lunatic of all degrees*" (p.20). This horror of tuberculosis is also evident later in the century, when according to a doctor working after World War Two: "*tuberculosis cast a great shadow across the land, not only because of its high mortality, but because of the serious stigma attached to it. Individuals and families were loath to talk about it or even to admit to having*

had the disease. It was a harrowing experience to have to tell a young person that the diagnosis was tuberculosis" (Williamson, 2000: 183). This stigma could lead GPs to certify an alternative cause of death, such as pneumonia, bronchitis or influenza out of concern for the surviving relatives (Bryder, 1988). However, the extent to which this occurred before 1950, and whether this was still a motivation for miscertification in the chemotherapy era, is unclear.

A number of studies have examined the accuracy of clinical diagnosis on death certificates by cause. However, as tuberculosis became such a rare cause of death post-1950 it is seldom included in these studies. One survey which looked at tuberculosis compared clinical diagnosis on the death certificate with autopsy findings in South Lothian between 1975-1977 (Cameron and McGoogan, 1981b). Originally seven death certificates recorded tuberculosis as the underlying cause of death in this study. Of these certificates only two cases of tuberculosis were confirmed as the underlying cause of death at autopsy. However, five new cases of tuberculosis were only discovered at autopsy.

Given the lack of detailed evidence dealing with the reliability of tuberculosis death data it is difficult to establish how representative these results are. It is possible that the lack of accuracy seen in the South Lothian study is a result of the small number of cases which were being seen by the late 1970s. As doctors became less familiar with the condition their ability to diagnose it may have declined, suggesting that diagnosis may have been more accurate earlier in the century when the disease was more common. One way in which to test the accuracy of the death rates generated by this thesis is to compare them with rates discussed in both the literature of the time, and secondary reviews of tuberculosis. Similar trends have been identified in both these studies and this thesis, indicating that the overall pattern of mortality rates discussed in this thesis are likely to be reliable.

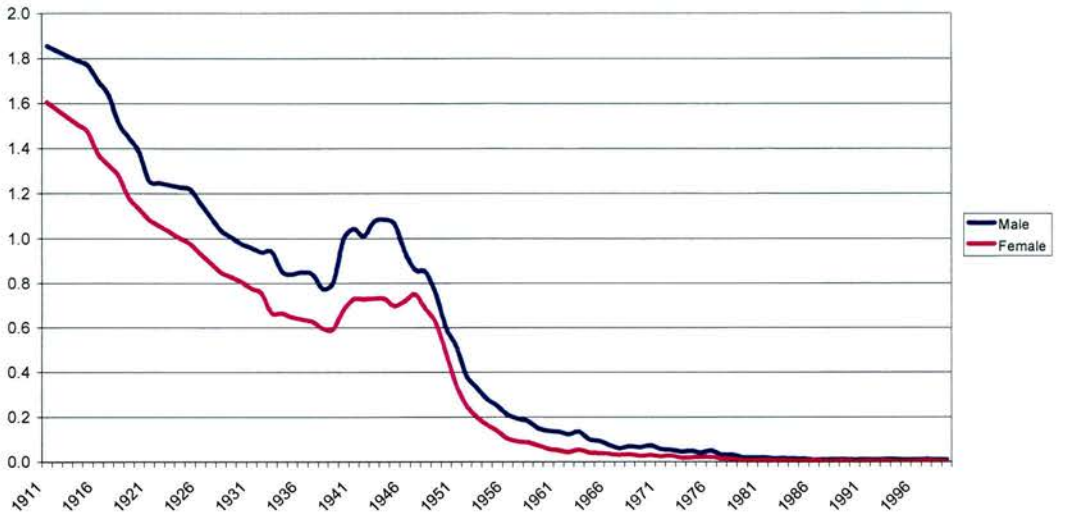
6.3.2. Pattern of decline

The trend of tuberculosis mortality since 1950 in Scotland is one of unbroken decline: a steep fall in deaths through the 1950s, so that by the 1960s its contribution to total mortality was small (see figure 6.1). This decline coincided with the discovery of the first chemotherapy drugs to be effective against tuberculosis. If this event had precipitated the decline of the disease, then it may be acceptable to state at this stage that medicine, through chemotherapy, was responsible for the post-1950 decline of tuberculosis. However, the

work of Thomas McKeown informs us that the decline of tuberculosis was long standing and predated the introduction of drugs. Therefore, it is not possible to accept that medicine was responsible for the entirety of this decline. Given these concerns, it was decided to investigate the post-1950 decline of tuberculosis within its longer term context, to confirm whether Scottish tuberculosis mortality followed a similar pattern to that found by McKeown in England and Wales. Considering influences on any earlier decline will allow a judgement to be made over which factors remained influential during the chemotherapy era.

In order to identify the longer term trends on Scottish tuberculosis mortality, additional years were added to the tuberculosis dataset. Initially the dataset was extended only back until 1930. Data on all tuberculosis deaths, by age and sex, were acquired from the GROS and digitised. Using this expanded dataset, death rates, standardised to the 1950 population, were generated. The new data indicated that there had been a surge in tuberculosis mortality during the war, although the death rate had been in decline between 1930 and 1938. To establish if the decline seen in the 1930s was a continuation of an earlier pattern of decline the dataset was expanded further. For the period 1911-1929 data for sample years (1911, 1915, 1921, 1925) were acquired from the GROS and digitised. Death rates, standardised to the 1950 population, were calculated using this dataset. The death rates for the years which were not digitised, between 1911-1929, were estimated by calculating the average rate between those years where data was available (a 'straight line' estimate).

Figure 6.5. Tuberculosis death rates, per 1000, standardised to the 1950 population, 1911-99*



*the rates for 1912, 1914, 1916-1920, 1922-24 & 1926-29 are estimates.

Source: Original analysis of records obtained from the GROS.

The Scottish tuberculosis death rate between 1911 and 1999 is shown in figure 6.5. This reveals an interesting pattern. The rapid decline in death rates seen post-1950 is still evident; however, this is not a continuation of a linear pattern of decline. Rather, the tuberculosis death rate during the 20th century experienced three distinct periods of decline, increase and decline, as outlined below.

1911-1938 Period of decline

The dominant trend before the Second World War was one of declining tuberculosis death rates, from 1.9 per 1000 amongst males, and 1.6 per 1000, amongst females, in 1911, to 0.8 and 0.6, respectively, in 1938.

1938-1947 The Second World War and its aftermath

During the War the trend of decline in tuberculosis death rates was reversed: there was an increase in deaths amongst both males and females.

1948-1999 The chemotherapy era

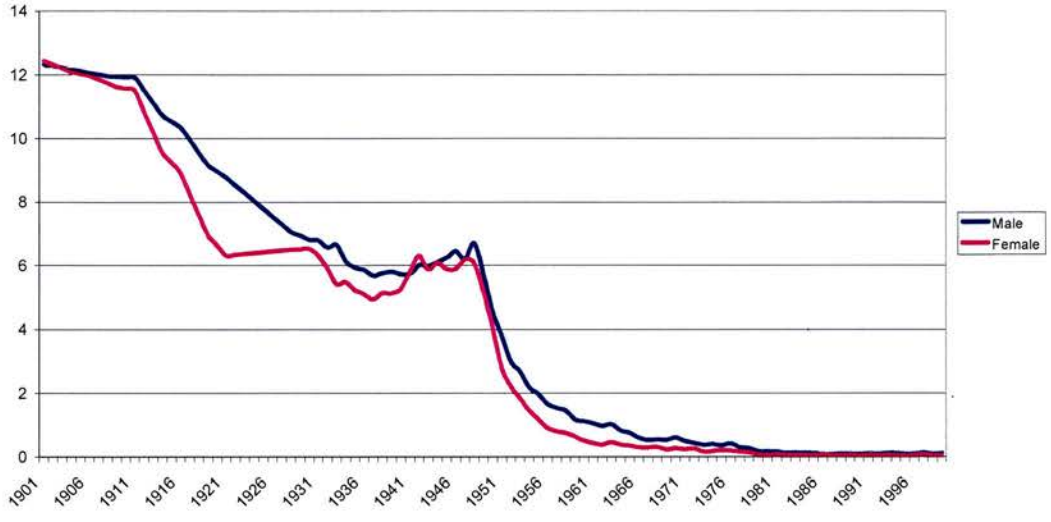
During this time period there was a continuous decline in the death rates of males and females. The majority of this decline took place in the 1950s.

The factors which influenced tuberculosis mortality will be discussed for each of these three time periods. Identification of the cause of mortality increase or decrease, in the absence of effective medical treatments, will shed light on what factors may have continued to exert an influence post-1950. The framework for assessing the relative contribution of medicine and standards of living to decline of mortality from case studies diseases was outlined in section 3.4. This has been adapted for tuberculosis. This chapter will consider whether the decrease (or increase) of tuberculosis mortality was the result of people recovering from the disease (survival), failing to develop the disease, or avoiding contact with the infection. The analysis will be performed for each of the time periods outlined above.

6.4. 1911 – 1938 Period of decline

The first four decades of the 20th century saw the percentage of total deaths attributable to tuberculosis almost halve (see figure 6.6) from 12% to 6.8% of male mortality and 6.3% of female. The identification of the causes of this earlier decline will reveal what factors, other than chemotherapy, can influence the decline of tuberculosis.

Figure 6.6. Percentage of deaths attributable to tuberculosis (all ages), 1901-99*



* the data for years 1902-10, 1912-1920 & 1922-29 are estimates

Source: Original analysis of records obtained from the GROS.

6.4.1. Survival (1911-1938)

6.4.1.1. The role of medicine (survival 1911-38)

The early 20th century saw a growth in interest by both the medical profession and the government in tuberculosis, a disease which had previously been neglected by both parties. The medical provision that existed was provided by the Poor Law. There are a number of reasons for the rise in prominence of tuberculosis at this time. In the case of the government, the poor physical state of recruits during the Boer War had prompted it to set up the 1904 Inter Departmental Committee on Physical Deterioration, which revealed the poor physical state of much of Britain's youth. For the medical profession, interest in tuberculosis had

been growing since the discovery of the tubercle bacillus, the causative agent of tuberculosis, in 1888. Prior to this discovery, the infectious nature of tuberculosis had been subject to debate. Armed with the knowledge that tuberculosis was infectious, and therefore theoretically curable and preventable, the medical profession began to look more closely at it. The reduction in deaths from the other major infections of the 19th century had also caused the relative importance of tuberculosis to increase, whilst the fact that it targeted the young raised concerns for national efficiency.

The government's response to this growth in concern about tuberculosis was to introduce funding provision for 'tuberculosis schemes'. The type of treatment established under these schemes in the early years of the 20th century was to determine the future of tuberculosis treatment until 1950. The development of the schemes began with the 1911 National Insurance Act which made financial provisions for tuberculosis suffers. The responsibility for the care of tuberculosis rested with the local authorities, with the treasury agreeing to match the amount invested by each local authority. These schemes were to be based upon that of Robert Philip in Edinburgh. Philip's system was designed to deal with the different stages of tuberculosis, from discovery to cure, and the isolation of advanced infectious cases.

The principle of the tuberculosis schemes was to offer a complete package for all stages of tuberculosis. The dispensaries, where new cases were identified, were intended to act as the first point of contact. The notification of tuberculosis became compulsory from 1912, providing a mechanism for the medical profession to keep track of all known cases of tuberculosis. The main component of these schemes were the sanatoria where early cases were to be sent to be cured. Finally, tuberculosis hospitals were established for advanced cases. Their emphasis originally was not to cure, but to isolate highly infectious cases. The tuberculosis schemes were therefore based almost entirely upon institutional care, with the erection and running of these buildings accounting for the vast majority of local authority tuberculosis budgets. While it was the responsibility of every local authority to develop a scheme based upon these lines, their initial development was delayed by the First World War, after which bed provision increased (see table 6.1).

Table 6.1. Bed provision for tuberculosis sufferers in Scotland

1921	3558
1929	5114
1938	5500

Source: Adapted from Bryder (1988).

The reality was that these schemes failed to attract sufficient 'early' cases of tuberculosis to allow the development of sanatoria (Bryder, 1988). The 'treatment' offered required a long stay in an institution and the scheme offered no financial provisions for any dependants left at home, discouraging many from entering whilst they still felt fit to work. As a consequence, much of the Scottish tuberculosis bed provision was in tuberculosis hospitals which were filled with advanced cases for whom there was little chance of recovery. The treatment offered by the sanatoria and hospitals was conservative in the early years of the schemes, based upon the principles of bed rest, fresh air and feeding (Bryder, 1988). The aim was to allow the lung to rest in the hope that this might affect a cure. Much faith, and finance, was placed in the tuberculosis schemes with many praising their results. However, the little evidence which exists of their performance leaves much to be desired, with over 80% of all cases registered in 1910 dying by 1930 (MacFarlane, 1990). The apparent failure of the schemes to deliver cures was recognised by some contemporaries. George Newman, the Chief Medical Officer, lamented in 1920 that three quarters of sanatorium patients were dead within 5 years of treatment. (MacFarlane,1990). Despite the fact that institutional care was failing to cure the sick, its development continued. The medical profession at the time could point to the declining death rate as evidence that the schemes were working, and blamed the deaths of those who had received institutional treatment upon their own 'feckless behaviour', once discharged (Lees, 1978).

By the late 1920s and 1930s the sanatoria and tuberculosis hospitals were seeking to deliver a more scientific form of treatment than that of bed rest and feeding. Doctors craved more recognition and patients were anxious to receive some type of active treatment (Bryder, 1988). Thus, during this time, a number of innovations were introduced into tuberculosis care. However, somewhat mixed results were achieved.

The main tuberculosis drug of this era was introduced in 1925. Sanocyrsin had been developed in Copenhagen and was based on a gold compound, leading to the period 1925 –

35 becoming known as the 'gold decade' (D'Arcy Hart, 1946). Initially there was a great deal of enthusiasm for the drug, until it emerged that the original trial data were flawed. When a proper controlled trial was eventually carried out sanocrysin was discredited (Amberson *et al*, 1931). In the meantime many patients had suffered its toxic effects. These included fever, albuminuria, loss of weight and skin eruptions (Smith, 1988). Despite its lack of clinical value many patients continued to be subjected to this treatment in the absence of any other drug.

The other major innovation in the treatment of tuberculosis was surgery, in particular the use of artificial pneumothorax and thorocaplasty. The operation for artificial pneumothorax had been performed since the early 19th century but only became popular in Britain in the 1920s. The principle of this operation was similar to that of bed rest: it aimed to allow the lung to rest and so heal itself. This was achieved by forcing air between the membrane of the lung and the chest wall causing the lung to collapse, and so rest. This operation became very popular, despite a long list of side effects which included pleural embolism, pleural shock, perforated lung leading to infection and pain, and sometimes death. About 50% of operations resulted in pleurisy (Smith, 1988). Doctors at the time had a great deal of faith in this operation, although what little evidence there is on its effectiveness would appear to show that this faith was misplaced. A MRC study of 1922 revealed 50% of patients were dead within 2.75 years of surgery (Burrell and Salisbury, 1922), with other studies claiming 70% were dead within 10 years (Smith, 1988). The other popular operation was thorocaplasty. This was a horrific procedure which involved the removal of part of the chest wall to affect a permanent collapse of the lung; one of its many side effects was to leave the patient hideously disfigured. In his extensive work on tuberculosis surgery, Smith was unable to find any reliable data on the effectiveness of this operation (Smith, 1988).

The period up to World War Two saw a major investment in the provision of care for tuberculosis sufferers. Evidence from the time suggests that, despite widespread belief in the effectiveness of the tuberculosis schemes, they achieved little. In Glasgow, a city with the second greatest bed provision in the UK, 73% of sputum positive cases notified between 1935 and 1938 were dead within 5 years and 83% within 9 years (MacFarlane, 1990). Similar results were achieved by the London council sanatoria, where 76% of patients treated in 1927 had died by 1932 (Bryder, 1988).

This thesis has considered the influence of the tuberculosis schemes on survival by calculating the percentage of deaths which took place amongst people known to have tuberculosis. Figure 6.7 shows trends over the period 1914-99, based on tuberculosis notifications (denominator), a proxy measure for tuberculosis incidence. For the period 1914–1938, the percentage of deaths barely changes, staying consistently over 70%, in line with the findings of MacFarlane and Bryder discussed above. This suggests that any decline in the death rate and the percentage of total deaths due to tuberculosis was not due to the curative effects of medicine. This is in spite of massive investment on the part of the local authorities and central government in the institutional care of tuberculosis. MacFarlane (1990) has suggested that it was precisely this investment which tied Scottish tuberculosis treatment into a system which was ineffective – the fact that they had invested so much meant they simply could not admit to failure.

Figure 6.7. Percentage of tuberculosis deaths to notifications, 1914-99*

*the data for 1915-20 & 1922-29 are estimates.

Source: Original analysis of records obtained from GROS and Scottish Centre for Infection and Environmental Health (SCIEH).

6.4.1.2. Standards of living (survival 1911-38)

It has been argued that the percentage of those suffering tuberculosis who went on to recover did not increase between the start of the 20th century and the outbreak of World War Two.

Therefore, although improving standards of living may have been directly linked to declining tuberculosis death rates, this could not have been by increasing cure rates amongst those who were suffering active disease.

6.4.2. Levels of infection (1911-38)

In order to develop active or clinical tuberculosis an individual must first have been exposed to the tubercle bacillus, without such exposure it is impossible for the disease to occur. This section will consider the influence of medicine and standards of living on the prevalence of infection with the tubercle bacillus in Scotland before World War Two.

6.4.2.1. Role of medicine (levels of infection 1911-38)

In the 1920s the BCG vaccine was developed in France to protect against infection with the tubercle bacillus. However, this was not employed in Britain until the late 1940s. The reasons for this are varied and will be discussed in greater depth in section 6.6.2.

One of the justifications for the tuberculosis schemes was that they removed infectious cases of tuberculosis from the community, thereby reducing the spread of infection. The value of this measure is dependent on the number of infectious cases left in the community. A good example of this is Glasgow, which, as has been discussed, had the largest bed provision in Scotland. Despite this, 75% of known cases of tuberculosis were still living in the community (MacFarlane, 1990). As so many cases were left untreated to spread disease, it is unlikely that the isolation of a minority of cases would have had a marked effect on the spread of infection.

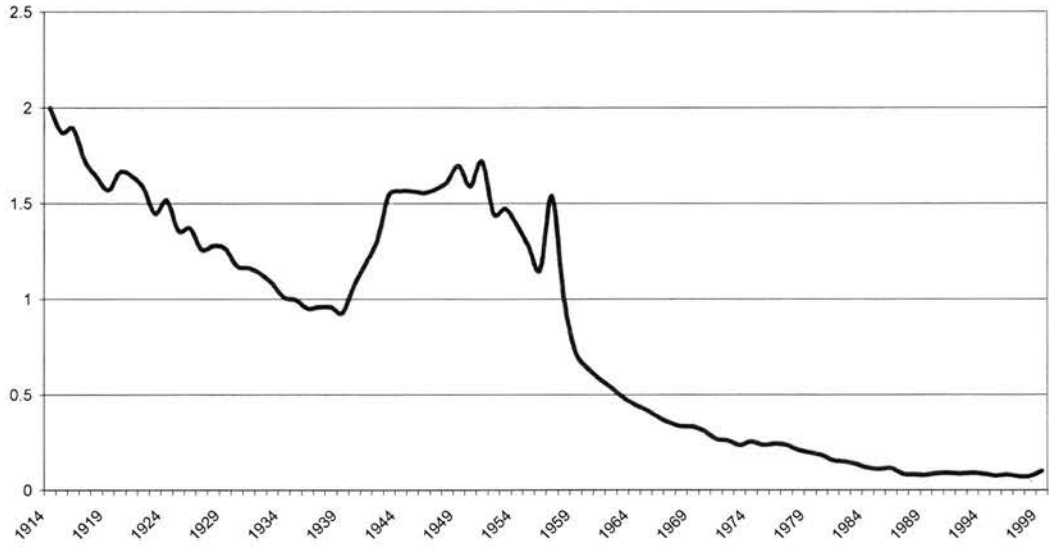
6.4.2.2. Standards of living (levels of infection 1911-38)

As the tubercle bacillus is air-borne, this means there is a direct link between overcrowding and the potential for infection. However, evidence of infection rates before World War Two indicate that by adulthood approximately 90% of the population had been exposed to the disease (Bryder, 1988). Therefore it is doubtful that declining levels of infection were responsible for the decline in the death rate seen at this time.

6.4.3. Failure to develop active disease after infection with the tubercle bacillus (1911-1999)

It has been argued here that, until the outbreak of World War Two, the decline in tuberculosis was not due to a greater proportion of those who developed active disease recovering or a reduction in the number of people being exposed to the infection. This leaves us to consider the final way in which it is possible to avoid death from tuberculosis, which is by not developing the disease once infected. The main determinant of why an individual develops tuberculosis is the state of the immune system, with a healthy immune system affording a greater degree of protection. The notification rate for the 20th century shown in figure 6.8, provides an indication of the level of active disease in the population. The defining trend in the pre-war period was downwards, pointing to a decline in the number of people going on to develop active disease. Thus, it can be argued that the reason for the decline in mortality during this period was that fewer people were going on to develop clinical disease.

Figure 6.8. Tuberculosis notification rate, per 1000, 1914-99



Source: Original analysis of records obtained from the GROS and SCIEH.

6.4.3.1. The role of medicine (levels of active disease 1911-38)

The focus of medicine at this time was upon curing those already sick, with little attention being paid to preventing others from falling sick. There is no evidence of any direct contribution from medicine in this area.

6.4.3.2. Standards of living (levels of active disease 1911-38)

Poverty has historically been recognised as a risk factor for tuberculosis. The link between poverty and tuberculosis has been confirmed by more recent studies of tuberculosis in Britain (Spence *et al*, 1993, Darbyshire, 1995, Hawker *et al*, 1999). However, what is it about poverty which makes an individual more susceptible to tuberculosis? Associated with poverty are the twin effects of poor diet and poor housing. Since diet was isolated by McKeown as the single greatest influence on the decline of tuberculosis, it will be considered first.

The link between malnutrition, a weakened immune system, and tuberculosis has long been recognised. Examples of this include a study of US Navy recruits which found that being overweight offered protection against tuberculosis, with recruits who were 10% or more underweight being 3.4 times more likely to be suffering from tuberculosis than those who were 10% or more overweight (Kumaresan, 2002). Thus, individuals who are not underweight have a greater resistance to developing clinical tuberculosis.

The period from the late 19th century through to the 1920s has been identified by MacFarlane (1990) as a time when the diet of the majority of the Scottish working class improved. He relates this to a trend of rising real wages, which were of particular importance in the west of Scotland where heavy industry was flourishing. The diet of the poorest 10% of the population at the end of the 19th century was appalling, with many surviving on bread and potatoes, with only a little meat and fat (Nelson, 1993). Lack of nourishment would have increased population susceptibility to clinical tuberculosis. This experience was gradually reversed for many, although huge disparities still existed between the diets of the richest and the poorest. Improving wage rates in combination with increasing imports of foodstuffs were improving both the quantity and variety of the working class diet. Evidence of changing diet is shown in table 6.2 (these data refer to the UK but provide an indication of consumption patterns in Scotland). Table 6.2 reveals a decline in the consumption of cheap staple foods such as potatoes and bread and an increase consumption of luxury items such as butter (associated with greater spending power).

Table 6.2. Consumption of selected food groups (in lbs), per person, per year, in the UK

	1880	1909-1913	1934-1938
Potatoes	296	243	190
Bread	280	211	195
Butter	12	16	25
Meat	91	131	129

Source: Adapted from Greaves and Hollingsworth (1966).

This improvement in choice was translated into increased calorific consumption, with an increase in average daily intake from 2760 in 1909-13 to 2810 in 1924-8 and 3050 by 1934-8 (Greaves and Hollingsworth, 1966).

It is therefore evident that, on average, the quantity and variety of the Scottish diet had improved in the period up to World War Two. However, it is also necessary to establish if protection against tuberculosis was directly conferred by eating greater quantities of food, or whether there was a benefit to be derived from eating specific types of food. Within the sanatoria and hospitals a substantial diet rich in meat, fat, eggs and milk was recommended. A recent study has established a link between the consumption of meat and tuberculosis (Russel, 2003). This study found that certain fatty acids in meat can prevent the M. TB disabling the body's cells and so allow the body's defence system to work more effectively. These findings are supported by evidence that the incidence of tuberculosis is higher amongst those who follow a strict vegetarian diet (Kumaresan, 2002). If it is accepted that meat provides protection against developing tuberculosis, which goes beyond the provision of nourishment, the consumption of meat during this time must be considered. From the 1880s there was a marked rise in meat consumption facilitated by the introduction of large-scale importation of frozen carcasses (Nelson, 1993). This resulted in the average consumption of meat per person per year increasing from 91lbs in 1880 to 131 in 1909-13 and 129 in 1934-8 (see table 6.2). In addition, the consumption of visible fats (e.g. lard) quadrupled between 1880 and 1934-8. (Greaves and Hollingsworth, 1966). Arguably therefore, the increased consumption of meat may have increased resistance to tuberculosis. The consumption of meat has also been highlighted by Gibson and Smout as an indicator of standards of living (Gibson and Smout, 1993).

Overcrowding is another aspect of standard of living which has been acknowledged to affect the tuberculosis death rate. Overcrowding affects risk by increasing an individual's potential for exposure to the tubercle bacillus. However, it has been argued in this thesis that infection with the bacillus was virtually universal by adulthood in the period before World War Two. Therefore it is unlikely that improvements in overcrowding could have led to a decline in the death rate by reduced exposure. However, overcrowding can also affect the likelihood of developing clinical disease by suppressing the immune system (Lees, 1978). Evidence of the effect of overcrowding on the tuberculosis death rate is shown in table 6.3 which illustrates the increase in risk with the reduction in apartment size.

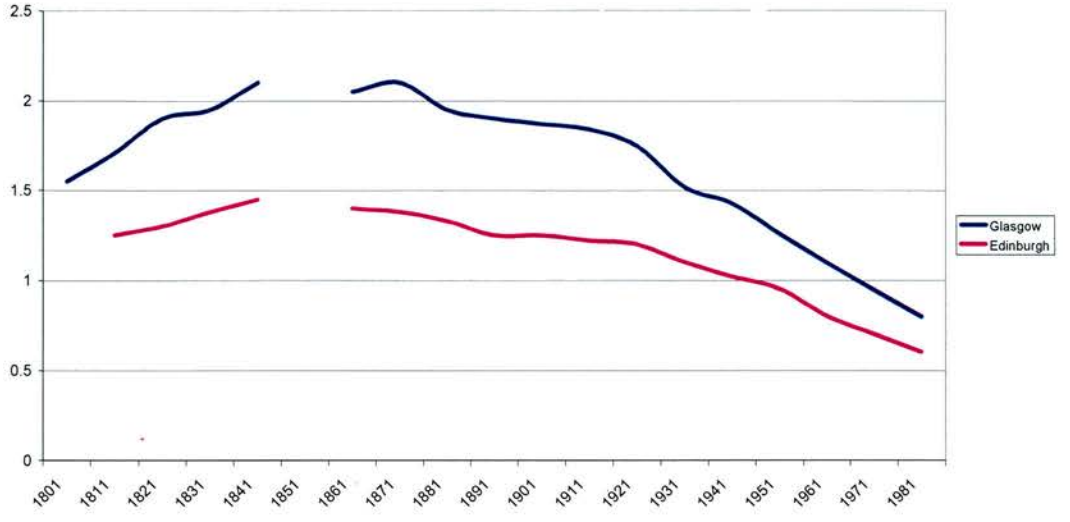
Table 6.3. Tuberculosis death rate, per 1000, 1901, by number of rooms in dwelling (Glasgow)

Number of rooms	Death rate per 1000
1 apartment	2.5
2 apartment	1.8
3 apartment	1.2
4 apartment	0.7

Source: Adapted from A.K. Chalmers (1930).

Williams (1994) has examined overcrowding levels in Scotland from the start of the 19th century until the 1980s. Using census data he has examined the trends in the number of persons per room in Glasgow and Edinburgh throughout this time period, shown in figure 6.9.

Figure 6.9. Persons per room, Glasgow and Edinburgh, 1801-1981*



* data for 1801-1841 are estimates.

Source: Adapted from Williams (1994).

One of the most striking features of this graph is the large increase in persons per room in Glasgow during the 19th century. Williams has attributed this to the rapid industrialisation of the city, combined with the influx of immigrants from Ireland and the Highlands. This surge of immigrants not only increased the population of the city, but led to a low wage economy with the majority able to afford only low rent accommodation. As a result the city became hugely overcrowded, with a lack of finance to encourage new building. The overcrowding of Glasgow may help to explain the fact that the city had the highest tuberculosis death rate in Europe. An improvement in overcrowding can be detected from the late 19th century. Williams has linked this to the improvement in real wages which was seen from this time. This may have contributed to the improvement in resistance to tuberculosis. However, the rate of improvement in overcrowding was slight before the war and Glasgow in particular was infamous for its dreadful housing conditions.

6.4.4. Conclusions 1911 – 38

During the period 1911–1938 there was a downward trend in tuberculosis mortality. It has been argued that this decline did not result from a larger proportion of those with tuberculosis recovering from the disease, despite the vast investment in medicine at the time. The evidence of infection levels reveals that until World War Two infection was ubiquitous among the adult population. Thus, the main reason why mortality declined at this time, was that fewer people were developing the disease once infected. There is no evidence to show that medicine contributed in any way to this trend. Rather, in agreement with McKeown, it can be concluded that improving standards of living and in particular improvements in diet played the more significant role.

6.5. 1939 – 47 The Second World War and its aftermath

The dominant trend of the war years and the years immediately following was one of increasing levels of tuberculosis. This affected both the death rate (see figure 6.5) and the number of people developing the disease (see figure 6.8). This phenomenon was not experienced in England and Wales. Central to this trend was Glasgow. The city's population had always suffered high tuberculosis mortality. Glasgow's position relative to the rest of Scotland worsened considerably, so that by 1940 41% of all pulmonary deaths occurred there, as compared to 28% in 1920 (MacFarlane, 1990). In 1943 the notification rate stood at 2.8 per 1000 in Glasgow as opposed to 1.5 in the rest of Scotland. The Glasgow death rate peaked at 1.4 per 1000 in 1948, which was almost double the national and Edinburgh rates (Lees, 1978).

As the war years were dominated by increasing mortality the task for this thesis is to identify why this was the case. This is of use in the study of the post-1950 period as it highlights what factors contributed to excess mortality. Central to this will be conditions in Glasgow which had an adverse effect on national figures.

6.5.1. Survival (1939-47)

6.5.1.1. The role of medicine (survival 1939-47)

During the war the first major breakthrough in tuberculosis chemotherapy occurred, with the discovery of streptomycin in 1943. However, as this drug was not introduced in the UK until 1948, it will be discussed in a subsequent section.

In section 6.4.1.1 it was stated that one way of measuring the effectiveness of medicine in curing those with clinical tuberculosis is to consider those who die of tuberculosis as a percentage of the number of notifications. Figure 6.7 reveals what at first appears to be an odd result, which is that the percentage dying appears to be declining throughout the war years. This was in absence of any appreciable advance in medical treatment. A possible explanation for this can be found when the number of notifications are reviewed (see figure 6.8). The number of notifications rose sharply during the war. Tuberculosis is a chronic disease, with many sufferers surviving for a number of years before succumbing to the disease. These new cases would still have been in the early stages of the disease during the war years and so would not have been adding significantly to the death rate at that time. Rather, although total deaths were increasing, they were not doing so at the same rate as

notifications, causing the apparent rise in the percentage who survived the disease. It is unlikely that, during the war years, the actual percentage of those going on to recover from tuberculosis increased.

This increase in incidence of the disease placed an additional demand on an already overstretched resource. As we have seen, the number of notifications of cases of clinical tuberculosis increased considerably, so that between 1945 and 1948 the number of patients awaiting treatment in Scotland rose by 51%, while the number of beds increased by only 5% (MacFarlane, 1990). This situation was exacerbated by a crisis in nursing, especially amongst tuberculosis nurses. This trend had begun in the 1930s. Tuberculosis nursing was unattractive for a number of reasons, including fear of infection, poor status, lack of recognition of training, and the fact that many of the tuberculosis institutions were in isolated locations (MacFarlane, 1990). However, it has been argued that, in the earlier period, when the tuberculosis death rate was declining, medicine did nothing to bring about the decline in the death rate. Therefore, it is unlikely that any *failing* in the system would actually have worsened the cure rate.

6.5.1.2. Standards of living (survival 1939-47)

It was stated in section 6.4.1.2, dealing with the pre-war period, that standards of living exerted little influence on prognosis once clinical disease had developed. This would have remained the case during the war years.

6.5.2. Levels of infection (1939-1947)

Prior to the Second World War the infection rate amongst the adult population was estimated to be around 90%. As no new medical innovations were introduced during the war, medicine would not have influenced this rate. The main determinant of infection levels was housing conditions, and the following section will discuss their deterioration over the period. This may have led to an increase in infections. However, as the majority of the population was already infected, it is unlikely that deterioration in conditions could have exacerbated the situation.

6.5.3. Failure to develop clinical tuberculosis after infection with the tubercle bacillus (1939-1947)

It has been argued that the rise in tuberculosis mortality seen in Scotland during the war was unlikely to have been caused by the deterioration of medical provision causing fewer patients

to recover from disease, as under peace time conditions the tuberculosis services had failed to influence prognosis. In addition, the conditions of war make it highly unlikely that infection levels would have declined. Thus, the reason for the war time surge in mortality must have been due to the reversal of the trend seen before the war – that is that more people were 'breaking down' with clinical disease. The explanation for this lies in the state of the immune system, and the weakening of defence mechanisms. As the role played by medicine remained unchanged from the pre-wars years, medicine could not have been responsible for increasing mortality. This leaves us to consider changing standards of living.

Diet was selected as the main determinant of the pre-war decline in tuberculosis. Therefore this thesis will consider whether worsening diet was the cause of the increase in tuberculosis during the war. The main feature of the wartime diet was the introduction of rationing. In the inter-war period a plan had been devised to deal with food shortages in the event of another war, allowing rationing to be introduced relatively rapidly after the outbreak of hostilities. This began with the rationing of specific items from January 1940. By December 1941 a points or coupon system had been introduced which allowed the consumer to purchase what they pleased, dependent on the number of coupons they had in their book (Buss, 1990). In addition to rationing, other measures were introduced by the government to ensure the nation was properly fed, including free school milk and school meals, with 1.6 million school children being in receipt of these by 1945 (Buss, 1990).

One area where people may have experienced a particular shortage compared to the pre-war years was in meat consumption. Average meat consumption amongst the working classes fell from 33.8 oz in 1936-7 to 18.8oz by 1944 (Buss, 1990). If meat does afford a degree of protection against tuberculosis this may have had an effect on incidence rates. However, in general the wartime diet has been widely praised as being healthy and nutritious, as well as promoting equality between the social classes (Zweiniger-Bargielowska, 2000). In addition if there had been a serious decline in quantity or quality of the diet this should have affected the whole population of the UK, and their tuberculosis death rate. However, it was only Scotland which saw an increase in mortality. This would suggest that diet could not have been the cause of Scotland's escalating tuberculosis rate.

An alternative explanation for the excessive mortality of World War Two and its aftermath has been advanced by MacFarlane, who placed Glasgow and its housing stock at the centre of the war-time surge in tuberculosis. In section 6.4.3.2 the role of standards of living on

population resistance to clinical tuberculosis was considered. It was argued that the decrease seen in overcrowding in Glasgow and Edinburgh during the first decades of the 20th century may have helped increase resistance. However, although there had been some improvement, on the eve of the war Glasgow, in particular, was still very overcrowded, with on average over 1.5 persons for every room (see figure 6.9). The conditions of war confounded the problems of urban overcrowding as people transferred to industrial centres to work in the munitions factories. In addition, the war drew a halt to any building work which may have alleviated the overcrowding problem. This view of Glasgow as a city whose overcrowding problem significantly worsened during the war would appear at first to contradict the trend which is shown in figure 6.9. This graph indicates a downturn in overcrowding in both Glasgow and Edinburgh between 1931 and 1951. However, this graph is compiled from data gathered by the census, and in 1941 there was no census in the UK. Therefore the apparent downturn seen in figure 6.9 may have been the result of improvements seen after the end of the war. Although overcrowding is normally associated with urban areas, there is evidence that during the war conditions also deteriorated in some rural areas. An example of this is Tarbert, Loch Fyne. In Tarbert the increase in over-crowding has been linked to the influx of children who were 'boarded out' to avoid the war-time dangers of the cities, as well as the re-location of the Clyde fishing fleet, to an area which was already over-crowded before the out-break of hostilities (Macintyre & Macdonald, 2000). Thus, if large sections of the population experienced a deterioration of their living conditions during the war, this may explain the increase in mortality seen at this time.

6.5.4. Conclusions (1939-47)

This study has concluded that, in the absence of effective chemotherapy, the only way in which deaths from tuberculosis were reduced was by fewer people developing the disease. Once disease was established there was nothing which medicine could do to alter the course of the disease for the majority, in spite of the vast sums being spent on tuberculosis schemes for the institutional care of sufferers. Medicine neglected the one area where something may have been achieved, that is, the improvement of conditions for those already infected to prevent individuals 'breaking down' with active disease. One of the greatest failings of medicine was, that by concentrating all its resources on expensive, ineffective institutions, it failed to contribute to improving social conditions. Thus, there is evidence that only two factors could have influenced the tuberculosis death rate in Scotland before 1948: improving diet (declining death rates), and poor housing conditions (increasing death rates).

6.6. 1948 – 1999 The chemotherapy era

Since 1948 tuberculosis has been a disease in a state of continual decline. This began rapidly (see figure 6.5), so that by the 1960s the death rate for males was only 0.1 per 1000 and 0.05 among females, with the share of total mortality attributable to tuberculosis reduced to just 1% among males and 0.4% among females in 1960. This section will consider whether this was the result of the new chemotherapy drugs which became available at this time, or whether improving standards of living, by reducing the number of people succumbing to the disease, were still making a contribution.

6.6.1. Survival (1948-1999)

For the first time a substantial decline in the percentage of deaths to notifications took place in the late 1940s and 1950s (see figure 6.7). This decline is linear and steep until the late 1950s when it plateaus at roughly 15% deaths to notifications, before a second drop to below 10% in the late 1970s. These figures are confirmed by contemporary reports which claimed that the period 1950-60 ultimately saw only 15%, instead of the previous 74%, of those treated for pulmonary tuberculosis die from progression of the disease (Hawthorne, 1969). For the first time the decline of the tuberculosis death rate was affected by recovery among people who had clinical disease, as opposed to the pre-1948 trend when the only influential factor was the number of people developing disease.

It has been suggested in earlier sections that, although the tuberculosis death rate was declining, improving standards of living could do little to alter the prognosis of those who had already developed clinical disease. Therefore, standards of living can be discounted in trying to explain the post-1948 increase in survival. This leads us to consider the role of medicine. The main medical treatments for tuberculosis are listed in box 6.1. It has been argued in this thesis that, in the earlier period, medicine had no impact on the decline of tuberculosis. The introduction of streptomycin in 1948 marks a watershed, as this was the first drug to be effective in the treatment of tuberculosis. Streptomycin was discovered in 1943 by Selman Waksman at Rutgers University in the USA after an intensive search to find a cure for tuberculosis (see Sakula, 1988). The discovery of streptomycin was initially greeted with a degree of scepticism, given the disappointment of earlier tuberculosis drugs such as sanocrysin. The MRC launched trials of the drug in 1947. These trials are not only significant to the history of tuberculosis but also to the history of clinical trials due to their

innovative approach to randomisation. Within the streptomycin group a considerable or moderate improvement was seen in 69% of cases at six months, whilst the control group saw an improvement in only 33% of cases. In addition only 18% of the streptomycin group experienced considerable deterioration or death as compared to 38% of the control group (Streptomycin treatment of pulmonary tuberculosis, 1948). This represented the first breakthrough in the medical treatment of tuberculosis. By 1948 streptomycin was being used on tuberculosis patients in Scotland and by 1949 a domestic supply of the drug had been established (Bryder, 1988). The effect of this upon the survival rates of those suffering tuberculosis is immediately evident in figure 6.7: the death rate, which had already seen some recovery from its war time high, entered a steep decline.

Box 6.1. Medical advances in the treatment of tuberculosis

1880	Discovery of the tubercle bacillus
1910-1950	Era of the sanatoria and TB hospital
1924	Sanocysin
1920/1930s	Rise of surgery
1943	Streptomycin discovered
1946	PAS
1947	Start of MRC streptomycin trial
1948	Introduction of streptomycin
1949	Start of BCG trial
1953	Isonaizid
1956	Control trial of bed rest
1957/1957	Mass mini radiography national campaign
1968	Ethambutol
1969	Rifmapacin
1977	MRC trial reduces treatment time from 18/24 months to 6 months

Streptomycin, however, was not without drawbacks. The drug had to be delivered intramuscularly, which made its administration difficult. Many patients also experienced toxic side effects, which included giddiness, problems with vision, and nausea. The most serious side effect was that a sizeable minority developed resistance to the drug. This occurred in several of the trial patients who died; they had initially done well but then experienced relapse (Streptomycin treatment of pulmonary tuberculosis, 1948).

The discovery of streptomycin was followed by several other drugs including PAS and isonaizid, which was a highly effective drug. These were later joined by ethambutol and rifmapacin in the late 1960s (see box 6.1). Together these drugs were theoretically capable of curing 100% of tuberculosis. However, problems still remained with cure rates in the 1950s, with the gold standard of 95% remaining elusive. A number of factors were responsible for this. Firstly, the length of the treatment was long (18–24 months), and the continued reliance on bed rest led to poor patient compliance. Patients would start their course of treatment and begin to feel better and so default, only to relapse later. Secondly, there was also the continuing problem of drug resistance (Girling, 1978).

These problems were gradually overcome through a series of controlled trials which demonstrated the best use of the available drugs. By 1956 it was shown that the outcome of a case was not affected by bed rest. This allowed patients to remain at home and in many cases maintain a normal life while undergoing treatment, thus reducing the rate of default. The degree to which this was put into practice can, however, be questioned. Out of a sample of cases notified in Scotland during 1968, the mean duration of hospital stay was still 4.2 months (Heffernan *et al*, 1977). 'Good chemotherapy' was developed in Edinburgh by John Crofton and his colleagues. They developed a system of combining drugs, which could turn all sputum positive (infectious) cases negative within eight months, and claimed a cure rate of 100% (Crofton, 1985). The introduction of rifmapacin in 1969, a drug of equal strength to isonaizid, signalled the end of prolonged treatment. In 1977 the East Africa and British MRC study reported on their trial of modern chemotherapy. The addition of rifampacin to isonaizid and streptomycin allowed a dramatic curtailment in the duration of drug therapy to six months, eliciting cure rates of over 95% (East African/ British Medical Research Council Study, 1977).

The period following 1950 witnessed a revolution in the medical treatment of tuberculosis. Gone were the prolonged confinements in institutions which offered little hope of permanent

recovery. Instead the new drugs offered a cure in the vast majority of cases. As the years progressed the treatment was to become increasingly less invasive. The current treatment recommended by the British Thoracic Society is only six months on a combination of different drugs (British Thoracic Society, 2003).

In tandem with the development of the new drug regimes a plan was devised to locate undiagnosed cases within the community so that they too could be treated. This took the form of mass miniature radiography (MMR), which allowed large numbers to be screened for pulmonary tuberculosis by mobile x-ray machines. MMR was originally developed in the 1930s and was initially used to screen recruits for the army (Bryder, 1988). Its use in the civilian population gradually increased from 35000 screened in 1945 to 438651 in 1956 (Carstairs and Howie, 1972). The years 1957 and 1958 marked the height of the use of MMR as the government launched a national campaign of screening in Scotland. MMR units were sent all over Scotland to locate undiagnosed cases, with all areas, including the Isles, receiving a visit. This campaign received massive press attention, newspapers called on the population to play their part in the war against tuberculosis, "*Every man, woman and child in Scotland has a part to play in the fight against tuberculosis. Already tuberculosis is on the run. Its defeat can only be hastened if we all join in the war*" (Campbletown Courier, 1958). Further encouragement was offered in the form of prizes for a random selection of those who went along to be x-rayed. By the end of 1957, 1319867, or over 1/5 of the population had been x-rayed (Carstairs and Howie, 1972). The effect of this is reflected in the notification rate, which peaked in 1958 as previously undiagnosed cases were discovered (see figure 6.8). The rapid decline seen in the notification rate after this date owes a great deal to the number of early cases which were located by the mass drive. MMR's role in locating cases was significant, with 30% of all notifications in 1956 and 48% in 1957 being located by this means (Carstairs & Howie, 1972).

Through the 1950s medicine would finally appear to have come to grips with tuberculosis. Not only was an effective means of curing the disease developed, but this was combined with a 'medically created' mechanism to locate cases while still in the early stage of the disease.

6.6.2. Levels of infection (1948-1999)

Between 1950 and 1999 Scotland made the transition from a country where infection with tuberculosis was virtually universal by adulthood, to one where it was rare amongst those born during this time. This coincided with the introduction of the BCG vaccination to protect against infection. BCG was not a new vaccine; it had been developed in France in 1923, by Albert Calmette and Camille Guerin and had been used in France since 1924 as well as being adopted by the League of Nations health committee. Its introduction was delayed in the UK for two reasons. The first was the Lubeck disaster of 1930, where 249 newborn babies had mistakenly been given virulent bacillus instead of BCG, leading to the death of 76 infants (Bryder, 1988). This event had caused many to question whether the vaccine should be used. In addition, many in the UK tuberculosis establishment were hostile to the use of BCG, arguing that its effectiveness was unproven and preferring instead to continue with the system of institutional care. The impact of the war brought matters to a head. The spiralling rates of tuberculosis led to increased pressure on beds, which, combined with a shortage of nurses within the tuberculosis institutions, led to calls for the usefulness of BCG to be investigated. This resulted in BCG being seriously considered for the first time and the Ministry of Health setting up a committee to advise on clinical trails of BCG. The controlled trial of BCG was launched in 1950 and used on a sample of male and female school leavers. In the sample, followed up over fifteen years , there was a 78% reduction in tuberculosis amongst those vaccinated as compared to the tuberculin negative (never previously exposed) group who were not vaccinated (Springett, 1978). The initial good results of the trial led to BCG being offered to all tuberculin negative school children at age 13 from 1954 (Capewell *et al*, 1986).

It is therefore evident that BCG is an effective method of protection against infection with tuberculosis. However, it is more difficult to measure the extent to which it was responsible for the decline in the population's infection levels. The value of BCG is dependent upon exposure to a person who is infectious. If an individual never comes into contact with an infectious case of tuberculosis there is no need for the vaccination. BCG was introduced at the same time as chemotherapy and an effective case finding mechanism in the form of MMR. These measures would have gone a long way to reducing the residual pool of infectious individuals, so helping to limit the number of new people exposed to the infection. In their audit of BCG, Pell and Capewell (1996) questioned the role which BCG played in the decline of tuberculosis notifications, attributing only 10% of the overall decline in

notifications to this measure. Instead, they highlighted the role played by chemotherapy and mass screening in reducing the pool of infectious individuals (Pell and Capewell, 1996). Despite this they still found that BCG was a useful tool and recommended its continued use on 13 year olds. The routine administration of BCG to school children was eventually withdrawn in 2005, in favour of a policy which targets those at greatest risk of exposure to infection.

6.6.3 Failure to develop active disease after infection with the tubercle bacillus (1948-1999)

It has been argued in this thesis that in the pre-chemotherapy era the main factor which influenced the decline of tuberculosis mortality was the decline in the development of tuberculosis following infection, and that the main influence on this had been standards of living. Given this, it is not possible to attribute the entirety of the post-1950 decline to the presence of effective drugs and vaccines without first considering any continuing input from improved standards of living. Figure 6.5 reveals that the start of the decline of the tuberculosis death rate from its post war-high had begun before 1948 and the introduction of streptomycin, indicating that some other influences were at work. The remainder of this section will consider the extent to which standards of living contributed to the steep decline in mortality after 1948.

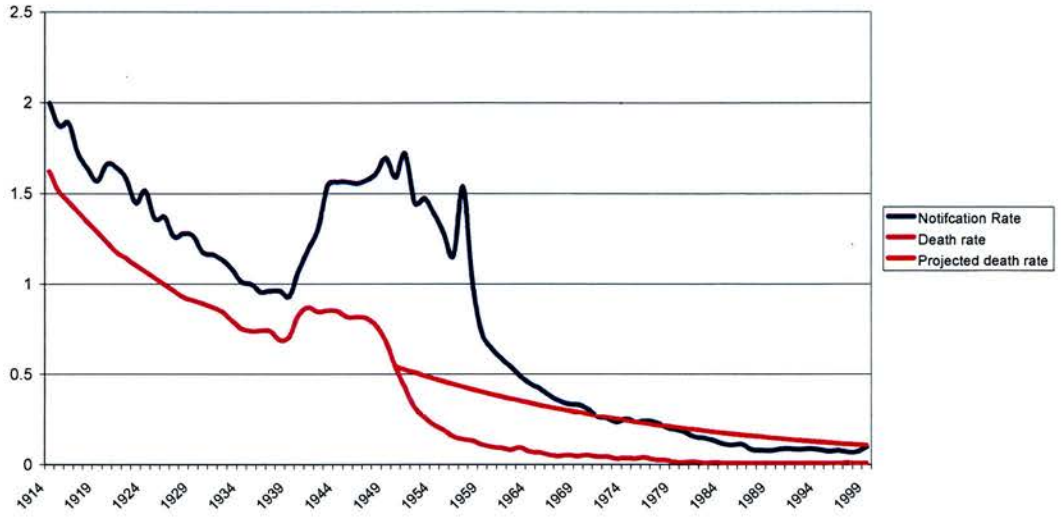
The 1950s witnessed an increase in standards of living for the vast majority of the population. Diet in particular improved, at least in respect of preventing clinical tuberculosis. Rationing continued in the UK until 1954. Its halting led to a rapid increase in consumption of items which had previously been scarce, in particular white bread, sugar, butter, eggs, meats and sweets (Buss, 1993). The consumption of meat amongst the working class in particular rose from an average of 11.8oz per person in 1951, to 34.2oz by 1959. (Buss, 1990). Given the link between the consumption of fatty produce and tuberculosis in the pre-1950 period, it is probable that this would have strengthened the immune systems of some individuals susceptible to tuberculosis.

The role of poor housing in Glasgow has been highlighted as the main factor influencing the war time surge of tuberculosis in Scotland. Therefore, it would be anticipated that any improvement in housing conditions would result in an increase in resistance to clinical tuberculosis. In figure 6.9 a rapid decrease in persons per room is evident in the post-war period, indicating that over-crowding was reduced significantly in the post-war period.

It is difficult, however to assess the extent to which the post-1948 decline of tuberculosis mortality was due to medical measures to locate and cure cases of the disease, or improvements in diet and housing which may have increased resistance to the disease. This thesis has attempted to overcome this problem by considering what would have happened to the tuberculosis death rate in the absence of effective medical measures. Figure 6.10 displays the tuberculosis death rate and notification rate between 1914 and 1999. The red line on the graph is the projected post-1948 death rate, if the pre-war gradient of decline had been re-established. This line was generated by calculating the average percentage decline in the death rate, per year, in the period 1911-1938, the percentage decline was then calculated for each year following 1948. The line represents what would have happened to the death rate of medical innovations for the treatment and discovery of tuberculosis had not been introduced. In 1999 the difference between the actual death rate (0.01 per 1000) and the projected death rate (0.11 per 1000) is relatively small. However, the main difference between these death rates can be seen in earlier decades. By 1959 the real tuberculosis death rate had declined to 0.11 per 1000, as compared to 0.4 per 1000 for the projected death rate. The difference between these two rates represents the number of lives which were saved by the introduction of effective medical treatments for the prevention, location and treatment of tuberculosis.

Estimating what would have happened if the pre-war pattern had been re-established provides an indication of trends in the death rate if chemotherapy had not been developed, but standards of living had begun to improve. If the difference between the actual and projected death rates in 1999 is considered there appears to be little difference in the death rate. The large increase in the notification rate during and after the war should also be noted. It has been shown that before the introduction of chemotherapy approximately 70% of those who developed the disease went on to die of it (see figure 6.7). The progression of the disease amongst those notified would also have influenced the post-war death rate.

Figure 6.10. Tuberculosis death and noification rates, per 1000, 1914-99, and the projected tuberculosis death rate, per 1000, 1948-99



Source: Original analysis of records obtained from the GROS and SCIEH.

6.7. Conclusions

When considering the relative contribution of medicine and standards of living to the decline of tuberculosis since 1950, it is impossible to ignore the earlier time period. This provides both a context for the later decline and also evidence of what, in addition to chemotherapy, could influence the decline. If the post-1950 period alone were considered, this could lead to the acceptance of a monocausual explanation, as the new chemotherapy was claiming cure rates of up to 100%. In reality the explanation for the decline of tuberculosis mortality in Scotland is more complex. Evidence from pre-1950 reveals that, in the absence of chemotherapy, the only factors which exerted a significant effect on mortality were the influence of diet and housing on the immune system, determining whether an individual would be able to resist developing clinical disease. These influences would not simply have disappeared in the chemotherapy era. What is more difficult to determine is the extent to which they would have contributed in the face of the first effective medical response to tuberculosis, which consisted of prevention through BCG, case finding through MMR and cure by chemotherapy. The best place to look for answers to this is the late 1940s and 1950s, and envisage what would have happened if chemotherapy had not come along at that time. Scotland, together with Portugal, shared the unenviable role of leading the European league table in tuberculosis deaths, with Glasgow having the highest death rate of any European city. Although diet may have been acceptable, and improving once rationing ended, this would have done little to answer the immediate problems of excessive tuberculosis mortality in a city where housing conditions could only improve slowly. Although the long term trend of declining tuberculosis mortality would no doubt have re-established itself once Scotland recovered from its wartime epidemic, it is unlikely that it could have done so as rapidly as was seen once chemotherapy was introduced. Without the introduction of chemotherapy it is likely that mortality would have remained high. Perhaps 70% of the excessive wartime notifications would have gone on eventually to die (as they had in the earlier time period).

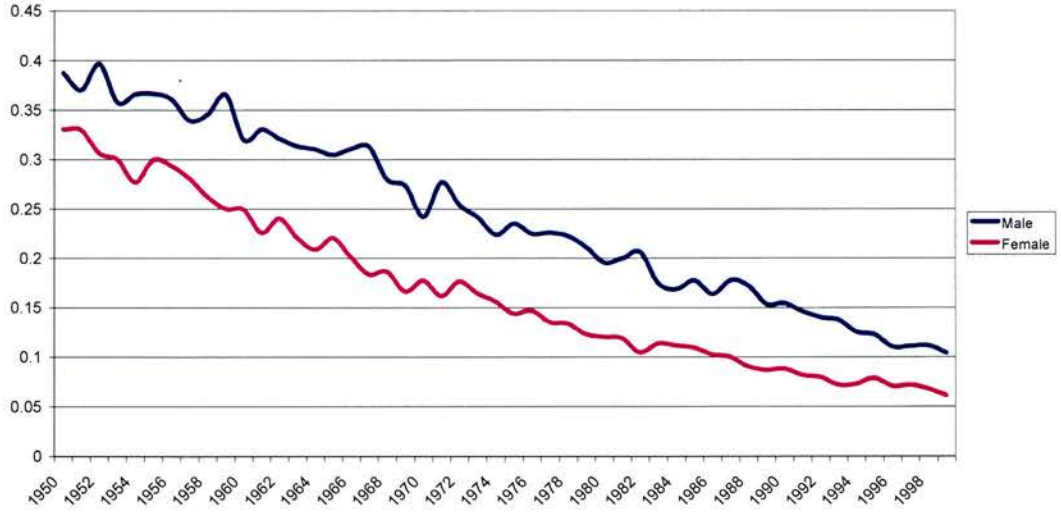
In terms of the three groups of influence on mortality which were defined in box 3.2 the majority of the decline from tuberculosis post-1950 can therefore be attributed to group 1 (medical interventions delivered at an individual level). Pre-1950 the main influences on mortality belonged to group 3 (standards of living), however, post-1950 this would only have exerted a minor influence on the mortality decline.

7. Stomach Cancer

7.1. Introduction

Stomach cancer is the second leading cause of cancer death in the world, killing 776000 in 1996 (WHO, 2003c). In 1950 stomach cancer was the second most common type of cancer in Scotland, exceeded only by lung cancer. By 2001 it was ranked as the fifth most common cancer amongst men and the seventh amongst women (ISD, 2005). Since 1950, death rates for stomach cancer, amongst both males and females, have been in constant decline. These rates are displayed in figure 7.1 which reveals that, between 1950 and 1999, the death rate per 1000 amongst males declined from 0.39 to 0.1, whilst female rates declined from 0.33 to 0.06. During this time period stomach cancer was the fourth largest contributor to the decline in all-cause mortality in Scotland (see chapter 5).

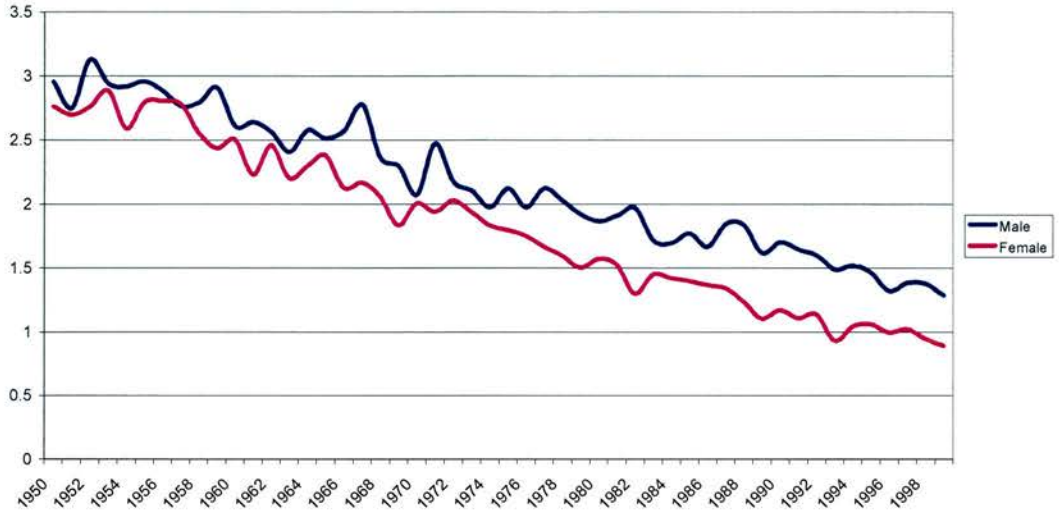
Figure 7.1. Stomach cancer death rates, per 1000, standardised to the 1950 population, 1950-99



Source: Original analysis of records obtained from the GROS.

The decline in the stomach cancer death rate is reflected in the reduction in the percentage of all-cause mortality which was attributable to this cause (see figure 7.2). In 1950 2.96% of all male deaths, and 2.76% of female deaths, were due to stomach cancer. By 1999 this had declined to 1.29% and 0.89% respectively.

Figure 7.2. Percentage of total deaths attributable to stomach cancer, 1950-99



Source: Original analysis of records obtained from the GROS.

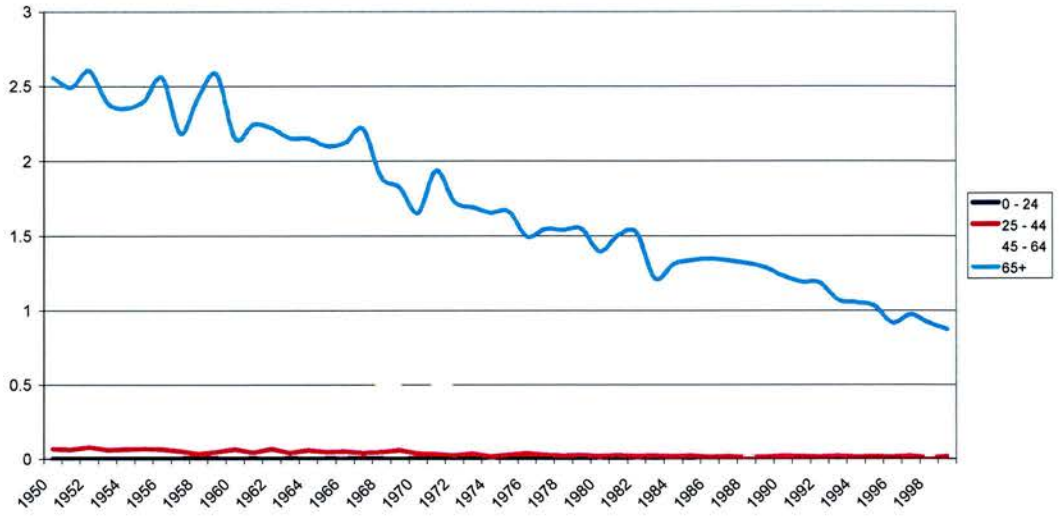
This chapter will review the factors which have influenced the decline in mortality from stomach cancer. Section 7.2 will provide background information for the disease, including age groups affected, symptoms and risk factors. The pattern of decline in stomach cancer will then be considered; this will isolate when stomach cancer mortality first began to decline in Scotland. The relative contribution of medicine and standards of living to either increasing cancer survival or declining disease incidence will then be reviewed.

7.2. Background

7.2.1. Age groups affected

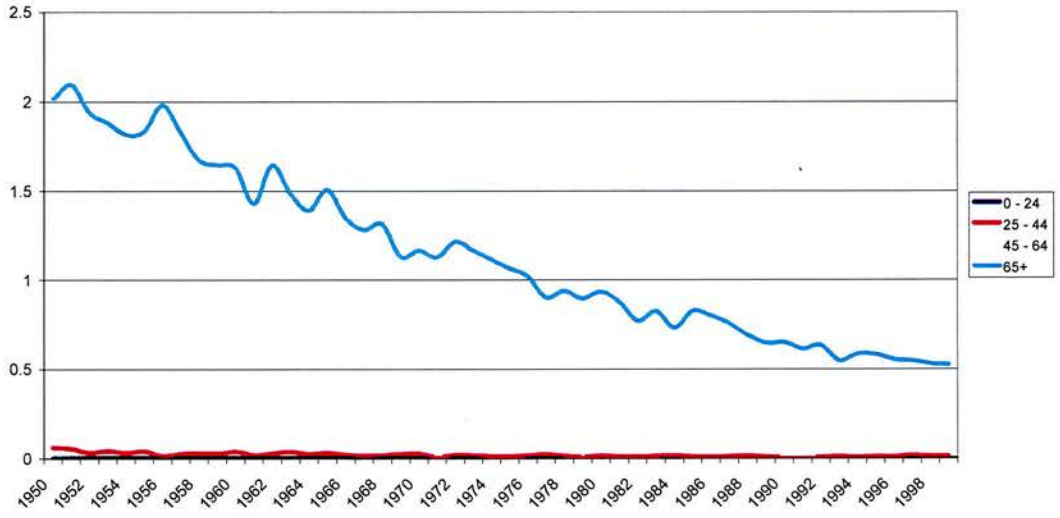
Stomach cancer is a disease which is associated with the elderly and is rare below the age of 40 (Bowkes & Benjamin, 2001). This is reflected in death rate by age group trends shown in figures 7.3 and 7.4. Death rates amongst the under 45s are very low, with deaths amongst the under 25s almost unknown. Amongst the 45-64 year olds in 1950 the death rate was 0.7 per 1000 amongst males and 0.4 amongst females. These death rates declined to 0.1 among males, and 0.01 among females, in 1999. It is the over 65s who experienced the highest death rates in 1950 and the steepest decline in mortality. Among males the death rate declined from 2.6 per 1000 in 1950, to 0.9 in 1999, while females experienced a decline from 2 per 1000 to 0.5.

Figure 7.3. Male stomach cancer death rates, per 1000, by age group, 1950-99



Source: Original analysis of records obtained from the GROS.

Figure 7.4. Female stomach cancer death rates, per 1000, by age group, 1950-99



Source: Original analysis of records obtained from the GROS.

7.2.2. Clinical description

The layers of, and structures surrounding, the stomach are described in box 7.1. Stomach cancer first develops on the mucosa layer of the stomach, from there it spreads more deeply into the stomach. In the next stage of the disease the cancer spreads through the outer layers of the stomach and the stomach wall. From there it can affect the abdominal nodes and spread to distant nodes. In its advanced form the cancer can affect the adjacent organs, and spread across the abdominal cavity to affect other parts of the abdomen (Buckman, 1997). The duration of this process depends on how aggressive the cancer is, but in most cases it can grow unsuspected for a number of years (Suvakovic, 1997).

Box 7.1. Description of the stomach and surrounding structures

Structure	Description
Mucosa	The innermost lining of the stomach, in which stomach acid and digestive juices are made.
Submucosa	The next layer of the stomach.
Serosa	The outermost layer of the stomach.
Lymph nodes	Lymph nodes form part of the body's immune system. They connect the lymph vessels which transport lymphatic fluid (made up of cells which help fight infection and diseases). Lymph nodes are present under the skin in the neck, underarm and groin, as well as inside the chest pelvis and abdomen.
Surrounding structures	These include; the oesophagus, liver, gall bladder, duodenum and pancreas.

7.2.3. Signs and symptoms

The symptoms of stomach cancer are mild or vague in the early stages; symptoms specific to the disease only occur once local disease is advanced (Bowkes & Benjamin, 2001). This can make the disease difficult to diagnose in its early stages. The general symptoms of stomach cancer include weight loss, nausea, pain in the epigastric region and back, difficulty in swallowing and blood in vomit or stool caused by internal bleeding (Bowkes & Benjamin, 2001). Once stomach cancer is suspected, diagnosis can be confirmed by a barium meal x-ray or gastroscopy (this involves the insertion of a fibre-optic instrument into the stomach via the mouth).

7.2.4. Risk factors

The principle cause of stomach cancer is a shortage of hydrochloric acid in the stomach, which can lead to gastric atrophy (inflammation) of the mucosa. Therefore any factor which causes a reduction in acid increases risk of developing stomach cancer. The main risk factors are infection with helicobacter pylori (*H. pylori*) and a diet high in salt preserved, cured or pickled foods and low in fruit and vegetables (Quinn *et al*, 2001). A family history of stomach cancer is also a risk factor for the disease (Bowkes & Benjamin, 2001). There are two main types of stomach cancer, intestinal or expanding, and diffuse or infiltrative. The diffuse type is strongly associated with hereditary risk and has not experienced a decline. This thesis will focus on the intestinal type which has been linked with the decline in mortality in the developed world (Imerie *et al*, 2001).

7.3. Mortality trends

7.3.1. Data reliability

The literature on stomach cancer mortality does not indicate any non-medical reasons why stomach cancer would not have been recorded as a cause of death on a death certificate (for example patient stigma, as in the case of tuberculosis). Instead, the main factor influencing the reliability of stomach cancer death data is the accuracy of the diagnosis. A study from Ireland has compared the accuracy of a diagnosis of stomach cancer in either part 1 or 2 of the death certificate during 1978 (Corridon, 1982). This study compared the diagnosis on the death certificate with diagnosis made from a review of all past medical records or autopsy result, where available. The diagnosis of stomach cancer was found to be correct in 68% of cases. In the majority of cases where the diagnosis was not confirmed an alternative diagnosis was not made, rather there was a lack of evidence to confirm the suspected diagnosis. Cameron and McGoogan's (1981b) study of death certificate reliability in South Lothian between 1975-1977 also considered stomach cancer. This study compared the cause of death entered in the death certificate with that found at autopsy. Stomach cancer was entered on the death certificate in 24 cases, and the diagnosis was confirmed in 17 cases (71%). However, a further 11 cases were only discovered at autopsy. In most instances where stomach cancer was over or under diagnosed the alternative diagnosis was either an incomplete diagnosis of cancer, or cancer of another site.

The death data for stomach cancer would therefore appear to be relatively accurate, although it is possible that the disease may have been under diagnosed. The death rates which have been generated for this thesis closely follow mortality trends which have been discussed in the literature (see Swerdlow *et al*, 1997, Scottish Cancer Intelligence Unit, 2000) indicating that overall the pattern of decline discussed in the following section is probably reliable.

7.3.2. Pattern of decline

Figure 7.1 indicates that stomach cancer mortality was in constant decline during the period 1950-1999. This implies that the mortality decline probably started before 1950. As with tuberculosis, accurately defining the start of the mortality decline was considered important as this allows the isolation of factors which were influencing the disease at that time. Therefore, it was decided that the stomach cancer death data from the pre-1950 period should also be digitised and added to the post-1950 data set. However, when the GROS death data for the pre-1950 period was reviewed it emerged that this was not possible. In versions of the International Classification of Disease in use before 1950 stomach cancer was not classified as a separate cause of death; rather, deaths were included within a more generic gastric category. In order to overcome this problem the literature on stomach cancer was consulted. Charlton and Murphy (1997) place the start of the decline in Britain in the 1930s. A more common way to identify the start of the decline has been to consider the first cohorts to have experienced sustained decline. In England and Wales the cohort born in the 1880s has been identified as the first to see declining death rates (Quinn *et al*, 2001). Evidence for Scotland has placed the start of the decline at a slightly later date, with the Scottish Cancer Intelligence Unit (2000) and McKinney *et al* (1995) dating the start of the decline to cohorts born at the very start of the 20th century.

The decline of stomach cancer mortality would therefore appear to date from the first decades of the 20th century. The remainder of this chapter will consider the factors which have influenced this decline throughout the 20th century. First, the contribution of medicine and standards of living to improving survival amongst those who had already developed the disease will be examined. The influence of medicine and standards of living on disease incidence will then be considered.

7.4. Survival

This section will consider the extent to which an increase in cure rates amongst those who are already ill with stomach cancer contributed to the decline in mortality. Within this context the relative contribution of medicine and standards of living to any improvement will be considered.

7.4.1. The role of medicine (survival)

Unlike tuberculosis, the treatment of stomach cancer in the 20th century was not subject to a defining event such as the discovery of streptomycin. Rather, the principal treatment of stomach cancer, the surgical excision of the growth, was in use throughout the century. Therefore, this study will look at whether improvements in surgical technique, care and diagnosis might have improved survival from stomach cancer. The examination of the medical treatments for stomach cancer will be split into two time periods. The period prior to 1975 will be reviewed first, as the treatment for the disease had remained fundamentally unchanged until this time. The post-1975 period, during which time a number of advances in surgical treatment and diagnosis occurred, will then be considered.

7.4.1.1. Pre-1975 (role of medicine - survival)

The main medical treatment for stomach cancer pre-1975 was the surgical removal of the cancer, using a procedure known as a partial or total gastrectomy. In a partial gastrectomy part of the stomach was removed in the hope of eliminating the cancer. The total gastrectomy, also know as a radical resection, involved the removal of the entire stomach, and possibly some of the abdominal nodes.

Throughout the 20th century the main factor influencing treatment for stomach cancer was the stage at which the disease was diagnosed. This determined the type of treatment which a patient received, if they received any at all. There are four main stages of stomach cancer which depend on the spread of the growth – that is, whether it is still confined to the stomach or has spread to the nodes or distant organs. The princples of 'staging' (defined as the stage to which the cancer has developed) have also remained unchanged; however, they were formalised in 1985 with the International Gastric Cancer Staging System (see box 7.2). The way in which stage was determined (and still is in many cases) was by laparotomy, which requires an incision in the abdomen so that the surgeon can establish how advanced the disease is.

Box 7.2. Unified International Gastric Cancer Staging Classification System

Cancer stage	Description
Stage 0	Tumour is limited to the mucosa, no involvement of lymph nodes.
Stage 1a	Tumour extends into mucosa, no involvement of lymph nodes.
Stage 1b	Tumour extends into submucosa, no involvement of lymph nodes.
Stage 2	Tumour extends into submocosa or penetrates the serosa without invading surrounding structures. Lymph nodes may be involved.
Stage 3a	Tumour extends into submucosa or penetrates the serosa without invading surrounding structures; lymph nodes may be involved. Or tumour extends through the serosa and directly invades surrounding structures, no nodal involvement.
Stage 3b	Tumour penetrates the serosa without invading surrounding structures, with the involvement of distant lymph nodes. Or tumour extends through the serosa and directly invades surrounding structures, with involvement of abdominal lymph nodes.
Stage 4	Tumour extends through the serosa, with involvement of distant lymph nodes. Or any of the above with evidence of metastasis.

Source: Adapted from Kennedy (1987).

When considering the role played by medicine in the decline of stomach cancer, the West Midlands provides some of the best evidence. A series of studies were conducted on the results of treatment in this area between the years 1935 and 1981 (Brookes *et al*, 1965, Allum *et al*, 1989). These studies are widely cited and seen as representative of the situation nationally. There is no reason to believe the evidence from them is not applicable to Scotland.

It has been stated that stage at diagnosis determines treatment. Once stage four has been reached the cancer is regarded as incurable, with most patients pre-1975 receiving no treatment either for palliation or cure if this stage was reached. Although stages 1 – 3 were regarded as potentially curable, success rates still varied considerably. In his review of 25 years of the treatment of stomach cancer, between 1957 and 1981, Allum *et al*(1989) calculated survival at 5 years by stage (seen as an indicator of cure). These are shown in table 7.1.

Table 7.1. Percentage diagnosed by stage, and five year survival by stage, in the West Midlands, 1957-1981

	% diagnosed by stage	Survival at 5 years
Stage 1	<1	72
Stage 2	20	32
Stage 3		10
Stage 4	79	Incurable

Source: Adapted from Allum *et al* (1989).

In the period up to 1975 there were no major improvements in surgery for stomach cancer. However, the survival rates by stage results reveal that if the cancer was caught at stage 1 there was a 72% chance of the patient being cured. This indicates that medicine pre-1975 had the means of curing stomach cancer in many cases if the disease was caught early. This made the staging of the disease critical to survival. Thus, the main contribution which medicine would have made during this time period to the overall decline of stomach cancer would have been through an increase in the proportion of sufferers identified at stage 1 of the disease. Stomach cancer is, however, difficult to diagnose in its early stages due to the relatively benign nature of its symptoms and pre-1975 diagnosis relied upon the identification of these symptoms. By the time patients had developed specific symptoms, the disease was liable to be in an advanced state. Once the disease could be detected by physical diagnosis, that is the examination of the abdomen by hand to locate swelling over the stomach or an enlarged liver (Bowkes & Benjamin, 2001), the disease had already progressed beyond the help of the surgeon. One method of technological diagnosis available pre-1975 was the barium meal x-ray. This was most effective at giving an accurate diagnosis of cancer. However, it relied upon the doctor being initially suspicious of stomach cancer to refer for the procedure.

The difficulty in forming a diagnosis of stomach cancer, and the lack of advance in diagnostic techniques, is reflected in the very small proportion of cases which were diagnosed at stage 1 (see table 7.1). Although cancer diagnosed at stage 1 was believed to have a survival rate of 72%, less than 1% of all cases diagnosed had stage 1 cancer. The fact that the vast majority of cases were diagnosed at a late stage meant that very few sufferers were offered the chance of a curative gastrectomy or radical resection. An indicator of the

effectiveness of early diagnosis is the percentage of all cases of stomach cancer which are eligible for potentially curative surgery. Table 7.2 lists studies of the percentage of sufferers being offered potentially curative surgery, and reveals that through the first three-quarters of the 20th century, only 20% of all cases were offered treatment.

Table 7.2. Percentage of all stomach cancer patients being offered potentially curative surgery

Study	% being offered surgery
Minnesota, 1906-1931 (Balfour, 1937)	19%
West Midlands, 1936-1949 (Brookes *et al*, 1965)	18%
West Midlands, 1957-1981 (Allum *et al*, 1989)	20.8%

Even for the minority of patients who were eligible for potentially curative surgery the result was by no means certain. The operation carried high post-operative mortality rates, and of those who survived the first 30 days only a small number went on to survive for 5 years. Five-year survival for those radically resected varied between 17% in the West Midlands between 1936 and 1949 (Brookes *et al*, 1965) and 23.1% in Oxford between 1938-1949 (Swynnerton & Truelove, 1952). The very poor survival rates for those undergoing surgery reflect the large proportion of cases diagnosed at stage 2 and 3, for whom the chances for cure were low.

The success of medicine in the pre-1975 era in curing stomach cancer was largely dependent on diagnosis. Medicine claimed to have the ability to cure the disease in 72% of cases if they could identify the disease early enough. However, the vast majority of cases had gone beyond any hope of a cure by the time they were identified by the medical profession, and even for the minority, to whom the potential of cure was offered, less than 25% survived to 5 years. Medicine's lack of progress in curing stomach cancer is reflected in table 7.3 which shows the 5-year survival for all cases of stomach cancer over time, and reveals a consistent pattern of over 95% of all those diagnosed with the disease not surviving.

Table 7.3. 5-year survival for all cases in the West Midlands

1935-1950	4.9%
1950-1959	4.9%
1957-1961	3.8%

Source: Allum *et al* (1989).

Thus, between the 1930s and the 1970s the 5-year survival of those with stomach cancer saw very little change, with rates worsening slightly between 1950-1959 and 1957-1961. It should be noted, however, that many doctors at this time were aware of the problem of diagnosis, and called for efforts to be made to improve the identification of cases at stage 1. As early as 1952 (Swynnerton & Truelove, 1952) calls were made for the pro-active discovery of cases by recommending that barium meal x-rays be carried out routinely on all patients over 40 presenting with symptoms of indigestion. This concept of targeted screening will be discussed in greater depth with reference to the post-1975 period.

7.4.1.2. Post-1975 (role of medicine - survival)

The main treatment for stomach cancer remained the surgical removal of the growth. However, the post-1975 period witnessed attempts to advance the effectiveness of this treatment to offer a greater chance of survival. The main influence on surgery post-1975 has been developments in Japan. The Japanese population has, in global terms, a relatively high incidence of the disease. This led to the development of specialist surgical techniques which have since been adopted around the world. One of the main methods adopted in the UK from the late 1970s was the D2 operation. In the earlier time period the operation for stomach cancer involved either the partial or total removal of the stomach (gastrectomy) and in certain cases the removal of the abdominal lymph nodes (lymphadectomy). The D2 operation was more extensive and involved the removal of more tissue with the intention of preventing any recurrence of the disease. In addition to improvements in surgical procedure, advances were also made in anaesthesia and post-operative care, such as intravenous feeding. Attempts have also been made to employ chemotherapy. However, any influence this may have had would only have affected the final years of this study. In addition, the benefit derived from chemotherapy is still uncertain, although one recent trial did show a

survival benefit of one third (36 months versus 27 months) amongst those receiving treatment (Cuschieri, 2002).

Data on the survival of patients receiving potentially curative surgery in the second time period are shown in table 7.4. Findings from the Leeds study indicate a higher survival rate than those of the MRC study. As the results of the MRC study are from a controlled trial, whereas the Leeds data are based on observational data, it is probable that the MRC data are the more reliable, as accurate measurement is a fundamental part of control trials. The results of this trial indicate that by the late 1990s 64% of those patients diagnosed with stage 1 cancer could expect to be cured. This appears to be a significant percentage until it is compared with the stage 1 survival from Allum *et al's* review of surgery in the West Midlands, 1957-81, which reported a success rate at stage 1 of 72% (see table 7.3). It is evident from table 7.4 that stage at diagnosis remained central to an individual's potential for survival post-1975, and that, in spite of advances in surgical techniques and aftercare, the percentage who are cured by stage remained remarkably unchanged over time.

Table 7.4. Survival by stage following potentially curative surgery

	Stage 1	Stage 2	Stage 3
Leeds study, 1985-1989 (Sue-ling *et al*, 1993)	93%	69%	28%
MRC Randomised Surgical Trials, 1986-1992 (Cuschieri *et al*, 1999)	64%	28%	11%

This leads to the consideration of advances in diagnosis: if medicine was not curing a higher proportion by stage, had it managed to identify a greater proportion of early cases to allow them the potential of a cure?

The main development in diagnosis in this time period was endoscopy, which first became available in the diagnosis of stomach cancer in the late 1960s. However, during that time the procedure was only used to confirm an already suspected diagnosis of stomach cancer. Post-1975 the procedure began to be used to identify cases pro-actively. In Japan endoscopy has been used as part of a national screening programme with everyone aged over 40 screened for stomach cancer. This has resulted in a large proportion of early gastric cancers being

identified and has had a corresponding effect upon survival rates (Hallissey *et al*, 1998). However, in Scotland, as in most Western countries, the low overall incidence of stomach cancer means that it does not make financial sense to screen the entire population for early cancer. Instead, attempts have been made to introduce Open Access Gastroscopy (OAG). The aim of OAG is to target those people who are at greatest risk of cancer and then refer them for early investigation to enable early diagnosis and treatment. This involves the direct referral of all patients over 40 who present with signs of dyspepsia for the first time.

Several problems have been identified with the operation of these schemes in the UK. One of the main problems centres upon the lack of symptoms, where, despite the relatively broad criteria for referral to OAG, many GPs do not recognise the early, vague, symptoms in order to refer them on. Another problem arises when GPs have already prescribed powerful anti-ulcer drugs (proton pump inhibitors) prior to sending patients to endoscopy. These drugs may mask the signs of early gastric cancer and lead to misdiagnosis (Griffin & Raimes, 1998).

In spite of problems with OAG, reviews of its effectiveness have produced improved results. A study of OAG in Birmingham from 1984 saw an increase in the diagnosis of early lesions from 1% to 26% of total cases, allowing an increase in potentially curable operations from 20% to 63% (Hallissey *et al*, 1990). In South Tees a comparison was made between stage at diagnosis amongst patients who were referred straight to OAG and those who had been diagnosed through the traditional route of hospital clinic. Just over a fifth (21.1%) of patients diagnosed via OAG had early or stage 1 cancer compared to only 10.6% of those diagnosed by conventional means. It should be noted that despite this improvement, 87% of all those diagnosed in South Tees still had stage 2 or above cancer (Sukakovic, 1987).

It would appear that post-1975 improvements were made in both the medical treatment and diagnosis of stomach cancer. However, these improvements can be challenged when the overall survival rates for those undergoing surgery in the randomised MRC surgical trials are considered. In spite of improvements in diagnosis, the 5-year survival rate was still only 34%, indicating that a large proportion of cases which received treatment had already reached a relatively advanced stage.

The overall effectiveness of medicine in curing those with stomach cancer in Scotland post-1975 can be assessed by reviewing 5-year survival amongst all cases (see table 7.5). In the

period up to 1981, survival resembled that seen in the earlier studies from the West Midlands. After 1981 the percentage surviving eventually doubled for both men and women, which may reflect improvements in detecting early cases of stomach cancer through targeted screening. However, although survival rates have doubled in Scotland the percentage remaining alive after 5 years is still very low, with nearly 90% of men and 86% of women succumbing to the disease. Therefore, although medicine may have seen some advance over the last two decades, it only influenced the prognosis of the disease in a small percentage of cases.

Table 7.5. Stomach cancer survival at five years, all cases

	1976-1980	1981-1985	1986-1990	1991-1995
Male	5.7%	8.2%	9.8%	11.5%
Female	7.8%	9.7%	14.3%	14%

Source: Trends in Cancer Survival in Scotland (2000).

7.4.1.3. Role of medicine in stomach cancer survival – conclusion

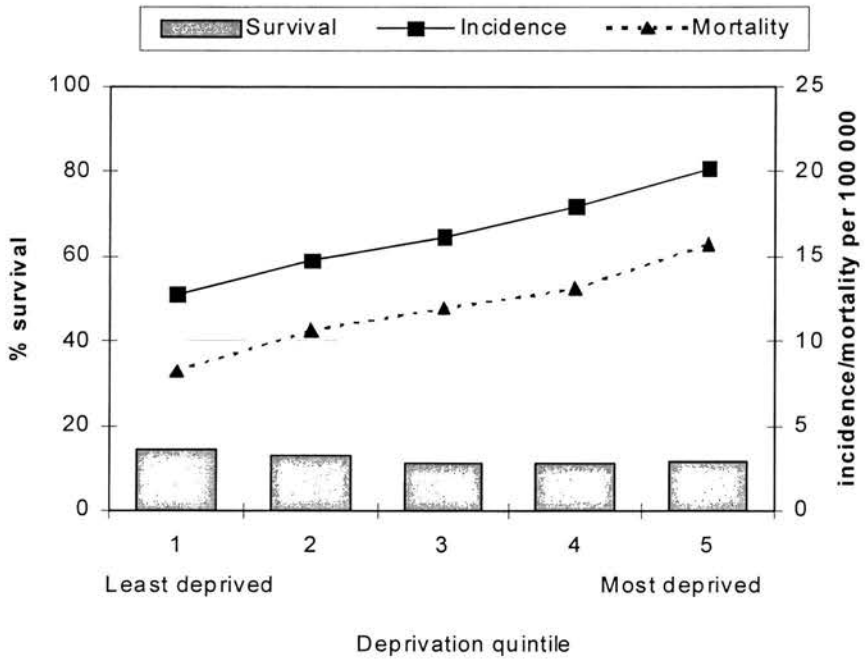
Throughout the 20th century medicine developed the ability to cure the majority of cases of stomach cancer, if they were diagnosed at an early stage. Medicine also developed an effective means of locating these cases, as can be seen from the success of the screening programme in Japan. However, in spite of this, the vast majority of people in Scotland who develop stomach cancer go on to die of the condition very soon after diagnosis, with median survival in 1991–95 being only 6 months (Trends in Cancer Survival in Scotland, 2000).

Why then did Scotland fail to achieve cure rates for stomach cancer at the level seen in Japan? One possibility is that in Japan the disease is relatively common, allowing surgeons to build up an expertise in the operation leading to greater operative success. Although this may be a contributory factor, it cannot explain the overwhelming proportion of deaths. The short time between diagnosis and death in Scotland gives an indication of how advanced many cases must be before they come to the attention of the medical profession, by which time there is little that can be done to prevent death. Therefore, the main problem lies in the ability to diagnose. It takes approximately 5 years for endoscopically detectable cancer to develop into advanced cancer (Tsukuma *et al*, 1983) and in Scotland cases are not being identified in this time.

7.4.2. Role of standards of living in stomach cancer survival

This section will consider the relationship between standards of living and stomach cancer survival. Mortality from stomach cancer is unequally distributed amongst the socio-economic groups, with the poorest suffering the greatest mortality rates. As standards of living are lowest amongst the poorest in society this suggests that mortality may be influenced by standard of living. However, figure 7.5 reveals that there is little difference in 5-year *survival* amongst the social classes, indicating that once an individual has developed stomach cancer a higher standard of living does not influence prognosis. Rather, standards of living are more strongly linked to disease incidence (see figure 7.5) with incidence of the disease increasing with each deprivation quintile. The relationship between standards of living and stomach cancer incidence will be discussed in depth in section 7.5.

Figure 7.5. Incidence, mortality and cause specific survival at 5 years by deprivation quintile. Patients diagnosed in 1991-5



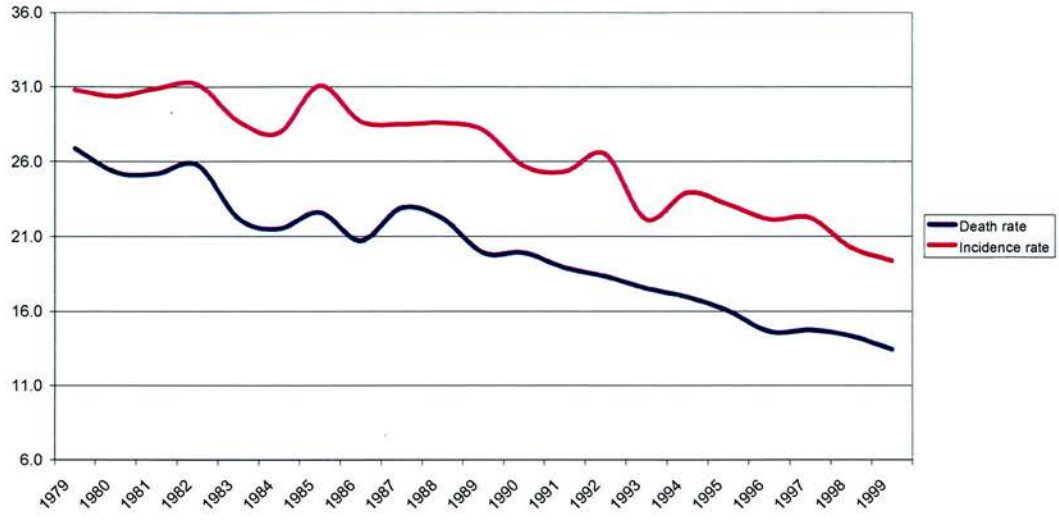
Source: Scottish Cancer Intelligence Unit, 2000.

7.4.3. Improvements in cancer survival – conclusions

In the case of medicine it has been found that, although the disease could be treated by the surgical excision of the growth, this was only possible for those in the early stages of the disease. Throughout the time period covered by this thesis the majority of cases of stomach cancer were only diagnosed once the disease had progressed beyond the stage when surgery could offer the potential for cure. It has also been shown that, although there is a relationship between standards of living and stomach cancer mortality, this did not influence *survival* once the disease was established. The inability of both medical advances and improving standards of living to significantly improve survival is reflected in the 5 year survival data (see table 7.5) which reveal that by the 1990s 88.5% of men, and 86% of women, went on to die from the condition.

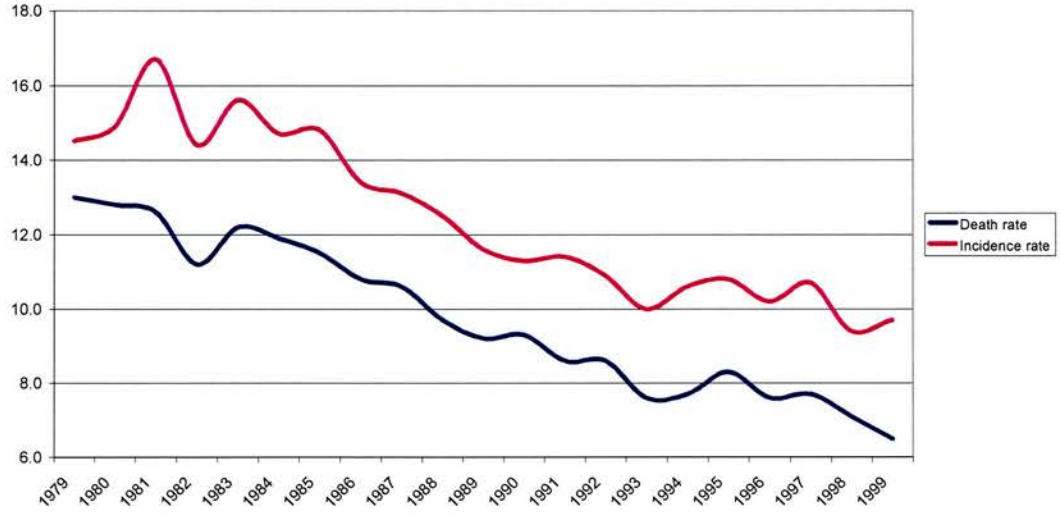
Another way in which the contribution of increasing survival to decreasing mortality can be estimated is to consider the incidence rate for the disease. Figures 7.6 and 7.7 were generated using data acquired from Information Services Division (ISD) of NHS Scotland. These provide the stomach cancer mortality and incidence rates for Scotland between 1979 and 1999. If improving survival was the main influence on the mortality decline it would be anticipated that the death rate would decline at a steeper gradient than the incidence rate. However, these graphs reveal that both the incidence and death rates declined at roughly the same rate. This indicates that the main factor influencing the decline in the mortality rate was the reduction in the number of people who developed the disease. The remainder of this chapter will consider the relative contribution of medicine and standards of living to stomach cancer incidence.

Figure 7.6. Male stomach cancer death and incidence rates per 100000, age standardised to the standard European population



Source: Adapted from data obtained from ISD.

Figure 7.7. Female stomach cancer death and incidence rates per 100000, age standardised to the standard European population



Source: Adapted from data obtained from ISD.

7.5. Decline in the incidence of stomach cancer

In the previous section it was concluded that the main determinant of the decline of stomach cancer post-1950 was not a greater proportion of people recovering from illness, but rather a decrease in the number of people developing the disease in the first place. This section will consider the relative contribution of medicine and improving standards of living to this decline in incidence.

7.5.1. Role of medicine (incidence)

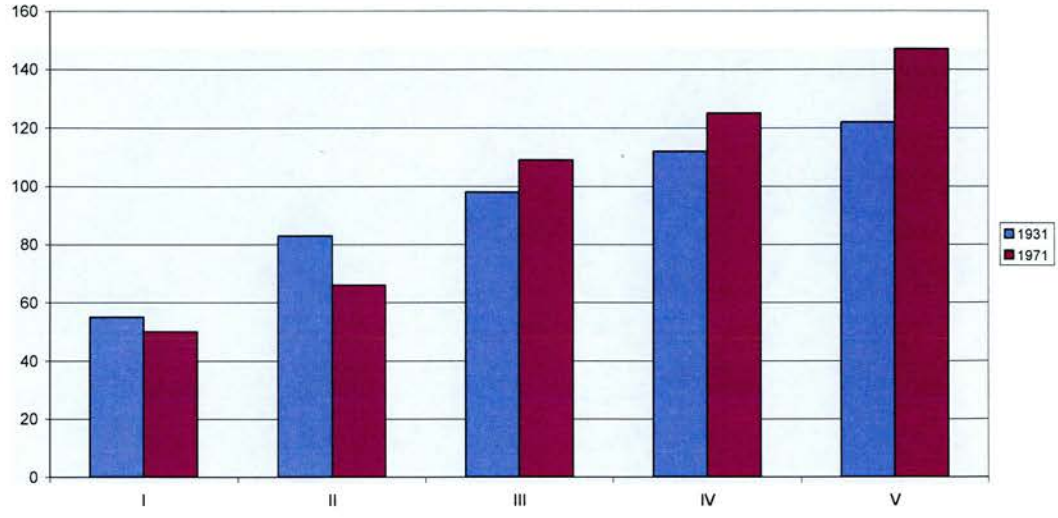
One way in which medicine can directly influence the incidence of stomach cancer is the screening for, and treatment of, helicobacter pylori (Briggs *et al*, 1996). The bacterium was first identified in 1984 and is a risk factor for stomach cancer. Since its discovery questions have been raised regarding both the viability and desirability of screening and treating the infection. Screening has been attempted in countries with a high incidence of stomach cancer, such as Japan. However, no programme is yet in existence in the UK. There is also concern about the implementation of such a scheme, as infection with *H. pylori* has been associated with protection against cancer of the oesophagus (Blasser, 1998).

Thus, although medicine may play a role in the future control of *H. pylori* as a risk factor for stomach cancer, it did not have any influence during the time period covered by this study. Rather, the main influence on *H. pylori* prevalence during the 20th century in Scotland were social conditions. The role of public health medicine in improving social conditions will be discussed jointly with that of improving standards of living in the following section.

7.5.2. Standards of living (incidence)

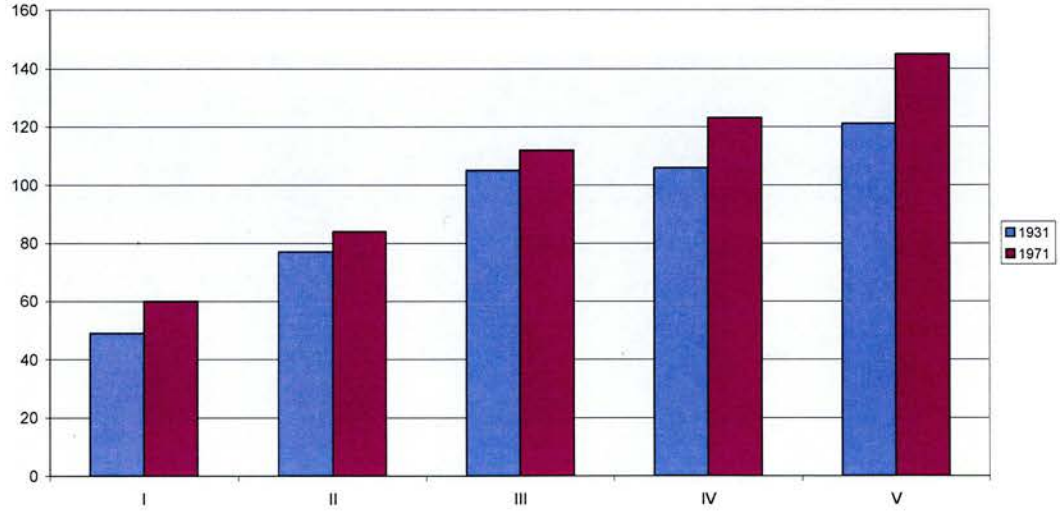
It was argued in section 7.4.2 that stomach cancer death rates are related to social class and that this is due to a higher incidence of the disease amongst the most deprived groups, rather than a differential survival rate. The link to social class can be traced through the 20th century. Figures 7.8 and 7.9 display the mortality ratio of stomach cancer amongst social classes I (high) - V (low) for the years 1931 and 1971. These charts reveal that a class gradient persisted in both years but also that the gap between the classes has widened. The mortality ratio between classes I and V is relatively higher in 1971 than 1931. This disparity amongst the social classes is also reflected in the Scottish incidence rate in 1991 – 95, when the rate was 20 per 100000 in social group 5 and 13 in group 1 (see figure 7.5).

Figure 7.8. Male standard mortality ratio for stomach cancer by social class (I = high, V = low)



Source: Adapted from Logan (1982).

Figure 7.9. Married female standard mortality ratio for stomach cancer by social class (I = high, V = low)

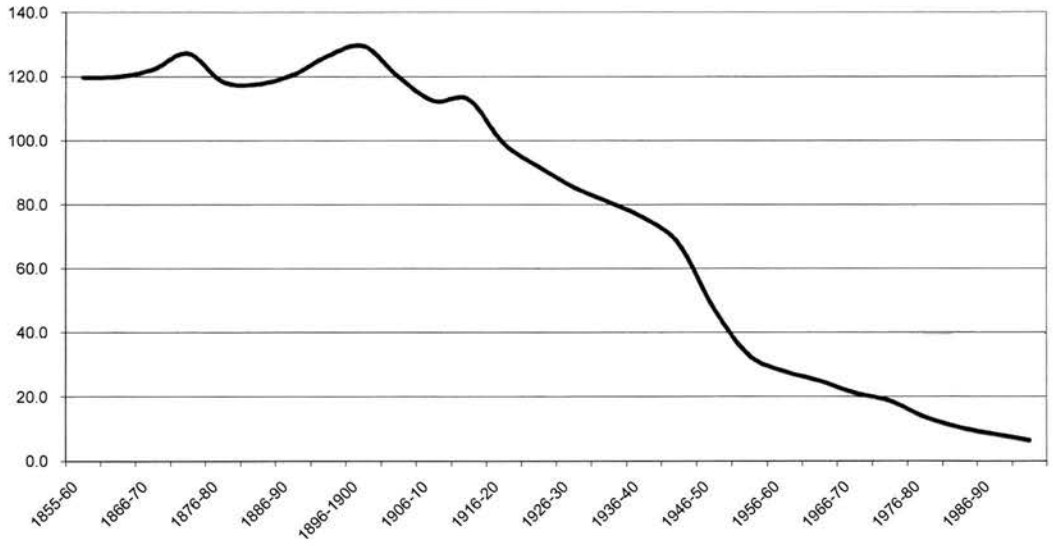


Source: Adapted from Logan (1982).

The fact that stomach cancer affects the social classes unequally, with the poorest at greatest risk, indicates that the incidence of the disease is likely to be influenced by social conditions.

Therefore any improvement in standards of living should influence the incidence of the disease. Leon and Davey Smith (2000) have investigated the link between deprivation and stomach cancer and have shown that the strongest link with deprivation occurs during infancy: poor socio-economic conditions in the first years of life increase an individual's risk of stomach cancer in later life. They established a link between cohorts who first experienced a decline in infant mortality and who later experienced a reduced risk of stomach cancer. These findings correlate with trends in the Scottish infant mortality rate (see figure 7.10). The decline in infant mortality occurred relatively late in Scotland (at the start of the 20th century) and it is this cohort who first experienced a decline in stomach cancer. Meanwhile in England and Wales infant mortality began to decline at a slightly earlier date, whilst stomach cancer mortality first began to decline there in cohorts born in the late 1880s.

Figure 7.10. Infant mortality rate, per 1000 live births, 1855-1995



Source: Adapted from data obtained from the GROS.

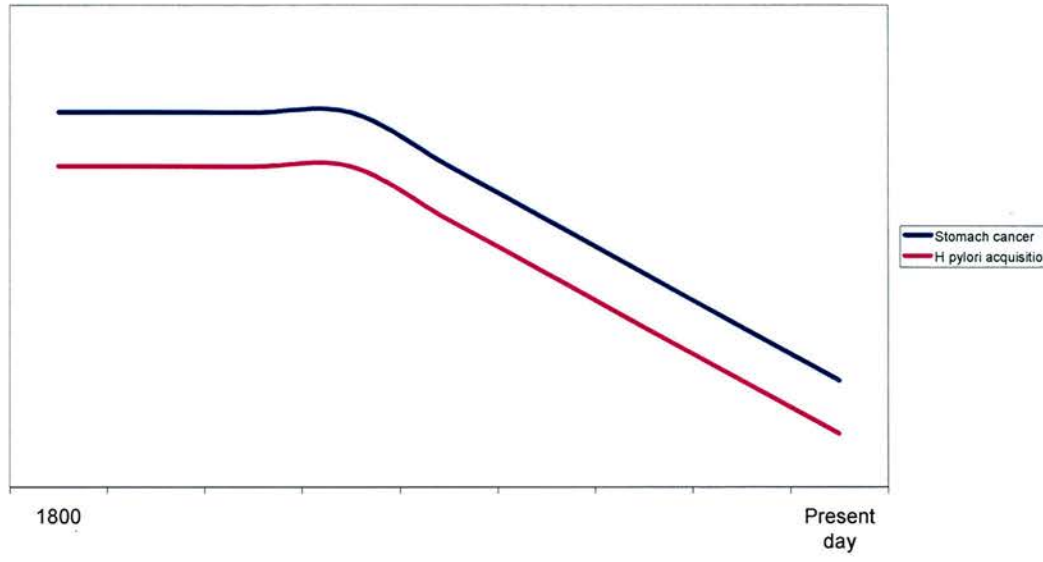
If it is accepted that improved conditions in infancy have a direct effect on the later development of stomach cancer, we must then consider what factors in infancy increase the risk of stomach cancer and whether they experienced any change in Scotland at the start of the 20th century.

7.5.2.1. Helicobacter pylori

Helicobacter pylori (*H. pylori*) is a bacillus which colonises the mucus layer of the stomach, and can lead to a decline in acid production and gastric atrophy. As the bacterium was only identified in the 1980s much still remains to be discovered about it. It has been recognised as a major risk factor in stomach cancer, with studies attributing between 55% and 80% of cases to the infection (Parsonnet, 1991, Helicobacter Cancer Collaboration Group, 2001, Moayyedi, 2002, WHOb, 2003). Nevertheless, the majority of people who carry the infection suffer no adverse effects.

It is believed that, in the developed world infection with *H. pylori* was virtually universal up until the end of the 19th century, but that prevalence has been declining since. As research into *H. pylori* is still in its early stages it is difficult to locate accurate information on the timing of the decline of the infection. Blasser (1998) has placed the start of the decline in *H. pylori* incidence in the 1800s (see figure 7.11). However, testing of cohorts born throughout the 20th century supports the theory that infection began to decline at the start of the century. This was the case in a study of infection prevalence amongst Welsh men which found rates of 75% amongst those born between 1905-10, 66.7% for those born 1925-30, declining to 29.3% amongst those born 1945-50 (Sitas *et al*, 1991). *H. pylori* infection rates are also strongly related to social class, with the most deprived experiencing much higher rates (see table 7.6).

Figure 7.11. H pylori acquisition and stomach cancer in Western countires



Source: Adapted from Blasser (1998).

Table 7.6. Prevalence of infection with *H. pylori* amongst Welsh men aged 30-75, by social class

Social class	Seroprevalance (%)
I & II	49.2
IIIN & IIIM	57.5
IV & V	62.2

Source: Sitas *et al* (1991).

Evidence that infection rates declined throughout the 20th century and their link to social class suggests strongly that infection with *H. pylori* is influenced by social conditions. This fits with the theory of how the disease is transmitted. The mode of transmission has not been confirmed but it is believed to be person to person, via the gastro-oral route (Imerie *et al*, 2001). It has also been shown that the risk of developing stomach cancer after infection with *H. pylori* is greatest if it is acquired in infancy, rather than in later life. Therefore, factors which may have influenced the decline of infection amongst infants born in Scotland at the start of the 20th century will be considered. Given the link between infection and social class, and the belief that the infection is passed by person to person transmission, overcrowding and general sanitation are most likely to be risk factors for *H. pylori.*

Attempts have already been made to establish the link between poor housing conditions in early life and later risk of stomach cancer. In England and Wales Barker *et al* (1999) examined stomach cancer mortality in local authority areas and were able, through examination of the 1931 census and 1936 National Survey of Overcrowding, to show that overcrowding in childhood was a, "*major determinant of stomach cancer, and might act by promoting the transmission of causative organisms*" (p.575). Therefore, if overcrowding is a risk factor for *H. pylori* infection, and the later development of stomach cancer, the gradual improvement in overcrowding in Scotland from the start of the 20th century (discussed in chapter six (see figure 6.9)) may have helped to reduce infection rates.

As infection in infancy carries the greatest risk of future stomach cancer, any factors which may have influenced the improvement in overcrowding of this age group must be considered. One of the main determinants of crowding in infancy is family size, with a large number of siblings increasing the likelihood of infection. The birth rate in Scotland had, by 1900, been in decline for nearly 20 years (see figure 7.12). It can be argued that this may have helped influence overcrowding amongst the very youngest in society, reducing the opportunity for the transmission of *H. pylori* amongst siblings in infancy.

Figure 7.12. Crude birth rate per 1000 living, 1855-1995

Source: Adapted from data obtained from GROS.

If the transmission of *H. pylori* is via the gastro–oral route, hygienic conditions in infancy should also be considered, as poor sanitary conditions would have been conducive to the spread of the infection. McKeown acknowledged that the late 19th century was a time when the public health services were successful in improving urban sanitary conditions. These works included the provision of refuse collection and especially the provision of pure water as well as advances in drainage and sewage. McKeown attributed the decline of the major water-borne infections to these actions. The date of the introduction of most of these initiatives, and the decline of diseases such as cholera and typhoid, which are linked to poor sanitation, does, however, pre-date the decline of *H. pylori* amongst infants by roughly 30 years. One explanation for this is that many of the public sanitary works influenced the world outside the home, rather than the domestic environment. The infection rates of infants would perhaps have been influenced most by changes in domestic hygiene. The infant mortality rate is acknowledged to be a sensitive indicator of conditions of domestic hygiene and if figure 7.10 is consulted this shows that in Scotland infant mortality did not begin its decline until 1900, suggesting that domestic hygiene in Scotland did not see significant improvements until this time. In addition, education by health visitors and the sanitary authorities of the public on the importance of infant hygiene and feeding practices, informed by the findings of Pasteur on germ theory, was introduced from the start of the 20th century (Morrel, 1991). If a link can be established between poor sanitation and *H. pylori* infection

in infancy, a case can be made for improvements, linked to public health medicine, dating from 1900 which may have helped reduce transmission.

It has been argued that *H. pylori* is a significant risk factor for stomach cancer. However, as it was only identified relatively recently much research remains to be done. Therefore, the conclusions reached in this thesis are tentative. If current research findings in this area are correct, a case can be made that the decline in *H. pylori* in Scotland did influence the decline of stomach cancer. It is probable that improvements in overcrowding, a decline in family size and improvements in domestic hygiene at the end of the 19th century and the start of the 20th century influenced the decline in *H. pylori* rates amongst cohorts born at this time. The connection between *H. pylori* infection prevalence and social conditions may also explain why infection rates and stomach cancer incidence rates have remained higher amongst the most deprived.

Although *H. pylori* is now acknowledged to be the main risk factor for stomach cancer, it is not the only one. A recent review of the English language literature (Imerie *et al*, 2001) estimated that less than 1% of people infected go on to develop stomach cancer. This suggests that additional factors are required for an individual to become at increased risk. This has been accepted by a joint WHO/FAO expert consultation, which found that "*infection with the bacterium is an established risk factor, but not sufficient cause, for the development of stomach cancer*" (2003:95). Therefore other factors influencing the incidence of the disease must also be considered.

7.5.2.2. Diet

Prior to the discovery of *H. pylori*, diet was thought to be the main risk factor for stomach cancer. Since the 1960s links had been made between high salt diets and the incidence of stomach cancer. Investigations of the relationship between diet and stomach cancer have continued following the discovery of *H. pylori*. A number of case-control studies have been undertaken to identify which food groups either increase or decrease the risk of developing stomach cancer. The main findings of these are displayed in box 7.3.

Box 7.3. Food groups found to influence risk of developing stomach cancer

Increased risk	Reduced risk
Preserved meats Salty foods Possibly starchy foods (See La Vecchia *et al*, 1987, Hu *et al*, 1988, Buitti *et al*, 1992). The common link between these foods is the presence of high concentrations of salt, which has been linked to gastric irritation leading to gastric atrophy which may generate lesions which can eventually progress to cancer (Correa, 1992).	Fresh fruit Fresh vegetables (See La Vecchia *et al*, 1987, Hu *et al*, 1988, Buitti *et al*, 1992, McCullough *et al*, 2000). The protective agents in these foods is vitamin C, or ascorbic acid, which creates an anti–oxidant affect which inhibits the development of gastric atrophy and pre-cancerous lesions (Correa, 1992).

Diet can therefore be seen to have a dual effect on the incidence of stomach cancer: consumption of certain foods increases risk, whilst others offer protection. Any decrease in the intake of one or increase in the other should influence the number of cases being seen. This study will therefore consider any changes in the intake of these food groups amongst cohorts born after 1900 in Scotland.

Before considering post-1900 improvements it is useful first to consider the diet of the late 19th century to assess the extent to which this may have contributed to previously high levels of stomach cancer. As we have seen, Scotland experienced a period of rapid urbanisation during the industrial revolution, which had an adverse effect on food supplies, as well as housing. The massing of much of the population away from sources of fresh foods and the lack of an infrastructure to transport such foods, together with the poverty experienced by many, led to reliance upon poor quality preserved foods. For the working classes salted bacon was the main source of meat, and potatoes were the main vegetable. Fresh fruit and vegetables, if they were consumed at all, made up the most expensive part of the diet (Kitchin & Passmore, 1949). According to the risk factors outlined in box 7.3 this was a diet which placed much of the Scottish population, and especially the working classes, at higher risk of stomach cancer. The 20th century witnessed changes in this diet brought about by developments such as refrigeration and improved transportation, as well increased purchasing power. Influences on the consumption of high and low risk food groups are discussed below.

Changes in the consumption of high risk foods

The most important change in this category was the shift from consumption of salted or smoked meat to fresh meat. The main influence on this was the development of refrigeration. The process of making ice in the laboratory was first discovered by Sir John Leslie in Edinburgh in 1810. As the 19th century progressed this technology was employed in the preservation of meat, especially in America. It was from here that Britain first began to receive its new source of fresh meat, with the first consignment of frozen mutton arriving in 1880. From 1900 America was joined by Argentina in the exporting of meat to Britain (Kitchin & Passmore, 1949). This supply of fresh frozen meat was to change the diet of all levels of society. Even the working classes were able to acquire it as from the 1890s it had become widely available at a price similar to bacon (Nelson, 1993). The increase in total meat consumption has also been discussed in chapter six and data on consumption levels from 1880 were provided in table 6.2.

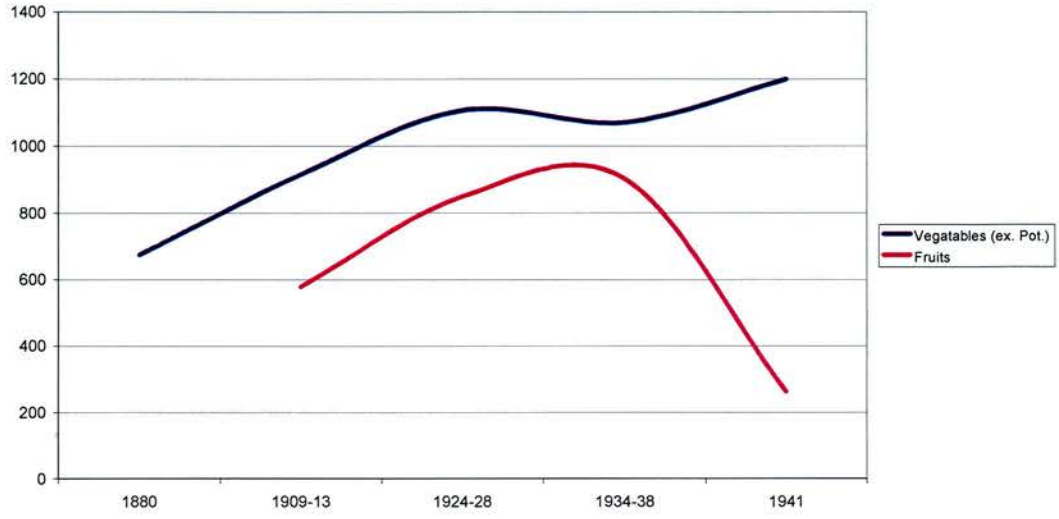
It can be argued therefore, that from the last decade of the 19th century, the diet of the majority underwent a significant change in Scotland. The availability of cheap non-salt preserved meat changed the focus of the nation's diet from preserved to fresh meat. Although the consumption of preserved meats still continues, it now forms a much smaller part of the diet rather than the central source of meat as it had been in the19th century.

Changes in the consumption of foods which reduce risk

At the end of the 19th century the consumption of fruit and vegetables was very low, especially amongst the working classes. However, despite the fact that Scotland is still a nation whose consumption of these foods is a matter of concern, intake increased throughout the 20th century. In common with the availability of fresh meat, refrigeration played a role in keeping produce fresh for longer, whilst improvements in transport links facilitated its distribution to urban centres. This improvement in the average consumption of fruit and vegetables is evident in figures 7.13 and 7.14. The data are from two sources and so not directly comparable (they also refer to consumption in the UK rather than Scotland alone). However, they give an indication of trends in overall consumption. Figure 7.13 shows an increase in the consumption of vegetables from the 1890s and of fresh fruit since the 1900s. The intervention of World War Two led to a decline in the consumption of fruit as foreign supply routes were cut. This recovered in the post war period and continues to increase to the present day (figure 7.14). The war did not affect the consumption of vegetables,

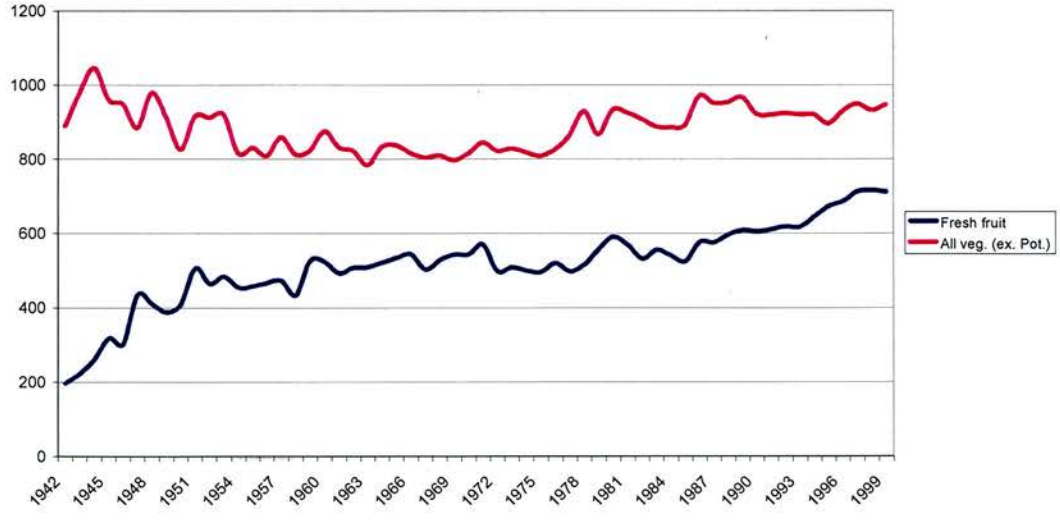
however their consumption did see a slight decline in the post-war period, before remaining consistently high.

Figure 7.13. Fruit and all vegatable (ex. potatoes) consumption, grammes per person, per week, 1880-1941 (UK)



Source: Adapted from Greaves & Hollingsworth, 1966.

Figure 7.14. Fruit and vegatable (ex. potatoes), grammes per person, per week, 1941 - 99 (UK)



Source: Adapted from the National Food Survey (2001).

The result of this increasing consumption of fruit and vegetables was an increase in vitamin C intake by all sectors in society (see table 7.7). Studies have shown that vitamin C provides anti–oxidant protection against cancer. By ensuring an adequate supply of vitamin C, the war was to provide an equalising role, as rationing was developed to ensure that the entire population was receiving their recommended daily intake.

Table 7.7. Average daily intake (mg) of vitamin C by social class

	Poor	Working class	Middle class
1900	13	25	50
1944	62	67	67
1980	51	54	71

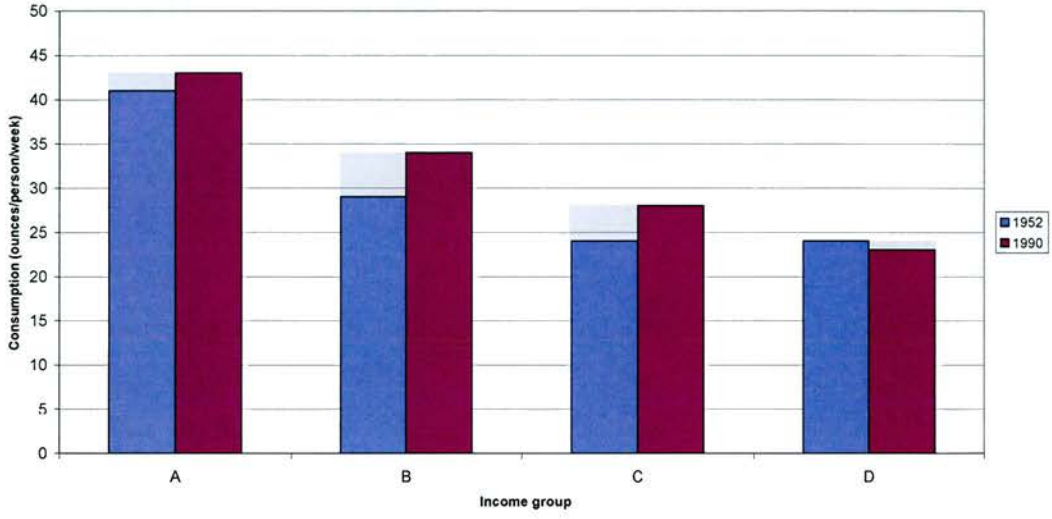
Source: Nelson, 1993.

7.5.3. Decline in the incidence of stomach cancer - conclusions

The main influences on the decline in the incidence of stomach cancer in Scotland were the decline in the infection rate from *H. pylori*, aided by a movement in diet away from foods that increase risk towards those which reduce it. It should be noted, however, that the incidence of stomach cancer in Scotland is still strongly influenced by social class. Possible reasons for this can be found in the main risk factors. *H. pylori* infection is significantly higher amongst the most deprived groups, especially amongst children, reflecting poorer living conditions, which may contribute to the continued transmission of the bacterium.

Class differentials in diet may also explain the excess incidence of stomach cancer amongst the lowest socio-economic classes. It has been argued here that consumption of fruit and vegetables increased overall throughout the time period covered by this study, thereby increasing natural resistance to the development of stomach cancer lesions. Nevertheless, there is an inequality in consumption amongst different social classes. The most affluent members of society have always enjoyed a higher consumption of these foods which has continued to increase. Meanwhile (see figure 7.15), those in the poorest class may actually have seen a deterioration in their consumption of fruit between 1952 – 90.

Figure 7.15. Consumption of fruit in the UK, 1952 and 1990, by income group (A = high income D = low income)



Source: Adapted from Austroker (1994).

7.6. Conclusions

This thesis has defined three groups of influence on mortality (see box 3.2). Group 1 has been defined as medical influences involving the interaction of a medical professional with an individual. In the case of stomach cancer it has been argued that the contribution of this group of medical influence on mortality decline was minimal. Between the 1930s and the late 1970s, 5-year survival from the disease remained virtually unchanged (4.9% in 1935-50 and 5.2% in 1977– 81) (Allum, 1989). The reason was not that medicine lacked the ability to cure (at least some cases) through surgical intervention, but that it lacked the means of diagnosing cases before the disease had advanced to a stage where surgery was futile. There was some improvement in medicine's ability to identify cases at an early stage since the late 1970s, with the introduction of OAG to target those at greatest risk for screening. In spite of these advances, survival in Scotland only improved from 5.7% for men and 6.6% for women since the late 1970s, to 11.5% for men and 14% for women by the mid 1990s (Scottish Cancer Intelligence Unit, 2000). This leaves almost 85-90% of people suffering from the disease with no hope of a cure. It is possible that continued advances in early diagnosis, through improvements in targeted screening, might lead to an improvement in the cure rate in the future.

The decline of stomach cancer mortality was attributable mainly to the decline in the incidence of the disease. The origins of this decline lie in the reduction in the main disease risk factors dating from the early 20th century. This meant that cohorts born from that time onwards had a lower risk of developing stomach cancer in later life.

In the case of *H. pylori* infection levels the conclusions reached in this thesis must be treated with a degree of caution, as research is still being conducted on the bacterium. However, it is probable that both improving standards of living (group 3) and public health (group 2) contributed to the decline in the prevalence of the infection. Improving standards of living were associated with a reduction in overcrowding levels, and also a reduction in family size, which may have inhibited the spread of infection. Public health helped to improve sanitary conditions, and was responsible for educating the public on the care of infants. The other stomach cancer risk factor is diet, and changes in diet relevant to stomach cancer have not been associated with medicine in this thesis. Rather, changes in diet have been linked to technological advances in food preservation, and the increasing affordability of both fresh meat, fruit, and vegetables.

Therefore, although clinical medicine may have increased success in the fight against stomach cancer in the future, by offering an increased chance of cure via surgery and disease prevention via *H. pylori* screening, in the time period covered by this thesis it contributed little to the overall decline in mortality. Declining disease incidence, as a consequence of rising standards of living and public health, was the central force driving the mortality decline.

8. Cardiovascular disease

8.1. Introduction

When the case study diseases were selected for study it was decided that, in order to make the task of analysis practical, only three diseases would be considered: tuberculosis, stomach cancer and IHD. This decision was later reviewed and the possible influences on the decline of stroke have also been considered. This chapter will consider the influences on the mortality decline from both IHD and stroke.

The relative contribution of medicine and standards of living to IHD mortality will be reviewed in the following section. The decline of stroke will be considered within the context of the findings for IHD. By identifying the risk factors and treatments for stroke which overlap with IHD, an indication of main determinants on the decline of stroke will been reached. In addition, possible additional influences on the decline of stroke will be considered. These are discussed in section 8.3. Including two diseases in the same chapter makes it a very long one. It is hoped that the reader will understand the benefits of concatenating the two diseases in the same chapter.

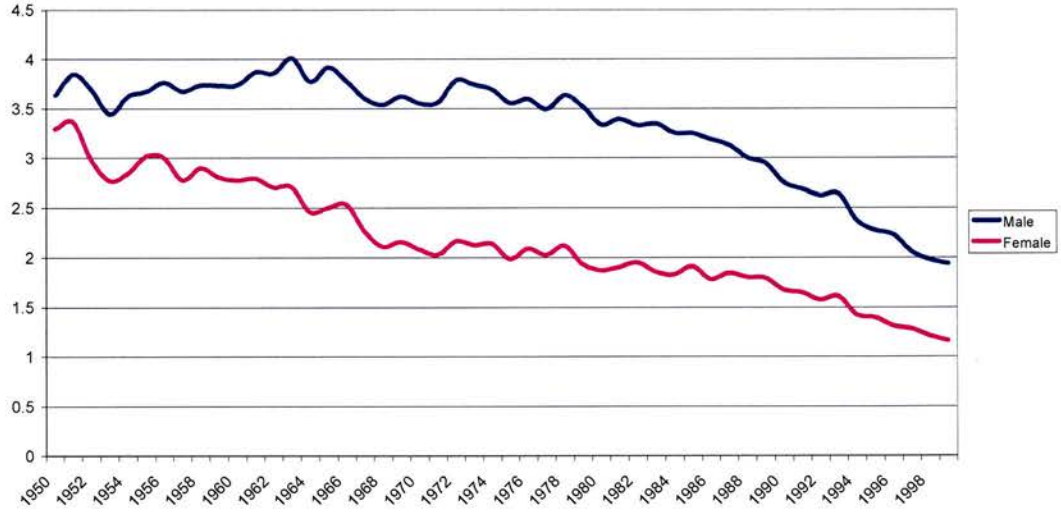
8.2. Ischemic heart disease (IHD)

8.2.1. Introduction

Ischemic heart disease (IHD) is the leading global cause of death, accounting for 23% of all deaths in the developed world (WHO, 2003). Scotland has one of the highest death rates for the disease and by 1989 had the highest IHD event rate globally (The Scottish Diet, 1993).

Although Scotland has a relatively poor international position in respect of IHD, mortality from the disease has declined, and was the single largest contributor to potential lives saved in 1999, when compared to the 1950 death rates. The all age standardised death rate for IHD is shown in figure 8.1. Male death rates declined from 3.6 per 1000 in men in 1950 to 1.9 in 1999, whilst female rates declined from 3.3 to 1.1.

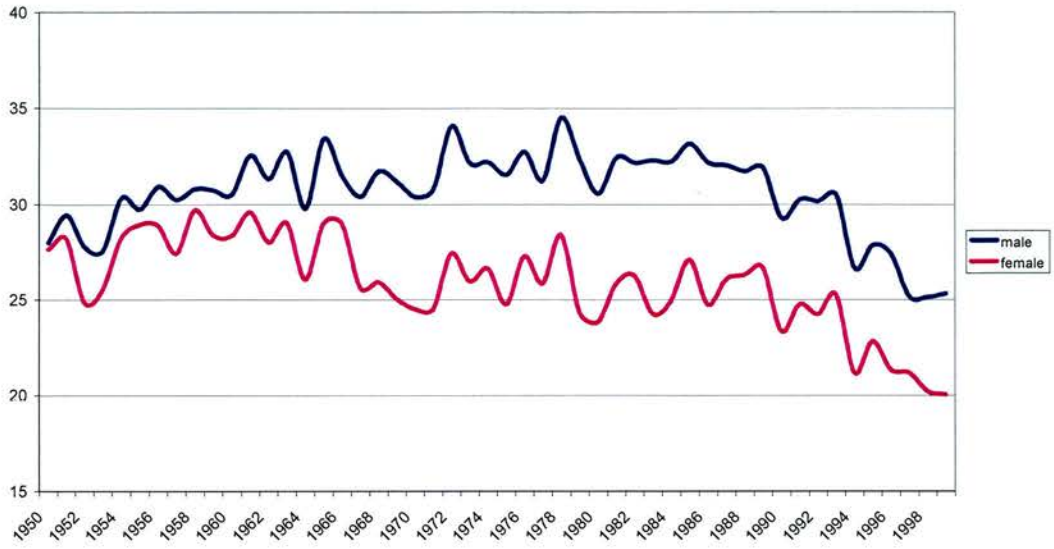
Figure 8.1. IHD death rates, per 1000, standardised to the 1950 population, 1950-99



Source: Original analysis of records obtained from the GROS.

Throughout the time period covered by this study, IHD was the most significant cause of death in Scotland. In 1950 it accounted for 28% of all male deaths and 27.6% of all female deaths. This percentage increased to 34.5% of male deaths in 1978 and 29.6% of female deaths in 1962, after which the percentage of total deaths attributable to IHD declined so that by 1999 it accounted for 25.3% of male deaths, and 20% of female deaths (see figure 8.2).

Figure 8.2. Percentage of all deaths attributable to IHD, 1950 - 99



Source: Original analysis of records obtained from the GROS.

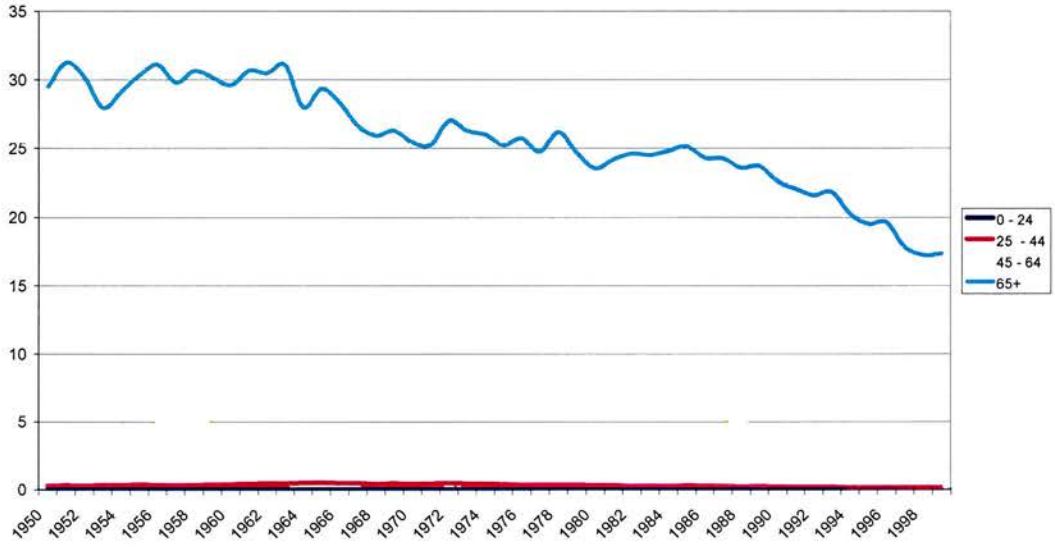
IHD was therefore, paradoxically, not only the largest contributor to the decline in mortality in Scotland since 1950, but was also the single largest cause of death since that time. In this chapter the factors which influenced the decline in IHD mortality will be considered. However, given Scotland's poor international position regarding IHD mortality, consideration will also be given to why death rates were originally so high. IHD offers a more complex case study than tuberculosis or stomach cancer, as the disease is linked to a wider range of risk factors (which will be discussed in the following section). The pattern of IHD mortality throughout the 20th century will also be considered. For much of the century death rates were increasing, rather than decreasing. The factors influencing the increase in IHD will be discussed in a section dealing with the pre-1975 time period, and the findings from this will then inform the discussion of factors influencing the decline of the disease after 1975.

8.2.2. Background

8.2.2.1. Age groups affected

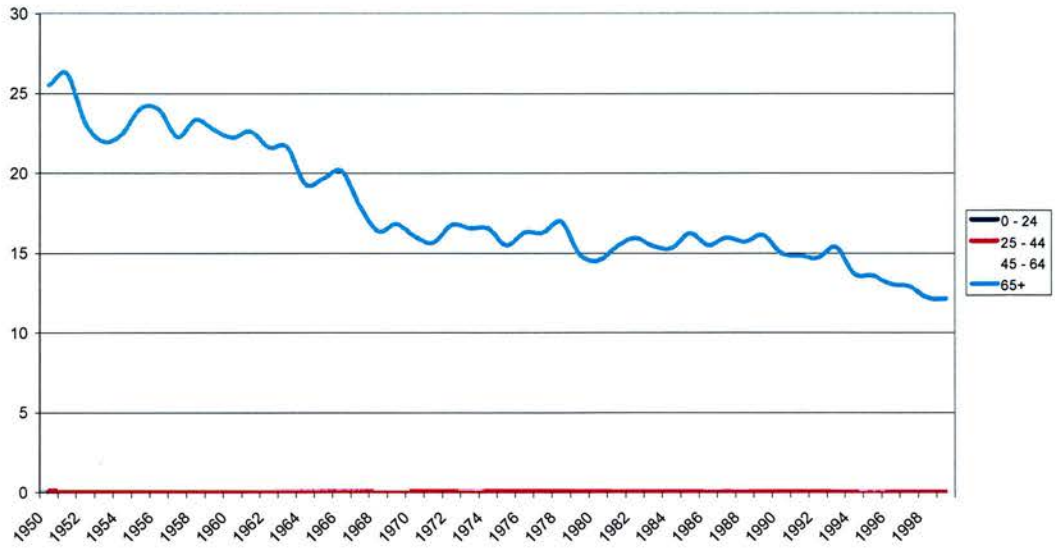
IHD is predominately a disease of later life, with the death rate highest amongst the over 65 age group (see figures 8.3 and 8.4). The high death rates experienced by the over 65s disguise the pattern of mortality amongst the younger age groups, and in particular amongst the 45-64 year olds. Deaths amongst this age group are of particular interest as they are now regarded as largely preventable (Andolo, 2004). Male and female death rates are displayed separately in figure 8.5. The IHD death rate amongst 45-64 year olds alone was higher than the all age death rates from tuberculosis (see figure 6.1) or stomach cancer (see figure 7.1). Although both male and female IHD death rates are high, the rate was particularly high for men, among whom the death rate remained above 6 per 1000 until the 1980s.

Figure 8.3. Male IHD death rates, per 1000, by age group, 1950-99



Source: Original analysis of records obtained from the GROS.

Figure 8.4. Female IHD death rates, per 1000, by age group, 1950-99



Source: Original analysis of records obtained from the GROS.

Figure 8.5. IHD death rate per 1000, aged 45-64, standardised to the 1950 population, 1931-99

Source: Original analysis of records obtained from the GROS.

8.2.2.2. Clinical description

Ischemic heart disease is also known as coronary heart disease (CHD): although both names are used in the literature, they refer to the same condition. Death from IHD normally occurs in the form of a myocardial infarction (MI) or heart attack. This occurs when one of the heart's coronary arteries becomes blocked, which stops the blood supply to the heart and causes part of the heart muscle to die. The longer the blood supply is curtailed, the more likely death is. In non-fatal cases the longer the delay in removing the blockage from the artery the more severe and longer lasting is the damage to the heart. Two factors can lead to an artery becoming blocked. The first is narrowing of the coronary arteries, which occurs when they become blocked with atheroma (fatty deposits) which accumulates on the sides of the artery walls, restricting the passage of the blood. The other factor is the condition of the blood. Blood which has an excess of fibrinogen, an agent of coagulation, can become sluggish and more likely to form clots. The combination of these factors, narrowed arteries and blood susceptible to clotting, increases an individual's likelihood of suffering a heart attack.

8.2.2.3. Signs and symptoms

For the majority, the first sign that they are suffering from IHD is when they experience a MI. This is an acute attack whose main symptom is pain accompanied by a feeling of sickness. Angina is also a symptom of IHD and can occur without the individual having first suffered a MI. It manifests itself as recurrent chest pain, which can be induced when extra strain is put on the heart, for example during exercise. In angina the artery is not totally blocked as in a MI; rather, the narrowing of the arteries leads to a reduction in blood flow.

8.2.2.4. Risk factors

IHD does not have one specific cause: a number of different factors can increase an individual's risk. These comprise anything which can cause the narrowing of the arteries or increase the risk of blood clots. The main risk factors are shown in box 8.1. Many of these risk factors are linked to specific behaviours, such as consumption of a high fat diet or smoking. The level of risk may be determined by a number of other factors, for example ignorance of the risk or poverty. When considering factors influencing IHD mortality and incidence these risk factors, and the underlying causes which influence their prevalence, will be reviewed. The list in box 8.1, while not exhaustive, includes the main risk factors which are discussed in the literature.

Box 8.1. IHD risk factors

High cholesterol	There are two different types of cholesterol: low-density lipoprotein (LDL) and high-density lipoprotein (HDL). An excess of LDL leads to the build up of atheroma on the walls of the arteries. Some cholesterol is produced naturally in the liver, and about 1 in 500 people in Scotland suffer from the genetic condition familial hypercholesteroloemia, which can lead to the excessive production of cholesterol (Report of a Working Party to the Chief Medical Officer for Scotland, 1993). However, for the majority of people the excess of LDL is caused by diet, in particular a diet which is high in saturated fats, such as those found in red meats and dairy products. Therefore, a diet which is high in these food groups increases the risk of high cholesterol which can lead to narrowed arteries.
Smoking	Smoking causes an excess of fibrinogen in the blood. Fibrinogen is a natural protein in the blood which is the agent of coagulation, an excess of which can lead to an increased risk of blood clots.
High blood pressure	Over time high blood pressure can lead to damage of the blood vessels by making them less flexible. This can encourage the build up of atheroma and the narrowing of the arteries. The presence of high blood pressure is directly linked to the level of salt in diet (WHO Technical Report Series, 2003).
Lack of physical activity	Physical activity improves the hearts functional capacity and discourages the build up of atheroma and the formation of blood clots. Correspondingly, a lack of regular physical activity encourages these conditions.
Poverty	Since the 1980s poverty has been recognised as a risk factor for IHD. This is in part due to the greater prevalence of the risk factors outlined above amongst the most deprived. A number of explanations have been advanced to explain why the most deprived have higher IHD death rates (Lynch *et al* , 2000), (see section 8.7.6.2).

8.2.3. Mortality trends

8.2.3.1. Data reliability

Several studies have considered the reliability of death certification for IHD. One of the longest running studies forms part of the Framingham Heart Health Study in the USA, which began in 1948. All deaths amongst the study population were reviewed by a panel of specialist doctors to assess the accuracy of the diagnosis on the death certificate. This study confirmed the diagnosis of CHD in 67.4% of cases. In most instances where a diagnosis of CHD was not proven, the diagnosis was changed to 'unknown', as there was insufficient evidence to confirm a diagnosis of CHD (Lloyd-Jones *et al*, 1998). In the UK a similar result was found in a 1971 study which asked doctors to self-assess their confidence in a diagnosis of coronary thrombosis (MI) on death certificates. In 70% of certificates doctors were either very or fairly sure of their diagnosis (Walford, 1971). Cameron and McGoogan's (1981a) review of death certificate accuracy in South Lothian during the mid 1970s also considered the reliability of certificates for acute MI. This study confirmed the diagnosis in 198 cases. However, 51 cases were only identified at autopsy and 58 cases were disproved at autopsy.

Two principle reasons for the inaccurate certification of IHD have been identified. One explanation is that IHD is a convenient diagnosis for a sudden death in cases in which the practitioner is sure that the death is natural (Walford, 1971). This can lead to an excess of cases where the actual cause of death is not confirmed. Most of the inaccuracy in IHD death certification has been found amongst deaths in the very old. The reason for this is that the very elderly frequently suffer from co-morbidity which can make it difficult for the certifying doctors to differentiate between obscure signs and symptoms to identify the *underlying* cause of death (Lloyd-Jones *et al*, 1998).

Death certification for IHD would appear to be accurate in approximately 70% of certificates. This thesis has attempted to confirm the mortality rates generated from the GROS data by comparing them with mortality trends reported in the literature. The findings of this thesis are similar to those discussed elsewhere (see Charlton *et al*, 1997). As the highest levels of inaccuracy were found amongst the very elderly mortality trends amongst younger age groups (see figure 8.5), where certification is more reliable, have been considered separately. IHD mortality trends are discussed below.

8.2.3.2. Pattern of decline

Between 1950 and 1999 the male IHD death rate declined by 53% and the female rate by 35%. However, focus on the start and end points of a time period fails to take into account the pattern of mortality in-between. Male IHD mortality did not see a linear decline from 1950; rather, the death rate increased to reach 4 per 1000 in 1963, then remained high until the late 1970s, after which it went into a period of decline, only reaching under 3 per 1000 in 1989, before declining rapidly to under 2 in 1999 (figure 8.1). The 45-64 age group (figure 8.5) followed a similar pattern but with a steeper increase and decrease. The female death rate has been in decline since the 1950s. However, the pattern was not one of constant steep decline; rather, after an initial period of gentle decline the rate stagnates until the late 1970s, after which a pattern of decline was re-established, accelerating in the 1990s. The female 45-64 age group did not experience the large rise in mortality seen amongst men. However, it did not see any sustained decline until the 1980s. Therefore, it is not until the late 1970s that both sexes and all age groups were experiencing a continuous decline in IHD mortality.

The stagnating or high death rates seen in the period before the late 1970s raise the question: had IHD mortality always been high, or were there influences at work peculiar to the post-1950 period which caused the increase in male, and stagnation of female, mortality? If the pattern of mortality witnessed post-1950 was the continuation of a longer term trend, then the factors underlying this must have been in existence prior to this time. However, if the high post-1950 mortality was recently established then its cause can be related to changing conditions at that time. In order to consider mortality trends dating back to 1930, deaths for IHD by sex and age group, for the years 1930 to 1949, were isolated from the GROS death data and added to the IHD dataset.

Figure 8.6 displays the standardised IHD death rate from 1931-1999. This reveals that at the start of this time period, IHD death rates were relatively low but experienced a steep increase up until World War Two, during which they declined. The post-war pattern of mortality was high and increasing for men, whilst the female rate, although remaining high, did not see any further increase. The dramatic increase in mortality since at least 1930 was noted by contemporary observers (see Ryle and Russel, 1949, Cassidy, 1946). If the factors underlying this large rise in mortality can be identified, they will provide a context in which the post-1975 decline in mortality can be considered.

Figure 8.6. IHD death rates, per 1000, standardised to the 1950 population, 1931-99

Source: Original analysis of records obtained from the GROS.

8.2.4. Existing literature

In the case of IHD, unlike tuberculosis and stomach cancer, there is an existing body of literature which has attempted to measure the contribution of medicine and other factors to the decline of this disease since the 1970s (see box 8.2). Four of these studies relate to countries where IHD mortality had begun to decline earlier than in Scotland. These found that the most significant factor influencing mortality decline was the reduction in the prevalence of risk factors. None of these studies reviewed the influence of all the main risk factors, but rather focused on the effect of smoking and cholesterol. In addition, little attention was given to the role played by medicine in either the prevention or treatment of IHD, except to a limited degree in the study from the USA. The Finnish study did not look explicitly at the role of medicine but did attribute the residual decline to medicine.

In their study of the influences on IHD mortality between 1975-1994 Capewell *et al* (1999) examined the contribution of medical treatments and risk factor decline. This thesis differs from that of Capewell *et al* in that it covers the overall decline in mortality, in which IHD forms a case study. However, when considering the decline of IHD there are also a number of differences between the aims of this thesis and that of the study of Capewell *et al.*

Capewell *et al* offer final conclusions on how much each factor, whether medical treatment or risk factor reduction, contributed to the overall decline in mortality, but they do not discuss the context in which these changes occurred. For example, reductions in smoking are isolated as having been the single largest contributor to the decline in mortality, responsible for 36% of the decline, but no explanation is given as to why this decline occurred. Did smoking decline because the public modified its behaviour in response to medical advice, in which case the reduction could be attributed to the work of medicine? However, consideration also has to be given to other possible influences. This study is interested not only in the actual relationship between risk factor prevalence and mortality, but in what caused the prevalence of the risk factor to decline: that is, whether it was due to medical or non-medical influences. In chapter three two groups of medical influence were defined. Examples of what this thesis has viewed as medical influences on IHD are provided in box 8.3.

Box 8.2. IHD case studies

Study	*Country*	*Years covered*	*Main findings*	*Comments*
Goldman and Cook (1984)	USA	1968-1976	Findings are based on a review of the literature. IHD mortality declined by 21%, 31% of decline attributed to medical interventions and 54% to a decline in cholesterol and smoking.	
Jackson and Beaglehole (1987)	New Zealand	1968-1980	Findings are based on data acquired form government departments from which the predicated decline associated with risk factors was calculated. IHD mortality declined amongst men aged 35-64, estimated that between 31% and 51% of the observed decline in mortality was linked to changes in diet and smoking.	Study only considered reduction in changes in diet and smoking and no attempt was made to measure the contribution of medicine.
Sigfussonn *et al* (1991)	Iceland	1968-1988	Findings are based on population surveys. IHD mortality amongst men aged 46-64 declined by 34%-37%. Study predicted that, taken together, reductions in smoking, cholesterol and blood pressure should have led to a reduction in IHD mortality of 35%, which closely matched the observed decline.	Study only considered the role of risk factor reduction and not medicine.

Vartiainen *et al* (1994)	Finland	1972-1992	Study reviewed influences on the decline of mortality amongst 30-59 year olds using data from population surveys. Until the mid 1980s IHD mortality declined in line with predications based on the reduction in smoking, cholesterol and blood pressure, after which it declined at an accelerated rate. Overall 80% of the decline of male mortality and 72% and the decline in female mortality were attributed to the decline in the prevalence of risk factors.	Although this study did not deal specifically with the influence of medical technologies it acknowledged that the accelerated decline seen since the 1980s was probably due to advances in medicine.
Capewell *et al* (1999)	Scotland	1975-1994	Study considered the contribution of risk factor reduction *and* medical treatments to the decline of IHD. The contribution of risk factor change was calculated from data in population surveys. The individual contribution of medical treatments was calculated using effectiveness data from trials, combined with patient uptake. In total 60% of the decline in mortality was attributed to risk factor reduction and 32% to treatments.	

Box 8.3. Medical interventions to reduce IHD risk factors

Direct interaction of medicine with at risk individual
This includes the treatment of high risk individuals, such as the use of hypertensive treatments in those with high blood pressure. In addition, any instance where an individual receives personalised advice would be included in this category, for example, counselling for smoking cessation.
Health education strategies
These are organised campaigns to change behaviour, these can take a number of different forms, including mass media campaigns designed warn the public of dangers and encourage risk factors reduction.
In-direct health education
That is, when the public is informed of a risk via a non medical medium. This normally takes the form of the media reporting research finding.
Government regulation and legislation.
By acting on medical advice the government is able to legislate against certain risk factors, to encourage reduction. Examples of this include taxation on cigarettes and food regulations.

This study will make use of the findings of Capewell *et al* on the contribution of medical treatments and risk factor reduction, but will also provide a discussion of how these changes came about. All the studies in box 8.2 have placed risk factor reduction as the main cause of the decline in IHD mortality. By considering why these risk factors declined in Scotland, a more wide-ranging discussion of the overall role of medicine in this decline will be possible.

One other distinguishing feature of this thesis, compared to the study of Capewell *et al,* is the timeframe. It is argued here that, in order fully to understand why IHD mortality declined since the 1970s, the reasons why it was originally so high must first be understood; therefore this study will extend over a longer time period. Timing is also important when considering the role of medical technologies. Capewell *et al* provide an estimate of the contribution of medicine over the whole of their time period. This study will look at the dates at which these interventions were introduced in order to measure their relative contribution at different time periods. In addition, the impact of medicine post-1994 will be considered.

Thus, this thesis will draw on the findings of the existing work on the decline of IHD, but will consider the broader influences on the disease. This will be done within the context of the rise (pre-1975) and fall (post-1975) of the disease. Within these time frames the relative influence on mortality of the actual numbers dying of the disease (survival), in contrast to the numbers developing the disease (incidence), will be considered.

8.2.5. Influences on mortality pre-1975

Within this section influences on the rise, and subsequent high levels, of IHD mortality will be discussed. This will be achieved by first considering whether mortality rose due to an increase in the numbers dying of the disease once already ill, or as a result of an increase in the numbers of people developing the disease in the first instance. By identifying the factors which caused an increase in mortality, changes in these factors post-1975 can then be discussed to establish the extent to which, and the reasons why, they decreased.

8.2.5.1. Survival (pre-1975)

In the previous case studies, discussion began by considering the ways in which medicine and standards of living may have *improved* survival from the disease over time. However, given the upward trend in IHD mortality seen since the 1930s (see figure 8.6), the question being posed is: why did so many people fail to survive IHD?

8.2.5.1.1. The role of medicine (survival pre-1975)

Pre-1975 medicine offered few treatments which could improve the survival of those suffering from acute MI or chronic IHD. Before 1950 one of the main reasons for this was a lack of interest by the medical profession in the condition, with medical text books in the 1920s paying '*scant notice*' to IHD (McNee, 1925: 44). The situation had not improved by 1949 when a review of IHD claimed that "*...therapeutics in so far as a number of useless remedies have been discarded, have not registered any outstanding gains...*" (Ryle and Russel, 1949: 370). A number of factors contributed to this apparent indifference by the medical profession to the treatment of IHD. One of these was that, prior to the 1920s, the disease had been viewed as relatively rare, and therefore had not been the focus of attention. In addition, many had not considered IHD as a disease which could be treated; rather, it was seen as an inevitable part of the ageing process.

After 1950 some treatments for IHD did start to become available, although the extent to which these improved survival is debateable. In the treatment of acute MI cardiopulmonary resuscitation began to be used, although initially this was mainly within a hospital setting (Cobb *et al*, 1975). This may have benefited the few who received it during the acute stage of the disease, but would not have addressed the underlying problem of narrowed arteries.

The main advance in treatment before 1975 was the introduction of coronary artery by-pass grafting (CABG). This surgery involves the removal of a length of vein from the leg, arm or chest, which is attached above and below the blocked section of the coronary artery, and restores blood supply by 'bypassing' the problem area. Experimental research into this procedure began in the early 20th century when attempts were made to perform the procedure on dogs. Experimentation on humans had to wait until the invention of the first effective heart and lung by-pass machine in 1953, which allowed the procedure to be performed on a non-working heart (Connolly, 1978). The first successful operation on a human took place in 1964. However, this was not reported until nine years later (Garrett *et al*, 1973). Instead, the first operation to be widely reported was that of Favaloro and colleges which took place in 1967 (Favaloro, 1968). This operation had a very low mortality rate of 4.1%, although at the time some doubts were raised about the role of the surgery in increasing survival in the long-term (McIntosh and Garcia, 1978). Although this operation became well publicised the extent to which it contributed to improving survival from IHD in Scotland before 1975 can be questioned. The first CABG did not take place in England until 1970 (Favaloro, 1998) and in 1981 only 350 operations were undertaken in Scotland (Pell, 2002). This suggests that CABG would not have been a widely used procedure before 1975.

Although some advances were made in the treatment of acute MI and operative techniques to clear arteries, it is unlikely that this had much effect on the majority of people who had IHD. Most people's first experience of the disease was when they suffered a MI, with many dying soon after this. The minority who survived to reach hospital could be resuscitated if they suffered a repeat attack but little could be offered in the way of long term treatment. Although CABG was available by the 1970s, this was still a relatively new procedure which would only have benefited the handful of patients who received it. Thus, as the century progressed medicine could offer some treatment, only to very few and the overall contribution to the reduction in IHD mortality would probably have been small. As medicine began to acknowledge the problem of IHD after the Second World War, interest and research into possible treatments grew, but the benefits of this was not felt until after 1975.

8.2.5.1.2. Standards of living (survival pre-1975)

The factors related to standard of living which influence prognosis once the disease is established are similar to those which influence disease incidence and will be discussed in depth in the following section.

8.2.5.2. Incidence (pre-1975)

Although medicine may have done little to cure those suffering from IHD before 1975, it is unlikely that it actually increased the numbers dying once they were already ill. A more plausible explanation for the dramatic rise of IHD in the early 20th century lies with the increase in the incidence of the disease. The relative contribution of medicine and standards of living to disease incidence is discussed below.

8.2.5.2.1. Role of medicine (incidence pre-1975)

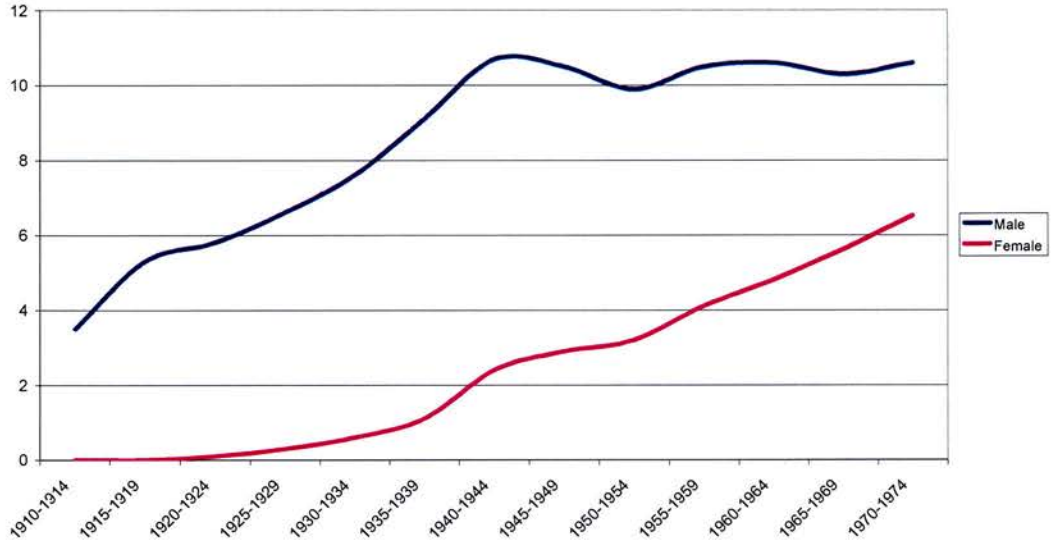
Medicine can only affect the incidence of a disease once it has identified the factors which cause ill health. As has been discussed, IHD was still viewed as a relatively rare condition in the early part of the 20th century; this meant that large-scale research into its causes did not begin until after World War Two when several epidemiological studies were launched. These studies required time for their findings to be disseminated and to have an impact on practice. Therefore, throughout much the pre-1975 period the medical profession, and the public, remained in ignorance of the true causes of the disease.

In the absence of systematic research into the causes of the disease, various other factors were believed to be responsible for the epidemic. One of the most popular beliefs was that the disease was induced by the 'special stresses' of the professional classes. This can be illustrated by the results of a study into the causes of the rise of IHD in Oxford between 1921-1945 (Ryle and Russel, 1949). In this study deaths from IHD were isolated and common factors were sought. The main finding was that the disease was most common in males of social classes I and II. When trying to explain the high mortality in this group none of the risk factors which are accepted today were considered, (for example a lack of physical exercise, or a tendency to eat a high fat diet). Rather, the explanation offered was that the main cause of the disease was mental stress, as those in the professional classes suffered from '*sustained mental activity*' and '*special stresses*' (p.387), whilst those employed in manual jobs found their work, "*...from the point of view of mental activity, relatively less exacting*" (p.379). Thus, the rise in IHD was attributed to the growth in education and professional jobs since the 19th century, which placed the men occupying them at higher risk because of the mental strain involved.

Ignorance on the part of the medical profession could in some cases lead to the encouragement of behaviours which are now regarded as risk factors. In the case of

smoking, it was common practice for a doctor to offer a cigarette to a patient to put them at ease (Doll, 1998). When criticised, some doctors were even prepared to defend the behaviour, such as the King's Physician who claimed in 1948 that, "*...the driving of a car in traffic would produce more adverse circulatory effects than the smoking of several cigarettes in an armchair at the club*" (Cassidy, 1946: 589). Meanwhile, in the absence of any medical warning to the contrary, the consumption of cigarettes continued to increase (see figure 8.7).

Figure 8.7. Manufactured cigarettes smoked per person, per day, 1910-74



Source: Adapted from Wald *et al,* 1988.

Smoking is considered one of the main risk factors for IHD. However, it was not until 1950 that the health effects of smoking first began to be seriously considered. In this year five studies were published, these early studies did not investigate the link with IHD, instead they linked smoking with lung cancer. Four of these studies originated in the USA, but one was a British study by Doll and Bradford Hill (1950). The results of this study led to the MRC setting up a prospective study of doctors and smoking. This began in October 1951 when all the doctors on the British Medical Register were sent a questionnaire on their smoking behaviour. At the time 62% of doctors smoked. The cause of subsequent death amongst these doctors was then recorded (Doll *et al*, 1994). Two and half years after this study began a link between smoking and lung cancer was demonstrated (Doll, 1956). These findings, however, were not immediately accepted. One of the main objections to the research involved cause and effect. It was argued that, as not everybody who died of lung cancer was a smoker, then the link with smoking could not be proven. This debate on the link with lung

cancer continued into the late 1950s before laboratory evidence confirmed the epidemiological findings.

Although the link between smoking and increased mortality, at least in the case of lung cancer, had been shown as early as the 1950s, this knowledge took time to accepted by the medical profession. By the 1960s most doctors recognised that smoking endangered health. The next challenge they faced was to communicate this knowledge to the public. The first attempt was the publication of the Royal College of Physicians Report on smoking in 1962. This received widespread media coverage, and was followed by a 5% drop in cigarette sales (Royal College of Physicians, 1983). However, at this time no organised strategy for the prevention of smoking was in place and sales soon recovered. In other nations, such as Finland, successful attempts were made earlier to implement successful anti-smoking strategies which had a corresponding effect on prevalence levels (Vartiainen *et al*, 1994). In Britain it was post-1975 before serious attempts were made to reduce smoking. The factors influencing high rates of smoking will be discussed in the following section.

One possible area in which medicine may have directly affected the incidence of IHD prior to 1975 was the control of hypertension, through the availability of medications which could help to control high blood pressure. However, although these treatments may have benefited the few who received them, this would not have had an effect at population level, as there was no organised programme to identify high blood pressure, leaving most hypertensives in the community undiagnosed.

It cannot be argued that medicine actually caused incidence to rise. However, it also did little to cause incidence to decrease. The lack of interest in the disease in the earlier decades of the 20th century, when it was still comparatively rare, meant that throughout much of the pre-1975 time period the main IHD risk factors remained unpublicised. Once these were identified by the post-1950 epidemiological studies, time was required to communicate these findings to the rest of the medical profession, after which strategies had to be devised to inform the public about these risks and influence their behaviour. The effect of these will be discussed in section 8.2.6.

8.2.5.2.2. Standards of living (incidence pre-1975)

It is unlikely that the steep rise in mortality observed since 1931 was brought about by an increase in the numbers of people who died once they developed IHD. Although medicine did not introduce many life saving technologies, neither did its treatment deteriorate to cause an increase in deaths amongst those already ill. Although data on the incidence of IHD pre-1975 are not available, a more plausible explanation is that the death rate increased because greater numbers were developing the condition. The main factor which dictates the incidence of a disease is the prevalence of its risk factors in the population. This section will consider what happened to the main IHD risk factors prior to 1975, and the influence which improvements in standards of living may have had on them.

- **Cholesterol**

The main cause of high cholesterol levels is a diet high in saturated fat (see box 8.1). Any increase in saturated fat in the diet is associated with an increased risk of developing IHD. Although studies were undertaken into the relationship between cholesterol and the risk of developing IHD, (Bronte-Stewart *et al,* 1956) this risk was not widely known amongst the population; rather, a diet high in saturated fat was regarded by many as a desirable consequence of rising standards of living (Nelson, 1993). A change in the levels of fat in the diet is evident from the end of the 19th century, especially amongst the poorest in society, whose diet had previously been deficient in saturated fat (Nelson, 1993). Several factors were responsible for the increase in fat in the diet, such as rising real income and the growth of meat imports. These were discussed in the sections 6.4.3.2 and 7.5.2.2. where it was argued that these changes in diet actually helped bring about the decline of tuberculosis and stomach cancer mortality. As well as increasing fat consumption overall, the increasing availability and affordability of fatty foods, such as red meat, led to the relatively greater growth in fat consumption amongst the poorest compared to the richest. In 1900 the poorest consumed an average of 39 grams of fat a day, compared to 99 grams amongst richest; by the 1930s this had increased to 84 grams amongst the poorest and 142 grams amongst the richest (Nelson, 1993). Therefore, although the richest in society were still eating substantially more fat, consumption (and related risk) had increased for all groups.

The intervention of World War Two led to a temporary reduction in fat consumption for all, as rationing of foods such as meat and dairy products limited access to fat. The trend of increasing fat consumption was re-established at the end of the war, as people enjoyed access to goods which poverty, or rationing had caused to be viewed as luxury items. Figure 8.8

reveals the effect that this had on fat consumption. These data refer to the whole of the UK but give an indication of the likely situation in Scotland. The overall trend in fat consumption from the mid 1940s is one of increase, staying consistently high until the early 1970s when the start of a decline is detected.

Figure 8.8. Consumption of fat in the UK per person, per day (in grammes) 1943-74

Source: Adapted from data obtained from the National Food Survey.

An increase in fat consumption among all parts of society is therefore apparent since the end of the 19th century. The adoption of a diet high in saturated fat does not immediately lead to an increase in IHD deaths; rather, there is a time lag as the arteries only become blocked slowly over the lifecourse. Therefore, it would be expected that the increase in fat consumption in the late 19th century would not bring about an increase in IHD risk until the 20th century, which has been observed. Considering the role of standards of living in the rise of this risk factor is interesting, as normally increased risk of disease is associated with poor living standards. However, for the majority, rising real wages brought about the increase in risk, as a diet high in meat and dairy produce was regarded as desirable. In the past meat had been a luxury item, and the absence of meat from the diet seen as an indictor of poverty. For many the increase in meat consumption represented a move away from the old diet of poverty, whilst increasing their risk of developing IHD.

- **Smoking**

It has been argued that smoking was not widely recognised by the medical profession as a dangerous activity until the 1950s, with the diffusion of this knowledge to the public only beginning in the 1960s and 1970s. This section will consider the reasons why smoking increased throughout the 20th century, and the effect which this had on the incidence of IHD.

The global smoking epidemic has been modelled, and provides stages through which a population travels in both their smoking behaviour and its health consequences (Lopez *et al*, 1994). In the first stage of the smoking epidemic smoking prevalence is comparatively low amongst men, at around 15%, whilst female consumption is very low. This results in a per capita consumption of less than 500 cigarettes annually. At this stage in the epidemic smoking would have little impact on the health of the population. In the second stage, male prevalence rises to reach a peak of 50-80%, whilst female smoking, although increasing, lags behind that of men. It is during this stage that deaths attributable to smoking rise, especially amongst men.

These stages in the smoking epidemic can be identified in Scotland before 1975, with smoking prevalence first beginning to increase in the early years of the century, reaching a peak of 80% amongst men by the end of World War Two (Doll, 1998). The health consequences of this on men can be seen in figure 8.5. This graph shows the dramatic increase in male IHD mortality, as compared to the more moderate female increase, in the age group 45-64. This age group is of interest when considering the mid 20th century as these are the men who would have been exposed to cigarette smoking for their entire adult life, and it is amongst this group that the adverse consequences of smoking first became apparent.

It is now acknowledged that smoking is a major risk factor for IHD, and that IHD mortality increased first amongst the group which was first exposed to mass cigarette smoking, by increasing disease incidence. However, this study is also interested in the reason *why* cigarette smoking increased to bring about this augmentation in risk. A number of factors influenced this, starting with the mechanisation of cigarette manufacture from 1883. This led to mass produced, ready rolled cigarettes, which became increasingly affordable as cigarette companies competed with each other for market share. In the case of men the intervention of two world wars served to accelerate the growth in smoking prevalence. Meanwhile, female smoking remained relatively low until the 1920s when cigarette

companies began to market their product directly at women. By the 1930s smoking was being linked with female independence (Elliot, 2001). Therefore, the epidemic of smoking experienced by Scotland, and its resultant increase in IHD risk, can be linked to industrialisation and mechanisation, leading to a cheap, convenient product, whilst marketing increased its desirability and increasing real wages its affordability.

- **High blood pressure**

High blood pressure is a risk factor for IHD. However, is it more strongly associated with stroke. Therefore, factors which may have influenced the decline of blood pressure during this time period will be discussed in the section dealing with stroke (8.3.5.2).

- **Physical activity**

It is difficult to measure accurately the extent to which levels of physical activity declined before 1975. A number of factors, in particular the increase in mechanisation during this time period, suggest that activity would have been decreasing for many. In the workplace, activities which previously required manual labour were replaced by machinery. In the household, domestic chores which would have required sustained activity, such as cleaning carpets and clothes, were increasingly replaced by less labour intensive technologies such as carpet sweepers and washing machines. In addition, although by the late 1960s only half of households had access to a car (Social Trends 33, 2004), the development of public transport throughout the century reduced the need to walk significant distances. Therefore, it seems likely that, although hard manual activity still formed a large part of the day for some, the daily physical demands placed on many had decreased.

- **Poverty**

Poverty is now regarded as one of the leading risk factors for IHD, with the poorest in society shouldering a greater burden of disease. However, for much of the pre-1975 time period this was not the case. Rather, up until the 1960s men in social classes I & II suffered an excess of mortality over those in classes IV & V (Marmot, 1978). This excess mortality amongst the most affluent was noted by the medical profession throughout the first half of the 20th century, and it was the need to explain this which led to theories on its causality, such as the 'special stresses' suffered by the professional man discussed in section 8.2.5.2.1. 'Special stresses' were not the main cause of high IHD rates amongst professional men; rather, they were the first to suffer from an excess of what we now know to be the risk

factors for IHD. It was amongst affluent men that smoking first became popular, they were the first group with access to a diet high in saturated fat, and the nature of professional occupations would have reduced the amount of physical activity in which they participated. For those who lived in poverty, on the other hand, smoking was not as affordable, meat was a luxury item and many had to work physically hard in their occupations. However, as the standard of living of many began to improve, so did their ability to pursue a lifestyle previously limited to the upper and middle classes, and the class divide in IHD mortality gradually began to narrow. By the 1960s the IHD death rate for all social classes was roughly equal.

8.2.5.3. World War Two

Prior to the 1970s, the only time when both sexes and all age groups experienced a decline in IHD mortality was during World War Two. Therefore it is useful to consider what happened to IHD risk factors during this time, as this may indicate what changes were required to bring about the sustained decline in mortality seen in later years.

There are a number of possible explanations for the wartime decline in IHD mortality. It has been argued here that the increase in fat consumption dating from the late 19th century was in part responsible for the increase in mortality; this trend, was reversed during the war. The introduction of rationing meant that the population's diet typically saw an increase in the consumption of bread, milk and potatoes and a reduction in foods high in saturated fat, such as butter, visible fats, meat, eggs and bacon (Zweiniger- Bargielonska, 2000). This may have prevented or delayed an increase in the level of an individual's cholesterol. In addition, the conditions of war led to the mobilisation of population groups in the war effort who may previously have been physically inactive, such as older men and women recruited to the industrial and farming jobs vacated by younger men. The mobilisation of the population in the war effort, together with the equalising effect of war conditions, may have increased feelings of social cohesion and lessened the psychosocial effects of poverty. Although there was an increase in smoking amongst both men and women during the war the health consequences would not be felt until later.

It is difficult to measure the contribution of these factors to the reduction of mortality. The main effect of the war was to create greater social equality than previously or subsequently. The consequence of this was that everyone (in theory) ate the same diet and had access to the same goods and activities; it is possible that this may have been the main influence of the

war on IHD. It should be remembered that the health consequences of the war were not the same for all the diseases considered in this thesis. Although the circumstances may have been favourable for IHD, they were unfavourable for certain other diseases, such as tuberculosis.

8.2.5.4. Conclusions (pre-1975)

The dominant trend in IHD mortality prior to 1975 was one of increasing or high death rates. It has been argued here that this was unlikely to have been the result of an increasing number of people failing to survive the disease once they were already ill. In the treatment of the disease few advances were made which could have benefited those who were ill. Although the groundwork for the major medical advances were laid at this time, their benefit was only felt post-1975.

A more probable explanation for the high pre-1975 IHD mortality is that the incidence of the disease increased. That is, a greater proportion of the population was developing the condition than ever before. This can be linked to the rise in prevalence of what are now recognised as the main risk factors for IHD: high fat diet, smoking and diminishing physical activity. In the absence of medical advice to the contrary, these were seen as the desirable consequences of improving standards of living. These activities were originally more prevalent amongst the more affluent classes, and this is reflected in their initially high death rates. However, as wages rose and industrialisation made these products more accessible, as well as physical activity less necessary, they were adopted by all ranks of society. The result was a growth in the levels of IHD.

8.2.6. Influences on mortality post-1975

The earlier trend of increasing and high death rates was reversed post-1975 as Scotland's IHD death rate declined significantly. This section will examine the main influences on this decline, and will consider the relative impact on mortality of the improving survival of those who have already developed IHD, and the effect of declining disease incidence. The findings from Capewell *et al* (1999) on the relative contribution of both medical treatment and risk factor reduction will provide a framework for this analysis.

8.2.6.1. Survival (post-1975)

8.2.6.1.1. The role of medicine (survival post-1975)

It has been argued that, with the possible exception of cardiopulmonary resuscitation and CABG, medicine made little contribution to improving survival before 1975. This situation changed post-1975 and medicine now offers a wide variety of treatments to improve survival during acute MI and to postpone subsequent death. Capewell *et al* (1999) calculated that between 1975 and 1994 treatments for IHD accounted for 32% of the observed fall in mortality. These treatments, which fall into a number of areas, are discussed below.

- **Treatment for acute MI**

Capewell *et al* estimated that between 1975 and 1994 advances in the treatment of acute MI were responsible for 10% of the observed reduction in mortality. These treatments include:

Resuscitation

This treatment aims to restore heart rhythm and maintain blood flow throughout the patients cardiovascular system, and can prevent death during the acute stage of MI. Manual cardiopulmonary resuscitation was available prior to 1975. This was joined post-1975 by defibrillators which produce an electric charge to restart the heart. The use of these treatments increased after 1975. Initially they were hospital-based, and as such could only benefit those who suffered a MI when in hospital. In the 1980s ambulances began to be fitted with defibrillators to allow out-of-hospital resuscitation, although the extent to which this improved survival at this time is debated. A study examining the effectiveness of defibrillators in ambulances in Glasgow between 1984-1990 found that they only led to a 1% improvement in overall survival. The main reason for this poor success rate was delay in calling for help after a MI, as sometimes members of the public failed to recognise the

symptoms of a MI, or first called their GP, thus delaying the arrival of the ambulance crew (Leslie *et al*, 1996).

Beta blockers and ACE inhibitors

These medications are given post MI to reduce the pressure on the heart. Beta blockers do this by slowing the heart beat and ACE Inhibitors by increasing blood flow. Although beta blockers were available relatively early in this time period, their use increased greatly from the mid 1980s, whilst ACE Inhibitors only became available later. This increase in usage is reflected in the number of MI survivors who received them, which rose from 32% of male and 32% of female survivors, who had a MI, receiving beta blockers in 1985-87, to 54% and 50%, respectively, in 1992-94. ACE Inhibitors were not available in 1985-87 but by 1992-94 28% of men and 33% of women who survived a MI received them (Tunstall-Pedoe *et al*, 2000). These treatments have also been used in the secondary prevention of MI (see below).

Thrombolytic treatments

Thrombolytic treatments have been revolutionary in the treatment of acute MI. A MI occurs when a blood clot blocks the blood supply to the heart; thrombolytics have been designed to break down the clot, thereby restoring blood flow. These drugs first became available in the mid 1980s, and when used in conjunction with aspirin, have been shown in trials to reduce mortality by 42% following MI (Lip *et al*, 2002a). The growth of these drugs is reflected in their increased use amongst those suffering non-fatal MI, from 10% of males and 9% of females in 1985-87 to 48% and 49%, respectively, in 1992-94 (Tunstall-Pedoe *et al*, 2000). The growing recognition of the effectiveness of this treatment led to its use became increasingly widespread during the 1990s.

- **Secondary prevention post MI**

These are treatments designed to prevent the recurrence of a MI after an individual has suffered an initial attack. They are aimed at maintaining clear arteries and preventing blood clots. Capewell *et al* (1999) have estimated that until 1994 8% of the decline in mortality can be attributed to these treatments.

Aspirin and Warfarin

Aspirin has been recognised as an effective medication since the time of Hippocrates when he used it (in its natural form of willow bark) to ease the pain of labour. Throughout history it has been used for a variety of purposes, although initially mainly in the relief of fever and

pain. In the 1940s the anti-coagulant effect of aspirin was first recognised, but it was not until the late 1970s and 1980s that it began to be used in vascular treatments. In a low dose (75mg) aspirin helps to stop the formation of blood clots by preventing the platelets in blood from sticking together (Mueller & Scheidt, 1994). The other anti-coagulant used to prevent the build up of blood clots is Warfarin. This drug originated from research in the USA into rat poison in 1940s, and the discovery of its anti-coagulant property was an unexpected by-product. Warfarin is a long-term treatment in the prevention of blood clots, and, although first available in the USA in 1950s, its use did not become widespread until the 1980s.

Cholesterol lowering drugs

Any treatment which can lower levels of LDL cholesterol will help to prevent narrowed arteries. Attempts have been made since the 1960s to create a drug which will achieve this; most early attempts were unsuccessful. An example of this was the treatment of men with oestrogen based on the belief that 'maleness' was a risk factor, whilst 'femaleness' offered protection. The intention was that by treating men with the female hormone this could transfer the alleged protection. The results of trials of this treatment were not encouraging. It was found to have a "*significant adverse effect on all clinical outcomes*" (Gould *et al*, 1995: 2280). Co-fibrate drugs became available in the 1960s and these were effective against IHD. The WHO co-fibrate trial found MI and IHD reduced by 20% in men undergoing the treatment; the major side effect was that it led to an increase in mortality from other causes (Oliver, 2000). It was not until the 1990s that the first effective and safe cholesterol lowering drugs became available in the form of statins, in particular simvastatin. In 1994 a large trial of patients in Scandinavia reported that Simvastatin reduced LDL cholesterol by 35% with no adverse effects, and, when compared with a placebo group, the Simvastatin patients saw a 42% reduction in coronary death (Scandinavian Simvastatin Survival Study Group, 1994).

- **Treatments for angina**

The treatments for angina are surgical interventions to improve the passage of the blood which, as well as reducing the symptoms of angina, decrease the risk of developing blocked arteries and MI. Capewell *et al* (1999) have estimated these treatments were responsible for 8% of the decline in mortality until 1994.

CABG

The principle of this surgical procedure has already been outlined section 8.2.5.1.1, and it was argued that although this treatment first became available pre-1975, its use was not widespread. Post-1975 CABG has become an increasing routine part of the treatment of IHD with 2380 people undergoing the surgery in 1994, increasing to 2870 by 2000 (ISD Scotland, 2004).

Angioplasty

This intervention is designed to 'open up' the narrowed section of the coronary artery, allowing improved blood flow. This is achieved by passing an inflated balloon, which is attached to a tiny catheter, through the blood vessel to open it up. Although for most of the post-1975 period this was used mainly as a planned surgery for those suffering chronic disease, it is increasingly being used as an emergency procedure to unblock arteries during acute MI (Lip *et al*, 2000). This is reflected in the growth of operations performed during the 1990s, from 896 in 1994 to 2527 in 2000 (ISD Scotland, 2004).

- **Treatment of heart failure**

Heart failure is a condition made up of a collection of physical signs and symptoms, including breathlessness, fatigue and fluid retention. Its main cause is IHD, therefore any improvement derived from the treatment of IHD would also have had a positive affect on those suffering heart failure. Before the 1990s treatments for heart failure focused on relieving the symptoms of the condition, but would not have affected survival. This changed with the introduction of ACE inhibitors, which Capewell *et al* (1999) estimated accounted for 8% of the decline in mortality until 1994. Use of this treatment has the potential for expansion as by 1998 only 30% of heart failure patients were receiving it (Heart Failure Strategy, 2004)

8.2.6.1.2. Standards of living (survival – post-1975)

Once diagnosed with IHD a change in lifestyle can influence the prognosis of the disease. Most of the changes which can be made, such as a low fat diet and taking regular exercise, are the same as those which would be taken to reduce the risk of developing IHD in the first place. It is difficult to say if these changes could have occurred in the absence of medical advice, and this issue will be discussed further in the section dealing with risk factor reduction.

8.2.6.1.3. Conclusions (survival – post-1975)

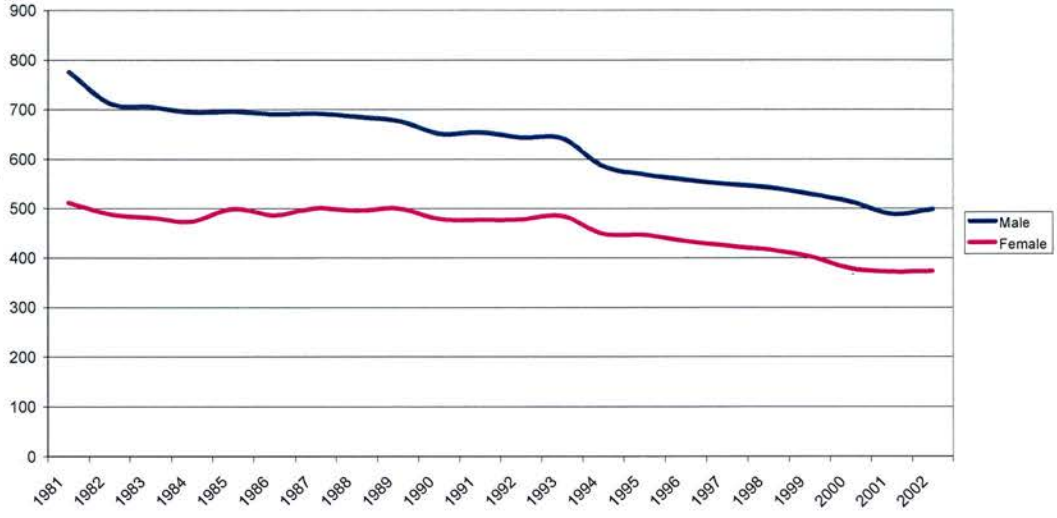
In 1975 medicine could offer few treatments to improve survival once IHD had developed. By 1999 treatment had undergone a revolutionary change. Capewell *et al* have estimated that the medical treatments aimed at improving survival, once the disease is established, accounted for 32% of the decline in IHD mortality between 1975 and 1994. This estimate, however, appears at first to contradict that found by the international studies of IHD reduction (see box 8.2), which either failed to consider medicine as an influence, or only attributed a minimal reduction in mortality to it. This thesis has reviewed the medical treatments selected by Capewell *et al* and has found that treatments with proven success are now available for almost all aspects of IHD. The timing of the international studies may explain why medicine at first appears to have been more successful in Scotland than elsewhere. The majority of these studies end before the 1990s. Most effective treatments, such as statins, thrombolytics and ACE Inhibitors, only became available in the mid 1980s, and so would not have been in use when the earlier studies were conducted. Given that these treatments date from the 1980s, and that earlier studies do not view medicine as a significant factor in the mortality decline, it can be argued that medicine only became effective in improving the survival of large numbers suffering from IHD after the mid 1980s. Once these treatments became available they did have an important effect on survival, with 28-day fatality rates declining from 49% for men and 49% for women in 1985-87, to 44% and 41%, respectively, in 1992-94 (Tubstall-Pedoe *et al*, 2000).

Given the lack of effective medical treatments prior to the mid 1980s, it can be argued that the majority of the 32% of the mortality decline attributed to medicine by Capewell *et al* between 1975 and 1994 occurred after the mid 1980s. Consideration must also be given to the role of medicine after 1994, during which time the use of IHD treatments became increasingly widespread. This can be illustrated by the fact that by 2003 80% of MI patients were receiving thrombolysis treatment within 30 minutes of arriving at hospital (Andolo, 2004). It is therefore likely that medicine contributed more than 32% to the overall decline in mortality.

Thus, since the mid 1980s medicine has become increasingly effective at preventing death amongst those who suffer from IHD. The death rate from IHD has, however, been in decline since the late 1970s, before most of the new IHD treatments became available. Therefore, an alternative explanation for this earlier decline in mortality, as well as the remainder of the later mortality decline, must be sought. Data are available on the incidence of IHD since

1981 in Scotland. Figure 8.9 reveals that the incidence of IHD amongst men has been in decline since at least 1981. Amongst women the incidence of IHD declines in the early 1980s then remains constant until the early 1990s when it again declines. In the following section factors influencing the incidence of IHD post-1975 will be considered.

Figure 8.9. IHD incidence rates, per 100000, standardised to mid-year population, 1981-2002



Source: Adapted from data acquired from ISD.

8.2.6.2. Incidence (post-1975)

Prior to1975 the main influence on the IHD death rate was the high incidence of the disease, and this has been linked to the growth in prevalence of the main IHD risk factors dating from the late 19th century. Figure 8.9 reveals that, since at least 1981, the incidence of the disease in males, and to a lesser degree females, has been in decline, predating the widespread use of effective medical treatments designed to improve survival once the disease is established. IHD incidence is determined by the presence of risk factors in the population and Capewell *et al* (1999) have calculated that 60% of the decline in IHD mortality between 1975 and 1994 can be attributed to the decline of the main IHD risk factors. However, no judgement is made on the contribution of medicine or other factors, such as changing standards of living, to this decline. This section will consider the relative contribution of each of these on the reduction in risk factor prevalence.

It has been argued that pre-1975 medicine could do little to affect the prevalence of the main IHD risk factors. However, the roots of post-1975 prevention strategies lie in this time. Most of the main IHD risk factors were uncovered in the 1950s and 1960s. The discovery of the link between smoking and ill health has already been discussed, with the association of increased IHD risk emerging from research into lung cancer. The other risk factors were identified by large epidemiological studies into IHD, including the Minnesota Businessman Study and the Framingham study, which established the link with cholesterol and high blood pressure (Epstein, 1996). The link with lack of physical activity was first advanced by Morris in the 1950s following his study of London Transport drivers and conductors (Morris *et al,* 1953). The association between poverty and IHD was made in the 1980s and the significance of this risk factor will be discussed at the end of this section. Thus, by the 1960s most of the main IHD risk factors were known. However, this did not lead to an immediate medical response. The prevention of IHD posed a new challenge to the medical profession. Traditionally its approach to prevention had been designed to cope with infections through regulation of the environment, using measures such as sanitary reform. The risk factors for IHD were at first viewed as independent of the environment, dictated by personal behaviour, and the potential for prevention of IHD through modification of these risk factors was not originally appreciated.

By the late 1970s the medical profession was increasingly aware of and had accepted the main risk factors; with this came a growing belief that medicine had a responsibility to prevent the disease. Calls were made for "*major new strategies... [from] ...both the medical*

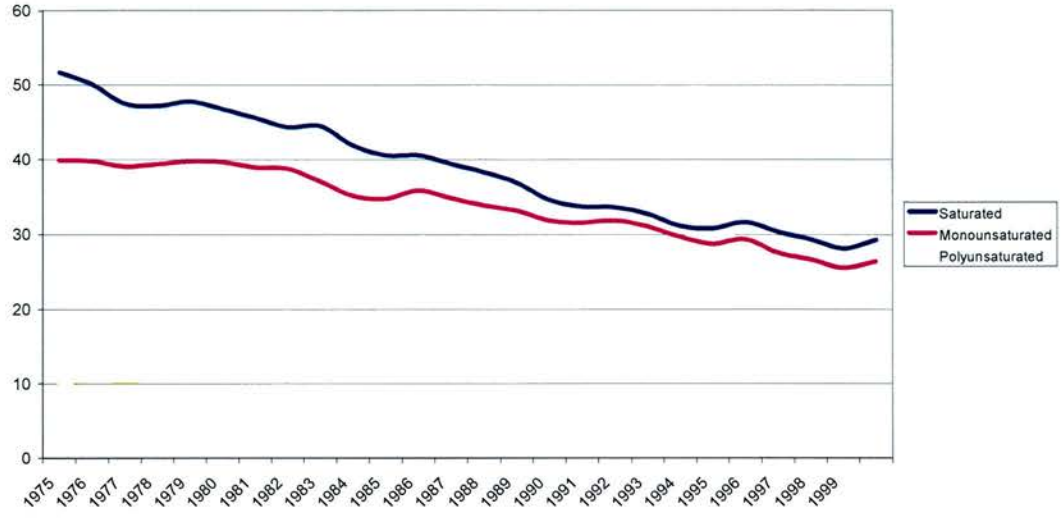
profession and the government to achieve a reduction in the commonest single cause of death in the UK" (Tudor-Hart, 1984). In Scotland disease prevention through health education was the responsibility of the Scottish Health Education Unit (SHEU), which was established in 1968 (later the Scottish Health Education Group (SHEG)). From 1991 it was called the Health Education Board for Scotland (HEBS) before becoming part of the new organisation NHS Health Scotland in 2003. This study views the role of medicine in the prevention of IHD in a broad context, including not only strategies specifically designed to combat certain risk factors, but also the indirect influence of medicine, such as diffusion of knowledge through media discussion of research findings (see box 8.3).

Identifying the role of medicine in risk factor reduction is difficult. To reduce the risk of IHD requires changes in personal behaviour. The main influence on personal choices, such as smoking and diet, had previously been standards of living, as choice was determined by affordability. Once the main IHD risk factors had been communicated to the public, levels of risk factors were not only influenced by affordability but by the knowledge of their health consequences. As risk factor prevalence post-1975 may have been decided by the dual actions of medicine and standards of living these influences will be considered jointly. Therefore, this section will review each of the main risk factors, consider the effect which changes in their prevalence would have had on IHD incidence, and also the extent to which any reduction in these was the result of the medical or non-medical influences. Consideration of the pattern of risk factor prevalence seen pre-1975, when most of the main risk factors were relatively unknown, will help reveal whether these changes were in direct response to medical influences, or whether they might have occurred in their absence.

- **Cholesterol**

Pre-1975 the Scottish diet was characterised by the increasing consumption of fat in the diet. (see figure 8.8) which has been linked to the growth in high cholesterol levels as a risk factor for IHD. Since the mid 1970s this trend has been reversed, with consumption of saturated fat consistently declining (figure 8.10 (these data refer to the entire of the UK but indicate the probable pattern of consumption in Scotland)). This reduction would have had a positive effect on population level of cholesterol. Capewell *et al* (1999) associated 6% of the overall decline in IHD to 1994 to this cause.

Figure 8.10. Consumption of fats at home in UK (grammes per day), 1975-1999



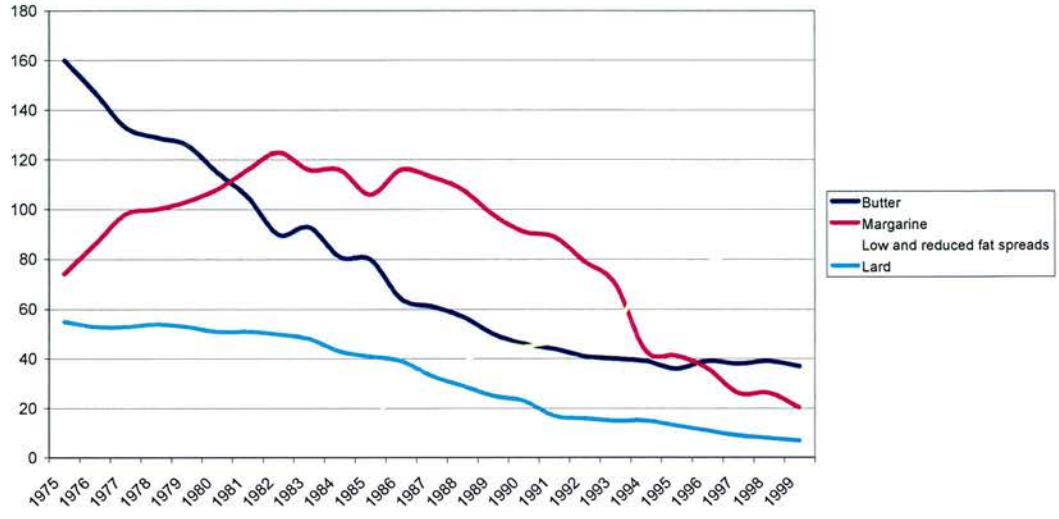
Source: Adapted from data obtained from the National Food Survey.

The dangers of a high fat diet first began to be discussed by the medical profession after the results of the Framingham and Minnesota Businessman studies were published. However, no official, medically designed campaign to promote the dangers of a high fat diet appeared until the 1980s. Rather, it was the food industry which first responded to this research, incorporating the concept of a 'healthy diet' into their commercial advertising, and broadcasting the knowledge of what this entailed (Walker, 1984). The food industry has remained an important influence in informing the public of the latest medical findings on diet. This role has expanded to include the development of foods specifically designed to reduce cholesterol, such as yoghurts, cereal bars and spreads which claim to reduce cholesterol by between 10-15% (Kelly and Stanner, 2003). Certain foodstuffs are now marketed expressly for their health benefits. A current (2004) example of this is Flora Light, which claims on its packaging to help '*to keep your heart healthy... Approved by the Family Heart Association*'.

The food industry has therefore played a significant role in encouraging change in the nation's diet. It incorporated the medical findings into its marketing before any large scale health education campaigns were in existence, and as it is likely that it played a part in the decline seen in fat intake since the mid 1970s. In addition, as the suppliers of the nation's diet, the food industry has also provided the means to change diet, by developing foods which fit the new concept of healthy eating; in particular the uptake of low fat spreads which

has been in evidence since the mid 1980s (figure 8.11). It should be noted that although the by-product of this food marketing and production was to help improve access to a healthier diet, the food industry's primary motivation would have been to ensure profits.

Figure 8.11. Consumption of spreads at home (grammes per week) in the UK, 1975-99



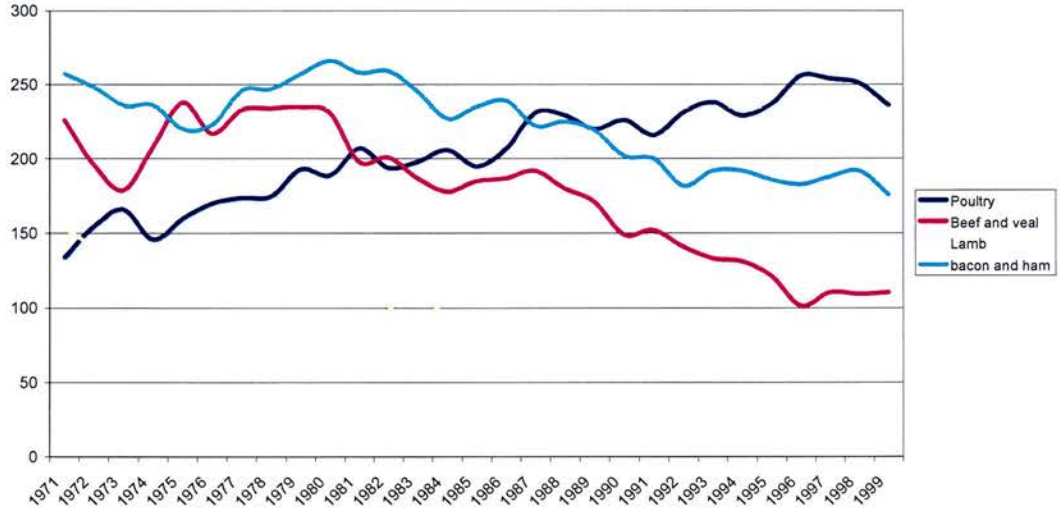
Source: Adapted from data obtained from the National Food Survey.

The government has also attempted to influence the nation's cholesterol levels by setting targets for fat consumption - no more than 35% of total diet to come from fat, of which saturated fat should make up less than a third (Kelly and Stanner, 2003). Several strategies have been implemented to achieve this goal. The most successful have been mass media campaigns which deliver a positive message on how to change diet, such as those recommending the consumption of five portions of fruit and vegetables daily and two portions of fish weekly. Less successful have been the campaigns delivering a negative message, that is those urging people to cut down consumption of certain products. This is reflected in the failure to achieve government targets on fat consumption, with 38% of the UK diet still consisting of fat in 2001 and 11% accounted for by saturated fat. Furthermore, Scotland lags behind the rest of the UK (Kelly and Stanner, 2003).

Thus, since 1975 medicine, through its influence on the food industry, government targets and health education appears to have succeeded, to a limited degree, in modifying population diet, away from foods with a high fat content. Before this date the main determinant of diet

had been income, and the desirable consequence of increasing income was perceived to be an increased consumption of those foods now regarded as less healthy. Given the trend of increasing and high fat consumption which was witnessed since the start of the 20th century, it seems unlikely that decline would have occurred in the absence of medical advice. In the case of certain changes in diet, it is possible that non-medical factors played a part. For example, although chicken is promoted as a healthier option than red meat, it is also a cheaper option which would have increased it attractiveness to consumers (figure 8.12). The growth in low fat spreads may also owe something to convenience, as it spreads straight from the fridge. Evidence from other countries (Vartiainen *et al*, 1994) has shown that strategies to modify diet and so lower cholesterol can be effective. Although the same degree of success has not been achieved in Scotland, medicine would appear to have been responsible for the majority of the 6% of decline in mortality, estimated by Capewell *et al*, through its direct and indirect influence on the population's eating habits.

Figure 8.12. Consumption of meat at home in the UK (grammes per week), 1971-1999



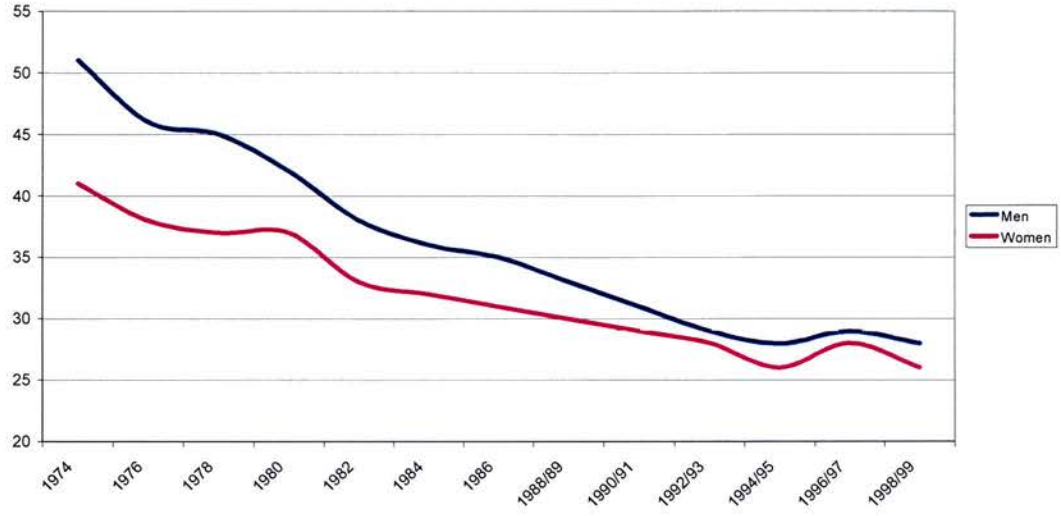
Source: Adapted from data obtained from the National Food Survey.

- **Smoking**

The dominant trend in smoking prior to 1975 was one of increasing or high death rates which correspond with stage 1 and 2 of the model of the smoking epidemic in the developed world. Post-1975 Scotland experienced stages 3 and 4, when smoking prevalence first began

to decline, followed by a decline in mortality attributable to it (Lopez *et al*, 1994). This is reflected in a decline in smoking prevalence between 1975-1999 from 51% to 28% among men, and 41% to 26% among women (see figure 8.13). Capewell *et al* (1999) calculated that this fall in smoking prevalence accounted for 36% of the total decline in IHD mortality between 1975 and 1994. The finding by Capewell *et al* (1999) that the reduction in smoking prevalence had a sizeable effect on IHD mortality is supported by a correlation analysis performed by this thesis. This analysis considered the influence which the reduction in smoking prevalence would have on IHD mortality rates 20 years later. This produced a result of 0.9 for both males and females, indicating a strong relationship between the decline in smoking levels and mortality rates when a 20 years time lag is considered.

Figure 8.13. Prevalence of adult cigarette smoking in Great Britain, 1974-99



Source: Adapted from data obtained from the General Household Survey.

This thesis has argued that, prior to 1975 medicine had little impact on smoking behaviour. However, the beginnings of health education were evident from the publication of the 1962 report of the Royal College of Physicians. Since the mid 1970s medicine has campaigned to reduce smoking levels through a number of different strategies. These include the anti-smoking campaigns in the 1980s which focused on highlighting the dangers of smoking and produced changes in awareness, knowledge and attitudes (Flay, 1987). As smoking continued, in spite of the widespread knowledge of the risk attached, health education began to develop more sophisticated campaigns to encourage cessation. An example of this in Scotland is the 1992 Smokeline campaign, which formed part of HEBS anti-smoking strategy. This campaign combined a mass media approach to inform the public, with a

telephone helpline offering personal support. A review of the effectiveness of this campaign found that it led to an acceleration of the underlying trend of reduction seen since 1975, with an estimated 5.9% of adult smokers in Scotland having called the helpline, and 1.4% of adult smokers believed to have given up smoking as a result (Platt *et al*, 1997). Mass media interventions for health education have been used to target specific groups, such as young people. Such campaigns have been found to be effective in preventing the uptake of smoking (Sowden and Arblaster, 2004).

Prevention strategies have also been employed at community level to encourage smoking cessation in specific geographical locations. These include the promotion of smoking cessation groups and nicotine replacement therapy. The evidence for the success of these schemes is not convincing; however, they are still recommended as an important part of health promotion (Secker-Walker *et al*, 2004). The other area where prevention strategies have been implemented is in the work place. Prior to the 1990s workplace smoking polices were mainly aimed at reducing the risk of fires. One of the main components of workplace strategies has been smoking bans, and these have been effective in reducing consumption during the working day, although there is no evidence on their overall effect on smoking prevalence (Moher *et al*, 2004). A ban on smoking in public places (including workplaces) is due to become compulsory in Scotland from March 2006.

Medical findings have also influenced government measures aimed at reducing smoking. This began with increased taxation on cigarettes. By the early 1980s, cigarettes were cheaper in real terms than they had been in 1960 (Royal College of Physician, 1983). Tax was increased by 14p in 1981, a policy which has continued, and has been shown to be effective in reducing smoking prevalence (Townsend *et al*, 1994).

In addition, attempts have been made to reduce smoking at the level of the individual. This has included personal advice from physicians to patients on how to stop smoking. This has had a small but significant effect (Silagy and Stead, 2004).

Thus, since the 1980s, medicine, through both health education strategies and government measures, was effective in influencing smoking prevalence. However, smoking has been in decline since the mid 1970s (see figure 8.13) when there were no specific strategies in place. Instead, the main source of public knowledge on the dangers of smoking came from media coverage of medical findings. The extent to which these measures were responsible for the post-1975 fall in smoking can be estimated by considering what may have happened to smoking prevalence in the absence of medical warning of the dangers. Pre-1975, smoking

prevalence had increased throughout the century. Amongst men this appeared to plateau after World War Two, with over 50% of men smoking; whilst the female rate, although lagging behind that of men, was increasing (see figure 8.7). Before the first health warning on the dangers of cigarette smoking, no sustained decline in smoking can be detected. It was only after the dangers of smoking first became evident, initially through the reporting of medical findings and then through prevention strategies, that any decline is apparent. It is therefore likely that, in the absence of this warning, male consumption would have remained high, whilst female rates would have continued their upward trend, resulting in an even greater burden of IHD.

- **Blood pressure**

Capewell *et al* (1999) have calculated that 9% of the decline in IHD mortality can be attributed to the treatment of hypertension; this is clearly a medical achievement. However, they have also attributed 6% of the decline to a fall in population blood pressure levels. Factors which have influenced this decline will be discussed in the section dealing with stroke (see section 8.3.5.2).

- **Lack of physical activity**

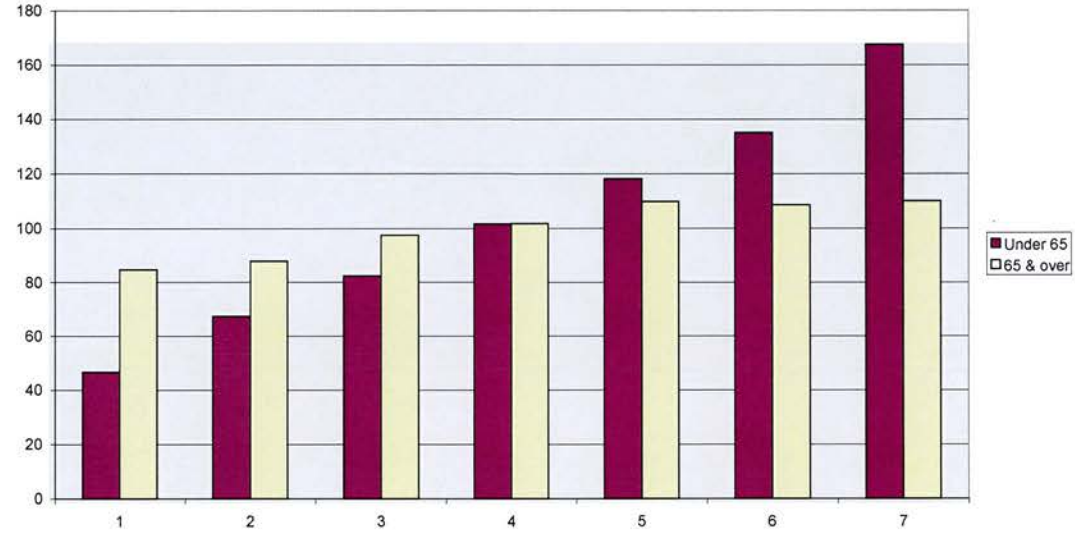
Physical activity was not considered by Capewell *at al* in their study of factors influencing the decline of mortality, although it is recognised as a major risk factor for IHD. If the decline in physical activity pre-1975 was instrumental in the rise of IHD death rates, then any influence which this factor may have had on the decline must also be considered. For physical activity to have influenced the fall in IHD mortality evidence must be produced that levels of activity have increased post-1975. This evidence on overall levels of physical activity is not available from 1975. However, by the mid 1990s it was estimated that over a third of the UK population was inactive or sedentary (Wimbush *et al*, 1998). Recognition of these high levels of inactivity, and the risk of IHD associated with them, has led to the development of strategies to increase activity. In Scotland this led to the launch of a mass media campaign to promote the health benefits of walking in 1995. This campaign was successful in improving adult awareness of walking as a good form of exercise; however, it did not produce any detectable change in adult walking behaviour (Wimbush *et al*, 1998). As well as medically inspired motives to improve physical activity, exercise has come to be regarded as a leisure activity. This is reflected in the growth of gyms and other facilities. However, as it is hard to assess the extent to which physical activity levels may have influenced IHD, it is difficult to attribute any possible change to medical or social factors.

Campaigns, such as the HEBS walking campaign, were successful in increasing awareness of the benefits of exercise, and may in the future produce a detectable impact on behaviour.

- **Poverty**

Capewell *et al* (1999) calculated that the decline in poverty resulted in a 3% fall in IHD mortality. When the role of poverty in the pre-1975 era is considered this at first appears a contradictory statement, given IHD's earlier association with affluence. However, differences in mortality between the social classes had been narrowing since the 1960s, as the death rates of the most affluent remained stable, whist those in more deprived groups continued to rise. When mortality began to decline, it did so first and fastest amongst the most affluent. By the 1990s the burden of IHD mortality lay with the poorest in society (figure 8.14).

Figure 8.14. Ischemic heart disease standardised mortality ratios (SMR) by deprivation catogory, 1994-98 (1 = less deprived, 7 = most deprived)



Source: Scottish Health Statistics (2000).

In order to comment on the mortality reduction associated with declining poverty, it is first necessary to discuss why, since 1975 IHD has become so strongly associated with it. One reason is the concentration of IHD risk factors in the most deprived groups. Consumption of saturated fat is higher amongst those in manual compared to non manual occupations, with

men in manual occupations consuming 41.7 grammes daily compared to 38.1 grammes amongst non-manual workers (the figures for women are 36.5 and 33.6, respectively) (Bolton-Smith *et al*, 1991). The prevalence of smoking by social class is shown in tables 8.1 and 8.2. These reveal that over time smoking has declined for most, but with a relatively greater decline for those in non-manual occupations. The exception to this is women in semi- and unskilled manual occupations in Scotland, of whom 51% and 59% smoked in 1998. The relationship between smoking prevalence and poverty (especially amongst women) has been investigated. The knowledge that smoking is harmful is almost universally known, however, it has been argued that giving up is more difficult for those living in poverty (Graham, 1994, Graham and Der, 1999). Smoking amongst the most deprived has been identified as both a necessity and luxury which helps the individual to deal with the stress of poverty. A lack of immediate reward after giving up smoking has also been identified. Similar socio-economic inequality is apparent in blood pressure levels (figure 8.15).

Table 8.1. Prevalence of male smokers aged over 16 in UK, by socio-economic group, 1976-1998

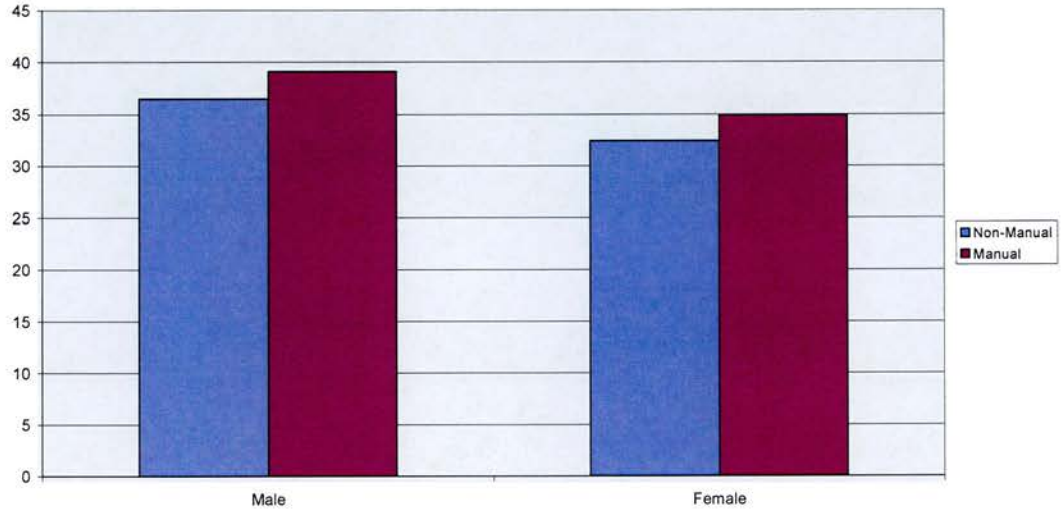
	1976	1986	1992	1994	1996	1998	1998 (Scotland only)
Professional	25	18	14	16	12	16	14
Employer/ manager	38	28	23	20	20	22	27
Intermediate/ junior	40	28	25	24	24	25	36
Skilled manual	51	40	34	33	32	34	40
Semi-skilled manual	53	43	39	38	41	40	53
Unskilled manual	58	43	42	40	41	44	48

Table 7. 5. Prevalence of female smokers aged over 16 in UK, by socio-economic group, 1976-1998

	1976	1986	1992	1994	1996	1998	1998 (Scotland only)
Professional	28	19	13	12	11	14	9
Employer/ manager	35	27	21	20	18	21	26
Intermediate/ junior	36	27	27	23	28	24	29
Skilled manual	42	36	31	29	30	30	36
Semi-skilled manual	41	35	35	32	36	33	51
Unskilled manual	38	33	35	34	36	32	59

Source: Office of National Statistics (2000).

Figure 8.15. Percentage of the population aged over 16 with high blood pressure in 1998 (England)



Source: Joint Health Surveys Unit (1999).

Thus, it is apparent that the prevalence of the main IHD risk factors is greatest amongst the poorest. However, these risk factors do not account for all the excess mortality amongst the deprived, and once they are controlled for poverty still remains as an independent risk factor (Rose and Marmot, 1981). The question therefore remains: why do the poorest have the highest prevalence rates for IHD risk factors, and why is poverty itself strongly and independently associated with IHD? Several explanations have been advanced to explain

this relationship (see box 8.4). This section will therefore consider, why in the late 20th century the poorest suffered disproportionately.

Levels of income inequality have varied throughout the 20th century and Wilkinson (1989) has argued that this has had a corresponding effect on disease incidence and mortality. He first considered the pre-World War Two period when mortality differentials between the social classes were narrowing, in spite of the depression and mass unemployment. He accounts for this by highlighting both the increase in the numbers eligible for unemployment benefit, and the increase in the value of the benefit. This meant that although the proportion of those out of work was high, there was now an infrastructure in place which would have prevented the extremes of poverty seen in earlier times. The intervention of World War Two, as has been discussed, led to a further diminishing of income differentials, as incomes and the market were increasingly controlled.

Box 8.4. Interpretations of relationship of poverty and health

Individual Poverty Health and mortality are determined by an individual's income.
Psychosocial Poverty In societies with wide inequalities in wealth, an individual's position in the social hierarchy determines health and mortality. In this interpretation individual income is important as it determines social rank, with the most deprived developing feeling of inferiority, which are translated into negative health consequences.
Neo – materialistic Poverty Health and mortality are influenced by individual resources and levels in investment in infrastructure. In this interpretation the dangers associated with lack of personnel income can be avoided if sufficient funds are invested public services to allow equality of access to areas, such as health care, education, transport, education and housing.

Source: Lynch *et al* (2000).

Post-World War Two there was a reversal of this trend, as the gap between the richest and the poorest widened. Although improvements were seen in all sectors of society, relative poverty increased, with 30% of the population living in relative poverty (judged by the

standard of supplementary benefit) in 1983, compared to only 8% in the early 1950s. The result of this was that, although mortality improved for all, the improvement was relatively greater amongst the most affluent. Evidence from the 1990s reveals that this trend has continued, with mortality rates for 1991-1992 162% higher amongst the most deprived compared to the most affluent (McLoone and Boddy, 1994). In the case of IHD it was found that mortality rates had improved in all classes, but had declined twice as fast amongst the most affluent compared to the most deprived. This increase in relative poverty has also been linked with a direct biological link between poverty and IHD. Wilkinson and Marmot (2001) have argued that psychosocial poverty can lead to lack of control over life, insecurity, anxiety, social isolation, bullying and depression. These can lead to stress on the neuroendocrine system which can increase risk of developing IHD (McEwan, 1998).

The result of this widening gap has been the concentration of IHD mortality amongst the poorest. If mortality had declined at the same rate in all classes, the overall decline in IHD mortality would have been far greater. However, low standards of living amongst sectors of society prevented larger falls in IHD. It is now recognised that health education is least effective amongst most deprived groups (Townsend *et al*, 1994), and attempts have been made to target health promotion at these groups. An example of this is the HEBS walking campaign which aimed to tackle the problem of low levels of leisure time exercise amongst the most deprived. Awareness of this campaign was highest amongst its target group. However, there was no corresponding change in behaviour (Wimbush *et al*, 1998). Lynch *et al* (2000) offer an alternative solution: strategic investment to redistribute public and private resources, in effect re-creating the conditions of World War Two when IHD rates were in decline. In the time period covered by this study neither of these policies was in effect and although the overall decline in poverty may have resulted in a 3% decline in mortality, the relative importance of poverty as a risk factor increased.

8.2.7. Conclusion

IHD is a disease which dominated mortality in Scotland throughout the 20th century. In the earlier years of the century mortality was greatest amongst the most affluent classes, as they received the most exposure to the disease risk factors. Industrialisation increased the overall risk in the population through the mechanisation of cigarette production, and reduction in physical activity, whilst the associated rising real wages increased the affordability of products which were previously luxuries, such as red meat. As the risk factors for the disease were either unknown or unpublicised, these changes were seen as desirable, and the prevalence of risk factors and mortality remained high. Medicine, meanwhile, could offer little hope of improved survival to those who were already ill.

In the 1970s the IHD death rates began to decline consistently. Capewell *et al* (1999) have attributed 32% of this decline to the medical treatment of IHD. This study agrees with Capewell *et al* 's finding that medicine was important in reducing mortality by improving survival. However, it has been argued that this benefit dates from the mid 1980s. Until this time medical treatments had been improving, but it was from 1980s that the treatments which make up the core of medical treatment of IHD, such as thrombolytics, ACE inhibitors and statins, became available. Since 1994, when Capewell *et al* 's study finishes, the use of these treatments has increased, resulting in a further improvement in survival, which is evident in the acceleration of the decline in deaths rates seen during the 1990s (see figure 8.1). Under the definitions of influences devised for this thesis these interventions fall into group 1 (medical).

The main influence on the decline of mortality before the 1980s was the decline in prevalence of the main IHD risk factors, to which Capewell *et al* have attributed 60% of the decline in mortality. The role of medicine in this area is difficult to assess. Its central role was to devise strategies for prevention. This requires a movement away from the traditional approach to prevention used against the infections (environmental control). Rather, the prevention of IHD required the development of health education to encourage individuals to change their behaviour. In addition, medicine also had a wider influence through the reporting of its findings in the media, the introduction of healthy eating into food marketing and production, and the development of government legislation and regulations. In attempting to change behaviour, medicine had moved into the area previously determined by standards of living. For example, the main consideration when buying food had been affordability or taste; to these health concerns were now added. These influences on

mortality have also been categorised as medical by this thesis, falling into group 2 (medically influenced).

The one area where medicine has not influenced mortality, and whose relative importance as a risk factor has increased, is poverty. It has been argued that in the first half of the 20th century IHD was regarded as a disease of affluence. This changed in the latter half of the century when the disease became associated with poverty. Although all sectors of society have seen improvements in their IHD mortality rates, the improvement has been greatest amongst the most affluent, whilst the position of the poorest has relatively worsened. If mortality rates had improved equally across the population, the overall decline in IHD mortality would have been greater.

Although Scotland's IHD mortality rate has been in decline, one area which must be addressed is the relative worsening of its relative position in international terms. This is due to the fact that mortality in other nations declined sooner and more steeply. One possible explanation for this is the delay in introducing preventive strategies until the 1980s. In other nations, steps had been taken to reduce risk factor prevalence at an earlier date, such as the N. Karelia project in Finland (Vartiainen *et al*, 1994), resulting in the earlier fall in mortality.

Despite the relative worsening of IHD rates amongst the poorest and Scotland's position internationally, the main trend in mortality since the 1970s has been downward. Although standards of living were the main determinant in the earlier rise in incidence and mortality, they played little part in the later decline. The earlier pattern of risk factor prevalence was examined and revealed no indication of risk factor reduction until medical advice became available. Since the 1980s, the role of medicine in the decline of IHD increased with the introduction of a comprehensive range of treatments aimed to improve survival after IHD has been established.

8.3. Stroke

8.3.1. Introduction

Stroke is the third largest cause of death in the developed world, exceeded only by IHD and deaths from cancer (Lip *et al*, 2002b). However, between 1950 and 1999 it was also the second largest contributor to the decline of mortality in Scotland, accounting for 18% of male and 20% of female lives saved. In addition, the total share of mortality attributable to stroke declined over this time period from 11% of all male, and 16% of all female, deaths, to 8.7% and 13.5%, respectively. This reduction in mortality is reflected in the declining death rate seen in figure 8.16.

Figure 8.16. Stroke death rates, per 1000, standardised to the 1950 population, 1950-99

Source: Original analysis of data obtained from the GROS.

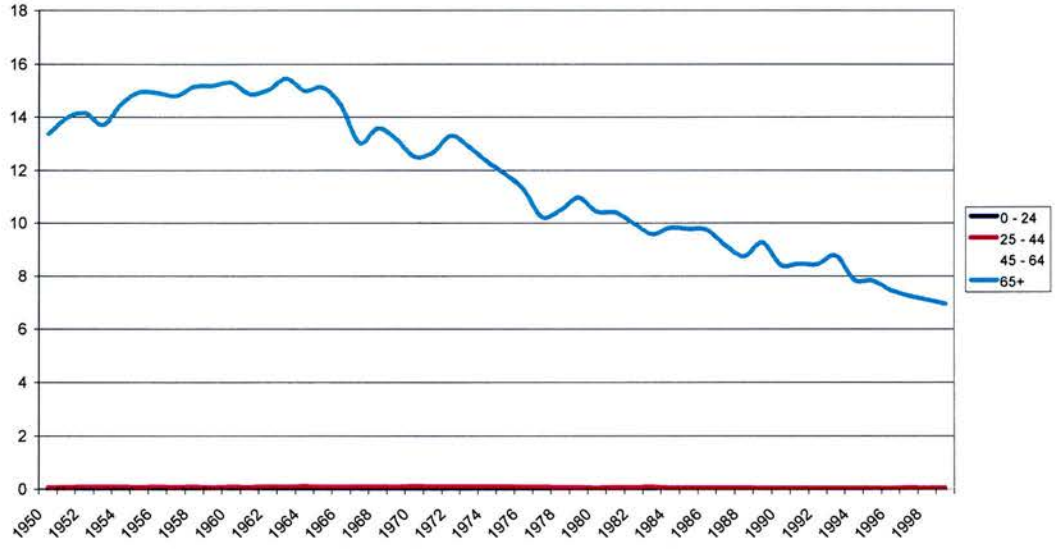
This section will consider the relative contribution of medicine and standards of living to the decline in stroke mortality. This will be achieved by drawing on the findings from the previous section on IHD in areas where an overlap is found. Thus, the first part of this section will consider pathology and aetiology of stroke to determine the applicability of the findings to IHD to stroke. The factors which influenced the decline of stroke will then be discussed.

8.3.2. Background

8.3.2.1. Age groups affected

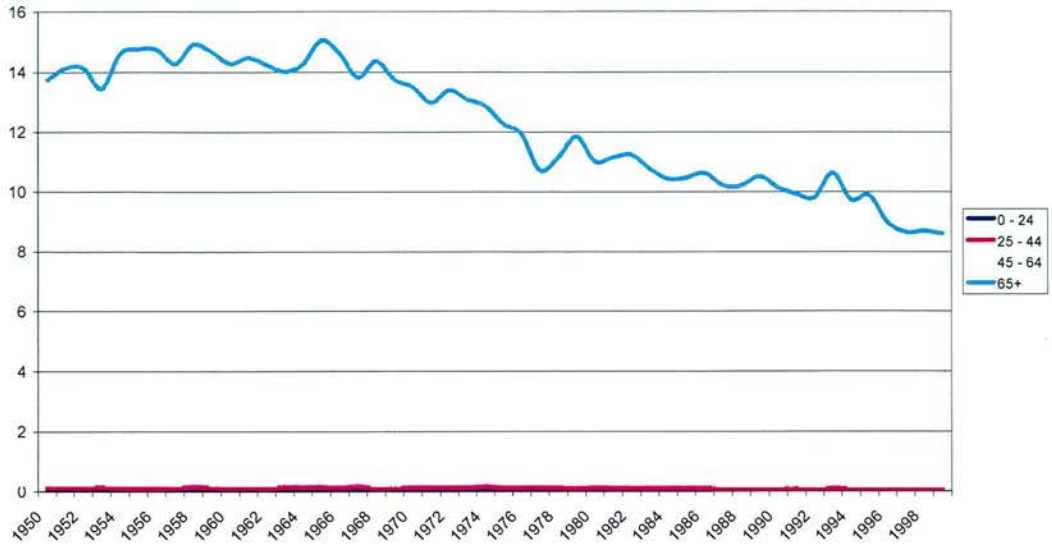
Trends in the stroke death rate by age group are displayed in figure 8.17 and 8.18. In common with IHD, the death rate from stroke is highest amongst the over 65s, whilst the death rate amongst the under 45s is so low is barely registers on the graphs. However, unlike IHD, the death rate in both the 45-64 age group and the over 65s is not significantly higher amongst males than among females.

Figure 8.17. Male stroke death rates, per 1000, by age group, 1950-00



Source: Original analysis of data obtained from the GROS.

Figure 8.18. Female stroke death rates, per 1000, by age group, 1950-99



Source: Original analysis of data obtained from the GROS.

8.3.2.2. Clinical description

A stroke occurs when the blood supply to the brain is disturbed; this leads to the affected part of the brain dying. However, unlike IHD, this disturbance is not the result of one cause, that is the blocking of one of the heart arteries depriving it of blood. Stroke is instead made up of two different syndromes (described below).

Ischemic stroke

This type of stroke is the brain equivalent of a heart attack or MI, and both have similar pathologies (Lawlor *et al*, 2002). The blood supply is curtailed by a blockage in an artery. In ischemic stroke this leads to the death of the part of the brain which is affected. As with IHD, the occlusion of the artery is caused by a combination of atheroma on the walls of the artery, and an excess of fibrinogen in the blood, which increases the risk of clots forming. (In the literature this type of stroke is also known as a cerebral infarct, but will be referred to as an ischemic stroke in this thesis). In the late 20th century approximately 81% to 85% of all strokes were of this type (Bamford, 1990, Bath and Lees, 2000).

Haemorrhagic stroke

This type of stroke is also known as a cerebral haemorrhage. Injury to the brain is not caused by blood deprivation as a result of the blocking of an artery. Rather, in haemorrhagic stroke a blood vessel in the brain bursts, which results in bleeding which damages brain

tissue. Haemorrhagic stroke is made up of two subtypes, intracerebral haemorrhage (ICH) and subarachnoid haemorrhage (SAH). In the late 20th century approximately 15% of all stroke was haemorrhagic, with ICH being twice as common as SAH (Bamford, 1990, Bath and Lees, 2000).

8.3.2.3. Signs and symptoms

Although ischemic and haemorrhagic stroke are the result of two different types of disturbance in the brain, they are not easy to distinguish clinically as they produce similar symptoms (Lawlor *et al*, 2002). Stroke can produce a wide variety of symptoms which may or may not be present depending on the part of the brain which has been affected. These include facial weakness, weakness down one side, sensory loss on one side, speech and communication difficulties, nausea and vomiting and lack of coordination and unsteadiness (Bath and Lees, 2000, Muir, 2001). The only reliable ways to diagnosis stroke subtype are by brain imaging using a CT scanner, or by autopsy (Bath and Lees, 2000).

8.3.2.4. Risk factors

The risk factors for stroke were not identified at the same time as those for IHD as there was initially some confusion over the relative contribution of the traditional cardiovascular disease risk factors (described in box 8.1) to the prevalence of stroke (Lawlor *et al*, 2002). Much of this confusion arose as it was not recognised that many of the risk factors operated differently according to stroke subtypes.

Ischemic stroke risk factors

Ischemic stroke is effectively the same type of disease as IHD, and as such this stroke subtype shares many of the same risk factors as IHD (see box 8.1). These are risk factors which cause the arteries to narrow, or the risk of blood clotting to increase. However, the relative importance of the risk factors differs slightly between the two conditions, with high blood pressure being more significant in ischemic stroke. Smoking and high cholesterol have also been identified as risk factors (Vartiainen *et al*, 1995). When considering the impact of changing risk factor prevalence on disease incidence, this section will focus on these three main risk factors.

Haemorrhagic stroke risk factors

Research into the risk factors for haemorrhagic stroke has shown that this subtype shares some risk factors in common with ischemic stroke. High blood pressure has been shown to be a more significant risk factor in haemorrhagic stroke than in either IHD or ischemic stroke (Song *et al*, 2004). Smoking has not been identified as a major risk factor in haemorrhagic stroke and high cholesterol has been shown to reduce risk (Hart *et al,* 2000). Family history was not found to be significant in ischemic stroke, but was significant for haemorrhagic, with the strongest association being with SAH (Kubota *et al*, 1997).

8.3.3. Mortality trends

8.3.3.1. Data reliability

Significant problems exist with the reliability of death data for stroke. In Cameron and McGoogan's (1981b) study of death certificate reliability, it was found that only 22% of clinically diagnosed stroke was confirmed at autopsy. These findings meant that the results which were generated from the Registrar General's data were initially treated with a degree of caution. Reasons for this lack of accuracy and possible ways in which this can be overcome are discussed below.

8.3.3.2. Pattern of decline

The stroke death rate in Scotland has been in decline over the period 1950 to 1999, from 1.5 per 1000 amongst males, and 1.9 per 1000 amongst females, to 0.6 and 0.7, respectively. Figure 8.16 reveals that both male and female rates stayed relatively constant until the 1960s when a sustained period of decline began. Data from England and Wales reveal that the constant rates of the 1950s are actually a plateau when seen in the context of an earlier period of decline dating from the beginning of the 20th century, with this earlier pattern of decline then being re-established in the early 1960s (Charlton and Murphy, 1997). In the case of IHD, death rates increased from at least 1930, to reach a peak in the 1970s, before a pattern of decline was established (figure 8.6). This 'epidemic' of disease did not occur in stroke; instead, the dominant trend throughout the 20th century was one of decline. This result is problematic for this thesis, as the analysis of the factors influencing the decline of stroke was originally intended to draw heavily on those found for IHD. However, if both diseases experienced a different pattern of mortality throughout the 20th century, this suggests that different factors were acting on each disease. One possible explanation is that

the death rates for stroke discussed so far refer to total stroke and not to different subtypes of stroke. It has been argued in this thesis that both ischemic stroke and IHD share many of the same risk factors. However, haemorrhagic stroke does not and so would not be expected to follow a similar pattern to IHD. Therefore, it is necessary to consider the pattern of mortality experienced by both stroke subtypes to get an accurate picture of what happened to stroke mortality. In order to achieve this, the Registrar General's Death Returns were consulted to identify what proportion of total stroke mortality was attributable to each stroke subtype. The results of this, for sample years, are shown in table 8.3. This table shows that it is not possible to accurately separate each stroke subtype using the Registrar General's Death Returns. The percentage of total stroke attributed to both ischemic and haemorrhagic stroke declined over the time period, whilst the percentage listed as undefined increased. This growth in undefined stroke reflects growing recognition of the difficulty in distinguishing the two subtypes in the absence of modern diagnostic scanning or autopsy, and means that the death returns cannot be used to analyse the pattern of mortality by subtype.

Table 8.3. Percentage of all stroke deaths listed in Registrar General's Death Returns, for sample years, according to subtype

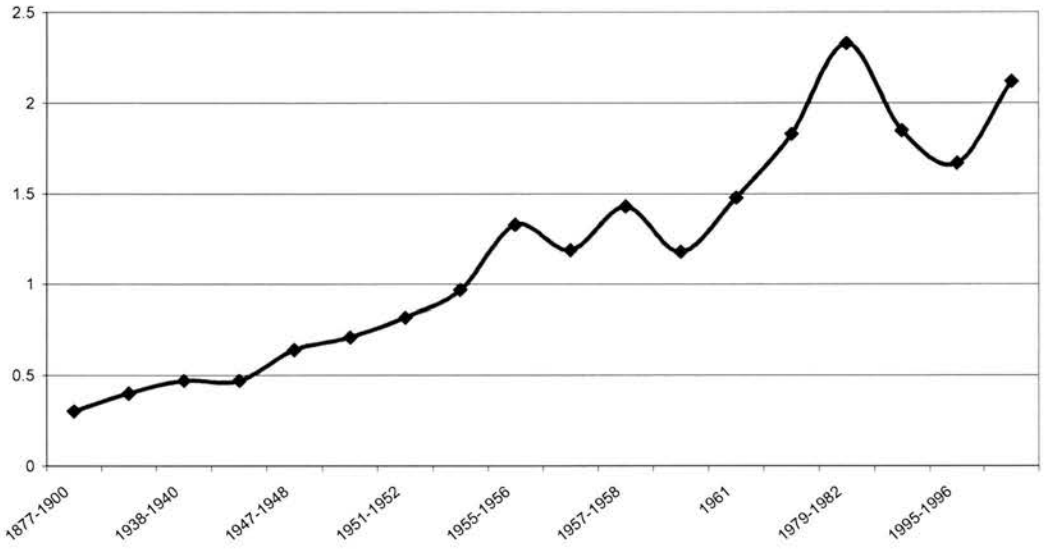
	1950		1975		1999	
	Male	Female	Male	Female	Male	Female
Haemorrhagic stroke	51	55	20	19	16	13
Ischemic stroke	45	41	34	36	10	8
Undefined stroke	4	4	45	45	74	79

Source: Original analysis of records obtained from the GROS.

The only accurate method of diagnosing stroke subtype over the entire of the time period covered by this thesis was autopsy. Lawlor *et al* (2002) have attempted to reconstruct the mortality trends of each stroke subtype by consulting this source. They acquired autopsy records from a number of English hospitals covering the period 1877 to 1999 and calculated the ratio of ischemic stroke to haemorrhagic (see figure 8.19). While the vast majority of stroke in the late 20th century was ischemic, Lawlor *et al* 's analysis of autopsy results

reveals that this is reversal of the situation at the start of the 20th century. Between the 1930s and the 1990s there was a fourfold increase in the ratio of ischemic to haemorrhagic stroke. This was the result of a combination of a decreasing number of deaths from haemorrhagic stroke, and an increase in the number deaths from ischemic stroke. Using these results, Lawlor *et al* (2002) plotted two different mortality trends for each stroke subtype. In the case of ischemic stroke they identified a pattern very similar to that seen for IHD in figure 8.6; that is, an increase in the death rate to a peak in the 1970s, after which the death rate fell. However, for haemorrhagic stroke a different pattern was identified as the death rate fell constantly from at least the start of the 20th century. This pattern has been reported in other studies, which have compared the pattern of decline of haemorrhagic stroke to that seen for stomach cancer (Davey Smith *et al*, 1998, Leon and Davey Smith, 2000).

Figure 8.19. Ratio of ischemic to haemorrhagic stroke, 1877-1999*



*data are based on autopsy results from a sample of English hospitals.
Source: Adapted from Lawlor *et al* (2002).

Therefore, although mortality for total stroke had been in decline for much of the 20th century, this disguised the trends for the two different subtypes. The increase in ischemic stroke mortality up to the 1970s was offset by the decline in the death rate from haemorrhagic stroke. This thesis will consider the factors influencing the mortality from each subtype separately as the differing mortality trends indicate that each was subject to different influences. The following section will consider the relative contribution of medicine and standards of living to mortality from ischemic stroke. As ischemic stroke

shares many of the risk factors, and follows a similar pattern of mortality increase and decrease, as IHD this analysis will draw heavily on the finding for IHD. The factors influencing the decline of haemorrhagic stroke will then be considered.

8.3.4. Ischemic stroke

The first part of the discussion on the factors influencing mortality from ischemic stroke will consider the effect of cure rates. The role played by disease incidence will then be reviewed.

8.3.4.1. Survival

It has been argued that increasing IHD case fatality was not responsible for the increase in mortality rates seen up until the 1970s. It is therefore also unlikely that this was the case for ischemic stroke. This section will consider the degree to which increasing survival of patients already ill with ischemic stroke may have contributed to the decline in mortality reported since the 1970s. As death from ischemic stroke and IHD are both caused by blood deprivation as the result of a blockage in the artery, it would be anticipated that treatments for IHD would be applicable to ischemic stroke. In the case of IHD, Capewell *et al* (1999) estimated that 32% of the decline in mortality between 1975-1994 was the result of treatment, and this thesis has argued that in the period up to 1999 this figure may be greater as the number of effective treatments were being more widely employed. The extent to which medical treatments influenced ischemic stroke is discussed below.

8.3.4.1.1. Treatments for acute stroke

As with IHD, there are two ways to prevent death from ischemic stroke once the disease has been established. The first is to prevent the patient dying during the acute stage of the disease (primary prevention) and the second to prevent the recurrence of stroke once the acute phase has passed (secondary prevention). Before any treatment for acute ischemic stroke is undertaken, "*of paramount importance is correct clinical diagnosis*" (Muir, 2001:i12). This is because many of the treatments which can help in ischemic stroke are aimed at thinning the blood and improving blood flow and so can prove dangerous to those with haemorrhagic stroke as they increase the risk of further bleeding. Therefore treatment should not begin until stroke subtype has been distinguished (Bath and Less, 2000). Given the similarities in the symptoms of both subtypes, the only way in which a diagnosis can

reliably be made is by a CT scan and this should be undertaken before treatment in commenced.

Thrombolytic treatments

In the 1990s the treatment of IHD with thrombolysis significantly decreased the death rate in the first few hours following a MI. The treatment dissolves the blood clot causing the blockage and rapidly reinstates blood flow (see section 8.2.6.1.1). In trials of thrombolysis for ischemic stroke the treatment has been found to increase significantly the chance of complete recovery if begun within three hours (Bath and Lees, 2000). However, thrombolysis use in stroke has a severe complication as it increases the risk of intracranial bleeding by about four times (Warlow and Wardlaw, 2003). During the time period covered by this thesis thrombolysis, for use in the treatment of stroke, was not licensed for use in the UK, and so would not have contributed to the decline of ischemic stroke mortality. This treatment may offer the potential for increasing survival rates in the future.

Antiplatelet treatment

The only form of antiplatelet treatment which has been evaluated for acute ischemic stroke is aspirin (Lip *et al*, 2002b). If this treatment is started within 48 hours it reduces the risk of death and prevents re-blockage (Bath and Lees, 2000). In addition aspirin use has not been found to increase the risk of haemorrhagic stroke (Lip *et al*, 2002b).

Anticoagulant treatment

As this treatment is aimed at thinning the blood any benefit which is derived in the treatment of acute ischemic stroke is accompanied by an increased risk of major bleeding (Bath and Lees, 2000, Lip *et al*, 2002b).

Neurosurgery

Surgery for ischemic stroke to remove the blood clot is not common. It is only undertaken if the pressure placed on the brain is likely to prove fatal.

Patient care by specialist nurses and stroke units

Complications can be problematic following ischemic stroke. These can include difficulties in swallowing, which can lead to pneumonia, and the development of deep vein thrombosis (Bath and Lees, 2000). Specialist care to avoid or cope with these complications can help to reduce case fatality rates (Muir, 2001).

8.3.4.1.2. Secondary prevention post ischemic stroke

In the secondary prevention of ischemic strokes, the dangers of wrongly treating a haemorrhagic stroke are not present as diagnosis is more certain. Therefore, many of the treatments which are used post-MI are appropriate to ischemic stroke prevention.

Aspirin, warfarin and ACE inhibitors

Warfarin has been found to significantly reduce the risk of recurrent stroke from around 12% to 5% (Muir, 2001). Aspirin is also recommended for the prevention of recurrent ischemic stroke. ACE Inhibitors can be used in the secondary prevention of ischemic stroke and have been found to significantly reduce risk (Muir, 2001). ACE Inhibitors, however, were only available from the early 1990s (see section 8.2.6.1.1).

Cholesterol lowering drugs

Prevastatin has been found to reduce the risk of stroke (Muir, 2001). Simvastatin, which proved highly effective in cholesterol lowering in cases of IHD, was not licensed for stroke prevention in the time period covered by this study.

8.3.4.1.3. Ischemic stroke survival conclusions

The treatment of ischemic stroke would therefore appear to offer a more complex challenge than that for IHD. The dangers associated with wrongly treating a case of haemorrhagic stroke with treatments designed to treat ischemia have mean that many of the most effective clot busting, and blood thinning drugs cannot be used. This thesis has argued that since the mid 1980s the new treatments for IHD, such as thrombolytics and ACE Inhibitors, have helped to reduce case fatality significantly if delivered to the patient soon after acute MI. Although thrombolytics have been shown in trials to be effective against ischemic stroke, this treatment is still not available in the UK, although this may change in the future for cases where diagnosis is certain. The treatments for the secondary prevention of ischemic stroke have more in common with those seen for IHD, and so this area of treatment may be similarly effective. This disparity between the treatment of IHD and ischemic stroke has been recognised by doctors treating stroke who have claimed that "*the heart attack doctors are not just in the lead, they are out of sight*" (Warlow and Wardlaw, 2003: 234). Thus, although this thesis has agreed with Capewell *et al* 's (1999) assessment that medical treatments were responsible for 32% of the decline of IHD between 1975 and 1994, it seems probable that medicine's contribution to the decline of ischemic stroke by improving survival was smaller.

8.3.4.2. Incidence of ischemic stroke

It has been argued that mortality from ischemic stroke followed a similar pattern to that of IHD and that both diseases share many of the same risk factors. This section will consider the applicability of the findings from IHD to the three main risk factors for ischemic stroke: high cholesterol, smoking and high blood pressure.

- **Cholesterol**

Research has shown a positive relationship between high cholesterol and the risk of ischemic stroke (Hart *et al*, 2000, Arieson *et al*, 2003). Therefore, it is probable that ischemic stroke would have been influenced by the same trends in fat consumption linked in section 8.2.5.2 to mortality from IHD. In the case of IHD, it was argued that increasing fat consumption dating from the late 19th century was partially responsible for the increase in IHD mortality up until the 1970s. Improving standard of livings, through increasing the affordability of higher fat products, was identified as the main reason for this. The decline in cholesterol levels seen in Scotland since the 1970s was attributed to the diffusion of medical research on the dangers associated with a high fat diet by a number of different means. These included the reporting of medical findings via the media, marketing of low fat products by the food industry and health education. Statin drugs for the treatment of those already suffering from high cholesterol were also identified as important in the reduction of IHD deaths.

- **Smoking**

Smoking has been identified as a risk factor shared by both IHD and ischemic stroke. In the case of IHD the rise of smoking was associated with the increase in mortality seen until the 1970s, and this was ascribed to rising standards of living which increased the affordability of cigarettes, and industrialisation which facilitated mass production. The decline in smoking prevalence was isolated by Capewell *et al* (1999) as the main contributor to the decline of IHD mortality between 1975 and 1994. This thesis has attributed the decline in smoking prevalence to medicine, as the findings of research on the dangers of smoking were communicated to the public, leading to behaviour change. Others initiatives to reduce smoking prevalence, including government regulation and legislation in areas such as taxation and provision of smoke-free areas, have also been viewed as medical as these measures would not have occurred in the absence of medical findings. Therefore, any reduction in ischemic stroke mortality due to the decline in smoking prevalence can be linked to medicine.

- **High blood pressure**

High blood pressure is a risk factor for both IHD and ischemic stroke; however, the association is stronger with haemorrhagic stroke. Therefore, the analysis of factors which may have influenced the relationship of high blood pressure with mortality from ischemic stroke will be discussed in the section considering haemorrhagic stroke.

8.3.4.2. Conclusions – ischemic stroke

IHD and ischemic stroke share a similar pathology and risk factors, and both experienced an increase in mortality up until the 1970s followed by a period of sustained decline. Mortality from both diseases would probably have been influenced by a similar set of circumstances. However, in the case of medical treatments to improve survival post ischemic stroke, this thesis has found that the range of treatments available was more limited than those for acute MI, which saw the introduction of several life saving treatments since the mid 1980s. Although in theory these treatments, aimed at dispersing blood clots and improving blood flow, are applicable to the treatment of acute ischemic stroke, the complications surrounding bleeding have meant that they were not licensed for use in the period covered by this study. Therefore, it is possible that declining case fatality as a result of improving medical treatments was a more significant contributor to the decline of IHD than to ischemic stroke. This would imply that the reduction in disease incidence may have been relatively more important in the decline of ischemic stroke. Ischemic stroke incidence is affected by the prevalence of acknowledged risk factors, and this thesis identified high cholesterol, smoking and high blood pressure as the main influences on ischemic stroke. In the case of high cholesterol and smoking, the decline in prevalence has been attributed to medicine by means of informing the public of the dangers associated with a high fat diet and smoking. As high blood pressure is a more significant risk factor for haemorrhagic stroke, it will be discussed in the following section, and the implications of this on ischemic stroke will be discussed in the overall conclusions for stroke.

8.3.5. Haemorrhagic stroke

The decline in the ischemic stroke death rate has been dated to the 1970s (Lawlor *et al*, 2002); however, mortality from all forms stroke has been in decline since the early 20th century (Charlton and Murphy, 1997). The most important influence on the decline of stroke before the 1970s was the reduction in mortality from haemorrhagic stroke, which has been dated to the early 20th century (Lawlor *et al*, 2002). This section will consider the relative contribution of medicine and standards of living to the decline of haemorrhagic stroke as a result of improving cure rates and declining incidence, and will trace the origins of this decline to the early years of the 20th century. Although ischemic stroke and haemorrhagic stroke are symptomatically similar, they have different causes and treatments. This means that haemorrhagic stroke is not influenced by many of the risk factors and treatments for IHD; and most of the findings for IHD are not applicable to haemorrhagic stroke.

8.3.5.1. Survival

The fatality of haemorrhagic stroke is determined by the severity of the initial bleed (Ariesen *et al*, 2003). Both ICH and SAH carry high case fatality rates at 30 days of 50% and 46% respectively, far in excess of that experienced by ischemic stroke at 10% (Bamford, 1990). A recent review of treatments for haemorrhagic stroke found that there are no good data on the treatment of haemorrhagic stroke (Muir, 2001). Another study indicated that treatments which do exist have limited success (Ariesen *et al*, 2003). In cases of haemorrhagic stroke all treatments by antiplatelet and anticoagulant drugs should be stopped to prevent further bleeding (Muir, 2001). The main medical intervention for haemorrhagic stroke in its acute stage is the provision of specialist care for sufferers to avoid complications. This includes the regulating of body temperature and blood glucose, and the prevention of infections and deep vein thrombosis. These measures have been found to deliver 'considerable benefit' to patients suffering from ICH (Muir, 2001). One possible surgical intervention for treatment of haemorrhagic stroke is shunting. This involves bypassing of the area of the bleed after a neurological examination (Muir, 2001); however, the benefit of this on mortality rates is not clear. There are no established guidelines for the secondary prevention of haemorrhagic stroke.

Given the lack of available treatments and of evidence of their effectiveness, it is unlikely that medical measures to cure individuals after haemorrhagic stroke would have had a significant effect on the decline of mortality. It is possible that improvements in the care of sufferers may have helped improve longer term survival by preventing the complications

associated with the condition; however, it is not possible to estimate the extent to which this may have reduced case fatality.

8.3.5.2. Incidence

As it appears unlikely that increasing rates of survival of those suffering haemorrhagic stroke had any effect on the overall decline in mortality, the most probable influence on the mortality decline was the decline in disease incidence. This section will consider the relationship of the main cardiovascular risk factors to haemorrhagic stroke, and the extent to which their prevalence may have influenced the decline of disease incidence. As haemorrhagic stroke has been in decline since the start of the 20th century, the discussion will consider risk factor prevalence dating from this time.

- **Cholesterol**

In common with IHD and ischemic stroke, cholesterol is a risk factor for haemorrhagic stroke. However, there is an inverse relationship between haemorrhagic stoke and cholesterol levels (Vartiainen *et al*, 1995, Hart *et al*, 2000, Ariesen *et al*, 2003): as cholesterol levels *increase*, the risk of developing haemorrhagic stroke *decreases*. The opposite effect of cholesterol levels on the two stroke subtypes may go some way to explaining the initial confusion when stroke risk factors were being identified. As higher cholesterol levels reduce the risk of haemorrhagic stroke, the increase in fat consumption in the 20th century up until the 1970s, which has been identified as a contributor to the increasing incidence of IHD and ischemic stroke, may have been partially responsible for the decrease in haemorrhagic stroke incidence rates.

- **Smoking**

Smoking has not been identified as a risk factor for haemorrhagic stroke (Ariesen *et al*, 2003). In the case of IHD and ischemic stroke, this risk factor was isolated as having a large effect on the increase in the incidence of both diseases. If smoking has no influence on haemorrhagic stroke this will help account for why this stroke subtype did not experience a similar surge in mortality during the first three quarters of the 20th century.

- **Blood pressure**

High blood pressure is a risk factor for IHD, ischemic stroke and haemorrhagic stroke. Consequently the findings from this section can be applied to each disease subtype.

However, the association of high blood pressure is stronger with ischemic stroke than IHD, and in the case of haemorrhagic stroke high blood pressure is the main risk factor for the disease (Eriksson *et al*, 2000, Song *et al*, 2004). Therefore, any decline in the prevalence of high blood pressure would have had a more significant effect on the decline of haemorrhagic stroke than on either IHD or ischemic stroke.

Routine data on population blood pressure levels are not available for the whole of the 20th century. However, a study based on Glasgow University students has indicated that levels have probably been in decline since the beginning of the 20th century. This study measured the blood pressure levels of successive students attending the university between 1948 and 1969, and found levels to be continually decreasing amongst cohorts born since at least 1928 (the first year covered by the study) (McCarron *et al*, 2001a). Thus, if blood pressure has been in decline since the early 20th century, and is the main risk factor for haemorrhagic stroke, this may in part explain the decreasing levels of mortality seen since this time. The remainder of this section will consider the relative contribution of medicine and standards of living to the decline of blood pressure in Scotland throughout the 20th century.

Medical measures to reduce blood pressure

Medical treatments to lower blood pressure were first developed in the 1950s (Charlton and Murphy, 1997). These were first available on a large scale from the 1970s (Vartiainen *et al*, 1995). Capewell *et al* (1999) attributed 9% of the decline in IHD mortality between 1975 and 1994 to the treatment of hypertension. As high blood pressure is more closely associated with stroke it can be argued that the contribution of drug regimes for the reduction of hypertension since the 1970s may have had a more significant influence on the decline of stroke. Thus, since the 1970s medicine has been able to offer an effective treatment for the regulation of high blood pressure, coinciding with the start of the decline in mortality from IHD and ischemic stroke. The relative contribution of medicine to blood pressure reduction is likely to have increased since the 1980s as from this time threshhold for defining hypertention was lowered, leading to a greater proportion of the population receiving treatment (Burt *et al*, 1995). However, it has been shown that blood pressure was declining in Scotland since at least 1928, long before any effective medical intervention for hypertension became available, and that it was this earlier decline in blood pressure which helped influence the decline of haemorrhagic stroke. The factors which may have influenced this initial decline in blood pressure will therefore be considered.

Early life influences

Lawlor *et al* (2002) have suggested that the earlier decline in mortality seen in haemorrhagic stroke "*might reflect a greater importance of early-life risk factors on the aetiology of cerebral haemorrhage*" (p1823). It has also been established that blood pressure in childhood continues through to adulthood (McCarron *et al*, 2001a) and that there are two main influences in early life, birth weight and growth in childhood, which affect this.

A number of studies have noted the relationship between birth weight and an individual's later risk of developing stroke. One study from Sweden examined the effect of prenatal growth amongst men and women born between 1915-1929. This found that "*impaired fetal (sic) growth is strongly associated with haemorrhagic stroke, but not with occlusive (ischemic) stroke*" (Hypponen *et al*, 2001: 1033). This inverse relationship between haemorrhagic stroke and birth weight was also noted by Lawlor *et al* (2002) and Eriksson *et al* (2000). In another study areas which experienced high maternal and neonatal mortality early in the 20th century were linked with high stroke death rates in the late 20th century (Davey Smith *et al*, 1998). It is believed that the relationship between low foetal birth weight and later risk of stroke arises because of impaired arterial growth which leads to elevated blood pressure throughout life. Thus, any factor which can help increase foetal growth can then influence an individual's later risk of developing haemorrhagic stroke. The main influence on foetal growth is the physical condition of the mother during pregnancy, which is determined in part by nutritional status; factors influencing this at the start of the 20th century are discussed below.

The second early life influence which affects later risk of developing high blood pressure is growth in childhood, reflected in adult height (Davey Smith *et al*, 1998). In a study reviewing risk factors for stroke subtypes in Renfrew/ Paisley, it was found that there was a stronger association between height and haemorrhagic stroke than ischemic stroke, with each 10cm increment in height resulting in almost a 30% reduction in risk (McCarron *et al*, 2001b). Height is an indicator of nutritional status as a child and the link between this and later levels of high blood pressure has been investigated using data from men who experienced the siege of Leningrad as children. The siege of Leningrad took place during the Second World War, during which the average daily ration was 300 calories per day. Boys who lived through the siege were later found to have a significant excess risk of high blood pressure, indicating the long-term outcome of poor nutrition in childhood (Sparen *et al*, 2004).

Both foetal weight and adult height are indicators of maternal nourishment (Eriksson *et al*, 2000) and of nourishment during childhood (Lawlor *et al*, 2002). Therefore any evidence of improving nutrition amongst mothers and children in the early years of the 20th century could be linked to the later reduced risk of developing high blood pressure and cardiovascular disease, in particular haemorrhagic stroke. It has already been shown in the discussion of factors influencing the decline of tuberculosis (see section 6.4.3.2) that nutrition had improved for many towards the end of the 19th century and earlier part of 20th century. This was seen as consequence of improving standards of living resulting from rising real wages. If nutrition had improved to a level where mortality from tuberculosis was declining, it is probable that the nutritional status of pregnant women and children would have improved to reduce later risk of high blood pressure associated with earlier malnourishment.

Salt

Excess salt consumption has been identified as a risk factor for high blood pressure (Ebrahim and Smith,1998). Evidence has also suggested that the effect of salt may be most damaging in infancy, leading to high blood pressure in later life (McCarron *et al*, 2001b). Any reduction in salt consumption, especially during infancy, should have an effect on stroke mortality. One of the main factors which influenced the decline in the incidence of stomach cancer was the reduction in the consumption of salted foods, in particular meats (see section 7.5.2.2). This was ascribed to the development of refrigeration dating from the 1880s which allowed salt to be replaced as means of preservation, and rising standards of living which improved the quality of diet. This decline in salt consumption would have also had the effect of reducing the risk of developing high blood pressure, especially for infants born at that time.

Medicine has also been influential in attempting to reduce levels of salt consumption in the post-1970s period. Population level strategies to publicise the dangers associated with excessive salt consumption have been found to result in small reductions in blood pressure which have produced substantial effects over the entire population (Hooper *et al*, 2004). However, concerns have been raised about the amount of salt which has entered the diet since the 1970s through the growth of ready prepared, processed foods, such as bread, ready meals and junk foods. It is estimated that 80% of salt in diet is derived from these sources (Consensus Action on Salt and Health, 2004). These may counteract much of the benefit derived from health education.

Blood pressure would appear to have been in decline throughout the 20th century. Before the 1970s the main influences on this decline were outside the sphere of medical influence. These were improvements in nutrition which improved both overall physical condition and reduced salt consumption, as a consequence of improving standards of living. Since the 1970s medicine has played a much more significant role, through both the direct treatment of hypertension and prevention as a result of health education concerning the dangers of excessive salt consumption.

Infection

One final influence on the decline of haemorrhagic stroke has been proposed. This is that the disease was linked to an as yet unidentified infection which has since declined (Davey Smith *et al*, 1998, Leon and Davey Smith, 2000). This theory has emerged out of research which has considered the origins of the decline of stomach cancer and haemorrhagic stroke, as well as number of other conditions. Similarities were noted in the pattern of decline of stomach cancer and haemorrhagic stroke, and both diseases were found to be strongly related to socio-economic conditions in childhood. In the case of stomach cancer this relationship with childhood circumstances has been shown to be the result of early infection with *H. pylori*, leading to the implication that haemorrhagic stroke may have a similar cause. However, as there is as yet no evidence linking haemorrhagic stroke to any infection, it is not possible to estimate its potential impact on death rates.

8.3.5.3. Conclusions – haemorrhagic stroke

Haemorrhagic stroke has been in decline in Scotland since the start of the 20th century (Lawlor *et al*, 2002). Given the lack of effective treatment for haemorrhagic stroke it is unlikely that medicine contributed significantly to the decline of this stroke subtype, although it is possible that improvements in the care of sufferers after the onset of illness may have helped reduce fatality from subsequent complications. The main influence on the decline of haemorrhagic stroke has probably been the decline of disease incidence dating from the start of the 20th century. This has been attributed to improving standards of living, which resulted in the improved nutrition of pregnant women and children, helping to ensure lower rates of blood pressure in adulthood. Improved nutrition, in the form of salt reduction, has also been linked to reduced blood pressure and the lower incidence of haemorrhagic stroke. One of the main reasons why incidence rates of haemorrhagic stroke did not increase

during the earlier part of the 20th century was that high cholesterol and smoking did not increase risk. The case of cholesterol is interesting as it would appear that the rising consumption of fat, which increased the risk for ischemic stroke, may have offered protection against haemorrhagic stroke.

8.3.6. Overall stroke conclusions

Mortality from total stroke has been in decline throughout the 20th century (Charlton and Murphy, 1997). However, analysis of the two subtypes of stroke has shown that they followed divergent mortality trends up until the 1970s, with ischemic stroke increasing whilst haemorrhagic stroke decreased, after which both subtypes experienced a decline. The main reason for these contradictory mortality trends were the different relationships of each subtype with the main cardiovascular disease risk factors. The increasing prevalence of smoking during the earlier part of the 20th century led to an increase in the incidence of ischemic stroke, but had little impact of haemorrhagic stroke rates. The increase in cholesterol up until the 1970s has also been identified as increasing the incidence of ischemic stroke; however, this trend may actually have contributed to the decline of haemorrhagic stroke. Blood pressure is the only risk factor, which affects both subtypes and which was in decline prior to the 1970s. This is the main risk factor for haemorrhagic stroke, and most of the decline of this subtype has been ascribed to the reduction of blood pressure as a result of improving standards of living, before the introduction of antihypertensive treatments in the 1970s. Although high blood pressure is also a risk factor for ischemic stroke, this subtype experienced no reduction in mortality in line with the reduction in blood pressure. One possible explanation for this is that any benefit derived from the reduction in blood pressure as a risk factor may have been cancelled out by the increase in both smoking and cholesterol levels.

Thus, up until the 1970s the main influence on the decline of total stroke was the reduction in the haemorrhagic subtype as a result of improving standards of living. The picture post-1970s is more complex as the decline of total stroke was due to the reduction of both ischemic and haemorrhagic stroke. It the case of haemorrhagic stroke medicine contributed little to the decline of the disease post-1970 as few treatments were available. Rather, declining incidence continued to be the main determinant of decline. As the medical treatment of ischemic stroke is not as advanced as that for IHD, it is unlikely that medical treatments to prevent death during the acute stage contributed as much to ischemic stroke.

Rather, a relatively larger proportion of the decline of ischemic stroke, than of IHD, may have been due to risk factor reduction as a result of the diffusion of medical findings.

The relative contribution of medicine and standards of living to the decline of total stroke depends largely on what proportion of the decline in mortality can be attributed to each stroke subtype. It has been shown that the Registrar General's Death Returns lack detailed data on stroke subtype, tending instead to enter a code of undefined stroke. Evidence uncovered by Lawlor *et al* (2002) has indicated that in the first part of the 20th century haemorrhagic stroke was four times more common than ischemic. They have also shown that ischemic stroke mortality did not begin to decline until the 1970s. Therefore, it can be argued that up until the 1970s the decline in haemorrhagic stroke mortality as a result of standards of living was the main influence on the decline of total stroke. However, by the end of the 20th century ischemic stroke was far more common than haemorrhagic, accounting for over 80% of total stroke. This suggests that the greatest potential for decline in the latter part of the 20th century came from ischemic stroke, and as such the main influence on mortality at this time was medicine.

8.4. Overall conclusions for cardiovascular disease

Taken together, the decline in mortality from IHD and stroke account for 56% of all male, and 69% of all female, potential lives saved in Scotland between 1950 and 1999. It has been argued in this thesis that the decline of IHD and ischemic stroke can be attributed to medicine. The influence of medicine on mortality decline can be divided between the two groups of medical influence which were defined in box 3.2. In the case of IHD at least 32% of the decline in mortality since 1975, and in particular since the mid 1980s, was ascribed to medical treatments (group 1 medical) for those already ill with the disease. Medical treatments probably contributed less to ischemic stroke, as fewer treatments were available for this condition in the time period covered by this thesis. Group 2 (medically influenced) was developed to encompass the wider influence of medicine on mortality decline, this involved the diffusion of medical findings on the main disease risk factors. A slightly higher proportion of the decline of ischemic stroke, than IHD, was probably due to the decline in risk factors.

When the decision was originally taken to include stroke as one of the diseases to be considered in this thesis, it was believed that the findings for IHD could be applied to the decline in stroke mortality. However, once the two different stroke subtypes were considered, it emerged that this was only true for ischemic stroke. Haemorrhagic stroke experienced a different pattern of decline, and was subject to a different set of influences. Haemorrhagic stroke was the only disease sub-type which experienced a decline as a result of non-medical influence (group 3). Certain by-products of improving standards of living, which increased the risk of IHD and ischemic stroke, either did not influence haemorrhagic stroke or may have contributed to its decline. Therefore, not all the decline of cardiovascular disease can be attributed to medicine, either through medical treatments or its influence on population behaviour. Instead, a small proportion of the decline was due to improving standards of living dating from the start of the 20th century.

9. Discussion and conclusion

9.1. Introduction

This thesis began with a discussion of Scotland's reputation as the 'sick man' of Europe, and data were provided which indicated that only Portugal had a higher all-cause death rate in Western Europe at the end of the 20th century (see figure 1.1). It has been argued that, starting from this unenviable situation, Scotland experienced a significant decline in mortality rates over the period 1950-1999. The principal aim of this thesis was to establish the relative contribution of medicine and standards of living to this mortality decline. It should be recalled that this thesis has only considered influences on mortality decline and has not attempted to measure any improvements which may have occurred in morbidity.

The main findings of the thesis will be considered in this chapter. First, the process of establishing the causes of death which contributed most to the decline in mortality, and the main influences on the decline of these causes of death, will be reviewed. Issues and problems related to the study of influences on mortality decline which emerged from this thesis will then be outlined. The relative contribution of medicine and standards of living to the mortality decline will be discussed. Finally, recommendations for achieving future mortality decline will be considered.

9.2. Causes of death which contributed the most to the post-1950 mortality decline

The data source employed, and the process of selecting the case study diseases were described in chapters four and five. From an original sample of 17 causes of death from the Registrar General's Death Returns, six causes of death were selected; their relative contribution to the decline in mortality was then calculated. Four causes of death which accounted for 70% of the decline in male mortality, and 78% of the decline in female mortality over the period 1950-1999, were then selected as case studies. The only cause of death which survived from McKeown's review of mortality decline pre-1971 was tuberculosis. This provided a direct link with the earlier study of mortality trends and allowed a comparison to be made between the findings of this thesis and those of McKeown on the pre-1950 mortality decline. As the focus of this thesis is on post-1950 mortality decline this also allowed an evaluation to be made of the influences on tuberculosis during a time when effective medical treatments were available. In the McKeown thesis the infectious diseases were credited with the majority of the mortality decline. However, when only the post-1950 period was considered, degenerative diseases dominated the mortality

decline. The inclusion of stomach cancer is noteworthy, as the main trend in cancer mortality was one of increase. As Scotland had the highest IHD death rate in Western Europe at the end of the 20th century it appeared paradoxical that this cause of death accounted for the largest share of the mortality decline between 1950 and 1999. However, focusing on this disease illustrates that, although Scotland may lag behind her neighbours in terms of overall mortality reduction, significant improvements have nevertheless been seen since 1950.

Before the overall contribution of medicine and standards of living to the mortality decline are considered, some of the issues and problems encountered in this thesis will be discussed. This is necessary as the approach taken to these issues will influence the final analysis of the relative roles of medicine and standards of living.

9.3. Issues and problems

9.3.1. Traditional concepts of medicine and standards of living are a false antithesis

One of the main challenges faced by this thesis was how to define what constitutes a 'medical influence' and an influence which was 'attributable to standards of living'. The necessity of developing categories into which specific influences could be placed was highlighted in the review of the literature on mortality decline (see chapter 2). Previous researchers on mortality decline were criticised for either using too broad or too narrow a definition of the different types of influence. The way in which each group is defined clearly determines the final conclusions on the relative contribution of medicine and standards of living. This thesis attempted to avoid some of the pitfalls experienced by earlier researchers by developing three categories of influence (see section 3.5). The first group was medical and included all influences on mortality which involved a personal interaction with the medical profession. The second group was also 'medical' but this group was intended to cover the influence of medicine on population behaviour and social conditions. The third group was 'standards of living', which included all influences which were not related to medicine.

Assigning of influences on mortality to group one was relatively straightforward, as these were mainly composed of clinical medical interventions, for example drug therapies, surgery or vaccination. The assigning of influences to groups two and three was more problematic.

Medical group two was developed in response to the criticism about the narrowness of McKeown's definition of medical interventions. Group two was intended to encompass those areas of medical influence which McKeown was accused of ignoring (public health) as well as the wider influence of medicine on population behaviour. However, the allocation of influences between groups two and three was difficult in relation to the latter part of the 20th century. This was especially true in the case of mortality reduction which was attributed to lifestyle change. This point can be illustrated by considering the influences on lifestyle choice in the early decades of the 20th century. Several influences on mortality during this time period which can be attributed to lifestyle were identified, for example the increase in meat consumption. In this instance mortality was directly influenced by an individual's standard of living, that is, if real wages rose, and the products became more affordable, then consumption increased. This thesis classed this type of influence as 'standards of living', as there was no evidence that medical knowledge informed the population that these lifestyle changes would improve health. However, as the 20th century progressed the separation of the influence of medicine and standards of living on lifestyle became increasingly complex, as decision making was no longer solely influenced by income, but also knowledge of what constitutes a healthy lifestyle. This is especially true of influences on the decline of IHD and ischemic stroke.

It is the overlap between of the role of medicine in informing the public of what constitutes a healthy lifestyle (via a number of different means) and the influence which standards of living had on lifestyle choices which caused the most difficulty in assigning influences to either group two (medical) or group three (standards of living).

This thesis attempted to identify the factors underlying lifestyle change by tracing trends in risk factor prevalence throughout the 20th century, to identify if changes in prevalence coincided with the diffusion of knowledge on risk, or if these changes occurred in the absence of medical knowledge. By the late 20th century the main influences on mortality were not environmental (i.e. sanitary conditions in the case of water-borne infections) but lifestyle related (i.e. diet and smoking). This meant that the traditional concepts of medicine and standards of living, as employed by McKeown, could no longer be applied. Rather, lifestyle was influenced by a diverse set of factors, including the diffusion of medical knowledge. This is not to say that standards of living no longer influence mortality. This is reflected in the way in which poverty can influence an individuals response to medical advice. Although the majority of the population are aware of what constitutes a healthy

lifestyle, the wealthier an individual the easier it is for them to follow that advice. Thus, the impact which medical advice has on risk factor reduction can be influenced by standard of living.

9.3.2. Consequences of improving standards of living can be positive or negative depending upon the disease

It has also become clear that within the same time period, the consequences of improving standards of living could be both positive *and* negative. This conclusion is especially pertinent when considering the changes which occurred in the earlier part of the 20th century which later influenced the mortality amongst cohorts born at this time. In the first decades of the 20th century the desirable consequences of an improving standards of living were perceived to be: an increase in the fat content of diet (a result, in part, of the increasing consumption of red meat), a decline in physical activity as work became less labour intensive, and access to tobacco. These are now viewed as risk factors for a number of different diseases. McKeown labelled the main killers of the late 20th century (i.e. IHD and lung cancer) the diseases of affluence. In these instances the health consequences of improving standards of living were detrimental. However, this thesis has found that the *increase* in some of these risk factors simultaneously influenced the *decline* in mortality from other causes. Increasing meat consumption has been linked to increasing cholesterol levels and in turn to the rise in mortality seen from IHD and ischemic stroke up until the 1970s. However, increase in fresh meat consumption was also linked to increased resistance to tuberculosis, as well as the decline in stomach cancer and haemorrhagic stroke as it replaced the previous reliance on preserved meats which were high in salt.

Improving standards of living have frequently been credited with helping to reduce mortality, for example the decline of infections in the McKeown thesis. However, the relationship of standards of living and health is multifaceted and should not always be perceived as positive. Rather, the consequences of improving standards of living must be considered within the context of the risk factors associated with specific diseases.

9.3.3. The concept of improving standards of living does not remain constant over time

Over time views changed as to what constituted a 'good standard of living'. What was considered a good standard of living in the earlier decades of the 20th century was not necessarily so at the end of the century. This can be illustrated by the association of a diet

high in fat, access to tobacco and limited physical activity, with a more affluent lifestyle during the earlier years of the century, whilst by the end of the century this lifestyle was associated with poverty. In this thesis the consequences of an improving standard of living have been considered within their historical context.

9.4. Relative contribution of medicine and standards of living to mortality decline in Scotland.

In chapter two the main theories on the influences on mortality decline were discussed. These fell into two broad camps: those which argued that medicine had contributed little to the mortality decline, and those which argued that the main influence on mortality decline was clinical medicine. Representative of these two standpoints were McKeown and Bunker, respectively. In his review of the influences on mortality decline in England and Wales between the 18th century and 1971, McKeown concluded that medicine had contributed little to the total decline, and that overall the main influence on mortality had been improving standards of living. McKeown used these findings to call for a new direction for medicine, which focused less on clinical cure, and looked more to disease prevention by concentrating on the origins of disease. Considering the influences on mortality decline in the USA from 1950, Bunker argued that there were no good data which would allow the contribution of improving standards of living to the mortality decline to be measured. Instead he calculated the increase in life expectancy which could be attributed to certain clinical medical interventions. In addition Bunker estimated that if everyone adopted the lifestyle of the fittest this would only lead to an overall gain in life expectancy of between 2 and 2.5 years. Based on this evidence Bunker recommended that "*increased investment in medical care would make the greatest and most predicable contribution to the reduction in death*" (Bunker, 2001: 1262).

McKeown and Bunker reached opposite conclusions and used their findings to justify differing recommendations for reducing mortality in the future. However, both these studies had drawbacks. In the case of McKeown, he used evidence from a time when few effective medical interventions were available, whilst Bunker did not consider the role which non-clinical factors may have played in mortality reduction.

This thesis has attempted to update the debate on mortality decline. In doing so it has considered what factors influenced the mortality decline post-1950, a time period when most

modern medical interventions first became available. In addition this thesis has attempted to measure the contribution of standards of living. The main influences on mortality decline attributable to the case study diseases are displayed in table 9.1. In this table the influences on mortality are divided according to the framework which was developed in chapter three and the main findings for each of these groups are discussed below. Then the main influences on the mortality decline will be considered.

Table 9.1. Influences on the decline of case study causes of death by group

	Tuberculosis	Stomach cancer	IHD	Ischemic stroke	Hemorrhagic stroke
Group 1. Medical – interventions conceived and delivered by health care professionals	Chemotherapy BCG MMR	Advances in diagnosis (gastronomy and OAG)	Primary prevention (resuscitation, beta blockers, ACE inhibitors, thrombolysis) Secondary prevention (aspirin, warfarin, cholesterol lowering drugs, CABG, angioplasty) Medical regulation of blood pressure	Primary prevention (anticoagulant treatment, neurosurgery, specialist treatment) Secondary prevention (aspirin, warfarin, ACE inhibitors, cholesterol lowering drugs) Medical regulation of blood pressure	Specialist care
Group 2. Medically influenced – interventions demonstrably informed by medical knowledge, but which may be conceived and/or delivered by other agencies		Sanitary reform (*H. pylori*) Diffusion of knowledge on infant hygiene and feeding)	Cholesterol reduction 1. Health education 2. Advertising incorporating healthy eating message 3. Diffusion of knowledge via media reporting of medical findings	Cholesterol reduction 1. Health education 2. Advertising incorporating healthy eating message 3. Diffusion of knowledge via media reporting of medical findings	Blood pressure reduction (health education on salt)

			Smoking reduction 1. Health education 2. Diffusion of knowledge via media reporting of medical findings 3. Legislation and regulation 4. Taxation Blood pressure reduction (health education on salt)	Smoking reduction 1. Health education 2. Diffusion of knowledge via media reporting of medical findings 3. Legislation and regulation 4. Taxation Blood pressure reduction (health education on salt)	
Group 3. Standards of living – developments which have a secondary impact on health, although they were created outside the medical sphere, with health not a priority	Overcrowding (resistance and infection) Diet (resistance)	Overcrowding (*H. pylori*) Diet 1. Refrigeration and availability of fresh meat 2. Availability and affordability of fruit and vegetables			Blood pressure reduction 1. Maternal nutrition and birth weight 2. Childhood nutrition 3. Decrease in salt in diet due to refrigeration Increase in cholesterol via fat in diet

9.4.1. Influences on mortality classed by group

Group one – medical – interventions conceived and delivered by health care professionals
Within this group are readily identifiable medical interventions, composed mainly of interventions aimed at curing individuals once they are ill. This is the grouping which, according to McKeown, contributed least to the decline in mortality up to 1971, and which, according to Bunker, contributed the most to the mortality decline since 1950. For each of the case study diseases this thesis has identified medical interventions which have contributed to the reduction in mortality, although the contribution of this group varies with each disease.

In the case of tuberculosis, this thesis reached a conclusion which those familiar with the findings of McKeown may find surprising. McKeown's conclusions were based on death data from England and Wales. Although his timing of the start of the decline of tuberculosis mortality in the 19th century has been questioned, his findings that mortality from tuberculosis had been declining throughout the 20th century, and that most of this decline occurred before the introduction of streptomycin, have not been challenged. Scotland, however, experienced a different pattern of tuberculosis mortality in the mid 20th century. Instead of a linear decline in mortality dating from the late 19th century, Scotland experienced a crisis in tuberculosis mortality during and after World War Two. This meant that continually improving standards of living were not responsible for the majority of the post-1950 mortality decline. The intervention of medicine, in the form of chemotherapy, mass diagnosis through MMR and to a lesser degree prevention in the form of BCG, was required to bring about a rapid decline in mortality in the early 1950s.

The main medical intervention for stomach cancer identified in this thesis was the surgical removal of the growth. Survival was largely dependent on the stage of the disease when the operation was undertaken; this made diagnosis crucial to any improvement in mortality. This thesis has argued that although medicine had the potential to cure using these medical interventions, in reality these contributed little to the overall decline in stomach cancer mortality since 1950. This is illustrated by the small improvement in five year survival between 1950 and 1999 (from approximately 4.9% in 1950 to 11.5 % among males and 14% among females in 1991-95).

Although IHD and ischemic stroke are pathologically similar, it has been argued that the contribution of clinical medical interventions varied between these two diseases. In the case of IHD, the introduction in the mid 1980s of a wider range of increasingly efficacious interventions aimed at improving survival was responsible for the acceleration in the decline of the death rate. However, the treatment of stroke lagged behind that of IHD. Treatments such as thrombolysis, which were responsible for significant increases in survival from IHD, were not available for the treatment of ischemic stroke in the time period covered by this thesis. Therefore, although medical interventions to improve survival from stroke did contribute to the decline in mortality, their contribution to the overall decline in ischemic stroke mortality was smaller than that seen for IHD.

Haemorrhagic stroke was the only disease covered by this thesis which did not see the introduction of a specific intervention to improve survival. The treatments which were available for ischemic stroke were not appropriate for the treatment of haemorrhagic stroke. It is possible that improvements in specialised nursing of stroke sufferers helped improve survival by preventing death resulting from post-stroke complications, although it is not possible to estimate the influence this may have had on mortality. The one medical intervention which did influence the decline in mortality from haemorrhagic stroke, as well as ischemic stroke and IHD, was the introduction of hypertensive medications for the treatment of high blood pressure.

Group two – medically influenced– interventions demonstrably informed by medical knowledge, but which may be conceived and/or delivered by other agencies

All influences in this group have been classed as 'medical'; these include influences which have traditionally been attributed to medicine, such as sanitary reform, as well as the wider influence of medicine in areas such as product advertising. This is a broader medical category than that employed by McKeown but was intended to take into account the wider influence of medicine outwith the traditional clinical sphere. The more diffuse medical influences contained in this category were not included in Bunker's study of mortality, which limited itself to clinical medicine.

In chapter seven it was established that one of the main influences on the decline in stomach cancer incidence was the declining prevalence of *H. pylori*. This thesis has attributed part of this decline to medical influences dating from the start of the 20th century. The first of these was the general effect of sanitary reform on overall hygienic conditions. McKeown linked

sanitary reform during the late 19th century with the decline of water-borne infections such as cholera and typhoid. Therefore, as *H. pylori* is believed to be transmitted via the gastro-oral route, it is probable that these improvements would have had a similar effect on *H. pylori*. The second medical influence on *H. pylori* which this thesis has proposed, is the increased diffusion of medical knowledge on infant hygiene and feeding practices dating from the early 20th century. It is believed that the risk of developing stomach cancer is greatest when *H. pylori* infection is acquired during infancy; therefore any measures to improve infant hygiene are likely to have had a beneficial effect on *H. pylori* prevalence.

Decline in cholesterol levels since the 1970s have been linked to the decline in mortality from both IHD and ischemic stroke. This has been attributed to diffusion of medical knowledge on the risk associated with a high fat diet. Until the results of the Framingham and other epidemiological studies were reported, no change could be detected in fat consumption. The transmission of these findings to the public was initially through press reporting and media advertising, before health education strategies were implemented.

The reduction in smoking prevalence since the 1970s has also been associated with the decline in mortality from IHD and ischemic stroke. This has been attributed to the diffusion of medical knowledge of risk, as well as government measures such as taxation and legislation and regulation.

Finally, a proportion of the decline in blood pressure, linked to the decline in mortality from IHD and both stroke subtypes, has been associated with health education on the dangers of excessive salt consumption.

Group three – standards of living – developments which have a secondary impact on health, although they were created outside the medical sphere, with health not a priority

Included within this group are changes in standards of living which resulted in a reduction in mortality but which were not motivated by an intention to improve population health. This group is comparable with McKeown's classification of 'standards of living'.

In the case of tuberculosis almost all of the reduction in mortality before 1950 was due to this type of influence. Although there was investment in tuberculosis schemes and a variety of treatments were introduced, these had little or no impact on mortality before 1950. However, this thesis is interested in factors which influenced *post-1950* mortality, and

although improving diet and housing after 1950 may eventually have caused the pre-war pattern of mortality decline to be re-established, they were no longer the main factors influencing tuberculosis mortality decline.

Improving standards of living have been isolated as one of the main influences on the post-1950 decline in mortality from stomach cancer. However, this has been linked to improvements in the late 19th and early 20th century which meant that cohorts born at that time went on to have a reduced risk of developing the disease. Improving standards of living influenced the two main risk factors for stomach cancer. In the case of *H. pylori* this took the form of reductions in overcrowding which lessened the risk of infection. Risk associated with diet declined through the combination of increasing availability of fresh meat as well as fruit and vegetables.

The decline of haemorrhagic stroke has been also been linked to improving standards of living dating from the late 19th and early 20th century. In particular, improvements in diet in infancy which reduced lifetime levels of blood pressure and later risk of brain haemorrhage. In addition, the increasing levels of cholesterol which were associated with the increase in risk of IHD and ischemic stroke have been linked to the decline of haemorrhagic stroke.

9.4.2. Main influences on post-1950 mortality decline

Both McKeown and Bunker placed a numerical value on the relative contribution of medicine to mortality decline. However, it is not possible to reach such a conclusion regarding the decline of mortality in Scotland since 1950. This thesis identified several different causes of death which influenced the post-1950 mortality decline and the contribution of each of these varied across this time period. Different factors also influenced the decline of each case study disease, with some having a detrimental effect on mortality from one cause whilst simultaneously helping to reduce mortality from another. Given the complex reasons for the mortality decline, conclusions regarding the main influences on the decline of each case study disease will be provided (table 9.2). This table has been divided into two time period (pre and post-1975).

Prior to1975 IHD and ischemic stroke did not contribute to the mortality decline, as mortality from these causes was either increasing or remained consistently high. Instead the main contributors to the mortality decline were tuberculosis, stomach cancer and haemorrhagic stroke. In the case of tuberculosis, although improving social conditions

following World War Two may have played a subsidiary role, medical interventions in the form of clinical cure, clinical prevention and an effective case finding mechanism were identified as the major influence on mortality decline after 1950. Medicine was not found to be the major contributor to the decline of stomach cancer or haemorrhagic stroke. Rather, the origins of their decline were dated to the late 19th and early 20th century. The main factor influencing this reduction in risk was improving standards of living. The consequence of this were improvements in diet which led to a reduction in blood pressure levels. In the case of stomach cancer, the reduction in the prevalence of *H. pylori* has been linked to sanitary reform and reductions in overcrowding levels.

Post-1975 mortality decline was driven by a different set of diseases and influences. Group 1 medical influences were still the main factor influencing the decline of tuberculosis. However, by 1975 tuberculosis only accounted for a very small share of total mortality, and did not contribute significantly to the mortality decline in this time period. In the case of stomach cancer and haemorrhagic stroke the factors which influenced the decline in mortality pre-1975 continued to exert an influence; however, it is possible that medicine also contributed a small amount to the decline. This took the form of improvements in the diagnosis of stomach cancer leading to a slight improvement in survival rates following surgery. Specialist nursing may also have improved survival following a haemorrhagic stroke.

The main contributors to the post-1975 mortality decline were IHD and ischemic stroke. The previous trend of increasing and high death rates was reversed post-1975. In the case of IHD and ischemic stroke no influence on the mortality decline has been attributed to group three (standards of living). The prevalence of the main risk factors for both these diseases were traced throughout the 20th century. The earlier increase in prevalence of risk factors was associated with *improving* standards of living, as the population believed them to be desirable in the absence of medical warnings of their danger. This thesis did not detect any change in the prevalence of the behaviours that we now think of as 'risk factors' until medicine had first established and reported them to be 'risk factors'. Although the means by which this knowledge entered the public domain may not always have been traditionally viewed as medical (for example food advertising or media reporting) these were entered into group two. The assigning of the decline in the prevalence of the main IHD and ischemic stroke risk factors to group two, instead of group three, illustrates the importance of a justifiable division between changes in lifestyle which were the result of the diffusion of

medical findings and standards of living. The other major influences on the decline of IHD and ischemic stroke came from group one. These were readily identifiable medical interventions aimed to improve survival of those suffering from these conditions.

Table 9.2. Main influences on the decline of mortality from case study diseases, pre- and post-1975

	Pre-1975	Post-1975
Tuberculosis	Chemotherapy BCG MMR Improving post-war social conditions	Chemotherapy BCG
Stomach cancer	Reduction in prevalence of *H. pylori* as a result of sanitary reform, and improving standards of living leading to a reduction in overcrowding levels Reduction in salt consumption as a result of improving standards of living increasing the variety and affordability of food Small contribution by surgery	Reduction in prevalence of *H. pylori* Reduction in salt consumption Developments in diagnosis leading to slight improvements in survival after surgery
IHD		Primary prevention Secondary prevention Diffusion of medical findings on main IHD risk factors
Ischemic stroke		Primary prevention Secondary prevention Diffusion of findings on main IHD risk factors
Hemorrhagic stroke	Improving standards of living leading to changes in diet which led to a reduction in blood, as well as possible benefit of increase in cholesterol	Reduction in blood pressure Possible improvement in survival as a result of specialist medical care

9.4.3. Impediments to the post-1950 mortality decline

Although this thesis is concerned with the *decline* of mortality in Scotland since 1950, it is interesting to consider why mortality did not decline further. Several possible reasons have been identified. These include the fact that most of the cancers which were sampled in chapter four did not experience a reduction in mortality after 1950 (see appendix 2). The reasons why these causes of death did not decline is outwith the scope of this thesis. However, given the complex relationship between lifestyle and mortality it would be interesting to consider whether factors which caused the case study diseases to decline may have had a detrimental effect on other causes post-1950.

One of the main points to emerge from the analysis of the case study diseases is the unequal distribution of mortality amongst different socio-economic groups. For most diseases the greater risk of mortality is amongst the most deprived groups. If the death rates of the most affluent group had been experienced by all, a larger decline in mortality would have occurred.

The relationship between deprivation and excess mortality is not new. In the 19th century Edwin Chadwick in his *Report on the Sanitary Condition of the Labouring Population on Great Britain* (1842) drew attention to the excess mortality suffered by the poor. McKeown also highlighted the relationship between poverty and the infections in his advocacy of the importance of improving standards of living in mortality decline. In the late 20th century, the relationship between deprivation and excess mortality from the degenerative diseases was stressed by *The Black Report* (1980). This report was the result of a Working Group set up by the Labour government in 1977. The findings of this report on the relationship between poverty and mortality differed from those seen in the 19th and earlier part of the 20th century, when absolute poverty was associated with death from infection. Instead this report emphasised the role of relative poverty as a result of inequalities between socio-economic groups. *The Black Report* did recommend the use of health education but stressed the importance of a comprehensive anti-poverty strategy involving a wide ranging programme of public expenditure to reduce inequality. In 1980, when the report was published, a Conservative government was in power and most of the recommendations were not implemented (Marmot, 2001). Although the *Black Report* did not have an immediate impact on policy it was partially responsible for the growth in academic interest in the field of inequality and health.

It was not until 1997, when Labour was again in government that some of the recommendations of the *Black Report* were included in government policy. These included measures such as the national minimum wage, new deal and tax credits, aimed at improving the conditions of the poorest in society. The implementation of these policies occurred too late to influence the mortality decline studied by this thesis, however a recent review has considered the impact they have had since 1997 (Shaw *et al*, 2005). The findings of this review are not encouraging, as it found that inequality between socio-economic groups was the widest it has been since the Victorian era, with the most affluent groups living 4.06 years longer in 2001-3 than the most deprived. This represented a widening of the gap compared to 1992-93. To reduce this inequality Shaw *et al* recommended "*more potent and redistributive policies*" (p.1016).

9.5. Policy implications and future research

In 2.7 the current direction of Scottish health policy was outlined. The suitability of this to future mortality decline will now be considered in light of the findings of this thesis. At the start of the 21st century the acute medical sector still accounts for the vast majority of spending on health in Scotland. Under the definitions devised for this thesis this medical sector would fall under group 1 (medical interventions involving an interaction with a health care professional). The potential for further mortality decline from this source will be considered. This thesis attributed the majority of the decline of tuberculosis to this group. However, it is unlikely that further substantial gains to total mortality decline will be achieved from this disease as by the end of the 20th century tuberculosis only accounted for a marginal share of mortality. In the case of stomach cancer, although this thesis only attributed a small share of the post-1950 mortality decline to clinical medicine there is potential for further mortality reduction. Evidence from Japan has shown that if the disease is identified at an early enough stage (via population wide screening) then the operative success rate is high. However, the financial feasibility of introducing such a programme in Scotland has been questioned.

IHD and ischemic stroke accounted for the majority of the post-1950 mortality decline, and in 2001 22% of all mortality was still attributable to IHD and 11.5% to stroke, indicating that there is still substantial potential for future mortality decline from these causes. This thesis has argued that since the mid 1980s, medical treatments for IHD (and to a lesser degree for stroke) have become increasingly effective in preventing the development of cardiovascular

disease, and in increasing the survival of those already ill. It is therefore likely that medical treatments will continue to contribute to mortality decline from these causes. The relative contribution of medical treatments to stroke may increase as many of the new 'clot busting' drugs which had previously only been available for IHD become licensed for use. Therefore, it is probable that future mortality reduction could be gained by clinical medicine in the case of IHD, stroke and stomach cancer.

The second group considered by this thesis were factors which were influenced by medicine which did not involve a personal interaction. In general this meant communicating medical findings on risk factors to the public which led to the reduction in risk factor prevalence in the population. This type of influence was found to be most significant in the decline of IHD and ischemic stroke. Initially the diffusion of the knowledge of risk factors was through non-medical sources, such as media reporting of findings and advertising. However, this changed from the 1980s and the communication of findings on risk factors is now part of health education. In current Scottish health budgets health education only accounts for 2% of total spending. Given the response of the population to the diffusion of knowledge of risk factors such as smoking in the past, it can be argued that this area of medicine should receive a larger share of total funding. Most of the population are now aware of the main underlying causes of ill health, in particular the implications of smoking, poor diet, lack of exercise and obesity on health. Despite this, the prevalence of these risk factors in Scotland is still relatively higher than those of its near neighbours. Therefore it would be anticipated that the greatest future benefit would be derived from more sophisticated methods of health education, which do not simply deliver the message of what is bad for an individual, but which also offer practical means of support for behaviour change. Past examples of this type of measure which have been successful include 'Smokeline', which combined a mass media campaign, with telephone number to offer individual support and advise for those wishing to quit.

The final area which this thesis considered was the role of standards of living. When the groups of influence on mortality were defined, this group was explicitly defined as non-medical and as such would not be covered by policy aimed at health improvement. The initial decline of stomach cancer and haemorrhagic stroke was linked to improving standards of living in the early 20th century. However, it has been argued in this thesis that as the 20th century progressed it became increasingly difficult to isolate the relative contribution of standards of living on population risk from the response of the population to the diffusion of

medical knowledge of risk. This has led to a re-evaluation of what is covered by the term 'standard of living'. In this thesis a poor standard of living has been linked to poverty and in the case studies covered deprivation has been linked with higher mortality rates. In the previous section the role of relative poverty in restricting mortality decline was highlighted. The role of poverty in preventing further mortality decline has now been identified in Scottish health policy (Scotttish Office, 1999). Initiatives have been outlined for a multi-agencey approach, to improve the financial situation of individuals, such as the minimum wage and improved pensions. However, these polices still fall short of the substantial redistributive policies advocate elsewhere (see Lynch *et al,* 2000, Shaw *et al*, 2005).

The three groups of influence identified in this thesis contributed to the decline in mortality in Scotland since 1950. However, the contribution of the case study diseases to total mortality has changed since 1950. Tuberculosis and stomach cancer now contribute far less to total mortality, and although stroke and IHD are still significant causes of death, deaths from neoplasms now dominate Scottish mortality (accounting for 26% of all deaths in 2001). In addition, the way in which the groups affect mortality has changed over time. Medicine and standards of living are no longer two distinct influences on mortality. Instead medical research has identified many of the main risk factors for ill health, including smoking, poor diet, lack of exercise and poverty, and the prevalence of these is determined by both knowledge of the risk and standard of living Current policies to improve health include measures for the clinical treatment and prevention of disease, health education and policies aimed at reducing the health consequences of relative poverty. Future research should consider the impact which these policies will have on mortality reduction from the causes of death which dominate mortality at the start of the 21st century.

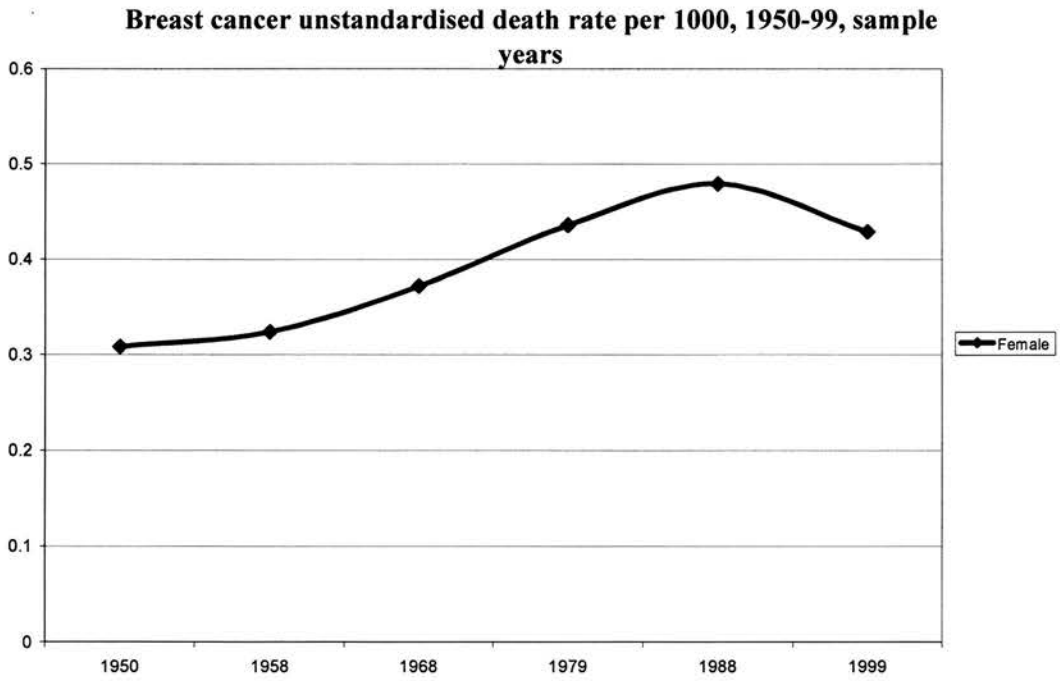
Appendix 1. ICD codes relating to sampled causes of death

Tuberculosis 6th revision 001-019 7th revision 001-019 8th revision 010-019 9th revision 010-018	**Stomach Cancer** 6th revision 151 7th revision 151 8th revision 151 9th revision 151
Colon Cancer (known as Neoplasm of the Large Intestine pre-9th revision 6th revision 153 7th revision 153 8th revision 153 9th revision 153	**Cancer of the Trachea, Bronchus and Lung** 6th revision 162 a. b. c. 7th revision 162.0 162.1 8th revision 162.0 162.1 9th revision 162
Breast Cancer 6th revision 170 7th revision 170 8th revision 174 9th revision 174	**Ovarian Cancer** 6th revision 175 7th revision 175 8th revision 183 9th revision 183
Prostate Cancer 6th revision 177 7th revision 177 8th revision 185 9th revision 185	**Diabetes Mellitus** 6th revision 260 7th revision 260 8th revision 250 9th revision 250
Ischemic Heart Disease 6th revision 420-422 7th revision 420-422 8th revision 410-414 9th revision 410-414	**Stroke** 6th revision 330-334 7th revision 330-334 8th revision 430-438 9th revision 430-438
Pneumonia 6th revision 490-493 7th revision 490-493 8th revision 480-486 9th revision 480-486	**Chronic Obstructive Pulmonary Disease** 6th revision 501, 502, 526, 527 7th revision 501, 502, 526, 527 8th revision 490, 491, 492, 518, 519 9th revision 490, 491, 492, 494, 496

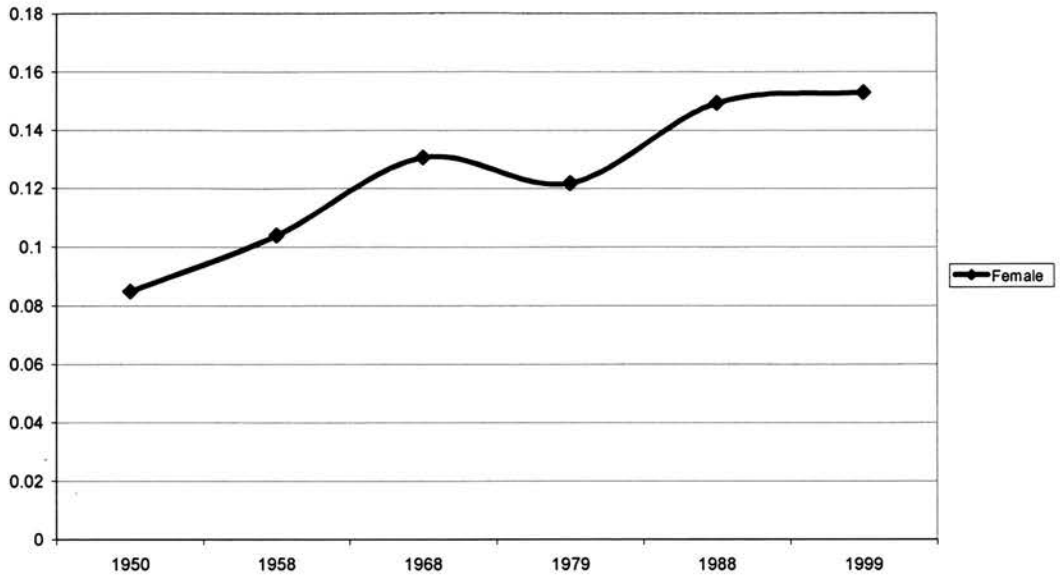
Hyperplasia of the Prostate 6th revision 610 7th revision 610 8th revision 600 9th revision 600	**Congenital Anomalies** 6th revision 750-759 7th revision 750-759 8th revision 740-759 9th revision 740-759
Certain Conditions Originating in the Perinatal Period (Infancy) 6th revision 760-776 7th revision 760-776 8th revision 760-779 9th revision 760-779	**Senility (without mention of psychosis)** 6th revision 794 7th revision 794 8th revision 794 9th revision 797
Road Traffic Vehicle Accidents 6th revision E810-E825 7th revision E810-E825 8th revision E810-E819 9th revision E810-E819	**Accidental Falls** 6th revision E900-E904 7th revision E900-E904 8th revision E880-E887 9th revision E880-E889
Suicide 6th revision E970-E979 7th revision E970-E979 8th revision E950-E959 9th revision E950-E959	

Appendix 2. Graphs of selected causes of death, for sample years, 1950-1999

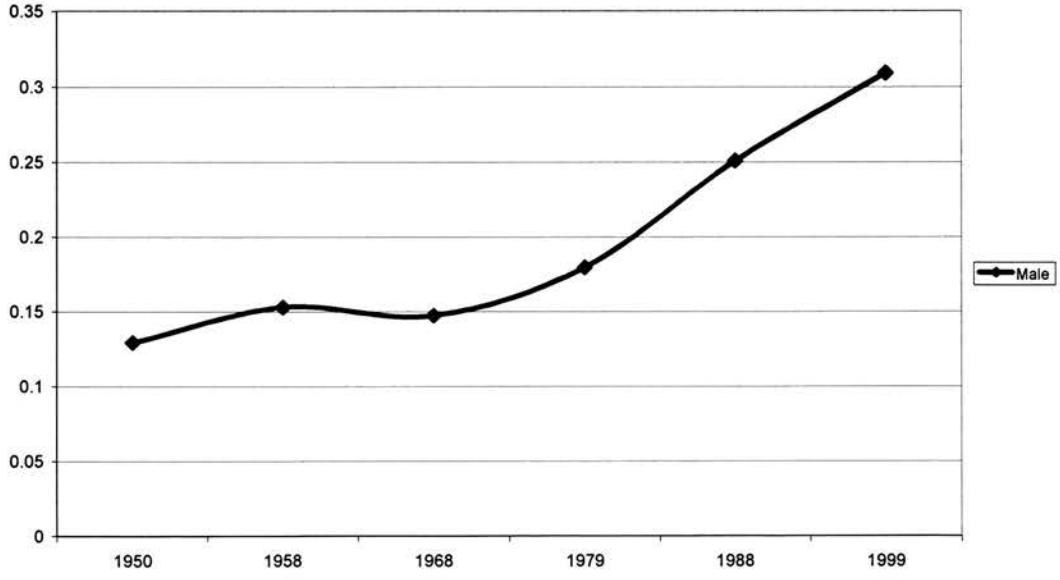
1. *Causes of death for which the death rate increased between 1950 and 1999 – excluded from further investigation*



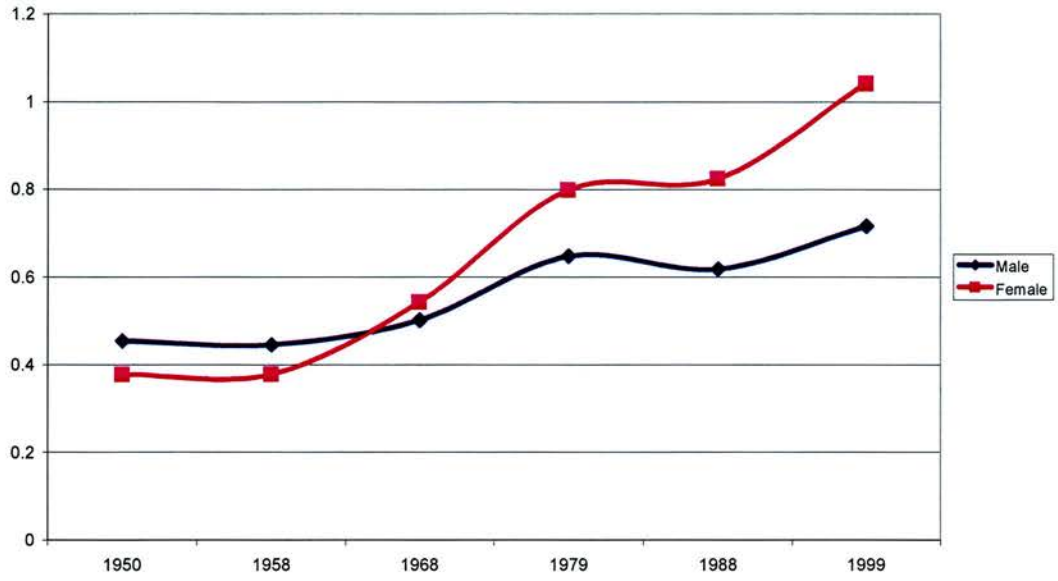
Ovarian cancer unstandardised death rate per 1000, 1950-99, sample years



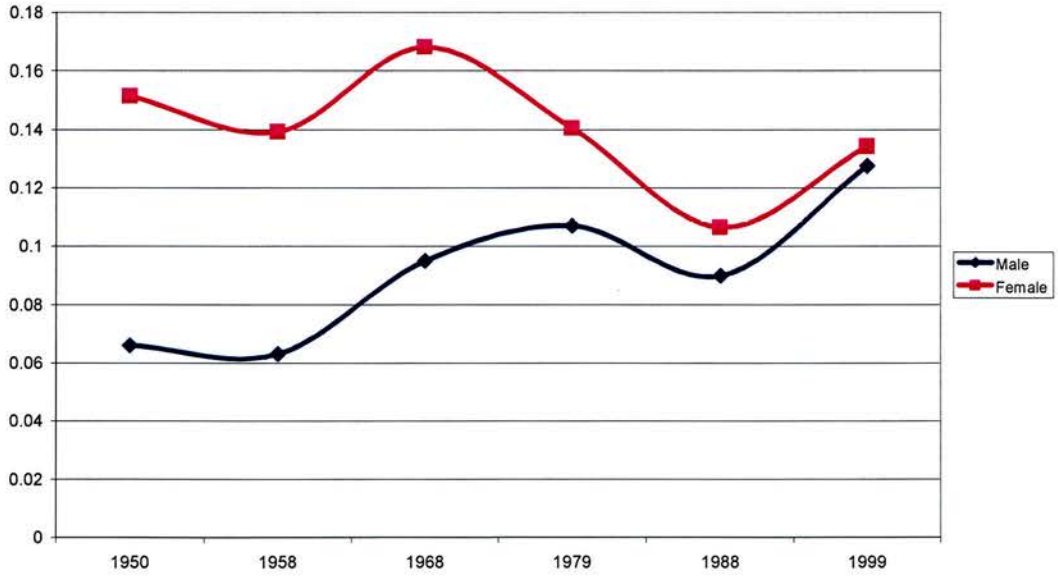
Prostate cancer unstandardised death rate per 1000, 1950-99, sample years



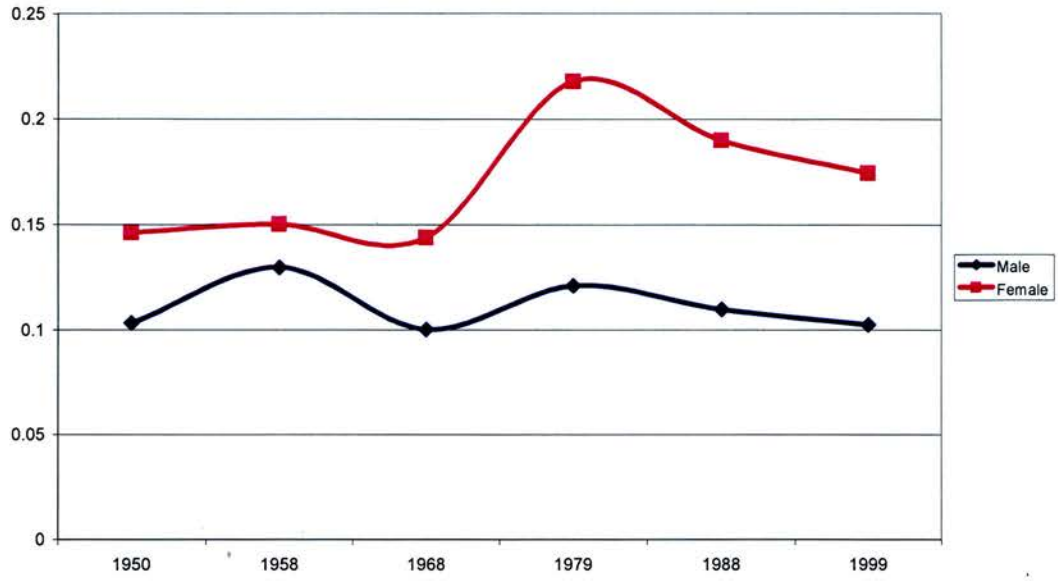
Pneumonia unstandardised death rate per 1000, 1950-99, sample years



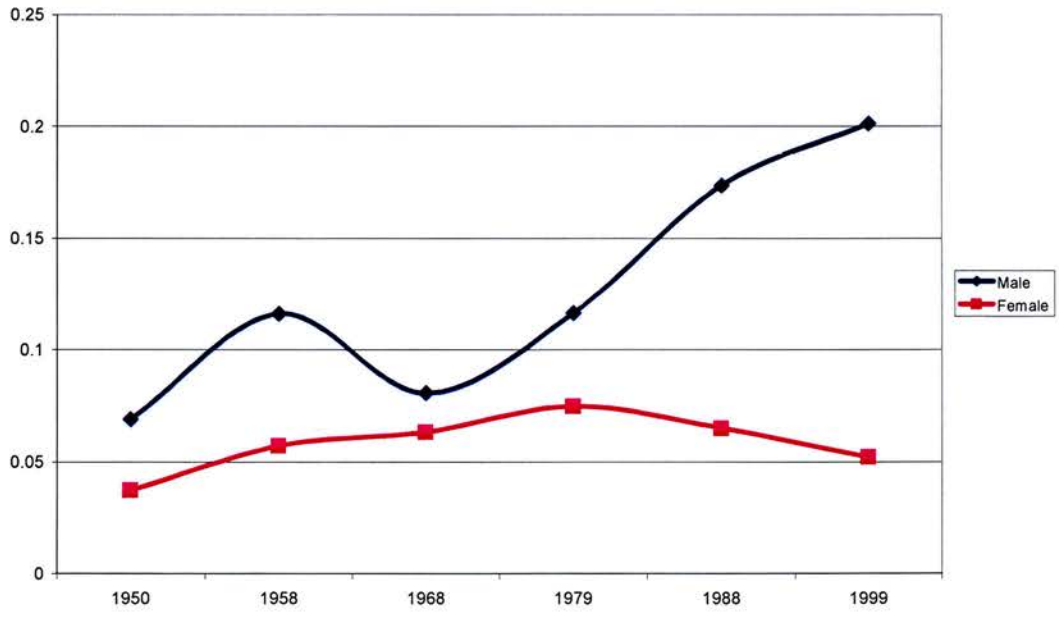
Diabetes mellitus unstandardised death rate per 1000, 1950-99, sample years



Accidental falls unstandardised death rate per 1000, 1950-99, sample years

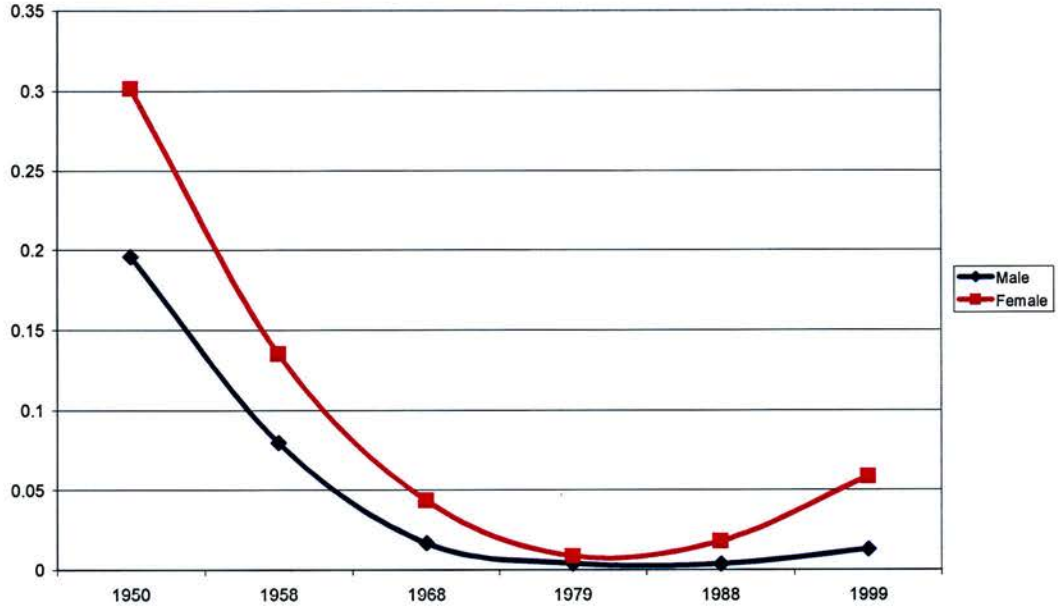


Suicide unstandardised death rate per 1000, 1950-99, sample years

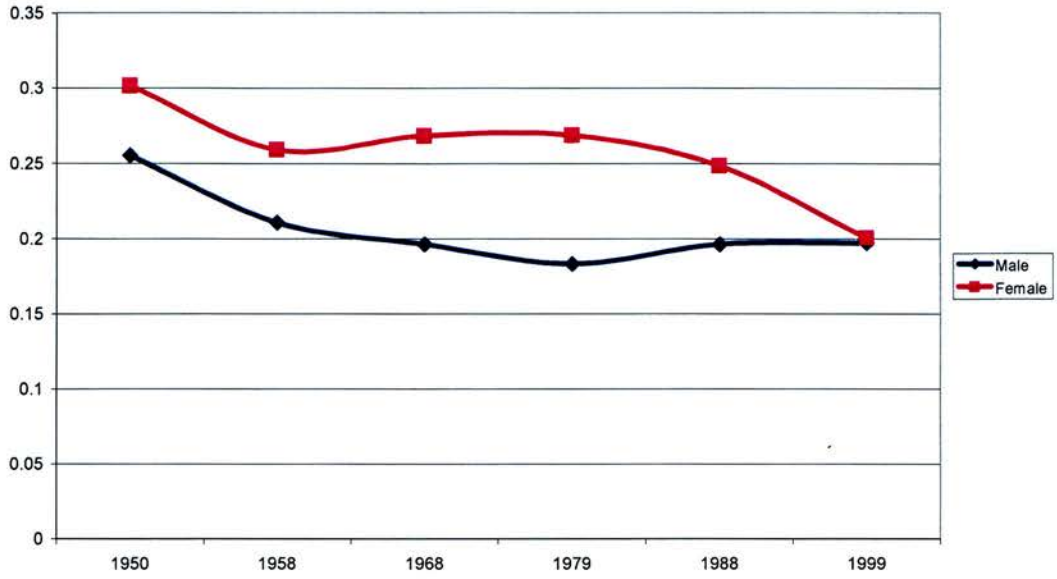


2. *Causes of death for which the death rate decreased between 1950 and 1999 – excluded from further investigation*

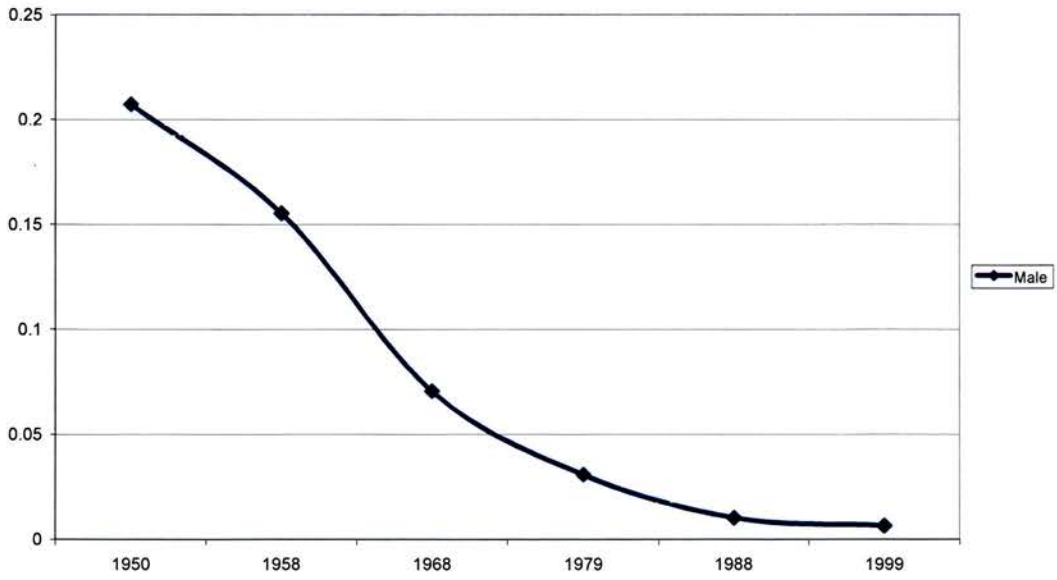
Senility unstandardised death rate per 1000, 1950-99, sample years



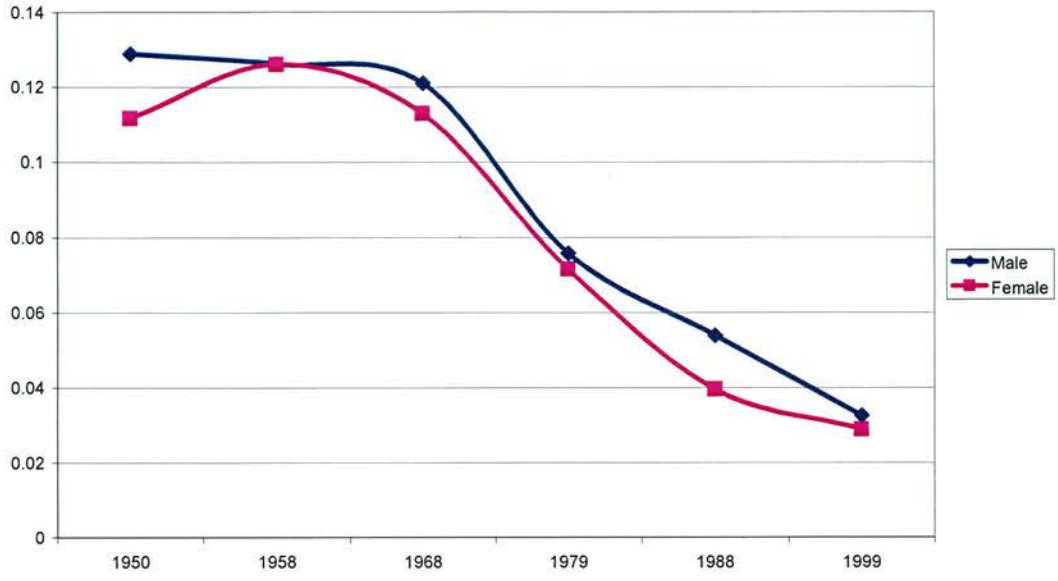
Colon cancer unstandardised death rate per 1000, 1950-99, sample years



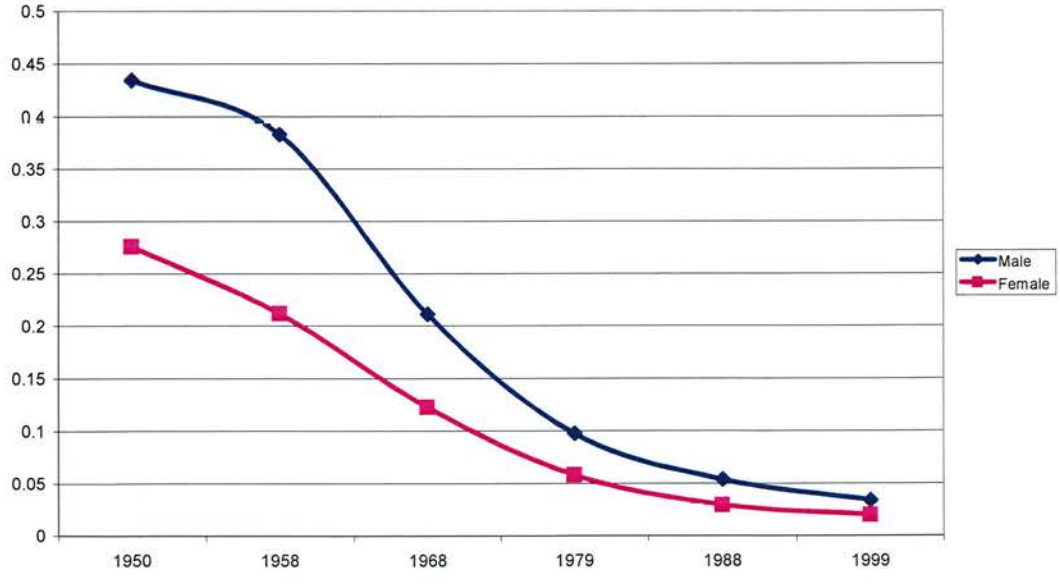
Hyperplasia of the prostate unstandardised death rate per 1000, 1950-99, sample years



Congenital anomalies unstandardised death rate per 1000, 1950-99, sample years

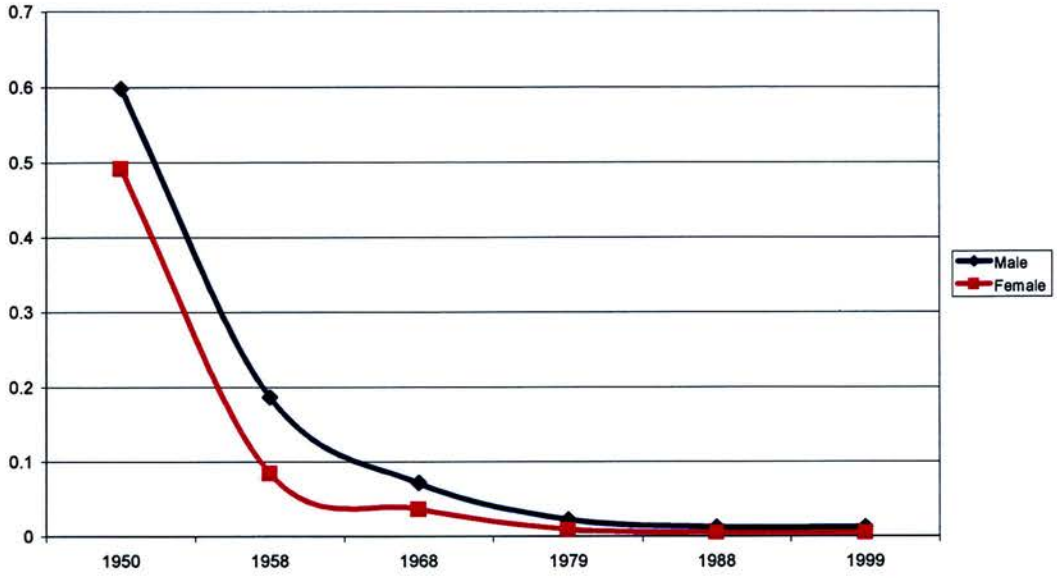


Conditions originating in the perinatal period (infancy) unstandardised death rate per 1000, 1950-99, sample years

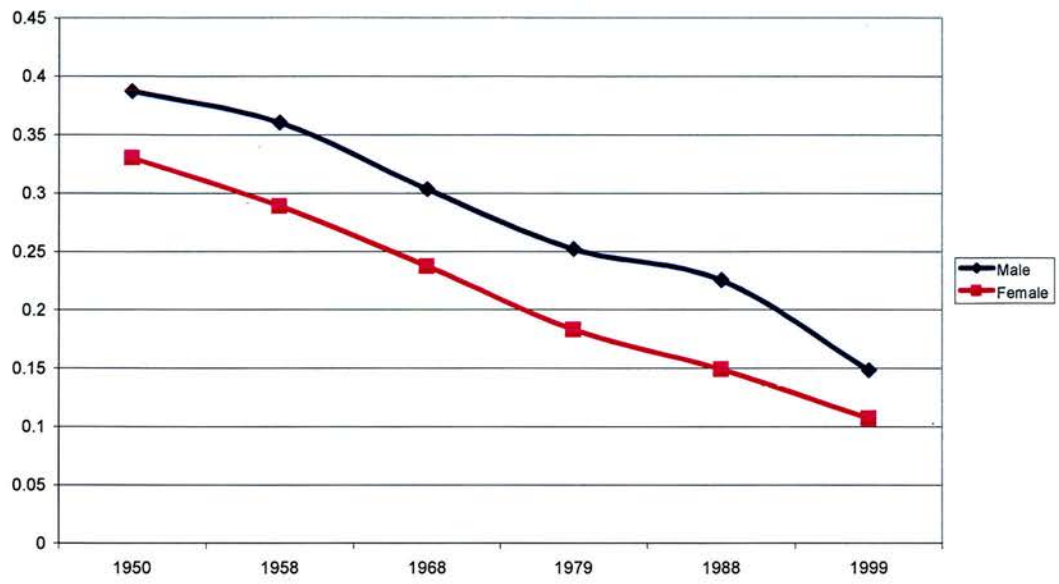


3. *Causes of death for which the death rate decreased between 1950 and 1999 – selected for further investigation.*

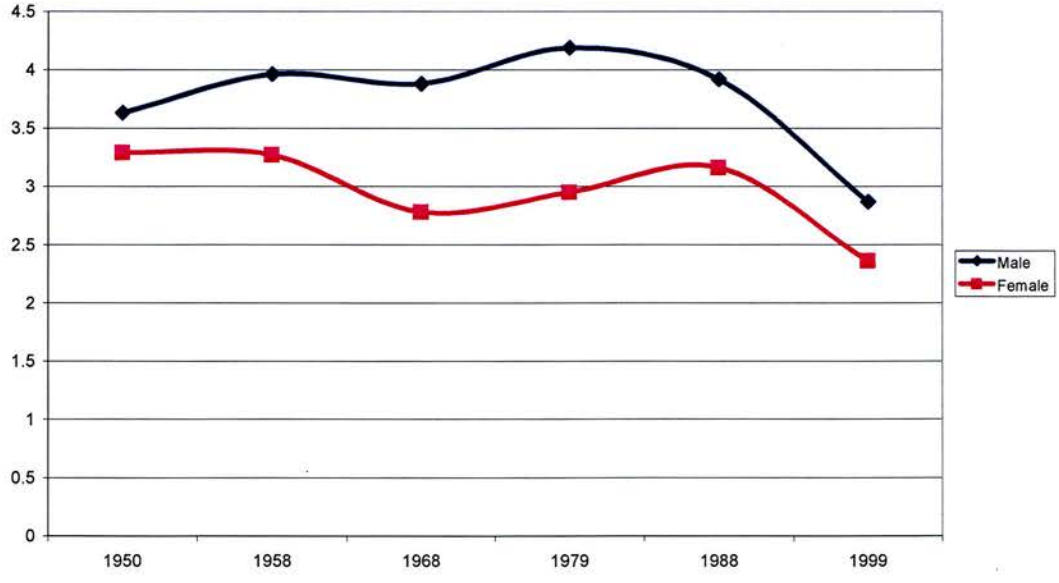
Tuberculosis unstandardised death rate per 1000, 1950-99, sample years



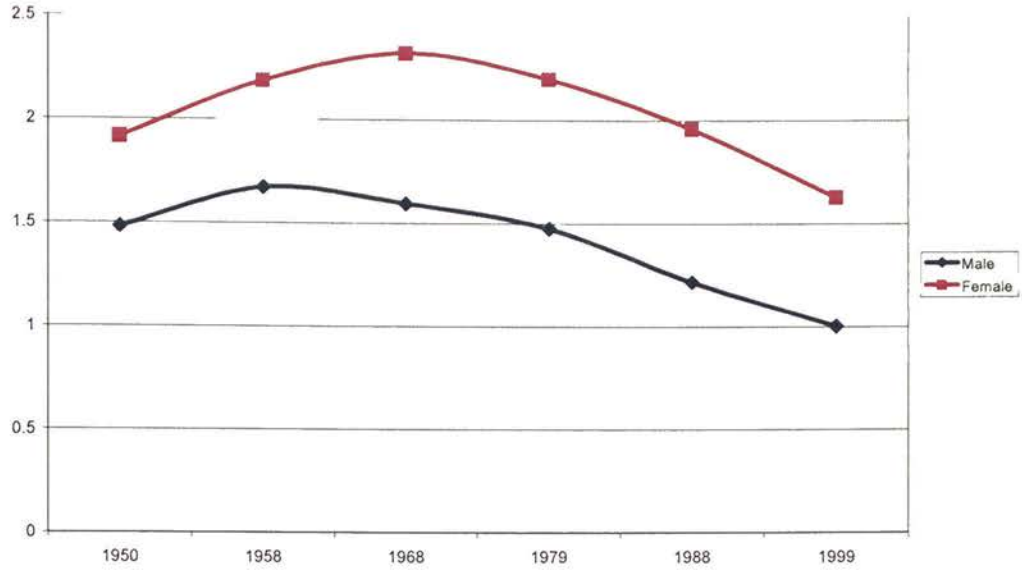
Stomach cancer unstandardised death rate per 1000, 1950-99, sample years



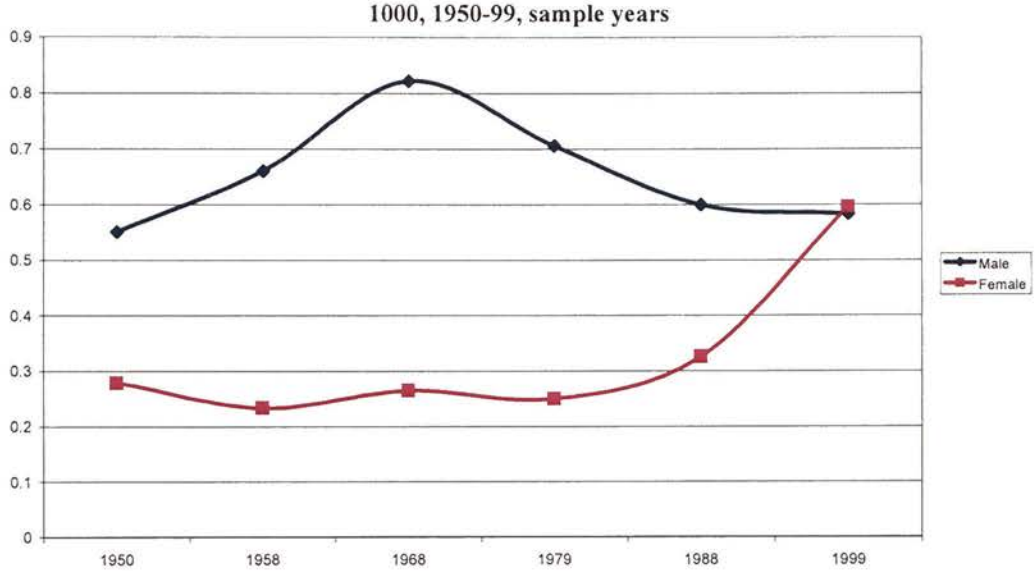
Ischemic heart disease unstandardised death rate per 1000, 1950-99, sample years



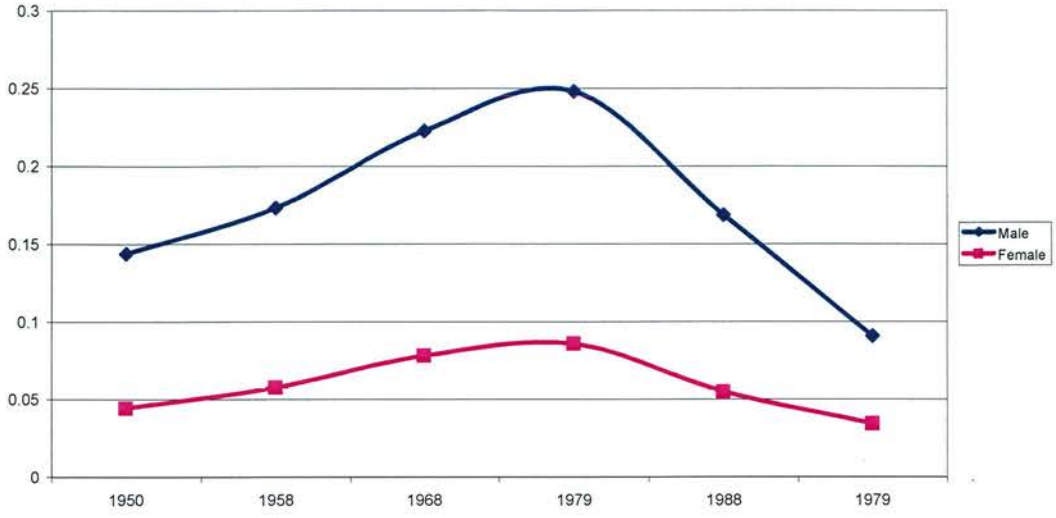
Cerebrovascular unstandardised death rate per 1000, 1950-99, sample years



Chronic Obstructive Pulmonary Disease death rates unstandardised per 1000, 1950-99, sample years



Road Traffic Vehicle Accidents death rate unstandardised per 1000, 1950-99, sample years



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