Total mortality inequality in Scotland: the case for measuring lifespan variation

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Abstract

Lifespan variation captures variation in age at death *within* a population as opposed to the inequality in average health that exists *between* populations. Higher lifespan variation equates to greater total inequality and is negatively correlated with life expectancy. Lifespan variation has not previously been measured for Scotland, where life expectancy and mortality rates are the worst in Western Europe. Routinely measuring lifespan variation in Scotland contributes to understanding the extent, and changing nature, of mortality inequalities.

Lifespan variation estimates were calculated using data from the Human Mortality Database and from Census population estimates, vital events data and the Carstairs Score. Analyses included joinpoint regression, Age-specific decomposition, Monte Carlo simulation, slope index of inequality, relative index of inequality, and Age-cause specific decomposition.

Males in Scotland experience the highest level of lifespan variation in Western Europe, increasing since the 1980s: the longest sustained increasing trend found in Western Europe. Increasing mortality rates across working adult ages account for Scotland's diverging trend. This age pattern of mortality was not evident in England and Wales. Lifespan variation for males in the most deprived quintile was higher in 2011 than in 1981 and the socioeconomic gradient steepened. Premature deaths from external causes of death accounted for an increasing proportion of lifespan variation inequalities. Without tackling the root causes of social inequality Scotland may struggle to reduce total inequality and improve its lifespan variation ranking within Western Europe.

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Author's declaration

I declare that, except where explicit reference is made to the contribution of others, that this dissertation is the result of my own work and has not been submitted for any other degree at the University of Glasgow or any other Institution.

Rosie Seaman

Conference presentations

The following conference presentations were based on analysis from the results in chapters 4, 5 and 7 of this thesis:

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Seaman, R., Leyland, A.H., & Popham, F. How have trends in lifespan variation changed since 1950? A comparative study of 17 Western European countries. *The European Journal of Public Health,* 26, 360-362. 7th European Public Health Conference, Glasgow (2014)

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I declare that the research carried out for these conference presentations was my own work: co-authors provided editorial advice.

Peer-reviewed publications

Some of the findings from chapter 4 and chapter 6 were incorporated into jointly authored articles that were written and accepted for publication during the course of this PhD:

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I declare that the research carried out for these publications was my own work: co-authors provided editorial advice.

Abbreviations

HMD: Human Mortality Database

ICD: International Classification of Disease

LGD: Local Government District

NRS: National Records of Scotland

NS-SEC: National Statistics Socio-economic Classification

RGSC: Registrar General's Social Class

SAH: Self-Assessed Health

SIMD: Scottish Index of Multiple Deprivation

SYOA: Single Year of Age

WHO: World Health Organisation

1 Introduction

1.1 Mortality in Scotland

It is now well established that mortality outcomes in Scotland are relatively poor within the context of Western Europe: the population of Scotland experience the highest all-cause mortality rate and are expected to have the shortest lives (McCartney et al., 2012b, Schofield et al., 2016, Leyland et al., 2007b). Inequalities in mortality in Scotland, measured between the most and least deprived socioeconomic groups, have also widened following the 1980s. This was because the most deprived experienced the highest level of absolute mortality to begin with and the smallest relative rate of improvement over time (Leyland et al., 2007a, Leyland et al., 2007b, Norman et al., 2011).

Research has paid particular attention to the widening gap between males of working age from the most deprived socioeconomic group compared to the least deprived socioeconomic group. Increasing premature mortality rates for the most deprived males of working age between 1981 and 2001 were largely due to deaths from causes that can be categorised as 'external': alcohol, substance abuse, suicides, accidents and assaults (McCartney et al., 2012a, Walsh et al., 2016, Schofield et al., 2016, Leyland et al., 2007a). In addition to representing a tragic loss of potential life, this age pattern of mortality can be considered counter intuitive: the economic cost of social interventions for reducing premature adult deaths from external causes tends to be lower than the economic cost of medical interventions for reducing old age deaths from chronic diseases (Nau and Firebaugh, 2012, Smits and Monden, 2009).

Widening mortality inequalities are not a problem unique to Scotland: they are a public health concern across many economically developed countries (Mackenbach et al., 2003, Mackenbach et al., 2016a). Given that most of these countries have comprehensive social welfare systems and some have longstanding, formal commitments to reducing mortality inequalities (The Scottish Government, 2010, Department of Health, 1999, Department of Health, 2013), their persistence over time is deemed to be one of public health's greatest disappointments (Bambra, 2011a, Mackenbach, 2012).

Mortality inequalities are traditionally measured by comparing the difference in average health status of predefined social groups. However this may not demonstrate the full extent of the problem. Measuring the average health difference between socioeconomic groups' risks assuming: that a homogenous gain in health has been achieved for everyone *within* the same socioeconomic group, or that health inequalities between individuals *within* the same socioeconomic group have stayed constant over time. Recent research attention has therefore been paid to the notion of *total inequality*: the variation in a health outcome that exists between all individuals *within* a population (Gakidou and King, 2002, Murray et al., 1999, Harper and Lynch, 2006). Limited research has applied the concept of total inequality to measure the full extent of mortality inequalities in Scotland (Popham and Boyle, 2010).

1.2 Interpretation of lifespan variation in this thesis

Lifespan variation is one outcome measure of total inequality: it is interpreted as the average number of years of life lost per death (van Raalte et al., 2011, Tuljapurkar, 2010, Shkolnikov et al., 2011) and higher lifespan variation equates

to greater inequality in the age at death (Murray et al., 1999, Gakidou and King, 2002, Harper and Lynch, 2006, Smits and Monden, 2009). The data required to calculate lifespan variation are death counts and population estimates: this data has a long history of being collected in Scotland (National Records of Scotland, 2016c).

Unlike improvements in life expectancy, which are achieved by reducing mortality rates at any age, reductions in lifespan variation are age dependent: mortality rates across premature ages of death must be reduced faster than mortality rates across older ages (Zhang and Vaupel, 2009, Seligman et al., 2016). This has led to lifespan variation being referred to as the ultimate expression of inequality (Smits and Monden, 2009). Lifespan variation in this thesis is considered to be a valid indicator of a society's ability to protect the most vulnerable members of the population from the social and economic risks associated with premature death (van Raalte et al., 2011, Smits and Monden, 2009, Seligman et al., 2016).

1.3 Definition of premature mortality in this thesis

In the context of this thesis a death is defined as premature if delaying it to an older age would contribute to a decrease in lifespan variation. The precise age which distinguishes premature and older deaths is different in each country and tends to have increased over time (as life expectancy increases) meaning that a much wider spread of ages contribute to decreasing lifespan variation relative to earlier in time (Gillespie et al., 2014, Zhang and Vaupel, 2009). This is one advantage of defining a death as premature in relation to lifespan variation: it provides a definition that is specific to the population and temporal context

being studied. Zhang and Vaupel (2009) applied advanced methods to determine exactly what this distinguishing age was in four countries between 1950 and 2005. Gillespie et al. (2014) similarly illustrated how shifts to threshold age and the widening spread of ages being defined as premature have impacted the correlation between life expectancy and lifespan variation. Although this thesis does not apply the advanced methods required to establish the precise distinguishing age the same technical definition derived from lifespan variation is still applicable: a death is considered premature if delaying it to an older age would have contributed to decreasing lifespan variation. Throughout the analysis chapters specific age groups are referred to when describing the results in order to aid interpretation.

1.4 Research Questions

The primary aim of this thesis was to measure, analyse and interpret lifespan variation, a measure of total inequality, for Scotland. Twelve research questions were developed to address this overarching aim. They are not mutually exclusive, answering each individual research question helped to inform the others. The 12 research questions, and the chapters which they are answered in, are listed below:

Chapter 4

1. Has Scotland's lifespan variation ranking within Western Europe changed over time?

2. Was the timing and relative rate of lifespan variation change in Scotland comparable with any other Western European country?

Chapter 5

3. Which ages of death contributed to the lifespan variation trend in Scotland?

4. Did the ages of death contributing to the lifespan variation trend in Scotland differ from the ages of death contributing to the lifespan variation trend in England and Wales?

Chapter 6

5. Was lifespan variation higher or lower in Scotland at a shared level of life expectancy with England and Wales?

6. Which ages of death account for the lifespan variation gap between Scotland and England and Wales at a shared level of life expectancy?

Chapter 7

7. Is there a socioeconomic gradient for lifespan variation in Scotland?

8. Has the socioeconomic gradient for lifespan variation in Scotland changed over time?

9. Are changes to the socioeconomic gradient for lifespan variation in Scotland related to Scotland's deteriorating lifespan variation ranking within Western Europe over time?

Chapter 8

10. Which ages and causes of death contributed to changes in lifespan variation, over time, for different socioeconomic groups in Scotland?

11. Which ages and causes of death contributed to the lifespan variation gap between the most and least deprived socioeconomic groups in Scotland?

12. Which ages and causes of death contributed to the lifespan variation gap between the most and least deprived socioeconomic groups, when life expectancy was similar, in Scotland?

1.5 Thesis structure

Chapter 2 provides an overview of the literature that relates to health, mortality inequalities and their empirical measurement. It highlights the empirical evidence that has sought to explain Scotland's relatively worse mortality experience within the context of Western Europe and in contrast to England and Wales, its closest geographical comparator. It also focuses on the international studies which have started to conceptualise and analyse mortality inequalities in terms of lifespan variation.

Chapter 3 outlines the data used and methods of analysis applied to understand lifespan variation inequalities in Scotland, including reflections on the strengths and limitations.

Chapters 4 to 8 report the results of the analysis carried out. Each of these chapters begins with a brief background section and summarises the relevant data sources and methods of analysis. Each of these chapters concludes by reflecting on the research implications in terms of this thesis and beyond.

Chapter 4 quantifies the population level lifespan variation trend in Scotland compared to 16 other comparable Western European countries using lifetables obtained from the Human Mortality Database.

Chapter 5 continues to focus on the population level lifespan variation trend in Scotland using the same data. It identifies the age patterns of mortality driving the lifespan variation trend within Scotland and within England and Wales in order to determine if any age patterns of mortality change in Scotland were distinguishable.

Chapter 6 extends the analysis of the population level trend in Scotland compared to England and Wales by measuring the lifespan variation gap at similar levels of life expectancy, albeit it at different points in chronological time. This allows lifespan variation to be studied independently of life expectancy.

Chapter 7 begins the process of analysing lifespan variation inequalities between socioeconomic groups in Scotland in order to demonstrate that measures of total inequality still adequately reflect inequities rather than just random variation. It uses population estimates derived from the Census in 1981, 1991, 2001 and 2011 and relevant all-cause mortality data. It quantifies changes to the socioeconomic gradient for lifespan variation and considers these changes within the context of Scotland's deteriorating ranking relative to the rest of Western Europe.

Chapter 8 uses the Census population estimates and the relevant cause-specific mortality data in order to quantify the age-cause specific contributions made to lifespan variation inequalities in Scotland. This helps build a fuller picture of what has driven total inequality in Scotland: evidence seeking to understand the determinants of widening mortality gaps in one country is likely to be of interest to other countries looking to ensure improvements in average population health are achieved alongside reductions in inequality. Lifespan variation inequalities

between socioeconomic groups are measured in terms of the lifespan variation change achieved over time by the most deprived compared to the lifespan variation change achieved over time by least deprived, the lifespan variation gap between the most and least deprived group at the same chronological time, and the lifespan variation gap between the most and least deprived group at a similar level of life expectancy.

Chapter 9 provides a summary of the main findings and the contributions these make to the existing body of literature. It considers some of the strengths and limitations of this thesis and highlights some areas that could benefit from further research.

2 Literature review

The purpose of this literature review was to explore some of the theoretical debates that have informed our understanding of health and health inequalities. It summarises the multiple interpretations of health inequalities and multiple measurements used in empirical research. This provides a foundation for linking the thesis findings into internationally relevant debates surrounding health inequalities and their empirical measurement. The literature review establishes which theoretical explanations might be most relevant for health inequalities in Scotland and identifies lifespan variation as a measure which might further our understanding of health inequalities in this context. Theories emphasising the structural determinants of health and recognising that relative deprivation could impact health via psychosocial and materialist mechanisms are highlighted as the most relevant for Scotland. These theories are then utilised throughout the empirical analysis chapters of this thesis. The literature review has four main sections.

2.1 Literature review outline

Section one (What is health?) attempts to compare and contrast different concepts of health, how it is defined and how the determinants are understood. This demonstrates the ambiguity of the meaning of health. It is important to outline definitions of health before discussing health inequalities.

Section two (Health inequalities) describes existing definitions of health inequalities. The distinction between health and health inequalities in this literature review aims to demonstrate that the determinants of population

health and the determinants of health inequalities may not necessarily be the same: the greatest gains in average population health may not always reduce inequalities.

Section three (empirical measurement of health inequalities) then outlines some of the theoretical explanations for the persistence and widening of health inequalities in economically developed societies. The theoretical explanations that are critically evaluated are those which have evolved from the Black report: artefact, health selection, social sorting, cultural and behavioural, materialist and structural, fundamental causes, psychosocial, and neo-materialist. The Black report was published in 1980 by the Department of Health and Social Security in the UK. It was produced by the working group on inequalities in health.

Section three ends with a critical evaluation of the traditional, rarely contested, concept of measuring health inequalities as differences in average health between socioeconomic groups. It highlights the theoretical and empirical limitations a traditional approach has for comparative research. It shows that applying a *total inequality* concept can help to overcome these limitations and can provide new insight into the changing nature of health inequalities. Lifespan variation is identified as one empirical measure which reflects the theoretical principles of total inequality.

Section four (Lifespan variation) summarises the existing studies which have used lifespan variation to measure health inequalities. Lifespan variation seeks to reflect the level of inequality in age at death that individuals and populations face, lower lifespan variation suggest less variability in age at death and lower

total inequality: individuals in the population are tending to die around a similar age. Although lifespan variation has been widely utilised in comparative studies few have explicitly studied this dimension of inequality in Scotland.

2.2 What is health?

In this first section I discuss different definitions of health. I begin by discussing how notions of health have evolved from biomedical and absolute interpretations to conceptualising health as a subjective state which has multiple determinants beyond the mechanisms that exist within the human body.

2.2.1 Interpretation of health within this thesis

The descriptions of health discussed in this literature review provide a basis for this thesis in the following ways. Firstly it shows that although a large body of research studying health exists there remains a lot that is unknown. Secondly it aims to demonstrate that health is a complex concept and that its determinants interact in a number of ways. This could suggest that the use of mortality data to capture health within this thesis may be inadequate. However, when the results examining health inequalities in Scotland are presented it will be evident that mortality is a valuable proxy for health: death data are being used within this thesis in order to operationalise a measure, a measure that reflects a complex social phenomenon as opposed to an isolated medically defined event.

2.2.2 Biomedical definition of health

The earliest notions of health saw it as the 'absence of physical disease or impairment' (Godlee, 2011). This definition reflects the biomedical discourse which Smith (2008) suggests is explicitly concerned with disease rather than

health. It is associated with the desire to uncover the aetiology of diseases within the human body and aims to make a distinction between sick individuals and healthy individuals (Rose, 2001).

A biomedical model is concerned with the problems of individuals and suggests that ill health is something which needs to be cured. Understanding health within this disease centred framework ensures that the focus is on the remediable aspects of illness and, in a clinical setting, aims to return individuals to the desirable disease-free state as soon as possible (Naidoo and Wills, 2015).

However, the biomedical model is a heavily criticised concept. This is because a disease centred approach does not account for what health means in the absence of disease or in the absence of a formal medical diagnosis (Yuill, 2010). Focusing on the absence of something that is objectively defined (e.g. a clinical diagnosis) frames health as what it is not, as opposed to what health is when it is interpreted subjectively (Barry and Yuill, 2012). For example individuals with objectively diagnosed diseases may never feel sick whilst individuals free from disease may experience feelings of sickness. It is important for concepts of health to focus on the protective factors that ensure good health, as opposed to focusing only on the risk factors that make individuals ill (Dahlgren and Whitehead, 2006). Therefore it is argued that a biomedical definition has limited scope because it focuses on health in negative and absolute terms and overlooks the broader determinants of health beyond medical care (Naidoo and Wills, 2015). In pragmatic terms, empirical research also regularly uses measures of health that are independent of clinical diagnosis (Kind et al., 1998).

2.2.3 The end of disease and the beginning of health

A more inclusive definition of health was constructed by the World Health Organisation (WHO). It aimed to communicate the notion that health is more than illness and disease, and is shaped by more than biological mechanisms and healthcare (Nettleton, 2006, Yuill, 2010). It defined health as a 'state of complete physical, mental and social well-being' (World Health Organisation, 1948). Alongside this definition the WHO proposed that the ability for all individuals to achieve well-being should be a consideration for all policies: responsibility for health should be shared between healthcare professionals and governments, policy makers and citizens themselves. The WHO definition is also valued for distancing the notion of health from only the negative concept of disease by incorporating 'well-being', a positive attribute of health (Barry and Yuill, 2012).

However the WHO definition has been the subject of some criticism because it continues to understand health as an absolute achievement rather than something which is relative. This critique is valid as meanings and expectations of health are dependent on beliefs and socio-political and cultural contexts. The attributes that are deemed to represent health in one society can easily be deemed to be unhealthy in another. Studies have also highlighted that individual interpretations of health vary across societies and across time in relation to the same clinical symptoms (Mitchell, 2005).

In fact, Smith (2008) suggests health is nothing more than an illusion. It is highly likely that everyone would be found to be diseased – either in terms of their physical health, mental health or social wellbeing – given that there are

thousands of diseases recorded and catalogued. This has prompted many to highlight that any notion of health as an absolute state will be unattainable for all of the population, all of the time (Godlee, 2011, Jadad and O'Grady, 2008). Rather very few would be found to be in a complete state of health as it is a social construct and cannot be understood or defined in absolute terms. In response many have advocated that health be conceptualised as a resource required for living rather than an aspirational end goal or a prescribed state of being that can be achieved (Kälin et al., 2004, Susser, 1993). This principle underpins the notion that health should be considered a basic human right.

2.2.4 Health as a human right

Health as a human right is another perspective which emphasises that the responsibility for health falls upon societies not healthcare institutions (Arcaya et al., 2015). Health as a human right can historically be linked to the argument that health should be framed as having 'great social and political value' which first emerged during the industrial revolution. Some of the earliest advocates of health as central to social development were, however, motivated by a utilitarian philosophy, not by the desire to improve health for health's sake (Susser, 1993). Asada (2005) further adds that the concept of health as a human right has previously been undermined because the dominant assumption has been that the ethics of health should only be concerned with individual patient-practitioner relations as opposed to population level dynamics.

Despite the contested origins of conceptualising health as a human right, societies have evolved to a perspective which attaches a higher value to health above the mere application of healthcare (Susser, 1993, Arcaya et al., 2015).

Proponents of health as a human right argue that health is essentially an ethical issue because the course of human development can be altered by decision makers (World Health Organization, 1998). This was strongly evidenced in United Nations, article 25 which formalised health as a human right in 1948 by stating that:

"Everyone has the right to a standard of living adequate for the health and well-being of himself and his family" (United Nations, 10 December 1948).

However this definition does not explicitly frame health as a human right but instead frames it in relation to the 'standard of living'. This perhaps reflects the ideological contention between the chain of causation of health: whether ill health causes poverty or whether poverty causes ill health (Susser, 1993).

It took until 1984 for the WHO to incorporate the notion of health as a human right when it argued that:

"Health is the extent to which an individual is able, on the one hand, to realise aspirations and satisfy needs: and on the other hand, to cope with the environment. Health is, therefore, seen as a resource for everyday life, not an object of living; it is a positive concept emphasising social and personal resources as well as physical capacities" (World Health Organization, 1984).

This revised WHO definition no longer sees health as a final outcome but as a requirement for living.

2.2.5 A state of equilibrium

Current debates surrounding the concept of health therefore propose that it is not a finite state but a state of balance individuals achieve within themselves,

and between themselves and their social and physical environment. Jadad and O'Grady (2008) propose that health should be defined as 'the ability to adapt and self-manage' and argue that health is something which exists in both the absence and presence of disease or impairment (Sartorius, 2006). Susser (1993) adds that understanding health as a human right seeks to reflect the notion that 'it is not a given, immutable fate'. This perspective allows for subjective interpretations of health and recognises that experiences of health vary every day.

Rather than understanding health as the absence of disease which is treated by medical intervention, framing health in these terms proposes that health be understood as a social necessity rather than an aspiration. Furthermore there is a conscious attempt to communicate the fact that healthcare is not the primary determinant of health: rather it sees health as having a relationship with multiple causes beyond biomedical mechanisms and structures (Naidoo and Wills, 2015). These causes include the physical and social environment alongside lifestyle and biological factors, which have been termed the social determinants of health.

Despite the evolution of concepts surrounding health a consensus has not yet been reached. Instead it is argued that opposing definitions of health should not be seen as mutually exclusive but rather used to demonstrate that understandings of health differ across contexts, time, situations and societies (Naidoo and Wills, 2015, Mitchell, 2005). These debates further emphasise the important distinction that needs to be made between the clinical care of individuals and public health of populations (Susser, 1993, Rose, 2001). Beckfield

and Krieger (2009) add that eco-social theories of health emphasise that the health status of a population is not fixed but is contingent upon societal, historical and ecological contexts.

Somewhat parallel to these evolving concepts of health are complex models seeking to identify the causes of health. These models go beyond biomedical cause and effect between vector-borne infections and disease outcomes. They are generally referred to as socioecological models.

Socioecological models are based on the theoretical principle that there is no single factor which can explain health: it is the outcome of interactions between many factors. They attempt to understand the ways in which societal conditions become embodied by populations. The following section will discuss three of these models: the epidemiological triad, the rainbow of determinants, and a life course model.

2.2.6 Modelling the determinants of health

Socioecological models seek to capture the hierarchy of social structures and the elements within systems that may influence health. Similarly to a doctor questioning a patient about their symptoms, family history and health behaviours when seeking to provide a medical diagnosis, these models question what aspects of our social and physical environment, and our responses to them, cause the patterning of health and disease. Models of causation aim to organise ideas and inform strategies for health promotion, health prevention or control of diseases (Bhopal, 2012). Dahlgren and Whitehead (2006) argue that socioecological models of health help to understand the root causes of health which are necessary if effective policies are to be formulated.

2.2.6.1 Epidemiological triad of causal factors

The most basic attempt at capturing the relationship between health and its wider determinants is the epidemiological triad of causal factors. The main idea underpinning this model, and many other models, is that health is a consequence of the interactions between the environment, the genetic or physical characteristics of the individual and the agent of disease (Bhopal, 2012). The aim of the triad was to communicate the notion of balance: all three elements need to be assessed to fully understand the causes of health in general, or the causes of a specific disease outcome. Figure 1 is a visual representations of the triad.

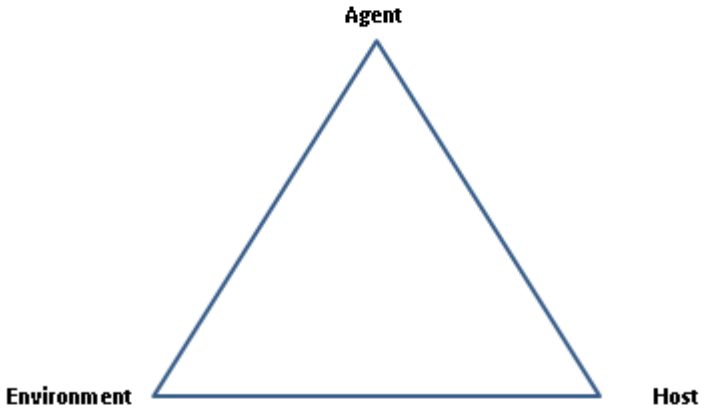


Figure 1 Epidemiological triad of causal factors adapted from (Bhopal, 2012)

The agent usually refers to the biological pathogen that must be present for the disease to occur, however its presence alone may not result in the disease outcome.

The host element highlights the risk factors that may make an individual more susceptible to the disease outcome.

The environment captures the elements which allow the opportunity for exposure to present itself or the environment which brings the host and agent together.

Although initially constructed to understand infectious disease, it is applicable when thinking about the determinants of non-communicable diseases. This is because it aims to identify the what (agent), who (host) and where (environment) of a specific health outcome.

Under this model ill health occurs because of an interaction between the agent and the vulnerable host within an enabling environment (Teutsch and Churchill, 2000). This concept was originally focused on infectious diseases such as the transmission of malaria (agent) from a mosquito (vector) to a human (host) (Centers for Disease Control and Prevention, 2012). However Bhopal (2012) gives the example of tuberculosis to demonstrate the wider application of the triangle model. Although tuberculosis has a single cause by definition, the tubercle bacillus, it has multiple determinants such as poor housing conditions (environment) or age (individual characteristic).Therefore over time the notion of what constitutes an agent has changed to include social, environmental and physical causes of diseases.

The application of the epidemiological triad to the social determinants is also possible. For example alcohol can be considered an agent of disease and injury (Bhopal, 2012). This is demonstrated in figure 2.

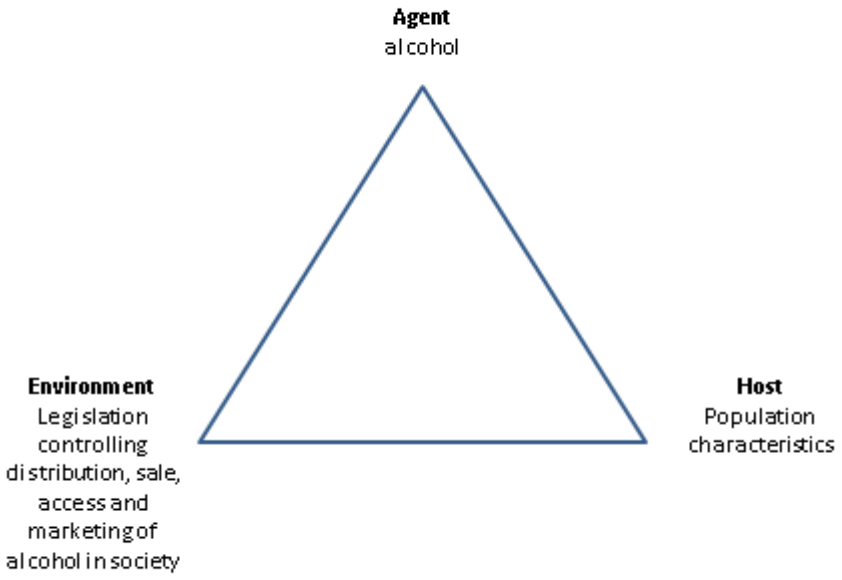


Figure 2 The application of the epidemiological triad to the social determinants of alcohol consumption adapted from (Bhopal, 2012)

Excessive alcohol consumption can cause liver cirrhosis and the manufacturing, distribution and marketing of alcohol are the vectors which bring together the agent (alcohol) and the host (the population) if the (social) environment enables it. Mapping the causes of liver cirrhosis in this framework can help identify different points of entry for public health policies seeking to reduce liver cirrhosis. For example minimum unit alcohol pricing is a legislative intervention for modifying the consumption of alcohol. It is a response to the evidence showing that alcohol harm amongst a population is related to the dose people

consume and the dose consumed is determined by price (Wagenaar et al., 2009). Minimum unit pricing aims to improve the social environment surrounding alcohol consumption via price regulation (Brennan et al., 2014, Purshouse et al.) in order to alter the link between the host (the population) and the agent (alcohol).

The epidemiological triad, although somewhat simplistic, successfully represents the different dimensions which influence health and attempts to untangle multi-dimensional relationships (Susser and Stein, 2009). However it is limited when trying to understand agents that may cause multiple health outcomes or health outcomes that have multiple causes rather than one cause. Bhopal (2012) adds that it is not always possible to conceptualise the cause of a disease as an agent and that the relationships between the host, agent and environment are rarely understood.

In response more advanced socioecological models continue to conceptualise the 'who', 'what' and 'where' of health but elaborate on the interacting relationships. One of the most widely recognised models to do this was the rainbow model of health introduced by Dahlgren and Whitehead (1991) shown in figure 3.

2.2.6.2 The rainbow of social determinants

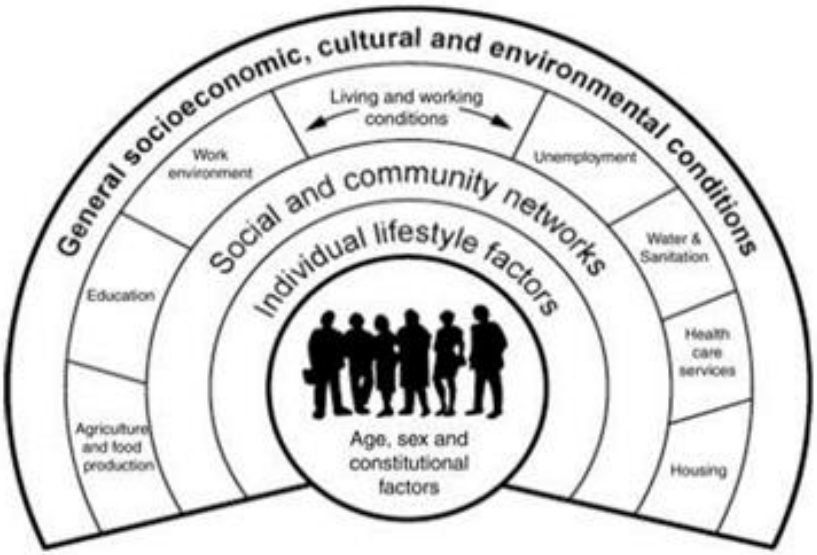


Figure 3 Rainbow model of health (Dahlgren and Whitehead, 1991)

Dahlgren and Whitehead (2006) state that the layers of the model should be interpreted as the main general determinants of health. At the centre are the characteristics individuals possess that can influence their health but are considered to be fixed when looking at one point in time, for example age and sex. Encompassing these are the interactions which, in theory, are modifiable by policy or public health interventions.

Individual lifestyle factors, such as exercise or smoking, are the positive and negative lifestyle factors which link with health outcomes.

Social and community networks may have an encouraging or discouraging influence on lifestyle factors.

Elements of living and working conditions can determine an individual's ability to maintain their own health.

Dominant economic, cultural and environmental circumstances exist in a given society and at a given point in time.

Dahlgren and Whitehead (2006) intended the model to be used to highlight the notion that the different layers are embedded and interact with one another. They argue that these layers can be influenced by the decisions made by individuals themselves but also by the decisions made by commercial or political stakeholders in society.

These decisions can impact on health by: influencing positive health factors, increasing protective factors, or reducing risk factors. Making a distinction between these three groups of determinants can be challenging. In general positive health factors are those which contribute to the maintenance of health such as economic or social security or emotionally beneficial relationships and control. Protective health factors are those which eliminate a risk. This could include immunisation programmes, the promotion of healthy eating or social support which encourages psychosocial health. Finally risk factors are the preventable causes of disease such as the physical, social and economic conditions which are detrimental to health. For example, the risk of poverty that may result from unemployment is a risk factor that is not inevitable as it can be mediated by social protection policies (Bambra, 2011c).

The rainbow model attempts to communicate the relationship between the social elements and physical or biological manifestations of health at a population level rather than at an individual level. It is powerful for mapping the

social determinants of health for specific populations at a given time. Its weakness is that it provides little insight into how health evolves over periods of time, how it is patterned over generations or how it is shaped by experiences at the earliest stages of life (Reid, 2009, Ben-Shlomo and Kuh, 2002).

2.2.6.3 Life course model of health

Much like the rainbow model a life course model provides insight in to biological, material, behavioural and psychosocial pathways (Ben-Shlomo and Kuh, 2002). However a life course model sees the biological characteristics of health as markers of an individual's past social position (Blane, 1999). It is concerned with the long-term effects of physical and social exposures that occur across the life course: before conception, gestation and childhood to adolescence, young adulthood and old age (Ben-Shlomo and Kuh, 2002). For example Barker and Martyn (1992) demonstrated that low birth weight was associated with coronary heart disease in adulthood. Framing this study within a life course context enabled the authors to argue that inadequate foetal nutrition may be an important risk factor for CHD like traditional adult risk factors such as smoking.

Ben-Shlomo and Kuh (2002) argue that research risks misinterpreting a life course model as merely the collection of exposure data across the life course when it should be applied to explicitly understand the temporal ordering of exposures and the interactions among exposures. It emphasises the importance of accumulation of advantage or disadvantage across the life and seeks to understand the social processes that aggravate or mediate the negative effects on health (Blane, 1999).

A life course model therefore provides a different approach for understanding where relationships between later life health outcomes and risk factors stem from. Figure 4 is one attempt for communicating that material, psychosocial and behavioural factors are mediating pathways linking social deprivation and health.

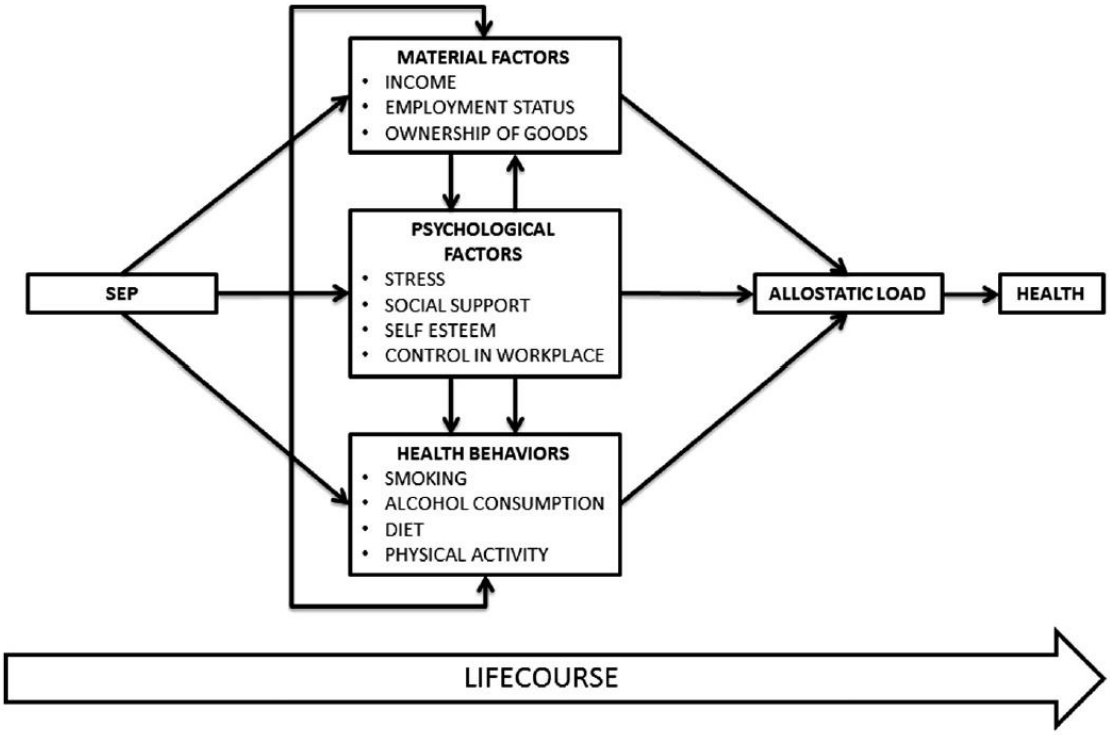


Figure 4 Life course model of allostatic load and material, psychosocial and behavioural factors (Robertson et al., 2015)

Ben-Shlomo and Kuh (2002) argue that the diagrammatical representation of variables over the life course should explicitly state the temporal ordering of exposures and their inter-relationships. This is the approach shown in figure 5 using the example of lung function. Although it captures the temporal ordering of factors specifically influencing lung function it has general applications for

understanding the principles considered in epidemiological research which adopts a life course approach.

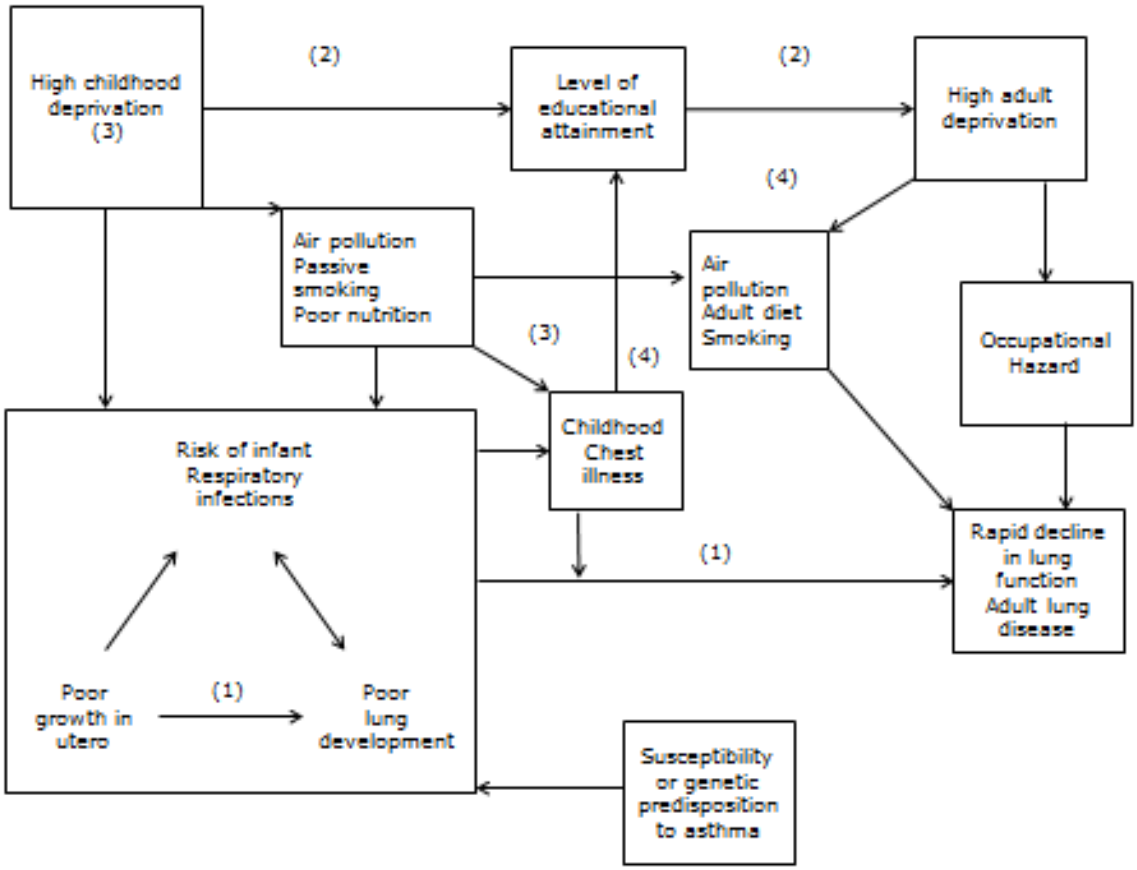


Figure 5 Life course model: temporal context and lung function (Ben-Shlomo and Kuh, 2002)

Figure 5 identifies four pathways:

(1) **Biological pathways** suggesting limited foetal growth is associated with decreased lung development, which over time causes respiratory problems later in life

(2) **Social pathways** that increase exposures which are detrimental for adulthood social circumstances such as high childhood deprivation being associated with lower educational attainment that has implications for future income or occupational opportunities

(3) **Sociobiological pathways** capturing the relationship between the social exposures that impact biological factors. For example high maternal deprivation may impact lung development in utero, lung function in childhood due to environmental exposures and adult function

(4) **Biosocial pathways** suggest that biological factors can have a detrimental impact on social processes. For example decreased lung function is associated with increased risk of childhood infections and lower education attainment as a consequence of school absence. Over the life course lower education attainment limits employment opportunities and individuals are more likely to be exposed to hazardous working condition. These hazardous conditions may be physical, environmental and or psychosocial.

Ben-Shlomo and Kuh (2002) recognise that this interpretation of the life course model is some-what crude. However it aims to highlight that distinguishing between social and biological pathways can be arbitrary and that both require attention to understand the causes of health.

The overview of competing concepts of health and models of the determinants alludes to the challenging nature of public health research. Jadad and O'Grady (2008) suggest that if health is too complex a concept to define operational measures of health may be unobtainable. However Godlee (2011) argues that this is why debating contrasting concepts is important: because they

fundamentally impact how we recognise health, how we interpret health and how we should attempt to measure it within research. Dahlgren and Whitehead (2006) further emphasise that the determinants of health be distinguished from the determinants of health inequalities because they are not conceptually the same. Confusing these may result in policies which are effective in improving levels of health but that are ineffective in tackling the health gradient.

2.3 Health inequalities

This second section focuses on different interpretations of health inequalities and some of the theoretical explanations that have been proposed to explain their persistence over time. This is relevant to this thesis for at least three reasons.

Firstly it demonstrates that health inequalities research should not be thought of as static but as a reflexive and evolving discipline. It needs to be responsive to new empirical evidence and the changing nature of health inequalities over time, and across different contexts.

Secondly it demonstrates the advancement within the discipline: from looking at absolute and material influences on health, disease aetiology and risk factors to introducing the psychosocial factors of relative deprivation into the theoretical framework (Bartley, 2004, Dowler and Spencer, 2007).

Thirdly it helps to understand the contested recommendation made by the World Health Organistation (2000): that health inequalities should be measured across individuals independent of socioeconomic group membership or irrespective of any other dimension of inequality. Lifespan variation is a measure which facilitates this conceptual approach and that has a number of pragmatic

advantages for carrying out comparative research. Critically evaluating this conceptual debate helps to demonstrate that there is no single approach for measuring health inequalities, and that different interpretations reflect different normative values. This chapter does not evaluate how we measure socioeconomic group membership. The theoretical and scientific reasons behind socioeconomic indicators are covered in detail in chapter 3 of this thesis.

2.3.1 Defining health inequalities

Although health inequalities have been documented across the world (Whitehead et al., 1992, Marmot, 2005, Mackenbach et al., 2008) no single working definition is adhered to.

One definition might refer to health inequalities as 'the observable or measurable differences in health' (Harper and Lynch, 2006). For example health differences that exist between countries can be seen as health inequalities. Sweden experiences high levels of population health and Scotland relatively low in comparison. Life expectancy in these countries is used to illustrate this in figure 6.

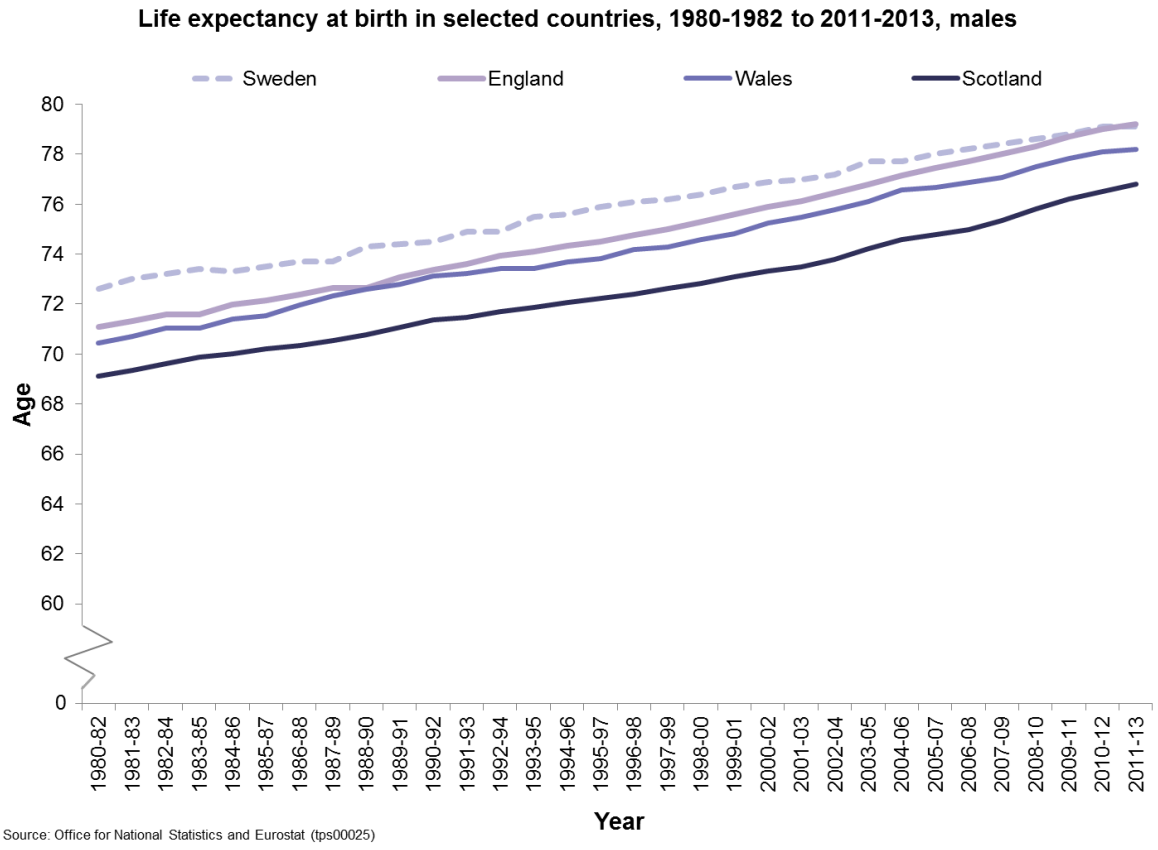


Figure 6 Comparisons of life expectancy trends in UK countries and Sweden (National Records of Scotland, 2014b)

Europe provides a valuable opportunity for comparative studies because of the range of political, economic and epidemiological histories which may help to identify opportunities for reducing health inequalities (Mackenbach et al., 2008). However, broad social ecological studies which measure differences in health across geographies may not strictly be studies of health inequalities as conceptualised by others (Bartley, 2004).

This is because an alternative definition of health inequalities states that they are the 'systematic differences in the health of people occupying unequal positions in society' (Graham, 2009). This perspective only deems differences in

health to be inequalities if they are patterned by a characteristic that reflect a socioeconomic inequality (McCartney et al., 2013).

Socioeconomic or demographic groups that are of interest may include gender, race and ethnicity, religion, sexual orientation, disability status, educational attainment or occupational status (Bartley, 2004, Marmot et al., 1991, Harper and Lynch, 2006). Table 1 illustrates the systematic gradient for life expectancy and deaths (age 0-64 years old) for males in Scotland in 2001 by socioeconomic deprivation as an example. This concept of health inequalities demonstrates that there is a social gradient for health that affects everyone (Marmot, 2005, Marmot et al., 2008). It is not simply that there is a gap between the richest and the poorest, although this is the starkest gap: even those almost at the top of the hierarchy experience worse health than those at the very top (World Health Organization, 2008, Marmot, 2001).

Table 1 Socioeconomic gradient for mortality in Scotland, 2000-2002, Males

		Life expectancy (years)	[1]Deaths age 0-64 per 100,000
Least deprived	1	77.7	176
	2	75.3	238
	3	73.9	302
	4	72.1	157
Most deprived	5	68.9	612

[1] (Leyland et al., 2007a)

2.3.2 Health inequalities or health inequities

Across disciplines the term health inequalities has been used to refer to measurable differences in health (Marmot, 2005, Bartley, 2004, Arcaya et al., 2015). However, it has been suggested that the term health inequality may not adequately reflect the extent to which a health difference is unjust, preventable and unnecessary (Arcaya et al., 2015, Marmot et al., 2012).

The example that is often given is that health differences by age are measurable but may not be unjust. A 20 year old will not have the same health as a 60 year old but the 60 year old will have previously experienced the health of a 20 year old and the 20 year old will one day experience the health of a 60 year old (Kawachi et al., 2002). In contrast Arcaya et al. (2015) suggest that health inequality is a term which is applicable in any circumstance where any quantity of health is unequal, but the term health inequities implies that a moral judgement be passed about whether the inequality is just.

Kawachi et al. (2002) highlights that health inequity requires a further distinction to be made between inequalities *achieved in health* and inequalities in *opportunities for health*. For example the consequences of smoking may be seen as an inequality but not an inequity: the individual smoker, potentially, had the same opportunity to achieve the same health as a non-smoker. However there are contrasting view points for where responsibility for health is situated. It could be argued that individuals should be held accountable for smoking or it could be argued that individual free choice is limited by the environment and by predisposing factors that lie outside of individual control (e.g. pricing, market availability, tobacco control legislation and health education). Health inequity

therefore has a strong normative dimension (Fabienne, 2001) and there is no full account of which health inequalities are distinguishable as unjust (Kawachi et al., 2002). Fabienne (2001) argues that this is problematic when trying to identify which normative judgement to pass when interpreting empirically measured health inequalities. It also risks being an affront to many people's sense of fairness and justice (Benzeval, 1995, Kawachi et al., 2002, Sen, 1992). Therefore a health inequity theoretical framework is a problematic objective for policies to pursue (Sen, 2002).

Instead it is proposed that health should be understood as the most important condition for human life: it is a basic, common need and right (Kawachi et al., 2002, Sen, 2002). This interpretation accepts that the distribution of health should be fair but that this does not require judgement to be passed. Health as human right does not discredit the valuable insights an equity argument presents. Instead it recognises that all health inequalities should be deemed unjust as they all tend to reflect fundamental differences in the distribution of resources (Kawachi et al., 2002, Fabienne, 2001).

In summary critically evaluating the concept of health inequity can act as a catalyst for specifying what it is in society that needs to be equalized (Sen, 2002). I chose to use the better established term health inequalities rather than health inequities throughout this thesis. The term health inequality does not inherently demand that a moral judgement be passed. The following section will discuss the theoretical explanations for health inequalities.

2.3.3 Theoretical explanations

Bartley (2004) suggested that the existence of health inequalities, as evidenced by a mortality gradient, came somewhat as a surprise when the Black report was published for at least two reasons. Firstly, the UK population had had access to free healthcare funded entirely by general taxation for over 30 years. Secondly, improvements in income were perceived to have been large enough so that poverty no longer directly impacted poor health and mortality. This prompted some to comment that a welfare state founded on progressive taxation and public service investments may be ineffective for reducing health inequalities. The Black report did not accept this perspective and instead proposed four explanations for the existence of health inequalities: the artefact explanation, health selection, cultural/behavioural and materialist/structural.

In the years since the Black report this fourfold classification has been heavily debated, a number of modifications presented, and several alternatives proposed (Mackenbach, 2012, Macintyre, 1997, Bambra, 2011a). Attempts at synthesising and disentangling these theories have also been made. For example Macintyre (1997) set out to distinguish between hard and soft interpretations of each theory. Mackenbach (2012) grouped a number of theoretical developments in terms of whether they focused on: (1) social mobility and the composition of socioeconomic groups over time, (2) the distribution of resources (material and psychosocial), (3) the ability for resources to enable health improvement (Grusky, 2010). Further critical reflections have focused on the ability for each theory to explain the *persistence* of health inequalities over time, against their ability to explain the *widening* of health inequalities over time (Mackenbach, 2012, Bambra, 2011a).

The following sub-section summarises these theories in terms of the Black reports' broad fourfold classification but adds in some more recent developments where appropriate. It does not provide an assessment of the validity of each of these theories, nor does it provide a full account of empirical studies which have tested them. Rather, it seeks to demonstrate that there is no single theoretical explanation for health inequalities: theories co-exist, stimulate critical development in one another, and are not mutually exclusive.

2.3.3.1 Artefact

Firstly the artefact explanation suggested that the measures used to capture social deprivation or social status may have no empirical significance (Townsend and Davidson, 1992, Fox et al., 1985). It was argued that empirical measures failed to reflect the decreasing proportion of the population in the lowest social class category. Therefore the health gap could be interpreted as an inevitable consequence of social mobility within the UK if upward mobility improved the health of the majority relative to those left behind. Townsend and Davidson (1992) were quick to discredit this perspective. It was reported that the reduction in the size of the lowest social class was relatively small. In addition this did not account for why the health of those near the top of the hierarchy was worse than those at the very top (Marmot, 2001). Therefore it was concluded that any potential measurement artefact would have had a minimal impact on the magnitude of health inequalities that were reported (Fox et al., 1985).

2.3.3.2 Health Selection

Secondly a health selection explanation proposed that the social structure of society reflected the health structure: healthy individuals experience upward social mobility and sick individuals find themselves in the most disadvantaged groups (Bartley, 2004). This interpretation of health selection flips the causal relationship: social position is dependent upon health rather than social position having implications for health (Townsend and Davidson, 1992, Boyle et al., 2009b). To overly simplify this scenario, health is the causal variable and occupational class is the outcome variable. It was strongly linked to Darwin's notion of natural selection, with Macintyre (1997) highlighting that 'natural' has both biological and moral connotations when referring to health inequalities.

The lowest infant mortality rate experienced by the highest social class was used as supporting evidence for this hypothesis: it was perceived to be a reflection of the fact that this sub-group of the population was comprised of the fittest and healthiest men and women (Fox et al., 1985). As a result it is hypothesised that health inequalities, between socioeconomic groups, could have widened as a consequence of healthy individuals experiencing upward social mobility and unhealthy individuals experiencing downward social mobility (Boyle et al., 2009b).

2.3.3.3 Social sorting

Earlier interpretations of selection effects saw health as a direct explanation for socioeconomic position (e.g. reverse causality). Later approaches saw selection effects as reflecting the relationship between health and socioeconomic position, which is confounded by many other factors (Mackenbach, 2012). For

example the argument that psycho-social and behavioural changes associated with downward selection are detrimental to health rather than health being the underlying process causing downward selection (Fox et al., 1985).

Although a social selection theory is plausible there is no consensus surrounding the potential impact it could have had on health gradients or how it can be incorporated into strategies for reducing them. Some of the debates have centred around whether the comparison of mobile and immobile populations is most appropriate for the health differences between areas or if the net difference between in and out flows provides more explanation (Boyle et al., 2009b, Norman et al., 2011). Further studies have critically evaluated whether increasing social mobility should be considered a viable policy option for reducing health inequalities (Boyle et al., 2009b).

2.3.3.4 Cultural and behavioural explanations

The third explanation suggested that health inequalities persisted because of different shared cultural practices that impacted health behaviour. There is large body of research findings documenting systematic differences in health behaviours across the social hierarchy: from the consumption of fats, sugars, and salt to exercise, smoking, and drinking (Lahelma et al., 2010, Laaksonen et al., 2008). Furthermore these behaviours can be clearly identified along the causal pathways in the onset of disease and death.

However Townsend and Davidson (1992) point out at least four reasons why health behaviours, by themselves, were an inadequate explanation. Firstly, they argued that empirical research capturing health behaviours need to recognise that health behaviours are only indicators of health which have limited

interpretation out of context. Secondly, a logical economic explanation for the social patterning of negative health behaviours is often missing when emphasising individuals as independent and autonomous decision makers in relation to their health (Bartley, 2004, Dowler and Spencer, 2007). Thirdly, was the prominent finding from the Whitehall study that health behaviours were unable to fully account for the mortality gap between social groups. Only 25% of the risk of death between occupation grades was due to the observed health behaviour patterns (Marmot et al., 1991). Fourthly types of health behaviours have changed over time, becoming more or less predominant, but the mortality gradient has remained (McCartney et al., 2013).

Despite all of this, commentators point towards the tendency for Government initiatives to rest upon the assumption that individuals act autonomously in relation to their health even though evidence refutes this (Dowler and Spencer, 2007, Department of Health., 2005, McCartney et al., 2013). Bartley (2004) argues that studies applying a cultural and behavioural framework tend to overly focus on behaviour as the cause, that behaviours are the responsibility of the individual, and that behaviours are 'freely taken' by individuals. Seeing health behaviours as determined entirely by individual choice removes any sense of inequity. Therefore a health behaviours perspective contributes to our understanding of the socioeconomic gradient but they do not fully explain its persistence or its steepening over time. Instead it is argued that this perspective should be used as a catalyst to push back the explanatory framework to help understand why differences in health behaviours exist and question the social contexts which they exist in (Macintyre, 1997). This task was better addressed within a materialist/structural hypothesis.

2.3.3.5 Materialist / structural explanations

A materialist /structural explanation seeks to understand the impact economic and social resources have for explaining the distribution of resources that determine health and mortality (Macintyre, 1997, Mackenbach, 2012). It states that health inequalities result from the unequal accumulation of exposures and experiences that stem from the material inequalities (Lynch et al., 2001).

It is not difficult to decipher why material poverty would have previously explained the burden of mortality on the lowest socioeconomic groups: historically overcrowded and poorly sanitised living conditions experienced by the lowest socioeconomic groups fuelled the spread of infectious disease (Link and Phelan, 1995, Phelan et al., 2010). It is more challenging to understand why this burden has persisted in capitalist economies that have driven economic growth, reduced over-crowding, improved sanitation, enabled material gains in wealth, and lowered absolute mortality rates (Townsend and Davidson, 1992, Bartley, 2004).

Although the Black report introduced this hypothesis by discussing exploitation, poverty and disease as causes of health inequality, Macintyre (1997) highlights that greater emphasis within the report was placed on clarifying material versus materialist interpretations.

A material interpretation assumes that material resources only impact health if access to resources falls below optimal condition. Under this interpretation excessive lifestyles only afforded by the rich may actually be seen as damaging to health (e.g. 'diseases of affluence'), still the highest socioeconomic group experience the lowest level of mortality (Townsend and Davidson, 1992).

Therefore a strict material perspective is flawed because it fails to see poverty in relative terms to the current level of economic prosperity or the prevailing social norms of society (Townsend, 1987, Townsend, 1979).

Materialist interpretations of poverty recognise that poverty is not a static entity but a relative concept that changes within different contexts (Townsend, 1987). Inequalities in access to material resources have remained a universal problem throughout history (Mackenbach, 2012) and, as societies have developed, new opportunities for inequalities have emerged. This is somewhat supported by the fact that health inequalities have remained despite radical changes in the diseases and risk factors assumed to explain them (Phelan et al., 2010). The evolving nature of new opportunities for inequality was summarised by (Link and Phelan, 1995) in their 'Fundamental causes' theory.

2.3.3.6 Fundamental causes

It was proposed that the persisting socioeconomic gradient for health, and ultimately death, were the consequence of the resources embodied within socioeconomic groups: money, knowledge, power, and social capital. These resources are beneficial for health against any prevailing disease profile in society and help to negotiate all types of mechanisms (Phelan et al., 2010). In addition mortality remains strongly patterned by material resources because the types of resources that may drive health improvements or enable individuals to negate risks are not finite: for example there is no absolute level of wealth, knowledge or education that can be achieved (Townsend and Davidson, 1992). The gradient in material welfare and health is therefore a consequence of the

most advantaged social groups disproportionately gaining more resources that enable them to improve health and experience the greatest fall in deaths.

Subsequently, the position of the most disadvantaged has become relatively worse off over time, but within the context of absolute improvements, meaning it may actually be harder to partake in the socially accepted functions of a richer society (Sen, 1992, Townsend and Davidson, 1992, Bartley, 2004). In this context Sen (1992) argued that relative material deprivation can lead to absolute deprivation in terms of *capabilities:* the ability to transform material resources into valuable activities via real freedom and equal access to opportunity. Bartley (2004) reiterates this by concluding that a materialist approach does not see the absence of enough material wealth as impacting health but that balancing psycho-social needs and biological needs is now dependent upon material wealth.

Therefore the distinction between a material and materialist hypothesis tends to be that money itself is not the causal factor impacting health but that the level of relative resources and the ability those resources provide to mitigate risks are (Bartley, 2004). Marmot and Wilkinson (2001) summarise that the difference is between the damage done by absolute poverty versus the health implications of relative inequality.

This theoretical framework clearly conveys the notion that poverty in modern societies is more related to social participation than bare minimum survival (Dowler and Spencer, 2007). The materialist framework for understanding health inequalities has also evolved to encompass different mechanisms, in particular

psychosocial resources and social protection policies under a neo-materialist perspective.

2.3.3.7 Psychosocial

Psychosocial mechanisms aimed to provide a biologically plausible account for why health is socially patterned: which in turn leads to the mortality gradient (Dowler and Spencer, 2007). Within this theoretical framework psychological conditions, which are seen as a direct consequence of social inequality, also have a direct impact on the body's biological processes.

Research evaluating the possible psychosocial mechanisms was initially motivated by the so called 'fight or flight' changes that happen in response to external stimuli that are perceived to be dangerous. Psychological mechanisms which link social exposure and physiological response may be worry, stress, anxiety, insecurity, depression and vulnerability (Artazcoz et al., 2005, Marmot and Wilkinson, 2001). Studies have demonstrated that perceived levels of stress or acute mental stress (psychosocial mechanisms) are associated with heightened cardiovascular responses and increases in blood pressure (biomedical responses) (Carroll et al., 2011, Light et al., 1999).

Psychological conditions, triggered by the social environment, might interact with the biological processes which facilitate the adoption of negative health behaviours. One example that links to health behaviours is that biological response to addictive substances may be altered under different psychological conditions (Bartley, 2004). This example takes account of the distribution of adverse social experiences, the distribution of health behaviours, and

subsequent distribution of health outcomes because social exposures are seen to be embodied in our psychology and biology.

Wilkinson and Pickett (2010) argue in favour of psychosocial mechanism by stating that health is not only damaged by material conditions but by our *responses* to our social circumstance. Their understanding of social circumstance was explicitly referring to the level of social stratification and the hierarchical structure of society at a macro level (Wilkinson and Pickett, 2006). They suggested that the distribution of wealth within a country may be more important for health than the absolute level of wealth and that this acts through psychosocial mechanisms (Graham, 2009, Wilkinson and Pickett, 2010, Wilkinson and Pickett, 2006). However it is still a theory that has been the subject of critical evaluation.

In a similar vein to critiques of health behaviour theories, Lynch et al. (2000) argue that psychosocial theories can be unintentionally decontextualized, risk being misappropriated with regressive political agendas, focus on the perception of inequality detracting attention from structural causes, and reinforce low expectations for structural change. Marmot and Wilkinson (2001) dispute this interpretation: social structure and relative deprivation have profound psychosocial impacts and to deny this burdens the responsibility for depression, anxiety and insecurity with individuals. They further emphasise that psychosocial mechanisms are important for clarifying a materialist perspective from a neo-materialist perspective.

2.3.3.8 Neo-materialist

A neo-materialist theory sees inequalities in material wealth as reflective of the negative exposures and lack of resources held by individuals due to systematic inadequacies in the social infrastructure (Lynch et al., 2000). The relationship between lower social inequality and better population health may be explained by the psychosocial benefits provided by an egalitarian welfare system that ensures higher levels of social protection for all of its citizens (Bambra, 2011a, Bambra, 2013).

Social protection policies are a means of redistributing wealth. They generally aim to protect the most vulnerable members of society from the effects of social and economic risks (Bambra, 2009, van Raalte et al., 2014). This approach suggests that high income inequality in itself is not problematic. Rather a country could demonstrate high income inequality without there being implications for health if other aspects of the social infrastructure improved.

This is somewhat of a contrast to a strictly psychosocial explanation for health inequalities where income inequality is always problematic because perceptions of relativity will always exist (Lynch et al., 2000). A neo-materialist view still recognises the importance of relative deprivation but sees the redistribution of materialist mechanisms as the solution for eradicating mortality inequalities: the redistribution of income and resources through tax, cash and non-cash benefits (Mackenbach, 2012).

There is a growing body of research which supports a neo-materialist hypothesis by demonstrating the links between welfare redistribution, social protection policies and lower mortality (Marmot et al., 2008, McCartney et al., 2016).

Perhaps the most widely cited examples are Scandinavian countries which tend to afford high levels of social protection to the unemployed and retired members of society but also ensure that publicly provided health care and education are of a high standard for all (Bambra, 2011a, Popham et al., 2013).

Access to these resources in these societies is independent of individual socioeconomic position explaining why there might be lower levels of health inequality (Marmot et al., 2008). This is often contrasted to the social structure in the USA where access to the resources which are required for social participation is more dependent upon individual socioeconomic position. It is perhaps not surprising then that the USA demonstrates one of the steepest mortality gradients of any economically developed country (Singh, 2003, Hummer and Hernandez, 2013).

However empirical evidence from Mackenbach et al. (2008) did not entirely agree with this theoretical approach. They find that the magnitude of health inequalities, when measured across socioeconomic groups, was not systematically lower in countries which have a history of egalitarian welfare policies. From this finding they concluded that a certain level of social protection may mitigate health inequalities but may not provide a universal explanation for their reduction across different countries.

Somewhat in contrast to this study Popham et al. (2013) demonstrated that inequalities in countries with egalitarian welfare systems could be interpreted as the smallest when measured using lifespan variation. This is considered a measure of total inequality as it reflects the distribution of health across all individuals rather than the difference in average health between socioeconomic

groups. The seemingly contradictory conclusions drawn from these two studies reflect the fact that different approaches for measuring inequalities were taken.

The differences between these two approaches can be debated in terms of: (1) the underlying normative judgements and (2) the pragmatic challenges. These are discussed in the third section of this literature review chapter.

2.4 Conceptual meaning and empirical measurement of health inequalities

It has become somewhat standard practice for quantitative research to strictly conceptualise health inequalities as the differences between socioeconomic groups (Marmot, 2001, Popham et al., 2013). Quantifying inequalities in this way has been widely utilised for setting policy targets. Scotland, for example, set out to improve health by 15% for the most deprived socioeconomic group as measured against coronary heart disease, smoking and suicide indicators (Munoz-Arroyo and Sutton, 2007).

However it is widely recognised that standard definitions and conceptual measurements of socioeconomic position are problematic (Mackenbach et al., 2016b). It is challenging to construct measures of socioeconomic inequality that adequately capture determinants of health that affect everyone or that are able to reflect the relative experiences that exist across an entire population (Munoz-Arroyo and Sutton, 2007). This has led to a lack of consensus among researchers and policy makers about which dimensions of socioeconomic inequality to measure progress against (Mackenbach et al., 2016b, Kawachi et al., 2002, Arcaya et al., 2015).

2.4.1 Normative judgements and pragmatic challenges

The underlying normative assumption driving the traditional measurement of inequalities is that differences in health are only inequalities if they are patterned by a socioeconomic inequality which renders the health difference distinctly unequal (McCartney et al., 2013). However, the normative judgement underpinning traditional concepts of health inequality presents a number of pragmatic problems (Popham and Boyle, 2010, Mackenbach et al., 2008). These can be summarised in reference to two commonly utilised measures of socioeconomic deprivation: educational attainment and occupational social class (van Raalte et al., 2011, Mackenbach et al., 2008).

Historical changes and cultural differences in terms of the socioeconomic position afforded by a certain level of education or an occupation restrict comparisons between countries and over time. The occupational structure of a society has changed over time (Macintyre, 1997) and the social and monetary value of an occupation is context specific. In a similar vein the educational profile of populations can be altered over a generation making temporal comparisons challenging and educational standards and practices vary between countries (Mackenbach et al., 1997, Krieger et al., 1997).

Mackenbach (2012) adds that incomparable socioeconomic data may impact the empirical results if variability in the data collection processes between countries affects the reported differences in the magnitude of inequalities. Dibben and Popham (2011) also suggest that the composition of socioeconomic groups within countries is fundamentally a product of the welfare structure in each country. This means that studying socioeconomic differences in health within countries,

and then making comparisons with contrasting countries, may not be appropriate for understanding the impact of different welfare systems on health inequalities. They present this argument in reference to the level of meritocracy within each country: if there is a link between health and intellectual ability that is independent of socioeconomic position then a meritocratic country would have a concentration of health in the highest socioeconomic group. This would be expected to result in steeper health gradient than a non-meritocratic country where ability and health are distributed more evenly between socioeconomic groups because the opportunity for social selection based on intellectual characteristics is restricted (Mackenbach, 2012). Despite these limitations, comparisons over time and between countries are deemed essential for assessing the effectiveness of social, political and economic initiatives that governments implement in order to reduce health inequalities (Murray et al., 1999).

One proposed solution is to instead measure total inequality. This interpretation of inequality does not require any socioeconomic data to be operationalised. This gives it an immediate pragmatic advantage over a traditional interpretation of inequality as the difference between socioeconomic groups (Popham and Boyle, 2010, Dibben and Popham, 2011, Le Grand, 1987, Harper and Lynch, 2006).

2.4.2 Total inequality

The conceptual meaning of total inequality is perhaps best demonstrated by comparing it with one of the most common traditional approaches used for monitoring health inequalities: the reporting of differences in average life expectancy between socioeconomic groups (World Health Organization, 2010,

National Records Scotland, 2011). Life expectancy at birth reflects the average number of years a new born can be expected to live for if the current mortality conditions of the population were to remain (World Health Organization, 2010). Although life expectancy at birth continues to be used in comparative studies, some have suggested that remaining life expectancy at adult ages may be a more appropriate indicator of inequality because infant deaths are now so rare in economically developed countries (Tuljapurkar, 2010, Smits and Monden, 2009). Despite this contested issue life expectancy at birth continues to be one of the most common indicators of population health (Murray et al., 2002, World Health Organization, 2010).

However a difference in life expectancy, between socioeconomic groups, fails to indicate a distinct dimension of inequality: differences in the distribution of age at death. Measuring the distribution of an outcome is common practice in other disciplines such as the study of income inequality in economics. The distribution of income across individuals is deemed to be essential as comparisons demonstrating similar averages may do so with different distributions (Murray et al., 1999). Therefore if average levels of income are not deemed informative enough then neither should average levels of health. Tuljapurkar (2010) adds that distribution of age at death should be considered a primary outcome variable in itself, much like the distribution of income can be.

The normative principles underpinning a total inequality perspective is that health, by itself, is fundamental to well-being and there should be a concern for inequalities in health regardless of whether it is correlated to any other dimensions of socioeconomic inequality (Tuljapurkar, 2010, Gakidou and King,

2002, Murray et al., 1999). This approach for measuring and comparing health inequalities was supported by the WHO when it proposed that the distribution of health across individuals, regardless of socioeconomic group membership, be monitored (World Health Organistation, 2000, Asada, 2005, Murray et al., 1999). Despite the potential for total inequality measures to provide additional insights in to the changing nature of health inequalities it has been met with criticism (Kawachi et al., 2002).

Opponents have particularly focused on the normative principles driving a total inequality approach arguing that it: removes the fundamental principle of injustice, that it measures inequalities that are not inequities, and simply reflects health differences or health disparities (Marmot, 2001, Dibben and Popham, 2011). It is assumed that if no dimension of socioeconomic inequality is measured then the socioeconomic factors amenable to interventions are being overlooked (Carter-Pokras and Baquet, 2002).

Navarro (2000) further points out that this approach has been endorsed by the WHO which is not primarily a scientific institution but a political one, whose position must be assessed accordingly. Carter-Pokras and Baquet (2002) similarly add that differing definitions used for health inequalities may reflect opposing political ideologies and have implications for resource allocation.

A counter argument is that a traditional approach risks ignoring important individual variations and the underlying array of health patterns (Murray et al., 1999): measuring the average health differences between socioeconomic groups risks assuming that a homogenous gain in health has been achieved for everyone

within the same socioeconomic group, or that health inequalities within these groups have stayed constant over time.

Dibben and Popham (2011) summarise this debate effectively: If total inequality were to reflect only random variation in health that is not unjust then the level of total inequality should be comparable between countries (and between socioeconomic groups). This is not the case (Smits and Monden, 2009, Shkolnikov et al., 2011, Seaman et al., 2016a) and *the difference in total inequality* should be understood as a consequence of inequity. Inevitable and unavoidable differences would be randomly distributed across individuals (e.g. not socially patterned) if there was no intervening social process at play. Therefore it is not only a traditional approach that can measure whether a health difference is unnecessary or unfair, total inequality can too.

2.4.3 Multiple dimensions of inequality

However the two approaches do not need to be interpreted as mutually exclusive. Instead Harper and Lynch (2006) suggest that contrasting the normative assumptions of the two approaches may help to highlight the underlying intentions of health inequality initiatives: whether the goal is to improve absolute health for everyone or to challenge relative inequalities. Gakidou et al. (2000) add that the debates surrounding the quantification of health inequalities should be concerned with: (1) explicitly defining quantities of interest and (2) justifying why they are of interest.

2.5 Lifespan variation

This thesis therefore recognises that opinions are divided when it comes to the conceptual meanings and measurement of health inequality. However a growing

body of research has provided insight into the changing nature of health inequalities by estimating and interpreting the relativity of total inequality. One of the most common measures used to do this is lifespan variation.

Lifespan variation is the variability in age at death that exists between all individuals within a population and is considered complimentary to average life expectancy. Multiple indices of lifespan variation exist and capture the degree of mortality compression in a population (van Raalte and Caswell, 2013, Tuljapurkar, 2010). Lifespan variation, like many indicators of population health and inequality, only conveys meaning when it is reported comparatively (Rose, 2001). A lifespan variation of 10 years experienced at one point in time, in one country, or by one socioeconomic group, can only be interpreted as favourable or unfavourable by making appropriate comparisons. Higher lifespan variation equates to greater inequality in the age at death (Murray et al., 1999, Gakidou and King, 2002, Harper and Lynch, 2006, Smits and Monden, 2009).

International studies have compared population level trends for lifespan variation over significant periods of time, compared lifespan variation trends stratified by socioeconomic position, and investigated the level of lifespan variation inequality independent of life expectancy (controlling for epidemiological time) (van Raalte et al., 2014, Smits and Monden, 2009, Gillespie et al., 2014).

2.5.1 Epidemiological transition

The theory of epidemiologic transition focuses on the changing patterns of health, disease and mortality and the associated demographic, economic and sociological changes. It proposes that disease intervention programmes may be a

determining factor for fertility change and for socioeconomic progression (Omran, 1971).

Mackenbach et al. (1997) suggest that studies seeking to understand the determinants of health inequalities should therefore control for epidemiological time rather than calendar time. This involves comparing populations when they achieve the same average health rather than comparing populations in terms of contemporaneous time.

The notion of epidemiological time has been highlighted in wider health inequalities literature as being particularly important. It is an approach that can be applied in order to control for differences between countries if they are known to be at different stages of a disease epidemic (e.g. cardiovascular disease (Vallin and Meslé, 2004)) or to control for any lag time that may be associated with diffusion of knowledge (Mackenbach, 2012, Smits and Monden, 2009). For example, it would be valuable to control for stages of the smoking epidemic or control for stages of the cardiovascular revolution (interventions introduced to control risk factors for cardiovascular disease) when comparing populations. These epidemiological developments started at different points in time for different countries and effects were socially patterned (Mackenbach et al., 2008, Vallin and Meslé, 2004).

Measures of total inequality facilitate multiple comparisons of inequalities: over time, between populations, and when controlling for epidemiological time. All of these comparisons will be made in the analysis chapters of this thesis. Traditional interpretations of inequality, as the average health difference between socioeconomic groups, are less capable of making all of these

comparisons because they require consistent measures of socioeconomic position (Mackenbach and Kunst, 1997). The literature review will now summarise some of the international empirical studies that have applied lifespan variation as a measure of inequality and identify the research questions that this thesis will answer.

2.5.2 International studies of lifespan variation

A number of international studies have applied a total inequality concept of health inequalities by measuring lifespan variation (Tuljapurkar, 2010, Shkolnikov et al., 2011, van Raalte et al., 2011, Smits and Monden, 2009, Gillespie et al., 2014). Lifespan variation seeks to reflect the level of uncertainty that surrounds life expectancy in a population, lower lifespan variation suggest less variability in age at death as individuals in the population are tending to die around a similar age. Being able to measure the level of uncertainty in age at death has implications for public sector investments in areas as diverse as: education, healthcare, retirement, and pensions (Gillespie et al., 2014). If individuals are informed about the level of certainty surrounding their expected age at death then they may be better positioned to make personal decisions about future finances and health (van Raalte et al., 2014).

Smits and Monden (2009) use three example countries to illustrate what is meant by lifespan variation. They compare Niger, Brazil and Japan in the year 2000: three countries at contrasting stages of development and therefore experiencing different distributions in age at death. Comparing these three countries demonstrates the standard processes of mortality development: falling childhood deaths, increasing life expectancy, emergence of a single modal age at death for

the population, and decreasing lifespan variation (Gillespie et al., 2014). Figure 7 shows how lifetable deaths are distributed across all ages in the three countries.

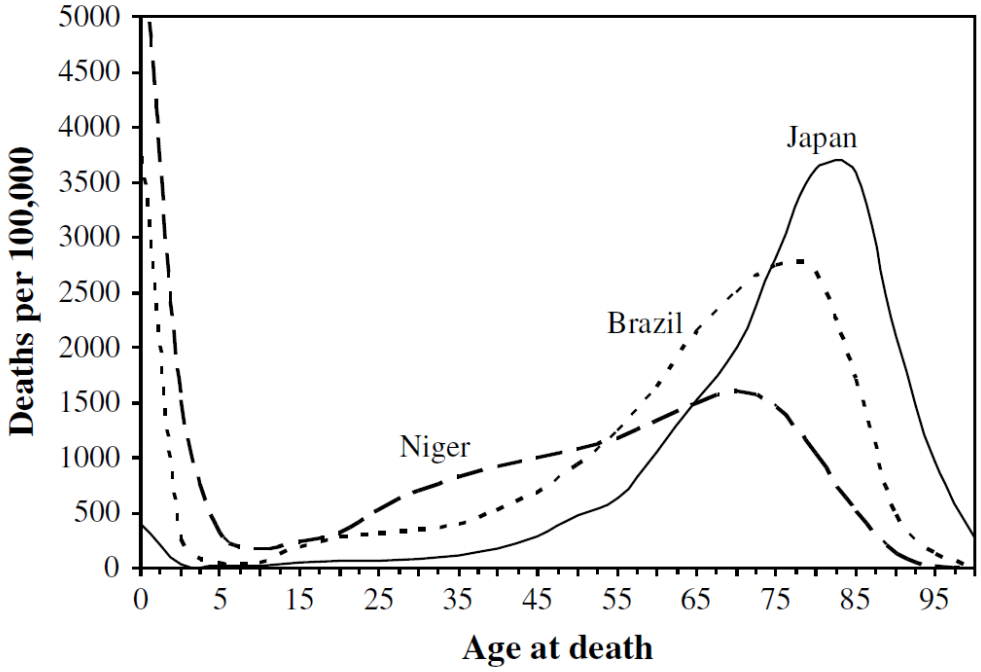


Figure 7 Age distributions of mortality, contrasting three countries at different stages of epidemiological transition (Smits and Monden, 2009)

It is evident that the greatest proportion of deaths in Japan is between the ages of 70 to 90 years old. This means the majority of the population would be expected to reach these ages if they were exposed to the same mortality conditions. This is in contrast to Niger and Brazil where infant mortality remains high and a smaller proportion of the population live to older ages. Hence in Japan there is less variation in age at death, which can be interpreted as lower inequality, whilst Niger experiences the greatest variation in age at death.

Although a number of indices of lifespan variation exist these are found to be highly correlated (van Raalte and Caswell, 2013). One of the most commonly used is *e*† (van Raalte and Caswell, 2013, Shkolnikov et al., 2011). *e*† is the average years of life lost per death in the population; it reflects the average remaining years of life expectancy when a death occurs (van Raalte, 2011a).A technical discussion of *e*† is included in the methods chapter of this thesis.

2.5.3 Compression and expansion: maintaining the association between life expectancy and lifespan variation

Historical trends in life expectancy have been well documented and a convergence in life expectancy at birth found for most developed countries (Edwards, 2011, McCartney et al., 2012b). Increases in life expectancy have historically been driven by steep mortality decreases at younger ages, outpacing mortality decreases at older ages (Shkolnikov et al., 2011, Gillespie et al., 2014).

As a result the countries with the highest life expectancy at any given time have tended to have the lowest level of lifespan variation (Shkolnikov et al., 2011, Edwards, 2011). This strongly suggests that increasing average population health and decreasing levels of total inequality are compatible public health aims (Edwards and Tuljapurkar, 2005, Shkolnikov et al., 2011, Smits and Monden, 2009).

Although improvements in life expectancy have almost universally continued (with Russia and some Central African countries being noted exceptions (Notzon et al., 1998, Bor et al., 2013) improvements in lifespan variation have not. Recent attention has been paid to understanding how age specific mortality

change impacts lifespan variation trends (Smits and Monden, 2009, Seaman et al., 2016a, Gillespie et al., 2014). There is some variability in the relationship between life expectancy and lifespan variation for some countries. Maintaining the optimal correlation depends on whether mortality compression continues to outpace mortality expansion (Gillespie et al., 2014). Mortality expansion is when the distribution becomes wider at the right hand tail because the age at death is increasing. Mortality compression is when the age at death distribution becomes narrower because younger age deaths (left hand tail) are being avoided and pushed into older ages[1].

Changes to mortality across young ages have historically driven the correlation between increasing life expectancy and decreasing lifespan variation. This is because mortality decline at young ages decreases lifespan variation by compressing the distribution of age at death. As life expectancy increases more ages at death are deemed to be premature and mortality compression is no longer reliant on infant and childhood deaths. However, at the same time mortality declines have shifted to older ages. These age processes of mortality change can alter the correlation between increasing life expectancy and decreasing lifespan variation.

Mortality decline at older ages can be associated with increases in lifespan variation if the age at death causes mortality expansion but will still be advantageous in terms of life expectancy. Mortality expansion increases lifespan variation by delaying the age at death (Gillespie et al., 2014, Zhang and Vaupel, 2009). It is not yet inevitable that mortality expansion will cause countries to experience increases in lifespan variation if premature mortality decline is

[1] Alternatively mortality has to increase more slowly among younger ages than older ages.

maintained. Ensuring decreases in premature mortality rates means that mortality compression counterbalances mortality expansion.

The USA is one of the most widely cited example countries where the age pattern of mortality decline has led to increases in lifespan variation (Shkolnikov et al., 2011, Gillespie et al., 2014). This can be explained by important changes to the distribution in age at death: expansion of mortality into extreme old age now coincides with smaller reductions in premature mortality (Oeppen, 2008). Gillespie et al. (2014) further demonstrated that age specific mortality change varied most across working adult ages when comparing the USA and Canada: in the USA male mortality rates increased between 1983 and 1994 by 30% for ages 34 to 37 years old. In Canada there was a smaller increase of 17% across slightly older ages (32 to 40 years old). This difference in the age specific pattern of mortality change caused lifespan variation trajectories in the USA and Canada to diverge.

Explaining why this is the case is challenging because many factors have caused the age processes of mortality to change. Some differences in age related mortality change between countries may reflect differences in terms of medical care and public health advancements. Others may indicate an inability for societies to provide an adequate social safety net to ensure all members of the population can access the primary goods required for living (van Raalte, 2011a, Vallin and Meslé, 2004, Tuljapurkar, 2010, Gillespie et al., 2014) with good health being one the most fundamental requirements (Fabienne, 2001, Sen, 2002).

When comparing the USA and Canada it is valid to hypothesise that the contrasting health care systems may play a role in the different distributions in age at death: access to health care at working adult ages in the USA depends largely on employer provided insurance and without it access to preventative healthcare, that could reduce the risk of premature death, is restricted (Gillespie et al., 2014, Wilper et al., 2009). However, if the only opportunity to access adequate healthcare is through employment then increasing premature mortality rates in the USA is a macroeconomic issue: it reflects the fact that the social safety net, for ensuring access to primary goods in society, in the USA is weak relative to a number of other countries (Brenner and Mooney, 1983, Shkolnikov et al., 2011). The ability for the health care system to account for differences in age patterns of mortality change is also weak for other countries experiencing increasing lifespan variation, such as Scotland. This is because health care in Scotland is provided irrespective of employment status, a system that was founded on the principal of universal health care being provided for free at the point of delivery (Timmins, 2001). Yet Scotland, like the USA, has demonstrated recent increases in lifespan variation and its trend has diverged from its closest geographical neighbours (Popham and Boyle, 2010).

2.5.4 Motivation for this thesis

Few studies measuring lifespan variation trends in cross national comparative context have included Scotland despite it being the country in Western Europe experiencing the poorest levels of life expectancy, having high rates of premature death and unenviable levels of socioeconomic deprivation (McCartney et al., 2012b, Leyland et al., 2007a). The study by Popham and Boyle (2010) is an exception that has applied a measure of lifespan variation to Scotland.

Given that the international literature demonstrated that the desired association between increasing life expectancy and decreasing lifespan variation was dependent upon premature mortality (Gillespie et al., 2014, Shkolnikov et al., 2011), it was fair to assume that Scotland would most likely be a country that has failed to reduce lifespan variation as well as its closest comparators. This is what Popham and Boyle (2010) identified: the lifespan variation trend in Scotland had been higher than in comparable countries, equating to greater inequality. However, of greater concern was the indication that lifespan variation in Scotland had recently increased, suggesting worsening levels of total inequality since around the 1990s. The worsening dimension of total inequality in Scotland, which has largely been overlooked in public health research, was the motivation for this thesis. Popham and Boyle (2010) also recognised that their study was unable to identify how different socioeconomic groups have contributed to worsening population level lifespan variation: this thesis contributes to filling these research gaps.

2.6 Research gaps

2.6.1 Statistically assessing the change in trends

Although international comparative studies have established differences in lifespan variation trends across countries, important research gaps still remain. One such gap is that Scotland's trend has only been compared to a token number of its closest comparator countries (Popham and Boyle, 2010). Another is that the timing of any changes in Scotland's trend have not been statistically assessed compared to other Western European countries. Evidence, using data from as early as the mid-19th Century, suggests that lifespan variation reductions

have been slowing since as early as the 1950s for some countries but did not start slowing until the 1970s for others (Edwards, 2011, Smits and Monden, 2009, Shkolnikov et al., 2011). These research gaps are the focus of chapter 4 in this thesis and are filled by answering the following research questions:

1. Has Scotland's relative lifespan variation ranking within Western Europe changed over time?

2. Was the timing and relative rate of lifespan variation change in Scotland comparable with any other Western European country?

2.6.2 Quantifying the impact of age specific mortality change within Scotland compared to within England and Wales

International studies have further progressed to quantifying the age processes of mortality, within countries, that have contributed to increases in life expectancy and decreases in lifespan variation (Auger et al., 2014, Vaupel et al., 2011, Gillespie et al., 2014). These studies have found that countries with the greatest improvements in life expectancy and lifespan variation tend to be those who have been most successful at reducing premature deaths (Shkolnikov et al., 2011, Vaupel et al., 2011). This is because reducing premature deaths causes mortality compression, i.e. a narrowing of the variation in age at death, whilst reducing older age deaths causes mortality expansion, i.e. a widening of the variation in age at death. While both processes improve life expectancy reductions in lifespan variation can only be achieved through more mortality compression than expansion (Popham et al., 2013, Shkolnikov et al., 2011, Gillespie et al., 2014).

In the USA relatively large reductions in older age mortality have been experienced alongside relatively poor reductions in premature mortality (Shkolnikov et al., 2011). These two mortality processes explains why its life expectancy is relatively low but somewhat higher than its lifespan variation suggests. Some have pointed out that the combination of these two mortality processes is counter-intuitive. The economic costs of social interventions for reducing premature adult mortality – deaths associated with external causes – tend to be relatively lower than the economic costs of medical interventions for reducing old age mortality – deaths from chronic diseases (Smits and Monden, 2009, Nau and Firebaugh, 2012, Vallin and Meslé, 2004). The same age at death characteristics may account for Scotland's population level lifespan variation trend as it is known to have experienced relatively high premature mortality (Leyland, 2004, Schofield et al., 2016).

Previous studies of premature mortality in Scotland found no change or even increasing mortality rates for certain working age groups over time (Leyland, 2004, Norman et al., 2011, Leyland et al., 2007a). For example mortality rates for males aged 15-29 years old increased between 1981 and 2001, while mortality rates for males aged 30-44 years old demonstrated an initial decrease before increasing, meaning that by the end of 2001 the rate had effectively remained unchanged (Leyland et al., 2007a).

Many studies have compared health trends within Scotland to those within England and Wales (Campbell et al., 2013, McCartney et al., 2012b). This comparison is justifiable as they share social and economic contexts, and a national government yet they have somewhat distinct mortality profiles. It is not

yet clear what age processes explain the unfavourable lifespan variation trend within Scotland compared to within England and Wales. The analysis in chapter 5 contributes to filling this research gap by answering the following research questions:

3. Which ages of death contributed to the lifespan variation trend in Scotland?

4. Did the ages of death contributing to the lifespan variation trend in Scotland differ from the ages of death contributing to the lifespan variation trend in England and Wales?

2.6.3 Lifespan variation differences at shared levels of life expectancy

The age patterns of mortality driving improvements in life expectancy over time tend to be ages where the greatest proportion of deaths occur (van Raalte et al., 2011). Existing studies that have illustrated how tackling premature mortality tends to drive improvements in lifespan variation over time (Shkolnikov et al., 2011, van Raalte et al., 2014). However it is argued that comparative studies should not simply evaluate trends in lifespan variation alongside trends in life expectancy. This is because life expectancy and lifespan variation are strongly correlated meaning that any reductions in lifespan variation over time would almost completely be reflected by increases in life expectancy.

Rather Smits and Monden (2009) propose that lifespan variation should be studied independently of life expectancy. This can be achieved if studies compare levels of lifespan variation when a certain level of life expectancy has been achieved, regardless of the year it is achieved. This is important if we are to understand more about why countries reach a shared level of life expectancy

with different levels of lifespan variation. In turn this may provide information for the different strategic options Governments have for improving average population health and reducing inequality. Smits and Monden (2009) demonstrate why this is by presenting two hypotheses.

Firstly *the forerunner hypothesis* suggests that countries reaching high life expectancy first may do so with a lower lifespan variation than countries achieving the same level of life expectancy later. This could stem from forerunners focusing on public health improvements and the social determinants of health that tend to reduce premature mortality (e.g. infant mortality, sanitation, reducing risk of accidental death, social protection policies) before moving onto more technical interventions and advanced medical practices associated with reductions in older age mortality (Nau and Firebaugh, 2012).

Secondly the *diffusion of knowledge hypothesis* argues that countries achieving the same level of life expectancy later may be better positioned to benefit from the diffusion of public health knowledge surrounding the social determinants of health. This could mean premature mortality is reduced more effectively and efficiently (Oeppen, 2008). So although arriving at the same life expectancy later they do so with a lower lifespan variation, especially if the *diffusion of knowledge* surrounding premature mortality and the social determinants of health outpace the impact of technical interventions which may reduce old age mortality resulting in more compression than expansion.

Evidence is mixed. Smits and Monden (2009) find evidence for the diffusion of knowledge hypothesis while Vaupel et al. (2011) find that lifespan variation is very similar between countries when reaching a shared level of life expectancy.

Therefore chapter 6 seeks to understand the age patterns of mortality that account for differences in lifespan variation between Scotland and England and Wales at similar levels of life expectancy. This analysis answers the following research questions:

5. Was lifespan variation higher or lower in Scotland at a shared level of life expectancy with England and Wales?

6. Which ages of death account for the lifespan variation gap between Scotland and England and Wales at a shared level of life expectancy?

2.6.4 Changes to the socioeconomic gradient for lifespan variation

As previously discussed, studies have long established the association between socioeconomic deprivation and mortality by measuring life expectancy; the least deprived experience the highest life expectancy and the most deprived the lowest (The Scottish Government., 2014, Singh and Siahpush, 2006). Fewer studies have measured inequalities in lifespan variation by socioeconomic deprivation (van Raalte et al., 2014, van Raalte et al., 2011).

These types of studies are important for at least three reasons. Firstly to help address the criticism that a total inequality concept only reflects random variation as opposed to capturing any form of social inequity (Dibben and Popham, 2011, Murray et al., 1999). If lifespan variation only reflected inevitable and unavoidable differences in age at death and there were no social process at play (inequity) then lifespan variation differences between populations would be minimal (Dibben and Popham, 2011).

Secondly to identify if the most deprived socioeconomic groups are exposed to a double burden of inequality: the shortest average life expectancy and the greatest amount of uncertainty about the age at which they will die. Thirdly to increase our understanding of the ways in which different socioeconomic groups contribute to lifespan variation inequalities at the population level.

One of the first large-scale international studies to systematically compare lifespan variation by socioeconomic group was by van Raalte et al. (2011). They used linked Census data and constructed three internationally comparable educational levels as an indicator for socioeconomic deprivation: less than secondary education, complete secondary education, and some tertiary education. The lowest educational category was found to experience the highest level of lifespan variation across all of the ten countries included (Belgium, Czech Republic, Estonia, Finland, France, Norway, Poland, Slovenia, Sweden and Switzerland). On average lifespan variation was 1.5 years higher for the lowest education category compared to the highest. However the time period covered for this study was limited, with data for the countries spanning ten years or less. Changes to the educational distribution make longer temporal comparisons challenging (van Raalte et al., 2014).

The study by van Raalte et al. (2014), looking at Finland only, was able to address this issue by measuring occupational socioeconomic status between 1971 and 2010. It reiterated the finding that lifespan variation was higher for the most deprived group but also found that the trend for the most deprived had diverged to increasing lifespan variation. This illustrated that the socioeconomic gradient for lifespan variation has steepened over time in Finland.

It is not yet known how lifespan variation in Scotland is patterned by any measure of socioeconomic deprivation (Popham and Boyle, 2010). Nor have any known studies formally quantified changes in the socioeconomic gradient for lifespan variation over time and related these to its deteriorating lifespan variation ranking within Western Europe. Chapter 7 therefore fills these research gaps by answering the following questions:

7. Is there a socioeconomic gradient for lifespan variation in Scotland?
8. Has the socioeconomic gradient for lifespan variation in Scotland, changed over time?
9. Are changes to the socioeconomic patterning of lifespan variation in Scotland related to Scotland's deteriorating lifespan variation ranking within Western Europe over time?

2.6.5 Age and cause of death contributions to lifespan variation within socioeconomic groups

The two key studies by van Raalte utilised measures of socioeconomic deprivation which only permitted the reporting of lifespan variation from adult ages (van Raalte et al., 2014, van Raalte et al., 2011). This is a theoretically and empirically valid approach: it reflects the argument that inequalities in developed countries may be better reflected when reporting life expectancy and lifespan variation from adult ages only as infant deaths are now rarer than in the past (Smits and Monden, 2009). However it does mean that there was something of a trade-off between capturing the full age distribution of mortality and applying a measure of socioeconomic deprivation which will adequately capture completed years of education or achieved occupation (van Raalte et al., 2011).

It is also a departure from the standard reporting of life expectancy from age 0 which is conventionally used to make international comparisons (World Health Organization, 2010).

Chapters 7 and 8 of this thesis utilised an area based measure of socioeconomic deprivation. An area level measure of socioeconomic deprivation can be applied to the entire population meaning that age truncation is not necessary. This approach has not previously been operationalised when studying lifespan variation trends. This is an important research gap to fill as area based measures of socioeconomic deprivation, compared to individual level indicators, are more widely available, often updated more frequently, and are able to capture multiple dimensions of deprivation (Payne, 2012, Kearns et al., 2000, Green, 2013). However this is not to say that area based measures are free from limitation.

Firstly, area based measures tend to reflect the average deprivation characteristics of the people living within an administrative area. Administrative areas are created primarily for administrative purposes which means this geographical context may not provide as much detail about the area level aspects most important for health (Leyland et al., 2007a). Secondly, people within areas are heterogeneous: some individuals living within a deprived area may be affluent and some individuals living within affluent areas may be deprived (Morgan and Baker, 2006, Leyland et al., 2007a). Thirdly, area level measures must be recognised as distinct from individual socioeconomic characteristics: one measure cannot be used as a substitute for the other. However area level proxy measures are a powerful tool: their wide availability

reflects the fact that socioeconomic inequalities are so embedded within society that they manifest across many aspects of life (Galobardes, 2012). The strengths and weaknesses of measures of deprivation are evaluated further in chapter 3 of this thesis (data and methods). The purpose of this section of the literature review is to: identify research gaps within the lifespan variation literature and demonstrate the value lifespan variation has for understanding mortality inequalities in Scotland.

Although van Raalte et al. (2011) demonstrate that higher lifespan variation for the most deprived groups in Finland is driven by differences in mortality rates across ages 35-55 years old they were unable to account for the contributions made below these ages. Accounting for deaths below these ages may be particularly important in Scotland because mortality rates for males aged 15-29 years old from the most deprived group increased between 1981 and 2001 (Leyland et al., 2007a). However this may be in contrast to the mortality at the youngest ages (below 15 years old) where Scotland has had relatively greater improvements in mortality rates at some time points compared to England and Wales (Campbell et al., 2013).

The potentially large contributions to lifespan variation in Scotland from these ages may have been overlooked if the population had to be truncated in order to apply an individual level measure of socioeconomic position. Therefore within the context of this thesis it was valuable to utilise a measure of socioeconomic deprivation from area level information because it allowed the whole age distribution of death in the population to be studied. The age distribution does not need to be truncated as is the case with alternative individual level

indicators. Studying the full age distribution of death by socioeconomic deprivation can help to highlight further the population sub groups that may be at risk of premature death (Seaman et al., 2015).

Additional insight into the social processes and aetiological pathways that are driving population level inequality can be gained by including cause of death into the analysis (van Raalte et al., 2014, van Raalte et al., 2015). Several studies have identified the age and cause specific components driving the crude trends in life expectancy and lifespan variation at the population level (Auger et al., 2014, Karanikolos et al., 2012, Kelly and Preston, 2016). Some of these studies have highlighted the consequence of harmful behaviours leading to premature mortality, with particular attention being paid to smoking (van Raalte et al., 2014, Kelly and Preston, 2016).

Smoking is known to be a major cause of death in Scotland with 1 in 5 deaths being attributed (Gray and Leyland, 2009). This cause of death is also considered to be one of the biggest drivers of health inequalities because it is so strongly patterned by socioeconomic deprivation. For example 34% of adults in the most deprived group in Scotland smoke compared to only 9% of the least deprived group (National statistics, 2015). Kelly and Preston (2016) estimated that population level life expectancy at age 50 in Scotland could be increased by 3.1 years for males and 3.6 years for females if smoking attributable deaths were removed but did not account for socioeconomic inequality and did not estimate lifespan variation.

In their study set in Finland van Raalte et al. (2014) did account for socioeconomic deprivation and demonstrated that smoking had a large impact on

differences in life expectancy but little impact on lifespan variation. This was because of the time lag associated with smoking attributable deaths.

Preventing deaths attributable to smoking could have limited impact on lifespan variation as they would reduce deaths in ages that potentially contribute to mortality expansion. This should not be interpreted as a moral judgement about which ages and causes of death should be prevented but aims to emphasise that causes of death have different implications for the age processes of mortality. This reflects the argument that the drivers of life expectancy can differ from the drivers of lifespan variation (Shkolnikov et al., 2011).

Diverging lifespan variation trends, either at the population level or amongst a particular socioeconomic group, tend to occur when there is a lack of mortality compression. This age process of mortality is found to be associated with premature deaths from causes not attributable to smoking. For example van Raalte et al. (2014) illustrated that external causes of death amongst the most deprived socioeconomic group in Finland explained its higher lifespan variation when it achieved a similar level of life expectancy as the least deprived group. This was despite the fact that the most deprived group tended to have a temporal advantage for reducing deaths from circulatory diseases, so experiencing lower mortality rates from circulatory diseases when reaching the same level of life expectancy as the least deprived.

In Scotland the causes of death which contributed to the rising mortality rates following the 1980s were suicide, alcohol and substance abuse and violence (Leyland et al., 2007b). These causes of death are strongly patterned by socioeconomic deprivation suggesting that that socioeconomic inequalities in

Scotland may account for the diverging lifespan variation trend at the population level (Popham and Boyle, 2010, Seaman et al., 2016a).

Although international studies have quantified the age and cause specific contributions driving lifespan variation inequalities by socioeconomic deprivation this has not been done for Scotland. This is an important research gap to fill because analysing the social patterning of causes of death can help to inform decision makers about which types of interventions could ensure future improvement. For example declines in mortality from circulatory diseases have been achieved by monitoring and medically treating risk factors such as high blood pressure, while deaths from lung cancer have fallen over time due to legislative changes and shifting social norms in smoking (Karanikolos et al., 2012, van Raalte and Caswell, 2013). Chapter 8 therefore answers the remaining research questions:

10. Which ages and causes of death contributed to changes in lifespan variation, over time, for different socioeconomic groups in Scotland?

11. Which ages and causes of death contributed to the lifespan variation gap between the most and least deprived socioeconomic groups in Scotland?

12. Which ages and causes of death contributed to the lifespan variation gap between the most and least deprived, when life expectancy was similar, in Scotland?

2.7 Literature review summary

In summary, this literature review chapter demonstrated some of the debates surrounding the study of health, health inequalities and empirical measurements in research.

The first section demonstrated that our understanding of health has changed over time, moving from a medical definition to a wider social interpretation.

The second section discussed the persistence of health inequalities in developed countries (Mackenbach et al., 2008, Marang-van de Mheen et al., 1998, Blane et al., 1990). A number of theoretical developments were summarised including the shift away from understanding absolute material poverty as the primary determinant of health.

The third section then discussed the debates surrounding the conceptual meaning of health inequalities and how the normative judgements we attach determines how we quantify the magnitude of health inequalities. This has been demonstrated by contrasting two, supposedly, different approaches: the traditional concept of differences between socioeconomic groups compared with a total inequality concept. It is not the aim of this thesis to make a distinction between a right and a wrong approach. Rather, it aims to build upon the body of international literature, demonstrating the value of using lifespan variation as a measure of total inequality for studying health inequalities within Scotland.

The fourth section identified gaps in the existing lifespan variation literature. Despite relatively poorer life expectancy at the population level and wider mortality inequalities between the most and least deprived being well documented for Scotland little is known about lifespan variation.

This thesis makes a unique contribution by analysing the population level lifespan variation trend in Scotland and demonstrating how this reflects socioeconomic inequalities in ages and causes of deaths. The following chapter details the data sources and quantitative analysis methods that were used to answer the research questions and make an empirical contribution to the existing body of literature.

3 Data and methods

3.1 Introduction

The overarching aim of this thesis is to increase our understanding of mortality inequalities in Scotland by estimating and analysing lifespan variation, a measure of total inequality. Life tables are required to calculate lifespan variation. These are constructed from death counts and population estimates. Life tables are explained in more detail later in this chapter. In Scotland the agency responsible for collating vital events statistics is National Records of Scotland (NRS). The research questions in chapter 4, chapter 5 and chapter 6 required population life tables while the research questions in chapter 7 and chapter 8 required life tables stratified by a valid measure of socioeconomic position. Lifespan variation was measured in this thesis by calculating e† which is interpreted as average number of years remaining at death and measures life years lost when a death occurs. A critical evaluation of e† is included later in this chapter.

Figure 8 summarises the broad research aims of each analysis chapter, the data used and the statistical tests, measure, or method applied in order to answer the specific research questions

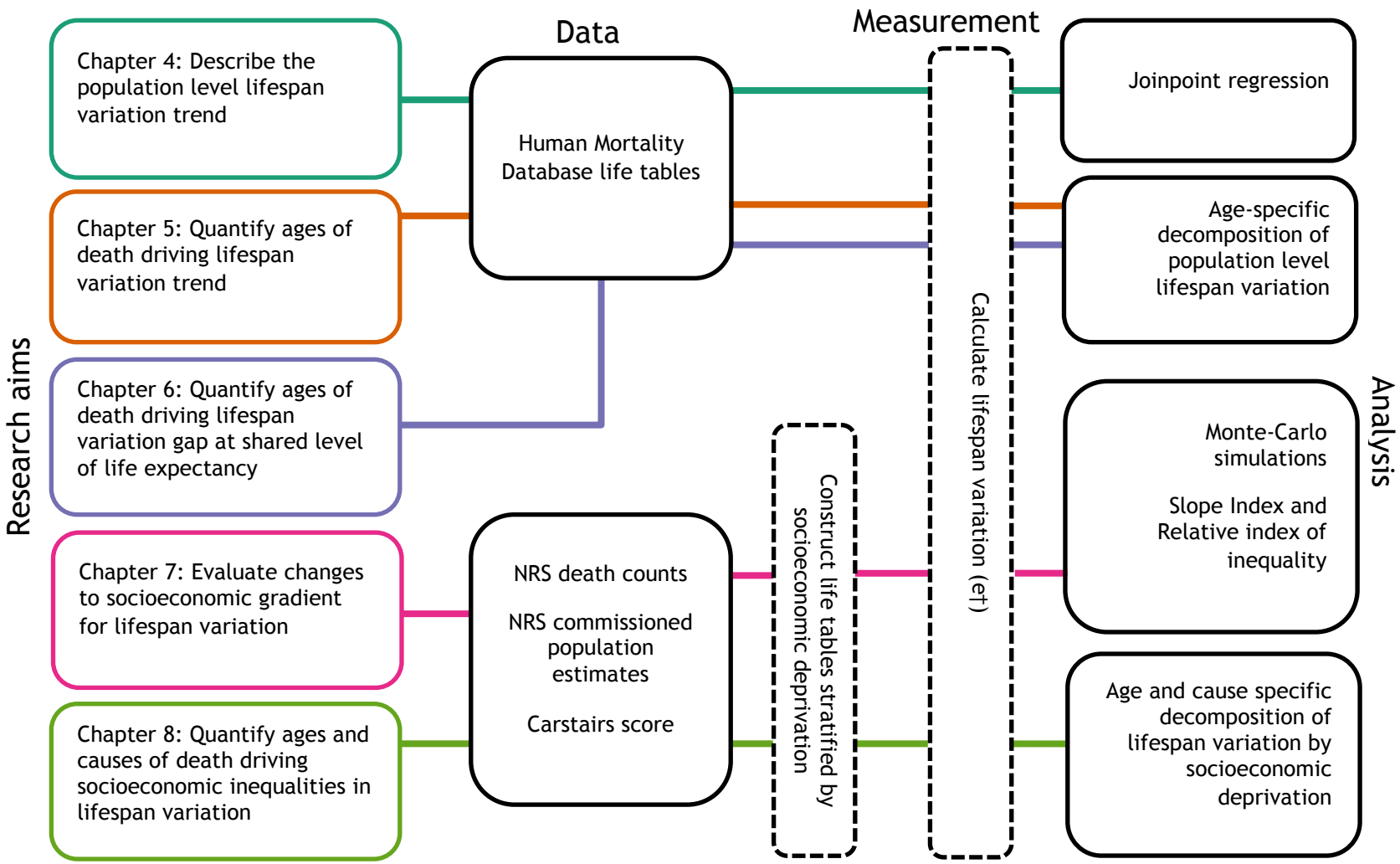


Figure 8 Summary of research stages

Life table data suitable for answering the research questions in chapter 4, chapter 5 and chapter 6 were available from the Human Mortality Database (HMD). The HMD is highly regarded and allowed annual lifespan variation estimates for the total population of Scotland to be calculated from 1950. This was then analysed and cross-national comparisons carried out using joinpoint regression (chapter 4) and age-specific decomposition (chapter 5 and 6). However this data source does not include any information on socioeconomic position.

Lifespan variation estimates stratified by a valid measure of socioeconomic position were required to answer the research questions in chapter 7 and chapter 8. Census population estimates for four most recent Census years (1981, 1991, 2001 and 2011) were obtained via a commissioned request from NRS. These were matched with the relevant death counts, produced by NRS but already held at the MRC/CSO Social and Public Health Science Unit, University of Glasgow. These were subsequently matched with the Carstairs score, a validated measure of socioeconomic deprivation that has been derived from four Census variables at every Census since 1981. The stages of data management carried out in order examine lifespan variation stratified by socioeconomic position are described in this chapter: these included statistically modelling mortality rates beyond the oldest age (85+) for which population estimates were available from the NRS commissioned request. Monte-Carlo simulations, the slope index of inequality and the relative index of inequality were used to answer the research questions in chapter 7. Age and cause specific decomposition of lifespan variation by socioeconomic deprivation were used to answer the research questions in chapter 8.

This chapter has two main sections. The first data section outlines the data sources used and critically evaluates their strengths and limitations against alternatives that were considered. The second section (methods, measures and statistical tests) demonstrates the evolution of the analysis from descriptive results through to those that allowed quantification and statistical inferences to be drawn.

3.2 Section 1: Data

The first section of this chapter will discuss the two data sets that were used: the life tables from the HMD, and the population estimates and death counts from NRS matched with the Carstairs score used to construct life tables. These data are considered secondary data: they were not intentionally collected for the purposes of this thesis (Lumme et al., 2015).

3.2.1 Human Mortality Database life tables

The research questions in chapter 4, chapter 5 and chapter 6 were concerned with assessing the population level lifespan variation trend for Scotland, over a significant time period and in a cross-national comparative context. In order to do this annual lifespan variation estimates had to be calculated for the total population of each country. Lifespan variation is calculated from life tables which are freely available from the HMD, upon registration.

The aim of the HMD is to formally record changes to population longevity in order to enable research into its causes and consequences (Wilmoth et al., 2007). In order to achieve this aim the HMD collects vital events statistics and population estimates from a range of countries and produces comparable period life tables. These data have a long history of being collected at the national

level in Scotland, with death records beginning in the middle of the 19th Century (National Records of Scotland, 2016c).

Chapters 4 utilises the period life table data for Scotland and 16 comparator populations. The comparator countries and the years for which data were available are given in table 2. Countries were grouped according to the geographical regions used by Mackenbach et al. (2008). Chapters 5 and chapter 6 utilise the life table data for Scotland and for England and Wales only.

Table 2 comparator countries and years for which complete period life tables were used for analysis in Chapter 4

Country	Years data available	Country group (Mackenbach et al., 2008)
Scotland	1950-2011	
England & Wales	1950-2011	West
Northern Ireland	1950-2011	
Ireland	1950-2009	
Denmark	1950-2011	
Finland	1950-2009	North
Norway	1950-2009	
Sweden	1950-2011	
Austria	1956-2010	
West Germany*	1956-2010	Continental
Switzerland	1950-2011	
Belgium	1950-2012	
France	1950-2012	
Netherlands	1950-2009	
Spain	1950-2009	
Italy	1950-2009	South
Portugal	1950-2012	

*West Germany is the area of unified Germany formally known as the Federal Republic of Germany (FRG). The territory has changed over time but the statistics here represent the regions that made up the FRG.

3.2.1.1 Description of period life table

A period life table is a vital tool for carrying out demographic research which displays a range of information about the mortality experience of a population (Wunsch et al., 2013). The life tables utilised for this thesis were period life tables. The population included in a period life table can be understood as a synthetic or hypothetical population. This is because it does not record how an actual birth cohort dies out (cohort life table) but rather models what would happen to a population if a given set of mortality conditions were experienced (Wunsch et al., 2013, Preston et al., 2001). Life tables are constructed separately for males and females because of their differing mortality experiences.

The definitions of the columns that make up a period life table are detailed in table 3.

Table 3 Period life table column definitions

x	Single year of age (0-110+)
N	population estimates by single year of age
D	Deaths by single year of age
n	Width of age interval
mx	Death rate between ages x and x+n
qx	Probability of death between ages x and x+n
ax	Average length of survival between ages x and x+n of persons dying in the interval
lx	Number of survivors at exact age x, assuming l(0) = 100,000
dx	Number of deaths between ages x and x+n
Lx	Number of person-years lived between ages x and x+n
Tx	Number of person-years lived after exact age x
ex	Life expectancy at exact age x (in years)

Age specific mortality rates (mx) are constructed from the number of deaths (D) in each given year divided by the population estimates (N). qx shows the probability of a person at that age dying in the next year of life. From this probability a number of statistics can be derived. Transforming age specific mortality rates into probabilities may lack precision if the numbers of deaths are low or if the population at risk is small (Preston et al., 2001). The life tables obtained from the HMD cover entire populations meaning limited consideration needs to be given to small sample sizes. For example the full population of Scotland has had an estimated population of at least 5,062,940 and between 53,661 and 65,747 deaths per year since 1954 (National Records of Scotland,

2014b). Although the total sample size is not small when analysing population trends this was a consideration when stratifying by socioeconomic deprivation. This issue is addressed later on in this chapter.

The life tables obtained from the HMD were complete period life tables as opposed to abridged life tables. Abridged life tables present the mortality experience broken by age groups: a complete life table presents the mortality experience for each single year of age. There are advantages and disadvantages to using both approaches.

The main motivation for constructing abridged life tables arises from problems stemming from age heaping, where by recording of age data may be misclassified because of rounding to years ending in 0 or 5. This can result in an overestimated and under estimation of age data when broken down by single years of age that can be overcome by grouping 5 year age groups together (Kostaki and Panousis, 2001). Small population sizes or rare events may also make abridged life tables more appropriate. However a more precise representation of the mortality experience of interest is provided when using complete life tables (Preston et al., 2001).

The HMD provided robust complete period life tables that were suitable for analysing Scotland's population level trend, relative to 16 comparator populations over a substantial period of time (table 1). However these data could not be used to stratify the population by socioeconomic position, which has a long established association with mortality inequalities. The literature review demonstrated why it is important to know how lifespan variation is

patterned by socioeconomic position in Scotland, and why assessing the socioeconomic gradient for lifespan variation over time is of interest.

3.2.2 National Records of Scotland data

The research questions in chapter 7 and chapter 8 were concerned with assessing changes to the socioeconomic gradient for lifespan variation in Scotland, over time. In order to do this lifespan variation estimates stratified by a valid measure of socioeconomic position had to be calculated. Lifespan variation is calculated from life tables which are constructed from the number of deaths in a given year divided by the relevant population estimates. Therefore the basic data required were deaths and population estimates stratified by a valid measure of socioeconomic position.

The most robust population estimates are derived from the Census which is carried out every ten years in Scotland. Census population estimates were utilised in chapter 7 and chapter 8 of this thesis. Census population estimates are updated annually to produce mid-year population estimates until the next Census. Mid-year population estimates refer to the 'usually resident' population on June 30th every year while Census population estimates refer to the population on Census day (Office for National Statistics, June 2016). NRS provide the HMD with the relevant vital events statistics and population estimates for Scotland. Although deaths and population estimates have a long history of being collected in Scotland (National Records of Scotland, 2016c) it is challenging to stratify the population by a consistent measure of socioeconomic position.

3.2.3 Measuring socioeconomic position in Scotland

The measurement of socioeconomic position has evolved over time and no single authoritative measure exists. Rather a number of measures are available each capturing different social and economic characteristics across different levels (e.g. individuals, households or areas) and across different stages of the life course (e.g. infancy, childhood, adolescence and adulthood) (Krieger et al., 1997, Bailey et al., 2003b). The inability for one measure to adequately capture all dimensions of socioeconomic position reflects the complexity of the construct (Galobardes et al., 2006). It is also important to recognise that optimal indicators of socioeconomic position may change over time and the relevance of indicators may differ between populations or cohorts (Næss et al., 2005). It is therefore important to consider the conceptual basis of different measures of socioeconomic position to ensure that the correct measure is used, results are interpreted appropriately, and that the measure used is appropriate for making the intended comparisons (Rose et al., 2005).

Four measure of socioeconomic position were considered for use within this thesis: Registrar-General's Social Class (RGSC), National Statistics Socio-economic classification (NS-SEC), the Carstairs score and the Scottish Index of Multiple Deprivation (SIMD). The first two are measures of individual level social class and the latter two are relative measures of area level socioeconomic deprivation. Household level measures are not discussed as it would not have been possible to derive a household level measure of socioeconomic position from the death data obtained.

In order to determine which approach and which measure of socioeconomic position was most appropriate for this thesis the following criteria were considered: (1) the relevance of the measure's conceptual and theoretical framework (2) the availability of the measure and its ability to reflect a consistent meaning over time (3) the ability for population data and death data to be matched to the measure. The measures are summarised in relation to these criteria in table 4 and a more detailed discussion follows.

Table 4 Summary of socioeconomic measures considered for this thesis

	Occupation based social class (individual level)		Relative socioeconomic deprivation (area level)	
	RGSC	NS-SEC	Carstairs	SIMD
Theoretical/conceptual framework	Conceptual basis constructed in retrospect. This alluded to culture being more important than material factors in explaining morality inequalities. Culture referred to knowledge of health and hygiene. Culture was perceived to be related to occupational status rather than to income or wealth.	Adapted Goldthorpe schema (sociological classification) which brought together social classes in terms of work and market situations. Internationally accepted and conceptually clear. Validated as a measure of health and as a predictor of health, education and numerous outcomes.	Conceptual basis established prior to construction. Variables selected on their ability to conceptualise relative deprivation in different areas. Variables selected on the basis that they would reflect the features of deprivation most relevant for health inequalities.	Datazones constructed to reflect physical boundaries, natural communities and households with similar social characteristics. Includes multiple domains that are weighted depending on the theoretical relevance for deprivation. Includes a measure of mortality and morbidity which means full indicator cannot be used for health research.
Availability & consistency	Introduced in 1913 as it applied to 1911 Census, cannot be directly derived for 2001 or 2011.	Introduced at the time of the 2001 Census and subsequently available for use in all official statistics and surveys.	Derived from four Census variables. Available for 1981, 1991, 2001 and 2011 Census.	Introduced in 2004. Subsequently available for 2006, 2009, 2012 and 2016.
Match with Census pop. & deaths	Risk of numerator/denominator bias as occupation in Census may differ from occupation reported on death certificate. SLS linked data can overcome this but only available from 1991 (compromises criteria 2).	Risk of numerator/denominator bias as occupation in Census may differ from occupation reported on death certificate. SLS linked data can overcome this but only available from 1991 (compromises criteria 2).	Matching with deaths and Census population estimates possible using postcode sector.	Matching with deaths and Census population estimates possible using datazones .

3.2.3.1 RGSC & NS-SEC

The RGSC is one of the oldest methods for categorising the socioeconomic position of individuals based on their occupation. It was first applied in 1913 to the 1911 Census. The 'five social classes' aimed to capture the social division between the upper, middle and working social classes.

The RGSC proposed that culture was more important for health than material factors (Rose and Pevalin, 2003). Culture was interpreted as knowledge about health and hygiene and was presented as being more related to occupation than to income or wealth. This conceptual interpretation sees the hierarchy of occupations as being important for health because it reflects the hierarchy of occupations in terms of social reputation rather than in terms of income. This was later changed and social class category was assigned to reflect occupational skill as opposed to perceived social standing, although only 7% of occupations were reassigned to a different social class (Rose, 1995). A clear conceptual reasoning for assigning occupations to each social class was missing when this measure was first derived and the theoretical interpretation of culture was only developed in retrospect (Rose, 1995).

Further criticisms were directed at the RGSC as it had failed to respond adequately to changes in the occupation structure over time: the decreasing proportion of people in the lowest social class for example which was partly caused by increased female participation in the labour market and partly caused by changes to the skill set demanded by the economy (Leyland et al., 2007a). This prompted the development of a new social class classification, the NS-SEC. On appearances the two measures of occupation based social class may seem

similar but it is not possible to read across the two measures (Rose and Pevalin, 2003, Leyland et al., 2007a).

The NS-SEC assigns a code to each occupation which corresponds to an occupation unit group conceptualised in relation to Goldthorpe's schema (Rose and Pevalin, 2003). The position of an occupation within the classification structure reflects level of job authority, control and autonomy but is not intended to be interpreted as a hierarchy, although it often is (Rose et al., 2005). Goldthorpe's schema framed social class in terms of employment relations as opposed to occupational status or skill. Employment relations are concerned with: individual's capacity to sell or buy labour, an individual's relationship with work, and the interactions with the employment structure that exists in society. The theoretical framework aims to communicate the idea that occupation based social classes do not exist in isolation but are defined in terms of one another e.g. an individual cannot be an employee if they do not have an employer (Krieger et al., 1997). Therefore NS-SEC is a measure of socioeconomic position which seeks to capture the fact that employment relations and regulations are mechanisms which impact the social structure of modern societies (Goldthorpe, 2007, Krieger et al., 1997). This approach is internationally recognised and a major strength of the empirical measurement is that it was developed alongside a coherent theoretical framework (Rose et al., 2005). However a clear conceptual basis does not mean that it was a suitable measure for use within this thesis. It was inappropriate for the following three reasons.

Firstly, analysing trends over time are often restricted to short time periods if using occupation based measures of social class. For instance the NS-SEC was introduced in 2001 which meant that it would not have been possible to apply it easily to the 1980s. This was when life expectancy improvements in Scotland began to stall and premature mortality rates changed (McCartney et al., 2012b, Leyland et al., 2007b).

Although the RGSC did enable social inequalities to be empirically studied for most of the last century it was not directly derivable for 2001 and 2011. It is also not appropriate to make direct comparisons between the RGSC and the NS-SEC (Leyland et al., 2007a). They have distinct interpretations of the features of employment that are important for social stratification which need to be recognised if they are to be used appropriately. Therefore it would not have been possible to construct stratified life tables that were theoretically and empirically consistent over the time period of interest using an individual level measure of occupation based social class.

Secondly, caution needs to be exercised when deriving health estimates for social class subgroups from so called 'unlinked data': these may be vulnerable to numerator/denominator bias (Shkolnikov et al., 2007). This is because the classification of social class derived from occupation on a death certificate is reported by proximal sources and may be less accurate than social class derived from self-reported occupation on the Census. This may result in the health estimates being overestimated for some occupational groups and underestimated for others (Carstairs and Morris, 1990, Shkolnikov et al., 2007). The most accurate data for analysing health differences between social class

groups is linked data (Shkolnikov et al., 2007). The Scottish Longitudinal Study (SLS) is the largest, most comprehensive linked data set available for Scotland. It links Census data from 1991 onwards with vital events, education and NHS data for a 5%, nationally representative sample of the Scottish population (Boyle et al., 2009a). These data could have been utilised in this thesis but as it only began in 1991 it was not deemed to be an optimal source for trend analysis. The temporal aspect of the SLS is one of its major limitations especially when compared to the Office for National Statistics Longitudinal Study for England (LS) which is linked to Decennial Census records from 1971 onwards (Gayle et al., 2008).

Thirdly, socioeconomic measures derived from occupation are not directly applicable to people who are not currently in employment (Galobardes et al., 2006). One possible approach to overcome this could have been to obtain the occupation based social class of spouses and assign based on this. This approach can also be used when both spouses are employment, with the highest ranking category in the household taken, to give a more detailed picture of the magnitude of mortality differences between social classes. However it is an approach that is criticised: household level social class is often identical to individual class and is still unable to ascertain a social class category for unmarried, cohabiting couples or single person households (Erikson and Torssander, 2008). Being unable to assign everyone in the population to a social class was problematic in the context of this thesis: it aimed to analyse lifespan variation inequalities for the entire Scottish population. This is one of the advantages of the Carstairs score and the Scottish Index of Multiple Deprivation

which were considered for use within this thesis: they are measures applied to areas rather than to individuals and are applicable to the entire population.

3.2.3.2 Carstairs Score & the SIMD

The Carstairs score is measure of relative deprivation that was first derived from the 1981 Census. It is derived from four Census variables (table 6) taken from small areas called postcode sectors; overcrowding, male unemployment, no car ownership and low social class. A postcode sector is a group of postcodes that are the same, minus the last two characters (for example G41 3**). Postcodes and postcode sectors are subject to boundary changes over time meaning they are not coterminous: a postcode and postcode sector boundaries are not necessarily the same over time (National Records of Scotland, 2016b). Table 5 details the number of postcode sectors in Scotland, the mean population size and the standard deviation at each Census year.

Table 5 Postcode sector, population size and standard deviation

Year	Total number of postcode sectors	Mean population size (SD)
1981	1010	4982.47 (3952.18)
1991	1001	4993.02 (3698.52)
2001	1010	5011.89 (3437.63)
2011	1012	5232.61 (3567.34)

The composite of the four variables used (see table 6) are considered to be a summary measure of population deprivation suitable for constructing a score which quantifies the level of relative deprivation in different areas (Carstairs and Morris, 1990). Although the formal definitions of the variables used for the

Carstairs score have changed between Census years the scores for each area over time are highly correlated. This suggests that the underlying information the variables aim to capture is similar or that deprivation has remained stable over time (Leyland et al., 2007a). However the relevance of the variables for capturing the meaning of deprivation across contexts and changes over time has been questioned (Norman, 2010). Criticisms have focused on the importance car ownership has for individual experiences of deprivation being fundamentally different between rural and urban contexts, and for overcrowding to occur out of choice and for cultural reasons rather than simply being a marker of deprivation (Fischbacher, 2014).

Table 6 Carstairs Score variables (Brown et al., 2014)

Carstairs Score	Census variables
Overcrowding	Persons living in a private household with a density of more than one person per room, as a proportion of all people in private households
Male unemployment	Economically active males seeking or waiting to start work, as a proportion of all economically active males
No car ownership	Persons in private households which do not own a car, as a proportion of all people in private households
Low social class	Persons in private households with an economically active head of household in social class IV or V, as a proportion of all people in private households with an economically active household reference person

3.2.3.3 Calculating the Carstairs score

The Carstairs score is a standardised index of area deprivation which is calculated from the unweighted combination of the four variables (Table 6) standardised using z-scores. A z-score is a statistical method for measuring the relationship between observations for a population sub-group and the mean of those observations for the total population. In the case of the Carstairs index the score for a variable in a postcode sector and the mean score for that variable for Scotland. A z-score of zero means the score is the same as the population mean score. A higher score indicates higher deprivation and a lower score (below zero) indicates lower deprivation.

The method used is as follows:

1. Calculate the proportion of each of the four variables for each postcode sector
2. Calculate the mean and standard deviation of the four variables for all of Scotland
3. Compute the z-score for each component variable by postcode sector
4. Add together the four z-scores for each of the component variables by postcode sector

Table 7 gives an example of how the Carstairs z-score is the sum of the standardised values of each component variable using data for 1991 for postcode sector G41 3.

Table 7 Example of calculating Carstairs score, Postcode sector G41 3 (1991)

Census variable		Mean	SD	calculation	z-score
overcrowding	0.0816	0.074	0.044	=(0.0816-0.074)/0.044	0.173
unemployment	0.0981	0.13	0.084	=(0.098-0.13)/0.084	-.381
No car	0.396	0.338	0.178	=(0.396-0.338)/0.178	0.326
Low social class	0.116	0.208	0.086	=(0.116-0.208)/0.086	-1.07
Overall Carstairs score for G41 3				=0.173+-0.381+0.326+-1.07	-0.952

The continuous Carstairs index scores are often categorised into quintiles (fifths of the distribution). The category cut-offs can delineate equal numbers of areas (postcode sectors) in each quintile or equal numbers of person. The latter 'population weighted quintiles' are preferable due to the uneven distribution of persons across postal sectors. Table 8,9,10 and 11 shows there are around 1 million persons in each quintile at every Census year.

One important limitation of the Carstairs Score is that the variables it is derived from are only collected once every ten years. This prompted the need for a measure that could be updated more frequently. This issue was addressed with the introduction of the SIMD in 2004.

3.2.3.4 SIMD

The SIMD is applied to small areas called datazones, each containing between 500-1000 household residents. Each datazone aims to reflect physical boundaries, natural communities and households with similar social characteristics (Flowerdew et al., 2007). The most recent publication was SIMD16 and included 6,976 datazones. The mean population size of a datazone in SIMD16 was 766.57 (SD 188.25) (The Scottish Government, 2016).

The SIMD takes 38 indicators of deprivation and collates these into 7 domains: employment, income, health and education; skills, training, and geographic access to services; crime and housing (Scottish Executive, 2006). Many of the indicators are updated annually and the domains are not solely reliant on Census data which are inherently costly. These are some of the advantages of the SIMD over the Carstairs score (Bailey et al., 2003b). However the full SIMD includes a health domain which is derived from indicators of mortality and morbidity, thus it is not suited for use in analyses trying to associate health and deprivation. Health inequalities research tends to apply the income domain of the SIMD only to overcome this problem (Seaman et al., 2015). This is deemed appropriate because the income domain is one of the most heavily weighted domains and is highly correlated with the overall score. On reflection this can make the Carstairs score more appropriate for analysing health inequalities at an area level as it intentionally sought out variables that were strongly associated with health but does not include a health dimension, meaning the full score can be applied.

Despite the empirical differences between the Carstairs score and the SIMD these approaches are both highly regarded for attempting to incorporate a multitude of measures to reflect the multifaceted nature of deprivation that exists across structures in society (Townsend, 1987). They build upon Townsend's (1987) concept of relative deprivation which emphasises that it is the potential individuals have to access the resources required to enable them to participate in the manner expected by society. Rather than only the potential to access material resources it is the potential to engage in relationships, partake in roles and adopt customary behaviours (Townsend, 1987). Deprivation

is defined by Townsend (1987) as 'a state of observable and demonstrable disadvantage relative to the local community or wider society to which an individual or group belongs'. It is distinct from the concept of absolute poverty because it is relative to the prevailing conditions. This means that the concept of deprivation is unique to every known society and its interpretations can evolve over time (Townsend, 1979).

3.2.3.5 Individual level and area level interpretations

The critical evaluation of the RGSC, NS-SEC, Carstairs score and SIMD has illustrated some of the theoretical and empirical differences between measures of socioeconomic position. These measures also differed in terms of whether they aimed to act as a proxy for area level determinants or as proxy for individual level determinants which require different interpretations that should not be confused. An example helps to highlight the contention between area level proxy measures which 'capture characteristics of populations' and individual level proxy measures which 'capture characteristics of individuals' (Leyland et al., 2007a). GPs aiming to reduce inequalities between individuals by providing preventative screening programmes may rely on the SIMD to target those deprived, they may target an individual in a deprived area who is actually well-off. So relying on an area level measure to reduce health inequalities between individuals is problematic if there is an assumption that the underlying characteristics of the population are socially homogenous (Fischbacher, 2014). Area level measures can therefore be accused of overlooking the fact that deprived and non-deprived individuals do not exclusively reside in deprived and non-deprived areas (Leyland et al., 2007a).

These limitations are counterbalanced by some empirical advantages. For instance health inequalities research reports findings for a range of geographical levels. Most of these higher geographies are built up from postcodes which are deemed to be the most convenient way of allocating an area reference to individuals based on their home address. Since home address is recorded for individuals across services, for example on hospital admissions and GP registers, this unit of measurement has an immediate advantage over social class or occupation that is not. Therefore area-level measures are valuable for increasing the opportunities available to researchers to help explain the causes of health inequalities (Carstairs and Morris, 1990). Area level measures can also provide an opportunity to capture subgroups of the population that were historically excluded from traditional measures of occupation based social class, in particular women, younger age groups and the unemployed. Although a household level measure also has the potential to do this it was not possible to derive a household level measure of socioeconomic position from the death data that was obtained.

3.2.3.6 Subsection summary

The literature review described the body of evidence demonstrating the social gradient that exists for mortality, particularly in Scotland. Assessing the socioeconomic gradient for lifespan variation will add to our understanding of mortality inequalities in Scotland. This has not previously been done despite long running data being widely available. Complete period life tables, constructed from death counts and population estimates are required to calculate lifespan variation. The previous subsection critically evaluated the options available to stratify the population of Scotland by socioeconomic position. Individual level

social class and area level socioeconomic deprivation have been compared and the strengths and weakness of each discussed.

The Carstairs Score was deemed to be most appropriate for the following reasons. It could be utilised to cover the time period between 1981 and 2011, albeit at ten year increments, which captures the time period when life expectancy improvement in Scotland began to stall (McCartney et al., 2012b). It had a clear conceptual basis prior to being constructed. The variables included were intentionally selected to account for inequalities in health which is the focus of this thesis (Carstairs and Morris, 1990). The Carstairs scores can be aggregated to population weighted deprivation quintiles (as noted above) allowing for a consistent meaning over time; even though deprivation will have changed over time there is always a conceptual 20% most deprived compared with a 20% least deprived. And finally a Carstairs Score for each part postcode sector in Scotland has previously been calculated systematically and were readily available at the time of research (MRC/CSO Social and Public Health Sciences Unit University of Glasgow, 2016).

The following subsection details the data management processes that were carried out to produce life tables for all-cause mortality by quintiles of deprivation, as measured using the Carstairs Score. The Carstairs score data file is freely available online (MRC/CSO Social and Public Health Sciences Unit University of Glasgow, 2016).

3.2.4 Constructing all-cause mortality life tables

Life tables are constructed from age specific all-cause mortality rates that are calculated from death counts and Census population estimates. Table 8 to table

11 detail the aggregated population estimates and death counts used to construct deprivation specific life tables at each Census year.

Table 8 1981 aggregated population estimates and death counts used to construct deprivation specific life tables

	Population estimates NRS commissioned Request				Total pop variable from Carstairs	All cause death counts		
		Males	Females	Persons [1, 2]	Persons	Males	Females	Persons [3]
1981	data total	2,428,471	2,606,841	5,035,312	5,032,295	94,143	96,318	380,713
	excluded from analysis	2,524	1,162	3,686	-	90	119	209
	quintile 1 (least dep.)	480,582	523,678	1,004,260	1,004,457	16,748	19,230	35,978
	quintile 2	483,834	516,776	1,000,610	1,000,695	18,335	19,279	37,614
	quintile 3	480,644	512,768	993,412	993,206	18,088	18,199	36,287
	quintile 4	493,061	532,936	1,025,997	1,026,153	20,602	20,412	41,014
	quintile 5 (most dep.)	487,826	519,521	1,007,347	1,007,784	20,280	19,079	39,359
	total included in analysis	2,425,947	260,5679	5,031,626	-	94,053	96,199	19,0252

[1]1981 population estimates by individual postcode included 21 Armed forces postcodes which a Carstairs Score is not calculated for. [2]Postcode data included 18 postcodes which could not be matched to higher level geography. [3]Deaths excluded from the analysis in 1981, 1991 and 2001 did not contain adequate information for assigning geography

Table 9 1991 aggregated population estimates and death counts used to construct deprivation specific life tables

1991	Population estimates NRS commissioned Request				Total pop variable from Carstairs	All cause death counts		
		Males	Females	Persons	Persons	Males	Females	Persons [3]
1991	data total	2,391,961	2,606,606	4,998,567	4,998,012	580,22	62,957	120,979
	excluded from analysis	275	28	303		196	560	756
	quintile 1 (least dep.)	493,588	528,558	1,022,146	1,022,146	9,758	12,053	21,811
	quintile 2	470,435	505,303	975,738	975,738	11,002	11,947	22,949
	quintile 3	480,450	521,528	1,001,978	1,001,726	11,255	12,193	23,448
	quintile 4	473,864	523,921	997,785	997,785	12,300	12,946	25,246
	quintile 5 (most dep.)	473,349	527,268	1,000,617	1,000,617	13,511	13,258	26,769
	total included in analysis	2,391,686	2,606,578	4,998,264	-	57,826	62,397	120,223

[3]Deaths excluded from the analysis in 1981, 1991 and 2001 did not contain adequate information for assigning geography

Table 10 2001 aggregated population estimates and death counts used to construct deprivation specific life tables

	Population estimates NRS commissioned Request				Total pop variable from Carstairs	All cause death counts		
		Males	Females	Persons	Persons	Males	Females	Persons [3]
2001	data total	2,432,494	2,629,517	5,062,011	5,062,011	81,611	90,056	171,667
	excluded from analysis	0	0	0	-	36	37	73
	quintile 1 (least dep.)	493,995	520,725	1,014,720	1,014,720	12,905	16,149	29,054
	quintile 2	490,600	521,874	1,012,474	1,012,474	15,332	17,181	32,513
	quintile 3	484,529	524,910	1,009,439	1009439	16266	18285	34551
	quintile 4	484,503	528,994	1,013,497	1,013,497	17,151	18,307	35,458
	quintile 5 (most dep.)	478,867	533,014	1,011,881	1,011,881	19,921	20,097	40,018
	total included in analysis	2,432,494	2,629,517	5,062,011	-	81,575	90,019	17,1594

[3]Deaths excluded from the analysis in 1981, 1991 and 2001 did not contain adequate information for assigning geography

Table 11 2011 aggregated population estimates and death counts used to construct deprivation specific life tables

	Population estimates NRS commissioned Request				Total pop variable from Carstairs	All cause death counts		
		Males	Females	Persons	Persons	Males	Females	Persons
2011	data total	2,567,444	2,727,959	5,295,403	5,295,403	77,340	84,401	161,741
	excluded from analysis	0	0	0	-	0	0	0
	quintile 1 (least dep.)	510,021	546,807	1,056,828	1,056,828	17,670	18,201	35,871
	quintile 2	512,340	546,570	1,058,910	1,058,910	15,891	16,946	32,837
	quintile 3	512,778	545,654	1,058,432	1,058,432	15,290	17,180	32,470
	quintile 4	513,485	544,783	1,058,268	1,058,268	15,089	17,083	32,172
	quintile 5 (most dep.)	518,820	544,145	1,062,965	1,062,965	13,400	14,991	28,391
	total included in analysis	2,567,444	2,727,959	5,295,403	-	77,340	84,401	161,741

Detailed individual death record data for Scotland since 1974, produced by NRS, are held by the MRC/CSO Social and Public Health Science Unit, University of Glasgow. Population estimates by single year of age and geographical level derived from the Census in 1981, 1991, 2001 and 2011 were obtained via a commissioned request from NRS.

The population estimates files obtained were not consistent across all four Census year (1981, 1991, 2001, and 2011) in terms of level of geography and final open ended age group. Extensive data management was required in order to match population estimates to death data and Carstairs Score on postcode sector and then aggregate into population weighted deprivation quintiles. It was also only possible to match these data with an open ended age category of 85+. A complete period life tables require the final open ended age category to be 110+. Therefore the mortality rates beyond age 85 + had to be modelled.

The data management processes that were carried out will now be described and the implications of missing data reflected upon. The data management processes are summarised in figure 9.

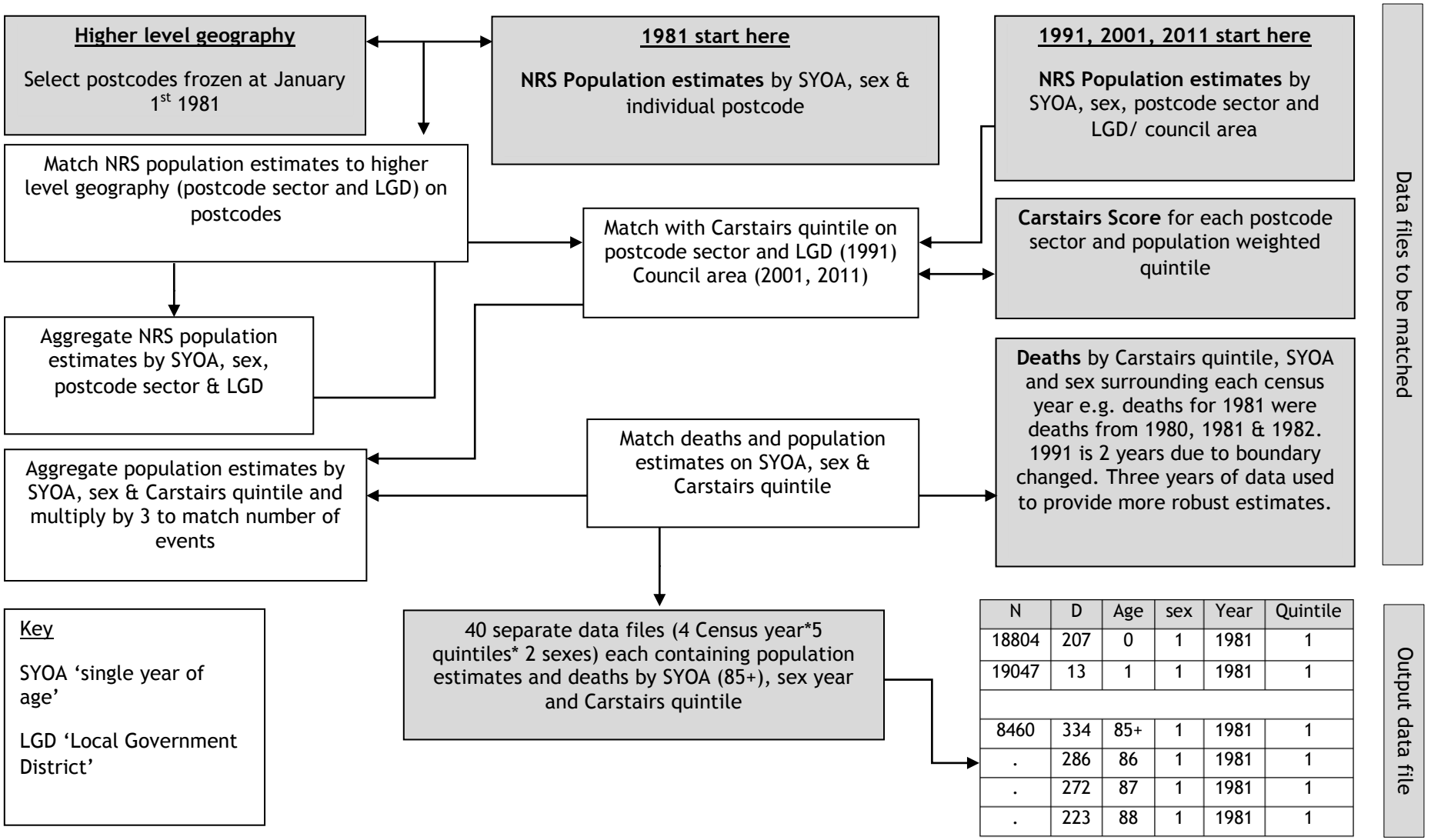


Figure 9 Summary of data management processes

3.2.4.1 1981

1981 population estimates by sex and SYOA (up to age 85+) were obtained for each individual postcode in Scotland (117,102 postcodes in total). Extensive data management processes were required to aggregate the population estimates by single postcode to postcode sectors in order to match with the Carstairs deprivation score. Deaths used for the Census year 1991 were from 1991 and 1992 to increase the number of events. Population estimates were multiplied by two accordingly. All other Census years used three years' worth of death data and population estimates were multiplied by three accordingly. Deaths occurring in 1990 had to be excluded because of changes to geographical boundaries.

3.2.4.2 Frozen postcodes

The geographical area attached to an individual postcode can change over time: the size of the geographical area can be changed, the postcode can be removed entirely from circulation, or reassigned to another area ('postcode recycling'). This means that postcodes are only unique geographical identifiers at one point in time. The population estimates derived from the Census are therefore covered by a standard 'frozen geography policy' (National Records of Scotland, 2013a). This means the underlying postcode database is 'frozen' as it stands on January 1st of each Census year and all Census outputs are based on the postcodes active on this date. The Carstairs Score is based on postcode sectors as derived from the Census meaning that the underlying postcodes were also frozen as of January 1st.

The postcodes detailed in the population estimates therefore uniquely identified each observation for 1981 but needed to be matched, and then aggregated, by higher level geography data. This is because the Carstairs Score is based on part-

postcode sector split by Local Government District (LGD) and cannot be matched directly to individual postcodes.

The total number of individual postcodes used in Scotland, at the time of research, was 184,560. Of these 117,102 postcodes were active on January 1st 1981 and had population estimates. 117,063 were successfully matched with higher level geography data. Of the 39 unmatched postcodes 21 were identified as 'Armed Forces' and were removed as the Carstairs Score is not calculated for 'Armed Forces'. There was no higher level geography data for the remaining 18 postcodes to be matched with. The total number of people residing in postcodes that that could not be matched with higher level geography data was 2,104 which represents 0.04% of the total Scottish population estimates in 1981. Therefore it is valid to assume that excluding these from the population denominator would not have greatly impacted the results.

Matching individual postcodes with higher level geography data then enabled the population estimates to be aggregated by postcode sector and LGD. The individual postcodes were aggregated up to 1,136 postcode sectors but of these only 1,010 postcode sectors have a Carstairs score. This is because some postcode sectors in Scotland have very small population sizes meaning that it would not be valid to calculate a Carstairs score. The 126 postcode sectors that did not match with a Carstairs score contained 1,582 individuals or 0.03% of the total population of Scotland in 1981.

Once population estimates were aggregated for the remaining 1,010 postcode sectors they were matched to the Carstairs score. The Carstairs score data set contained a total population variable which provided an opportunity to validate

the aggregation and matching processes applied to the population estimate data by single year of age obtained from NRS for 1981. The total population variable for each postcode sector in the Carstairs score was not identical to the sum of the single year of age population estimates obtained from NRS for 84 postcode sectors. The difference equated to 699 fewer individuals in the NRS commissioned data. A small difference was expected. This is because the NRS commissioned data would have been updated more recently than the total population variable in the Carstairs file. It is not uncommon for population estimates derived from the Census to be revised at a later date which would explain this small discrepancy.

A small number of deaths, 209 in total, had to be excluded from the 1981 analysis. These deaths represented 0.05% of all deaths in Scotland in 1981. They were excluded because the death certificate data were not detailed enough to attach the death to the correct geography for matching with Carstairs score. At this point it is appropriate to outline how a death is assigned to a geographical location in Scotland.

3.2.4.3 Geographical location of deaths

There can be up to three sources of geographical information available on a death certificate: place of normal residence, place of death, and place of occurrence. Place of normal residence is the deceased most recent address of residence. Place of death should reflect exactly where the person died, this could be a detailed hospital address or it could be a location such as area of countryside or motorway. Place of occurrence is used in the case of external causes of death and records where the event occurred if known. It does not specify a precise location but

rather identifiable places such as school, residential home or construction area (General Register Office for Scotland, 2016).

NRS adopts the following policy for assigning geography to each death. Deaths of Scottish residents that occur in Scotland are assigned to their place of normal residence. Deaths of non-Scottish residents that occur in Scotland are assigned a geography based on their place of death. Deaths of Scottish residents that occur outside of Scotland are currently not included (General Register Office for Scotland, 2015).

3.2.4.4 1991

1991 population estimates by sex and SYOA (up to age 90+) were obtained for each postcode sector and LGD. This removed the need to match the data with higher level geography and could be matched immediately with the Carstairs score and matched with deaths. Two postcode sectors included in the NRS commissioned population estimates were for shipping addresses and were excluded as no Carstairs score would have been calculated. The population estimates for these two postcode sectors contained 303 individuals or 0.01% of the Scottish population in 1991. The NRS commissioned population estimates showed 252 more individuals than the Carstairs population.

A total of 756 deaths could not be included in the analysis for 1991, again due to inadequate geographical information on the death certificates. These deaths represented 0.6% of the total number of deaths in 1991.

3.2.4.5 2001 and 2011

Population estimates for 2001 and 2011 were obtained by sex and SYOA for each postcode sector and LGD. The open ended age group in 2001 was 85+. The older

age groups in 2011 were grouped as follows: 85-89, 90-94 and 95+. These were matched with the Carstairs score and matched with deaths. No population data had to be excluded from the analysis for 2001 or 2011. There was also no difference between the NRS commissioned population estimates and the Carstairs population estimates for 2001 and 2011.

73 deaths could not be included in the analysis for 2001. These deaths did not have adequate information on geography and represented 0.04% of the total deaths in Scotland in 2001.

At this stage the open ended age group for the population estimate was not consistent across the four census years and it was not yet possible to construct complete life tables stratified by socioeconomic position. Deaths were by single year of age and there was no open ended age group, the last age was the oldest death that had occurred. A final open ended age category of 85+ was applied to the population estimates and deaths across all Census years and the mortality rate past this age modelled. The modelling process is now described.

3.2.5 Modelling mortality rates at extreme old age

To construct complete period life tables a mortality rate for each SYOA up to age 110+ was required. Given that the data available provided a number of known parameters about the mortality experience and that the population level mortality rate was available from the HMD up to 110+ it was possible to model a precise description of the deprivation specific mortality rate beyond ages for which data were available. The HMD provide a standard protocol for splitting open age categories and smoothing death rates (Wilmoth et al., 2007). The method applied

in this thesis used the results for Scotland as a whole from the HMD method and fitted the shape of the curve to each deprivation quintile.

Figure 10 shows the log of mortality rates for males in 1981 by deprivation quintile. These log mortality rates were calculated from the Census population and mortality data for ages ≤85. The log mortality rate for all males in Scotland in 1981 from the HMD is plotted as an external comparator. This is for ages 0-110+

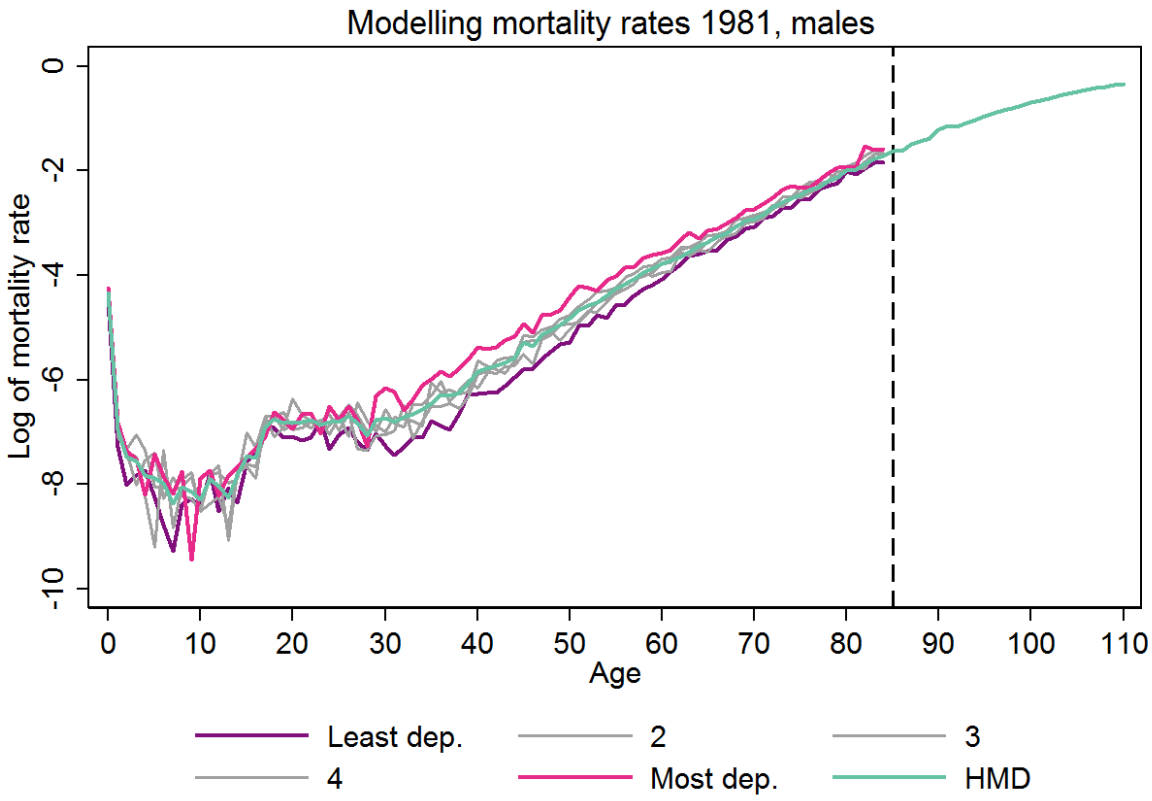


Figure 10 Log of mortality rate by age and deprivation quintiles (≤84) compared to HMD (age 0-110+), 1981, males

The following briefly summarises the history of predicting mortality rates beyond a final age whose values cannot be calculated directly. It is followed by a discussion

on the modelling process that was used in this thesis alongside a worked example. This process was necessary in order to construct complete deprivation specific period life tables up to age 110+ years old.

3.2.5.1 Gompertz's law of mortality

The relationship between mortality and age is considered to be one of the oldest topics studied in demography (Preston et al., 2001). One of the earliest insights into mortality and age was by Gompertz (1825) who was aiming to evaluate the age at which individuals are most likely to die. He produced a mathematical formula which suggested a population's mortality rate increases exponentially with age and found that the likelihood of dying doubles with every eight to ten years of age up until 80 years old (Carnes and Olshansky, 2002). The formula showed that the log of the mortality rate is a linear function of age (Preston et al., 2001). This finding demonstrated the regularity and predictability of death and was labelled the 'law of mortality'.

However Gompertz only intended the law of mortality formula to be used to describe the relationship between age and underlying risk of mortality. This meant it did not allow for causes of death attributable to external accidents or infections which are assumed to be independent of age (Preston et al., 2001). Makeham (1860) adapted the law of mortality to account for the constant age-independent component of mortality. However this age-independent component tends to only have a negligible impact in developed countries with relatively low mortality.

It is further argued that Gompertz's law of mortality over-predicts mortality at the youngest ages and that it overlooks the fact that the rate of increase in the mortality rate at extreme old ages may slow. Despite these critiques Gompertz's

law of mortality has held over time and across populations, and is especially accurate across ages 30 to 80 years old. Therefore Gompertz's law of mortality demonstrated that it is possible to use age to model the log mortality rate if objective data are unavailable.

Considering all of this the log mortality rate could be modelled as a linear function of age. However an interaction could be included to allow the relationship between age and the log mortality rate to slow, or a better model on the log scale could be achieved with a Poisson regression. These three approaches were tested to establish which was most appropriate for modelling a mortality rate beyond the ages for which data were available for Scotland.

3.2.5.2 Modelling mortality rates

The predictor variables used were age (continuous) and deprivation (categorical). Models were further stratified by year (1981, 1991, 2001 and 2011) and sex as the mortality experience will have changed over time and is known to differ between males and females.

In total the mortality rate was modelled across the 40 different data sets (4 years× 5 quintiles×2 sexes) that were produced from matching the population estimates and death counts on Carstairs score (see figure 9). The modelled predictions were evaluated by plotting them against the mortality rates for the total population of Scotland from the HMD up to ages 110+. The final model selected for use was the Poisson model which included a fractional polynomial for age. The stages carried out to justify this decision are described in the following section using data for males in 1981 as a worked example.

3.2.5.3 Linear regression of log mortality rate on age (model 1)

The first model tested whether the relationship between age and the log mortality rate was linear. The log mortality rate was predicted for ages 60 to 110+, irrespective of deprivation. Age 60 was chosen as the relationship between age and mortality rate was likely to be linear past this point.

The mortality rate data that were available from the HMD for ages 60 to 110 and the fitted linear regression are shown in figure 11. From this plot it appears that the linear regression is a good fit but may not accurately account for mortality at extreme old ages where the HMD data shows the linear increase in log mortality rate slowing.

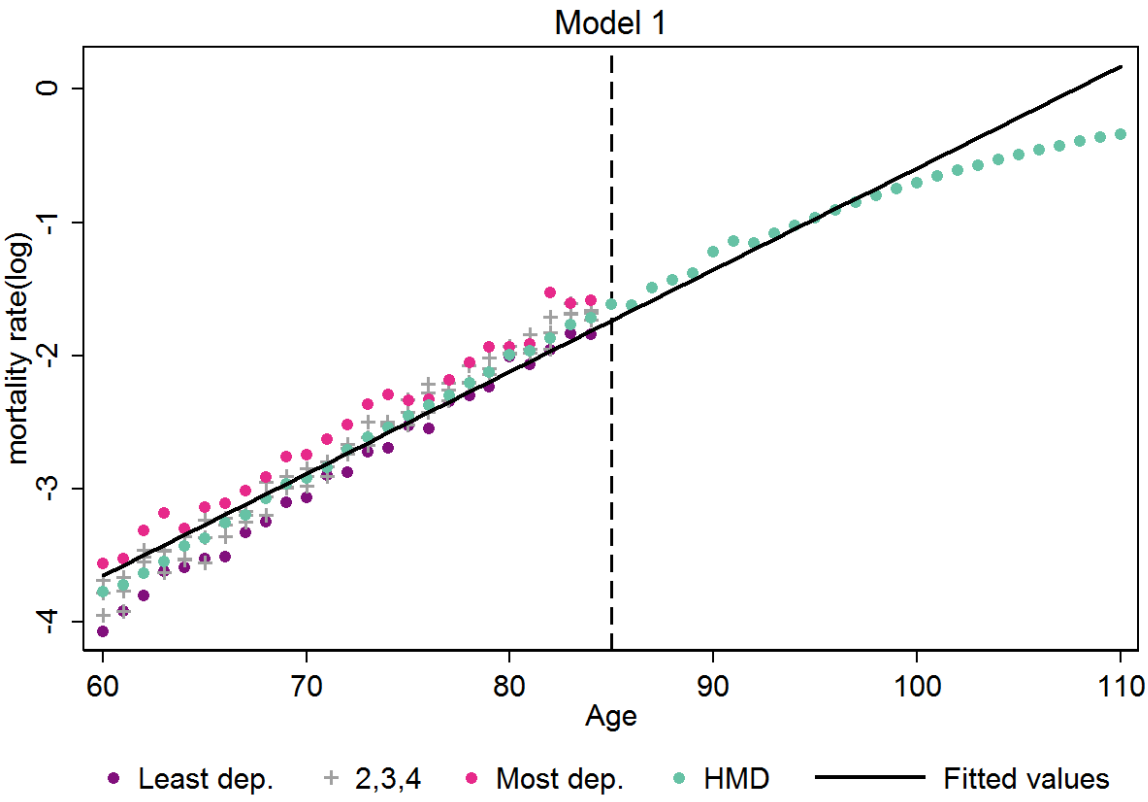


Figure 11 Log of mortality rate by age from HMD (up to age 110+) compared with fitted linear regression

In addition it was deemed to be overly simplistic because the model did not include an indicator for deprivation which was assumed to be important because the literature has established a strong association between mortality and socioeconomic deprivation (Carstairs and Morris, 1989b, Townsend, 1987). This assumption appears to be valid in figure 12.

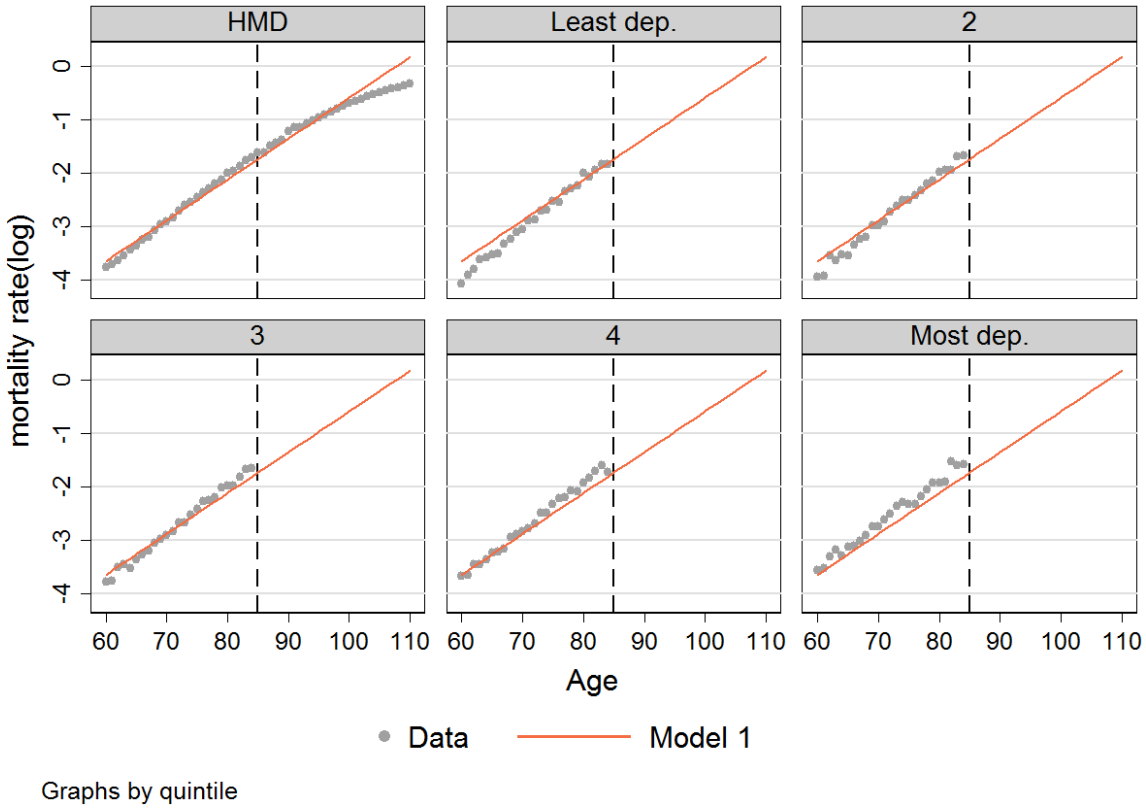


Figure 12 Log mortality rate by age for HMD (up to age 110+) and for deprivation quintiles (up to age 85+) compared with fitted linear regression (Model 1)

Figure 12 shows that model 1 may overestimate the log mortality rate compared to the data from ages 60 to 85 for the least deprived and may under estimate for the most deprived. The data line and the model 1 line are not as close across ages 60 to 85 for the two extreme deprivation groups, the most and least deprived.

3.2.5.4 Linear regression of log mortality rate on age with age interaction (model 2)

Model 2 aimed to account for the slowing increase in log mortality rate at extreme old ages by allowing for a non-linear relationship between age and the log mortality rate by including age and age squared, it also includes a categorical indicator variable for deprivation.

The improved fit of the model is demonstrated in figure 13. It is difficult to distinguish between the scatter plots for the data and the line for model 2 for the ages where both were available. This indicates that the model would accurately predict the log mortality rate into the ages where data were not available assuming that the same pattern applies.

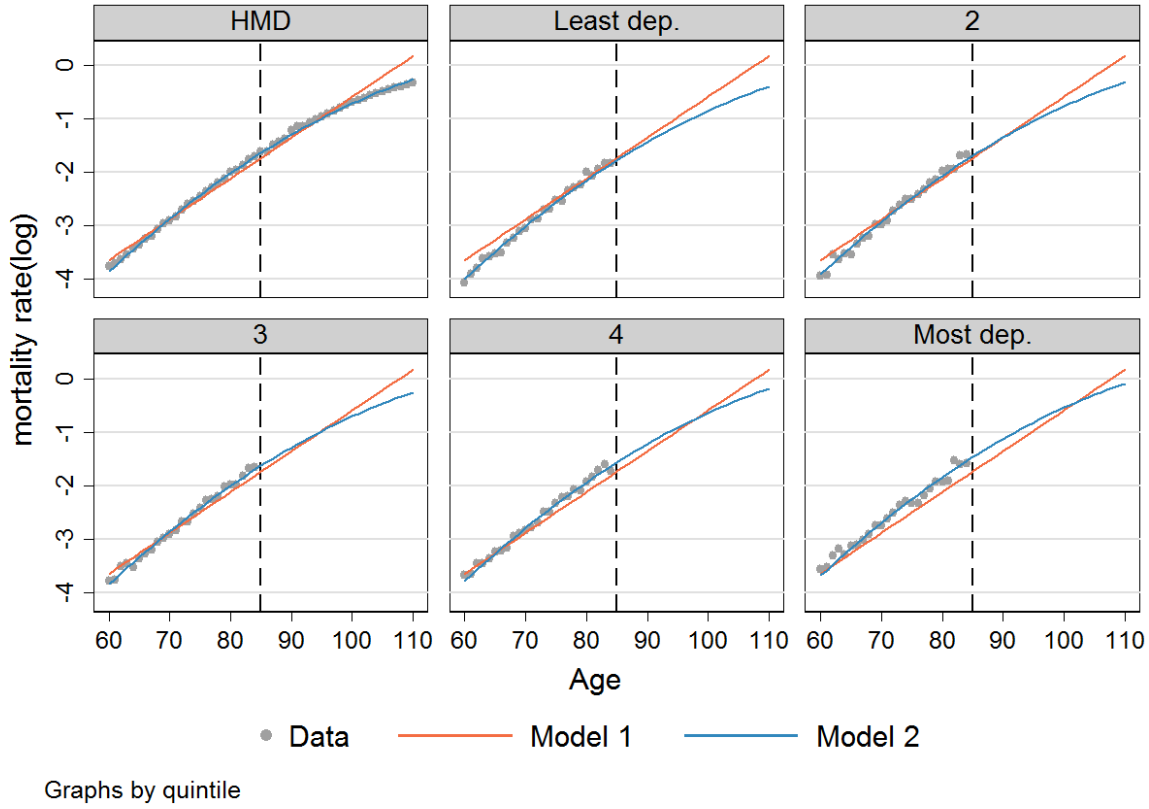


Figure 13 Log mortality rate by age for deprivation quintiles (up to age 85+) compared with fitted linear regression (model 1) and fitted linear regression controlling for deprivation and including age interaction (model 2)

However the linear regression models predict the log mortality rate which means that the results would need to be back transformed to obtain a mortality rate for including in a full life table (Yang, 2012). A Poisson regression model removes this issue and as a result can be considered more appropriate than a linear regression model. It is also possible to use a fractional polynomial to account for the non-linear relationship at older ages which selects the 'best fit' for age rather than simply allowing the linear relationship to change for different ages. These issues were addressed in model 3a and 3b. Model 3b was the final model selected.

3.2.5.5 Poisson regression of log mortality rate on age

A Poisson regression models the log of the expected count as a function of the predictor variables (Yang, 2012). It removed the need to back transform the log mortality rate as it predicts the mortality rate rather than the log (model 1 and model 2). The log mortality rate taken from the predicted mortality rate from the Poisson regression model 3a and log mortality rate predicted from the linear regression model 2 are therefore very similar, demonstrated in figure 14.

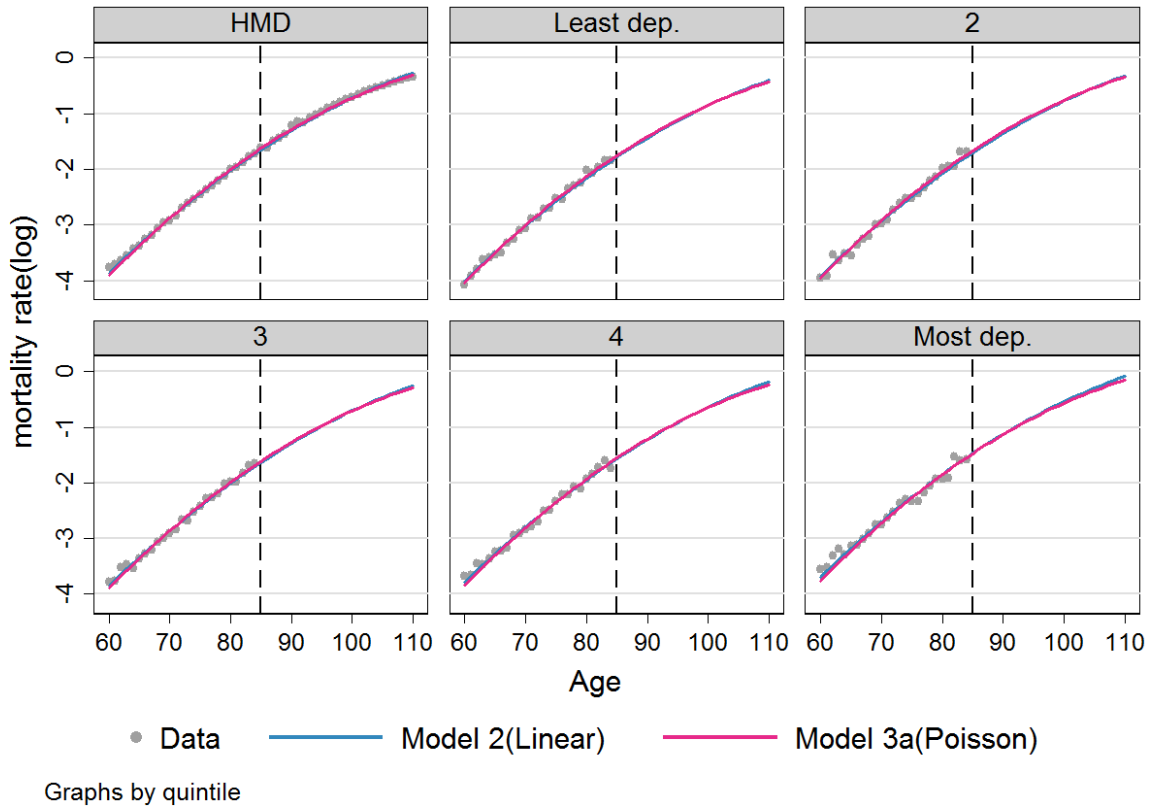


Figure 14 Log mortality rate by age for deprivation quintiles (up to age 85+) compared with fitted linear regression controlling for deprivation and including age interaction (model 2) and Poisson regression (model 3a)

Using age squared is a valid approach however the model could be improved by including the function of age as a fractional polynomial. Fractional polynomials provide a more flexible parameterization for continuous variables which produce a wider range of curved shapes than those available from linear or quadratic functions (Stata, 2016, Sauerbrei et al., 2006). Model 3b therefore included a fractional polynomial function for age (term$^{(2)}$,term $^{(3)}$) in the Poisson regression for predicting the morality rate past age 85+ across deprivation quintiles. The predicted mortality rate from the Poisson regression (model 3a) and the mortality rate predicted from the Poisson regression model including the fractional polynomial function for age (model 3b) are compared in figure 15 alongside the data.

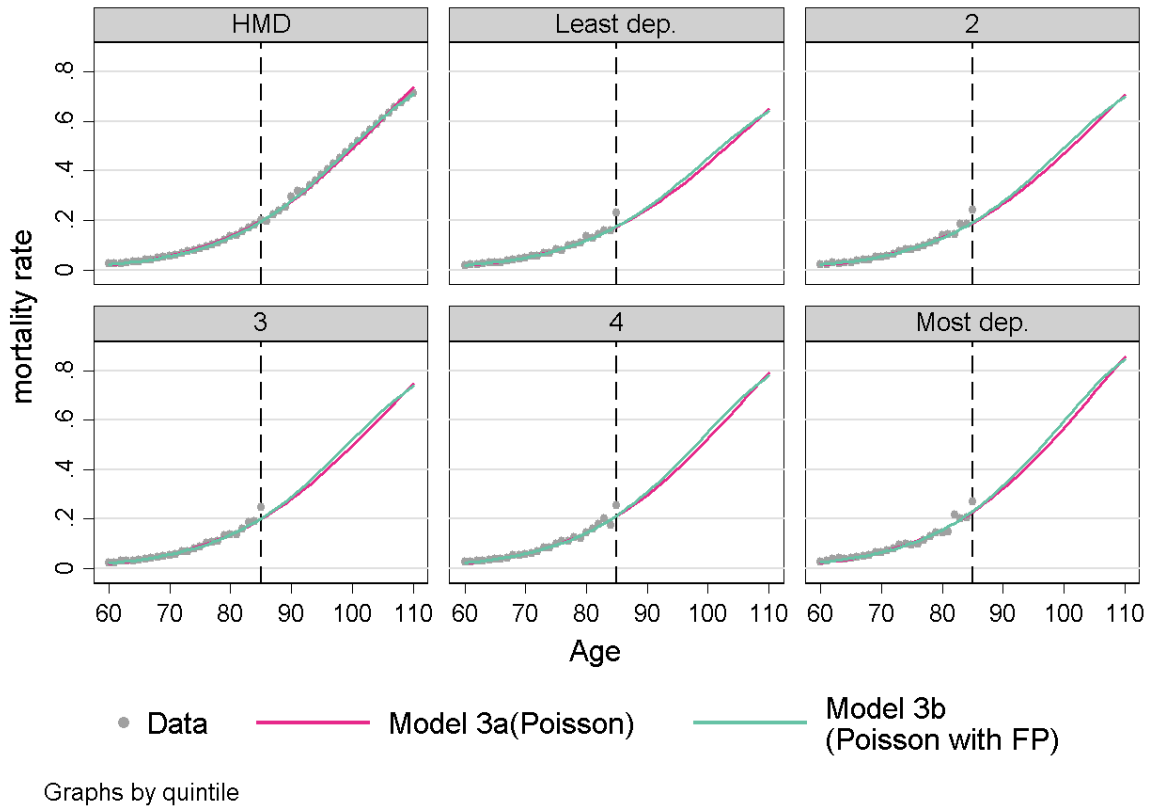


Figure 15 Mortality rate by deprivation quintile (up to age 85+) compared with Poisson regression (model 3a) and Poisson regression with Fractional Polynomial function for age (model 3b)

The results from all the models are compared with the data for the HMD in figure 16 for ages 85 to 110+, the ages for which data were unavailable.

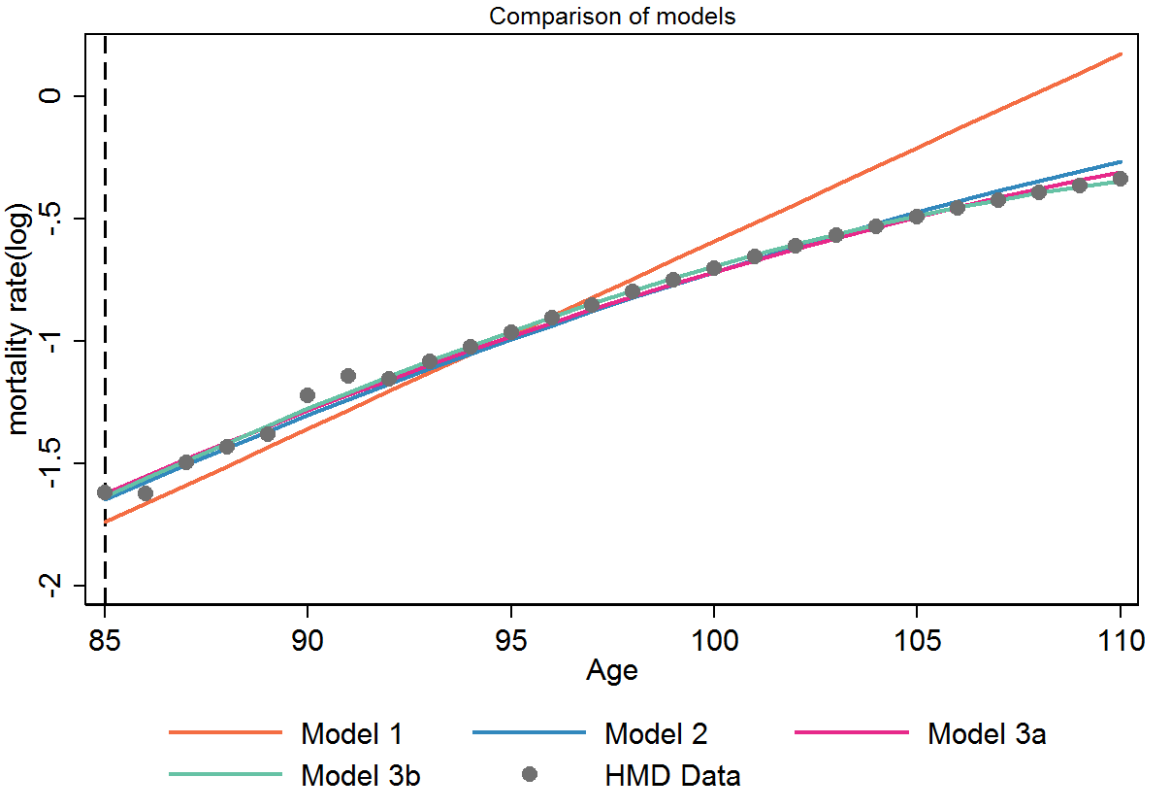


Figure 16 Log mortality rate by age for HMD (up to age 110+) comparing fitted linear regression (model 1), fitted linear regression controlling for deprivation and including age interaction (model 2), Poisson regression (model 3a), and Poisson regression with fractional polynomial (model3b)

Both the linear model with age squared (model 2) and the Poisson models (model 3a and model 3b) fit the mortality rate from the HMD better than the simple linear regression (model 1). This is a strong indication that the Poisson model including a fractional polynomial function for age would provide reliable estimates for the mortality rates by deprivation quintile for which data could not be obtained. Model 3b was therefore applied to all 40 datasets (4 years×5 quintile×2 sexes).

Using these predicted mortality rates (for ages 85-110+) it was possible to produce full deprivation specific period life tables up to age 110+ years. These were required to calculate lifespan variation the mortality outcome of interest to this thesis. The following section describes e† the measure of lifespan variation analysed throughout this thesis.

3.2.6 Calculating lifespan variation

e† is the sum of remaining life expectancy at each age, weighted by the number of deaths at that age. This intuitive interpretation of e† is advantageous: it is the average number of years remaining at death and measures life years lost when a death occurs (van Raalte, 2011a).

This thesis reports lifespan variation from age 0 ($e_0^\dagger$). Other studies report lifespan variation conditional on survival to older ages (van Raalte et al., 2012, van Raalte et al., 2011, van Raalte et al., 2014). However these studies calculated lifespan variation for populations stratified by measures of occupation and education respectively. This meant that the measure of socioeconomic position being utilised was not applicable to younger ages: ages when people not yet have an established occupational status or have yet to complete their education. Reporting lifespan variation from age 0 is valid within the context of this thesis for the following reasons.

Firstly, the chapters in this thesis concerned with calculating lifespan variation stratified by socioeconomic position used an area measure of socioeconomic deprivation (Carstairs score) which is applicable to the whole population, regardless of age.

Secondly, existing studies have identified that Scotland's mortality disadvantage following the 1980s may be driven by an excess of deaths across ages as young 15 to 29 years old (Leyland et al., 2007b). These ages would have been truncated out of the analysis had lifespan variation contingent upon survival to age 31, the age used by (van Raalte et al., 2014), been applied.

Finally, life expectancy is commonly reported and interpreted from age 0: the expected length of life a new born can be expected to live given the current mortality conditions (Preston et al., 2001). Therefore it was valid to include all ages when studying lifespan variation in Scotland.

3.2.6.1 e† equation

Lifespan variation was measured using $e_0^\dagger$. This is the average remaining life expectancy lost at each age, multiplied by the number of deaths at that age, divided by the total number of deaths in the life table. For example in a population of 100,00, if 2,000 people die age 60, and life expectancy at 60 was 20 years and life expectancy at 61 years was 19 years, lost life expectancy for those dying age 60 would be $\frac{2{,}000\times(20+19)/2}{100{,}000} = 0.39$ years. Carrying out this equation for each age in the life table, and then summing the results for all ages, gives you the total years of life expectancy lost for the period that the life table refers to (Popham et al., 2013). $e_0^\dagger$ estimates were calculated in StataSE13 and the equation used in this thesis for $e_0^\dagger$ can be summarised as:

$$e_0^\dagger = \frac{\int_0^\omega f_x \, e_x \, dx}{\iota_0}$$

(van Raalte et al., 2014)

Where

f_x is the life table number of deaths at age x

e_x is the average life expectancy at age x and x+1

ι_0 is survivorship at the starting age of the integral (here, starting age is 0 years)

ω is the last open ended age (here 110+)

When $e_0^\dagger$ is deemed to be relatively high this means some members of the population are dying well before the average expected age at death and contributing large amounts of lost-years of potential life (van Raalte et al., 2014). If more members of the population are surviving to similar ages and dying closer to the expected average age at death then $e_0^\dagger$ will become smaller. If it was possible for everyone in the population to die at the same age then remaining life expectancy when death occurred would be zero and $e_0^\dagger$ would subsequently be zero.

e† is correlated with a number of other indices of lifespan variation. van Raalte and Caswell (2013) demonstrated the correlation between e† , the Gini-Coefficient, Theil's index, The mean logarithmic deviation, the Standard deviation, Variance and Interquartile range. However e† may be more sensitive to older age mortality. Therefore van Raalte et al. (2014) suggest that e† might show more conservative estimates of lifespan variation differences by socioeconomic deprivation because inequalities in lifespan variation tend to be driven by the differences in premature working age mortality which e† is less sensitive to. Overall the high correlation between measures of lifespan variation provides reassurance that the broad trends and substantive conclusions reported in this

thesis would have been similar irrespective of the statistical measure of lifespan variation used (Vaupel et al., 2011).

3.3 Section 2: Statistical tests, measures and methods

The overarching aim of this thesis is to add to our understanding of mortality inequalities in Scotland by measuring lifespan variation, a measure of total inequality. The previous section of this chapter described the data utilised to construct life tables in order to estimate lifespan variation. It concluded by describing e†, the measure of lifespan variation that is analysed throughout this thesis.

This second section discussed the statistical tests, measures and methods that were used to analyse the lifespan variation estimates. The chapters that these appear are summarised, once again, in figure 17 (right hand panel).

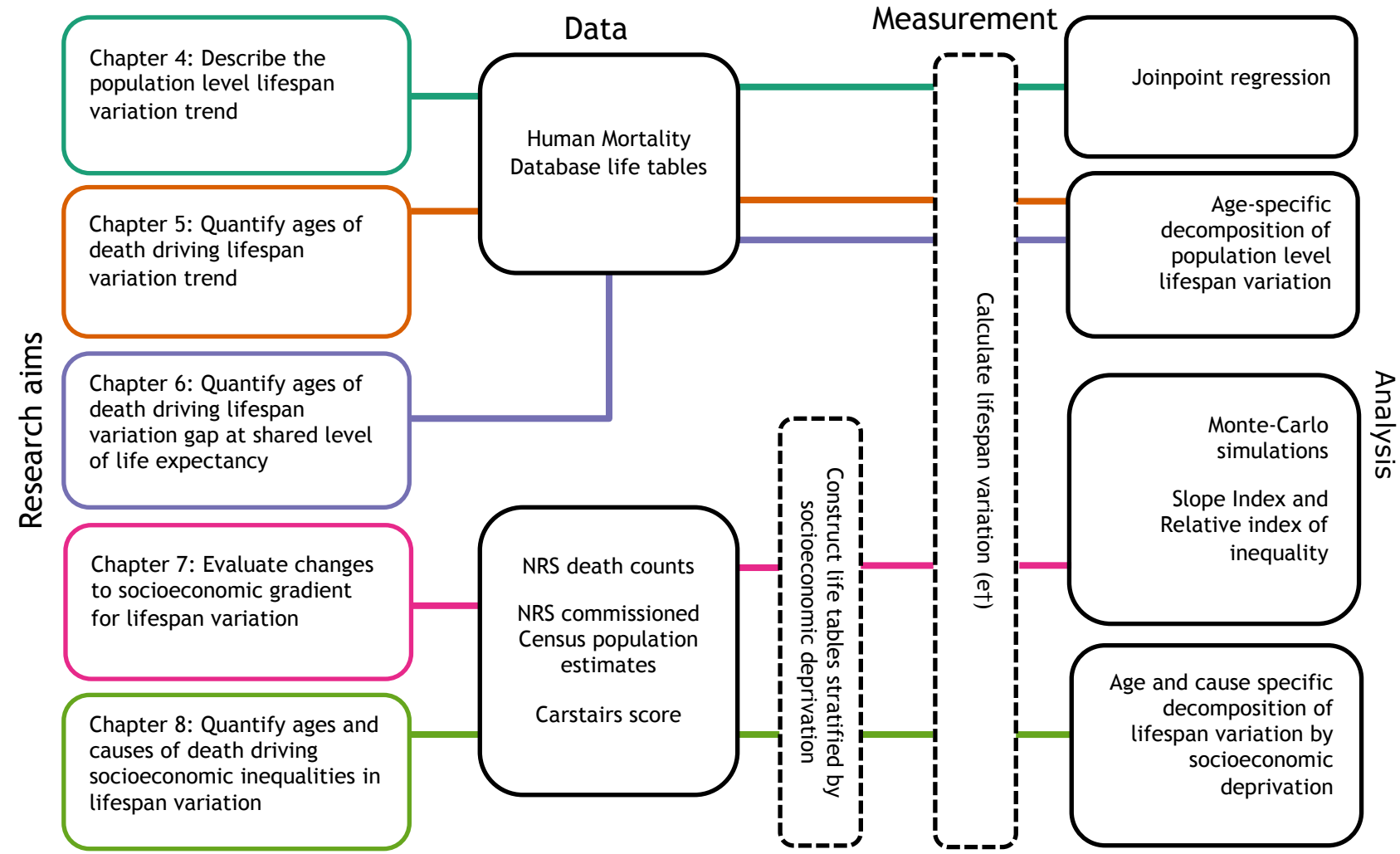


Figure 17 Summary of research stages

3.3.1 Joinpoint regression

Chapter 4 is concerned with assessing the change in the population level lifespan variation trend for Scotland, in a cross-national comparative context. Looking at Scotland's annual lifespan variation ranking within Western Europe since 1950 identifies if its ranking has changed over time and sufficiently answers the first research question. Joinpoint regression was then used to formally describe the changes in the lifespan variation trend data for Scotland and 16 comparable Western European countries. Joinpoint regression analysis answers the second research questions: it estimates the timing of any significant change in the magnitude or direction of a health outcome variable and quantifies the rate of change (annual percentage change) (Hegerl et al., 2013). The literature has referred to joinpoint regression analysis: piecewise regression, segmented regression, broken line regression, and multi-phase regression (Kim et al., 2000, Goovaerts and Xiao, 2011, Hegerl et al., 2013). This thesis refers to this method of analysis as joinpoint regression.

Joinpoint regression models were calculated using the software available from Surveillance Research Cancer Control and Population Sciences National Cancer Institute (2013). The lifespan variation estimates, calculated from the HMD life tables, were imported into the joinpoint regression software. The joinpoint analysis starts with a straight line and tests whether one or more statistically significant joinpoints should be added. The number of joinpoints tends to be specified at a maximum of three with a minimum of 5 observations between each (Goovaerts and Xiao, 2011, Hegerl et al., 2013). This avoids the joinpoints from being too close together or too close to either end of the time series. In practical

terms this also decreases the number of solutions produced through the iterative process and saves on computing time. For these reasons the default number of joinpoint was used in this thesis (a maximum of three joinpoints) (Hegerl et al., 2013). Sensitivity analysis was carried out by increasing the maximum number of joinpoints to five: this did not change the substantive conclusions reported in the results.

3.3.1.1 Annual percentage change

Trends in e† measured for each country can be calculated by the annual percentage change (APC) from the slope of the regression model over the relevant time interval. If the values of the 95% confidence intervals for the APC contained zero then the APC was not statistically significant and there was uncertainty surrounding the direction of change in trend.

Figure 18 shows an example output graph from the joinpoint regression analysis with lifespan variation estimates (scatter) and the modelled trend (line).

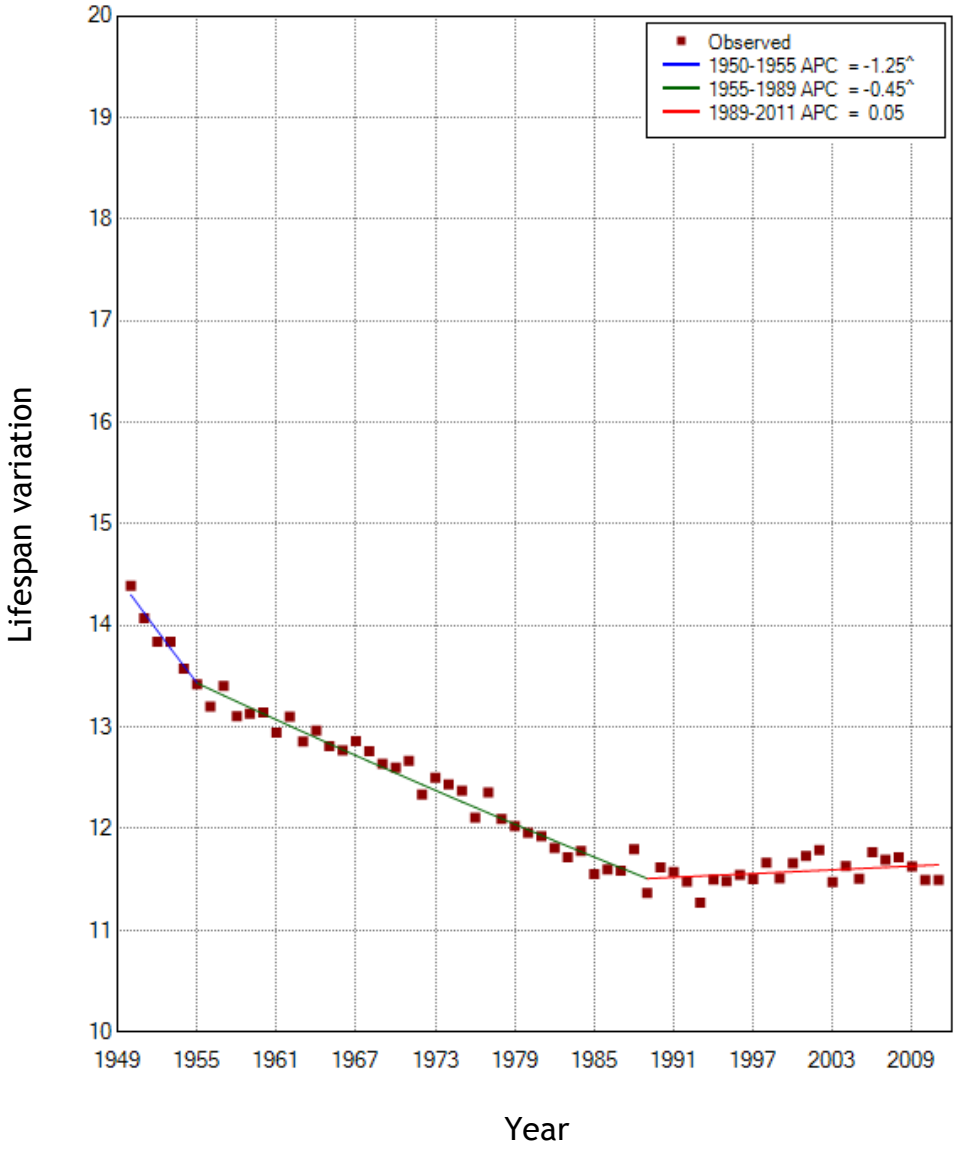


Figure 18 Joinpoint regression output example

From the modelled data the years that the lifespan variation trend changed were identified. 95% confidence intervals for the year of change were reported in the full model output. The annual percentage change (APC), between joinpoints, is also reported with 95% confidence intervals in the full model output. This allows for statistical inferences to be made about the significance of the timing of change and the significance of the level of change (Hegerl et al., 2013).

3.3.2 Age-specific decomposition

Chapter 5 and chapter 6 extended the analysis of the population level data by estimating the age-specific contributions made to the total difference in lifespan variation (Shkolnikov and Andreev, 2010, Preston et al., 2001). The two chapters differed in terms of how the time dimension was addressed in the analysis.

Chapter 5 identifies the ages of death that have contributed to the changing levels of lifespan variation over chronological time within Scotland. It then assesses how these compare to the ages of death that have contributed to the changing levels of lifespan variation over time within England and Wales.

Chapter 6 is concerned with assessing levels of lifespan variation when life expectancy was the same and is concerned with the notion that the two countries may be at different stages of the epidemiological transition (epidemiological time) (Smits and Monden, 2009). It aims to identify the ages of death that have contributed to the lifespan variation gap when Scotland achieved the same level of life expectancy as England and Wales, albeit later in time.

Both chapters are concerned with understanding the contributions from differences in age-specific mortality rates to the difference between two life table based quantities. This meant that the same age-specific decomposition method of analysis could be employed in both chapters.

There are many ways to decompose a difference and Preston et al. (2001) suggests the choice is, to an extent, arbitrary. Decomposition methods have been widely applied in research to partition absolute changes over time for one population (chapter 6) or absolute difference between two populations (chapter 7) into age-

specific components (Shkolnikov et al., 2011, Auger et al., 2014, van Raalte et al., 2014).

Age-specific decomposition was carried out using the stepwise decomposition algorithm within the Microsoft Excel Spreadsheets developed by Shkolnikov and Andreev (2010). This approach is appropriate for decomposing, by age, differences in any aggregate measure that is estimated from age-specific mortality rates and accounts for all ages including the oldest open ended age.

In order to obtain the decomposition components the mortality rates for the relevant comparator life tables had to be imputed into the Microsoft excel spreadsheet developed by (Shkolnikov and Andreev, 2010). The decomposition components were then exported to StataSE13 in order to produce figures for communicating the results.

The steps carried out for the age-specific decomposition are summarised as follows:

1. Age specific mortality rates for two populations (A and B) are obtained
2. Lifespan variation from observed mortality rates calculated
3. Starting with age 0 - replace age specific mortality rate for A with age specific mortality rate for B ($B \rightarrow A$), all other observed mortality rates stay the same
4. Recalculate lifespan variation for A with age specific mortality rate at age 0 replaced from B ($B \rightarrow A$),

5. Take the lifespan variation calculated with the replaced mortality rate for A from the lifespan variation calculated from the observed mortality rate for A to get the age specific contribution

6. Repeat steps 3,4,5 replacing each single year of age (0-110+) in a stepwise process

7. Repeat steps 3,4,5,6 in opposite direction - replacing age specific mortality rate for B with age specific mortality rate for A $(A \rightarrow B,)$

8. Take the average contribution for each single year of age from the two replacement directions $(B \rightarrow A, A \rightarrow B,)$ to get the age specific contribution

3.3.3 Slope index of Inequality and Relative Index of Inequality

The research questions in chapter 7 are concerned with the impact socioeconomic deprivation has for lifespan variation and quantifies changes to the socioeconomic gradient for lifespan variation over time.

To analyse the gradient in lifespan variation by socioeconomic deprivation the Slope Index of Inequality (SII) and the Relative Index of Inequality (RII) were used. The SII communicates the absolute level of inequality in a health variable for social groups. The RII communicates the inequality in relative terms (Mackenbach and Kunst, 1997).The SII and RII are valuable methods of analysis to use because they allow trends to be assessed by taking into account of all the data points not just the extremes (Munoz-Arroyo and Sutton, 2007).

The SII is an attempt to estimate the absolute health difference between the most and least deprived groups. It is interpreted as the absolute effect on health from

moving from the lowest ranking socioeconomic group to the highest ranking socioeconomic group.

Carstairs quintiles of socioeconomic deprivation rank the categories from 1 (least deprived) to 5 (most deprived). The population of each socioeconomic deprivation category are part of the cumulative total population. Each socioeconomic deprivation category is assigned a variable which refers to the midpoint of their range in the cumulative distribution of the total population.

This is straight forward when using population weighted quintiles as each contains 20% of the population. Therefore the first 20% has a range from 0% to 20% and the midpoint is 0.10, the second 20% has a range from 20% to 40% and a midpoint value of 0.30…the final 20% of the population has a range from 80% to 100% and is assigned a midpoint value of 0.90 (Munoz-Arroyo and Sutton, 2007).

The SII is then the value of the slope coefficient of an ordinary least squares regression between the dependent health variable and the independent deprivation cumulative population variable: the larger the coefficient the greater the impact of deprivation (Allik et al., 2016).

The Slope index regression equation is summarised as:

$\bar{y}_j = \beta_0 + \beta_1 \bar{R}_j$ where

j is the category of socioeconomic deprivation

$\bar{y}_j$ is the average health status

$\bar{R}_j$ is the average relative ranking of socioeconomic deprivation category in the cumulative distribution of the population

β_0 is the estimated health status of an individual in the lowest ranked socioeconomic deprivation category

β_1 is the difference in average health status between an individual in the lowest ranked socioeconomic deprivation quintile and an individual in the highest ranked socioeconomic deprivation quintile (this is the SII value)

The SII is typically reported as the absolute difference in the health outcome. For lifespan variation this is the absolute difference in years between the notionally most deprived and notionally least deprived quintile. The RII summarises this as a relative percentage difference and is derived from the same regression model.

The RII reported in this thesis was obtained by dividing the SII value by the mean value of the outcome variable across all socioeconomic deprivation categories.

3.3.4 Monte-Carlo simulations

The lifespan variation estimates, stratified by socioeconomic deprivation, the SII, and the RII results were subject to further statistical analysis in chapter 7: 95% confidence intervals were calculated by carrying out a Monte-Carlo simulation method. 95% confidence intervals allow the true population value of lifespan variation, stratified by socioeconomic deprivation, to be statistically assessed. This analysis is important to carry out because lifespan variation is calculated from mortality rates and the numbers of deaths in the population are inherently vulnerable to random, naturally occurring fluctuations. As a result the number of deaths that occur in a population can be interpreted as one possible series that could have occurred under the given set of circumstances (Curtin and Klein, 1995).

In order to produce 95% confidence intervals 1,000 simulated death counts were required for every age (0-110+) by socioeconomic deprivation (quintiles 1 to 5) in each census year (1981, 1991, 2001 and 2011) and for males and females separately.

Each single round of simulations produced 4,440 age specific death counts that were used to calculate 4,400 age specific mortality rates. From the mortality rates 40 complete period life tables were produced (2 sexes, 4 years, and 5 deprivation quintiles). The total number of simulations carried out produced 4,440,000 simulated age-specific death counts that were used to construct 40,000 simulated life tables. The 2.5 and 97.5 percentiles from the simulated results were selected for the 95% confidence interval limits.

The method used in this thesis is the fifth simulation method (MC) described in detail by Lumme et al. (2015). This method was appropriate as the socioeconomic group variable being used is grouped by proportions (quintiles). The numbers of people in each quintile can be assumed to be stable as they are from a large dataset: each quintile reflects 20% of the total population of Scotland. The fixed proportions of each quintile means the ranking of the socioeconomic groups' remains fixed when the simulation is carried out and only the variability in the health outcome (death counts used to calculate mortality rate) is modelled.

The steps of the MC simulation are as follows:

1. Obtain the population and death counts in each age group and deprivation group

2. Simulate the number of events subject to the constraint from the observed number of events in age group. The probabilities used for the multinomial distribution are estimated from the observed data: the number of deaths in each age and deprivation group over the total deaths in each age group
3. Repeat step 2 1,000 times to obtain 1,000 sets of data
4. Calculate age specific mortality rate (0-85+). Apply mortality rate prediction method (Poisson regression with fractional polynomial). Obtain mortality rate for ages 85-110+ for 1,000 data sets
5. Construct life tables from the mortality rates (0-110+) for the 1,000 simulated data sets
6. Calculate lifespan variation for the 1,000 simulated life tables
7. Calculate the SII and RII for the 1,000 simulations
8. Sort the simulated lifespan variation estimates and select the 2.5 and 97.5 percentiles to get 95% confidence interval limits
9. Sort the simulated SII estimates and select the 2.5 and 97.5 percentiles to get 95% confidence interval limits
10. Sort the simulated RII estimates and select the 2.5 and 97.5 percentiles to get 95% confidence interval limits

The 95% confidence intervals for the SII and RII allowed any uncertainty surrounding the socioeconomic gradient to be statistically evaluated. If the limits of the 95% confidence intervals for the SII or RII cross zero then there is

uncertainty as to whether there is a true health difference between the most and least deprived socioeconomic groups (Allik et al., 2016). Lumme et al. (2015) summarises the value of this method of analysis when stating that 'uncertainty in an indicator is an essential question when making comparisons of equality'.

3.3.5 International Classification of Disease

The analysis in chapter 8 uses the death record data collated by NRS and held by the MRC/CSO Social and Public Health Sciences Unit, University of Glasgow. Number of deaths by sex, single year of age and Carstairs deprivation quintile across 5 cause-specific categories of death (derived from the International Classification of Disease (ICD)) were obtained. The five cause specific categories in this thesis are: external, cancers, circulatory, respiratory and an all other category. The cause-specific categories were harmonized across version 9 and version 10 of the ICD. The ICD has been used by WHO member states since 1994. It has evolved from being a systematic classification of disease used for statistical purpose to being applied in medical administration, epidemiology and health services research (O'Malley et al., 2005).

Although the ICD is a standard diagnostic tool for population level health analysis (World Health Organization, 2012) the vast amount of data on mortality patterns is potentially incomprehensible. In its current iteration there are 120,000 total codes (World Health Organization, 2004). As a result O'Malley et al. (2005) highlight that researchers need to be reflexive about the extent to which ICD codes can be considered a representation of true and observed variables which reflect a person's true cause of death. The cause of death categories were informed by those which have previously been used in existing studies (Leyland et al., 2007a,

van Raalte et al., 2014). The cause-of-death categories are mutually exclusive meaning that the each death is only assigned to one category. If no specific cause of death was available or the death was ill-defined then it was assigned to the "all other causes" category.

Table 12 details the ICD codes that were included in the external, cancers, circulatory and respiratory categories.

Table 12 ICD codes assigned to cause of death categories

Category		ICD-9 (1981 and 1991)	ICD-10 (2001 and 2011)
External	Comprising chronic liver disease, accidents, intentional self-harm and events of undetermined intent, mental and behavioural disorders due to use of drugs or alcohol, and assault	5710-5719,E800-E929, E950-E959,E980-E989, 3040-3049,3052-3059, 2910-2919,3030-3039, 3050, E960-E969	K70-K709, K73-K749, V01-X599,Y85-Y869, X60-X849, Y870,Y872,Y10-Y349, F11-F169,F18-F199, F10-F109, X85-Y099, Y871
Cancers	Comprising all malignant neoplasm	140-208	C00-97
Circulatory	Comprising Ischaemic heart disease and cerebrovascular disease	390-459	I00-99
Respiratory	Comprising chronic obstructive pulmonary disease and chronic lower respiratory diseases	460-519	J00-99
Other causes	All remaining ICD codes attributed to 'other causes' category including deaths that had no cause of death listed or if the cause of death was ill defined. Remaining cause of death categories are infectious disease and diabetes	-	-

3.3.6 Cause specific mortality rates beyond age 85

The numbers of cause-specific deaths by sex and SYOA were extracted for the years surrounding each Census. 3 years' worth of death data (2 years for 1991) were extracted to increase the number of events as deaths are inherently vulnerable to random fluctuations (Lumme et al., 2015, Curtin and Klein, 1995). The numbers of cause-specific deaths were then matched with Carstairs score data on the postcode sector and local government district variable. The Census population estimates by Carstairs score were then matched. This allowed the numbers of cause-specific deaths by sex, SYOA and Carstairs score to be transformed into cause-specific mortality rates for ages 0-85+.

In order to obtain cause specific mortality rates, up to an open ended age interval of 110+, the modelled mortality rate used in chapter 7 was proportioned out to reflect the cause specific proportions as they stood at age 84 for each sex, quintile of deprivation and year combination. This approach assumed that the proportion of the total deaths by sex, quintile of deprivation and year remained constant across each single year of age for age ≥85. This gave cause-specific mortality rates for age 0 to age 110+ for each sex, quintile of deprivation and Census year (40 cause-specific data sets in total). The sum of the cause-specific mortality rates for each sex, SYOA and quintile is always equal to the all-cause mortality rate used for the analysis in chapter 7 of this thesis.

3.3.7 Decomposing a difference in lifespan variation by age and cause of death

Cause-specific mortality rates for age 0-110+ were then entered into the Microsoft Excel program developed by Andreev and Shkolnikov (2012). The decompositions in chapter 8 report a difference in $e_0^\dagger$ by single year of age and 5 cause specific categories of death (see table 12) utilising the stepwise decomposition method applied in the Microsoft Excel program developed by Andreev and Shkolnikov (2012). This approach has previously been applied in the literature to examine socioeconomic inequalities in lifespan variation (van Raalte et al., 2014, van Raalte et al., 2012). The age-cause specific decomposition method is similar to the age-specific decomposition (described in section 3.3.2) and can be summarised as follows:

1. Two lifetables (A and B) each contain age-cause specific mortality rates that are the equal to all cause age-specific deaths rates combined (calculated as rows sums of age specific death rates)

2. Replacement for age x and cause c includes the calculation of the change in the aggregate mortality measure resulting from the replacement of mortality rate A by B

3. Replacement is performed for each combination of causes of death other than c

4. Replacement progress is made from the first age 0 to the last age ω and is run twice ($A \rightarrow B, B \rightarrow A$) and the average of the two replacement cycles taken

3.4 Chapter summary

This chapter had two sections. The first described the data required to calculate lifespan variation, the measure of total inequality which is the primary focus of this thesis. It outlined the data management processes required to estimate lifespan variation stratified by socioeconomic deprivation. The strengths and limitations of the data were considered and alternative measures of socioeconomic position critically evaluated. The modelling process applied to obtain mortality rates for ages ≥85 was outlined and e†, the measure of lifespan variation used throughout this thesis, described. The second section discussed the range of statistical tests, measures and methods of analysis utilised within this thesis. The data chosen and the analysis applied were appropriate for answering the research questions which were presented in the literature review chapter of this thesis: different research questions may have demanded different methods of analysis. This issue is reflected on throughout the thesis especially when considering future research implications.

4 Scotland's lifespan variation trend in Western Europe

4.1 Introduction

The literature review chapter of this thesis began by exploring some theories surrounding health inequalities and their measurement in empirical research. Health inequalities in Scotland have been the focus of much research attention: it experiences the lowest level of life expectancy in Western Europe, widening mortality inequalities between socioeconomic groups, and increasing premature mortality rates for some ages (Leyland et al., 2007b, McCartney et al., 2012b). Little is known about lifespan variation in Scotland despite suitable data for estimating lifespan variation being widely available. Lifespan variation is a novel measure of inequality that reflects the concept of total inequality: the amount of inequality that exists across all individuals *within* a population. The concept of total inequality has been contested in the health inequalities literature and contrasted against a traditional concept of inequality: the difference in average health *between* populations (Gakidou and King, 2002, Murray et al., 1999, Le Grand, 1987). However both concepts of inequality should be measured simultaneously in order to evaluate the extent to which the twin aims of public health are being achieved: increases in average health alongside reductions in inequality. Existing studies suggest these aims are not incompatible as populations that have experienced greater reductions in total inequality have experienced the greatest improvements in average population health and narrower inequalities between socioeconomic groups (van Raalte et al., 2011, Vaupel et al., 2011).Measuring and analysing lifespan variation in Scotland will

help to identify how it can decrease the level of inequality in age at death the population experiences which could subsequently improve its average population health ranking within Western Europe. Chapter 3 outlined the data sources and quantitative analysis methods required to achieve this.

This chapter begins the process of analysing the population level lifespan variation trend for Scotland and the findings that are described build on those which have already been published (Seaman et al., 2016a). It contributes to the overarching aim of the thesis by answering the following research questions:

1. Has Scotland's lifespan variation ranking within Western Europe changed over time?

2. Was the timing and relative rate of lifespan variation change in Scotland comparable with any other Western European country?

Data used in this chapter are from the Human Mortality Database. The chapter reports the annual estimates of lifespan variation that were calculated for 1950 to 2011, the most recent year for which data were available when the research was carried out. The analysis seeks to formally assess changes to the lifespan variation trend, from the calculated estimates, for Scotland relative to the changes in 16 other Western European countries.

The chapter has four main sections. The first background section begins by discussing changes to Scotland's life expectancy trend relative to Western Europe and identifies that less is known about changes to Scotland's lifespan variation trend. The second section then briefly describes the analysis methods used to fill these research gaps. The third section reports the statistical findings.

The fourth section provides a summary and identifies the research implications of these findings and how they informed the analysis going forward in this thesis.

4.2 Background

Mortality in Scotland is high by Western European standards. Although the all-cause mortality rate has fallen across Scotland premature mortality rates for some ages from suicide, substance and alcohol abuse, and violence have risen over the past three decades. These unfavourable mortality trends are reflected in Scotland's life expectancy which is now the lowest in Western Europe (McCartney et al., 2012b, Leyland et al., 2007b).

4.2.1 Life expectancy

Life expectancy estimates the average number of years a new-born can expect to live for, given the current mortality conditions (Preston et al., 2001). It is one of the most common indicators of population health that allows both comparisons between countries to be made and changes over time to be monitored (World Health Organization, 2010). Increasing life expectancy means that the average age at death is being delayed. Annual increases in life expectancy, which have almost universally been experienced, are therefore interpreted as improving average population health. A lesser studied but equally important dimension of mortality to measure over time and in cross-national comparative research is lifespan variation (Smits and Monden, 2009, Shkolnikov et al., 2011).

4.2.2 Lifespan variation

Lifespan variation estimates the amount of inequality in age at death that exists between individuals. Decreasing lifespan variation means that deaths are being

compressed around a common age, and that the age at death is becoming more homogenous (Tuljapurkar, 2010, Smits and Monden, 2009). Having a relatively homogenous age at death is seen as a sign of low inequality and can help inform societal level decisions about healthcare, retirement and pensions (van Raalte et al., 2014). Therefore, as previously discussed in the literature review chapter, lifespan variation cannot simply be interpreted as random variation: it is a measure of mortality that reflects social and systematic inequality.

4.2.3 International evidence

International studies have found that historically most countries have been able to achieve the correlation between increasing life expectancy and decreasing lifespan variation (Smits and Monden, 2009, Tuljapurkar, 2010, Shkolnikov et al., 2011). This suggests that the twin aims of public health – to increase average health and reduce inequality- are compatible. However there are indications that some countries have failed to achieve both: countries which have been able to achieve increases in life expectancy without reducing lifespan variation at a comparable rate to other countries. The USA is one example country that has already been studied (Shkolnikov et al., 2011).

4.2.4 Distribution of age at death

The reason why the relationship between life expectancy and lifespan variation differs for the USA is due to the age distribution of mortality it experiences. While improvements in life expectancy are achieved by reducing mortality at any age, improvements in lifespan variation are generally achieved by reducing premature deaths to ensure mortality compression. Mortality compression refers to narrowing of the shape of the age distribution of death by reducing deaths

across working adult ages. The opposing process is mortality expansion. Expansion refers to the widening of the shape of the age distribution of death which is mostly driven by pushing more deaths into older ages (van Raalte, 2011a, Oeppen, 2008). Therefore countries with stalling lifespan variation trends tend to be those who have been less successful at reducing premature mortality and those achieving less compression than expansion (van Raalte et al., 2011). This is of direct relevance for Scotland as it is known to have a persisting premature mortality problem (Norman et al., 2011, Leyland, 2004).

4.2.5 Changing trends in Scotland

Changes to Scotland's life expectancy trend were analysed by McCartney et al. (2012b). Historically its life expectancy ranked around the European median but the rate of improvement in Scotland began to slow following the 1950s (McCartney et al., 2012b, National Records of Scotland, 2014b). This resulted in several countries in Europe overtaking Scotland because they were able to improve their life expectancy more rapidly. These countries included Spain, Portugal, Italy and Finland. McCartney et al. (2012b) reported a further stalling in Scotland's life expectancy following the 1970s. Consequently Scotland is now only achieving the life expectancy that the best performing countries in Europe achieved 40 years earlier: 76.9 and 80.9 years for men and women respectively (The Scottish Government., 2014). This is a relatively low life expectancy for such a high income country (Popham and Boyle, 2010).

4.2.6 The correlation between life expectancy and lifespan variation

Given the established correlation between life expectancy and lifespan variation evidenced in the international literature, it is hypothesised that Scotland's

lifespan variation trend may have also faltered around the same time. Popham and Boyle (2010) calculated lifespan variation for Scotland for each decade since 1950. They demonstrated Scotland's average lifespan variation for each decade up until the 1980s was close to the middle of the distribution of the countries identified as comparators. Following the 1980s Scotland's lifespan variation began to stall or slightly increase. By the mid 2000's Scotland's lifespan variation was the worst in Western Europe. The only country with larger variation in lifespan, included in the study by Popham and Boyle (2010), was the USA. This evidence, combined with that of McCartney et al. (2012b), suggests that Scotland may be burdened with the shortest average age at death and the greatest inequality in age at death relative to the rest of Western Europe.

4.2.7 Research gaps

Although Popham and Boyle (2010) estimated lifespan variation for each decade since the 1950s and alluded to the timing of lifespan variation change in Scotland they did not statistically assess the change in trend or systematically compare annual lifespan variation estimates in Scotland with Western Europe. It is these important research gaps that the following analysis chapter fills.

Analysing the timing of any changes to lifespan variation in Scotland is important for helping to inform future studies towards the possible causes of health inequalities. However in order to establish if Scotland's lifespan variation trend should be considered adverse the enquiry needs to be extended to include comparable countries (Rose, 2001). Western Europe provides a valuable opportunity for cross national comparative research as long running data are available for countries with contrasting political, cultural, economic and

epidemiological profiles (Mackenbach et al., 2008). This chapter employs joinpoint regression to identify and compare changes to lifespan variation trends since 1950. The emphasis is on understanding Scotland's trend, and not on comparing all trends in the other Western European countries.

4.3 Data & methods

Data used in this chapter were sex specific life tables for Scotland and 16 other Western European countries. A detailed description of a life table has already been given in chapter 3 of this thesis (data and methods). Lifetables for the years 1950 to 2011 were obtained from the Human Mortality Database, one of the most highly regarded providers of comparative mortality data. These were the most recent years of data available for all of the countries at the time of research. When making formal comparisons between trends the length of time series has to be the same. Data were available for Scotland from 1950-2011 but when making formal comparisons these had to be restricted to match the years that were available depending on the comparator country. The Western European countries used and the years for which data were available to make comparisons are detailed in table 13. These countries have been used in existing studies as comparators with Scotland (Mackenbach et al., 2008, Leon et al., 2003).

Table 13 comparator countries and years for which complete period life tables were used for analysis in Chapter 4

Country	Years data available	Country group
Scotland	1950-2011	
England & Wales	1950-2011	West
Northern Ireland	1950-2011	
Ireland	1950-2009	
Denmark	1950-2011	
Finland	1950-2009	North
Norway	1950-2009	
Sweden	1950-2011	
Austria	1956-2010	
West Germany*	1956-2010	Continental
Switzerland	1950-2011	
Belgium	1950-2012	
France	1950-2012	
Netherlands	1950-2009	
Spain	1950-2009	
Italy	1950-2009	South
Portugal	1950-2012	

*West Germany is the area of unified Germany formally known as the Federal Republic of Germany (FRG). The territory has changed over time but the statistics here represent the regions that made up the FRG.

4.3.1 Calculate lifespan variation

The first stage of the analysis calculates lifespan variation from these lifetables. e† was used to measure lifespan variation and a detailed justification for this measure is provided in chapter 3 (data and methods chapter). e† was calculated in Stata SE13. A total of 2,076 lifetables were used to calculate annual estimates of lifespan variation.

4.3.2 Joinpoint regression

The second stage of the analysis statistically assesses any change in lifespan variation trends for Scotland and the 16 other Western European countries. This was achieved by carrying out joinpoint regression analysis using the software provided by the Surveillance Research Cancer Control and Population Sciences National Cancer Institute (2013). A detailed description of joinpoint regression analysis is given in chapter 3 of this thesis (data and methods). It tests whether multiple straight lines joined together at 'joinpoints' are statistically a better fit to the trend data than a straight line (no joinpoints). In doing so it identifies the best fitting time point at which the trend for any health outcome has changed significantly in magnitude or direction (Hegerl et al., 2013). The joinpoint regression reports the year of change and the level of annual percentage change (APC) both with 95% confidence intervals.

4.4 Results

4.4.1 Lifespan variation estimates: Scotland's annual ranking within Western Europe

The annual e† estimates calculated for each country are detailed in appendix 3 and appendix 4, for males and females respectively.

The range in lifespan variation between Western European countries has compressed over time indicating that trends have, to an extent, converged. Despite the convergence Scotland's ranking has worsened, indicating that some of the comparable countries have been more successful at reducing total inequality. This is the case for males and females.

In 1956 males in Scotland ranked 7th out of the 17 countries included in the analysis and had a lifespan variation of 13.2 years. Portugal ranked 17th and had a lifespan variation of 18.35 years. The country that ranked first was the Netherlands with 12.25 years. By 2009 (the latest year for which all countries had data available for when the research was undertaken) Scotland ranked 17th and had a lifespan variation of 11.63 years. Sweden was ranked 1st with 9.76 years, closely followed by the Netherlands with 9.91 years. Portugal had improved its position for males and ranked 14th with 11.17 years.

In 1956 females in Scotland ranked 8th and had a lifespan variation of 12.11 years. The country ranked 1st was Netherlands with 10.93 years. The country with the worst rank was Portugal with 17.41 years. By 2009 females in Scotland ranked 17th and had a lifespan variation of 10.35 years. In 2009 Spain ranked 1st with a lifespan variation of 8.71 years. Portugal had improved its lifespan variation to 9.19 years and ranked 9th. The Netherlands ranking had fallen to 10th place with 9.37 years.

Scotland's worsening lifespan variation trend and the countries overtaking it are not easy to visualise in a table format and, although the annual estimates of lifespan variation allude to differences, formal statistical analysis can provide further insight.

4.4.2 Joinpoint regression: Scotland

Joinpoint regression models were therefore fitted to the annual lifespan variation estimates in order to formally identify when the trend in Scotland started to worsen, allowing comparator countries to overtake it, and to measure the relative level of change.

4.4.2.1 Males

Figure 19 shows the lifespan variation estimates (scatter) with the fitted joinpoint regression (line) for males in Scotland. The dashed lines from the x-axis identify the years when a statistically significant change in trend was identified.

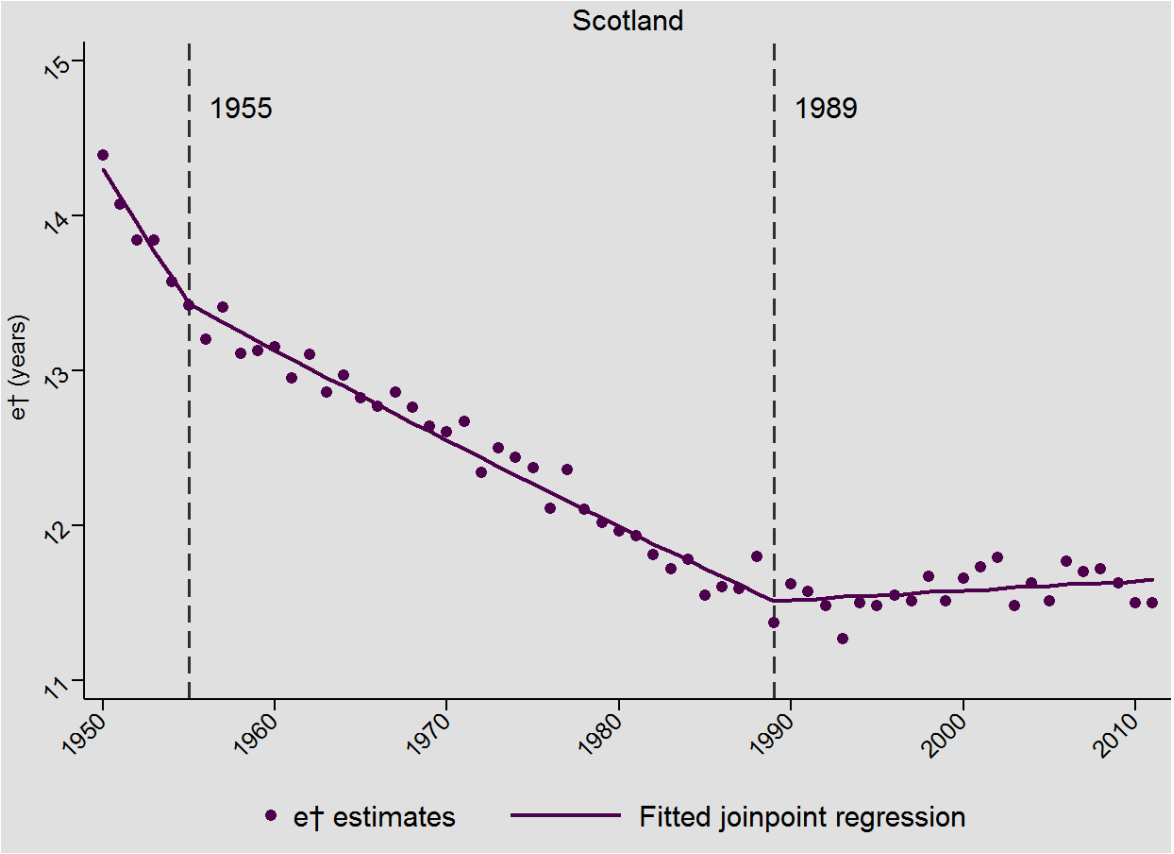


Figure 19 Lifespan variation estimates and fitted joinpoint regression for Scotland, Males

Although the lifespan variation trend for males in Scotland in 2011 was lower than in 1950, indicating an overall decrease, the joinpoint regression analysis identified two statistically significant changes to the rate of change over this time series. The first change in trend identified was in 1955 (1952 to 1958). Prior to this change lifespan variation had been decreasing at annual rate of -1.2% (-1.8% to -0.7%). After 1955 the annual percentage change in lifespan variation fell to -0.5% (-0.5% to -0.5%). This level of annual percentage change in lifespan variation was maintained until 1989 (1986 to 1993). From 1989 to 2011 the trend changed to be slightly increasing by 0.1% per year. However the 95% confidence intervals for the increasing percentage change following 1989 cross zero (-0.0% to 0.1%) indicating that there is some uncertainty about the statistical significance of this increasing rate of change.

4.4.2.2 Females

Figure 20 shows the lifespan variation estimates (scatter plots) with the fitted joinpoint regression (line) for Scotland for females.

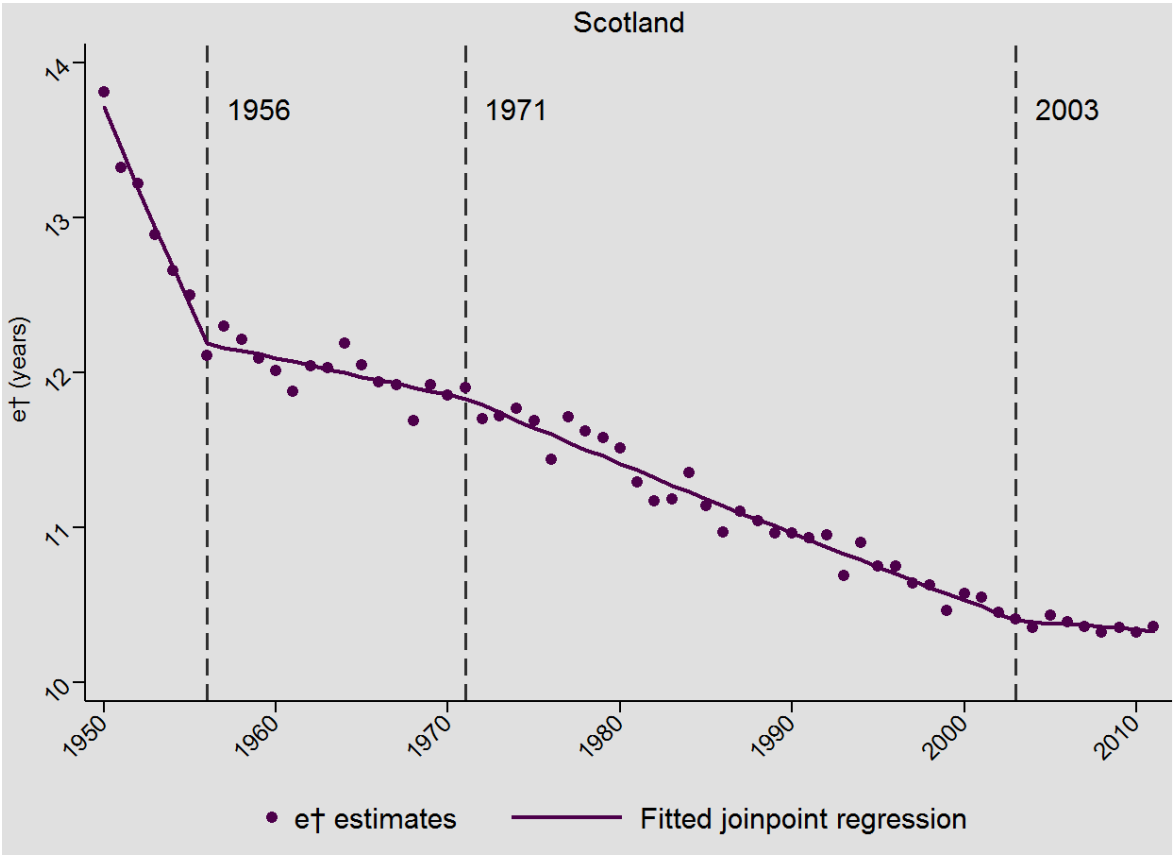


Figure 20 Lifespan variation estimates and fitted joinpoint regression for Scotland, Females

The lifespan variation for females in Scotland in 2011 was lower than in 1950. The joinpoint regression analysis identified three statistically significant changes to the rate of change over this time series. The first change in trend identified was in 1956 (1954 to 1958). Prior to this change lifespan variation had been decreasing at an annual rate of -1.9% (-2.3% to -1.6%). After 1956 the annual percentage change in lifespan variation fell to -0.2% (-0.3% to -0.1%). This level of annual percentage change in lifespan variation was maintained until 1971 (1963 to 1981). Following 1971 the decreasing rate of change improved again to -0.4% (-0.4% to -0.4%). The trend then changed again in 2003 (1980 to 2009). From 2003 to 2011 lifespan variation decreased by -0.1% for females in Scotland. However the 95% confidence intervals for the decreasing percentage change following 2003 include zero (-0.3% to 0.2%) indicating that there is some

uncertainty as to whether females in Scotland did experience a decreasing rate of change.

4.4.3 Joinpoint regression: cross-national comparisons

Figure 21 and figure 22 demonstrate how well the joinpoint regression fitted the annual estimates of lifespan variation for the 16 comparator countries. Figure 21 is for males and figure 22 is for females. Note the different scales for each country and the years which the time series begins at. The length of time series differed for each country but when making formal comparisons between trends the length of time series has to be the same. Data were available for Scotland from 1950-2011 but when making formal comparisons this had to be restricted to match the years that were available for the comparator country.

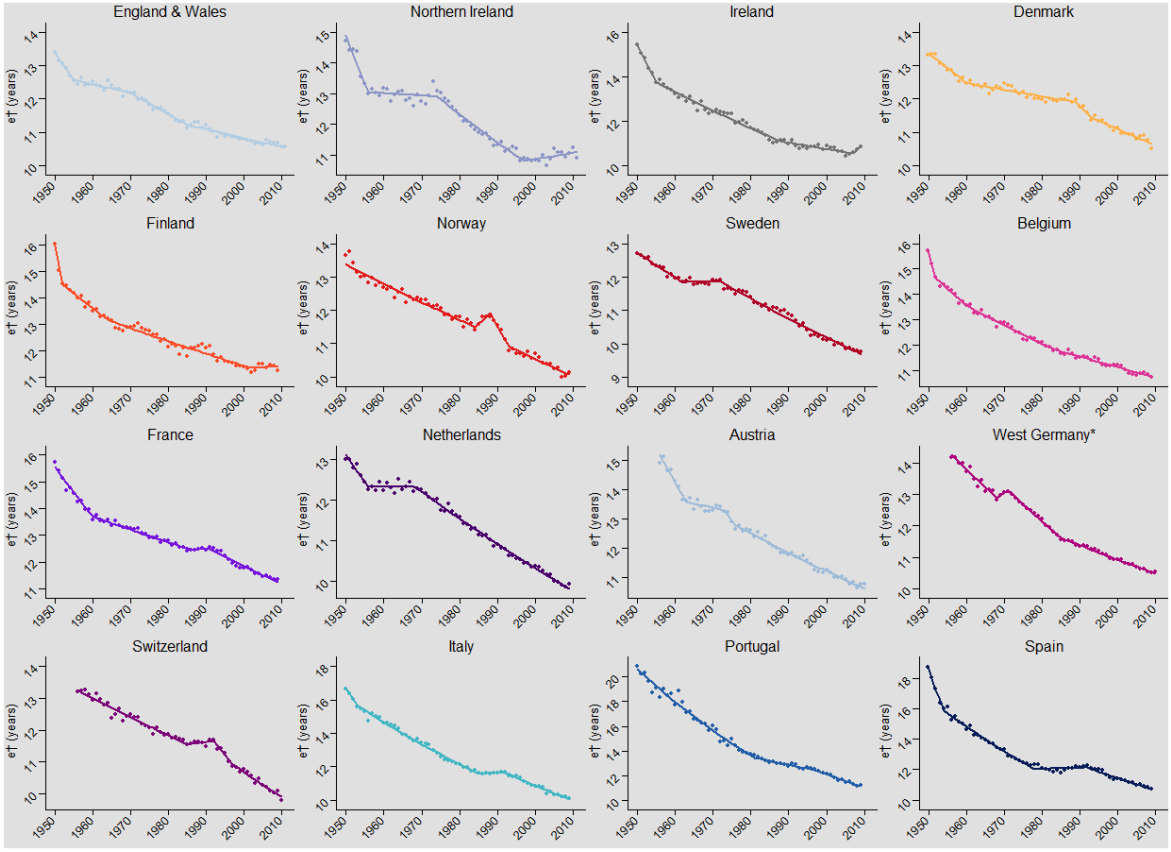


Figure 21 Lifespan variation estimates and fitted joinpoint regression for 16 Western European comparator countries, Males

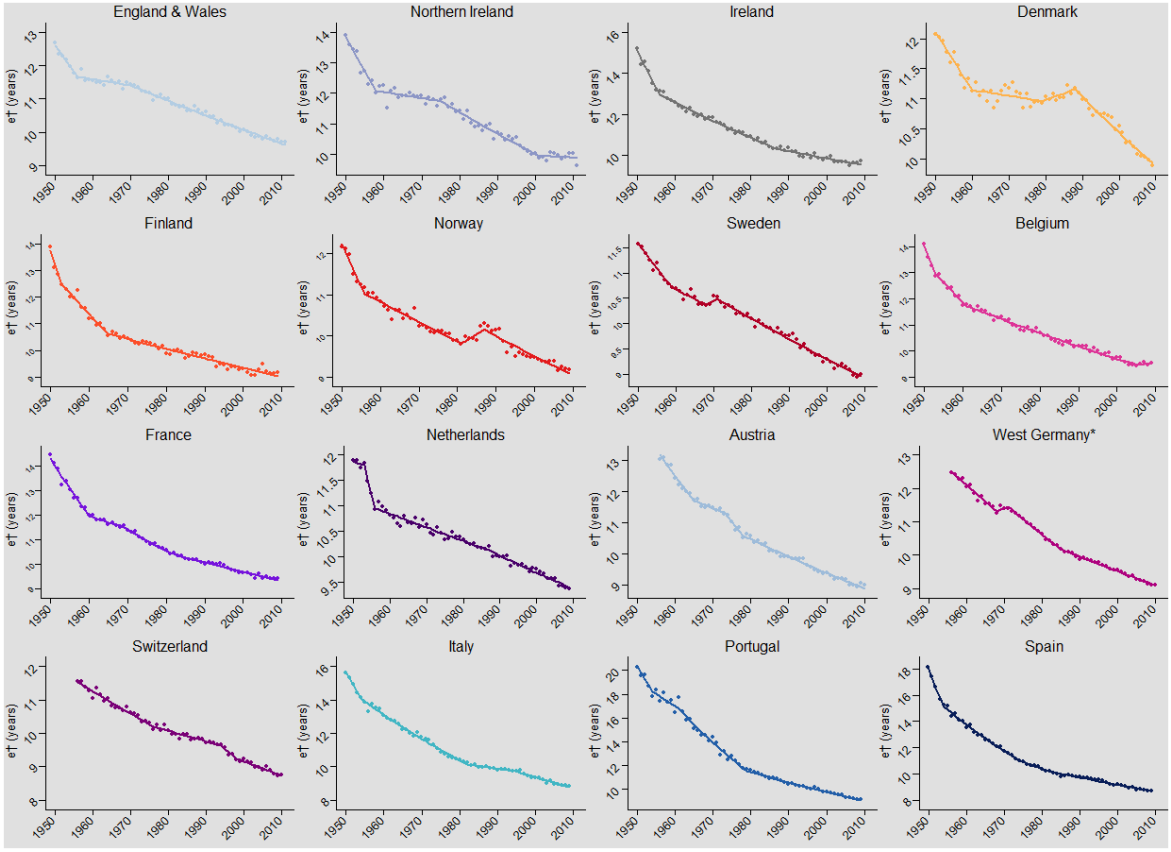


Figure 22 Lifespan variation estimates and fitted joinpoint regression for 16 Western European comparator countries, Females

The relative position of the each lifespan variation trend over time and the joinpoints identified for all countries can then be compared in figure 23 for males and figure 24 for females. The countries are grouped by geographical region to allow for easier interpretation.

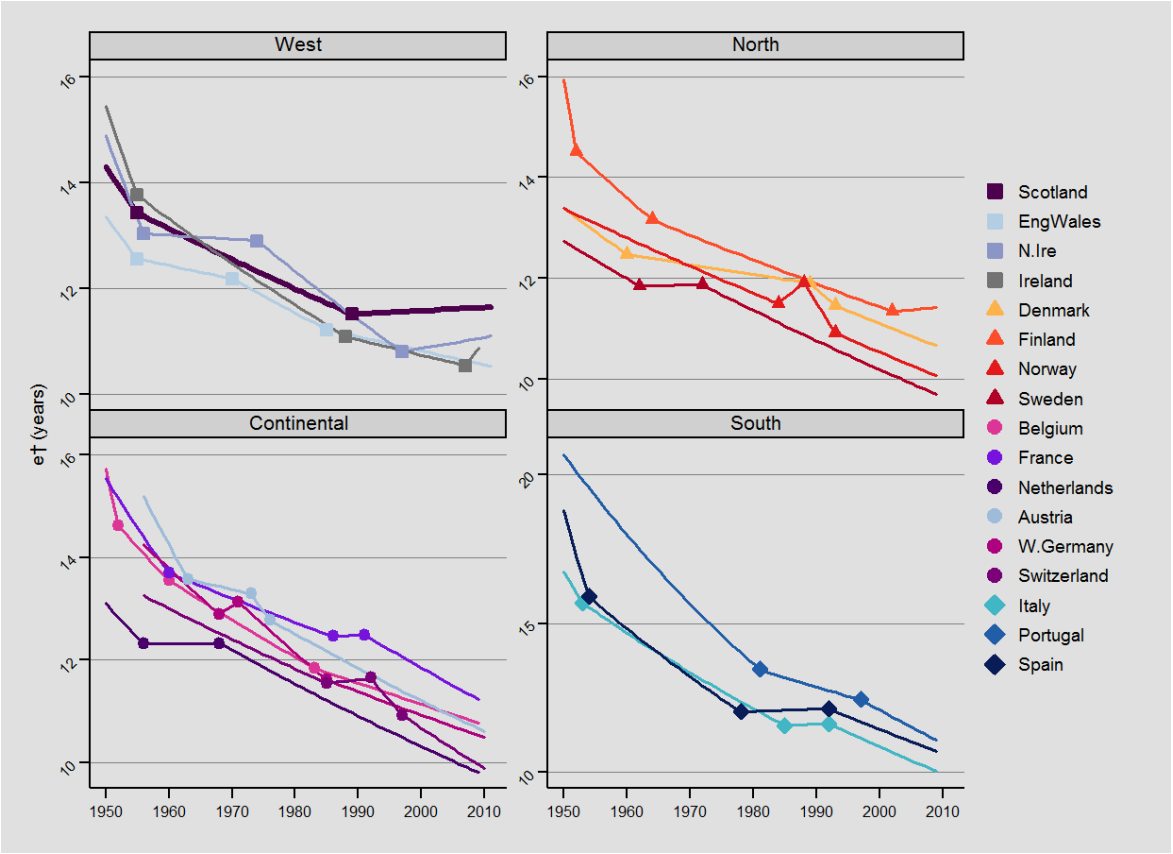


Figure 23 Modelled trends for lifespan variation for all 17 Western European countries grouped by geographical region, Males

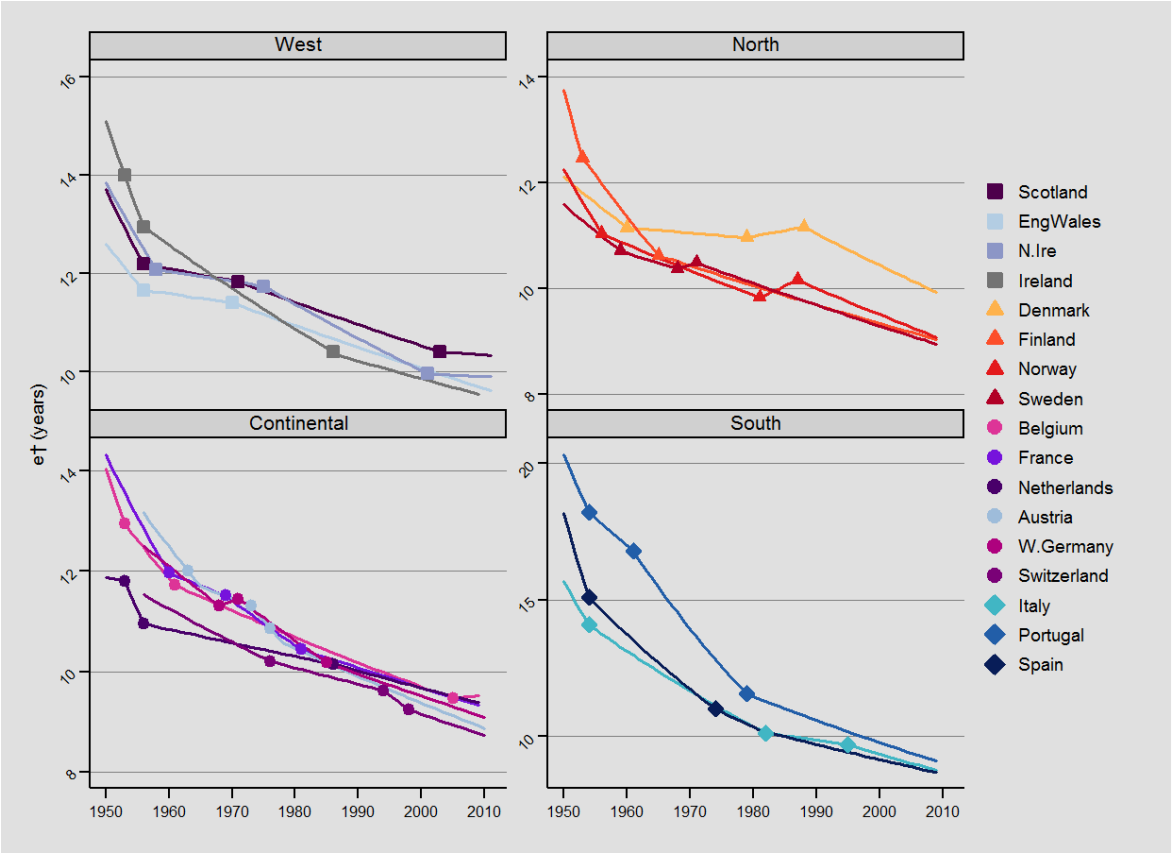


Figure 24 Modelled trends for lifespan variation for all 17 Western European countries grouped by geographical region, Females

For both males and females across Western European countries there is a general decreasing trend. For females there is a greater convergence of lifespan variation.

4.4.3.1 Comparing the timing of change

The year of change (95% CI) and APC (95% CI) for each country are detailed in appendix 5 for males and appendix 6 for females. The years of change for Scotland are compared to each of the years of change for each Western European country. This is illustrated in figure 25 for males and figure 26 for females. This gives a sense of how the timing of the change in trends for Scotland compared with the timing of the change in trend in each of the 16

comparator countries: most of the comparator countries maintained a decreasing trend over the time period studied. No other Western European country experienced a change in trend towards stagnating or slightly increasing lifespan variation at this time.

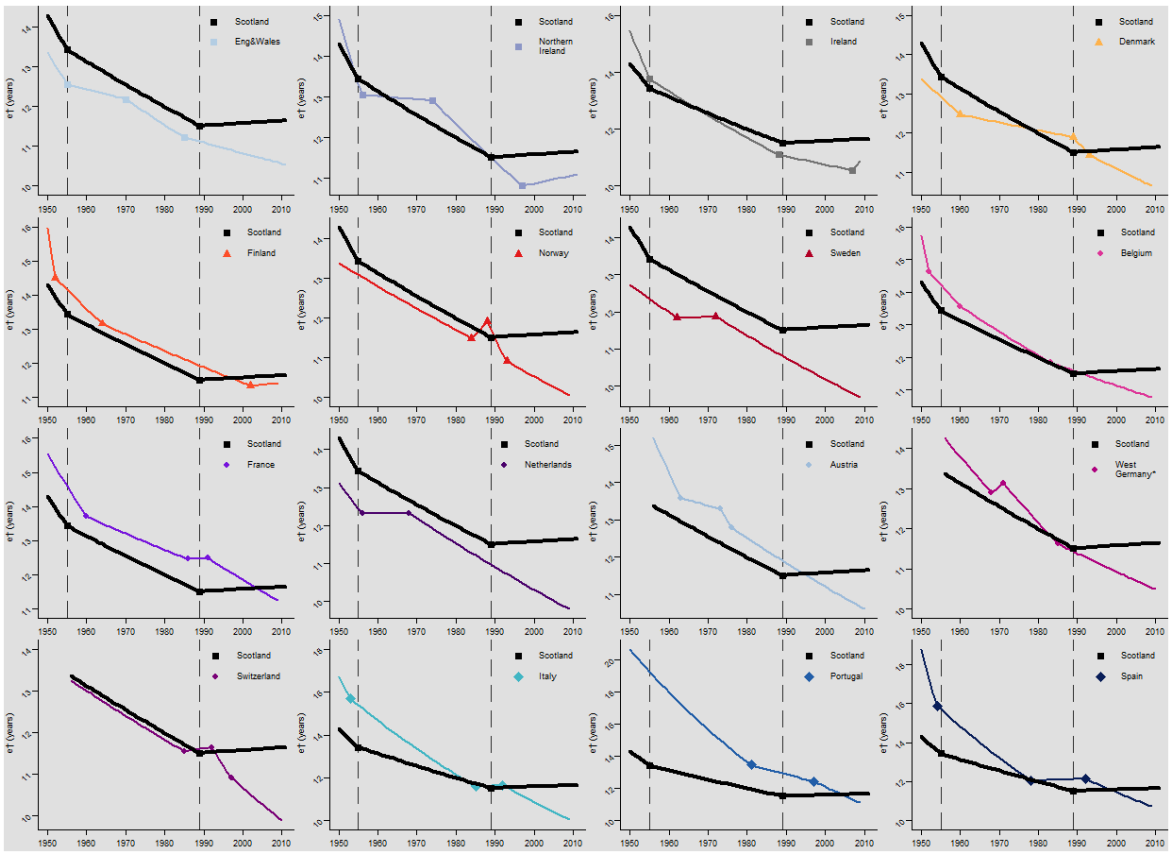


Figure 25 Change in lifespan variation trend in Scotland compared to each Western European country, Males

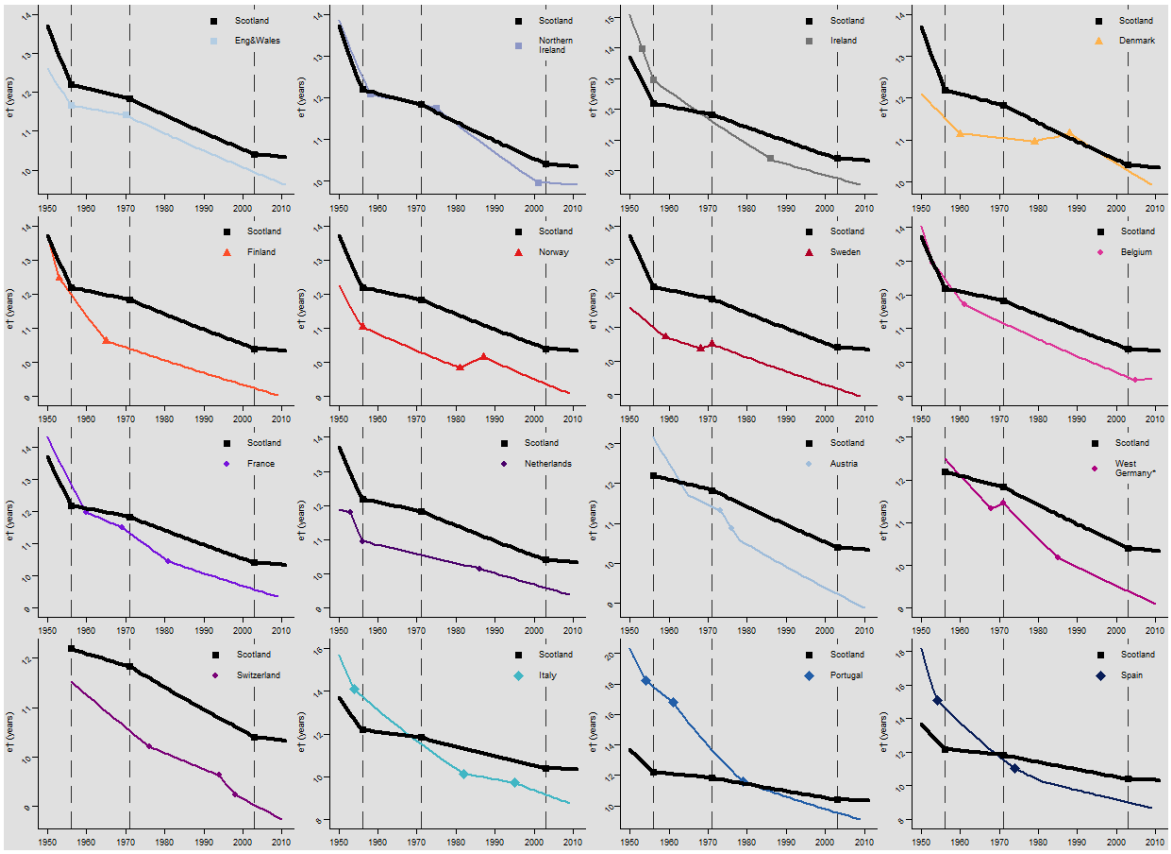


Figure 26 Change in lifespan variation trend in Scotland compared to each Western European country, Females

4.4.3.2 Comparing the level of change

It was also important to evaluate the level of change in each country compared with the timing of change. In general the timings of change and levels of change experienced in Scotland relative to the 16 other Western European countries can be summarised in terms of: periods of rapid decline, periods of stagnation or incline, and the most recent change in trend.

Periods of rapid decrease

There is a general trend of decreasing lifespan variation for males and females across all Western European countries. Rapid decreases in lifespan variation for all countries are found in the years immediately after 1950. Males in Scotland

experienced a -1.2% (-1.8 to -0.7) decline in lifespan variation between 1950 and 1955. This is in contrast to Spain where males experienced the steepest decline of -4.1% (-5.0 to -3.2) between the period 1950 and 1954.

At the start of the time period Sweden demonstrated one of the lowest absolute levels of lifespan variation but was experiencing a relatively smaller decrease of -0.6% (-0.7 to -0.5) between 1950 and 1984. Between 1950 and 1956 females in Scotland were experiencing a decrease of -1.9% (-2.3 to-1.6).

Periods of stagnation or incline

Some countries have experienced periods of stagnating or slightly increasing lifespan variation but have tended to return to a downward trend quite quickly. Females in Norway for example experienced an increase of 0.5% (-0.2 to 1.3) between 1981 and 1987. The confidence intervals for the APC cross 0 indicating that the there is a lack of certainty as to whether the lifespan variation trend has been increasing. This is the case when consulting the data in appendix 4: between 1981 and 1987 lifespan variation increases between some years in Norway but decreases between others. Scotland has always experienced decreasing lifespan variation until the most recent change in trend.

Most recent change in trend

For males the most recent change in trend for a few countries has been towards stagnating or slightly increasing. Males in Scotland have experienced an APC of 0.1% (-0.0 to 0.1%) since 1989 (1986 to 1993). Finland's most recent change in trend was in 2002 (1981 to 2007) with a subsequent APC of 0.1% (-0.4 to 0.5). The wider confidence intervals for Finland indicate that there is a lack of certainty as to whether the lifespan variation trend has been increasing.

Northern Ireland and Ireland have also recently demonstrated increasing or slightly stagnating trends.

All of the most recent changes in trends for females have been towards continued decline. However these are not significant declining trends for Scotland, Belgium and Northern Ireland.

For females there is a strong convergence in trends not evident for males. The convergence in trends means that several countries are competing to have the lowest level of lifespan variation. This is in contrast to males: Sweden has almost exclusively had the lowest level of lifespan variation over the time period covered. The most recent period for females in Scotland appears to be somewhat of an exception to this convergence. In 2009 Scotland had a lifespan variation of over 10 years (10.4 years). This was partly due to it experiencing a slowing in the rate of change: slowing from -1.9% (-2.3% to -1.6%) in 1950-1956 to -0.1% (-0.3 to 0.2%) in 2003-2011. This is in contrast to its nearest neighbours England and Wales where the slowing rate of change has not been as large. It experienced an APC of -1.3% (-1.6 to -0.9) in 1950-1956 to -0.4% (-0.4 to -0.4) in 1970-2011. The joinpoint regression analysis gives an indication as to which countries have been experiencing similar changes in trends and helps to inform an interpretation of Scotland's relative position.

4.5 Discussion

4.5.1 Summary of results

This chapter has formally assessed Scotland's lifespan variation trend in comparison to the trends in 16 other Western European countries. The range in lifespan variation between Western European countries became smaller over the

time period indicating a convergence of trends. However Scotland's annual lifespan variation ranking in Western Europe deteriorated from 7th (males) and 8th (females) position to 17th position. This was partly because other countries were able to reduce lifespan variation at a faster rate and overtake Scotland, despite having higher absolute levels of lifespan variation at the beginning of the time period. It was also partly due to males in Scotland experiencing a diverging lifespan variation trend from decreasing (at a relatively slower rate) to slightly increasing lifespan variation, and to females in Scotland experiencing one of the greatest reductions in the rate of relative change over time. Most countries have been able to maintain a decreasing trend since 1950 and there is a particularly notable convergence of trends for females in the comparator countries.

Although periods of slightly increasing lifespan variation are evidenced in a number of countries these have returned to decreasing. The most recent change in trend for males in Scotland is the longest sustained divergence in lifespan variation of the countries included in the analysis. Although the trend is still decreasing for females in Scotland the picture is still of concern: they have had the highest level of absolute lifespan variation for the past 22 years. Therefore males and females in Scotland are now in the unenviable position of ranking highest in level of lifespan variation: meaning that the population experiences the highest level of total inequality in Western Europe.

Lifespan variation reflects another dimension of health inequality that Scotland performs poorly on when measured against comparable Western European countries, thus adding further support for examining Scotland as a case study country. Formally investigating the lifespan variation trend in Scotland

demonstrates that although it is a country with relatively higher levels of lifespan variation this has not always been the case.

4.5.2 Comparison with existing studies

The timing of the most recent change in lifespan variation trend for Scotland is somewhat comparable with the trend analysis of life expectancy by McCartney et al. (2012b). This study concluded that Scotland's life expectancy trend was previously comparable with Western European countries but from around 1980 improvement in life expectancy faltered. No formal quantification of the life expectancy trend was carried out by McCartney et al. (2012b) meaning direct comparisons cannot be drawn even though the same time periods have been covered and the same HMD data utilised. Despite this the similar timing of the change in trends is not an unexpected result given that the international literature has established a strong correlation between life expectancy and lifespan variation (Smits and Monden, 2009, Oeppen, 2008).

4.5.3 Strengths and limitations

The findings reported here contribute to the body of research concerned with the timing and extent of change to health inequalities across Western Europe (Mackenbach et al., 2008, Popham et al., 2013, Smits and Monden, 2009). They were produced using the highest quality lifetable data available and demonstrate the most up-to-date estimates of lifespan variation across Western European countries. Calculating annual lifespan variation estimates for Scotland and 16 Western European countries, previously identified as valid comparators (Leon et al., 2003, McCartney et al., 2012b), allowed Scotland's relative ranking in each year to be assessed, answering research question one.

The extensive time period covered facilitated the use of joinpoint regression analysis. This is a well-established method for analysing trends in a number of health outcomes such as cancer (Goovaerts and Xiao, 2011, Bosetti et al., 2008) and suicide (Coope et al., 2014, Hegerl et al., 2013) but which has rarely been utilised for understanding lifespan variation trends. This method of analysis identified the timing of change and relative rate of change to the population level lifespan variation trend in Scotland, answering the second research question. Although the methods of analysis employed in this chapter adequately answer the research question this does not mean that the results should not be reflected upon.

4.5.4 Stage in epidemiological transition

The finding that Scotland's trend was distinguishable from most other Western European countries is perhaps not surprising given that a long time period was covered during which different countries were at different stages of the epidemiological transition. This is important to consider when analysing historical trends and when comparing countries at the same point in chronological time as they may have been experiencing different processes of modernization (Vallin and Meslé, 2004).

Omran (1971) summarised processes of modernization in terms of the general mortality patterns and the factors driving them. He stated that societies tend to experience three stages as they develop into industrialized countries with advanced health care. The first stage tends to be characterised by high and fluctuating mortality due to factors such as pestilence and famine. The second stage is defined by rapid reductions in mortality due to receding pandemics. The

third stage is characterised by a flattening in the reduction of mortality and the increasing presence of degenerative and man-made diseases as opposed to infectious diseases (Vallin and Meslé, 2004).

Therefore differences in trends in lifespan variation may be associated with differences in the timing of mortality change being experienced. This has prompted some to argue that cross-national comparative studies of health inequalities should compare countries in terms of epidemiological time rather than comparing countries in in terms of chronological time (Mackenbach et al., 1997, Smits and Monden, 2009). The analysis in chapter 6 seeks to reflect this debate and a more detailed discussion is provided there.

4.5.5 Whole country comparisons

In a similar vein it could be argued that comparing whole countries may be seen as an overly crude approach: there is wide heterogeneity within countries restricting any interpretation (Goovaerts and Xiao, 2011). The analysis in this chapter did not assume that the factors driving lifespan variation within each country would be the same: rather it acknowledged that the countries included in the analysis captured a variety of social, political, economic and epidemiological profiles. The chapter was concerned with the timing and level of change and recognised that the analysis did not permit any conclusions about the causes within each country to be drawn.

4.5.6 Premature mortality

Although the analysis in this chapter could not identify any single reason as to why Scotland's lifespan variation trend diverged, it is hypothesised that premature mortality will be part of the problem. This is because premature

mortality has already been identified as driver of relatively higher lifespan variation in the USA (Smits and Monden, 2009, Shkolnikov et al., 2011). However this is not to say that premature mortality is the root cause of poorer population health and higher inequality in Scotland: rather it should be interpreted as a marker for the types of mechanisms linking social determinants and health outcomes (McCartney et al., 2013).

van Raalte et al. (2011) argue that deaths at working ages could indicate the failure of social protection policies to mitigate the risk of premature death for the most vulnerable members of society. In a similar vein McCartney et al. (2012b) argue that the stalling of life expectancy in Scotland may, in part, be associated with the neo-liberal policies that were introduced during the 1980s. Neo-liberalism is associated with policies that increase inequality, have a negative impact on health outcomes and are associated with higher risk of premature death (Peacock et al., 2014, Coburn, 2004). The neo-liberal policies of the 1980s had a disproportionate impact on Scotland, where the effects of deindustrialisation were more concentrated (Walsh et al., 2010).

Within Scotland increasing rates of premature mortality have been the focus of much research attention. Mortality rates for males aged 15-29 years old increased between 1981 and 2001 (Leyland, 2004, Norman et al., 2011). This is the time when trends in life expectancy and lifespan variation in Scotland changed suggesting that both of these trends may be partly explained by premature mortality. Scotland's higher premature mortality problem is often contrasted to the more favourable experience in England and Wales. However Campbell et al. (2013) demonstrated that Scotland has had relatively greater

improvements in mortality rates below ages 15 years old at some time points. Fully understanding the implications these age differences in the distribution of death have for population level trends is important for highlighting sub-groups that are disadvantaged. Distinct age patterns of mortality can allude to the types of social and aetiological process that may be causing Scotland's unfavourable population level trends (Seaman et al., 2015).

4.5.7 Research implications

The findings demonstrating the timing of change therefore informed the subsequent analysis in this thesis. The timing of stalling life expectancy improvements in Scotland identified by McCartney et al. (2012b) corresponds with the timing of the diverging lifespan variation trend. This could suggest that the same changes to the age distribution of death in Scotland are responsible for both of these outcome measures but this cannot be assumed. Although the two outcome measures are highly correlated they capture distinct dimensions of mortality: life expectancy improvements are achieved if the mortality rate at any age is reduced over time but improvements in lifespan variation are age dependant. Changes to age distribution of death and the impact these have had on Scotland's lifespan variation trend have not yet been quantified.

4.6 Conclusion

Overall the study by McCartney et al. (2012b) and the analysis reported in this chapter strongly suggest that sometime following the 1980s mortality processes in Scotland began to change: these underlying changes were large enough to have a visible, and long lasting impact on the population level trends. The timing of these population level changes coincides with the timing of rising premature

mortality rates for some age groups: alluding to the importance of tackling premature mortality to ensure improvements in averages alongside reductions in inequality. Future research quantifying the role premature mortality plays in ensuring the twin aims of public health are achieved has implications far beyond this thesis and to countries other than Scotland.

5 Age decomposition of change in lifespan variation trend over time

5.1 Introduction

The previous chapter began the process of analysing the population level lifespan variation trend within Scotland. It identified that Scotland ranks worst for lifespan variation compared to 16 other Western European countries. Scotland previously occupied a more favourable ranking but following the 1980s failed to maintain the same relative rate of improvement for females and diverged towards slightly increasing lifespan variation for males. This meant other Western European countries, that had previously experienced higher lifespan variation, were able to overtake Scotland. The findings described in this next chapter extend the analysis of the population level lifespan variation trend within Scotland by carrying out age-specific decomposition.

There are four main sections in this chapter. The first background section summarises the age patterns of mortality decline that have historically been required to maintain a decreasing lifespan variation trend. How changes to the age-specific patterning of mortality have contributed to the changes in the population level lifespan variation trend within Scotland has not yet been the focus of any formal analysis. Therefore the chapter aims to answer the following two research questions:

3. Which ages of death contributed to the lifespan variation trend within Scotland?

4. Did the ages of death contributing to the lifespan variation trend in Scotland differ from the ages of death contributing to the lifespan variation trend in England and Wales'?

Answering these research questions begins to inform our understanding of why the lifespan variation trend in Scotland diverged when it did. Analysing the ages of death driving changes to lifespan variation in Scotland is important for helping to understand the causes of higher inequality. In order to establish if the ages of death driving Scotland's lifespan variation trend can be considered different or problematic the enquiry needs to be extended to include an appropriate comparison (Rose, 2001). England and Wales are appropriate comparators to use: they are Scotland's closest geographical neighbour and share social and economic contexts and a national government. The second section briefly describes the age-specific decomposition analysis methods utilised to fill this research gap. A more detailed description of the decomposition analysis methods can be found in chapter 3 (data and methods). The third section reports the age-specific decomposition results. Age-specific decomposition results in this chapter are being used to understand which ages of death have driven the population level lifespan variation trend within Scotland over time. The fourth section summarises the results and outlines how they informed the thesis going forward.

5.2 Background

Reducing mortality rates across any age leads to increases in life expectancy but decreases in lifespan variation are age dependent. Maintaining decreases in lifespan variation over time depends on a country's ability to consistently reduce mortality rates faster across ages that contribute to mortality compression than

across ages that contribute to mortality expansion (Oeppen, 2008, Zhang and Vaupel, 2009). Reducing deaths across ages that are well below the average life expectancy generally lead to mortality compression and decrease lifespan variation. A death was defined as premature within the context of this thesis if delaying it to an older age would decrease lifespan variation.

Zhang and Vaupel (2009) showed that it is possible to define the precise age which separates whether a death is considered "early" or "late" in terms of the effect it will have on lifespan variation. The age which distinguishes "early" and "late" deaths is different in each country and tends to have increased over time as life expectancy increases. Deaths that occur before the threshold age are deemed to be "early". Reducing "early" deaths leads to mortality compression which is beneficial for lifespan variation. Deaths that occur after the threshold age are deemed to be "late". Reducing "late" deaths leads to mortality expansion which is detrimental in terms of lifespan variation. For example the distinguishing age in Japan in 1950 was 65 but by 2005 it was 84 (Zhang and Vaupel, 2009). The threshold age in Scotland, and how it has changed over time, will not be identified in this chapter. It is recognised that this could be the focus of future research and demands more attention than could be given in this thesis.

5.2.1 Diminishing returns

Historically the greatest mortality reductions have tended to be concentrated across ages where the greatest proportion of the population dies (Vaupel, 1986). In broad terms this has meant a shift from saving lives across infancy and childhood – deaths more likely to have common causes that can be eradicated –

to younger and older adult ages where a variety of causes may be interacting with one another and that may be difficult to target (Oeppen, 2008, Nau and Firebaugh, 2012). The shifting age profile of mortality ensured the greatest impact in terms of life expectancy (Oeppen, 2008, Vaupel, 1986). However it also means that it has become harder to maintain the same rate of decline in lifespan variation: as premature deaths become rarer the marginal lifespan variation gains made from reducing "early" deaths tends to diminish (Vaupel, 1986). Tuljapurkar et al. (2000) add that over time the level of resources required to reduce mortality rates has increased but the marginal effectiveness has decreased because the causes of mortality are increasingly complex.

Despite these challenges most economically developed countries have continued to experience a decreasing lifespan variation trend. Decreases in lifespan variation are increasingly contingent upon reducing mortality rates across "early" ages of death (Oeppen, 2008, van Raalte, 2011a, Zhang and Vaupel, 2009). Scotland's inability to reduce mortality rates at these ages might partly explain why it is one of only a few countries that have not decreased lifespan variation.

5.2.2 Age-specific mortality change in Scotland

Scotland is found to have experienced higher lifespan variation relative to England and Wales when comparing the same time points. This finding was evidenced in chapter 4. In any given year Scotland has also been less effective at reducing premature mortality rates compared to England and Wales, with mortality rates for some age groups increasing between 1981 and 2001 (Norman et al., 2011, Leyland, 2004, Campbell et al., 2013). Norman et al. (2011)

demonstrated that the mortality rate for males in the areas of Scotland consistently classified as the most deprived increased between 1991 and 2001: from 649 per 100,000 to 743 per 100,000. Leyland et al. (2007b) illustrated that increasing premature mortality rates for ages 15-29 years between the same time period were mostly due to deaths from external causes: alcohol misuse, substance abuse, and suicides. Although age specific mortality change over time has tended to always be to Scotland's detriment compared to England and Wales (Campbell et al., 2013) the premature mortality patterns in Scotland are of particular concern; they were masked when looking at all-cause mortality rates for the total population of Scotland which have always decreased.

5.2.3 Research gaps

It is unlikely to be a coincidence that the timing of these increasing premature mortality rates in Scotland strongly corresponds with the timing when lifespan variation in Scotland diverged to a slightly increasing trend (Seaman et al., 2016a). Considering the existing evidence, it is valid to speculate that premature mortality in Scotland may account for the population level divergence in lifespan variation trend following the 1980s. Yet this question has not yet been the subject of any formal analysis. It is this research gap that this chapter will fill.

This chapter formally quantifies the ages of death driving the population level lifespan variation trend in Scotland over time compared to the ages of death driving the population level lifespan variation trend in England and Wales. It is important to understand if any of the age patterns of death driving higher and diverging lifespan variation in Scotland were distinguishable from the age

patterns in England and Wales, its closest comparator country. Any differences in the age distribution of death may point to differences in the aetiological pathways and social mechanisms that can better explain why population level lifespan variation in these two seemingly comparable countries differs, especially for males.

5.3 Data and methods

Data used in this chapter were sex specific lifetables for Scotland and England and Wales. Although England and Wales are two separate countries the governmental department responsible for collecting and reporting vital events data in both is the Office for National Statistics (ONS) (Philipov, 2015). It is National Records of Scotland who have responsibility over Scottish vital events statistics (Jasilionis, 2015, National Records of Scotland, 2014a). Therefore it is common practice in research and in official reporting of vital event statistics for England and Wales to be combined (Campbell et al., 2013, Majeed, 2013). As a result the data obtained from the Human Mortality Database were only available in this format. Chapter 3 provides a detailed description of a life table. Sex specific life tables for the years 1950-2010 for Scotland and for England and Wales were used: this was a total of 240 life tables.

Data were obtained for males and females separately because of their different mortality experiences. The sex differential in mortality is addressed within the context of this thesis by stratifying the analysis by sex rather than analysing the gap between males and females. This thesis recognises that there is a lifespan variation difference between males and females but that this type of comparison should be valued as a distinct research question in itself (Rigby and Dorling,

2007, White et al., 2014). Although some descriptive comparisons are made between the male trend and female trend this is not the focus of any formal decomposition analysis in this thesis.

5.3.1 Five year average mortality rate

The annual age-specific mortality rates were aggregated for each five year time period, beginning in 1950, and a five year average mortality rate calculated in Stata SE13. This was to smooth the age specific mortality rates. The decision to use five years' worth of data was informed by the approach used by van Raalte et al. (2014) when decomposing lifespan variation trends in Finland between 1971 and 2010. It made the number of decompositions to be carried out more manageable and the results clearer to interpret than annual changes in lifespan variation which were analysed in chapter 4. An alternative approach would have been to decompose the difference between the first and last year of each decade, which was applied by van Raalte (2011b) when studying the lifespan variation trend for Japanese females. Decomposing the difference between five years provided a more detailed analysis of the trends than only looking at the last year of each decade.

5.3.2 Age specific decomposition

The analysis in this chapter was used to partition the absolute change in lifespan variation within each country, between each five year time period, into age specific components. This was achieved by importing the five year mortality rates calculated in Stata SE13 into Excel spreadsheets developed by (Shkolnikov and Andreev, 2010). These spreadsheets apply Andreev's stepwise decomposition algorithm. A description of the 'general stepwise replacement algorithm' is

given in the data and methods chapter of this thesis. In summary it is an approach which is appropriate for decomposing, by age, changes in any aggregate measure that is estimated from age-specific mortality rates and accounts for all ages including the oldest open ended age.

This chapter applies stepwise decomposition to calculate the age-specific contributions to the lifespan variation change within Scotland over time compared to the lifespan variation change within England and Wales over time. Decomposing lifespan variation changes within each country and then comparing the results answers the two research questions. If the age specific mortality change in Scotland differed from the age specific mortality change in England and Wales this might suggest which aetiological processes and social mechanisms may be responsible for Scotland's diverging lifespan variation trend. The absolute change in life expectancy was also decomposed by age but is not reported. Although the life expectancy analysis is of interest it was not required to answer the specified research questions in this chapter.

5.4 Results

5.4.1 Estimates of lifespan variation for five year time periods

The estimates of lifespan variation (e†) calculated for each five year time period are detailed in table 14.

Table 14 e† estimates calculated from five year average mortality rate

	Males		Females	
Years mx averaged	**Scotland**	**Eng. & Wales**	**Scotland**	**Eng. & Wales**
1950-1954	13.94	13.04	13.18	12.28
1955-1959	13.25	12.49	12.24	11.68
1960-1964	13.00	12.41	12.03	11.52
1965-1969	12.77	12.25	11.90	11.46
1970-1974	12.51	12.07	11.79	11.33
1975-1979	12.19	11.72	11.61	11.07
1980-1984	11.84	11.40	11.30	10.84
1985-1989	11.58	11.21	11.04	10.66
1990-1994	11.49	11.04	10.88	10.43
1995-1999	**11.53***	10.85	10.65	10.16
2000-2004	**11.61***	10.72	10.45	9.94
2005-2009	11.55	10.67	10.31	9.79

***Increase in lifespan variation**

When using five year averaged mortality rates the trends for lifespan variation generally correspond with the annual trends identified in the first chapters of this thesis and reported here. For both males and females, absolute levels of lifespan variation are higher in Scotland than England and Wales. This is the case at any given time point covered in the analysis. Of particular relevance to this thesis is the evidence that following the 1980s lifespan variation in Scotland changed from decreasing to increasing for males.

5.4.2 Absolute and relative change between five year time periods

The absolute change and relative percentage change in lifespan variation between each five year time period within Scotland and within England and Wales were then calculated allowing further interpretation. The relative change was calculated by dividing the change in lifespan variation by the absolute lifespan variation estimate at the previous time point. The absolute changes in lifespan variation are reported in table 15 for males and table 16 for females but are perhaps easier to visualise in figure 27 and figure 28.

Table 15 e† change over time, Males

	Scotland		England and Wales	
Years mx averaged	absolute (years)*	%	absolute (years)*	%
(A) 1950-54 to 1955-59	-0.69	-4.93	-0.55	-4.22
(B) 1955-59 to 1960-64	-0.25	-1.91	-0.07	-0.60
(C) 1960 -64 to 1965-69	-0.23	-1.79	-0.17	-1.33
(D) 1965-69 to 1970-74	-0.26	-2.03	-0.18	-1.48
(E) 1970-74 to 1975-79	-0.32	-2.52	-0.35	-2.88
(F) 1975-79 to 1980-84	-0.35	-2.88	-0.31	-2.68
(G) 1980-84 to 1985-89	-0.26	-2.18	-0.19	-1.68
(H) 1985-89 to 1990-94	-0.10	-0.84	-0.18	-1.57
(I) 1990-94 to 1995-99	***0.04***	***0.39***	-0.18	-1.66
(J) 1995-99 to 2000-04	***0.08***	***0.72***	-0.13	-1.22
(K) 2000-04 to 2005-09	-0.07	-0.56	-0.05	-0.47

*these columns are the values that were decomposed by age

Increasing lifespan variation reflects increasing inequality in age at death

Table 16 e† change over time, Females

	Scotland		England and Wales	
Years mx averaged	absolute (years)*	%	absolute (years)*	%
(A) 1950-54 to 1955-59	-0.94	-7.16	-0.60	-4.89
(B) 1955-59 to 1960-64	-0.21	-1.75	-0.17	-1.43
(C) 1960-64 to 1965-69	-0.12	-1.02	-0.06	-0.48
(D) 1965-69 to 1970-74	-0.12	-0.98	-0.13	-1.12
(E) 1970-74 to 1975-79	-0.18	-1.53	-0.26	-2.33
(F) 1975-79 to 1980-84	-0.31	-2.63	-0.23	-2.10
(G) 1980-84 to 1985-89	-0.26	-2.33	-0.18	-1.63
(H) 1985-89 to 1990-94	-0.15	-1.40	-0.23	-2.17
(I) 1990-94 to 1995-99	-0.24	-2.19	-0.27	-2.59
(J) 1995-99 to 2000-04	-0.19	-1.79	-0.22	-2.12
(K) 2000-04 to 2005-09	-0.14	-1.37	-0.15	-1.54

*these columns are the values that were decomposed by age

Increasing lifespan variation reflects increasing inequality in age at death

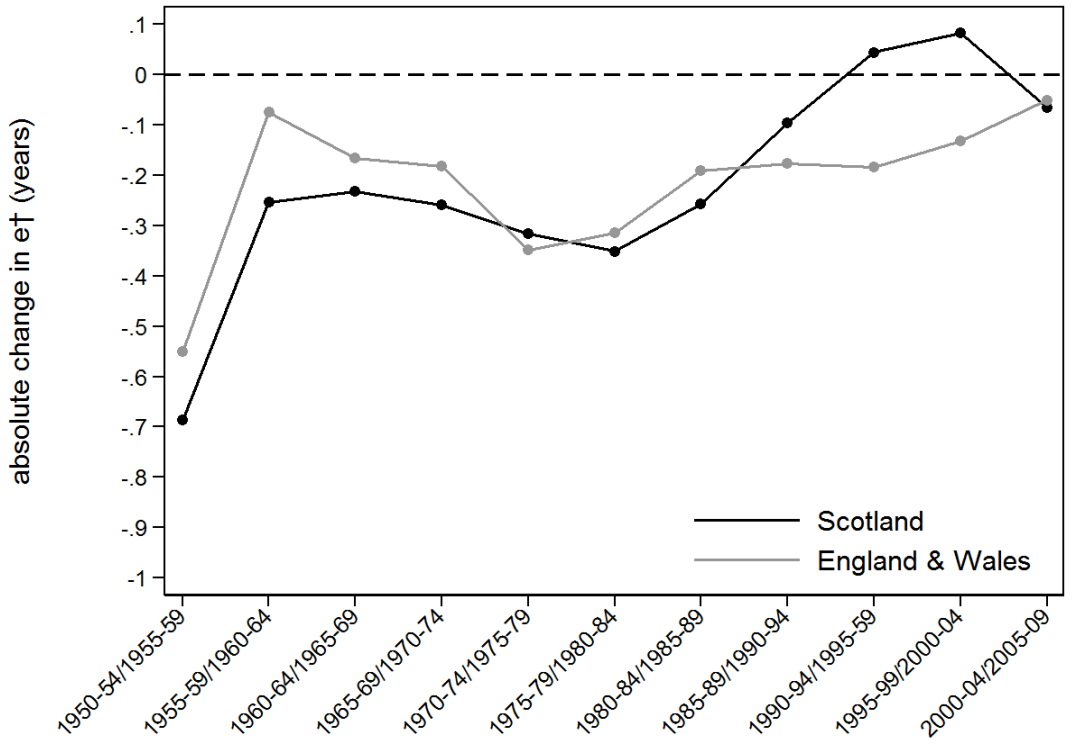


Figure 27 Change in lifespan variation over time, Males

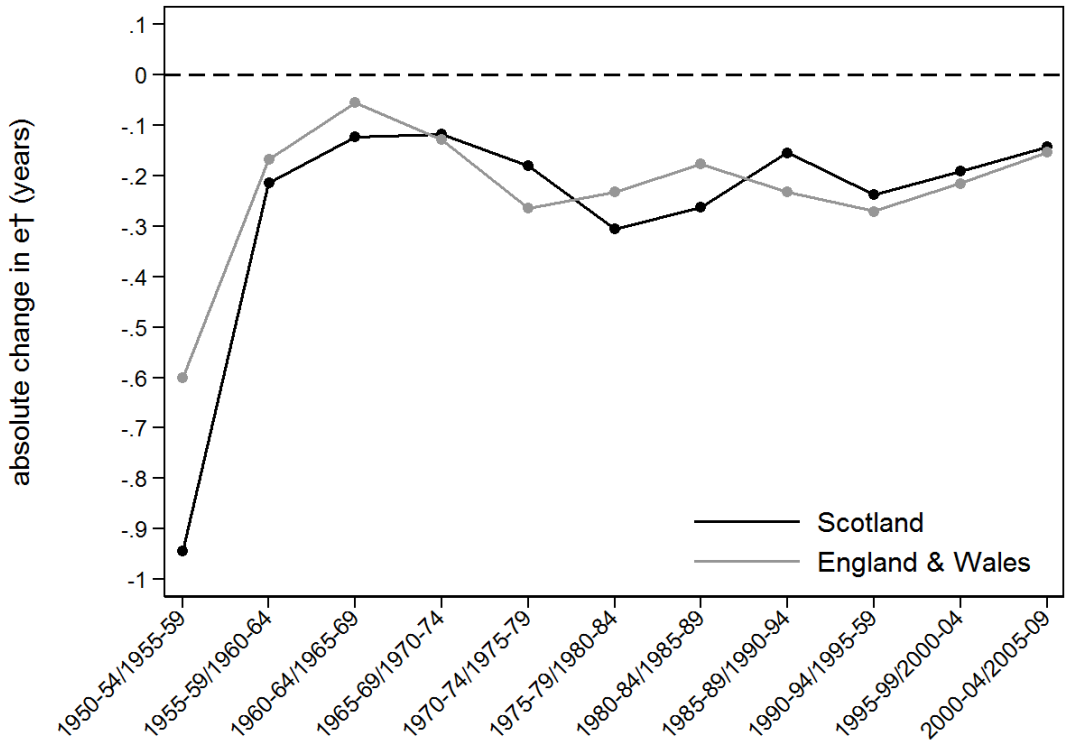


Figure 28 Change in lifespan variation over time, Females

5.4.2.1 Males

At the earliest time points the decrease in lifespan variation was greater for males in Scotland than males in England and Wales. For males in Scotland there was a -0.25 years decrease in lifespan variation between 1955-59 and 1960-64. This was greater than the -0.07 years decrease in England and Wales for the same time period. During the middle of the time period the decrease in lifespan variation for males in Scotland and males in England and Wales is similar: the grey and black lines are closer. However this is always within the context of higher absolute levels of lifespan variation in Scotland. The change in lifespan variation in Scotland then diverges from England and Wales and crosses the zero line. Between 1990-94 and 1995-99 there was a lifespan variation increase of 0.04 years. This is not the case for males in England and Wales: between 1990-94 and 1995-99 there was a still a decrease in lifespan variation of -0.18 years. This is evident as the grey and black lines are furthest apart. Scotland then achieves a small decrease in lifespan variation between 2000-04 and 2005-2009 and is similar to the decrease in England and Wales, yet still within the context of higher absolute levels of lifespan variation.

5.4.2.2 Females

The lifespan variation trend for females in Scotland and England and Wales was always decreasing in this period. There have been times when females in Scotland were experiencing relatively more favourable decreases, but within the context of higher absolute lifespan variation. For example between 1960-64 and 1965-69 lifespan variation decreased in Scotland by -0.12 years while England and Wales reduced it by -0.06 years. Over time the decreases for females in Scotland have changed to be slightly smaller than the decreases in England and

Wales and within the context of higher absolute levels of lifespan variation. For example between 1985-89 and 1990-94 in Scotland lifespan variation decreased by -0.15 years but in England and Wales it decreased by -0.23 years. The divergence in Scotland evident for males is not evident for females.

5.4.3 Scotland and England and Wales

5.4.3.1 Death distributions

The fact that Scotland experienced a higher absolute level of lifespan variation over the entire time period, alongside the differences in the level of change in lifespan variation achieved and the increasing lifespan variation trend indicates that the age distributions of death must differ between Scotland and England and Wales. Figure 29 provides a comparison of the age distributions of death (%) for males in Scotland and males in England and Wales for the earliest time period (1950-54) and the latest time period (2005-09).

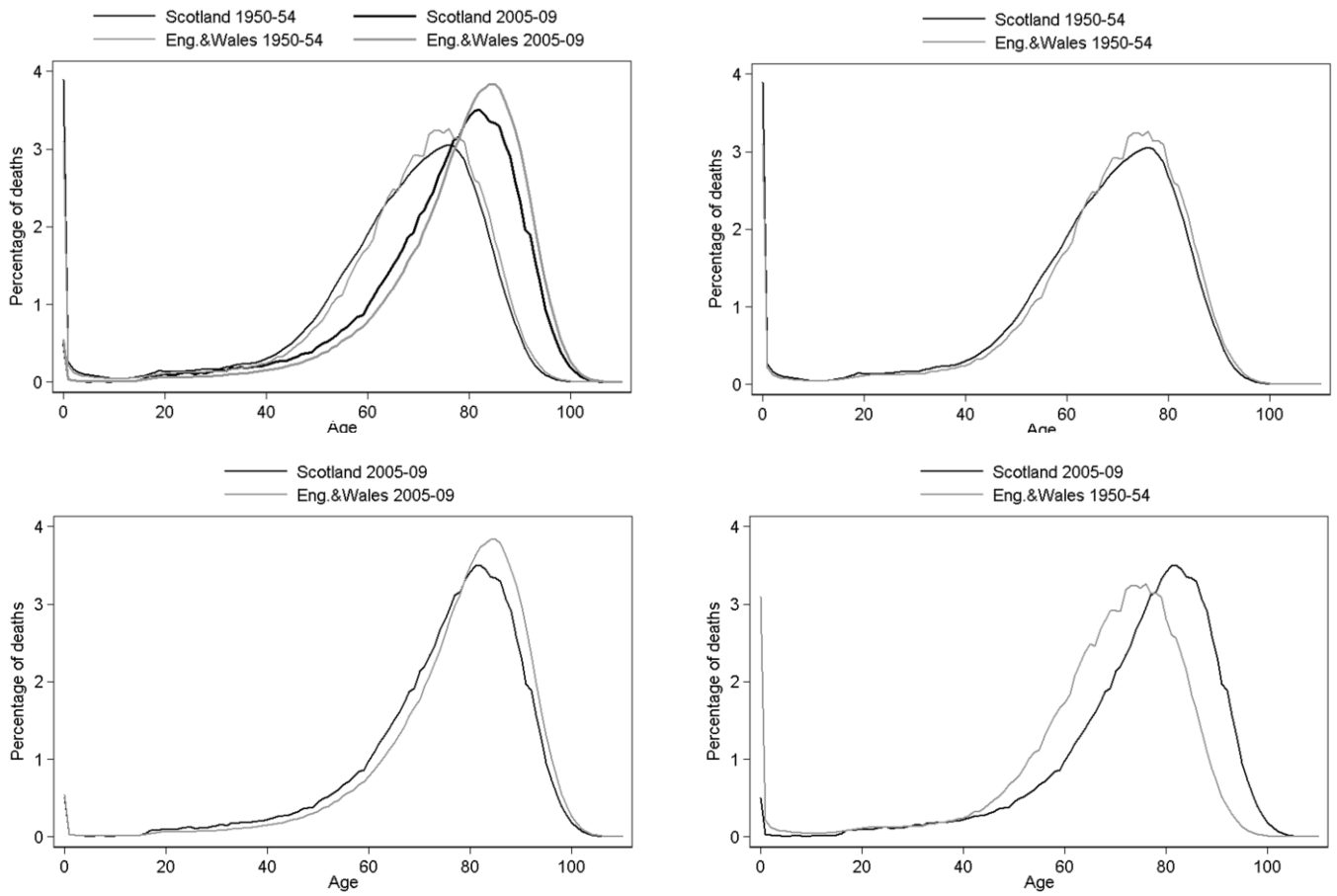


Figure 29 Comparison of death distribution over time in Scotland and in England and Wales, Males

For males the death distributions for the two countries appear more similar in 1950-54 than in 2005-09. In 1950-54 the left hand tail of the distribution is long and the percentage at deaths across these ages is slightly higher in Scotland than in England and Wales. By 2005-09 this gap had widened and premature deaths towards the left hand are higher in Scotland than in England and Wales. A greater percentage at deaths in England and Wales occur at older ages in 2005-09 than in Scotland in 2005-09. However perhaps of most concern is that the percentage at deaths across the left hand tail of the distribution for males in Scotland in 2005-09 is very similar to the percentage at deaths across the left hand tail of the distribution for males in England and Wales in 1950-54. Infant mortality is an exception.

Figure 30 provides a comparison of the age distributions of death (%) for females in Scotland and females in England and Wales for the period 1950-54 and 2005-09.

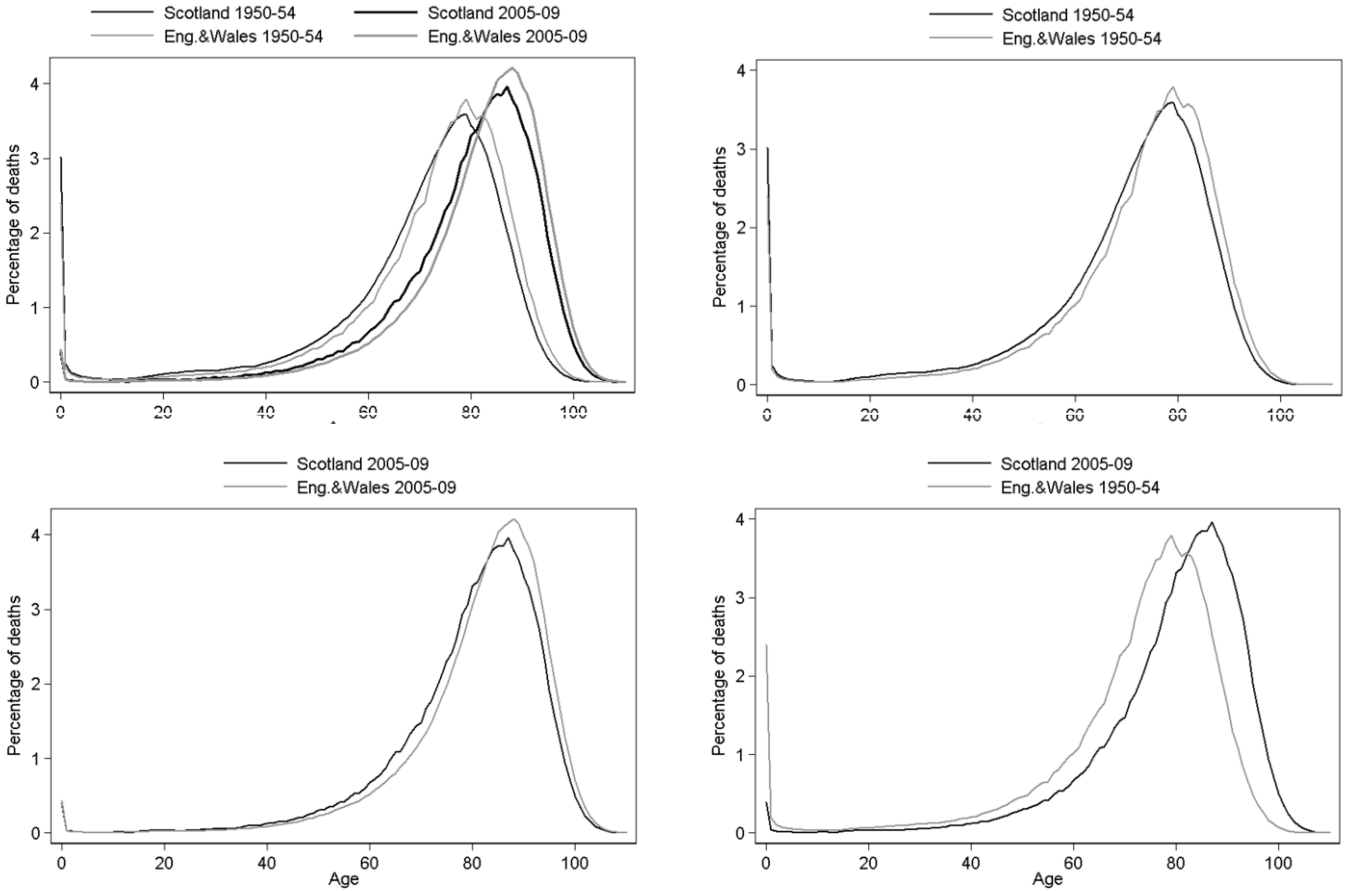


Figure 30 Comparison of death distribution over time in Scotland and in England and Wales, Females

For females the age at death distributions for the two countries are similar in 1950-54 than in 2005-09. In 1950-54 the percentage at deaths at ages in the left hand tail of the distribution is slightly higher in Scotland than in England and Wales. By 2005-09 this gap had widened and premature deaths towards the left hand are higher in Scotland than in England and Wales. A greater percentage at deaths in England and Wales occur at older ages in 2005-09 than in Scotland in 2005-09. However, unlike for males, the percentage at deaths across the left hand tail of the distribution for females in Scotland in 2005-09 is lower than the percentage at deaths across the left hand tail of the distribution for females in England and Wales in 1950-54.

The remaining analysis in this chapter quantifies the impact changes to the age specific mortality rates have had on the population level change in lifespan variation. It compares Scotland with England and Wales in order to identify if any of the age contributions in Scotland are distinguishable.

5.4.4 Age decomposition of change in lifespan variation

Age-specific decomposition results for lifespan variation throughout this chapter are presented as spike graphs (figure 31 to figure 38). The spike graphs show the age specific components that make up the total change in lifespan variation between each five year time period. The spike graphs in this chapter and the age process they reflect are interpreted in the following two ways.

Firstly, spikes which are below the zero line are desirable in terms of decreasing lifespan variation. These correspond to ages at which mortality change has contributed to decreasing lifespan variation. Decreases in lifespan variation are

achieved if mortality rates are lower in the later time period across “early” ages of death which drive mortality compression.

Secondly, spikes which are above the red zero line correspond to ages at which mortality change has contributed to increasing lifespan variation. Increases in lifespan variation may occur through two different processes. Higher mortality rates across “early” ages of death later in time which prevent mortality compression and lower mortality rates across “late” ages of death later in time which cause mortality expansion.

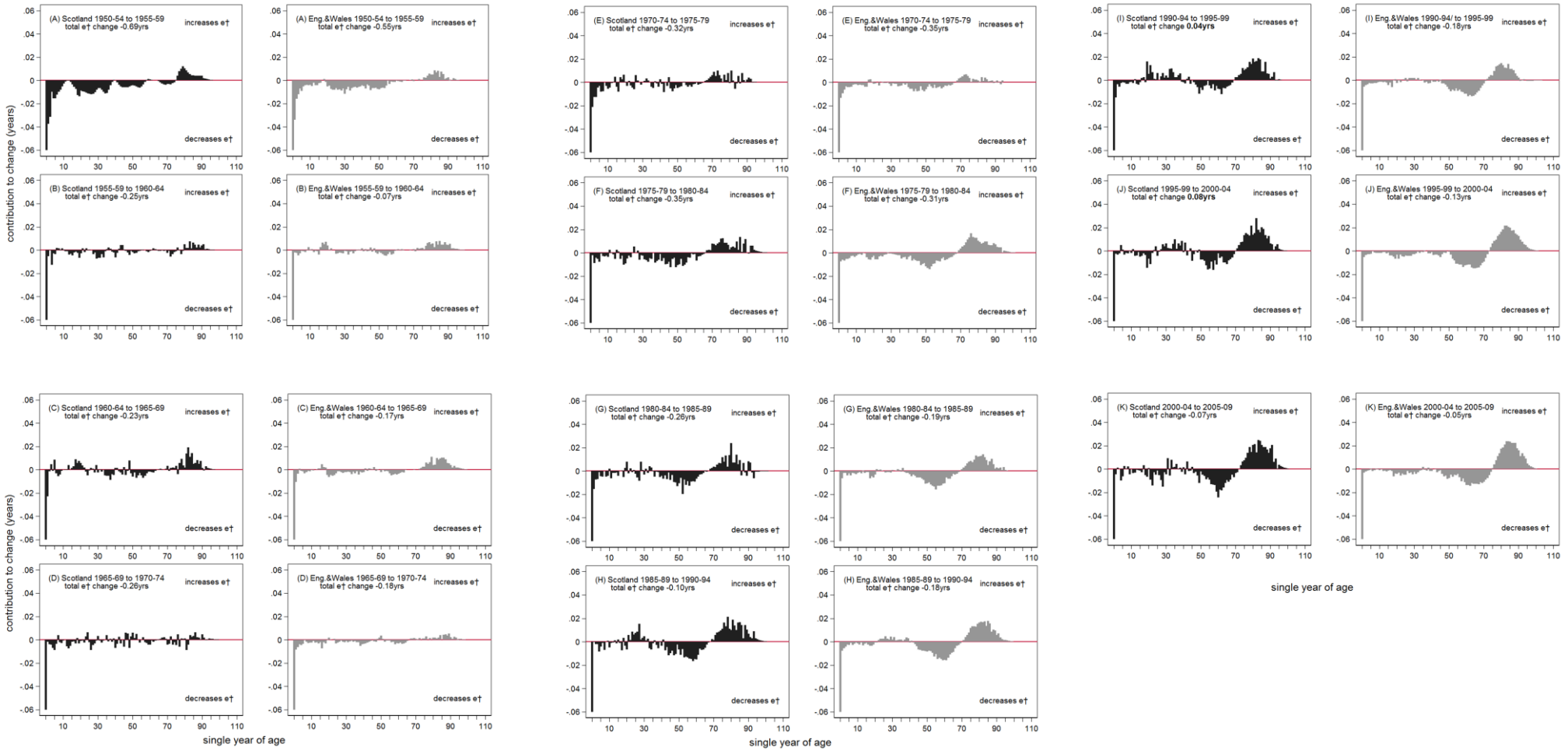


Figure 31 Age specific components of total change in lifespan variation between each five year time period in Scotland (black spikes) compared to in England and Wales (grey spikes), Males

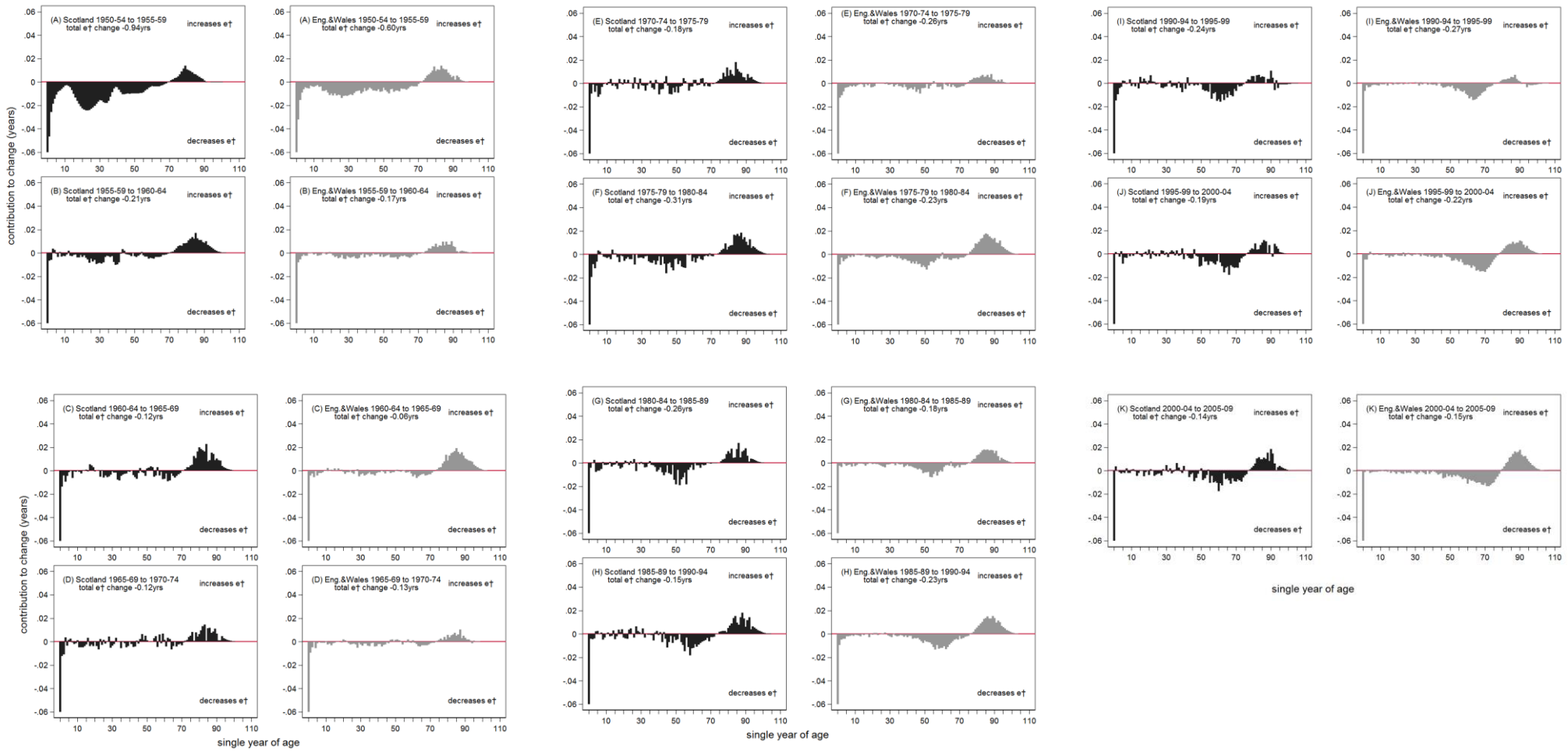


Figure 32 Age specific components of total change in lifespan variation between each five year time period in Scotland (black spikes) compared to in England and Wales (grey spikes), Females

Over time some of the age specific patterns and the size of the contributions in Scotland are similar to England and Wales but there are also notable differences. The age specific patterns driving lifespan variation over time can be described in terms of whether the change in mortality rate within each country, later in time, (1) contributed to mortality compression, (2) prevented mortality compression, or (3) contributed to mortality expansion.

5.4.4.1 Dense contributions driving mortality compression

Between 1950-54 and 1955-59 Scotland experienced a decrease in lifespan variation of -0.69 years for males. For the same time period males in England and Wales experienced a decrease of -0.55 years. Between these two time periods the general pattern of mortality decline in Scotland was similar to England and Wales as illustrated in figure 33.

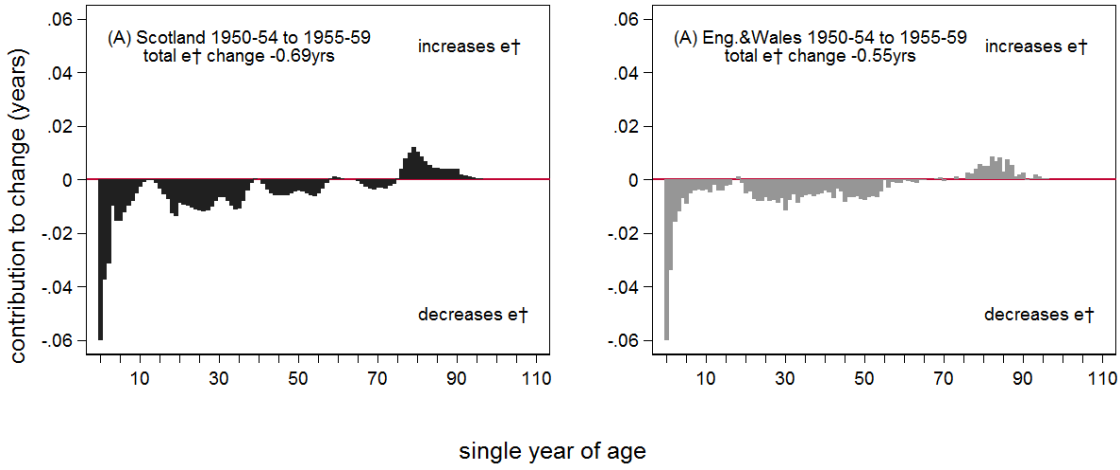


Figure 33 Age contributions from mortality change between 1950-54 and 1955-59 to total change in lifespan variation, Males

Contributions to mortality compression were made from reducing mortality rates across most ages in Scotland and in England and Wales. These contributions can be described as dense contributions: the spikes are consistent across all ages without any fluctuations across the zero lines. The spikes for most ages in Scotland and England and Wales are consistently below the red zero line. Lower mortality rates, later in time, across ages 0 to 74 years old contributed -0.79 years to the total change in lifespan variation in Scotland. In England and Wales lower mortality rates across these ages contributed -0.63 years to the total change in lifespan variation. Although lower mortality rates across some older ages were contributing to mortality expansion these contributions were offset. In Scotland ages 75+ contributed 0.10 years to the change in lifespan variation. In England and Wales ages 75+ contributed 0.07 years to the total change in lifespan variation.

A similar pattern is illustrated for the total change in lifespan variation in the earliest period for females in figure 34. However the contributions from lower mortality rates later in time were larger than for males because the improvements in lifespan variation were greater for females. For females in Scotland falling mortality rates from ages 75+ contributed 0.11 years to the total change in lifespan variation: in England and wales ages 75+ contributed 0.16 years to the total change in lifespan variation. These contributions were offset by greater decreases in mortality rates across ages 0-74 years old: for females in Scotland these ages contributed -1.06 years to the total change in lifespan variation, for females in England and Wales the contribution to the total change was -0.76 years.

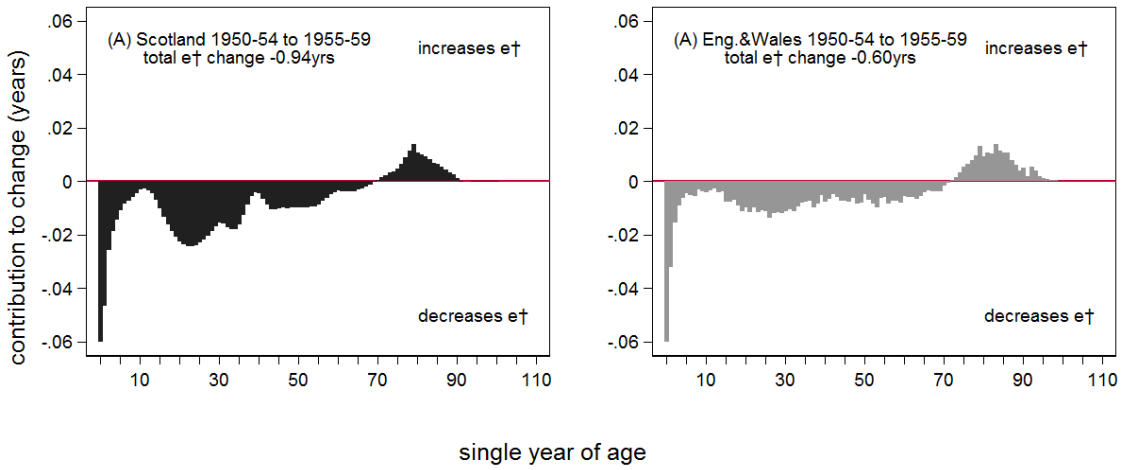


Figure 34 Contributions from mortality change between 1950-54 and 1955-59 to total change in lifespan variation, Females

5.4.4.2 Fluctuating contributions

As time progresses the dense pattern driving mortality compression, and contributing to decreasing lifespan variation, changes. For example the dense contributions are no longer evident between 1965-69 and 1970-74 in either Scotland or England and Wales. Rather fluctuating patterns are seen. Between these time periods males in Scotland experienced a decrease in lifespan variation of -0.26 years. For the same time periods males in England and Wales experienced a decrease of -0.18 years. Between 1965-69 and 1970-74 the general pattern of mortality decline in Scotland was similar to England and Wales as illustrated in figure 35.

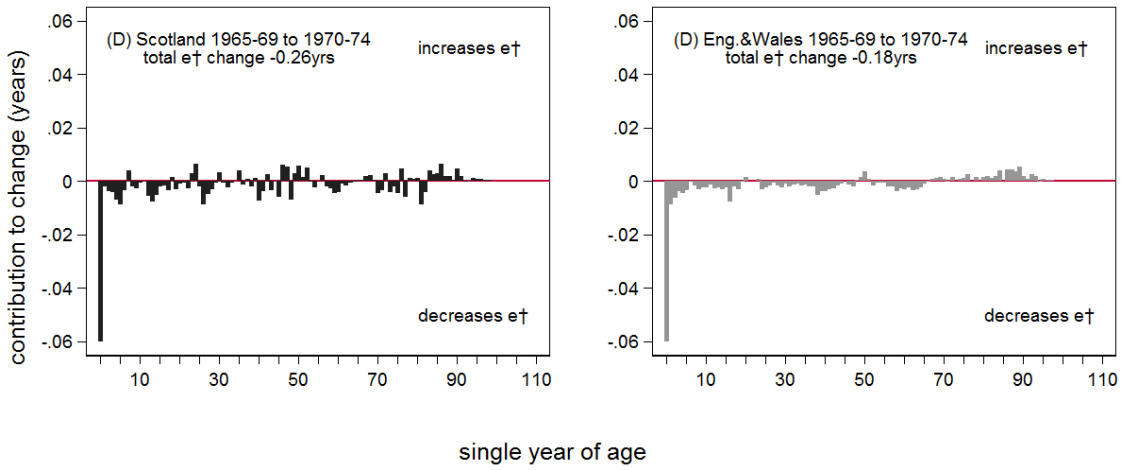


Figure 35 Contributions from mortality change between 1965-69 and 1970-74 to total change in lifespan variation, Males

For males in Scotland contributions to the total change in lifespan variation between 1965-69 and 1970-74 from each age fluctuate and cross the zero line but are relatively small. In England and Wales the density of the contributions is reduced and there is some fluctuation but to a much lesser extent than in Scotland. Decreasing mortality rates during the first year of life are continuing to drive mortality compression. This, alongside limited contributions from older ages to mortality expansion, means that an overall decreasing trend was maintained. The same fluctuating pattern is seen when comparing females in Scotland with females in England and Wales as illustrated in figure 36.

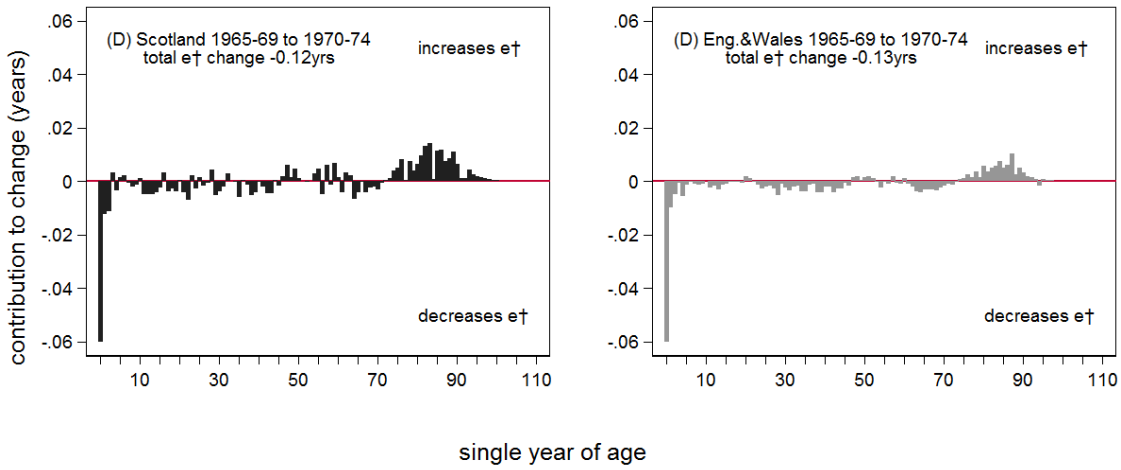


Figure 36 Contributions from mortality change between 1965-69 and 1970-74 to total change in lifespan variation, Females

Fluctuating contributions to lifespan variation in Scotland indicates that mortality rates across all ages were not consistently lower later in time. The absence of a fluctuating pattern in England and Wales demonstrates that mortality rates were consistently lower later in time, although not as large as the reductions made between earlier time points.

5.4.4.3 Offsetting mortality expansion

The most recent changes in the age specific patterns of death do not demonstrate a fluctuating pattern. In both countries there is evidence of a shift towards a two peaked distribution but the age patterns of change in Scotland and in England and Wales are quite different.

A two peaked distribution is generally characterised by mortality compression at the left of the distribution and mortality expansion at the right hand of the distribution. In order to maintain decreases in lifespan variation the contributions from the left hand of the distribution must offset the contributions

from the right hand of the distribution. Scotland did not achieve this between 1990-94 and 1995-99 or between 1995-99 and 2000-04 (figure 37) but England and Wales did.

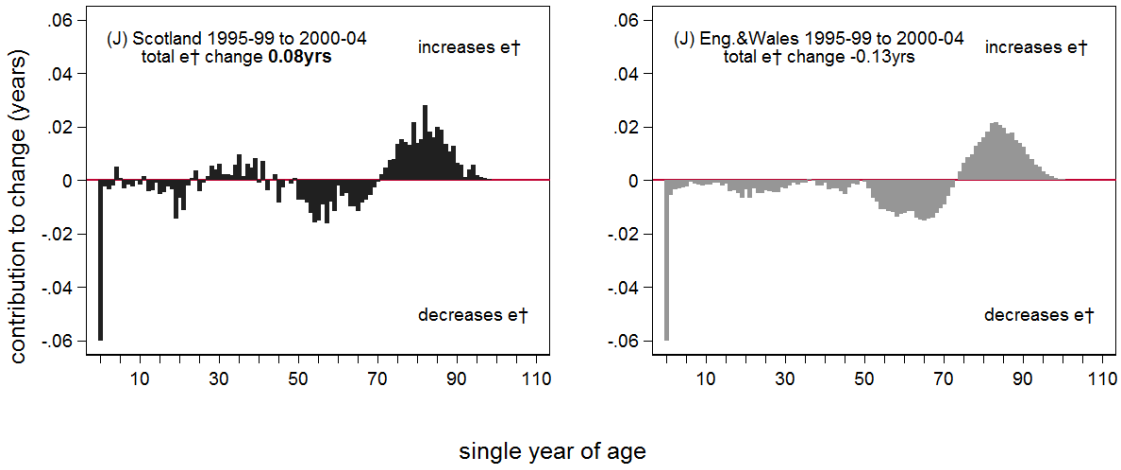


Figure 37 Contributions from mortality change between 1995-99 and 2000-04 to change in life expectancy, Males

Scotland's increasing lifespan variation was not due to mortality across infant/early life ages. Mortality rates across ages 0 to 15 years old have tended to always be lower in the later time period resulting in mortality compression and contributing to decreasing lifespan variation.

Mortality rates across ages 16 to 69 years were not consistently lower in Scotland in the later time period. For some premature adult ages mortality rates were higher in the later time period, evidenced by the contributions to increasing lifespan variation. This partly explains why Scotland's trend changed. Higher mortality rates across ages 20 to 40 years old in 1995-99 compared to 1990-94 contributed 0.09 years to the total change in lifespan variation. Higher mortality rates across ages 20 to 40 years old in 2000-04 compared to in 1995-99 contributed 0.04 years to the total change in lifespan variation.

For males in Scotland decreasing mortality rates across ages 70+ contributed to increasing lifespan variation by expanding the age distribution of death. 0.22 years of lifespan variation were added to the total change between 1995-99 and 2000-04 from expansion. This could not be counterbalanced by the -0.18 years contributed from compression from ages <70 resulting in an overall increase in lifespan variation of 0.04 years for males in Scotland (lifespan variation changes over time detailed in table 14).

A similar pattern continued for males in Scotland between 1995-1999 and 2000-04: decreasing mortality rates across ages 70+ contributed to increasing lifespan variation by expanding the age distribution of death. 0.29 years of lifespan variation were added to the total change between 2000-04 and 1995-99 from expansion. This could not be counterbalanced by the -0.21 years contributed from the compression from ages <70 resulting in an overall increase in lifespan variation of 0.08 years.

This is not the case for males in England and Wales and there are no detrimental contributions, in terms of lifespan variation, from premature adult ages. For males in England and Wales mortality expansion from decreasing mortality rates at older ages is being offset by the contributions from mortality compression. Decreasing mortality rates causing expansion contributed 0.26 years to the total lifespan variation change in England and Wales. Mortality compression contributed -0.40 years in England and Wales.

These differences between Scotland and England and Wales are also found for females. This is illustrated in figure 38. However the detrimental contributions

for females in Scotland are not as large as they are for males in Scotland so an overall decreasing trend was maintained.

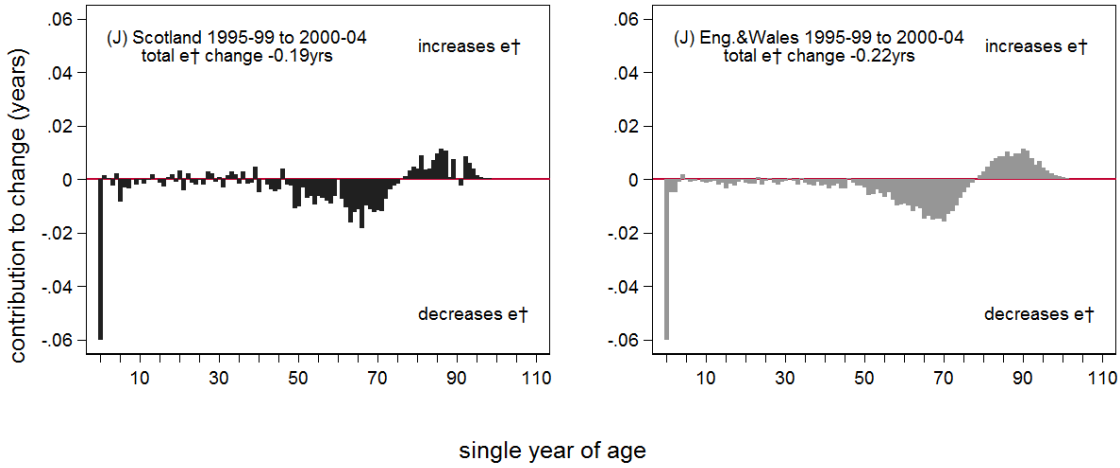


Figure 38 Contributions from mortality change between 1995-99 and 2000-04 to change in lifespan variation, Females

5.5 Discussion

5.5.1 Summary of results

This chapter extended the evaluation of the population level lifespan variation trend in Scotland, in a cross-national comparative context, by applying age decomposition analysis. Age decomposition analysis formally quantified the ages of death driving the lifespan variation trend in Scotland and allowed any differences, compared to England and Wales, to be identified. England and Wales were deemed to be the most appropriate comparator countries because they are Scotland's closest geographical neighbour and they share social, economic and political contexts. Despite this Scotland and England and Wales have contrasting mortality experiences. The analysis identified that detrimental contributions, in terms of lifespan variation, were made from increasing premature mortality rates in Scotland only: these contributions largely

accounted for the increasing lifespan variation trend. This age pattern was distinguishable from England and Wales; a distinction that was more evident when making comparisons between males in the two countries than between females.

The finding that the risk of death across some premature adult ages was higher for males in Scotland in 2000-04 than in 1995-99 and in 1995-99 than 1990-94 was not unexpected and is consistent with the existing body of literature that was published following the 2001 Census (Leyland et al., 2007b, Norman et al., 2011). The analysis in this chapter adds to the existing body of knowledge comparing Scotland with England and Wales and focusing on their contrasting experiences of premature mortality. It has a number of research strengths.

5.5.2 Strengths

Firstly, measuring lifespan variation to understand the mortality experience in Scotland is novel. Lifespan variation is calculated from death counts and population estimates. Suitable, high quality data have been available for Scotland for many years and lifespan variation is a measure of inequality which has an intuitive interpretation: it captures the average number of life years lost when death occurs within a population. Despite this, age decomposition analysis of lifespan variation has not previously been carried out for Scotland.

The changes to the lifespan variation trend within each country were decomposed by age using an established method developed by (Shkolnikov and Andreev, 2010). Using a five year average mortality rate was informed by van Raalte et al. (2014) study of lifespan variation trends in Finland between 1971 and 2010. This ensured that the number of decompositions carried out were

manageable and straightforward to interpret in contrast to annual changes. An alternative approach would have been to decompose the change between the first and last year of each decade (van Raalte, 2011b) but this approach would have relied on annual mortality data. Although population level mortality data are fairly stable they are not immune to random fluctuations. Therefore a further benefit of calculating an average mortality rate is that most random fluctuations would have been smoothed.

A further strength is the significant time period covered and the robust comparator country used to understand the relative context of Scotland's age pattern of mortality. England and Wales were selected on the basis that they share social, political and economic contexts with Scotland, along with a national government, as well as being Scotland's closest geographical neighbours. Despite all of these commonalities the two countries have contrasting mortality experiences, with the difference in lifespan variation being the focus of this thesis.

Studies comparing Scotland with England and Wales recognise that these two countries share a national government and have been exposed to many of the same social, economic and political contexts: factors which are deemed to be important for population health (Bambra, 2011a, Campbell et al., 2013). Although Scotland's trend could have been studied in isolation it is only possible to recognise the adversity of a problem in a country by extending the enquiry beyond the country of interest to account for any distinguishable differences in exposures (Rose, 2001); differences in exposures that may account for their contrasting mortality experiences.

5.5.3 Explanations for Scotland's premature mortality problem

A number of hypotheses have been proposed for Scotland's mortality disadvantage within Western Europe and relative to comparable countries, such as England and Wales. For example,Walsh et al. (2016) argue that Scotland was more vulnerable to the impacts of deindustrialisation and the implications this had for deprivation, poverty and the level of inequality in society. The underlying mechanisms associated with these theories could be associated with higher premature mortality.

The analysis in this chapter cannot ascribe any one reason why Scotland has been at a relative disadvantage in terms of premature mortality. Nor can this thesis provide any insight into the so called 'Scottish effect': the inability for area level social deprivation to account for Scotland's excess mortality relative to England and Wales (Popham and Boyle, 2011). This thesis does not compare Scotland with England and Wales while controlling for socioeconomic deprivation but it appreciates the important questions this body of research has presented. Similar to the literature specifically concerned with the 'Scottish effect' this thesis draws attention to Scotland's premature mortality problem and how this has contributed to widening mortality inequalities over time.

However this chapter has identified that the different population level lifespan variation trends in Scotland and England and Wales were driven by differences in the age patterns of mortality change (Shkolnikov et al., 2011, van Raalte, 2011a). This does not mean that the different age patterns themselves are the final explanation. Rather evaluating differences in the age patterns of mortality decline is only able to identify the countries which are lagging behind and

highlight the population sub-groups who are at risk of an untimely death. This is no easy undertaking especially because the causes of differences in premature deaths in adulthood are particularly complex with deaths at these ages being influenced by a range of mechanisms that may take time to change (Nau and Firebaugh, 2012, Vallin and Meslé, 2004). This is in contrast to historical changes in mortality decline associated with deaths in infancy. These were more uniformly achieved by all economically advanced countries because they tended to have a common underlying cause that could be directly tackled (Smits and Monden, 2009, Vallin and Meslé, 2004).

5.5.4 Lifespan variation independent of life expectancy

This chapter focused on comparing chronological changes in the age pattern of mortality decline in Scotland with the contemporaneous chronological changes within England and Wales. It quantified the impact any changes have had in terms of lifespan variation. This thesis has so far demonstrated, in relation to Scotland, that increases in life expectancy are not inevitably coupled with decreases in lifespan variation. Therefore the relationship between inequality and average population health is complex and these two measures are not simply a substitute for one another.

However in any given year countries with the highest life expectancy tend to have the lowest level of lifespan variation prompting some to question if lifespan variation can provide any additional insight over and above what we can deduct from measuring life expectancy. Criticisms focus on the fact that studies of crude changes in lifespan variation will almost completely reflect the crude changes in life expectancy (Smits and Monden, 2009). Therefore it has been

argued that it is perhaps more appropriate to study differences in lifespan variation between two populations when life expectancy is the same, regardless of the whether the same level of life expectancy was achieved at the same chronological time (Smits and Monden, 2009). This allows the relative difference in lifespan variation independent of life expectancy to be examined. Identifying the ages of death contributing to the difference in lifespan variation could indicate the types of aetiology that have caused unequal mortality reductions between relatively comparable countries experiencing similar levels of average population health (Smits and Monden, 2009, Wilmoth and Horiuchi, 1999).

5.5.5 Research implications

This critical insight into the limitations of the analysis in this chapter (looking only at crude changes over chronological time) therefore informed the analysis going forward. It is not yet known if there are any distinguishable differences in the age specific patterns of mortality when Scotland and England and Wales achieved comparable levels of average population health regardless of chronological time. This is the focus of the analysis in chapter 6.

It is important to reassess the concept of chronological time in order to better highlight important differences in the complex determinants of mortality decline, in particular across premature adult ages of death. The reasons why some determinants may differ in one country compared to another could be differences in terms of: public health strategies, medical advances, levels of social protection, temporal context or stage of epidemiological transition (Omran, 1971, Vallin and Meslé, 2004, Wilmoth and Horiuchi, 1999).

Given that Scotland and England and Wales share similar social and economic contexts and a national government it could be assumed that these important determinants of age specific mortality decline are the same. However there have been some notable differences highlighted in the literature (Walsh et al., 2016, McCartney, 2012, Hanlon et al., 2005). For example the impact of deindustrialisation was more profound for Scotland and it has historically been exposed to higher levels of deprivation and greater levels of relative inequality. Therefore the cross national comparative context of this chapter and chapter 6 help to inform our understanding of the types of determinants that might explain why the age pattern of mortality decline in Scotland is distinguishable from England and Wales, and which social determinants within Scotland could be deemed most important for explaining lifespan variation.

5.6 Conclusion

The results in this chapter formally identified the ages of death which have driven the crude lifespan variation trend for Scotland compared to the ages of death in England and Wales. The decomposition analysis allowed distinguishable age patterns of mortality decline in Scotland to be identified and formally quantified the contributions made by changes to the age pattern of mortality. Identifying differences in the age pattern of death between Scotland and Wales helped understand the pathways and mechanisms accounting for Scotland's higher levels of lifespan variation and its faltering improvement following the 1980s.

6 Increasing inequality at shared levels of life expectancy

6.1 Introduction

The previous chapter examined changes to the age patterns of mortality driving lifespan variation changes over time. It formally quantified the contributions each age at death made to changes in lifespan variation over time within Scotland compared to within England and Wales. The age patterns driving the increase in lifespan variation in Scotland were distinguishable from the age patterns in England and Wales, particularly for males. Higher premature mortality rates in Scotland in recent years prevented mortality compression. This meant the contributions to increasing lifespan variation from fewer deaths at older ages (mortality expansion) were not offset.

This chapter continues to focus on lifespan variation in Scotland compared to England and Wales. It utilises some of the same lifetable data obtained from the HMD used in chapter 5 and applies the same age-specific decomposition analysis. It extends our understanding and interpretation of population level lifespan variation by comparing Scotland with England and Wales when the same level of life expectancy was achieved, albeit at different points in time. This allows lifespan variation to be studied independently of life expectancy which Smits and Monden (2009) argue may be more appropriate for understanding the different social determinants that may be most important for increasing average health alongside reducing inequality.

There are four main sections in this chapter. The first background section summarises the literature which has evaluated the reasons why countries achieving a high level of life expectancy at different points in time may do so with different levels of lifespan variation. Scotland has tended to achieve the same level of life expectancy as England and Wales later in time but it is not yet known whether this has been with any lifespan variation advantage or disadvantage or what ages of death account for any lifespan variation gap. Therefore this chapter answers the following research questions:

5. Was lifespan variation higher or lower in Scotland at a shared level of life expectancy with England and Wales?

6. Which ages of death account for the lifespan variation gap between Scotland and England and Wales at a shared level of life expectancy?

Answering these questions helps to understand which differences in the age-specific processes of mortality change may be most important in terms of achieving the association between high average population health and low inequality. The second section briefly describes the data used for the analysis and the interpretation of age-specific decomposition as it is applied within this chapter. The third section describes the results. The final section outlines how the findings from the population level analysis chapters informed the decision to study deprivation specific trends in lifespan variation going forward in this thesis.

6.2 Background

In any given year, countries with the highest levels of life expectancy tend to have the lowest levels of lifespan variation (Smits and Monden, 2009, Vaupel et

al., 2011). Scotland currently experiences the lowest level of life expectancy and highest level of lifespan variation in Western Europe (McCartney et al., 2012b, Seaman et al., 2016a). This demonstrates the strong negative correlation between life expectancy and lifespan variation.

Given the correlation, alongside the fact that they represent distinct dimensions of mortality, Smits and Monden (2009) argued that it may be more appropriate for cross national comparisons to focus on the lifespan variation gap when similar levels of life expectancy were achieved, regardless of when they were achieved. This approach allows differences in lifespan variation to be studied independently of life expectancy.

This reinterpretation of the time dimension is important to explore as countries reaching the same level of life expectancy, particularly at differing times, tend to do so with underlying differences in age specific mortality rates, resulting in different levels of lifespan variation (Shkolnikov et al., 2011, Auger et al., 2014, Nau and Firebaugh, 2012). There are at least three possible scenarios that can be considered. The first two were set out by Smits and Monden (2009) and this thesis proposes the third scenario.

Firstly, countries reaching a level of life expectancy later in time may do so with low levels of lifespan variation. Life expectancy pioneers have historically reduced premature mortality rates the most in order to achieve their high levels of life expectancy, which in turn equates to low lifespan variation. This means countries arriving at a high life expectancy later in time will do so with very similar levels of lifespan variation to the pioneers. This is termed the forerunner hypothesis.

Secondly, countries reaching the same level of life expectancy later do so with lower lifespan variation than life expectancy pioneers. This is because they have a temporal advantage and can capitalise on the lessons already learnt about reducing premature mortality rates from the countries ahead of them. Or as Vallin and Meslé (2004) state "the most recent arrivals advance more rapidly than the pioneers". This is termed the diffusion hypothesis.

Thirdly, and perhaps surprisingly, there may be some developed countries achieving the same life expectancy later in time but with considerably higher levels of inequality. This suggests that there are countries that are failing to benefit fully from their temporal advantage. This is somewhat counter-intuitive as the economic cost of reducing premature adult mortality – deaths associated with external causes – tend to be lower than the economic costs of reducing old age mortality – deaths from chronic diseases (Smits and Monden, 2009, Nau and Firebaugh, 2012). Vallin and Meslé (2004) therefore suggest that not all societies are equally prepared to draw on the benefits of earlier innovation or replicate outside practices. This thesis termed this the laggard hypothesis.

It is therefore argued that any relative difference in lifespan variation, at a shared level of life expectancy, may reflect contrasting influences on mortality rates between countries (Smits and Monden, 2009). These contrasting influences may include differences in terms of: the social determinants of health, public health strategies, temporal contexts, or stages of epidemiological transition (Wilmoth and Horiuchi, 1999, Omran, 1971, Vallin and Meslé, 2004).

These broad types of determinants of mortality are relevant for Scotland and have already been incorporated into some of the hypotheses proposed for

tackling Scotland's premature mortality problem. For example the association between deindustrialisation, social deprivation and the level of social protection provided to protect those most at risk of premature death (Walsh et al., 2016).

6.2.1 Research gap

The current literature looking at mortality in Scotland has tended to place an emphasis on the upstream explanations that link structural changes in employment and the political landscape to poverty, deprivation and in particular inequality in Scotland. Yet the relative level of lifespan variation, a novel measure of inequality, in Scotland has not previously been the focus of any formal analysis. It is this research gap that this chapter will fill.

Measuring the lifespan variation gap between Scotland and England and Wales, at similar levels of life expectancy, will help to distinguish between the three internationally relevant scenarios put forward for describing population level lifespan variation inequalities: the forerunner hypothesis, the diffusion hypothesis, or the laggard hypothesis. Formally quantifying the ages of death that account for the lifespan variation gap will provide further insight into the types of aetiological pathways and social mechanisms that have caused uneven patterns of mortality decline despite shared levels of average population health.

6.3 Data & methods

Data used in this chapter were the mortality rates from the sex specific life tables for Scotland and England and Wales provided by the Human Mortality Database. Chapter 3 provides a detailed description of a life table. The mortality rates from the life tables for the years 1949-2013 (the latest available year at the time of analysis) were used for the analysis in this chapter: this was a total

of 260 life tables. Results are reported for males and females separately because their mortality experiences differ.

Age specific mortality rates from the lifetables were used to calculate a three year rolling average annual mortality rate for each age and year from 1950 to 2012. The rolling average mortality rates give equal weight to each year. This approach for smoothing annual mortality rates, which are inherently vulnerable to random fluctuations, is consistent with the approach used by National Records of Scotland (2014b). The rolling average mortality rates were converted to life table probabilities of dying for males and females in Scotland and England and Wales separately. From these lifetable probabilities life expectancy and lifespan variation were calculated using methods implemented in a Microsoft Excel spreadsheet provided by the Max Planck Institute for Demographic Research and authored by Shkolnikov and Andreev (2010). It was appropriate to use 5 years' worth of data when comparing contemporaneous trends over time in order to make the number of decompositions more manageable. Smoothing over 3 years' worth of mortality data allows for more detail and is commonly applied within empirical research(Leyland et al., 2007b). For these reasons 3 years' worth of data were aggregated going forward in this thesis.

The analysis reported in this chapter utilised stepwise decomposition methods to calculate the age-specific contributions to any lifespan variation gap between Scotland and England and Wales at shared levels of life expectancy. The same level of life expectancy was also decomposed by age in order to illustrate that inequalities in the age distribution of death can exist even when life expectancy is comparable (Auger et al., 2014). This also establishes if the mortality rates

contributing to the lifespan variation gap were also detrimental to Scotland's life expectancy even when overall life expectancy was similar. However the primary focus of this thesis is on lifespan variation as a measure of inequality. For this reason any discussion of the analysis of life expectancy is limited.

6.4 Results

6.4.1 The association between life expectancy and lifespan variation over time

Figure 39 illustrates the level of lifespan variation that was achieved when the same level of life expectancy was achieved in Scotland compared to in England and Wales, for males. Figure 40 illustrates the same for females. Three comparisons are highlighted in colour; when the lifespan variation advantage to Scotland was greatest, when the lifespan variation gap was smallest and when the lifespan variation disadvantage to Scotland was greatest.

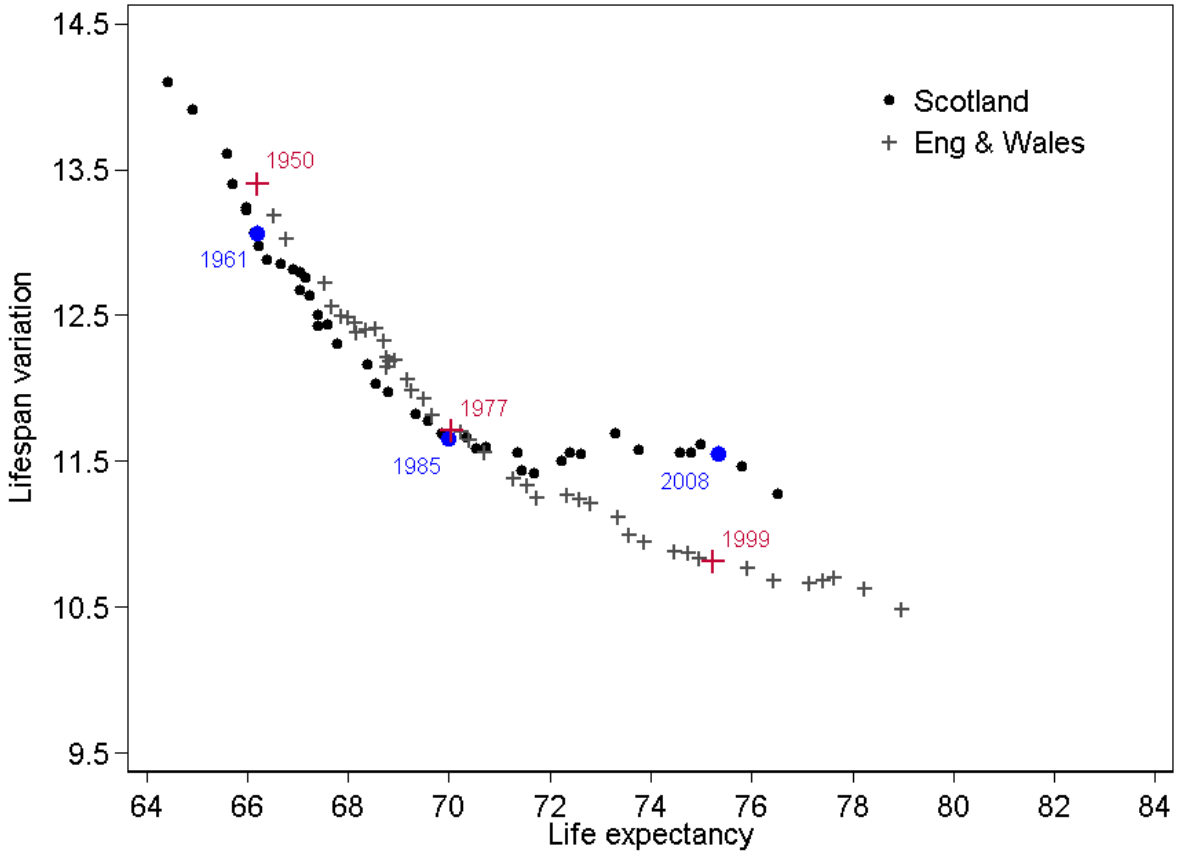


Figure 39 Association between life expectancy and lifespan variation, Males

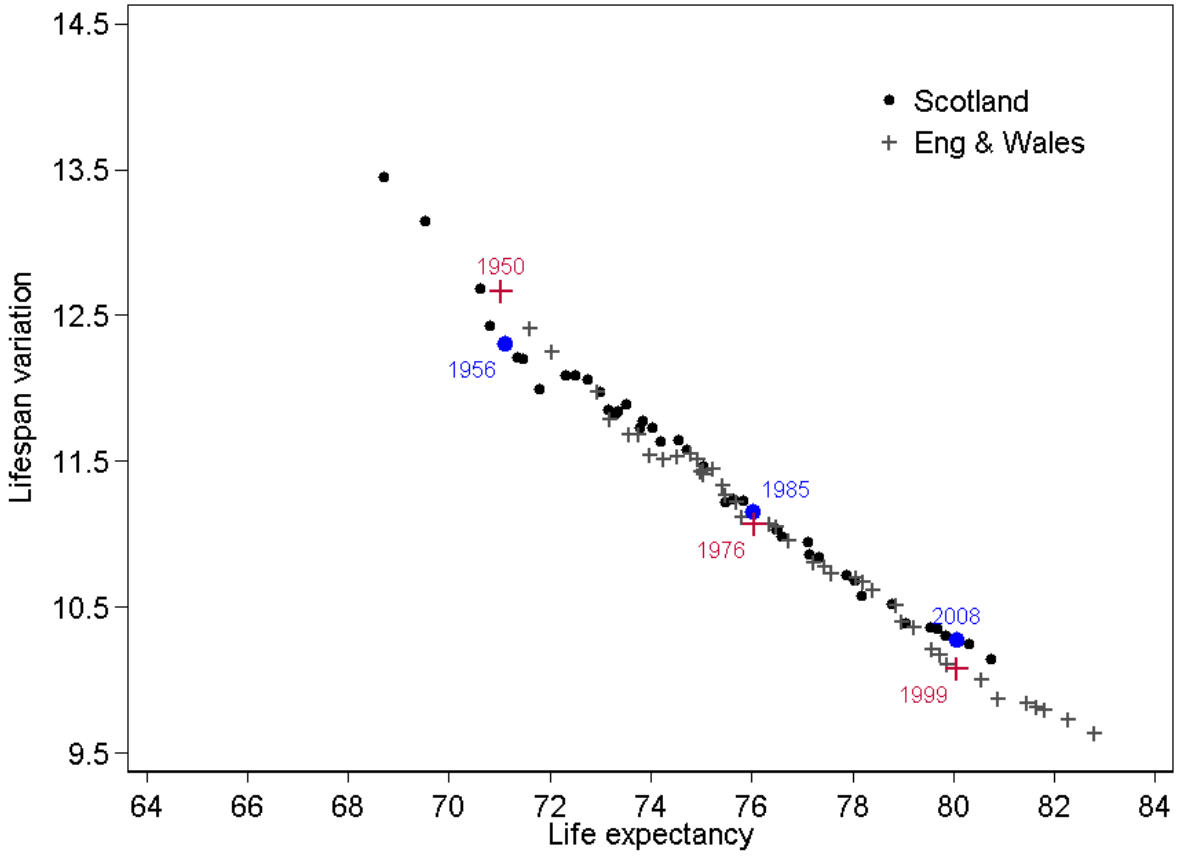


Figure 40 Association between life expectancy and lifespan variation, Females

Scotland has tended to achieve a similar level of life expectancy approximately ten years later than England and Wales. Scotland was previously able to achieve the same level of life expectancy later in time than England and Wales with slightly lower lifespan variation. Following 1985, for males, Scotland lost its lifespan variation advantage when it achieved a similar level of life expectancy later in time than England and Wales. For example males in Scotland reached the same life expectancy in 2008 as males in England and Wales in 1999 (about 75 years) but lifespan variation was greater in Scotland (years highlighted in figure 39). This same change is observed for females but the level of higher lifespan variation, at a shared level of life expectancy, is not as large as seen for

males in more recent years (the scatter plots for females in Scotland diverge less).

6.4.2 Death distributions

The lifespan variation gap at shared levels of life expectancy means that the age distributions of death must differ.

Figure 41 provides a comparison of the age distributions of death (%) for males in Scotland and males in England and Wales when a similar level of life expectancy was achieved. The three comparisons made are those highlighted in colour in figure 39.

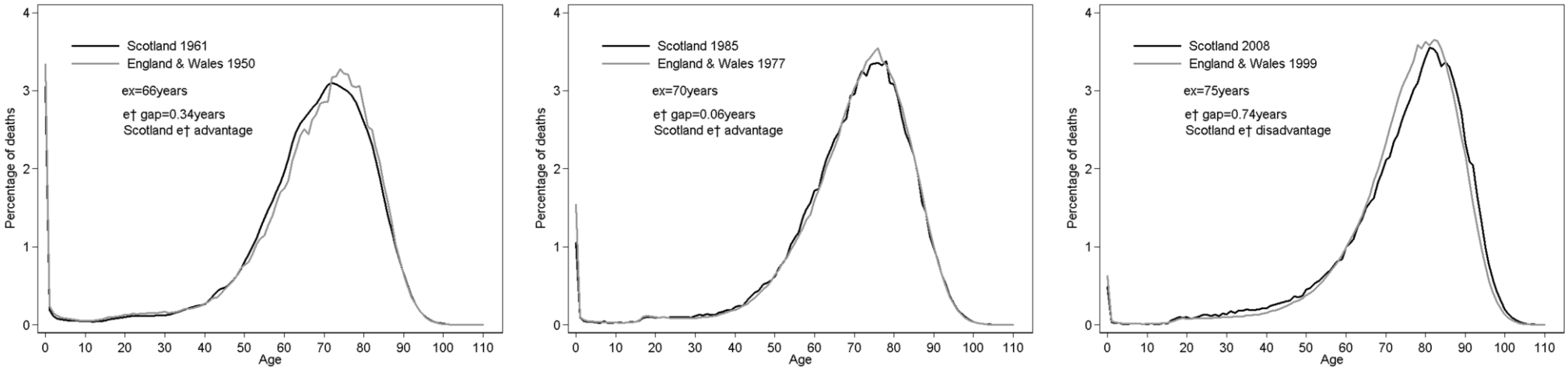


Figure 41 Comparison of death distributions in Scotland and England and Wales when shared level of life expectancy achieved, Males

For males the death distributions in Scotland and in England and Wales at a shared level of life expectancy are never exactly identical; this is not unexpected. Some of the differences in the age distributions are beneficial for Scotland in terms of lifespan variation while others are detrimental in terms of lifespan variation.

At a shared life expectancy of 66 years lifespan variation was 0.34 years lower in Scotland. This life expectancy was achieved in both countries with a similar percentage at deaths at younger ages and older ages. Lower lifespan variation in Scotland, at this level of life expectancy, will partly be associated with the slightly smaller percentage at deaths during the first year of life but will also be because of the lower percentage at deaths across ages 70 to 90 years (black line below the grey line). It is surprising that at this shared level of life expectancy there was a higher percentage at deaths across ages 50 to 70 years old (the black line is higher than the grey line) in Scotland.

At a shared life expectancy of 70 years lifespan variation was 0.06 years lower in Scotland. This is a much smaller lifespan variation gap than at a shared life expectancy of 66 years and the distributions of death are very similar. However it is concerning that the percentage at deaths from around age 30 to around 65 are higher in Scotland than in England and Wales despite the fact that Scotland achieved this life expectancy 8 years later.

At a shared life expectancy of 75 years lifespan variation was 0.74 years higher in Scotland. This is a much larger lifespan variation gap than seen at lower levels of life expectancy and in the opposite direction to the comparison at a shared life expectancy of 66 years. It is therefore not surprising that there was a more

notable difference in the age distributions of death. The percentage at deaths from around age 20 to around 60 are higher in Scotland than in England and Wales despite the fact that Scotland achieved this life expectancy 9 years later.

Figure 42 provides a comparison of the age distributions of death (%) for females in Scotland and females in England and Wales when a similar level of life expectancy was achieved. The three comparisons made are those highlighted in figure 40.

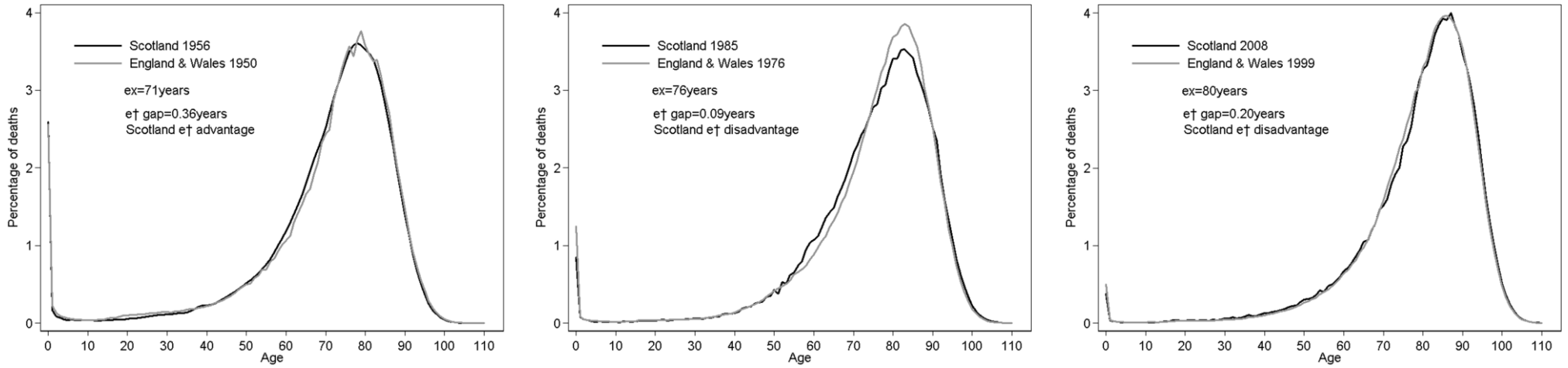


Figure 42 Comparison of death distributions in Scotland and England and Wales when shared level of life expectancy achieved, Females

For females the death distributions in Scotland and in England and Wales at a shared level of life expectancy are never identical. Some of the differences in the age distributions are beneficial in terms of lifespan variation while others are detrimental to Scotland.

At a shared life expectancy of 71 years lifespan variation was 0.36 years lower in Scotland. This life expectancy was achieved in both countries with a similar percentage at deaths at younger ages and older ages. Lower lifespan variation in Scotland, at this level of life expectancy, will partly be associated with the slightly smaller percentage at deaths during the first year of life: the percentage at deaths occurring across childhood and early life ages are also noteworthy (black line lower than grey line). However it will also partly be because of the lower percentage at deaths from age 80 years old which means there was limited expansion (black line very close to or below the grey line). It is also surprising that at this shared level of life expectancy there was a higher percentage at deaths across ages 50 to 70 years old (the black line is higher than the grey line) in Scotland despite the fact that it was achieved 6 years later in time.

At a shared life expectancy of 76 years lifespan variation was 0.09 years higher in Scotland. The lifespan variation gap has flipped to be to Scotland's disadvantage and will partly be associated with the slightly higher percentage at deaths at older ages leading to mortality expansion. It is of concern that the percentage at deaths from around age 50 to around 70 are higher in Scotland than in England and Wales despite the fact that Scotland achieved this life expectancy 8 years later.

At a shared life expectancy of 80 years lifespan variation was 0.20 years higher in Scotland. This is a larger lifespan variation gap than the earlier comparison. Although the gaps between the distributions are not as wide as the earlier comparisons there appears to be differences across more ages. The lifespan variation disadvantage in Scotland will partly be associated with the slightly higher percentages of deaths at older ages because these will contribute to mortality expansion: this is detrimental in terms of lifespan variation but indicates a greater proportion of the population are living to older ages. Perhaps of greater concern is that the percentage at deaths from around age 30 to around 65 are higher in Scotland than in England and Wales despite the fact that Scotland achieved this life expectancy 9 years later. However the difference between the two lines across these premature adult ages is not as large for females as it is for males. There is a small gap between the distribution from around age 70 to around age 80 years old with a lower percentage at deaths in Scotland.

6.4.3 Age-specific decomposition

The contributions that any differences in the age patterns of mortality made to the total lifespan variation gap between Scotland and England and Wales when achieving a similar level of life expectancy, albeit at different points in time, were quantified by carrying out age-specific decomposition. Decompositions were carried out for males at 11 shared levels of life expectancy. Decompositions for females were carried out 10 shared level of life expectancy. The shared levels of life expectancy and the lifespan variation gap that was decomposed by age are detailed in table 17 for males and table 18 for females.

Table 17 Decompositions carried out at shared levels of life expectancy, Males

Decomposition number	Shared life expectancy (approx.)	Year achieved (*England*: Scotland)	Lifespan variation gap*
1	66	*1950*:1961	-0.34
2	67	*1953*:1971	-0.36
3	68	*1959*:1976	-0.17
4	69	*1971*:1981	-0.22
5	70	*1977*:1985	-0.06
6	71	*1981*:1990	0.05
7	72	*1986*:1995	0.29
8	73	*1990*:2000	0.46
9	74	*1994*:2004	0.57
10	75	*1999*:2008	0.74
11	76	*2002*:2010	0.66
*negative lifespan variation gap(-) is to Scotland's lifespan variation advantage i.e. lower lifespan variation at shared level of life expectancy			

Table 18 Decompositions carried out at shared levels of life expectancy, Females

Decomposition number	Shared life expectancy (approx.)	Year achieved (*England*: Scotland)	Lifespan variation gap*
1	71	*1950*:1956	-0.69
2	72	*1952*:1962	-0.28
3	73	*1955*:1967	0.19
4	74	*1961*:1974	0.44
5	75	*1968*:1980	0.28
6	76	*1976*:1985	0.17
7	77	*1982*:1992	0.27
8	78	*1987*:1997	0.26
9	79	*1993*:2004	0.34
10	80	*1999*:2008	0.52
*negative lifespan variation gap(-) is to Scotland's lifespan variation advantage i.e. lower lifespan variation at shared level of life expectancy			

The following section describes how to interpret the decomposition graphs within the context of this chapter. The decomposition results are illustrated in figure 43 and figure 44 for males. The decomposition results are reported in figure 45 and figure 46 for females.

6.4.3.1 Description of decomposition graphs

There are two graphs for each decomposition. The first graph in column (a) shows the contribution of age differences in mortality rates to life expectancy (grey spikes). This is to demonstrate that age specific mortality can differ between countries even when life expectancy is comparable (Auger et al., 2014). If there were no differences in the age specific mortality rates, at the same level of life expectancy, there would be no spikes as there would be zero difference at every age. Chapter 4 established that there will be differences in the age specific mortality rates because there were differences in lifespan variation between Scotland and England and Wales.

The second graph in column (b) is the main focus of the analysis: it shows the age contribution from differences in age-specific mortality rates to the lifespan variation gap between Scotland and England and Wales when a shared level of life expectancy was achieved (black spikes).

Comparing across column (a) and column (b) establishes if the differences in the age specific mortality rates were to Scotland's life expectancy advantage (above the red line in column a) or life expectancy disadvantage (below the red line in column a) while simultaneously being to Scotland's lifespan variation advantage (above the red line in column b) or to Scotland's lifespan variation disadvantage (below the red line in column b).

The title of each graph states the years that are being compared and the level of life expectancy or lifespan variation difference that the decomposition results refer to (previously detailed in table 17 and table 18). The earlier year always refers to when England and Wales achieved this life expectancy and the later year always refers to when Scotland achieved this life expectancy. The contributions from the first year of life (age zero) are truncated in the graph. This was because the contributions dominated the scale when carrying out the earliest decompositions (years when infant mortality was still relatively high). The full contributions from the first year of life are provided in appendix 7 for males and appendix 8 for females.

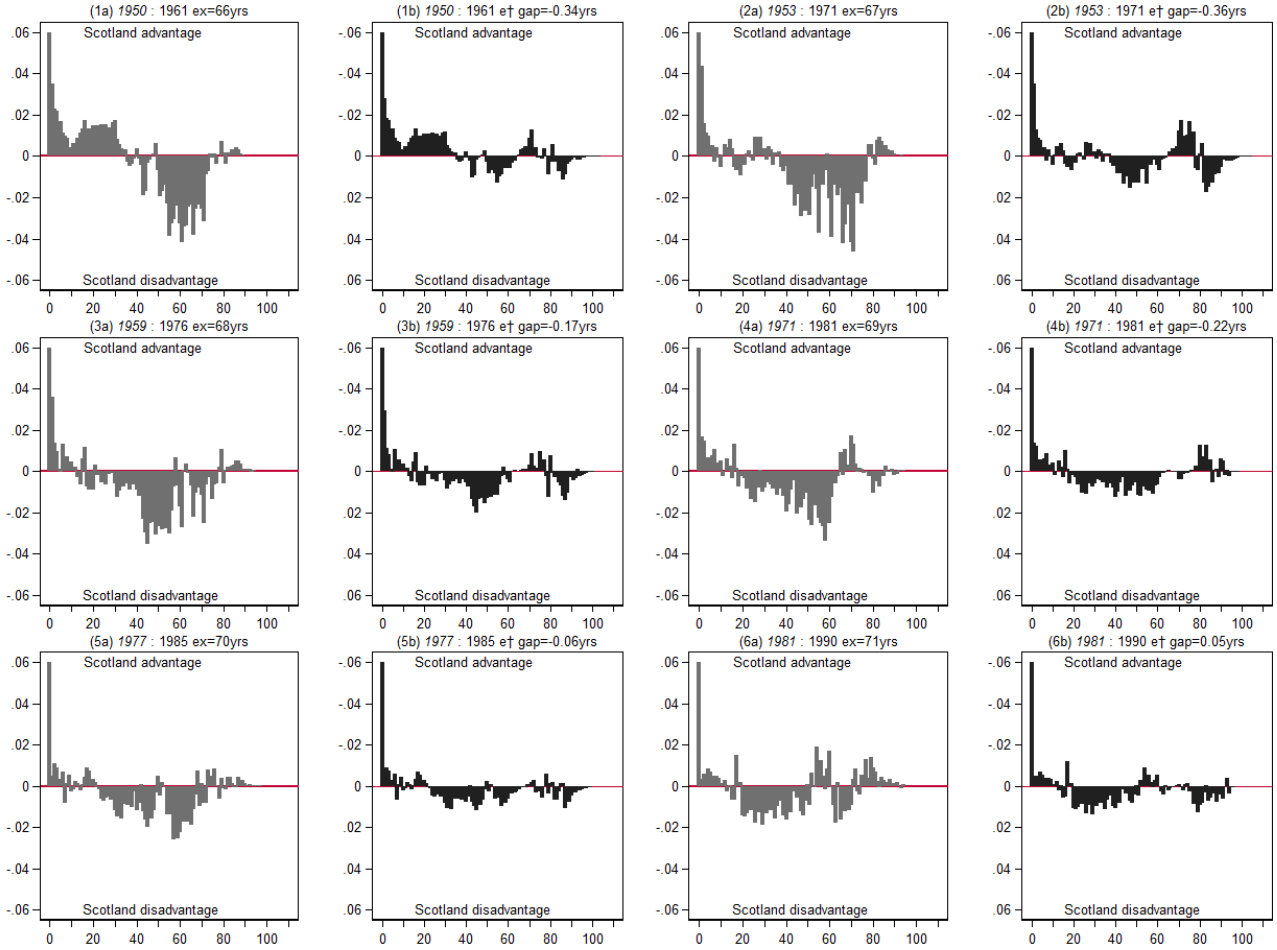


Figure 43 Age decomposition results at shared level of life expectancy, Males

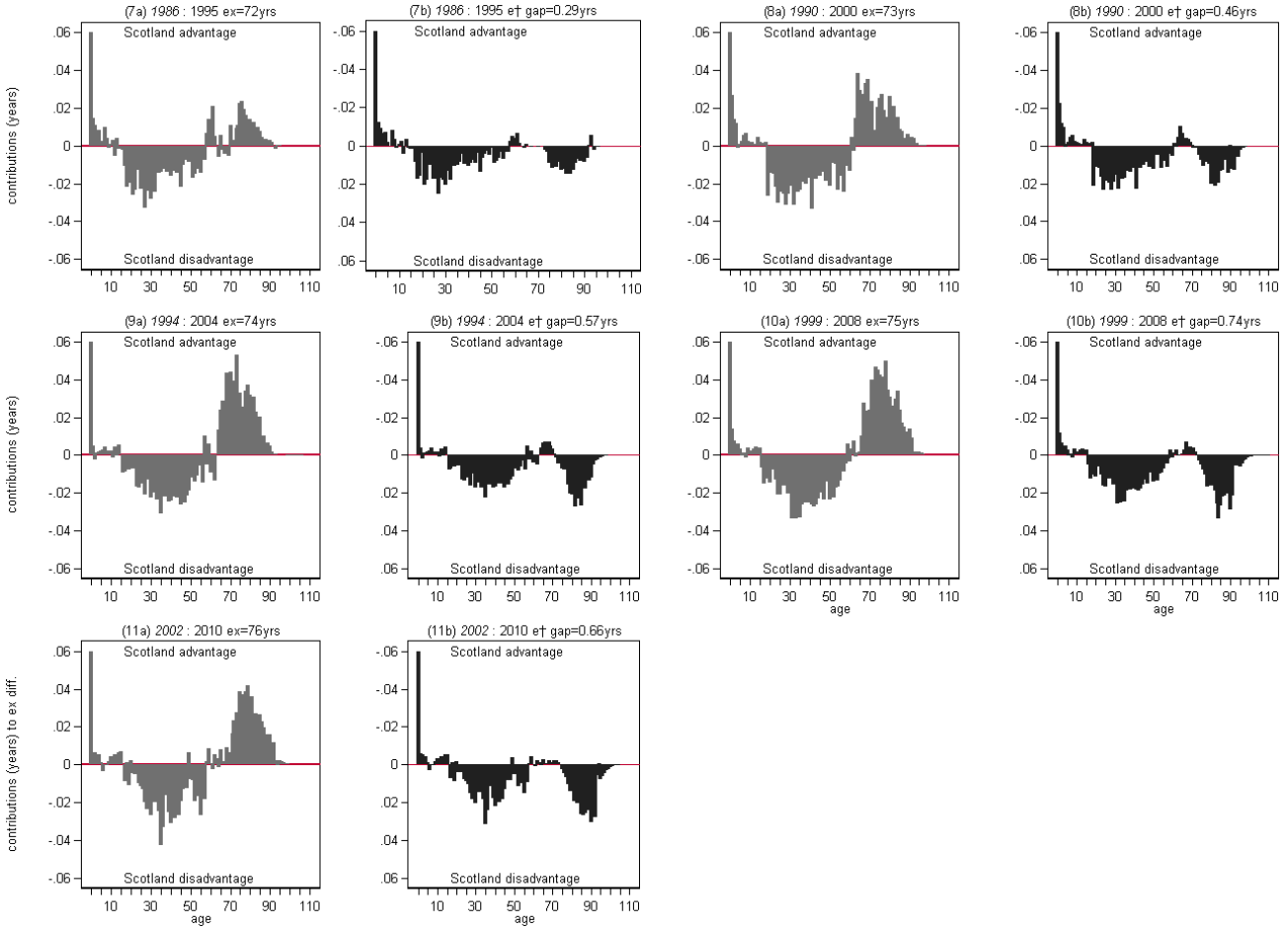


Figure 44 Age decomposition results at shared level of life expectancy, Males

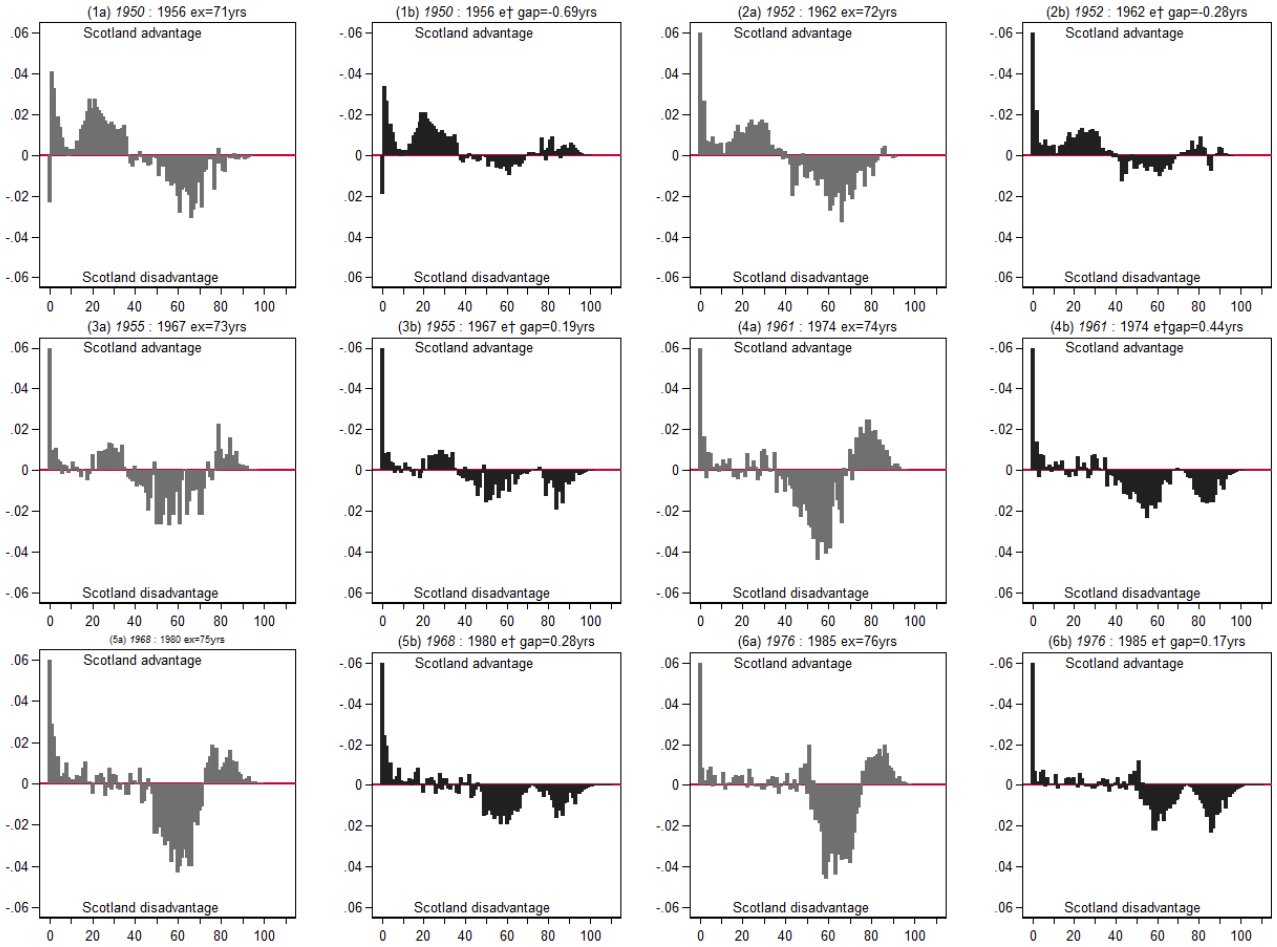


Figure 45 Age decomposition results at shared level of life expectancy, Females

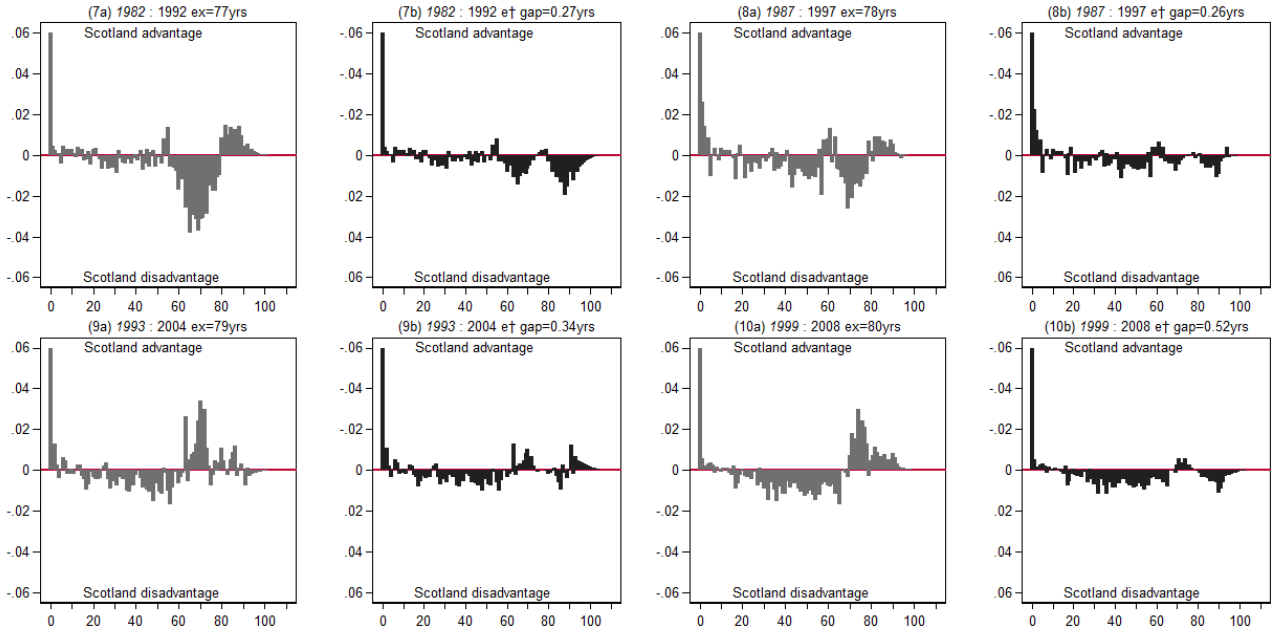


Figure 46 Age decomposition results at shared level of life expectancy, Females

6.4.3.2 Decomposition at shared levels of life expectancy

There are some distinct differences in the age distributions of death between the two countries revealing inequalities in age at death despite shared levels of life expectancy. Scotland reached the same level of life expectancy as England and Wales with lower mortality rates across some ages and higher mortality rates across others. These inequalities in the age patterns can broadly be described in terms of infant/early life mortality (age 0-15years old), premature adult mortality (16-64years old) and older age mortality (65+years old).

Scotland has always reached the same level of life expectancy later with lower or similar mortality rates across infant/early life ages (0-15 years old). The advantage this gave Scotland's life expectancy and lifespan variation is particularly evident for males at a shared life expectancy of 66 years old. Scotland achieved this in 1961 with a lifespan variation of 13.06 years. England and Wales achieved this in 1950 but with a lifespan variation of 13.40 years. Lower mortality rates across infant/early life ages therefore simultaneously improved average population health and reduced inequality because they compressed the age distribution. The same pattern is found for females.

Over time the gains made from lower infant/early life mortality rates in Scotland diminish. The much lower mortality rate during the first year of life in Scotland in 1971 compared to mortality during the first year of life in England and Wales in 1953 contributed 0.57 years of life expectancy advantage to Scotland (though this advantage is balanced out by life expectancy disadvantages at other ages) and -0.46 years of lifespan variation advantage. The slightly lower mortality rate during the first year of life in Scotland in 2000,

compared to England and Wales in 1994, contributed 0.09 years of life expectancy advantage to Scotland (though this advantage is balanced out by life expectancy disadvantages at other ages) and -0.08 years of lifespan variation advantage. This shows that mortality during the first year of life became more comparable over time between Scotland and England and Wales at a shared level of life expectancy and that Scotland's higher lifespan variation was not primarily due to higher mortality during the first year of life.

Scotland had lower old age mortality rates (≥65) at some levels of shared life expectancy. For example when comparing Scotland in 2008 with England and Wales in 1999. Lower mortality rates at older ages in Scotland do not simultaneously increase life expectancy and decrease lifespan variation. This is because they contribute to mortality expansion. The contributions from lower old age mortality are to Scotland's life expectancy advantage (column (a) grey spikes above the red line) but to its lifespan variation disadvantage (column (b) black spikes below the red line).

This contributed to Scotland achieving a similar life expectancy later but more unequally than England and Wales. For example in 2008 Scotland achieved a life expectancy of 75 years old with a lifespan variation of 11.55 years. England and Wales achieved this level of life expectancy in 1999 but with a lifespan variation of 10.81 years.

However, the main contribution to Scotland's greater lifespan variation at the same level of life expectancy was higher mortality rates across ages that are considered to be premature adult deaths (16-64years old).

Higher mortality rates for males age 16-64 years in Scotland, when it achieved the same level of life expectancy as England and Wales but later, have always been detrimental to its life expectancy and lifespan variation (contributions always below the red line). This age pattern becomes more predominant over time. It is perhaps most evident when the lifespan variation gap was greatest (0.74 years) at a shared life expectancy of 75 years old: in Scotland this was achieved in 2008 but had already been achieved by England and Wales in 1999.

A similar pattern was evident for females but to a much lesser extent. Higher mortality rates for females age 16-64 years in Scotland, when it achieved the same level of life expectancy as England and Wales but later, have mostly been below the red line but some ages have fluctuated across the red line. The spikes below the red line are shorter for females than the spikes for males indicating that there is a less of mortality difference.

6.5 Discussion

6.5.1 Summary of results

Scotland has achieved the same level of life expectancy as England and Wales around ten years later. Initially it did this with lower lifespan variation. It lost this advantage following the 1980s. Although higher lifespan variation was partly driven by expansion caused by falling mortality at older ages, higher working age mortality in Scotland explained much of the higher lifespan variation. This demonstrates that Scotland has failed to reach a high level of average health with relatively lower inequality because unfavourable working adult mortality rates have failed to offset lower old age mortality rates.

6.5.2 Strengths and limitations

The analysis in this chapter used the most robust, up-to-date data at the time of research available from the Human Mortality Database (Philipov, 2015, Jasilionis, 2015). The annual mortality rates available from the HMD were used to calculate a three year rolling average to account for any inherent random fluctuations in the annual mortality rates.

The significant time period covered in this chapter is a further strength. The time periods when lifespan variation was lower at shared levels of life expectancy and when lifespan variation changed to be higher at shared levels of life expectancy were all captured.

The comparator country, England and Wales, was chosen because it shares social and economic contexts as well as a national Government with Scotland and is its closest geographical neighbour. These countries also have had a longstanding, formal commitment to reducing mortality inequalities (The Scottish Government, 2008, Department of Health, 1999, Department of Health, 2013). The different mortality experience of these countries has been the focus of much research (Campbell et al., 2013, Carstairs and Morris, 1989b). The findings in this chapter provided further evidence demonstrating the timing of mortality change in Scotland, notably since the 1980s (Campbell et al., 2013, Norman et al., 2011, Leyland et al., 2007b). It is important that there is consensus across studies around the timing of change if we are to begin to evaluate the reasons for change.

6.5.3 Comparisons with existing studies

This is not the first piece of research to identify a country which experiences higher relative inequality in terms of lifespan variation or evaluate the important age characteristics of mortality decline. The USA is perhaps one of the most widely studied countries (Smits and Monden, 2009, Shkolnikov et al., 2011). Comparative studies are important if we are to understand different levels of inequality (lifespan variation) relative to neighbouring countries at the same level of average population health (life expectancy). The changing level of lifespan variation experienced in Scotland when it achieved the same level of life expectancy as England and Wales, but later, makes it a valuable case study. Case study countries such as Scotland and the USA can help us to understand why age patterns of mortality have not been declining uniformly across comparable countries.

Higher lifespan variation in Scotland, when arriving at a particular life expectancy later than England and Wales, was partly explained by lower old age mortality rates. This caused mortality expansion which adds uncertainty by delaying the age at death. These lower old age mortality rates suggest that Scotland may have experienced a temporal advantage across these ages but this temporal advantage did not translate into lower mortality rates across all ages. So when achieving the same life expectancy Scotland seems to have benefitted temporally in terms of old age mortality and younger age mortality but not premature adult mortality. The USA demonstrated a similar lack of mortality compression due to higher adult premature mortality over time. In Scotland the lack of mortality compression was coupled with higher levels of expansion associated with lower old age mortality rates compared to England and Wales

when both countries had roughly the same life expectancy (Shkolnikov et al., 2011). Unlike Scotland, the USA also had relatively worse young age mortality rates further adding to its higher level of lifespan variation (Shkolnikov et al., 2011). Like Scotland, the USA has become relatively more unequal than other countries in recent decades and when achieving the same life expectancy (Smits and Monden, 2009). So while not exactly the same, the USA and Scotland have similar problems with high premature mortality that may suggest underlying root causes.

Comparative research strongly suggests that the root causes of premature mortality in the USA relate to its poor performance in tackling the social determinants of health and high income inequality (Woolf and Aron, 2013). A recent extensive review of mortality in Scotland also suggests that the lasting effects of deindustrialisation, deprivation and higher inequality have resulted in it experiencing an excess of 5,000 deaths per year compared to England and Wales (Walsh et al., 2016). Social determinants of health and mortality may be amenable to social and economic policies and the magnitude of inequality in society alleviated (Bambra, 2011b, Coburn, 2000, McCartney et al., 2013).

Krieger et al. (1997) argue that society's commitment to minimising social and economic risk and ensuring health for all is a mediating pathway between risk factors (e.g. poverty, deindustrialisation and high income inequality) and eventual outcome (e.g. premature death). This is supported by the finding that societies with more egalitarian welfare systems, for redistributing income and thus reducing inequality, experience lower mortality rates and narrower mortality inequalities between socioeconomic groups (Vagerö and Lundberg,

1989, Popham et al., 2013). Therefore it is hypothesised that the degree of relative inequality in economically developed societies is an important determinant of mortality that is amenable to social, political and economic interventions (Wilkinson, 1997, Wilkinson, 2005)

Considering this, the mechanisms linking deprivation and premature adult age deaths in Scotland may be associated with its inability to reduce income inequality and lower the economic and health risks associated with poverty. Understanding how social and economic policies can reduce inequality and minimise the risk of premature death has implications for countries across Western Europe but is particularly important for Scotland as it contains some of the poorest communities experiencing some of the highest premature mortality rates in the UK (The poverty Site, 2016, McCartney et al., 2012b).

6.5.4 Research implications

The analysis of population level lifespan variation in Scotland has demonstrated that premature mortality is part of the explanation for its divergence. However it is not clear how lifespan variation inequalities are patterned by socioeconomic deprivation within Scotland.

It is valid to hypothesis that lifespan variation will be highest for the most deprived group: they are known to experience the highest risk of premature death. International studies have also demonstrated that most deprived experience a double burden of mortality inequality: the shortest expected length of life and the greatest amount of inequality in individual age at death (van Raalte et al., 2011). However reducing mortality rates at older ages can also lead to high lifespan variation. This is because reducing premature deaths

causes mortality compression, i.e. a narrowing of the variation in age at death, whilst reducing older age deaths causes mortality expansion, i.e. a widening of the variation in age at death. While both processes improve life expectancy reductions in lifespan variation can only be achieved through more mortality compression than expansion (Popham et al., 2013, Shkolnikov et al., 2011). In some Western European countries narrower inequalities in lifespan variation between socioeconomic groups were due to decreasing mortality rates amongst older ages in the least deprived group (van Raalte et al., 2011). The scale of lifespan variation inequalities between socioeconomic groups has not previously been measured for Scotland neither have changes to the scale of the socioeconomic gradient over time been quantified.

6.6 Conclusions

The results in this chapter add to the body of evidence seeking to describe lifespan variation inequalities between countries despite experiencing shared levels of average population health. This approach may better account for differences between populations in terms of epidemiological time. Some have argued this concept of time is more important to control for than calendar time: the magnitude of health inequalities between countries might be the result of them being at different stages of the epidemiological transition (Mackenbach et al., 1997, Smits and Monden, 2009) e.g. the smoking epidemic (Mackenbach et al., 2008) or cardiovascular revolution (Vallin and Meslé, 2004).

This chapter set out to identify which of three proposed hypotheses might best describe the lifespan variation gap between Scotland and England and Wales. The forerunner hypothesis proposed that a country leading the way in terms of

life expectancy would also lead the way in terms of lifespan variation: a country arriving at the same life expectancy later in time would do so with a very similar level of inequality as the forerunner. The diffusion hypothesis suggested that the country arriving at the same life expectancy later in time would be better positioned to reduce mortality rates across all ages, especially premature deaths, and would experience much lower inequality at a similar level of life expectancy. The laggard hypothesis suggested that a country arriving at a certain level of life expectancy later in time may do so with higher inequality because they have not fully benefited from their temporal advantage: they may have benefited from medical advancements for reducing deaths at ages that expand the age distribution of death but have failed to address the social determinants of health that are important for reducing deaths at ages which compress the distribution of death.

The current lifespan variation gap between Scotland and England and Wales would support a laggard hypothesis but previously would have supported a diffusion hypothesis. Scotland always seems to have benefited from its temporal advantage in terms of infant/childhood deaths and older age deaths: it always had lower mortality rates at these ages when arriving at the same level of life expectancy as England and Wales, albeit later on. However the lifespan variation gains available from reducing infant and childhood deaths diminish as deaths at these ages, thankfully, become rarer. Therefore it becomes increasingly important for mortality rates across working adult ages to be lower for the country arriving at the same life expectancy later in time. Scotland failed to achieve this, relative to England and Wales. The failure to tackle premature mortality has caused Scotland to experience higher lifespan variation at the

same level of life expectancy as its closest geographical neighbour, despite arriving at the same level of life expectancy ten years later. Without tackling premature working age mortality Scotland will not be able to simultaneously achieve a high level of population health with a low level of inequality.

7 Changes to the socioeconomic gradient for lifespan variation

7.1 Introduction

The previous three chapters have focused on measuring lifespan variation at the population level for Scotland within a cross national comparative context. Chapter 4 identified that Scotland has been unable to reduce lifespan variation at the same rate as other countries within Western Europe causing its relative ranking to worsen over time. This was particularly evident for males in Scotland who have experienced increasing lifespan variation since the 1980s.

This diverging lifespan variation trend is in contrast to Scotland's nearest neighbour England and Wales. Chapter 5 identified that the age patterns of mortality driving the increasing lifespan variation trend within Scotland were distinguishable from the age patterns within England and Wales. In particular were the higher premature mortality rates in recent years in Scotland: these prevented mortality compression. This meant Scotland was unable to offset the detrimental contributions, in terms of lifespan variation although not life expectancy, made from lower mortality rates across older ages. Lower mortality rates across older ages increase inequality by expanding the age distribution of death. England and Wales did not experience higher premature mortality rates later in time and successfully offset detrimental contributions from old age mortality in order to maintain a decreasing trend over time.

Chapter 6 analysed the lifespan variation gap independent of life expectancy and demonstrated that Scotland had a lifespan variation advantage, although it has tended to achieve the same level of life expectancy around ten years later than England and Wales. Following the 1980s Scotland lost its lifespan variation advantage and started to achieve the same level of life expectancy later in time *and* with higher inequality.

Although lower mortality rates across older ages contributed to Scotland's lifespan variation disadvantage, of concern are the contributions made from higher premature mortality rates when achieving a similar level of life expectancy later in time. This suggests that Scotland has been able to benefit from its temporal advantage in terms of old age mortality, capitalising on lessons already learnt, but not in term of premature mortality. This can be considered counter intuitive as the economic cost of reducing premature adult mortality – deaths associated with external causes and the social determinants of health – tend to be relatively lower than the economic costs of reducing old age mortality – deaths from chronic diseases.

It is hypothesised that socioeconomic deprivation in Scotland explains much of its premature mortality problem: premature deaths are more strongly associated with socioeconomic deprivation. Traditionally life expectancy or age standardised mortality have been used to measure the impact socioeconomic deprivation has on mortality. It is now well established that the most socioeconomically deprived groups experience the worst average mortality outcomes: they have the shortest average life expectancy and the highest age-standardised mortality rates. International studies measuring lifespan variation

have started to demonstrate that there is a further dimension of socioeconomic inequalities to consider: the most deprived socioeconomic groups also experience the greatest amount of inequality in age at death.

These international studies have: quantified the lifespan variation gap between the most and least deprived over time, examined how lifespan variation has changed over time for each deprivation group, and measured the lifespan variation gap between socioeconomic deprivation groups when a similar level of life expectancy has been achieved irrespective of chronological time. None of these analyses have been carried out for Scotland despite it being the country in Western Europe with the poorest level of average life expectancy and widening mortality inequalities between socioeconomic groups (McCartney et al., 2012b, Leyland et al., 2007b).

The findings in this chapter begin analysing lifespan variation stratified by socioeconomic deprivation within Scotland. It is the first analysis chapter in this thesis to utilise the population estimates obtained from National Records of Scotland, death count data and the Carstairs score data surrounding the four Census years: 1981, 1991, 2001 and 2011. It extends the analysis and interpretation of lifespan variation by producing 95% confidence intervals and calculating the slope index of inequality and relative index of inequality.

7.1.1 Chapter outline and research questions

The structure of this chapter is consistent with the previous analysis chapters and has four main sections. The first section summarises studies which have examined increasing socioeconomic inequalities in mortality outcomes in Scotland. It also highlights some international studies which have previously

examined socioeconomic inequalities in lifespan variation which have not previously been studied in Scotland. It is also unclear how the changing nature of the socioeconomic gradient for lifespan variation in Scotland is related to its lifespan variation ranking with Western Europe. Therefore the following chapter answers the following research questions:

7. Is there a socioeconomic gradient for lifespan variation in Scotland?
8. How has the socioeconomic gradient for lifespan variation in Scotland changed over time?
9. Are changes to the socioeconomic gradient for lifespan variation in Scotland related to Scotland's deteriorating lifespan variation ranking within Western Europe over time?

The second section briefly describes the data used and the analysis methods applied to answer these research questions. The third section details the results. The final section outlines how the results were used to inform the final analysis chapter.

7.2 Background

Systematic inequalities in mortality between the most and least deprived groups, whether measured by education, income or occupation, are found across all economically developed countries (Mackenbach et al., 2008, Health, 2008). These inequalities in death ultimately stem from inequalities in the social determinants of health (Mackenbach and Kunst, 1997, Walsh et al., 2016). The persistence of mortality inequalities over time, despite the introduction of

comprehensive welfare programmes, is considered to be “one of the greatest disappointments of public health” (Mackenbach, 2012).

Phelan et al. (2010) further argue that mortality inequalities in economically developed countries are a public health puzzle because the major causes and risk factors for premature death which historically accounted for much of the social patterning (e.g. infectious disease and malnutrition) have largely been eradicated. This evidence suggests that the nature of mortality inequalities has changed over time, and that changing risk factors reflect changes in the underlying causes.

7.2.1 Mortality inequalities in Scotland

Leyland et al. (2007b) somewhat echo this reaction in relation to Scotland, stating that the observed increase in mortality inequalities during the 1980s and 1990s were greatest for younger ages and for “newer” causes of death (Leyland et al., 2007b).For example increasing death rates for males aged 20-34 years old during the 1990s were driven by suicide, drugs, alcohol and assaults (Mcloone, 2003).

These increasing mortality rates were also strongly patterned by socioeconomic deprivation: Leyland et al. (2007a) observed that the all-cause mortality rate for males age 15-29years old from the least deprived group fell by 25% between 1991 and 2001 but increased by 4% for the most deprived. Consequently the mortality gradient in Scotland has steepened over time (Leyland et al., 2007a, Leyland et al., 2007b) despite there being a formal commitment made to reducing inequalities for the benefit of everyone (The Scottish Government, 2008).

7.2.2 Socioeconomic gradient in lifespan variation

It has been long established that the most deprived socioeconomic groups are expected to live for the shortest amount of time and have experienced the slowest rate of improvement in age-standardised morality rates over time (Leyland et al., 2007a). One proposed explanation for this is that relative deprivation may be a stronger determinant of population health in economically developed countries than absolute levels of material wealth. In the most unequal countries the health of the most deprived group disproportionately suffers but the health of the least deprived members is also relatively poor than their counterparts in more equal countries (Wilkinson, 2005, Wilkinson and Pickett, 2010, Wilkinson and Pickett, 2006). This implies that exposure to relative inequality is a population health problem, prompting Wilkinson (1997) to argue that national trends in mortality are likely to be a reflection of the psychosocial burden of relative deprivation. This is supported by international studies showing that the level of relative inequality within countries tends to be more strongly correlated with mortality than average levels of income or absolute measures of material wealth (Marmot and Bobak, 2000, Kaplan et al., 1996, Smith and Egger, 1996, Lynch et al., 2001). Therefore Scotland's poorer population level mortality trend, within the context of Western Europe, may be associated with relative inequality within the country.

This hypothesis was explored by McCartney et al. (2012b) when they measured the correlation between life expectancy and the index of economic freedom, paying particular attention to Scotland's trend within the context of Western Europe. They argued that the index of economic freedom was a valid measure of neo-liberal economic policies, which are reported to have had a detrimental

impact in terms of population level health and reducing relative inequality. This study concluded that the inverse correlation between the Index of economic freedom and life expectancy demonstrated that a greater change towards neoliberalism (e.g. a change towards increasing inequality) was related to smaller gains in life expectancy. Less common are studies seeking to understand the relationship between relative inequality and lifespan variation.

Higher lifespan variation equates to greater inequality in age at death. Therefore lifespan variation is considered a measure of inequality in itself, irrespective of its correlation with any other measures of social or economic position (Murray et al., 1999, Gakidou and King, 2002, Harper and Lynch, 2006). Smits and Monden (2009) supported this perspective when they argued that inequality in lifespan variation is the ultimate expression of inequality. Consequently, lifespan variation can be seen as a valid indicator of a society's ability to protect the most vulnerable members of society from the social and economic risks associated with premature death (van Raalte et al., 2011). It is therefore a particularly relevant measure to apply to Scotland, given that Scotland's observed premature mortality problem is embedded within the historical context of social and economic policies which may have contributed to poverty and relative inequality (Walsh et al., 2016, Dorling et al., 2007, McCartney et al., 2012b).

The few existing studies which have analysed the relationship between lifespan variation and a measure of socioeconomic position show a strong socioeconomic gradient: those occupying the lowest socioeconomic position experience the highest level of lifespan variation. This finding holds when measuring

socioeconomic position by education across ten European countries (van Raalte et al., 2011) and by occupational social groups in Finland (van Raalte et al., 2014). Alongside a traditional socioeconomic gradient for lifespan variation van Raalte et al. (2014) identified that manual occupational groups experienced higher lifespan variation than non-manual occupation groups at the same level of remaining life expectancy. This difference at the same level of remaining life expectancy increased over time and was driven by increasing differences in premature mortality between occupational groups: older age mortality was found to be extremely similar between occupation groups at the same level of life expectancy.

7.2.3 Research gaps

In summary, differences in lifespan variation between socioeconomic groups and at similar levels of life expectancy are important to measure as they may reflect inequalities in exposures to the social determinants of mortality between population sub-groups (Smits and Monden, 2009). Despite this, socioeconomic inequalities in lifespan variation over time or at shared levels of life expectancy have not previously been assessed for Scotland, nor have changes to the magnitude of the socioeconomic gradient for lifespan variation been formally quantified. It is also unclear if any changes to the socioeconomic gradient for lifespan variation correspond to Scotland's deteriorating ranking within Western Europe. It is these research gaps that this chapter will fill.

This chapter calculates lifespan variation for quintiles of socioeconomic deprivation, as measured by the Carstairs score, for 1981, 1991, 2001 and 2011. It then quantifies the magnitude of the socioeconomic gradient for lifespan

variation by estimating the slope index of inequality and relative index of inequality at each time point. The lifespan variation estimates, stratified by socioeconomic deprivation, are then compared with the lifespan variation estimates for Scotland and 16 other Western European countries (not stratified by socioeconomic deprivation) which were reported in chapter 4.

7.3 Data & methods

Data used in this chapter were Census population estimates and death counts by single-year of age matched with the Carstairs score of area level socioeconomic deprivation.

7.3.1 Population estimates and mortality data

Census population estimates were derived from the four most recent census years for which the Carstairs score was available: 1981, 1991, 2001, and 2011. These were multiplied by three to increase the size of the denominator population and to match with the number of years of death data. 1991 could only be multiplied by two as the geographical unit, which the population estimates are derived from, was subject to boundary changes in 1990 meaning there would have been inconsistencies.

Death counts used were from the years surrounding the four most recent census years: 1980-82, 1991-92, 2000-02 and 2010-2012. Using three years' worth of death data (two years in the case of 1991-92) increased the number of events by single year of age in order to smooth the age distribution of death which is vulnerable to random fluctuations (Lumme et al., 2015). Using three years' worth of population and death data is an established approach within empirical studies of mortality (Leyland et al., 2007b, Brown and Leyland, 2010).

Population estimates and death data were matched with the Carstairs score using postcode sector information. Postcode sectors were then aggregated into quintiles of deprivation each representing 20% of the Scottish population.

7.3.2 Predicting mortality rates

There Census population estimates were available up to an open ended age category of 85+ but a mortality rate by single year of age up to 110+ was required. Therefore a Poisson model, with a fractional polynomial function for age was used to estimate mortality rates beyond age 85+. The modelling process applied is outlined in chapter 3.

7.3.3 Lifetable probabilities

The mortality rates calculated from the population estimates and death counts, stratified by socioeconomic deprivation, were then converted into lifetables. A total of 40 deprivation specific lifetables were produced: one for each quintile of deprivation representing the mortality experience in 1981, 1991, 2001 and 2011 for males and females separately. A full description of how lifetable probabilities were produced is outlined in the data and methods chapter.

From these lifetable probabilities lifespan variation was calculated using e†. This is the same measure of lifespan variation that has been used throughout this thesis. The formula used to calculate e† and a critical evaluation of this measure are available in the data and methods chapter.

7.3.4 Slope index and relative index of inequality

In order to evaluate the scale of the deprivation gradient for lifespan variation the Slope Index of Inequality (SII) and the Relative Index of Inequality (RII) were calculated. A detailed discussion of the steps carried out to calculate the SII and

RII is given in chapter 3. In summary the SII communicates the absolute level of inequality in a health variable: in the case of this thesis, lifespan variation for social groups. The RII communicates the inequality in relative terms. These approaches allow trends to be assessed by taking into account the whole population and not just the extremes of deprivation (Munoz-Arroyo and Sutton, 2007, Schneider et al., 2005).

7.3.5 Monte Carlo simulations

95% confidence intervals were produced for the lifespan variation estimates and the slope and relative indices of inequality by carrying out 1,000 Monte-Carlo simulations. This produced 1,000 simulated mortality rates for each age (0-110+), sex, Census year and quintile of socioeconomic deprivation.

Calculating 95% confidence intervals allowed the estimate of lifespan variation, stratified by socioeconomic deprivation, to be statistically assessed and the statistical significance of any differences between quintiles of deprivation to be established.

In relation to the SII and RII, if the limits of the 95% confidence intervals cross zero then there is uncertainty as to whether there is a true health difference (Allik et al., 2016).

The steps required to carry out the Monte-Carlo simulation method are discussed in chapter 3. In summary it is important to calculate 95% confidence intervals as the mortality data used to calculate lifespan variation is inherently vulnerable to random fluctuations as a result the number of deaths that occur in a population can be interpreted as one possible series that could have occurred under the given set of circumstances (Curtin and Klein, 1995). It is also important as

'uncertainty in an indicator is an essential question when making comparisons of equality' (Lumme et al., 2015).

7.4 Results

Table 19 details the lifespan variation estimates for males, with 95% confidence intervals, for each deprivation quintile in Scotland in 1981, 1991, 2001 and 2011. Table 20 details the same for females. These results answer the first research question. It shows there is a strong deprivation gradient for lifespan variation evident for both males and females. This can be visualised in figure 46 (males) and figure 47(females).

Table 19 Lifespan variation estimates (with 95% CI) by socioeconomic deprivation, Males

Years	1981 (95% CI)	1991 (95% CI)	2001 (95% CI)	2011 (95% CI)
Least dep.				
Quintile 1	11.18(11.04-11.33)	10.76(10.59-10.93)	10.40(10.26-10.54)	10.44(10.31-10.58)
Quintile 2	11.63(11.49-11.77)	11.10(10.93-11.26)	11.11(10.98-11.25)	10.94(10.80-11.09)
Quintile 3	11.73(11.59-11.87)	11.35(11.19-11.52)	11.36(11.21-11.52)	11.21(11.08-11.33)
Quintile 4	11.83(11.70-11.95)	11.82(11.65-11.99)	11.82(11.69-11.95)	11.57(11.44-11.70)
Quintile 5	12.19(12.06-12.32)	12.28(12.12-12.43)	12.92(12.79-13.05)	12.25(12.14-12.39)
Most dep.				

Table 20 Lifespan variation estimates (with 95% CI) by socioeconomic deprivation, Females

Years	1981 (95% CI)	1991 (95% CI)	2001 (95% CI)	2011 (95% CI)
Least dep.				
Quintile 1	10.81(10.66-10.95)	10.53(10.39-10.69)	9.87(9.75-10.01)	9.87(9.76-9.98)
Quintile 2	11.08(10.95-11.21)	10.50(10.37-10.65)	10.13(10.01-10.27)	10.13(10.01-10.25)
Quintile 3	11.22(11.08-11.34)	10.87(10.71-11.01)	10.52(10.38-10.63)	10.24(10.11-10.34)
Quintile 4	11.32(11.18-11.44)	11.01(10.85-11.17)	10.61(10.49-10.74)	10.33(10.23-10.45)
Quintile 5	11.79(11.66-11.92)	11.72(11.56-11.85)	11.30(11.17-11.42)	10.91(10.82-11.03)
Most dep.				

7.4.1 Deprivation gradient

At each year the least deprived quintile experience the lowest level of lifespan variation and the most deprived quintile experience the highest. This means that the least deprived quintile experience the least amount of inequality in age at death while the most deprived quintile experience the most amount of inequality in age at death.

The analyses in chapters 4, 5 and 6 were concerned with the increasing lifespan variation trend at the population level for males in Scotland. The lifespan variation trend diverged for males during the 1980s. This change in trend was found to be statistically significant. The analysis in this chapter finds that the only group to experience a statistically significant increase in lifespan variation between 1991 and 2001 was quintile 5, the most deprived 20% of the Scottish population.

Over time the deprivation gradient for lifespan variation has steepened, particularly for males. This can be visualised for males in figure 47 and for females in figure 48. There is some improvement in the socioeconomic gradient in 2011 but it is still steeper than the socioeconomic gradient in 1981.

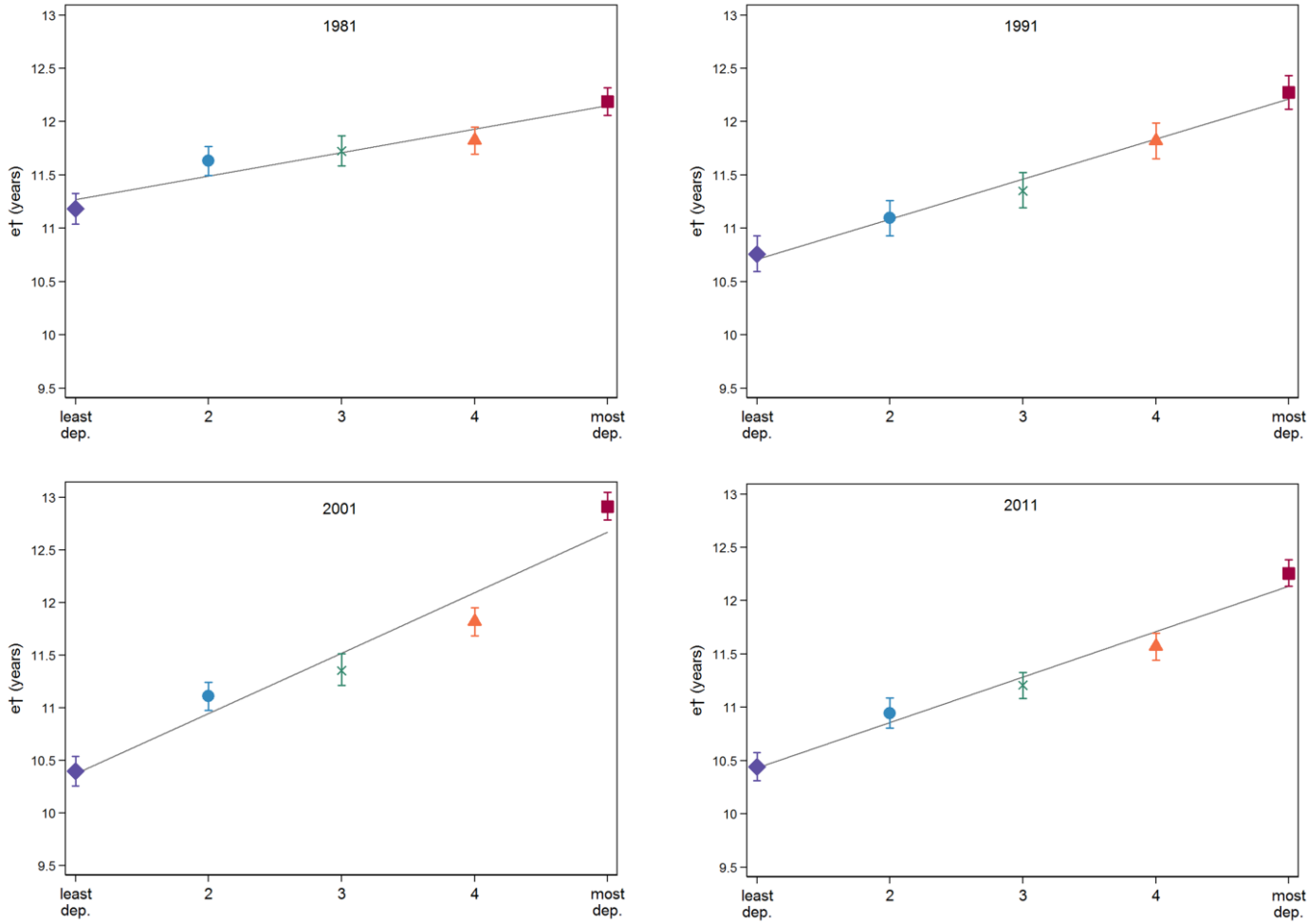


Figure 47 Socioeconomic gradient for lifespan variation, Males

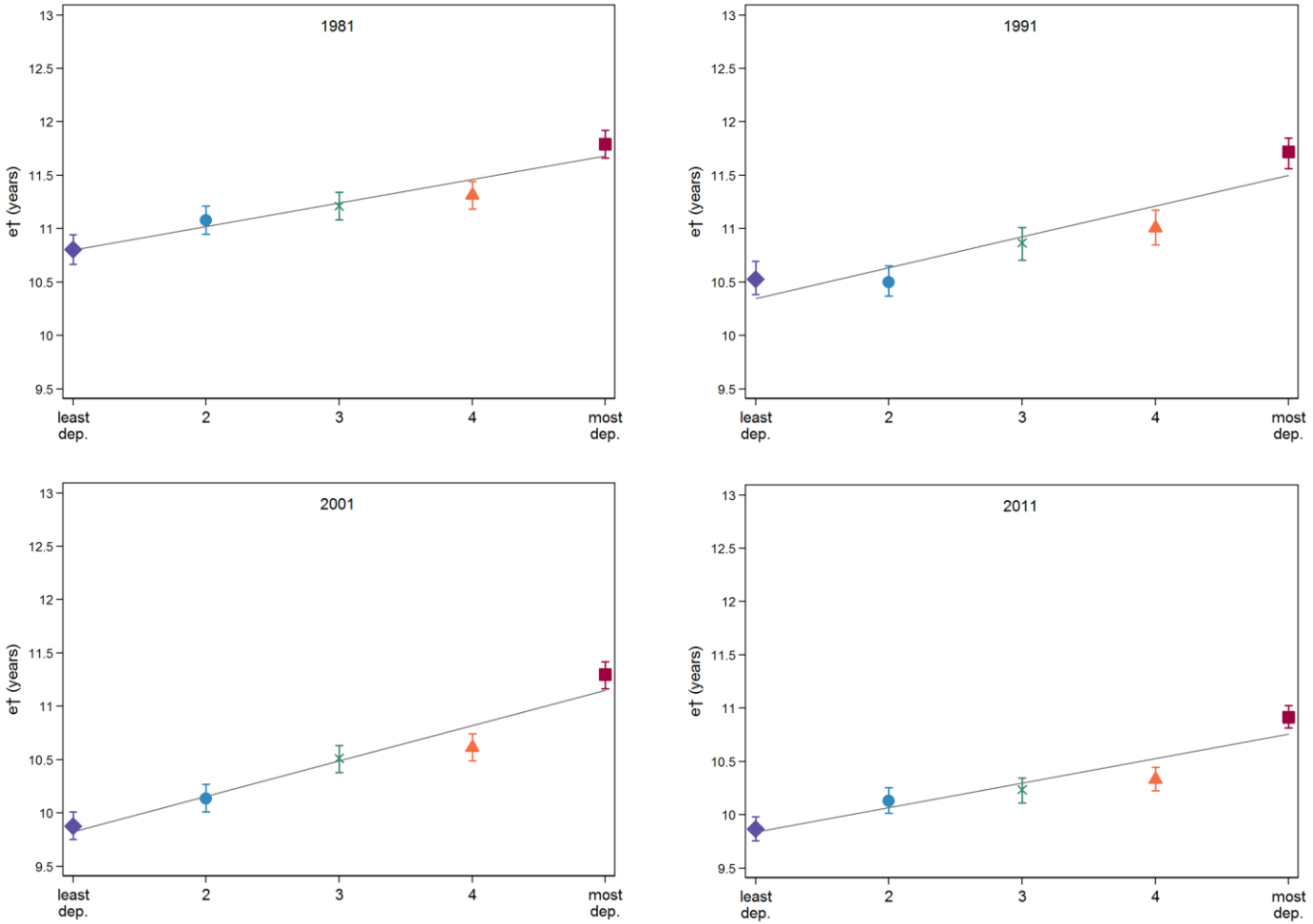


Figure 48 Socioeconomic gradient for lifespan variation, Females

7.4.2 Change in lifespan variation achieved over time

The steepening of the gradient over time for males is driven by the fact that the improvements in lifespan variation were unequally distributed across socioeconomic groups. The unequal change in lifespan variation experienced over time for each quintile can be visualised in figure 49 for males and figure 50 for females.

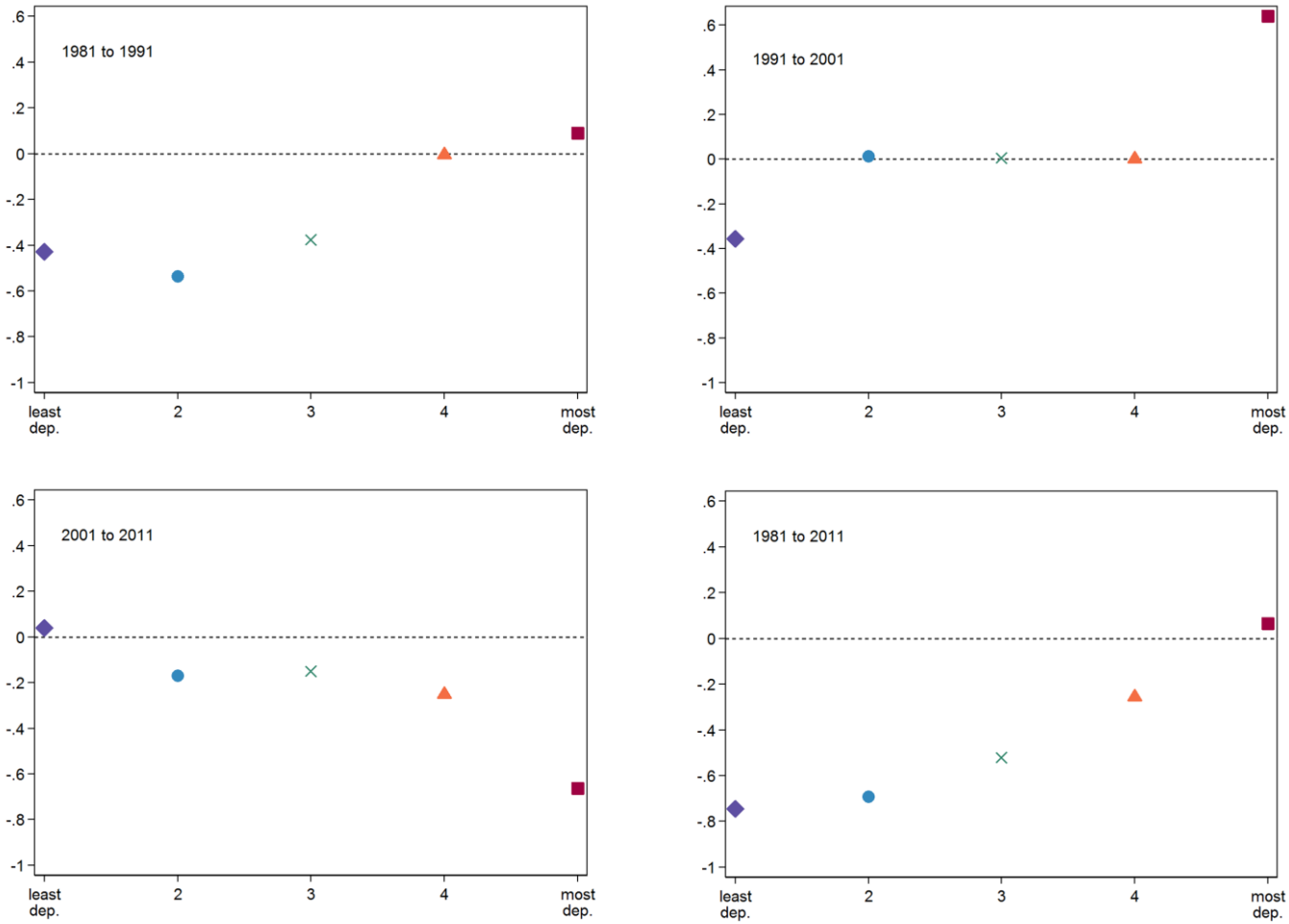


Figure 49 change in lifespan variation over time by deprivation quintile, Males

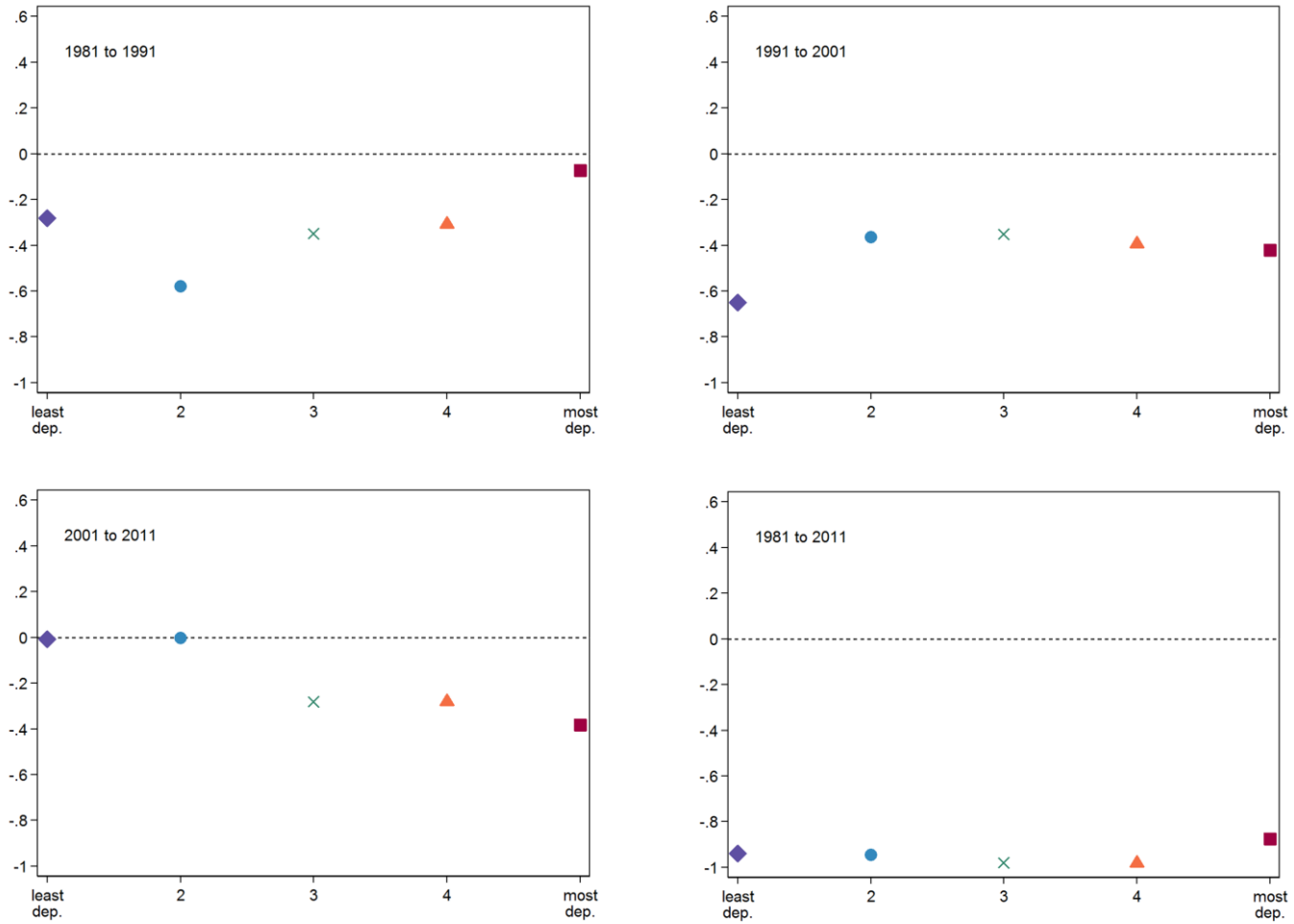


Figure 50 change in lifespan variation over time by deprivation quintile, Females

Between 1981 and 2011 males from the most deprived quintile experienced increases in lifespan variation but the least deprived experienced decreases. For females, the most deprived quintile experienced decreases in lifespan variation that were similar to the decreases achieved by the least deprived quintile but within the context of higher absolute levels of lifespan variation.

It is interesting to note that between 2001 and 2011 the most deprived experienced the greatest decrease and the least deprived experience an increase for males and no change for females. However this is still within the context of higher absolute levels of lifespan variation for the most deprived and lower absolute levels of lifespan variation for the least deprived. Therefore this did not negate the fact that between 1981 and 2011 males from the most deprived quintile still experienced an overall increase in lifespan variation while males from the least deprived experienced an overall decrease. For females the total change between 1981 and 2011 is not as strongly patterned by socioeconomic deprivation.

7.4.3 Slope index and relative index of inequality

The magnitude of the socioeconomic gradient for lifespan variation was quantified by calculating the slope index of inequality and the relative index of inequality. The results are reported in table 21 and answer the second research question: the magnitude of the socioeconomic gradient for lifespan variation did change over time. At first it steepened but there has been some improvement between 2001 and 2011.

Table 21 The socioeconomic gradient for lifespan variation (e†)

	Males		Females	
	SII (95% CI)	**RII (95% CI)**	**SII (95% CI)**	**RII (95% CI)**
1981	1.10 (0.86 to 1.36)	0.09 (0.07 to 0.12)	1.10 (0.86 to 1.33)	0.10 (0.08 to 0.12)
1991	1.88 (1.60 to 2.18)	0.16 (0.14 to 0.19)	1.45 (1.19 to 1.68)	0.13 (0.11 to 0.15)
2001	2.87 (2.64 to 3.11)	0.25 (0.23 to 0.27)	1.66 (1.41 to 1.88)	0.16 (0.13 to 0.18)
2011	2.13 (1.90 to 2.36)	0.19 (0.17 to 0.21)	1.15 (0.96 to 1.35)	0.11 (0.09 to 0.13)

For males the SII in 1981 is interpreted as 1.10 years difference in lifespan variation between the most and least deprived quintiles within Scotland. By 2011 this difference had increased to 2.13 years. The RII shows that lifespan variation was 9% higher for males in the most deprived quintile in 1981 compared to the least deprived quintile. By 2011 this difference had increased to 19%. The absolute difference was greatest in 2001 at 2.87 years which is the equivalent of a 25% difference in lifespan variation between the most and least deprived quintiles.

The results for females are similar but the socioeconomic gradient is not as steep and the changes over time are smaller. For females the SII in 1981 is interpreted as 1.10 years differences in lifespan variation between the most and least deprived quintiles within Scotland. By 2011 this difference had increased to 1.15 years. The RII shows that lifespan variation was 10% higher for females in the most deprived quintile in 1981 compared to the least deprived quintile. By

2011 this difference had increased to 11%. The absolute difference was greatest in 2001 at 1.66 years which is the equivalent of a 16% difference in lifespan variation between females in the most deprived quintile compared to females in the least deprived quintiles.

7.4.4 Scotland's deteriorating ranking within Western Europe

Much attention has been paid to Scotland's faltering mortality improvements relative to the rest of Western Europe (McCartney et al., 2012b, Leyland et al., 2007a). This prompted the analysis in Chapter 4 of this thesis, which focused on Scotland's faltering lifespan variation trend over time and identified that it currently had the highest level of lifespan variation within Western Europe. Figure 51 (males) and figure 52 (females) looks at these differences again compared with the lifespan variation differences stratified by socioeconomic deprivation within Scotland. This answers the fourth research question put forward at the beginning of this chapter: whether changes to the magnitude of the socioeconomic gradient for lifespan variation in Scotland are related to its deteriorating ranking within Western Europe.

Figure 51 and figure 52 situate Scotland (red dot) within the group of Western European countries that were analysed in chapter 4 and for deprivation quintiles for Scotland (blue dots). No statistical inference can be derived from the descriptive analysis comparing Scotland's deteriorating ranking with the lifespan variation estimates stratified by socioeconomic deprivation.

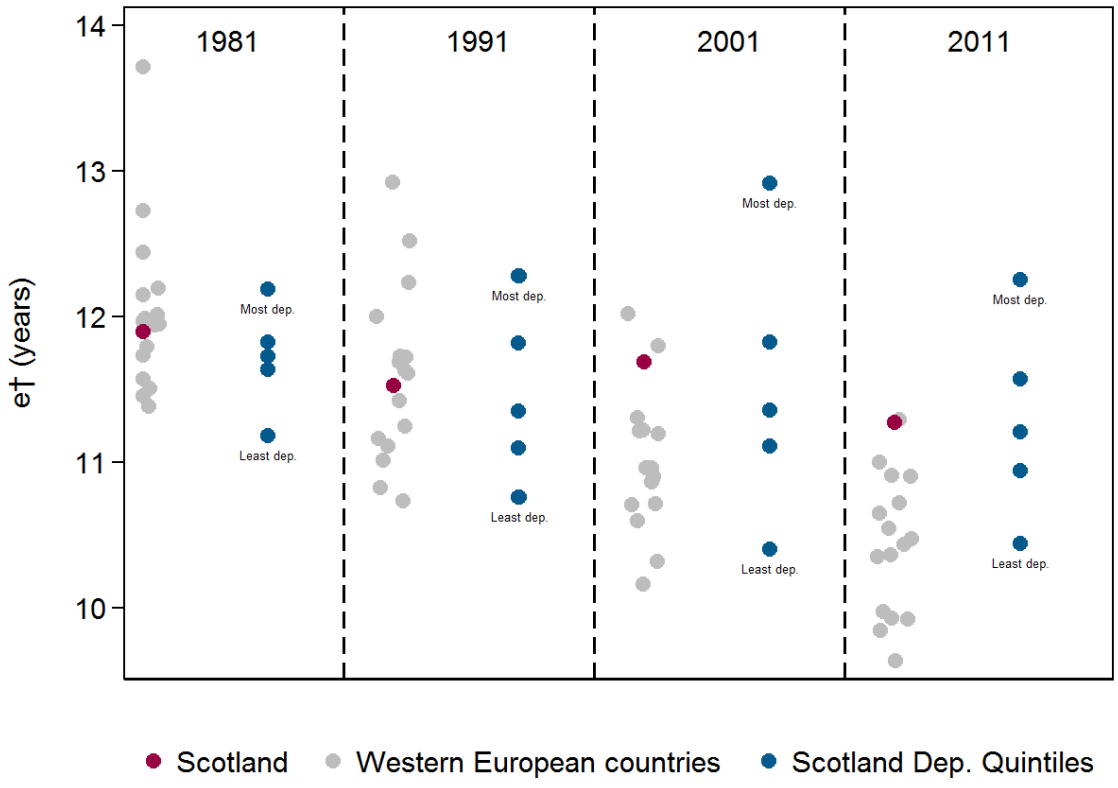


Figure 51 Comparison of lifespan variation estimates for Western Europe countries and lifespan variation estimates by socioeconomic deprivation, Males

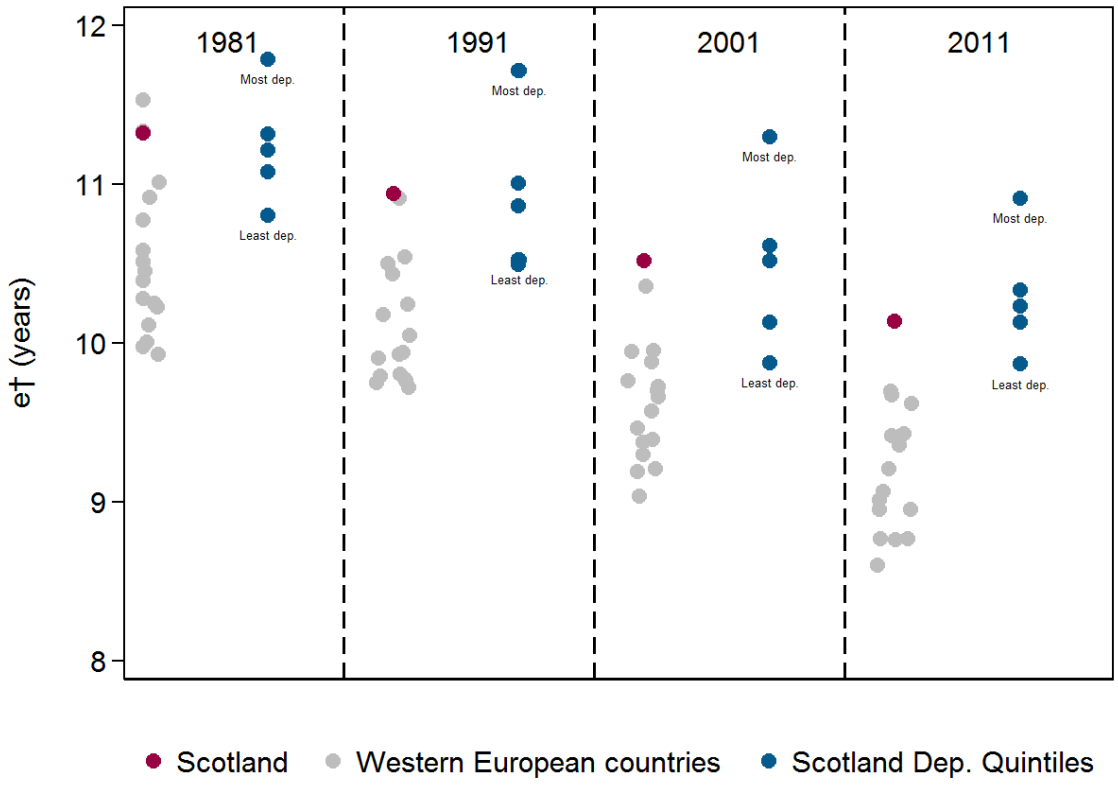


Figure 52 Comparison of lifespan variation estimates for Western Europe countries and lifespan variation estimates by socioeconomic deprivation, Females

For males and females in Scotland lifespan variation in 2011 was highest in Western Europe, with lifespan variation for males in France being very close. The blue dots, on the right hand of each panel, provide some explanation for why Scotland's lifespan variation ranking in Western Europe has deteriorated over time by showing the 5 quintiles of deprivation.

For males in 1981 only the most deprived quintile had lifespan variation higher than Scotland overall: the remainder had a lifespan variation that was close to or lower than Scotland overall. The quintiles of deprivation with lower lifespan variation were comparable to some other Western European countries. Countries with lower lifespan variation than Scotland fell in the lifespan variation range of 11.28 years to 11.80 years: three of quintiles of deprivation also fall within this

range while the least deprived quintile had a lifespan variation of 11.18 years which was lower than Sweden the top ranking country.

This pattern changes over time: for males in 2011 the two most deprived quintiles had lifespan variation higher than Scotland overall and the other three had lower lifespan variation. The quintiles of deprivation with lower lifespan variation were comparable to other Western European countries. Countries with lower lifespan variation than Scotland fell in the lifespan variation range of 9.64 years to 11.23 years: the least deprived quintile had a lifespan variation of 10.44 years but this was still higher than West Germany, Spain, Ireland, Norway, Italy, Switzerland, The Netherlands and Sweden. The most affluent 20% of the Scottish population experience a higher level of lifespan variation than the average level of lifespan variation experienced in these whole countries.

The findings are even more worrying for the quintiles which have higher lifespan variation than that of Scotland and therefore all other countries within Western Europe. Males from the most deprived quintile experiences 0.98 years *more* inequality in age at death than the average for males in Scotland. This is in contrast to males from the least deprived quintile which experienced 0.83 years *less* inequality in age at death than the average for males in Scotland.

Although the absolute level of lifespan variation for females in Scotland is lower than for males their position over time falls further behind the rest of Western Europe. This is partly due to the greater convergence over time, in terms of lifespan variation, for females across Western European countries: a convergence not so evident for males. It is also because of Scotland's poorer levels of lifespan variation.

For females in 1981 only the most deprived quintile had lifespan variation higher than Scotland overall: the remainder had a lifespan variation that was close to or lower than Scotland overall. Countries with lower lifespan variation than Scotland fell in the lifespan variation range of 9.90 years to 11.03 years: only the least deprived quintile had a lifespan variation that fell within this range.

This pattern worsened over time: for females in 2011 the three most deprived quintiles had lifespan variation higher than Scotland overall and the other two had lower lifespan variation. The quintiles of deprivation with lower lifespan variation were not comparable any other Western European country. Countries with lower lifespan variation than Scotland fell in the lifespan variation range of 8.60 years to 9.77 years: the least deprived quintile had a lifespan variation of 9.87 years.

The findings are even more worrying for the quintiles which have higher lifespan variation than that of Scotland and therefore all other countries within Western Europe: the most deprived quintile experiences 0.78 years more inequality in age at death than the average for Scotland. This is in contrast to the least deprived quintile which experienced 0.27 years less inequality in age at death than the average for Scotland.

7.5 Discussion

7.5.1 Summary of results

The analysis in this chapter identified that the socioeconomic gradient for lifespan variation in Scotland has steepened over time. Although there was some improvement in the gradient between 2001 and2011 the gradient was still steeper than it had been in 1981. In 1981, 1991, 2001 and 2011 the most

deprived group experienced the highest absolute level of inequality and the least deprived experienced the lowest absolute level, this is true for males and for females. For males the gradient became steeper over time because the least deprived group experienced an overall decrease in lifespan variation between 1981 and 2011 but the most deprived group experienced an overall increase. For females, all socioeconomic groups experienced an overall decrease and the change in socioeconomic gradient was not as dramatic.

Over time a greater proportion of the population is experiencing higher levels of lifespan variation than the Scottish population average. This is the case for males and females. In 1981, only the most deprived quintile experienced higher lifespan variation than the Scottish average. By 2001 the bottom two quintiles were experiencing higher lifespan variation than the Scottish population average. It is important to remember that each quintile represents 20% of the Scottish population: each is a substantial proportion than cannot be written off as outliers (Leyland et al., 2007a). It was therefore not surprising to find that the steepening socioeconomic gradient corresponded with Scotland's deteriorating lifespan variation ranking within Western Europe. By 2001 the least deprived 20% of the Scottish population experienced a level of total inequality higher than the average level experienced by entire populations of comparable Western European countries.

7.5.2 Strengths and limitations

The population data for Scotland used in this chapter were derived from Scotland's Census. This is one of the most reliable sources of population data and its main purpose is to provide an accurate population count (National

Records of Scotland, 2013b). A further strength of this chapter is the significant period of time covered (1981-2011) and the robust measure of socioeconomic deprivation used.

The Carstairs score has a strong theoretical framework and was constructed specifically in order to understand mortality inequalities (Carstairs and Morris, 1989a). It is the only measure of deprivation in Scotland that is available covering a thirty year time period (Brown et al., 2014). However it is not without limitations.

The score's ability to capture the changing nature of deprivation over time has been questioned (Tunstall et al., 2011). Particular attention has been paid towards the changing levels of car ownership and the meaning this has for relative deprivation being context specific (e.g. urban versus rural) (Norman, 2010).

The Carstairs score is an area level measure of deprivation rather than a measure derived from individual level variables of socioeconomic position. This means the results reported may be subject to the ecological fallacy: the extent of an association found at the area level may differ from extent of an association found at the individual level (Diez, 2002).

Despite these acknowledged short-comings, area level measures of deprivation are an invaluable tool for research. They also have pragmatic advantages for governments seeking to identify how resources should be distributed (Allik et al., 2016, The Scottish Government and National Statistics, 2012).

It was possible to statistically assess the true population value of lifespan variation stratified by socioeconomic deprivation by producing measures of uncertainty for lifespan variation.

Unfortunately no statistical inference can be derived from the descriptive analysis comparing Scotland's deteriorating ranking with the lifespan variation estimates stratified by socioeconomic deprivation. However each quintile which falls above the average lifespan variation for Scotland provide an indication of the extent of inequality in Scotland; this has implications in terms of inequality within a European perspective (Leyland et al., 2007a). This analysis is therefore important because it positions the widening inequality as a key factor which has contributed to Scotland's unenviable mortality experience in Europe.

7.5.3 Comparisons with existing studies

The social patterning of lifespan variation in Scotland is consistent with international studies that have analysed lifespan variation inequalities in different countries by education (van Raalte et al., 2011) and in Finland by occupation (van Raalte et al., 2014), the most deprived group experience the greatest amount of inequality in age at death. However the increases in lifespan variation over time for the most deprived in Scotland are somewhat starker than the stagnating trend identified for the most deprived group, in terms of occupation, in Finland between 1971 and 2010 (van Raalte et al., 2014).

The study by van Raalte et al. (2011) concluded that Finland had the largest socioeconomic inequalities in lifespan variation but Scotland was not included. Several authors have highlighted that international comparative research including Scotland as a separate entity from the rest of the UK is rare. Despite

the fact that it is a country that is comparable to many Western European countries in terms of wealth, economic development and epidemiological transitions (Leon et al., 2003, Popham and Boyle, 2010) it is not comparable in terms of life expectancy (McCartney et al., 2012b). Although the analysis in this chapter contributes to filling this research gap it is not possible to make direct comparisons with the existing international studies of lifespan variation.

Although van Raalte et al. (2011) and van Raalte et al. (2014) applied the same measure of lifespan variation (e†) as this thesis these two studies calculated lifespan variation as conditional upon survival to age 35 and 31, respectively. It was not possible to produce complete lifetables (beginning at age 0) because information on occupation and education, the measures being used to reflect socioeconomic position, are not meaningful for younger ages. In pragmatic terms it was also the youngest age for which data were available across all countries of interest (van Raalte et al., 2011). Similarly, Smits and Monden (2009) suggested that studies interested in the social distribution mechanisms of adult mortality should consider restricting analysis to ages 15+ because the causes of death driving mortality change over time differ across ages: infectious disease and effective medical intervention historically reduced infant and childhood deaths rapidly, while adult mortality is influenced by more complex mechanisms that change slowly (Vallin and Meslé, 2004). Smits and Monden (2009) further estimated that 80% of all deaths in 2000 (one of the most recent years for which they had data) occurred in ages 15+.

This thesis acknowledges these valid arguments but chose to measure e† from age 0 for the following three reasons. Firstly it was deemed appropriate to

measure e† from age 0 because life expectancy is traditionally reported and interpreted at age 0. Smits and Monden (2009) recognise this issue when they report lifespan inequality and life expectancy at all age groups for reasons of comparison. Secondly the decomposition methods, applied in chapter 5, 6 and 8, account for the contributions made from deaths at every age allowing the relative distribution of contributions to be assessed. Finally it was possible in the context of this thesis to measure lifespan variation from age 0 because the measure of deprivation used was applicable to the whole population unlike the measures used by van Raalte et al. (2014) and van Raalte et al. (2011).

7.5.4 Research implications

The evidence presented in this chapter, showing the strong patterning of lifespan variation by socioeconomic deprivation, means that the age distribution of death must differ between socioeconomic groups. The impact age specific change in mortality has had in terms of lifespan variation was quantified by van Raalte et al. (2014) and demonstrated that the contrasting lifespan variation trends between 1971 and 2010 across occupation groups were largely due to higher premature mortality rates associated with external causes of death. This type of formal analysis has not yet been carried out for Scotland. However it cannot simply be assumed that the same premature mortality problem evidenced in Finland, will hold true for Scotland: reductions in older age mortality can also prevent reductions in lifespan variation (van Raalte et al., 2011).

However evidence from existing studies of Scotland strongly suggest that higher premature mortality is a problem for Scotland (Leyland et al., 2007b, Mitchell et

al., 2005, McCartney et al., 2016, Schofield et al., 2016). For example increasing mortality rates for ages 15-29 years old were evident at the population level between 1981-1991 and 1991- 2001 (Leyland et al., 2007a). This deteriorating mortality pattern was largely caused by deaths from suicide, alcohol harm, substance abuse and violence (Norman et al., 2011, Leyland et al., 2007b, Leyland, 2004, Schofield et al., 2016).

Leyland et al. (2007a) demonstrate the extreme divergence in the social patterning of external causes when discussing deaths from chronic liver disease, which represent alcohol harm. Even though chronic liver disease was considered a rare cause of death in 1981, the ratio of deaths for males from the least deprived group to males from the most deprived group was still 1:4.8. By 2001 this ratio had increased to 1:17.5. A similar result was evidenced for females.

The analysis in chapters 5 and 6 of this thesis identified that premature mortality, from all-causes of death, were detrimental in terms of Scotland's population level lifespan variation trend. Chapter 7 demonstrated that lifespan variation in Scotland is strongly patterned by socioeconomic deprivation. Existing studies of mortality in Scotland have demonstrated increasing premature mortality rates from external causes of death which the international literature argues contribute to lifespan variation inequalities between socioeconomic groups. Considering all of this evidence it is valid to hypothesis that premature deaths from external causes will have also contributed to lifespan variation inequalities in Scotland.

This is important to establish as different ages and causes of death reflect different aetiological pathways and contrasting root causes . It is also a research

question which will have relevance beyond this thesis: evidence seeking to understand the determinants of widening mortality inequalities (as being measured by lifespan variation) in one country is likely to be of interest to other populations (Schofield et al., 2016, Mackenbach et al., 1997).

7.6 Conclusion

This chapter demonstrated that lifespan variation is strongly patterned by socioeconomic deprivation in Scotland. The socioeconomic gradient for lifespan variation has widened over time but showed signs of narrowing in 2011. This was the case when measuring the socioeconomic gradient in absolute and relative terms. Opposing trends between the most and least deprived quintiles in Scotland contributed to the widening lifespan variation gradient: for the least deprived quintile there was a decrease in lifespan variation between 1981 and 2011, for the most deprived quintile there was an increase. Over time a greater proportion of the Scottish population have experienced a lifespan variation level higher than the Scottish average.

Considering all of this evidence it is perhaps not surprising that Scotland's lifespan variation ranking has deteriorated: earlier analysis in this thesis identified that it now experiences the highest level of inequality in age at death within Western Europe. Scotland may not be able to improve its position if it does not ensure reductions in lifespan variation are experienced by all socioeconomic groups. Identifying if any age and cause specific patterns of mortality change are distinguishable between socioeconomic groups could help to understand how Scotland can achieve this.

8 Age and cause specific decomposition of lifespan variation inequalities

8.1 Introduction

The previous chapter of this thesis began the process of analysing lifespan variation stratified by socioeconomic deprivation within Scotland. It demonstrated the strong socioeconomic gradient for lifespan variation in Scotland: a gradient that widened over time in absolute and relative terms. Although there was some improvement in the gradient between 2001 and 2011, the most recent time point, this was still steeper than it had been in 1981.

Initially, only the most deprived quintile in Scotland experienced a higher level of inequality than the population average. Over time more quintiles of deprivation experienced a higher level of lifespan variation than the Scottish average. Each quintile represents 20% of the Scottish population, a significant proportion. It was somewhat surprising to find that the lifespan variation experienced by the most affluent quintile in Scotland has fallen further behind the average lifespan variation experienced by whole countries in Western European.

Evidence presented in chapter 5 of this thesis demonstrated that the age pattern of mortality driving the population level lifespan variation trend in Scotland was distinguishable from the age pattern driving the trend in England and Wales: Scotland experienced higher mortality rates across working adult ages. The detrimental contributions from these higher premature mortality rates increased

during the 1980s. This was around the same time that the population level lifespan variation trend in Scotland diverged towards increasing lifespan variation (as formally identified in chapter 4 of this thesis).

Chapter 6 of this thesis demonstrated that higher premature mortality rates in Scotland also explained the increasing lifespan variation gap between Scotland and England and Wales when achieving similar levels of life expectancy. This was despite the fact that Scotland was achieving a level of life expectancy comparable to England and Wales approximately ten years later in time.

Considering this, alongside the wider literature demonstrating persisting premature death rates in Scotland, it is valid to hypothesise that widening socioeconomic inequalities in lifespan variation could largely be due to the higher risk of premature death that is disproportionately experienced by the most deprived socioeconomic groups in society. However this has not previously been the focus of any formal analysis for Scotland. Nor have cause specific contributions to increasing socioeconomic inequalities in lifespan variation been quantified. It is these research gaps that this chapter will fill.

The analysis in this chapter extends our understanding of the social patterning of lifespan variation by visually inspecting differences in the age distributions of death and decomposing lifespan variation inequalities into age and cause specific contributions. This analysis was carried out in order to answer the final three research questions of this thesis:

10. Which ages and causes of death contributed to changes in lifespan variation, over time, for different socioeconomic groups in Scotland?

11. Which ages and causes of death contributed to the lifespan variation gap between the most and least deprived socioeconomic groups in Scotland?

12. Which ages and causes of death contributed to the lifespan variation gap between the most and least deprived socioeconomic groups, when life expectancy was similar, in Scotland?

This chapter continues to utilise the same population estimates obtained from National Records of Scotland, death count data and the Carstairs score data surrounding the four Census years: 1981, 1991, 2001 and 2011. The aggregate lifespan variation estimates reported in this chapter are therefore the same as the lifespan variation estimates that were reported in chapter 7.

This is the only chapter in this thesis to utilise International Classification of Diseases (ICD) data in order to assign each death to one of five, mutually exclusive, cause specific categories: external causes, cancers, circulatory diseases, respiratory diseases, and other causes. Like all of the previous chapters, this chapter is made up of the following four sections: background, data and methods, results, discussion and conclusion.

8.2 Background

Studies comparing lifespan variation inequalities between entire countries have identified how differences in the ages of death contribute to the cross-sectional lifespan variation gap (Shkolnikov et al., 2011, Vaupel et al., 2011) and the gap at shared levels of life expectancy (Seaman et al., 2016b, van Raalte et al., 2014). The literature has also progressed to quantify the ages and causes of death driving: socioeconomic trends in lifespan variation, the lifespan variation gap between socioeconomic groups at the same time point, and the lifespan

variation gap between socioeconomic groups when life expectancy was similar (van Raalte et al., 2014, van Raalte et al., 2011).

8.2.1 Socioeconomic trends

In most Western European countries the least deprived socioeconomic group has tended to experience the most favourable trends in lifespan variation and the most deprived the most unfavourable (van Raalte et al., 2011, van Raalte et al., 2014). Chapter 7 of this thesis demonstrated that this finding was replicated when studying socioeconomic trends in lifespan variation in Scotland. Countries with high population levels of lifespan variation have also been found to have steeper socioeconomic gradients for lifespan variation (van Raalte et al., 2011).

International studies have demonstrated that socioeconomic differences in lifespan variation trends are due to inequalities in the age and cause specific patterns of mortality decline (Shkolnikov et al., 2011, van Raalte et al., 2014). Although all socioeconomic groups have shifted the age distribution towards older ages, the most deprived are found to have experienced a smaller shift across working adult ages (van Raalte et al., 2014). This causes a lack of mortality compression amongst the most deprived socioeconomic group(s) which can result in a stagnating, or even increasing, lifespan variation. This is in contrast with the experience of the least deprived group :mortality compression over time, resulting in a decreasing lifespan variation.

8.2.2 Cross-sectional gap

The lifespan variation gap between socioeconomic groups at the same point in time was assessed for ten European countries by van Raalte et al. (2011). This study sought to understand if the same ages and causes of death causing the

most deprived socioeconomic group (as measured by education) to experience lower life expectancy also caused them to experience higher lifespan variation. The main reason for the lifespan variation gap between socioeconomic groups was different mortality rates across working adult ages: a persisting left hand tail across the age distribution was evident for the most deprived group.

However in a few countries there was a smaller gap between the socioeconomic groups because the least deprived were experiencing mortality expansion which also adds uncertainty to age at death. While measuring the cross-sectional lifespan variation gap is important, it is also important to capture and interpret lifespan variation independent of the level of life expectancy achieved (Smits and Monden, 2009).

8.2.3 Gap at comparable level of life expectancy

Chapter 6 of this thesis critically evaluated the hypothesis proposed by Smits and Monden (2009): that countries paving the way in terms of life expectancy may do so with higher inequality than those following behind them. Countries lagging behind, in terms of life expectancy, may experience a temporal advantage in terms of reducing inequality because they are positioned to capitalise on lessons already learnt for reducing premature mortality across ages that simultaneously increases life expectancy and decreases inequality.

van Raalte et al. (2014) applies a similar hypothesis, but in reference to lifespan variation inequalities between socioeconomic groups as opposed to countries, stating that the knowledge surrounding health improvement and motivation for health behaviour change would be diffused over time from the least deprived quintile to the most deprived quintile. Under this argument we would expect

lifespan variation to be higher amongst the most deprived when looking at chronological time but at a shared level of life expectancy lifespan would be similar or lower. When studying Finland, van Raalte et al. (2014) found that this was not the case. Lifespan variation was also higher amongst the most deprived socioeconomic group, as measured by occupation, when achieving the same level of life expectancy but later in time as the least deprived. This was despite the fact that the most deprived appeared to experience a temporal advantage (lower mortality rates) for circulatory diseases.

The lifespan variation disadvantage, at comparable level of life expectancy, was largely explained by the most deprived socioeconomic group experiencing higher premature mortality rates from causes of death that can be categorised as external. It could be argued that deaths from external causes are more likely to be associated with the social determinants of health and be amenable to redistributive policies (Bambra, 2011a). However it is important to recognise that stagnating lifespan variation can also be the result of saving lives at older ages: deaths that are likely to be caused by chronic medical conditions and co-morbidities which are more likely to be amenable to medical and health care advances. However deaths at older ages may also be the result of lagged effects of being exposed to socioeconomic determinants of health at earlier stages of the life course (Nau and Firebaugh, 2012).

Studies measuring socioeconomic inequalities in lifespan variation and seeking to identify the age and cause specific drivers are valuable for informing the direction of future empirical research, interpreting the theoretical explanations that exist for health inequalities, anticipating how socioeconomic inequalities

might change, and providing a basis for critically evaluating appropriate social policy responses for addressing socioeconomic inequalities. van Raalte et al. (2014) put forward three scenarios to illustrate these arguments: the fluctuating scenario, the divergence scenario, and the stagnating scenario.

8.2.4 Critical evaluation of lifespan variation scenarios

van Raalte et al. (2014) was particularly concerned with understanding why life expectancy tends to be strongly predictive of lifespan variation for all socioeconomic groups when analysed at one single point in time but that the ability for life expectancy to predict lifespan variation for entire countries is much more ambiguous. In the context of this thesis, the following critical evaluation is used to demonstrate why empirical studies of lifespan variation are valuable for assessing the merit of existing theories of health inequalities (Mackenbach, 2012). van Raalte et al. (2014) effectively summarises how empirical lifespan variation findings could provide evidence for: health behaviour explanations, social polarisation and psychosocial mechanism theories, selection effects, or fundamental causes (table 22). Identifying which scenario is applicable to lifespan variation inequalities in Scotland may help to understand the causes of mortality inequalities (McCartney et al., 2012a).

8.2.5 Fluctuating

The first scenario hypothesised that the lifespan variation gap between the most and least deprived groups over time could fluctuate from periods of widening to periods of narrowing. Under this scenario the lifespan variation gap between the most and least deprived could be very small at shared levels of life expectancy as the most deprived group simply lag behind the least deprived. This scenario

could be interpreted as support for either a health behaviours explanation or for a fundamental causes' framework.

It was suggested that that the least deprived socioeconomic group could pave the way for adopting health behaviours that reduce premature mortality. Over time the knowledge and motivation for health behaviours would be diffused to lower socioeconomic groups: causing the lifespan variation gap to widen during periods of rapid health improvement as the least deprived adopt new health behaviours before returning to previous levels or narrowing as new health behaviours diffuse to the most deprived.

This hypothesis was explored by looking at the role smoking played in the diverging trends between socioeconomic groups, as measured by occupation (van Raalte et al., 2015). It strongly suggested that smoking, one of the most detrimental of all health behaviours, did not contribute to the lifespan variation gap between the most and least deprived socioeconomic groups. Yet Kelly and Preston (2016) demonstrated that smoking was the main driver of lower life expectancy (conditional upon survival to age 50) in Scotland.

The inability for smoking to account for lifespan variation inequalities in Finland while accounting for life expectancy in Scotland was somewhat surprising considering that smoking is strongly patterned by socioeconomic deprivation. However it is important to consider the aetiological pathways of different causes of deaths. There is a substantial time lag between the exposure (smoking) and the outcome (death). This means that smoking has a minimal impact on deaths at younger ages but is the predominant behavioural factor for deaths at older ages (van Raalte et al., 2015). Unfavourable levels of lifespan variation tend to

occur in populations where there is a lack of compression because of unfavourable premature mortality rates: deaths that are less likely to be caused by smoking. However a change in mortality rate at any age will have implications for life expectancy. Similar findings were also evidenced by Shkolnikov et al. (2011) when studying differences between the USA and England and Wales as a whole: external causes of death accounted for inequalities in lifespan variation but not in life expectancy.

The notion that health behaviours are the mechanism driving population health has been heavily criticised. This hypothesis fails to recognise that health behaviours are indicators of health that need to be interpreted with the relevant context, relies on the notion that individuals act as autonomous and independent decision makers in relation to their health, and inadequately accounts for the fact that health behaviours have changed over time but the mortality gradient has remained (McCartney et al., 2013, Bartley, 2004, Townsend and Davidson, 1992, Dowler and Spencer, 2007). These theoretical limitations are somewhat addressed by interpreting a fluctuating scenario as support for a fundamental causes explanation for health inequalities.

A fundamental causes theory hypothesises that a socioeconomic gradient for health is always present to some degree regardless of the changes to the disease profile of the population(Link and Phelan, 1995, Phelan et al., 2010). This is not because of inequalities in the distribution of health behaviours or individually based risk factors but because socioeconomic status embodies the types of resources (so called fundamental causes) that impact health: money, power, prestige, and knowledge. These fundamental causes are always relevant for

protecting health regardless of changes to the disease profile and the distribution of these resources is not altered by interventions. As the disease profile changes or a new intervention is implemented the socioeconomic gradient may fluctuate from narrowing to widening but the highest socioeconomic will always maintain an advantage.

8.2.6 Diverging

The second scenario suggested that the least deprived may experience mortality compression but the most deprived would not to the same extent. This would cause the lifespan variation gap between socioeconomic groups to widen over time and to widen at shared levels of life expectancy.

van Raalte et al. (2014) hypothesised that a divergence scenario could be the consequence of increasing income inequalities, caused by the decline of heavy industries and the rise in precarious employment. The disproportionate impact these societal level changes had on the most deprived socioeconomic groups is well documented with many arguing that these processes have resulted in polarised societies (Bambra, 2010, Dorling, 2009). Societal level changes may have feasibly impacted the age and cause specific patterns of mortality experienced by socioeconomic groups if the mechanisms that help to mitigate social and economic risks were restricted for the most deprived group only. This could have had damaging effects on the psychosocial responses that are hypothesised to be important for avoiding premature death causing a lack of compression (Marmot and Wilkinson, 2001, Wilkinson and Pickett, 2006).

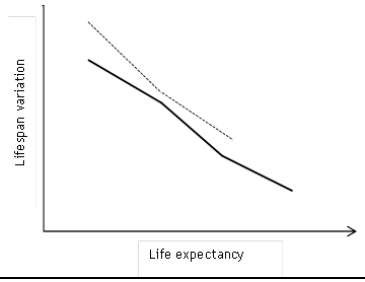
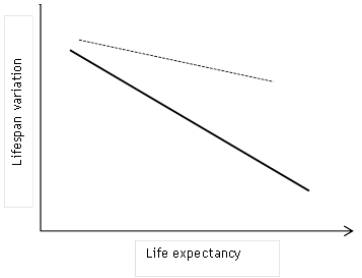
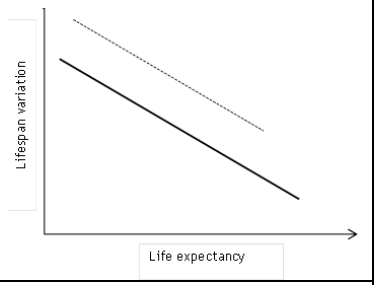
Another interpretation of a divergence scenario is that it would support a health selection effects explanation for increasing mortality inequalities: either in

terms of ill health causing downward mobility or in terms of a meritocracy increasing upward mobility for individuals with favourable health characteristics. If either of these mechanisms were active then socioeconomic groups would each be expected to become more homogenous in terms of health, leading to widening inequalities. This would be reflected in growing differences in causes of death when comparing socioeconomic groups, with external causes of deaths becoming concentrated within the most deprived groups.

8.2.7 Stalling

The third scenario hypothesised that the absolute lifespan variation gap between socioeconomic groups could remain constant over time. van Raalte et al. (2014) suggest that this would support a fundamental causes framework for health inequalities: the least deprived socioeconomic group are always able to maintain a health advantage regardless of the types of mortality risk that the population is exposed to over time because they are able to exploit their material, psychosocial and knowledge resources (Link and Phelan, 1995, Phelan et al., 2010). Causes of death would not be increasingly patterned by socioeconomic deprivation. Instead, the least deprived would continue to demonstrate a constant advantage across all causes.

Table 22 Summary of lifespan variation scenarios describing inequalities between most and least deprived socioeconomic groups

Most dep. ----- least dep.	**Fluctuating Scenario** Health behaviours <u>or</u> Fundamental causes 	**Diverging Scenario** Social polarisation & Psychosocial mechanisms <u>or</u> Selection effects 	**Stalling Scenario** Fundamental causes 
Description of scenario	Lifespan variation gap between the most and least deprived groups over time fluctuates: from periods of widening to periods of narrowing Magnitude of lifespan variation gap depends on spend of health improvement in given time period Lifespan variation gap between the most and least deprived minimal at shared levels of life expectancy.	Lifespan variation gap widening over time and widening at shared life expectancy Least deprived experience mortality compression causing decreasing lifespan variation Most deprived do no experience mortality compression causing increasing lifespan variation	Lifespan variation gap between socioeconomic groups could remain constant over time All socioeconomic groups experience decreasing lifespan variation but least deprived always experience greatest decrease and always maintain an advantage Higher lifespan variation for most deprived at shared levels of life expectancy
Age-cause specific patterns	Ages and causes of death driving lifespan variation gap associated with health behaviours e.g. smoking Lower mortality rates experienced by least deprived first before diffused to most deprived Most deprived may benefit from lower mortality rates at shared life expectancy as they capitalise on the lessons already learnt	Premature ages of death driving higher lifespan variation in the most deprived socioeconomic group External causes of death that are hypothesised to be associated with social determinants of health e.g. vulnerability to social and economic risk Homogeneity of cause of death within different socioeconomic groups	Least deprived always experience lower mortality rates across all ages but ages and causes of death not distinctly different between socioeconomic groups

8.2.8 Research gap

The critical evaluation of the three scenarios proposed by van Raalte et al. (2014) illustrates why the age and cause specific pattern of mortality change and the impact on socioeconomic inequalities in lifespan variation can be used to evaluate existing hypotheses for health inequalities. This issue is of direct relevance for studying Scotland.

The mortality differential in Scotland, relevant to a number of comparable Western European countries, has been the focus of much research attention and a number of theories proposed (Hanlon et al., 2005, Walsh et al., 2016, Campbell et al., 2013, Walsh et al., 2010). Seventeen theories were categorised by McCartney et al. (2012a) as either: artefactual explanations, downstream explanations, midstream explanation, or upstream explanations. The explanations which received the most support were those which understood Scotland's mortality problem within the context of, and in response to the consequences of, increasing relative deprivation.

Scotland is a country which experienced acute levels of deindustrialisation which is evidenced to have had detrimental effects in terms of poverty, relative inequalities and ultimately health (Graham et al., 2012, Walsh et al., 2010). McCartney et al. (2012b) add that these social inequalities were exacerbated further by the pursuit of neoliberal policies during the 1980s. Neoliberal policies have been found to be associated with detrimental health outcomes, with a limited time lag following their implementation, in Eastern Europe and the USA (Stuckler et al., Beckfield and Krieger, 2009). Like these countries, previous mechanisms for social protection in Scotland may have dissolved over time

(Walsh et al., 2016). These theoretical explanations recognise the important role that the political economy plays in determining the distribution of resources, and in particular those resources that enable individuals to fully participate in the socially accepted standard of living (Townsend, 1987).

Further insight into these hypothesized drivers of population mortality can be gained by studying lifespan variation. van Raalte et al. (2014) explicitly stated that the proposed scenarios and the interpretations they presented should be used to inform the analysis of lifespan variation inequalities in other economically developed countries. It is still ambiguous as to which scenario, if any, may adequately reflect the lifespan variation experience of Scotland. Identifying the ages and causes of death which have been responsible for widening inequalities in lifespan variation, and the most recent indications of improvement, will provide further clarity. It is this research gap which is the focus of the final analysis chapter of this thesis.

8.3 Data & methods

Data used in this chapter were the same as in the previous chapter: Census population estimates and death counts by sex and single year of age, matched with the Carstairs score of area level socioeconomic deprivation. A detailed summary of these data are available in chapter 7 of this thesis.

8.3.1 Cause specific mortality rates

The analysis in chapter 7 required an all-cause mortality rate by single year of age. The analysis in this chapter required cause specific mortality rates by single year of age, the sum of which would equate to the all-cause mortality rate. The

cause of death categories needed to be mutually exclusive in order for the cause specific rates to sum to the all-cause mortality rate used in chapter 7.

The International Classification of Diseases (ICD) was used to assign each death into one of the following five categories: external causes, circulatory diseases, respiratory diseases, cancers, or other causes. Cause of death categories were harmonized to ensure that there was comparability between ICD9 and ICD10. Full details on the relevant ICD codes included in each category can be found in the data and methods chapter of this thesis.

The choice of categories was informed by the analysis carried out by van Raalte et al. (2014) and by Leyland et al. (2007a). The existing literature on premature mortality in Scotland has highlighted the impact deaths from suicide, alcohol harm, substance abuse and violence have had in terms of inequality (Leyland, 2004, Walsh et al., 2016, Schofield et al., 2016). These causes of death have been referred to as external causes or diseases of despair and are known to be socially patterned (van Raalte et al., 2014, Alexander, 2008). It was important to include circulatory diseases as these causes of death have driven mortality decline in economically developed countries in the time period covered (van Raalte et al., 2014, Vallin and Meslé, 2004). Respiratory diseases and cancers reflect the other major causes of death in Scotland.

8.3.2 Smoothing procedure

Census population estimates were only available up to an open ended age group of 85+. The analysis of lifespan variation estimates by socioeconomic deprivation required cause-specific mortality rates up to an open ended age interval of

110+. This was to ensure comparability with the HMD data used for estimating the population level lifespan variation trend.

In order to obtain cause specific mortality rates, up to an open ended age interval of 110+, the all-cause mortality rate used in chapter 7 was proportioned out to reflect the cause specific proportions as they stood at age 84 for each sex, quintile of deprivation and year combination. This approach assumed that the proportion of the total deaths by sex, quintile of deprivation and year remained constant across each single year of age for age ≥85. This gave cause-specific mortality rates for age 0 to age 110+ for each sex, quintile of deprivation and Census year (40 cause-specific data sets in total). Although results are reported for all ages, deaths at older ages generally have multiple underlying causes (van Raalte et al., 2011). Therefore the distinction between the causes of deaths beyond age 85 should be interpreted with caution.

8.3.3 Decomposition analysis

Age and cause specific decomposition analysis was performed by importing the calculated age-cause-specific mortality rates in to an adapted version of the Visual Basics Application program provided by Shkolnikov and Andreev (2010) and applying the stepwise decomposition algorithm.

This results chapter focuses on the lifespan variation estimates for the most and least deprived quintile only. This made the number of decompositions to be carried out more manageable and the interpretation of the results clearer.

The results section reports a total of 12 decompositions. The first section reports the decomposition of the change in lifespan variation achieved between 1981 and 2011 for the least deprived quintile and for the most deprived quintile. The

second section reports the lifespan variation gap between the most and least deprived quintile at each year (1981, 1991, 2001 and 2011). The final section reports the decomposition of the lifespan variation gap between the most and least deprived quintile when they achieved the most comparable level of life expectancy, albeit at different points in time. All decomposition analysis was carried out for males and females separately because their mortality experiences are known to differ.

The age distributions of death are plotted from the all-cause mortality rate (the sum of the cause specific mortality rates) and briefly discussed before each decomposition result: this allows a visual interpretation of where age differences in mortality occurred and how the age distribution of death has changed over time. Plotting the age distribution of the all-cause mortality rates, as opposed to the age distribution of the cause-specific mortality rates, is consistent with how the results have been presented in previous chapters of this thesis and makes comparisons more straight forward. The age distributions of the cause-specific mortality rates (age 0 to 110+) are reported in appendix 9 to appendix 12 for the most and least deprived quintiles only.

8.4 Results

8.4.1 Change in lifespan variation over time

Figure 53 demonstrates the lifespan variation trends by sex and socioeconomic deprivation at each census year: 1981, 1991, 2001 and 2011. These are the same lifespan variation estimates reported in chapter 7 of this thesis.

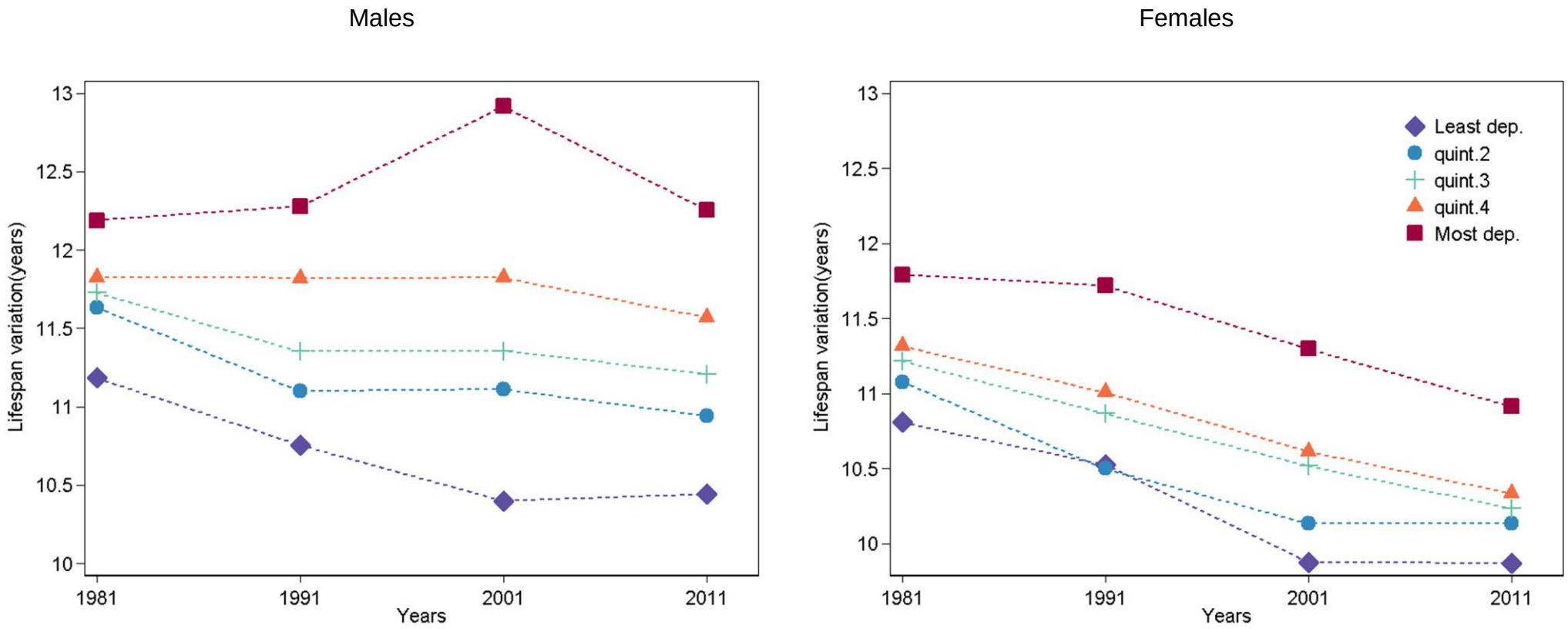
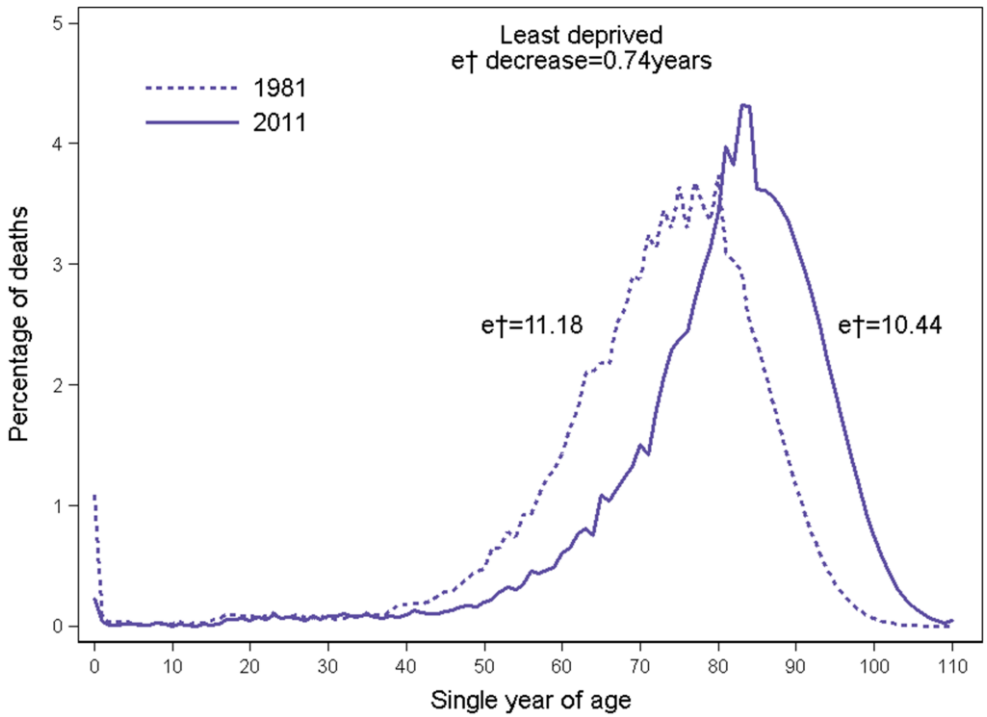


Figure 53 Lifespan variation trends by socioeconomic deprivation

As was previously evidenced in chapter 7 of this thesis, lifespan variation demonstrates a strong social pattern. Improvements in lifespan variation have generally been achieved for females across all socioeconomic groups but this is not the case for males. Over time males from the most deprived socioeconomic group have experienced increasing lifespan variation which is interpreted as increasing inequality in age at death within this group. This is in contrast to males from the least deprived quintile who have generally experienced decreasing lifespan inequality in age at death within this group, although improvement appears to have stagnated between 2001 and 2011. It is important to point out that the lifespan variation trends by socioeconomic deprivation are not exclusively linear. These results suggest that over time the least deprived have consistently experienced mortality compression but the most deprived have not.

Plotting the age distributions of death from the life tables that were calculated from the all-cause mortality rates for each sex, quintile of deprivation and year can help to better understand this. Figure 54 compares the age distributions of death in 1981 with the age distribution in 2011 for males in the least deprived quintile and males in the most deprived quintile. Figure 55 shows the same for females.



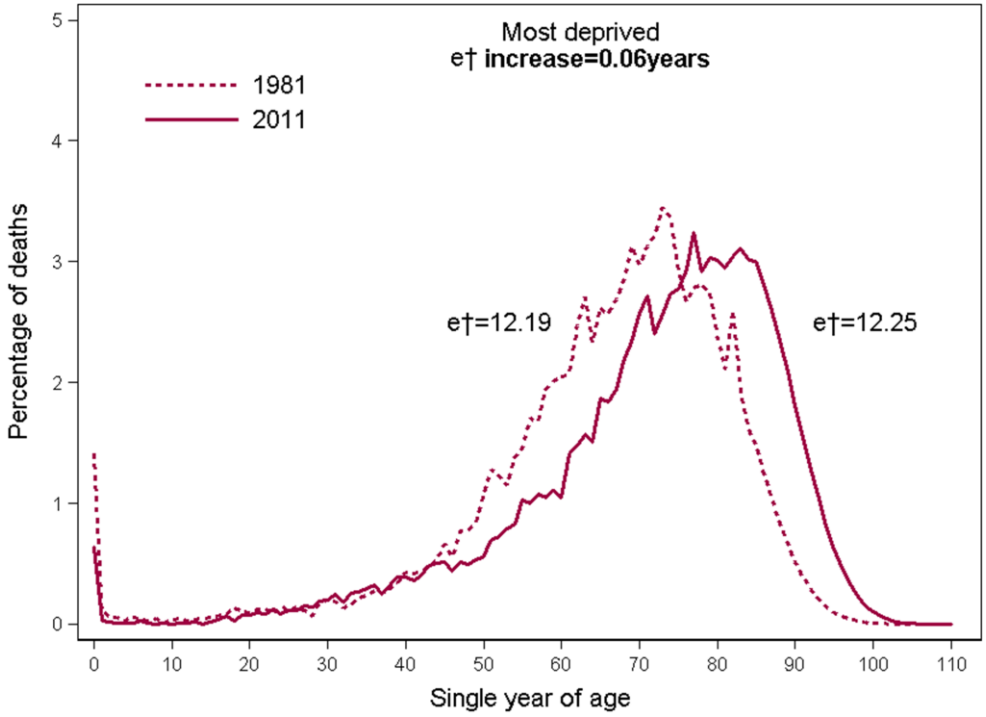


Figure 54 Age distribution of death in 1981 compared to 2011 by socioeconomic deprivation (Carstairs quintile), Males

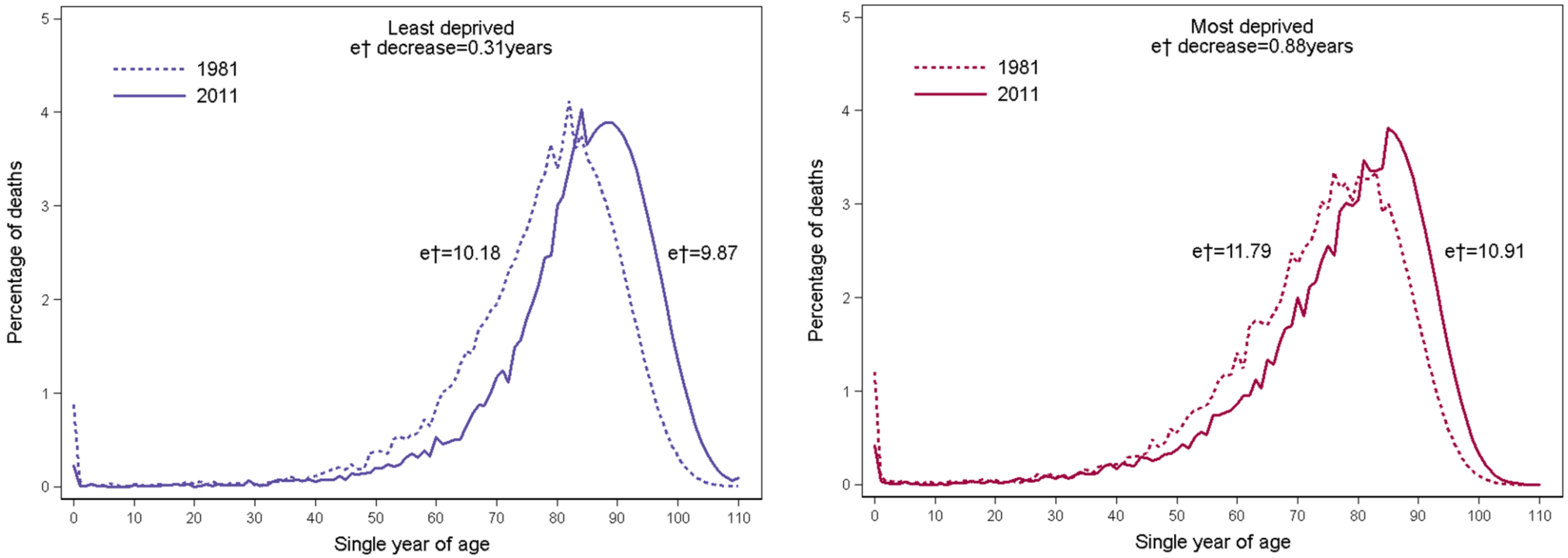


Figure 55 Age distribution of death in 1981 compared to 2011 by socioeconomic deprivation (Carstairs quintile), Females

Over time the age distribution of death has shifted toward older ages. This is the case for males and for females from the most and least deprived quintiles of deprivation. However there are some notable differences to the change in the age distribution of death achieved between 1981 and 2011.

The shift towards older ages is greater for the least deprived and the width of the distribution appears narrower. For males and females from the most deprived quintile there is a lingering tail to the left hand side of the distribution. This is particularly evident for males, where the left hand tail of the distribution for 2011 is slightly higher than the left hand tail of the distribution in 1981 around ages 30-40. This suggests that there has been a lack of mortality compression because the proportion of premature adult age deaths has not declined and this is reflected in the increasing lifespan variation estimates.

Figure 56 shows the decomposition results which quantified the age and cause specific contributions to the change in lifespan variation between 1981 and 2011 by socioeconomic deprivation for males. Figure 57 shows the same for females.

Spikes above the zero line reflect the ages at which mortality change between 1981 and 2011 increased lifespan variation, thus contributed to increasing inequality. Spikes below the zero line reflect the ages at which mortality change between 1981 and 2011 decreased lifespan variation, thus contributing to decreasing inequality. Each spike reports the combined total of the contributions made by each cause of death at each single year of age.

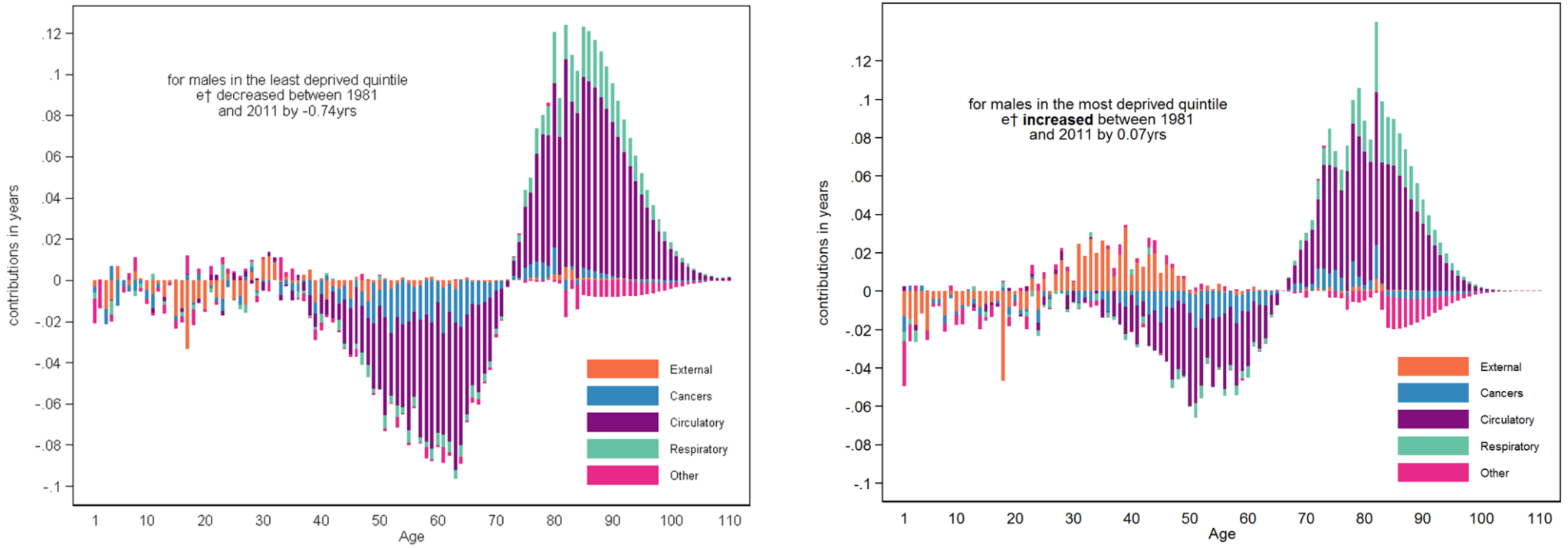


Figure 56 Age and cause specific contributions to change in lifespan variation between 1981 and 2011, by socioeconomic deprivation, Males

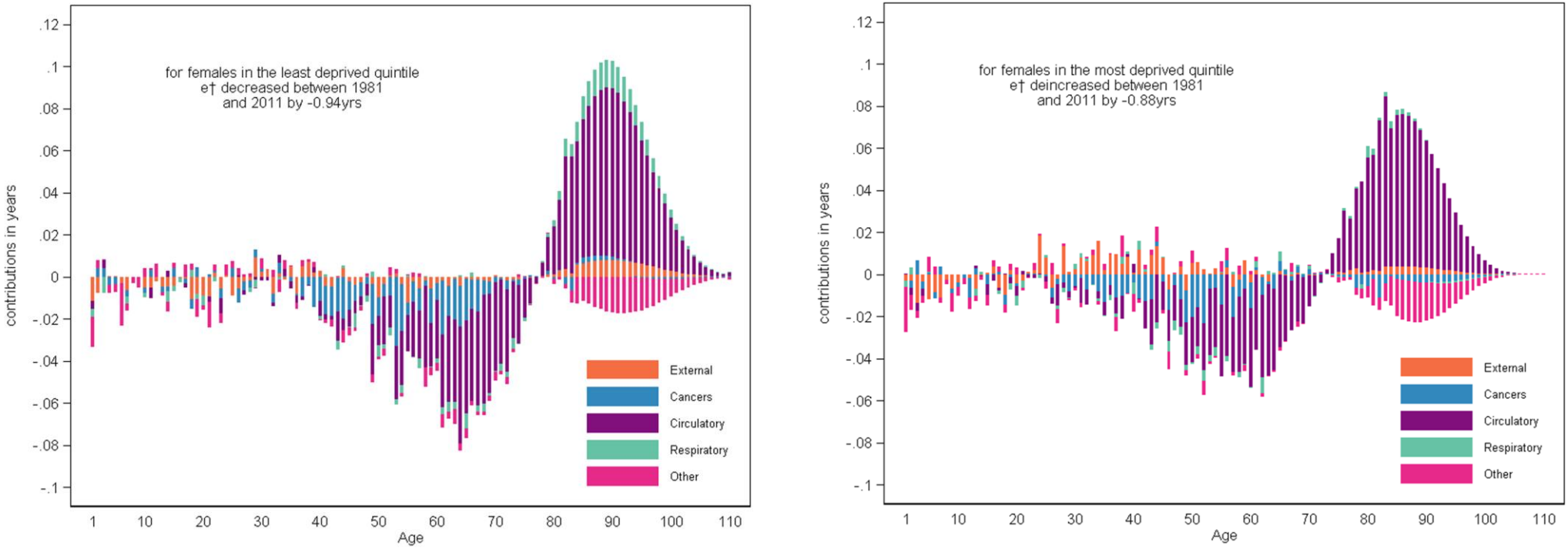


Figure 57 Age and cause specific contributions to change in lifespan variation between 1981 and 2011, by socioeconomic deprivation, Females

For males from the least deprived quintile, lifespan variation was lower in 2011 than it had been in 1981 because mortality compression across younger ages outpaced mortality expansion across older ages. The reduction in lifespan variation for the least deprived quintile largely came from lower mortality rates from circulatory causes and cancers. However lower mortality rates from circulatory diseases in 2011 compared to in 1981 have also contributed to mortality expansion, by saving lives at older ages. This could be expected as circulatory diseases are the leading causes of death during this time period (van Raalte et al., 2014). This age and cause specific pattern is also evident, but to a much lesser extent, for males from the most deprived quintile: the length of the spikes contributing to decreasing lifespan variation is much shorter indicating that mortality rates from circulatory diseases were not reduced as much as in the least deprived quintile.

These smaller gains do not explain why lifespan variation for males from the most deprived quintile lifespan variation was higher in 2011 than it had been in 1981. This was caused by mortality compression failing to outpace mortality expansion. For males in the least deprived quintile a total of 2.00 years of inequality were added from mortality expansion but this was counterbalanced because -2.74 years of inequality were reduced from mortality compression. This equated to a total decrease in lifespan variation of -0.74 years between 1981 and 2011. For males in the most deprived quintile a total of 1.73 years of inequality were added from mortality expansion but this was not counterbalanced because only 1.66 years of inequality were reduced from mortality compression. This equated to a total increase in lifespan variation of 0.07 years between 1981 and 2011.

Although some notable contributions to mortality expansion came from reducing deaths from circulatory diseases across older ages for males from the most deprived quintile, of concern are the contributions made to increasing lifespan variation from external causes of death. Males' age 27 to 55 years old from the most deprived quintile experienced higher mortality rates from external causes of death in 2011 than in 1981. This expanded the age distribution of death and added 0.37 years of inequality to the total change between 1981 and 2011. These contributions to increasing lifespan variation between 1981 and 2011 were not evident for the least deprived quintile. A similar pattern was found for the most deprived females, but the contributions from external causes of death to increasing lifespan variation were not as large as they were for males. Therefore females from the most deprived quintile experienced a decrease in lifespan variation between 1981 and 2011.

8.4.2 Lifespan variation gap at same time point

Figure 58 compares the age distributions of death for the most deprived quintile with the age distribution of death for the least deprived quintile at the four time points: 1981, 1991, 2001, and 2011. Figure 59 shows the same for females.

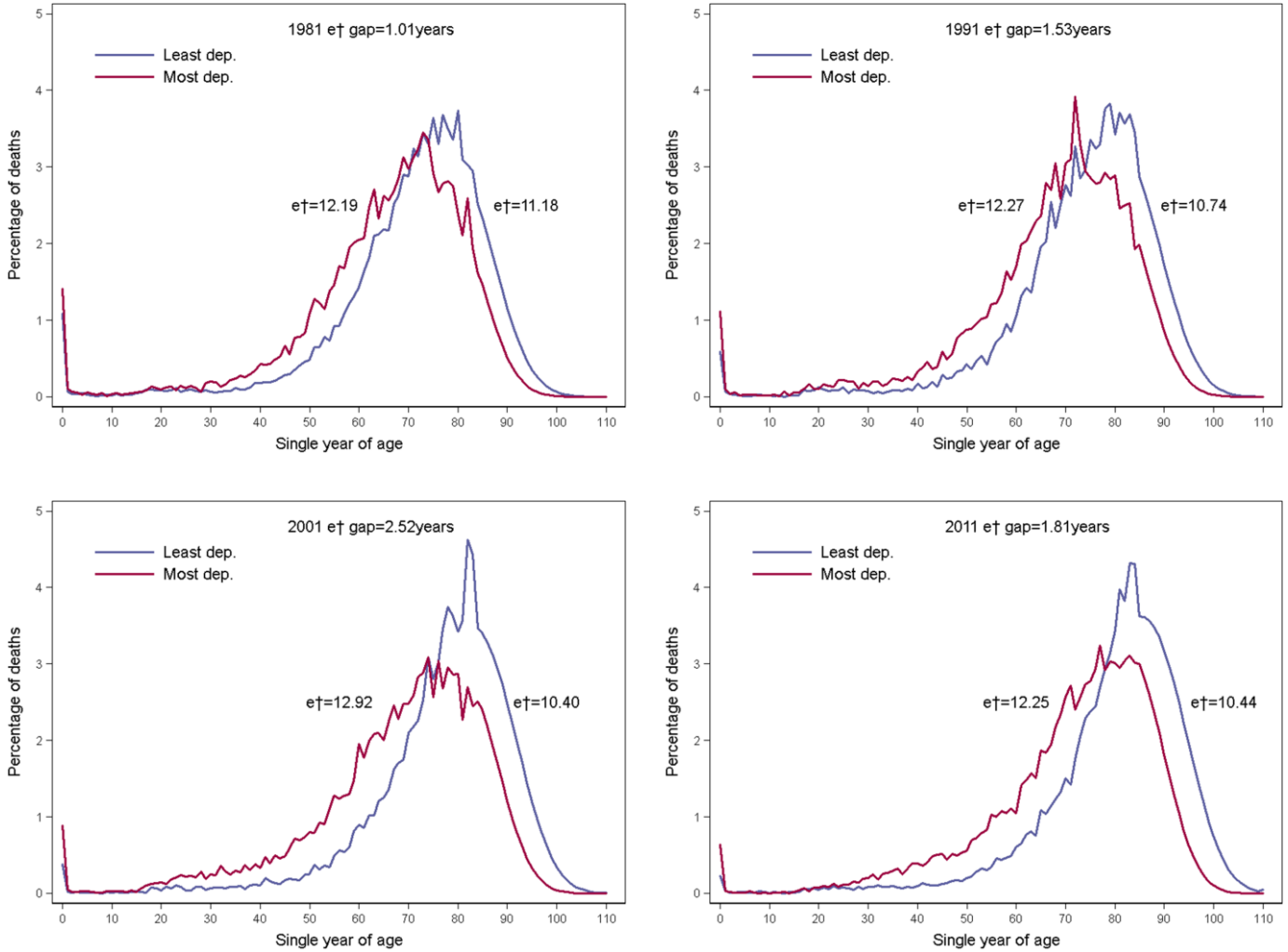


Figure 58 Age distribution of death comparing most deprived quintile with least deprived quintile, Males

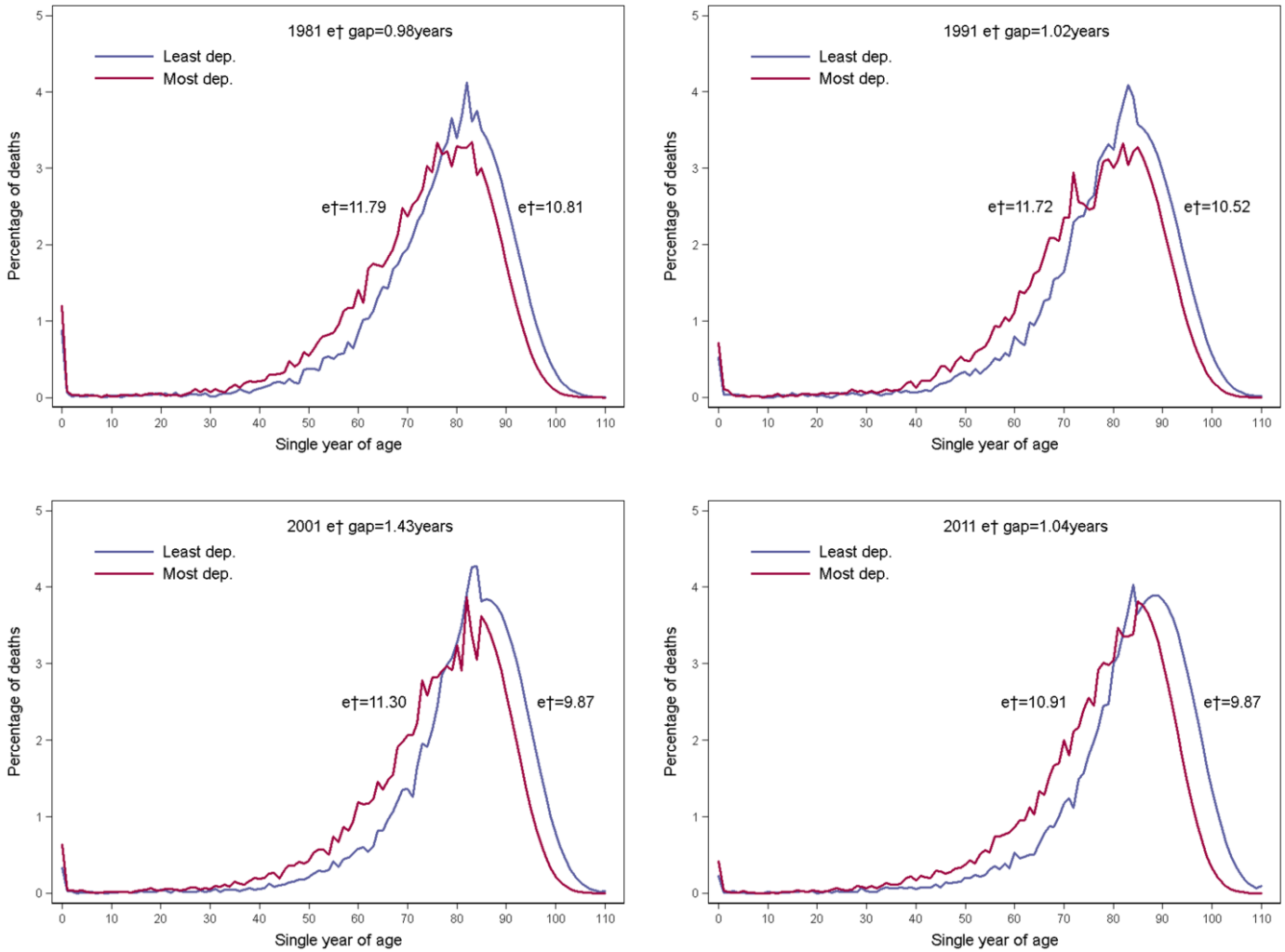


Figure 59 Age distribution of death comparing most deprived quintile with least deprived quintile, Females

At all four time points the age distribution of death for the least deprived quintile is shifted further towards older ages and the width of the distribution appears narrower. This is the case for males and females. The age distributions in 1981 are more similar than the age distribution in 2011. As time has progressed the age distribution of death experienced by the most and least deprived quintiles have become increasingly different. Of particular concern is the difference in the left hand tail of the distribution. After 1981 the purple line (least deprived) gets further away from the red line (most deprived) over younger ages. The gap between the lines is particularly stark for males in 2001: it is not a coincidence that this was when the lifespan variation gap was widest. This suggests that there has been a lack of mortality compression for the most deprived because the proportion of premature adult age deaths has not declined and is reflected in the increasing lifespan variation estimates.

Figure 60 shows the decomposition results which quantified the age and cause specific contributions to the lifespan variation gap between the most and least deprived quintile in: 1981, 1991, 2001 and 2011. Figure 61 shows the same for females.

Spikes above the zero line reflect the ages at which the difference in mortality between the most and least deprived added to the lifespan variation gap. Spikes below the zero line reflect the ages at which the difference in mortality between the most and least deprived has reduced the lifespan variation gap between the most and least deprived. Each spike reflects the combined total of the contributions made by each cause of death at that single year of age. Higher mortality rates in the most deprived quintile across ages that contribute to

mortality compression would increase the lifespan variation gap. Higher mortality rates in the most deprived quintile across ages that contribute to mortality expansion would decrease the lifespan variation gap. If there was no difference in the age specific mortality rates there would be no spikes: there is zero difference between the most and least deprived quintile across every age (Seaman et al., 2016b).

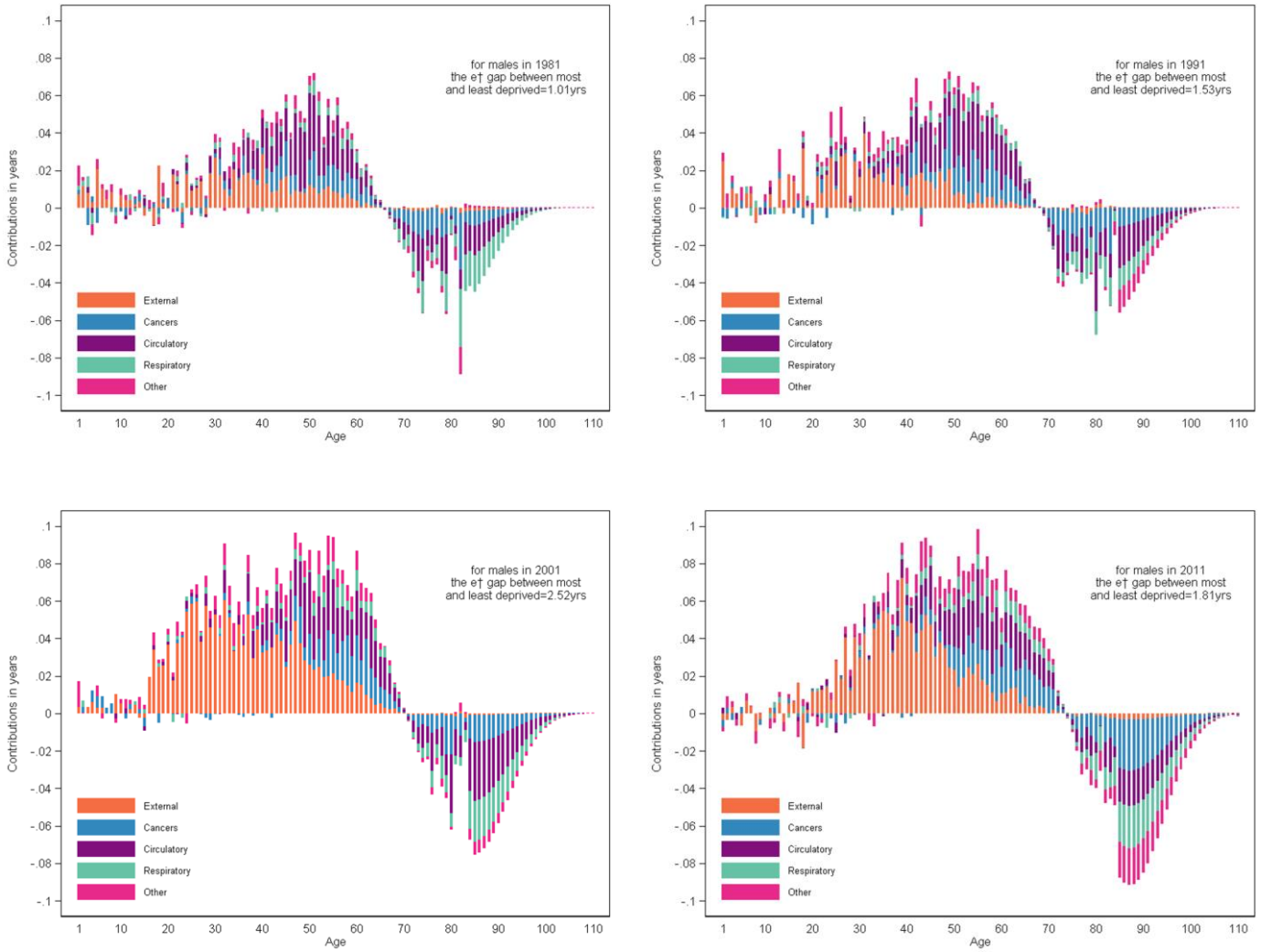


Figure 60 Age and cause specific contributions to lifespan variation gap at each time point, Males

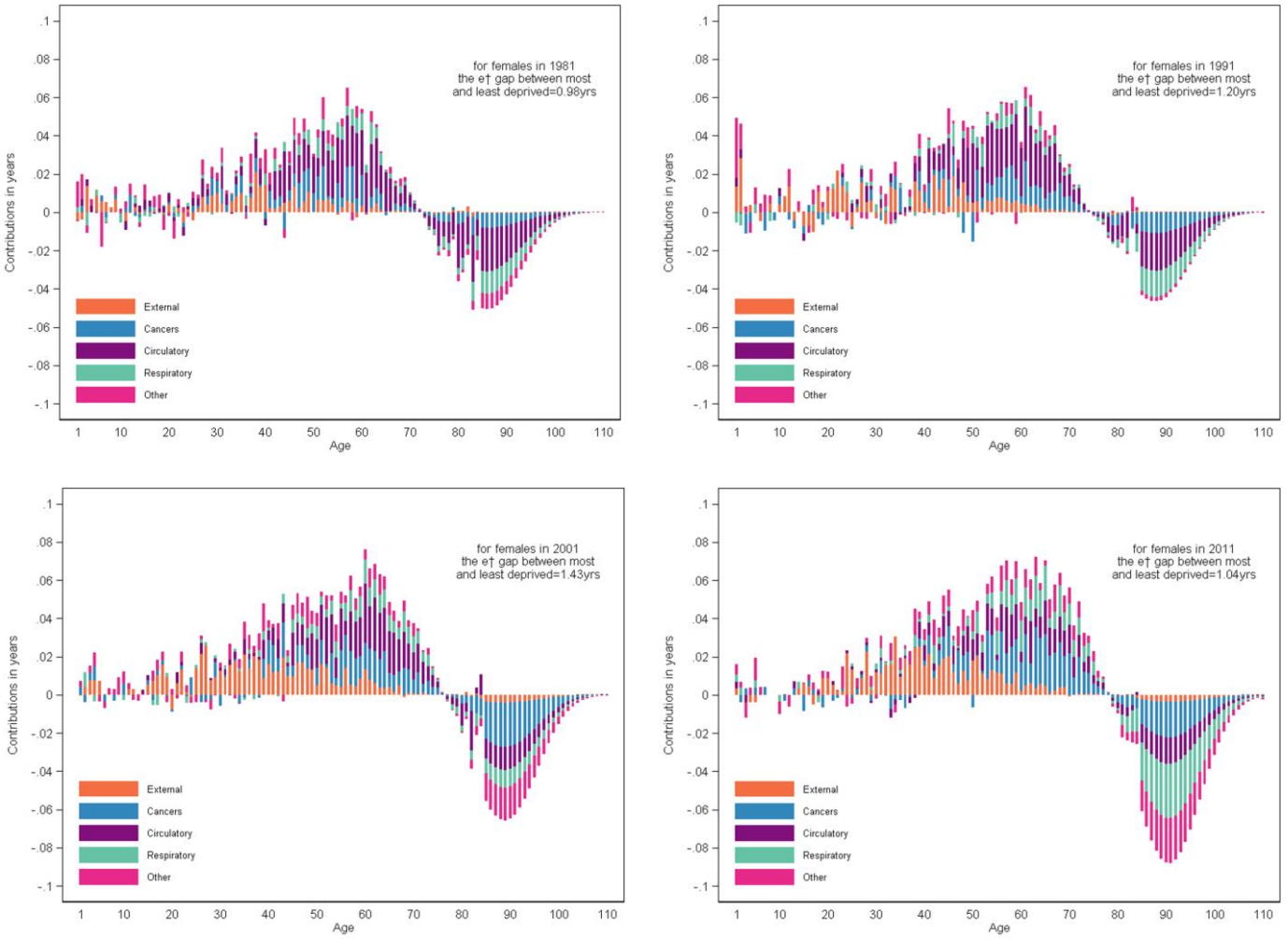


Figure 61 Age and cause specific contributions to lifespan variation gap at each time point, Females

Lifespan variation has always been higher in the most deprived quintile compared to the least deprived quintile. For males and females the gap was smallest in 1981 and highest in 2001. It is perhaps important to begin by addressing the spikes below the line which have reduced the lifespan variation gap. Most of these contributions occur across ages (ages which contribute to mortality expansion) where the mortality rate was subject to smoothing (the carrying forward of the proportion of causes from age 84 to ages ≥85 can be seen in the graphs). However it demonstrates that the higher mortality rate amongst the most deprived quintile was to their lifespan variation advantage because they did not experience as much expansion as the least deprived quintile but means that older age death rates are not improving as quickly as the least deprived.

However it is differences in external causes of mortality across working adult ages (ages that contribute to mortality compression) that provide much of the explanation for the lifespan variation gap between the most and least deprived quintile at every time point. This is particularly strong for males and the external causes of death dominate the decomposition analysis in 2001 when the lifespan variation gap was widest. Table 23 details the aggregate contributions made to the lifespan variation gap between the most and least deprived by each cause of death for males. Table 24 details the equivalent information for females.

Table 23 Aggregate contributions to lifespan variation gap between most and least deprived quintile, by cause of death, Males

	1981		1991		2001		2011	
	years	%	years	%	years	%	years	%
External	0.55	55.05	0.71	46.43	1.64	65.26	1.26	69.50
Cancers	0.03	3.26	0.08	5.04	0.16	6.24	0.03	1.42
Circulatory	0.30	29.77	0.36	23.48	0.22	8.59	0.34	18.74
Respiratory	-0.16	-15.86	-0.06	-3.76	-0.11	-4.43	-0.13	-7.35
Other	0.28	27.78	0.44	28.82	0.61	24.34	0.32	17.68
Total gap	1.01	100.00	1.53	100.00	2.52	100.00	1.81	100.00

Table 24 Aggregate contributions to lifespan variation gap between most and least deprived quintile, by cause of death, Females

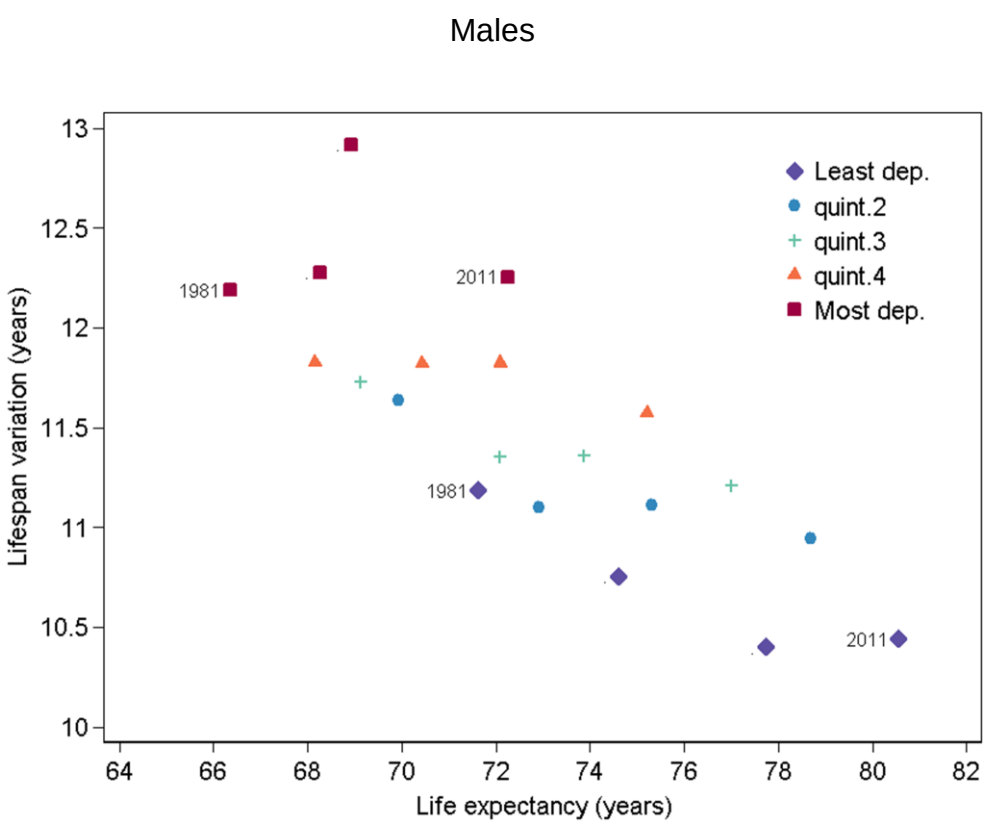
	1981		1991		2001		2011	
	years	%	years	%	years	%	years	%
External	0.24	23.94	0.38	31.82	0.55	38.61	0.53	50.45
Cancers	0.18	17.93	0.11	9.05	0.08	5.86	0.23	21.56
Circulatory	0.28	28.36	0.40	33.67	0.42	29.69	0.22	20.55
Respiratory	0.00	0.05	-0.03	-2.49	0.10	6.72	-0.08	-7.76
Other	0.29	29.72	0.33	27.95	0.27	19.12	0.16	15.21
Total gap	0.98	100.00	1.19	100.00	1.42	100.00	1.05	100.00

For males, higher mortality rates in external causes of death accounted for 55% of the total lifespan variation gap in 1981. By 2011 external causes of death accounted for 70% of the total lifespan variation gap. The percentage explained by external deaths also increased for females: from 24% in 1981 to 50% in 2011. Higher mortality rates from circulatory causes amongst the most deprived compared to the least deprived have also added to the lifespan variation gap but these contributions explain a smaller percentage of the gap than external causes of death.

Although external causes of death are the main explanation for the lifespan variation gap at each time point these may not account for the lifespan variation gap when a shared level of life expectancy was achieved.

8.4.3 Lifespan variation gap at a similar level of life expectancy

Figure 62 plots life expectancy along the x axis and lifespan variation along the y axis for all quintiles of socioeconomic deprivation. The analysis in this chapter focuses on comparing the most deprived (red square) and least deprived quintile (purple diamond) only. Given that life expectancy has increased for all socioeconomic groups between each time period then it can be deduced from the graphs that the lowest level of life expectancy relates to the first time point (1981) and the highest life expectancy relates to the last time point (2011).



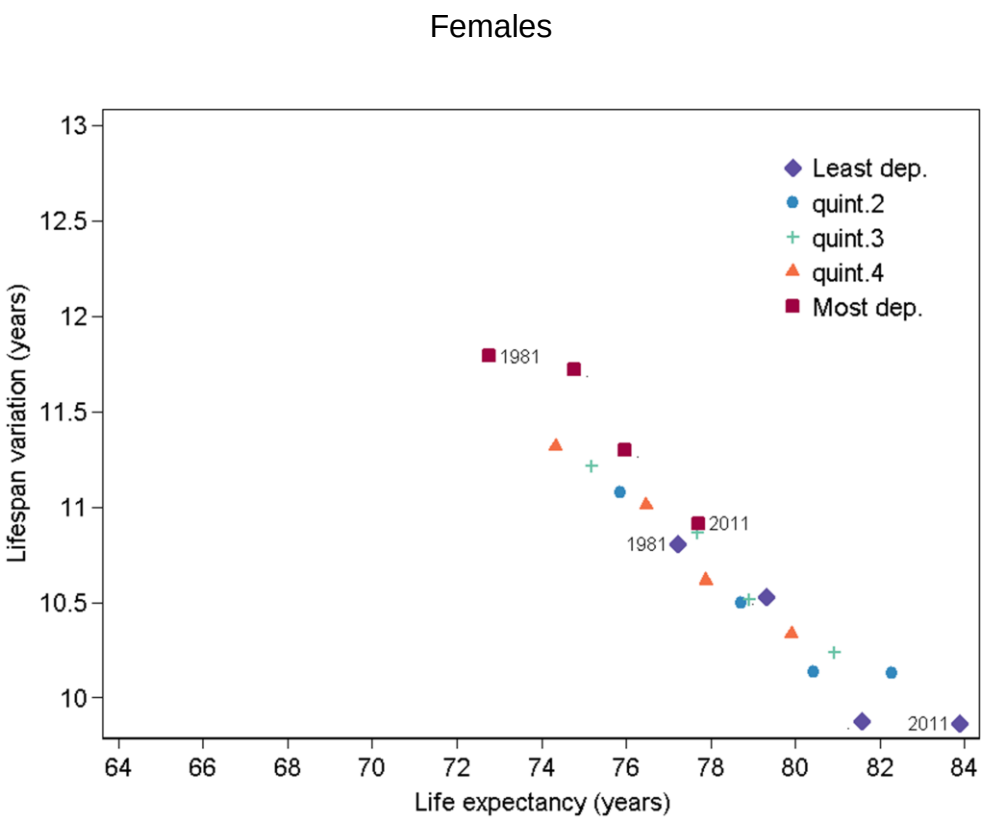


Figure 62 Trends in lifespan variation by trends in life expectancy, by sex and socioeconomic deprivation (Carstairs quintile)

At the same level of life expectancy, the least deprived quintile had lower levels of lifespan variation. A finding of particular interest is that the highest level of life expectancy achieved by the most deprived quintile in 2011 was achieved by the least deprived in 1981. The fact that it is only possible to make one comparison of the lifespan variation gap when a similar level of life expectancy was achieved is in itself a point of concern. It is perhaps even more surprising to find that the life expectancy achieved by the most deprived in 2011 was actually slightly higher (red square slightly further along the x axis) than the life expectancy achieved by the least deprived in 1981 but that the most deprived experienced this with higher inequality. This is particularly evident for males (the gap on the y axis between the red square and the purple diamond is much bigger than the gap for females).

Figure 63 compares the age distributions of death for the most deprived quintile in 2011 with the age distribution of death for the least deprived quintile 1981: the only two time points when the level of life expectancy was comparable. This shows that the difference in the relationship between life expectancy and lifespan variation is due to differences in the age distribution of mortality.

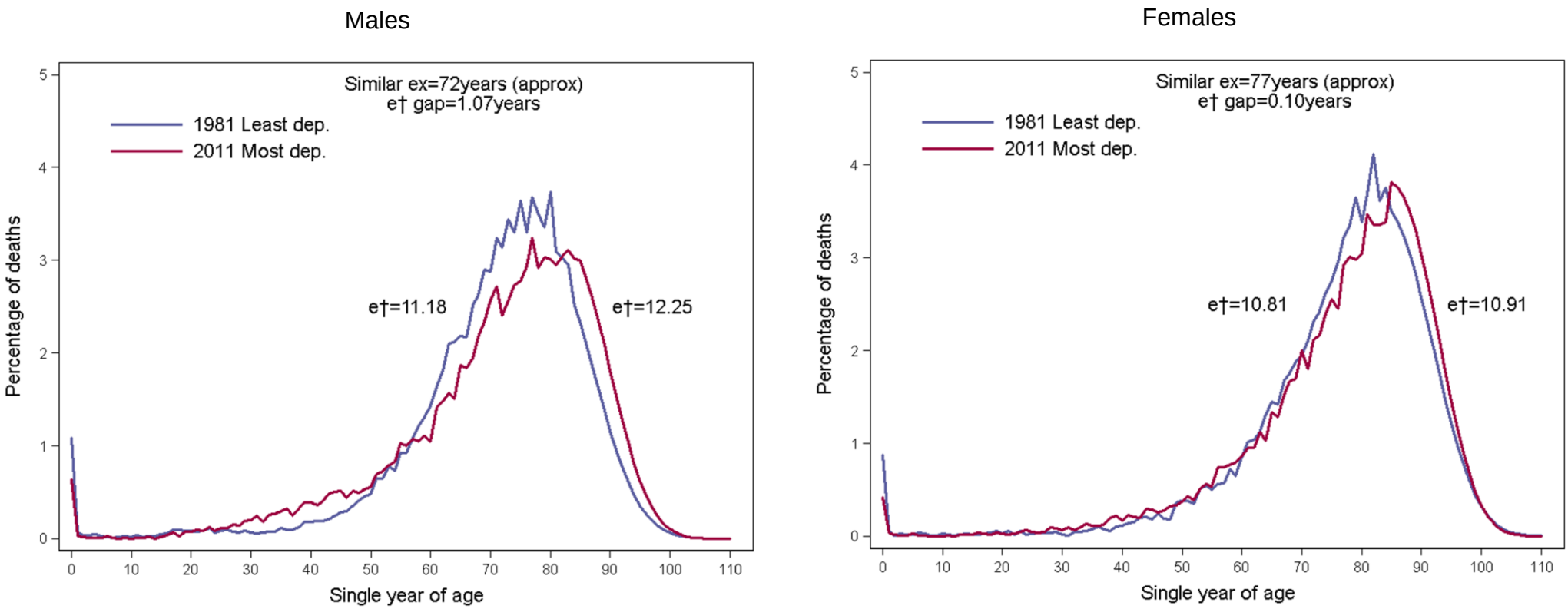
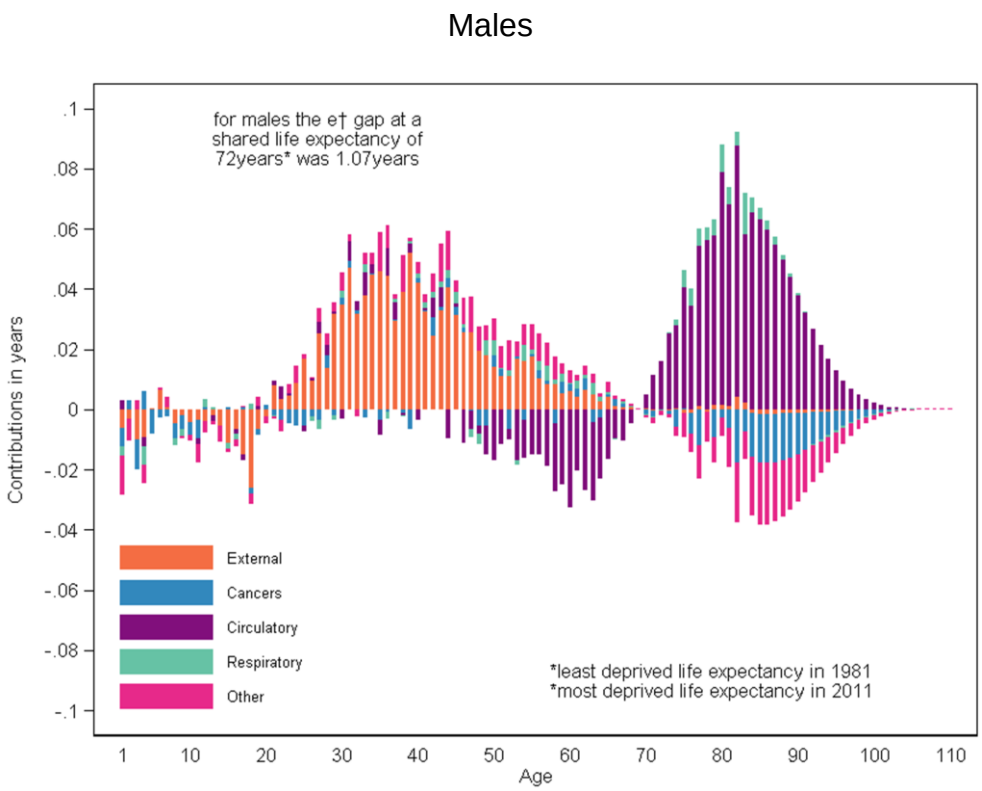


Figure 63 Age distribution of death at shared level of life expectancy, by sex and socioeconomic deprivation (Carstairs quintile)

For males and females the age distribution for the most deprived quintile in 2011 is shifted further along older ages, indicating a gross shift of the distribution. However the proportion of deaths under the left hand tail of the distribution is higher for the most deprived quintile in 2011 than the least deprived quintile in 1981. This is much more apparent for males than for females. For males the gap between the red line and the purple line across the left hand tail of the distribution is much wider. A stronger message to take from this is the fact that the proportion of premature adult deaths amongst the most deprived quintile in 2011 was higher than the proportion of premature adult deaths in the least deprived quintile 30 years earlier. The gap between the red line and the purple line for females is much smaller across the left hand tail than the gap for males but there is still a gap. Figure 64 reports the age and cause specific decomposition of the lifespan variation gap between the most and least deprived quintile when life expectancy was similar.

Spikes above the zero line reflect the ages at which the difference in mortality between the most and least deprived, when life expectancy was comparable, added to the lifespan variation gap. Spikes below the zero line reflect the ages at which the difference in mortality rates, when life expectancy was similar, reduced the lifespan variation gap between the most and least deprived. Each spike reflects the cumulative total of the contributions made by each cause of death at that single year of age. If there was no difference in the age specific mortality rates there would be no spikes indicating there is zero difference between the most and least deprived quintile across every age (Seaman et al., 2016b).



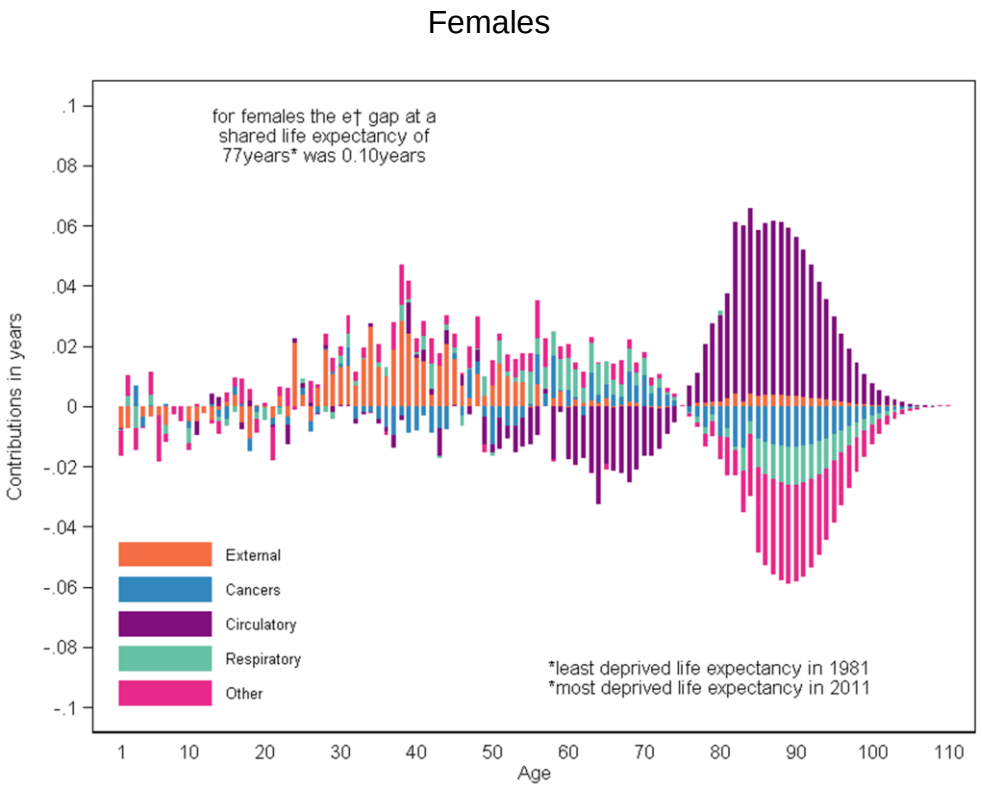


Figure 64 Age and cause specific contributions to lifespan variation gap at shared level of life expectancy, by sex and socioeconomic deprivation (Carstairs quintile)

It is important to state that the contributions from older ages should be interpreted with caution as these were subject to smoothing (the carrying forward of the proportion of causes from age 84 to ages ≥85 can be seen in the graphs). This is important to consider when discussing the contributions from circulatory diseases (purple contributions). These contributions are above and below the zero line at different ages. Lower mortality rates from circulatory diseases between ages 50 and 70 years old compressed the age distribution of death for the most deprived. Lower mortality rates from circulatory diseases experienced by the most deprived quintile in 2011 that occurred across ages >70 expanded the age distribution of death. These findings strongly suggest that the most deprived experienced a temporal advantage in terms of lower mortality rates from circulatory diseases reflecting the health care advances made during the time period being studied (van Raalte et al., 2014, Vallin and Meslé, 2004). However this does little to explain the lifespan variation gap, at a similar level of life expectancy, because these lower mortality rates occurred across ages that both add to and reduce the lifespan variation gap.

The higher lifespan variation experienced by the most deprived quintile compared to the least deprived quintile, despite a shared level of life expectancy, was largely explained by differences in external causes of death across working adult ages. This is particularly evident for males, and is reflected by the fact that the lifespan variation gap is larger than for females (1.07 years and 0.10 years respectively).

8.5 Discussion

8.5.1 Summary of results

The findings in this chapter were produced to provide a clearer interpretation of the explanations for lifespan variation inequalities between socioeconomic groups in Scotland between 1981 and 2011. Differences in mortality rates from circulatory diseases between socioeconomic groups were evident. However these deaths largely occurred across ages of death that contributed to both mortality expansion and mortality compression meaning they did little to explain socioeconomic inequalities in lifespan variation. External causes of death across working adult ages explain most of the lifespan variation inequalities in Scotland. This finding was not unexpected considering the evidence in chapter 5 and chapter 6 of this thesis, demonstrating the impact premature deaths have had on lifespan variation at the population level, and the substantial body of work that has focused on premature deaths from external causes in Scotland (Schofield et al., 2016, Leyland, 2004, Norman et al., 2011). Yet this age and cause-specific pattern of mortality is counterintuitive: these deaths are more likely to be amenable to the social determinants of health and should be less costly to reduce than deaths at older ages associated with medical and health care advanced (Nau and Firebaugh, 2012, Vallin and Meslé, 2004). This is in addition to the fact that deaths at these ages fundamentally represent a large loss of potential years of life.

8.5.2 Explanations for premature mortality in Scotland

The reasons causing these deaths are complex and a number of theoretical explanations have been put forward to account for them (Mackenbach, 2012,

Lynch et al., 2001, Marmot, 2001). Particular attention has been paid to the theories most likely to apply to Scotland. Emphasis has been placed on the upstream, structural explanations for health inequalities and the potential for social policies to escalate or mitigate risks associated with premature death (McCartney et al., 2012a, Walsh et al., 2016, Schofield et al., 2016).

Unfortunately no definitive conclusions can be drawn from the analysis in this chapter about which types of theories may best explain mortality inequalities in Scotland. However the analyses chose to draw upon the theories that were summarised in relation to three lifespan variation scenarios (van Raalte et al., 2014) and aimed to evaluate if one of these scenarios would adequately describe the empirical findings for lifespan variation inequalities in Scotland. The scenarios proposed by van Raalte et al. (2014) aimed to assess whether the following theories might account for lifespan variation inequalities: health behaviours, social polarisation and psychosocial mechanisms, selection effects, or fundamental causes. Although the theoretical interpretations of each scenario are open to debate they are important for highlighting that the ages and causes of death that have the greatest impact in terms of inequality may be distinct from the ages and causes of death that have the greatest impact in terms of average population health. Both of these issues should be considered by governments when choosing public health strategies (Smits and Monden, 2009).

A diffusion scenario would be characterised by fluctuating socioeconomic inequalities in lifespan variation: the least deprived would gain from health improvements first causing the gap to widen before knowledge was diffused to

the most deprived causing the gap to narrow. It was hypothesised that this scenario would suggest that health behaviours were the dominant mechanism driving the persistence of inequalities in mortality.

A diverging scenario would be characterised by widening socioeconomic inequalities in lifespan variation. If the empirical evidence reflected this scenario it was hypothesised that explanations focused on the impact of social polarisation on psychosocial mechanism were perhaps most appropriate for explaining inequalities in the age and cause-specific patterns of death. It may also indicate that socioeconomic groups are becoming increasingly homogenous in terms of health if there are social sorting processes at play.

The final scenario proposed that lifespan variation inequalities may remain constant over time because the most deprived are always positioned to negate risk of premature death, irrespective of changes to the mortality risk profile. This would suggest that a fundamental causes hypothesis would be a strong theoretical framework.

The findings in this chapter indicate that a fluctuating scenario may not adequately account for socioeconomic inequalities in lifespan variation in Scotland: not all socioeconomic groups experienced a decrease in lifespan variation. Instead it is suggested that the relationship between average population and total inequality differs depending on socioeconomic deprivation which would be more likely under a diverging or stalling scenario.

A diverging scenario described by van Raalte et al. (2014) is somewhat reflected in the empirical findings for Scotland. An increasing lifespan variation trend was found for the most deprived and a decreasing trend found for the

least deprived. This resulted in a widening cross-sectional gap over time although there were some recent improvements in 2011.

A diverging scenario fits well with the upstream explanations that have previously been put forward for mortality inequalities in Scotland (McCartney et al., 2012b, Walsh et al., 2016). These explanations recognise the link between widening mortality inequalities in Scotland and the consequences these have for its public health standing within Western Europe (Walsh et al., 2016, McCartney et al., 2012b, McCartney et al., 2012a). However, it is important to recognise that each scenario is open to interpretation and a more detailed interpretation would have been achieved if annual trend data have been available. The research in this chapter was restricted to decennial data which is not sufficient enough to draw any definitive conclusions. This limitation was particularly problematic for making comparisons at shared levels of life expectancy between the most and least deprived quintiles: only one comparison was possible. Despite these limitations the international literature also finds support for a diverging scenario (van Raalte et al., 2014).

8.5.3 Comparisons with existing studies

In Finland the most deprived group (as measured by occupation) experienced stagnating lifespan variation and the least deprived decreasing lifespan variation between 1971 and 2010. It is interesting to note that the increase in lifespan variation was greater for the most deprived in Scotland than for the most deprived in Finland. Unfortunately these two studies are not directly comparable. The two studies utilised different socioeconomic indicators that are derived from differing theoretical principles. Although both studies focused

on the same general time period the analysis in this chapter was only able to report decennial lifespan variation estimates by socioeconomic deprivation. This is because the Carstairs measure of area level socioeconomic deprivation is derived from the decennial Census. The study in Finland was able to report lifespan variation for every five years. Although the measure of occupational based socioeconomic status was available annually, five years' worth of data were aggregated to increase the statistical power of the lifetables. The measure of occupation applied by van Raalte et al. (2014) only permitted the reporting of lifespan variation conditional upon survival to age 31. This chapter reported lifespan variation from age 0 which again means the studies are not directly comparable.

8.5.4 Strengths and limitations

Reporting lifespan variation from age 0 corresponds to the reporting of life expectancy from age 0. The decomposition methods used allowed the contributions from deaths at every single year of age, up to an open ended interval of 110+, to be estimated. It was also possible to report lifespan variation from age 0 in the context of this thesis because the measure of deprivation used was applicable to the whole population: it utilised an area level measure of socioeconomic deprivation not an individual level of occupational socioeconomic position. However it has been suggested that studies interested in the social distribution mechanisms of adult mortality should consider restricting analysis to ages 15+. This is because the causes of death driving mortality change over time differ across ages. Infectious disease and effective medical intervention historically reduced infant and childhood deaths rapidly. Adult mortality is influenced by more complex mechanisms that

change slowly (Vallin and Meslé, 2004, Smits and Monden, 2009). Smits and Monden (2009) support this perspective by stating that 80% of all deaths in 2000 (one of the most recent years for which they had data) occurred in ages 15+.

Both approaches are recognised throughout the literature however it is important to critically reflect on the choices made throughout the research process to ensure that results are interpreted appropriately. The contributions made to lifespan variation inequalities from ages ≥85, for example, need to be interpreted with caution as the cause specific mortality rates at these ages were subject to a smoothing technique previously applied (van Raalte et al., 2011). Smoothing was required as population estimates were only available up to an open ended age interval of 85+ but the analysis required a mortality rate up to age 110+. It was also a valid approach as deaths at these older ages tend to be associated with multiple causes and it may not always be possible to distinguish between them (van Raalte et al., 2014).

Despite these limitations the analysis in this chapter makes an important research contribution by being the first to formally quantify the impact unequal age and cause specific patterns of mortality, within socioeconomic groups, have had on lifespan variation inequalities in Scotland.

Lifespan variation is interpreted as the average years of life lost per death in the population. This has an intuitive public health meaning that may be more accessible to interpret than traditional measures of mortality inequalities such as risk ratios. It is also a measure which reflects the theoretical principle that inequality in health is an inequality irrespective of its correlation with any

other dimension of social inequality (Murray et al., 1999, Gakidou and King, 2002, Tuljapurkar, 2010).

The lifespan variation estimates were calculated using the most robust and up-to-date mortality and population data available. The research applied a validated measure of socioeconomic deprivation that was derived specifically for studying health inequalities and covered a significant period of time. The substantial period of time covered is also an advantage with the existing literature suggesting that this is when mortality inequalities in Scotland began to diverge from its closest comparator countries (Campbell et al., 2013, McCartney et al., 2012b).

8.6 Conclusion

This chapter identified that external causes of death, across premature adult ages, account for increasing socioeconomic inequalities in lifespan variation in Scotland. Scotland must reduce deaths at these ages in order to simultaneously achieve increases in life expectancy and reductions in inequality.

This type of evidence is valuable for extending our understanding of why health inequalities have widened in most Western European countries, not only Scotland. This is despite the fact that most of these countries have long established welfare states and formal commitments to tackling inequality (Bambra, 2011a, The Scottish Government, 2008, Mackenbach, 2012). However the underlying causes of premature death are complex.

The results reported in this thesis, culminating in this chapter, strongly supports the types of theoretical explanations that recognise the impact relative deprivation has for distributing the risk of premature death. It is

argued that countries less successful at reducing these risks are those which place less value on the role of social protection policies: policies which at the very least are designed to protect the most vulnerable members of society and at the very most may minimise the negative consequences of relative deprivation that entire populations are exposed to (Wilkinson and Pickett, 2006).

In pragmatic terms social protection policies could be adopted to redistribute the structural determinants of inequality such as income, wealth, employment and housing. Targeting the structural determinants could minimise material and psychosocial inequalities that stem from increasing relative deprivation and ultimately ensure that all members of society are able to mitigate the social and economic risks associated with an untimely death. However, this is still fundamentally a theoretical problem: this thesis has demonstrated that the concept of inequality is contested and how its structural determinants are interpreted is an inherently political and ideological issue.

9 Discussion and conclusion

Chapter 9 concludes this thesis by building upon the discussion sections of each of the analysis chapters. It has four sections and a final conclusion. The first section revisits the concept of total inequality and the motivation for measuring lifespan variation in Scotland. The second section summarises the main empirical findings from the quantitative analyses carried out and relates these to some existing, international studies of lifespan variation and the explanations put forward for Scotland's premature mortality problem and steep inequalities. The third section critically evaluates the strengths and limitations of this thesis in terms of data used and the analysis methods applied. The fourth section highlights some future research directions by discussing the potential for incorporating subjective (self-assessed) data on health into empirical measures of total inequality.

9.1 Novelty of measuring lifespan variation

This thesis measured, analysed and described lifespan variation in Scotland. Lifespan variation is a novel measurement of mortality inequality that reflects the concept of total inequality. Total inequality proposes that the distribution of health should be considered a primary outcome variable in itself, much like the distribution of income is in economics (Tuljapurkar, 2010). In economics it is accepted that similar averages can be achieved with very different distributions. Therefore income studies routinely measure the distribution of income across individuals (Murray et al., 1999). The distribution of income is also important for population health and health inequalities. It could be assumed that increasing

average incomes would have been enough to reduce the mortality gradient. However, mounting evidence suggests that it is the distribution of income (relative deprivation) that is more important for population health and the mortality gradient: the more egalitarian the distribution of income the higher life expectancy tends to be (Wilkinson, 1989, Marmot and Wilkinson, 2001, Le Grand, 1987).

Despite this evidence, traditional approaches for measuring health inequalities have tended to only focus on average health and measure the extent of inequalities in terms of the difference in average between populations (either between countries or between socioeconomic groups within countries). This approach cannot ascertain whether a homogenous gain in health has been achieved for everyone *within* the same country or *within* the same socioeconomic group, or whether health inequalities between individuals *within* the same country or socioeconomic group have stayed constant over time (Murray et al., 2002, Gakidou and King, 2002, Le Grand, 1987). Focusing on the distribution of health also allows elements of the total amount of variance in population health to be distinguished: not all elements of total health variance will be related to socioeconomic deprivation but being able to better measure the elements that are is valuable (Le Grand, 1987, Wilkinson, 1989). The distribution of health is therefore an important dimension of inequality to capture.

Lifespan variation measures the age distribution of death: higher lifespan variation equates to greater variation and higher inequality in age at death between individuals within a population. International studies have

demonstrated the value lifespan variation has for providing insight into the changing nature of health inequalities: countries with the highest level of life expectancy tend to experience the lowest level of lifespan variation (Vaupel et al., 2011). Further studies have found that the most deprived socioeconomic groups experience a double burden of mortality inequality. In addition to having the shortest life expectancy they experience the greatest amount of inequality in age at death. This is in contrast to the least deprived socioeconomic group who experience the highest life expectancy and lowest level of inequality in age at death (van Raalte et al., 2014, van Raalte et al., 2011). This burgeoning body of evidence suggests that reducing inequality should be seen as beneficial for improving average health (Wilkinson, 2005) and increasing average health and reducing inequality are not incompatible public health aims. These are issues that have direct relevance when studying mortality inequalities in Scotland: it has experienced the slowest improvements in life expectancy in Western Europe and widening mortality inequalities between socioeconomic groups (Leyland et al., 2007b, McCartney et al., 2012b). Although mortality inequalities in Scotland have been the focus of much research attention total inequality, using lifespan variation, has rarely been measured. This is despite the fact that long running data, suitable for estimating lifespan variation are available. It was this research gap that this thesis has contributed to filling.

Most of the theories put forward for poorer average health and higher mortality in Scotland have emphasised socioeconomic deprivation and increasing relative inequality (McCartney et al., 2012a, Walsh et al., 2016): these are widely considered to be the most significant determinants of health, and ultimately death, across economically developed countries (Mackenbach and Bakker, 2003,

Bambra, 2013, Beckfield and Krieger, 2009). van Raalte et al. (2014) and (Smits and Monden, 2009) effectively demonstrated that lifespan variation is a useful tool for critically evaluating existing theoretical explanations for persisting health inequalities in economically developed countries. This thesis has demonstrated that measuring lifespan variation can help to better understand the full extent of mortality inequalities in Scotland and has used lifespan variation to aid the critical interpretation of the reasons for Scotland's poor population health standing within Western Europe.

9.2 Summary of main findings

The aim of this thesis was conveyed in the following twelve research questions:

Chapter 4

1. Has Scotland's lifespan variation ranking within Western Europe changed over time?

2. Was the timing and relative rate of lifespan variation change in Scotland comparable with any other Western European country?

Chapter 5

3. Which ages of death contributed to the lifespan variation trend in Scotland?

4. Did the ages of death contributing to the lifespan variation trend in Scotland differ from the ages of death contributing to the lifespan variation trend in England and Wales?

Chapter 6

5. Was lifespan variation higher or lower in Scotland at a shared level of life expectancy with England and Wales?

6. Which ages of death account for the lifespan variation gap between Scotland and England and Wales at a shared level of life expectancy?

Chapter 7

7. Is there a socioeconomic gradient for lifespan variation in Scotland?

8. Has the socioeconomic gradient for lifespan variation in Scotland changed over time?

9. Are changes to the socioeconomic gradient for lifespan variation in Scotland related to Scotland's deteriorating lifespan variation ranking within Western Europe over time?

Chapter 8

10. Which ages and causes of death contributed to changes in lifespan variation, over time, for different socioeconomic groups in Scotland?

11. Which ages and causes of death contributed to the lifespan variation gap between the most and least deprived socioeconomic groups in Scotland?

12. Which ages and causes of death contributed to the lifespan variation gap between the most and least deprived socioeconomic groups, when life expectancy was similar, in Scotland?

Figure 65 summarises the findings of this thesis in relation to the relevant chapters, the data utilised, and the analysis carried out. Chapter 4 through 6 utilised data from the HMD for the population of Scotland to estimate lifespan

variation. Chapter 7 and 8 utilised Census population estimates, vital events data and the Carstairs score of area level deprivation to construct deprivation specific lifetables, from which lifespan variation was estimated.

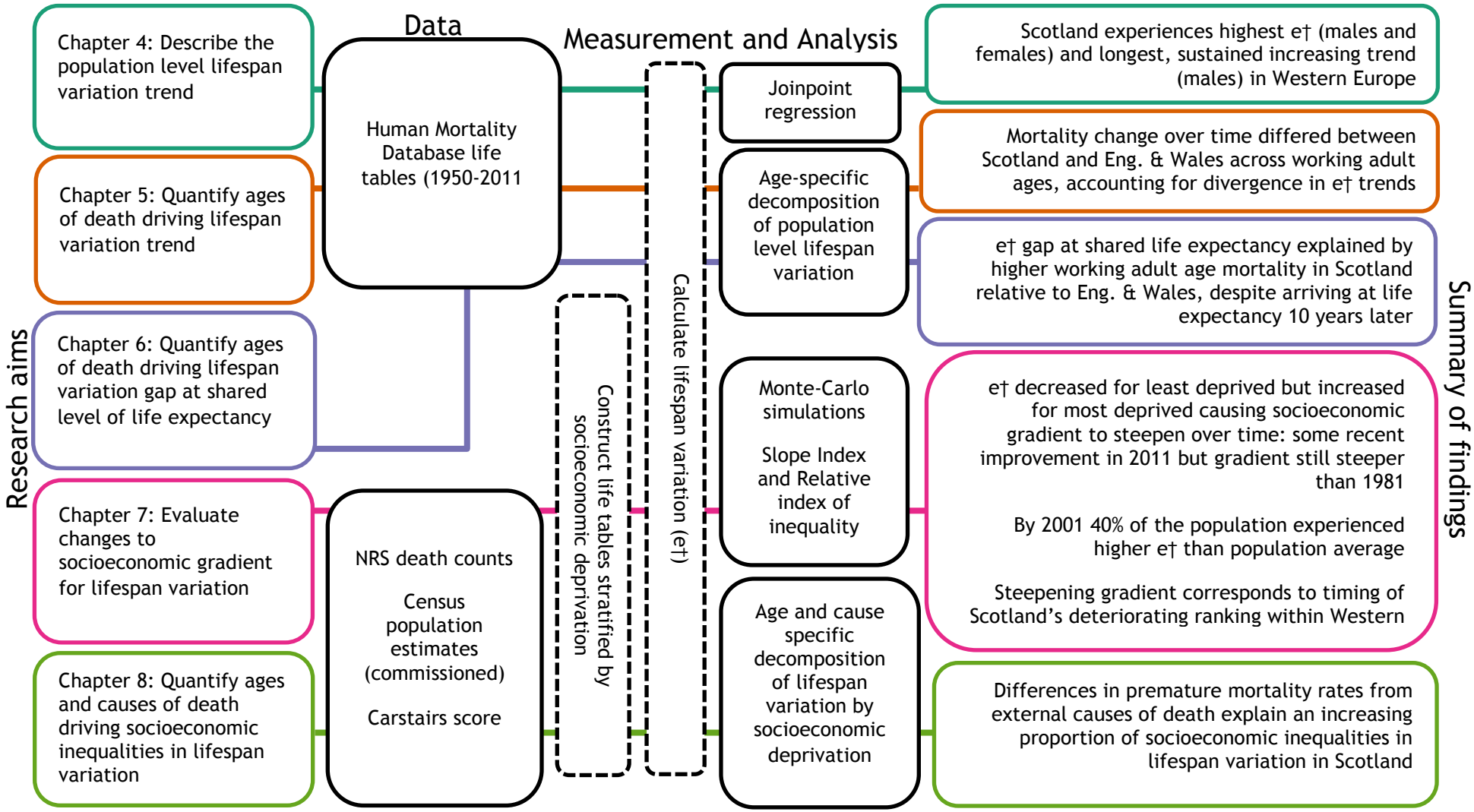


Figure 65 Summary of research stages

9.2.1 Scotland's lifespan variation trend within Western Europe

Chapter 4 demonstrated that Scotland is in the unenviable position of ranking highest in terms of inequality in age at death in Western Europe. This was the case for both males and females. Lifespan variation for females in Scotland consistently decreased following 1950 but the trend demonstrated one of the greatest reductions in the rate of change. This meant that lifespan variation for females in Scotland has not converged with the lifespan variation for females in most other Western European countries.

The results of the lifespan variation trend for males in Scotland were of further concern. The trend diverged from decreasing lifespan variation to increasing lifespan variation during the 1980s, the longest, sustained increasing trend found in any of the Western European countries included in the analysis. These results were not unexpected, considering that literature review reported the strong correlation between life expectancy and lifespan variation (Vaupel et al., 2011, Smits and Monden, 2009) and that relative rate of improvement in life expectancy in Scotland has faltered since the 1980s (McCartney et al., 2012b). The timing of Scotland's deteriorating lifespan variation ranking, relative to comparable countries, was previously presented by Popham and Boyle (2010). They estimated lifespan variation for each decade, beginning in 1950. Chapter 4 of this thesis was able to utilise more up-to-date data than Popham and Boyle (2010) and formally analysed (joinpoint regression) the trend data but the substantive conclusions reported are consistent.

9.2.2 Age specific mortality change over time in Scotland distinguishable from age specific mortality change in England and Wales

Differences in lifespan variation between countries are generally found to be explained by differences in younger working age mortality rates. This finding has been replicated across many international studies (Gillespie et al., 2014, Shkolnikov et al., 2011, Vaupel et al., 2011). It has also been replicated in this thesis beginning in chapter 5, which aimed to establish if the age specific mortality change over time in Scotland differed from the age specific mortality change over time in England and Wales. England and Wales are valuable comparator countries to use for studying Scotland: they are geographical neighbours, share social, political and economic contexts and a national government. The findings in chapter 5 were consistent with previous cross national studies of the age specific mortality changes driving differences in lifespan variation: mortality change across working adult ages in Scotland was distinguishable from that in England and Wales, causing Scotland to experience higher lifespan variation and a diverging trend. This adds to the body of evidence which has suggested Scotland has a premature mortality problem (Campbell et al., 2013, Leyland, 2004, Norman et al., 2011).

9.2.3 Increasing inequality in age at death at shared levels of life expectancy

It is hypothesised that cross national differences in the age distribution of death could be underpinned by differences in stages of the epidemiological transition (Nau and Firebaugh, 2012, Smits and Monden, 2009). This was the impetus for the analysis in chapter 6 of this thesis. The theory of epidemiologic transition focuses on the changing patterns of health, disease and mortality and the associated demographic, economic and sociological changes. It also proposes

that disease intervention programmes may be a determining factor for fertility change and for socioeconomic progression (Omran, 1971). The fact that stages of epidemiological transition started at different points in time, for different countries, (e.g. the smoking epidemic (Mackenbach et al., 2008)) or population interventions introduced to control risk factors for cardiovascular disease (Vallin and Meslé, 2004)) prompted the suggestion that it would be more appropriate for cross national comparative studies of health to control for epidemiological time as opposed to calendar time (Mackenbach et al., 1997, Smits and Monden, 2009).

Previous studies have attempted to control for epidemiological time by comparing differences in lifespan variation, and the underlying age distributions of death, at shared levels of average population health (life expectancy) (Smits and Monden, 2009, Seaman et al., 2016b). Three possible scenarios were subsequently presented for describing lifespan variation inequalities between comparable countries at shared levels of life expectancy.

Firstly, the forerunner hypothesis proposed that countries leading the way in terms of life expectancy would have done so by reducing deaths across ages that lead to mortality compression and countries arriving at the same life expectancy later in time would only be able to do so with a very similar level of lifespan variation. Secondly, the diffusion hypothesis suggested that countries arriving at a high level of life expectancy later in time would do so with lower lifespan variation because they are able to capitalise on the lessons already learnt from forerunner countries. Thirdly, this thesis added the laggard hypothesis which proposed the opposite: countries arriving at a high level of life expectancy later

in time would do so with higher lifespan variation because they have benefited from interventions that increase life expectancy but contribute to mortality expansion (e.g. reducing deaths around the ages where the greatest proportion die (Vaupel, 1986)) but have not capitalised on the interventions required to reduce inequalities.

International studies have suggested that most countries arriving to a high level of life expectancy do so with very similar or lower lifespan variation that the countries preceding them: only a few countries do so with higher lifespan variation. The analysis in chapter 6 controlled for epidemiological time in order to establish which scenario may best describe Scotland relative to England and Wales. Scotland achieved the same high level of life expectancy as England and Wales, later in time, with lower inequality. However following the 1980s Scotland lost any lifespan variation advantage: it was achieving the same high level of life expectancy as England and Wales, later in time, with higher inequality. This was explained by the fact that Scotland was experiencing higher premature mortality rates across working adult ages than England and Wales despite arriving at a comparable level of life expectancy up to ten years later.

Lifespan variation differences between countries, despite controlling for average population health (e.g. stage of the epidemiological transition), are thought to reflect differences in terms of a country's capacity to replicate outside practices or to capitalise on earlier innovations for mortality reduction (Vallin and Meslé, 2004, Nau and Firebaugh, 2012). Historically rapid reductions in mortality rates at infancy and childhood have been achieved: these are ages of death that simultaneously improve average population health and reduce

inequality. Deaths at these ages were also highly responsive to changes to the social determinants of health in Western Europe (e.g. sanitation, housing, education, welfare) and represented the most years of life expectancy to be gained (Oeppen, 2008). However as countries transition through stages of mortality development potential life expectancy gains from younger ages diminish and a single, older modal age at death emerges (Gillespie et al., 2014). This also means that the spread of ages of death considered to be premature expands to include working adult ages (Zhang and Vaupel, 2009, Oeppen, 2008). Reducing deaths at premature ages no longer represents the most gains in terms of population level life expectancy but they are still the only means of reducing inequality (mortality compression): even though each individual death at an older age loses fewer years of expected life than a death at a younger age, the total loss of years from deaths at older ages will be greater because a larger proportion of the population now dies at these ages than premature ages (Vaupel, 1986, Oeppen, 2008, Gillespie et al., 2014). A simplified summary suggests that averting deaths at older ages may be more likely to be associated with costly medical interventions or the lagged effects of social determinants of health at earlier stages of the life course (Nau and Firebaugh, 2012). Of course medical interventions and social determinants are not mutually exclusive processes and both can help to avert deaths at any age. However, populations risk increasing lifespan variation if they only focus on achieving steady declines at older ages via medical interventions without continuing to tackle deaths at premature ages via the social determinants of health. Therefore the laggard phenomena, as demonstrated for Scotland, could be considered counter intuitive.

9.2.4 Increasing socioeconomic gradient for lifespan variation associated with Scotland's deteriorating ranking within Western Europe

It is hypothesised that Scotland's higher premature mortality rates, and subsequent higher lifespan variation, relative to England and Wales may be associated with socioeconomic deprivation (Schofield et al., 2016, Scott et al., 2013, McCartney et al., 2013). The findings in chapter 7 and chapter 8 were produced to increase our understanding of the impact socioeconomic deprivation has for higher lifespan variation in Scotland within the context of Western Europe.

Lifespan expectancy inequalities in Scotland are known to have increased over time, in both absolute and relative terms. This was also the case when measuring lifespan variation. Despite there being some improvement in the socioeconomic gradient for lifespan variation between 2001 and 2011 it was still steeper in 2011 than it had been in 1981. The steepening of the socioeconomic gradient was associated with Scotland's deteriorating ranking within the Western Europe.

Traditionally life expectancy or age standardised mortality have been used to evaluate the impact socioeconomic deprivation has on mortality (Mackenbach et al., 2003, Munoz-Arroyo and Sutton, 2007, Murray et al., 2002, Mackenbach and Kunst, 1997). It has now been well established that the most socioeconomically deprived groups have the shortest average life expectancy and the highest age-standardised mortality rates. International studies, measuring lifespan variation, have demonstrated that there is a further dimension of socioeconomic inequality to consider: the most deprived socioeconomic groups also experience the

greatest amount of inequality in age at death. Some of these studies conclude that countries with steeper socioeconomic gradients for average health measured have lower average population health, indicating that inequality is detrimental for everyone (Wilkinson and Pickett, 2006, Vaupel et al., 2011). Chapter 7 provided further support for this conclusion.

9.2.5 Ages and causes of death accounting for increasing socioeconomic inequalities in lifespan variation within Scotland

Distinct age and cause specific patterns of mortality can allude to the types of social and aetiological processes that may be causing inequalities in lifespan variation in Scotland to widen over time (van Raalte et al., 2015, van Raalte et al., 2014). The findings in chapter 8 illustrated differences in mortality rates from circulatory diseases between socioeconomic groups. The least deprived experienced lower mortality rates from circulatory diseases and the most deprived experienced higher mortality rates from circulatory diseases. However this cause specific pattern of mortality occurred across ages of death that contributed to both mortality expansion (older ages) and mortality compression (working adult ages) meaning they tended to counter balance one another and did little to explain socioeconomic inequalities in lifespan variation. Higher premature mortality rates from external causes of death for the most deprived group occurred across younger ages which prevented mortality compression. This age and cause specific pattern of mortality explained an increasing proportion of lifespan variation inequalities in Scotland over time.

This finding demonstrates the emergence of “newer” causes of death (Leyland et al., 2007b) and suggests socioeconomic inequalities in mortality persist

despite the nature and risk factors for mortality changing over time (Phelan et al., 2010, Link and Phelan, 1995). van Raalte et al. (2014) described three lifespan variation scenarios that may occur and outlined which theories of health inequalities these might provide support for.

Firstly, the fluctuating scenario hypothesised that the socioeconomic gap in lifespan variation between the most and least deprived groups could change between periods of narrowing to periods of widening, but the lifespan variation gap at shared levels of life expectancy would be minimal. This scenario could be interpreted as support for either a health behaviours explanation or a fundamental causes' explanation (Link and Phelan, 1995, Phelan et al., 2010). Over time the knowledge and motivation for health behaviours could be diffused to lower socioeconomic groups. However, the gap would widen during periods of rapid health improvement as the least deprived adopt new health behaviours for reducing premature mortality and narrow once the behaviour diffuses to the most deprived. This explanation has been heavily criticised and does not seem to account for lifespan variation inequalities: smoking, the most detrimental of health behaviours, was unable to account for socioeconomic differences in lifespan variation (van Raalte et al., 2015).

An alternative explanation for a fluctuating scenario would be the fundamental causes' theory. This hypothesises that a socioeconomic gradient for health is always present regardless of the disease profile of the population (Link and Phelan, 1995, Phelan et al., 2010). This is not because of inequalities in the distribution of health behaviours but because socioeconomic status embodies the types of resources (so called fundamental causes) that impact health: money,

power, prestige, and knowledge. Fundamental causes are always relevant for protecting health but the socioeconomic gradient may fluctuate from narrowing to widening as the disease profile changes or a new intervention is implemented allowing for fundamental resources to be utilised (Scott et al., 2013).

Secondly, the diverging scenario suggested that the least deprived may experience mortality compression but the most deprived would not, causing the lifespan variation gap to widen over time and to widen at shared levels of life expectancy. This scenario could be due to the consequence of increasing income inequalities, the decline of heavy industries and the rise in precarious employment, all of which have had a disproportionate impact on the most deprived socioeconomic groups (Bambra, 2010, Dorling, 2009). This could have impacted the psychosocial responses that are hypothesised to be important for avoiding premature death causing a lack of compression in the most deprived group only (Marmot and Wilkinson, 2001, Wilkinson and Pickett, 2006).

Another interpretation of a divergence scenario is that it would support a health selection explanation, either in terms of ill health causing downward mobility or in terms of a meritocracy increasing upward mobility for individuals with favourable health characteristics. If either of these mechanisms were active then socioeconomic groups would be expected to become more homogenous in terms of health, causing trends to diverge.

Thirdly, the stalling scenario hypothesised that the absolute lifespan variation gap between socioeconomic groups could remain constant over time because the least deprived socioeconomic group are always able to maintain a health advantage regardless of the types of mortality risk that the population is

exposed to. Again this would provide evidence for a fundamental causes explanation: the most deprived are able to exploit the material, psychosocial and knowledge resources that are most important for health (Link and Phelan, 1995, Phelan et al., 2010).

The trends identified in chapter 7 showed that the lifespan variation gap widened between 1981 and 2001 but showed signs of narrowing in 2011. This feature was described in the fluctuating scenario proposed by van Raalte et al. (2014). In Scotland the most deprived experienced higher lifespan variation when achieving a comparable level of life expectancy as the least deprived, despite reaching this level of life expectancy 30 years later. This was a feature described in the diverging scenario but not in the fluctuating scenario. It was suggested that a fluctuating scenario could be interpreted as evidence for the fundamental causes' theory to explain increasing mortality inequalities, while a diverging scenario could be interpreted as evidence for increasing income inequalities to account for increasing mortality inequality.

9.2.6 Explanations for Scotland's premature mortality problem

Fundamental causes' theory and increasing income inequalities are plausible explanations for Scotland's premature mortality problem, which this thesis has demonstrated caused it to experience the highest level of lifespan variation in Western Europe and widening lifespan variation inequalities between socioeconomic groups. Both of these explanations have been considered in the existing literature measuring population health and inequality in Scotland in terms of average health status (Scott et al., 2013, Walsh et al., 2010, McCartney et al., 2012a).

Scott et al. (2013) explicitly set out to test the applicability of the fundamental causes' theory to Scotland. Their findings show support for this explanation because the socioeconomic gradient for suicide, alcohol related and HIV deaths has increased over time but there is no clear socioeconomic gradient for non-preventable causes of death. Phelan and Link (2005) hypothesised that this would be the case: the most advantaged socioeconomic groups would be able to operationalise their resources most effectively in relation to reducing preventable causes of death causing the socioeconomic gap to widen. Access to fundamental resources would be of little benefit in relation to causes of death that were not yet fully understood and there would be limited differences between socioeconomic groups.

Walsh et al. (2010) set out to identify if high relative deprivation, poverty and adverse health in Scotland were associated with its exposure to deindustrialisation. The process of deindustrialisation disproportionately impacted the most deprived areas in the Britain (Dorling et al., 2007) with some of the worst affected areas being concentrated within the West Central region of Scotland (Hanlon et al., 2006, Graham et al., 2012). Between 1971 and 2005 this area of Scotland lost 62% of industrial employment, one of the most severe losses out of 10 areas across Europe, and a subsequent slowing in the rate of life expectancy improvement (Walsh et al., 2010). It is hypothesised that increased alienation, anxiety and reduced control are psychosocial mechanisms which link deindustrialisation, higher relative deprivation and risk of premature death (Marmot and Wilkinson, 2001, McCartney, 2012, Wilkinson, 1997, Aldabe et al., 2011).

The potential for psychosocial mechanisms to link structural change and account for increasing mortality inequalities is plausible but contested. Lynch et al. (2000) argue that psychosocial interpretations are vulnerable to de-contextualization and risk being associated with regressive political agendas. It is assumed that psychosocial mechanisms focus on perceptions of inequality that may not be amenable to intervention, rather than objective material experiences that are structurally determined. Marmot and Wilkinson (2001) dispute this: social structure and relative deprivation have profound psychosocial impacts and to deny this burdens the responsibility for depression, anxiety and insecurity with individuals. This argument is supported by the finding that some societies have been able to minimise the impact structural changes have on mortality by providing an adequate safety net during times of social or economic uncertainty, which minimises the negative effects of psychosocial health (Bambra, 2009). This suggests that increased adverse health outcomes experienced in Scotland are not simply an inevitable outcome of deindustrialisation and increasing income inequality but are the consequence of a society's failure to manage and mitigate these processes (McCartney et al., 2012b).

It is important to note that this thesis cannot provide any insight into explanations for the so called 'Scottish effect': the inability for area level social deprivation to account for Scotland's excess mortality relative to England and Wales (Popham and Boyle, 2011, Hanlon et al., 2005, Schofield et al., 2016, Shelton, 2009). This thesis does not compare Scotland with England and Wales while controlling for socioeconomic deprivation but it acknowledges that the questions this body of research has raised are important. Similar to studies

explicitly concerned with the 'Scottish effect' this thesis draws attention to Scotland's premature mortality problem. Similar to the 'Scottish effect' literature, this thesis recognises that the determinants of population health are too complex to be accounted for by one single theory. However, this thesis does support the explanations that seeks to understand differences in Scotland, in terms of structural changes, that could explain the aetiological pathways leading to the higher risk of premature death that the population experiences (McCartney et al., 2012a, Walsh et al., 2010). Although it is a weakness that no definitive conclusions can be drawn from this thesis there are a number of strengths worthy of highlighting.

9.3 Strengths and limitations

The findings presented in this thesis provide new insights into the full extent, and changing nature, of mortality inequalities in Scotland by estimating and decomposing lifespan variation. The data used and analyses applied were appropriate for answering the relevant research questions: different research questions may have demanded different data and analyses.

9.3.1 Lifespan variation estimates from complete period life tables

Lifespan variation estimates in this thesis were calculated from complete period life tables. A lifetable provides a wealth of information about the mortality experience of the given population making it a vital tool for carrying out research (Wunsch et al., 2013). The population included in a period life table can be understood as a synthetic or hypothetical population because it models what would happen to a population if a given set of mortality conditions were experienced (Wunsch et al., 2013, Preston et al., 2001). The use of complete

life tables (e.g. single year of age) in this thesis is a major strength: they are a more precise representation of the mortality experience of interest than abridged life tables (Preston et al., 2001).

Lifespan variation in this thesis was reported from age zero. However, it has been argued that health inequalities in developed countries may be better reflected when reporting lifespan variation from adult ages only as infant deaths are now rarer than in the past (Smits and Monden, 2009). Existing studies have therefore reported lifespan variation conditional upon survival to older ages when stratifying the population by education or occupation (van Raalte et al., 2012, van Raalte et al., 2014). It is only possible to report lifespan variation conditional upon survival to adulthood when using occupation or education to measure socioeconomic deprivation because information on completed years of education or achieved occupation is required. However it does mean that there is something of a trade-off between capturing the full age distribution of mortality and applying a measure of socioeconomic deprivation. It is also a departure from the standard reporting of life expectancy from age 0 which is used to make international comparisons (World Health Organization, 2010).

This thesis utilised an area level measure of socioeconomic deprivation which allows the full age distribution of death to be captured and enables the reporting of lifespan variation from age 0 which is consistent with the reporting of the more commonly understood concept of life expectancy. It was also appropriate within the context of Scotland as premature mortality rates for males age 15-29 years old increased between 1981 and 2001 (Leyland et al., 2007a). This age pattern of mortality that would have been truncated out of the

results had measures of occupation or education been applied and lifespan variation conditional upon survival to adulthood reported.

9.3.2 Routine data

The secondary data utilised for estimating lifespan variation are considered robust as they were obtained from two highly regarded sources: the HMD and NRS.

The HMD collects vital events statistics and population estimates from a range of countries and applies a standard procedure for constructing period life tables, thus making it suitable for cross national comparative research and the most suitable data source for use in chapters 4 through 6 of this thesis. The lifetable data provided by the Human Mortality Database aims to facilitate the identification of changes to population longevity (Wilmoth et al., 2007) and enabled this thesis to hypothesise about some of the causes and consequences in Scotland (e.g. increasing variation in age at death (Gillespie et al., 2014)). However, the HMD lifetable data does not permit the study of population sub-groups meaning that an alternative data source had to be identified in order to study the effects of socioeconomic deprivation on lifespan variation in Scotland.

Fortunately there are number of alternative data options available for studying the relationship between socioeconomic deprivation and health inequalities in Scotland, some of which were the focus of critical evaluation in chapter 3. This thesis utilised the Carstairs score of area level deprivation because it met the following criteria that were set out in chapter 3: the conceptual and theoretical framework of the Carstairs score was relevant to the research questions, the Carstairs score was available for an extended time period and reflected a

consistent meaning of deprivation over time, and it was possible to match the Carstairs score with population estimates and death data obtained from NRS. Although the Carstairs score was deemed to be the most appropriate measure for use within this thesis it is not without limitation.

9.3.3 Reflection on the Carstairs score

It has been argued that the Carstairs score may be an out of date measure of socioeconomic deprivation (Schofield et al., 2016, Tunstall et al., 2011) and the relevance of the variables used for capturing the meaning of deprivation across contexts and over time has been questioned (Norman, 2010). For example the meaning of car ownership is fundamentally different for individuals in rural contexts compared to urban contexts. It is also acknowledged that overcrowding may occur out of choice and for cultural reasons rather than simply being a marker of deprivation (Fischbacher, 2014).

While still acknowledging these limitations the Carstairs score has a number of strengths. Firstly, it is a measure of deprivation in Scotland that is available covering a thirty year time period (Brown et al., 2014). Secondly, the Carstairs score has a strong theoretical framework and was constructed specifically in order to understand mortality inequalities (Carstairs and Morris, 1989a). The four variables used (overcrowding, male unemployment, no car ownership and low social class) were perceived to be a summary measure of population deprivation suitable for constructing relative measure of area level deprivation (Carstairs and Morris, 1990). Thirdly, the scores for each postcode sector at each Census year are highly correlated despite changes to the formal definitions of the variables. This indicates that the underlying information the variables aim to

capture is similar or that deprivation has remained stable over time (Leyland et al., 2007a).

It is important to acknowledge that the use of an area level measure of deprivation means the results may be subject to the ecological fallacy: the extent of an association found at the area level may differ from extent of an association found at the individual level (Diez, 2002). Despite this, area level measures of deprivation remain an invaluable, pragmatic tool for research (Bailey et al., 2003a) and they are a means for governments to identify how resources should be distributed (Allik et al., 2016, The Scottish Government and National Statistics, 2012).

9.3.4 Excluded deaths

Although this thesis utilised routinely collected data some death counts could not be included when constructing lifetables for chapter 7 and chapter 8 in this thesis. Deaths had to be excluded if they did not contain sufficient information on sex, age or postcode sector. These missing deaths represented a very small proportion of the total number of deaths and it is unlikely that they would have impacted the substantive results that have been reported.

9.3.5 Predicting mortality rates

A further restriction of the data used in chapter 7 and chapter 8 was the final open ended age category of the Census population estimates was age 85+. In order for the lifespan variation estimates to be consistent with those calculated from the HMD (chapter 4 through 6) a final open ended age category of 110+ was required. This limitation was overcome by predicting the mortality rates as a function of age for age ≥85. Three modelling approaches were tested and the

HMD mortality rates used as an external comparator. The final model applied was a Poisson regression which included a fractional polynomial function for age for predicting the morality rates for ages ≥85, by sex and deprivation quintile.

9.3.6 Random fluctuations

Chapter 7 permitted the population estimates of lifespan variation stratified by socioeconomic deprivation to be statistically evaluated by carrying out 1,000 Monte Carlo simulations to produce 95% confidence intervals. This is important to consider because lifespan variation is calculated from mortality rates and the numbers of deaths in the population are inherently vulnerable to random, naturally occurring fluctuations. Therefore the number of deaths that occur in a population can be interpreted as one possible series that could have occurred under the given set of circumstances (Curtin and Klein, 1995). This was a weakness of the estimates utilised in chapter 4 through 6, for which 95% confidence intervals were not produced.

Producing 95% confidence intervals for the 2,076 lifespan variation estimates used in chapter 4 was beyond the scope of this project. Although this would have allowed the results in chapter 4 to have been interpreted with more certainty it is valid to interpret the estimates as robust for the following two reasons. Firstly, the confidence intervals produced in chapter 7 were narrow and were based on smaller population sizes. Secondly, the lifespan variation estimate calculated for the population of Scotland, from the HMD lifetable data, did not fall outside the distribution of the lifespan variation estimates for each quintile of deprivation.

The joinpoint regression analysis applied in chapter 4 produced 95% confidence intervals for the timing of any significant change in trend and 95% confidence intervals for the APC change for each segment of the regression line. It is unlikely that carrying out the joinpoint regression analysis on upper and lower lifespan variation estimates would have changed these substantive conclusions.

9.3.7 Potential for error in cause of death categories

The analysis in chapter 8 constructed cause specific categories of death from ICD codes. The categories created were external, cancers, circulatory diseases, respiratory diseases and all other. Grouping deaths by different diagnoses allows patterns to be studied. However it is worth noting that processes involved in coding of deaths and the construction of broad cause of death categories are vulnerable to error (O'Malley et al., 2005). How deaths are classified for statistical purposes is also an administrative process that can be subject to change over time (National Records of Scotland, 2016a). The potential for these issues to have impacted the substantive results is minimal: the ICD-9 and ICD-10 codes were harmonised so that the causes of death assigned to a category were consistent over time and time trends in cause specific mortality could be interpreted despite the ICD change (Rooney and Smith, 2000, Rooney et al., 2002). The construction of categories was also informed by, but is not identical, to those already used in published research (Leyland et al., 2007b, van Raalte et al., 2014).

It was not possible to produce cause-specific mortality rates for ages ≥85 because Census population estimates were only available up to an open ended age interval of 85+. Therefore a smoothing process was required in order for the

lifespan variation estimates to be consistent with the estimates in chapter 4 through 7. The cause specific mortality rate at age 84 was carried forward to ascertain the proportion of causes for ages ≥85. This approach was previously applied by van Raalte et al. (2014). The sum of the cause specific mortality rates for ages ≥85 was always equal to the all-cause mortality rate that was used in chapter 7.

9.4 Future research directions

This thesis has used mortality data, as an indicator of health, to identify systematic inequalities in age at death (lifespan variation) that exist within Scotland. Lifespan variation reflects the novel concept of total inequality, an area of research that requires further attention. The following subsection explores the potential for subjective measures of self-reported health to be incorporated into measures of total inequality.

The literature review of this thesis began by debating different interpretations and definitions of health. Early concepts were focused on health being the absence of disease and definitions of health were structured in response to the need to control and understand communicable diseases (World Health Organisation, 1948). Mortality data provides researchers with a robust, proximal measure for health under this interpretation and has a number of pragmatic advantages. Firstly, it is a legal requirement for a death to be recorded in most developed countries meaning that long running, routinely collected data are available for entire populations rather than for samples of the population. Secondly, mortality data reflects an absolute state of being that cannot change.

Over time definitions of health have evolved to consider the subjective experience of health in both the presence and absence of disease and the notion of well-being (Arcaya et al., 2015, Jadad and O'Grady, 2008). Mortality data remains an appropriate indicator for health in these terms: although individual deaths are clinically defined events the age and cause specific expectations surrounding death have changed over time and vary between populations meaning death is relative in nature. The social patterning of death is also reflective of complex social phenomenon beyond that of clinical health care.

The counter argument to this is that death inadequately reflects the complexities of health because it is a single, irreversible, absolute state (Huber et al., 2011, Walley et al., 2001). Declining death rates and changes to the disease profile in many developed countries mean clinical definitions of health are no longer enough to capture the full extent of worsening health inequalities in societies which are increasingly characterised by chronic disease as opposed to the risk of premature death (Mitchell, 2005).

Studies measuring inequalities in self-reported health have emerged in response and demonstrate that differences in average self-reported health between populations have also widened over time (Kunst et al., 2005). However, it is important to note that self-reported measures have limitations. Mitchell (2005) questions the comparability of self-reported measures of health given that the meaning of illness, the willingness to report illness, and the level of awareness and expectations surrounding health have changed over time and differ between countries. Johnston et al. (2009) add that reporting error in self-report measures of health might result in an under estimation of the health gradient

compared to that found when using objective measures of health. Therefore death may still be considered a more accurate indicator of actual health rather than perceived health because we can be more certain about the elements of poor health that precede a death (Green, 2013, Mitchell, 2005).

This debate suggests that objective and subjective measures of health are mutually exclusive approaches, which is not the case. Healthy life expectancy (also referred to as healthy life years or disability free life expectancy) is an empirical measurement that incorporates death data and self-reported health data in a life table. Healthy life expectancy indicates the average number of years a person can be expected to live for free from illness and can be used to indicate differences in health status that people experience while they are still alive (Wood et al., 2006). Average levels of healthy life expectancy are now being used across Europe to monitor population health, quality of life and evaluate the health of an ageing workforce (European Commission, 2016, Salomon et al., 2012). A number of approaches exist for estimating the proportion of the population in the life table that are healthy. For example using cross sectional data on limiting long term illness, life in good self-assessed health, or years of life lived without a disability (Wood et al., 2006, Robine et al., 1998).

In Scotland a male born in 2015 is expected to live for 76.9 years, 59.9 of which would be in a healthy state. A female born in Scotland in 2015 is expected to live for 81.0 years and 62.3 years of this would be in a healthy state (ScotPHo, 2016). Healthy life expectancy in Scotland also demonstrates a strong socioeconomic gradient: years of life expected to be spent in a healthy state are

lowest for the most deprived. An important finding is that the gradient for healthy life expectancy is steeper than the gradient for life expectancy. The difference in healthy life expectancy, between the most and least deprived in Scotland, is 2.5 times greater than the difference in average life expectancy (Wood et al., 2006). It is not clear what level of variation in healthy life expectancy exists in Scotland or how variation in healthy life expectancy is experienced by different socioeconomic groups. This could be the focus of future research and help inform our understanding of how morbidity inequalities have changed in response to changes in the mortality profile of the population.

9.5 Conclusion

To summarise, this thesis has identified that both males and females in Scotland experience the highest levels of total inequality, as measured by lifespan variation, in Western Europe. The lifespan variation trend for females in Scotland has not converged with the rest of Western Europe, although it has consistently been decreasing since 1950. The lifespan variation trend for males in Scotland was decreasing up until the 1980s but then diverged; it has yet to return to a decreasing trajectory meaning that it is the longest, sustained increasing trend in Western Europe. Increasing total inequality in Scotland is a dimension of mortality change that would have gone undetected when only looking at the average mortality experience of a population.

Premature mortality in Scotland accounts for the high level of lifespan variation and the unfavourable trends it has experienced. This age pattern of mortality change in Scotland is distinguishable from England and Wales, and also accounts

for the lifespan variation gap when these comparable countries were achieving a similar level of life expectancy.

Higher lifespan variation in Scotland is unlikely due to it being at a different stage of the epidemiological transition. It is perhaps more likely to be an indication of an inadequate social safety net for ensuring all members of the population can access the primary goods required for living (van Raalte, 2011a, Vallin and Meslé, 2004, Tuljapurkar, 2010, Gillespie et al., 2014), with good health being one the most fundamental requirements (Fabienne, 2001, Sen, 2002). This is further evidenced by the finding that Scotland's deteriorating population level lifespan variation ranking within Western Europe is associated with widening lifespan variation inequalities between socioeconomic groups within Scotland. The least deprived have experienced decreasing lifespan variation but the most deprived have experienced increasing lifespan variation, causing the socioeconomic gradient for lifespan variation in Scotland to steepen over time. Although there has been some recent improvement the socioeconomic gradient for lifespan variation in 2011 was steeper than in 1981. Premature deaths from external causes explained a greater proportion of lifespan variation inequalities within Scotland in 2011 than in 1981.

Measuring lifespan variation in Scotland provides a more detailed account of the changing nature of mortality inequalities: age at death within Scotland is more unequal today than it was 30 years ago despite improvements in average life expectancy. Routine measurement of lifespan variation is required to ascertain whether increases in life expectancy and reductions in total inequality are being achieved simultaneously: these public health goals are not incompatible.

Appendices

Appendix 1 Pre-copyedited, author-produced PDF of an article accepted for publication in The European Journal of Public Health following peer review.

This is a pre-copyedited, author-produced PDF of an article accepted for publication in The European Journal of Public Health following peer review. The version of record 'Seaman, R., Leyland, A.H., & Popham, F. (2016). How have trends in lifespan variation changed since 1950? A comparative study of 17 Western European countries. The European Journal of Public Health, 26, 360-362' is available online at: http://eurpub.oxfordjournals.org/content/26/2/360.abstract

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Short Report

How have trends in lifespan variation changed since 1950? A comparative study of 17 Western European countries

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Lifespan variation adds to life expectancy by measuring the inequality surrounding age of death that a population faces. Countries that tackle premature mortality generally have decreasing lifespan variation but this is the first study to compare and statistically assess when and to what extent trends in lifespan variation have changed across Western Europe. Lifespan variation was measured using $e^\dagger$ and joinpoint regression analysed the timing and rate of change. Trends have been mostly downward with the recent exception of men in Scotland, Northern Ireland, Ireland and Finland where trends have flattened or show slight increases. Future research aimed at identifying the ages and causes of death, driving trends in these countries, is key to preventing increasing inequalities.

Introduction

Research has established life expectancy and mortality differences between Western European countries[1,2] and has now started to explore lifespan variation differences.[3,4] Lifespan variation adds to life expectancy by summarising the inequality in age of death. Decreasing lifespan variation suggests decreasing uncertainty surrounding average lifespan as deaths are compressed around a common age. This is important as lifespan variation may not change even if life expectancy is increasing. For example, if a population has reduced mortality at older ages faster than it has reduced premature mortality it will have achieved increases in life expectancy but not mortality compression.[3] Therefore, measuring life expectancy and lifespan variation helps to demonstrate the extent to which improvements in average population health and reductions in mortality inequality have been achieved simultaneously.[5,6]

The Western European countries which have achieved the highest life expectancy and lowest lifespan variation are generally those which have tackled premature mortality. Scotland[4] and Finland[6] are two countries where lifespan variation may have changed to be stagnating or even increasing despite improvements in life expectancy. However, it is not clear whether these changes in trends for lifespan variation are significant or when they began exactly. It is also not clear if similar changes in trends for lifespan variation have been seen in other Western European countries. These are important research questions to answer for two reasons, firstly to identify countries that are failing to reduce inequalities to the same extent as comparable countries, and secondly to identify when any changes in trends may have started in order to inform future studies into the causes of mortality inequalities. Using trend analysis techniques we explore trends in lifespan variation in 17 Western European countries from 1950 onwards.

Methods

The Human Mortality Database (HMD) provides annual, sex specific lifetables for individual countries from which we calculated lifespan variation. Seventeen Western European countries that had data available and which have been used in existing research were identified.[2] We used lifetables from 1950 to 2011, although some countries had data only from the mid-1950s (see Supplementary table for exact years).

Several measures of lifespan variation exist and are highly correlated (technical summary available elsewhere[7]). We chose $e^\dagger$ as it covers the whole age range. It is interpreted as the average number of years of life lost per death, giving it an intuitive meaning. It is calculated by summing remaining life expectancy at each age weighted by the proportion of deaths at that age, and was calculated for each year, sex and country using Stata SE13.

Once $e^\dagger$ was calculated the data were imported into the joinpoint regression programme 4.0.4.[8] Joinpoint regression identifies and quantifies changes to trends, and calculates the time points at which a change in trend is statistically significant by testing whether a multi-segmented line is a significantly better fit than a straight or less-segmented line. It is used to evaluate both the time point and the level of change across a time series for any given health outcome. Detailed information on the joinpoint regression programme is given elsewhere.[8] We modelled the log of lifespan variation in order to calculate annual percentage change (APC).

Results

Figure 1 shows the modelled trends for lifespan variation for men in all 17 Western European countries, grouped by geographic area. The equivalent graph for women is provided as a Supplementary figure.

Men

Since 1950 there has been a general decreasing trend for lifespan variation amongst men in Western Europe with some increases for short periods before returning to a downward trend. Rapid decreases in trends were found in the years immediately after 1950 with the steepest decline of −4.1% (95% CI −5.0 to −3.2) found in Spain between 1950 and 1954. However more recently, Scotland, Northern Ireland, Ireland and Finland have changed to a flattening or slightly increasing trend that has yet to be reversed.

Appendix 2 Pre-copyedited, author-produced PDF of an article accepted for publication in Social Science and Medicine – Population Health following peer review.

This is a pre-copyedited, author-produced PDF of an article accepted for publication in Social Science and Medicine – Population Health following peer review. The version of record 'Seaman, R., Leyland, A.H., & Popham, F. (2016). Increasing inequality in age at death at shared levels of life expectancy: A comparative study of Scotland and England and Wales. *SSM - Population Health*, 2, 724-731' is available online at:

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Article

Increasing inequality in age of death at shared levels of life expectancy: A comparative study of Scotland and England and Wales

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ABSTRACT

There is a strong negative correlation between increasing life expectancy and decreasing lifespan variation, a measure of inequality. Previous research suggests that countries achieving a high level of life expectancy later in time generally do so with lower lifespan variation than forerunner countries. This may be because they are able to capitalise on lessons already learnt. However, a few countries achieve a high level of life expectancy later in time with higher inequality. Scotland appears to be such a country and presents an interesting case study because it previously experienced lower inequality when reaching the same level of life expectancy as its closest comparator England and Wales. We calculated life expectancy and lifespan variation for Scotland and England and Wales for the years 1950 to 2012, comparing Scotland to England and Wales when it reached the same level of life expectancy later on in time, and assessed the difference in the level of lifespan variation. The lifespan variation difference between the two countries was then decomposed into age-specific components. Analysis was carried out for males and females separately. Since the 1950s Scotland has achieved the same level of life expectancy at least ten years later in time than England and Wales. Initially it did so with lower lifespan variation. Following the 1980s Scotland has been achieving the same level of life expectancy later in time than England and Wales and with higher inequality, particularly for males. Decomposition revealed that higher inequality is partly explained by lower older age mortality rates but primarily by higher premature adult age mortality rates when life expectancy is the same. Existing studies suggest that premature adult mortality rates are strongly associated with the social determinants of health and may be amenable to social and economic policies. So addressing these policy areas may have benefits for both inequality and population health in Scotland.

1. Background

There is a strong association between life expectancy and lifespan variation. Life expectancy reflects the average length of life in the population and lifespan variation reflects the variability surrounding the length of life (van Raalte et al., 2011). In any given year, countries with higher life expectancy have lower lifespan variation (Smits & Monden, 2009; Vaupel, Zhang & van Raalte, 2011). Lifespan variation is regarded as a measure of inequality and so there is an association between improving average population health and reducing inequality (Shkolnikov, Andreev, Zhang, Oeppen & Vaupel, 2011; van Raalte et al., 2011).

1.1. Compression and expansion

Reducing the mortality rate at any age will increase life expectancy but improvements in lifespan variation are only achieved if reductions in premature mortality rates are greater than reductions in older age mortality rates. This is because reducing premature mortality rates compresses the age distribution of death while reducing older age mortality rates expands the distribution (Oeppen, 2008; Smits & Monden, 2009; Vaupel et al., 2011). Therefore the desired negative correlation between increasing life expectancy and decreasing lifespan variation is contingent upon more compression than expansion (Shkolnikov et al., 2011). Hence countries reaching the same level of life expectancy at different times can do so with underlying differences in age specific mortality rates, resulting in different levels of lifespan variation (Anger et al., 2014; Nau & Firebaugh, 2012; Shkolnikov et al., 2011).

1.2. Lifespan variation differences independent of life expectancy

Given the strong correlation between life expectancy and lifespan variation Smits and Monden (2009) argue that cross national compar-

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Appendix 3 Annual e† estimates for Western European countries 1950-2012 – males

year	Scot	Eng&Wales	N.Ire	Denmark	Finland	Norway	Sweden	Belgium	France	Ireland	Nether.	Austria	W.Ger	Switz	Italy	Portugal	Spain
1950	14.39	13.39	14.71	13.30	16.05	13.66	12.70	15.72	15.75	15.43	13.01	16.62		14.09	16.69	20.85	18.76
1951	14.07	13.13	14.41	13.35	15.04	13.76	12.64	15.20	15.41	15.04	13.01	16.07		13.88	16.39	20.28	18.06
1952	13.84	13.07	14.43	13.34	14.53	13.41	12.57	14.67	15.15	14.86	12.79	15.36		13.76	16.06	20.31	17.31
1953	13.84	12.91	14.37	13.07	14.48	13.15	12.60	14.33	14.67	14.37	12.89	15.03		13.62	15.60	19.65	16.41
1954	13.57	12.69	13.56	12.99	14.28	13.00	12.38	14.38	14.78	14.17	12.60	15.06		13.45	15.49	18.71	16.01
1955	13.42	12.55	13.32	12.86	14.16	13.01	12.33	14.26	14.56	13.74	12.45	15.01		13.31	15.32	19.14	16.11
1956	13.20	12.44	13.00	12.85	13.99	12.82	12.31	14.17	14.25	13.85	12.25	14.90	14.17	13.22	14.75	18.35	15.28
1957	13.41	12.63	13.14	12.72	14.06	12.96	12.27	14.03	14.29	13.65	12.32	15.15	14.19	13.24	15.18	19.03	15.54
1958	13.11	12.40	13.01	12.53	13.64	12.74	11.99	13.64	13.97	13.47	12.23	14.66	14.00	13.27	15.04	18.48	15.15
1959	13.13	12.43	12.92	12.62	13.81	12.82	12.08	13.72	13.96	13.38	12.45	14.69	13.99	13.12	14.97	18.69	15.03
1960	13.15	12.51	13.17	12.47	13.51	12.68	11.96	13.57	13.58	13.23	12.23	14.29	13.74	12.95	14.63	17.75	14.65
1961	12.95	12.40	13.14	12.55	13.57	12.62	11.97	13.60	13.75	13.05	12.43	14.10	13.87	13.16	14.65	18.87	14.88
1962	13.10	12.37	12.75	12.40	13.27	12.68	11.85	13.29	13.53	13.14	12.30	13.65	13.50	12.96	14.49	17.94	14.25
1963	12.86	12.27	12.96	12.41	13.31	12.37	11.87	13.23	13.49	12.90	12.16	13.62	13.25	12.79	14.44	17.10	14.25
1964	12.97	12.54	13.06	12.36	13.21	12.55	11.96	13.39	13.56	13.09	12.51	13.70	13.45	12.85	14.26	17.22	14.10
1965	12.82	12.40	13.08	12.43	13.15	12.64	11.78	13.12	13.35	12.80	12.26	13.33	13.09	12.38	13.94	16.57	13.97
1966	12.77	12.29	12.77	12.16	12.87	12.23	11.81	13.11	13.53	12.46	12.33	13.64	13.17	12.48	13.87	16.51	13.78
1967	12.86	12.29	12.85	12.27	12.83	12.39	11.82	13.00	13.35	12.87	12.45	13.43	13.12	12.68	13.70	16.25	13.69
1968	12.76	12.06	12.61	12.38	12.76	12.30	11.80	12.73	13.29	12.52	12.22	13.27	12.83	12.27	13.56	16.16	13.44
1969	12.64	12.20	12.96	12.31	12.88	12.36	11.78	12.89	13.31	12.35	12.28	13.27	12.98	12.44	13.67	15.68	13.33
1970	12.60	12.19	12.76	12.46	12.89	12.32	11.91	12.90	13.26	12.43	12.25	13.30	13.08	12.50	13.45	16.04	13.32
1971	12.67	12.19	12.67	12.42	12.92	12.30	11.88	12.81	13.23	12.52	12.17	13.45	13.11	12.41	13.39	15.74	12.92
1972	12.34	11.98	12.94	12.39	13.03	12.18	11.90	12.72	13.27	12.41	12.08	13.42	13.02	12.40	13.34	14.71	12.82
1973	12.50	12.01	13.38	12.24	12.85	12.12	11.63	12.52	13.07	12.36	12.00	13.27	12.89	12.18	12.99	14.85	12.69

1974	12.44	11.95	13.09	12.14	12.78	12.13	11.65	12.49	13.03	12.32	12.03	13.23	12.76	12.14	12.79	14.40	12.59
1975	12.37	11.82	13.01	12.20	12.76	12.05	11.64	12.22	12.89	12.32	11.74	12.91	12.68	12.08	12.57	15.00	12.46
1976	12.11	11.66	12.85	12.03	12.61	11.86	11.48	12.21	12.90	11.91	11.73	12.66	12.57	11.88	12.43	14.42	12.38
1977	12.36	11.73	12.74	12.12	12.59	11.86	11.61	12.32	12.94	11.94	11.90	12.77	12.49	12.06	12.34	14.07	12.30
1978	12.10	11.73	12.56	12.02	12.37	11.73	11.58	12.27	12.73	12.05	11.72	12.58	12.39	11.94	12.30	13.88	12.34
1979	12.02	11.64	12.45	12.01	12.41	11.79	11.53	12.13	12.79	11.92	11.62	12.65	12.31	11.84	12.20	13.78	12.35
1980	11.96	11.56	12.28	11.98	12.13	11.80	11.39	12.11	12.80	11.69	11.57	12.61	12.22	11.87	12.13	13.75	12.09
1981	11.93	11.46	12.09	11.91	12.23	11.49	11.24	11.99	12.63	11.63	11.42	12.37	12.01	11.74	11.97	13.69	12.05
1982	11.81	11.37	12.09	12.02	12.16	11.72	11.21	11.93	12.69	11.57	11.39	12.54	11.96	11.78	11.96	13.53	11.98
1983	11.72	11.30	11.94	11.95	11.87	11.60	11.12	11.78	12.54	11.50	11.28	12.31	11.81	11.72	11.74	13.32	11.87
1984	11.78	11.32	11.82	11.93	12.09	11.41	11.18	11.81	12.56	11.37	11.31	12.42	11.70	11.68	11.79	13.18	11.94
1985	11.55	11.13	11.73	11.98	11.79	11.57	11.02	11.69	12.42	11.15	11.13	12.15	11.55	11.53	11.62	13.07	11.79
1986	11.60	11.21	11.66	11.98	12.12	11.81	11.08	11.62	12.44	11.02	11.12	12.06	11.52	11.57	11.56	13.09	11.94
1987	11.59	11.31	11.70	12.12	12.11	11.79	11.05	11.82	12.44	11.08	11.13	11.99	11.52	11.64	11.63	13.01	12.11
1988	11.80	11.29	11.52	11.89	12.16	11.81	10.99	11.68	12.49	11.14	11.03	11.84	11.51	11.63	11.59	12.94	12.07
1989	11.37	11.12	11.31	11.97	12.25	11.69	11.02	11.49	12.50	11.15	10.86	11.83	11.38	11.61	11.61	12.88	12.21
1990	11.62	11.23	11.34	11.76	12.10	11.54	10.88	11.53	12.46	10.99	10.91	11.78	11.34	11.47	11.63	12.71	12.16
1991	11.57	11.10	11.42	11.79	12.16	11.40	10.85	11.57	12.54	11.13	10.82	11.82	11.38	11.70	11.68	12.95	12.22
1992	11.48	11.03	11.13	11.58	11.86	11.13	10.69	11.53	12.52	10.91	10.79	11.72	11.36	11.68	11.70	12.90	12.27
1993	11.27	10.86	11.18	11.35	11.59	10.79	10.52	11.43	12.42	10.76	10.63	11.67	11.25	11.40	11.46	12.58	12.08
1994	11.50	10.96	11.28	11.50	11.75	10.85	10.61	11.53	12.42	10.83	10.63	11.74	11.25	11.43	11.42	12.50	12.06
1995	11.48	10.87	11.22	11.32	11.62	10.71	10.40	11.44	12.23	10.77	10.56	11.58	11.20	11.26	11.43	12.64	12.01
1996	11.55	10.90	10.80	11.37	11.56	10.69	10.25	11.24	12.08	10.83	10.52	11.44	11.12	11.01	11.33	12.57	11.98
1997	11.51	10.86	10.91	11.25	11.54	10.60	10.27	11.18	11.94	10.84	10.44	11.25	11.09	10.86	11.12	12.49	11.65
1998	11.67	10.84	10.86	11.16	11.43	10.75	10.21	11.20	11.84	10.93	10.44	11.18	10.96	10.83	10.98	12.39	11.44
1999	11.51	10.80	10.82	11.06	11.43	10.56	10.12	11.24	11.77	10.74	10.34	11.15	10.93	10.68	10.90	12.19	11.35
2000	11.66	10.80	10.84	11.14	11.40	10.69	10.10	11.23	11.77	10.90	10.36	11.24	10.94	10.77	10.82	12.10	11.43

2001	11.73	10.77	10.81	10.97	11.32	10.57	10.12	11.20	11.79	10.81	10.34	11.16	10.92	10.67	10.83	12.06	11.34
2002	11.79	10.73	11.01	10.96	11.19	10.42	9.97	11.06	11.69	10.80	10.24	10.98	10.82	10.56	10.75	11.85	11.29
2003	11.48	10.66	10.66	10.93	11.25	10.38	10.01	10.90	11.56	10.64	10.15	11.00	10.77	10.32	10.40	11.58	11.15
2004	11.63	10.67	10.87	10.93	11.51	10.38	9.99	10.86	11.56	10.62	10.16	11.03	10.77	10.46	10.52	11.68	11.15
2005	11.51	10.61	11.21	10.80	11.51	10.23	9.85	10.83	11.46	10.43	10.00	10.93	10.69	10.25	10.33	11.43	11.00
2006	11.77	10.75	11.09	10.89	11.37	10.27	9.87	10.88	11.49	10.50	9.96	10.77	10.63	10.19	10.33	11.49	11.03
2007	11.70	10.71	11.10	10.90	11.47	9.99	9.83	10.93	11.42	10.58	9.89	10.82	10.64	10.08	10.21	11.31	10.91
2008	11.72	10.67	10.94	10.75	11.41	10.02	9.79	10.87	11.34	10.72	9.86	10.62	10.53	10.01	10.23	11.15	10.83
2009	11.63	10.67	11.06	10.52	11.25	10.14	9.76	10.75	11.36	10.87	9.91	10.78	10.52	10.08	10.07	11.17	10.74
2010	11.50	10.58	11.25	10.55			9.71	10.76	11.33			10.78	10.54	9.79		10.93	
2011	11.50	10.56	10.90	10.42			9.64	10.77	11.29				10.56	9.84		11.05	
2012								10.65	11.12							10.75	

Appendix 4 Annual e† estimates for Western European countries 1950-2012 – females

year	Scot	Eng&Wales	N.Ire	Denmark	Finland	Norway	Sweden	Belgium	France	Ireland	Nether.	Austria	W.Ger	Switz	Italy	Portugal	Spain
1950	13.81	12.67	13.91	12.08	13.88	12.17	11.58	14.11	14.45	15.21	11.89	15.10		12.68	15.62	20.27	18.14
1951	13.32	12.33	13.59	12.03	13.10	12.12	11.52	13.59	14.11	14.43	11.89	14.34		12.43	15.34	19.56	17.39
1952	13.22	12.28	13.42	11.97	12.87	11.98	11.39	13.27	13.88	14.56	11.75	13.72		12.34	14.93	19.63	16.65
1953	12.89	12.17	13.36	11.77	12.47	11.50	11.26	12.88	13.25	14.10	11.84	13.45		12.05	14.44	18.70	15.66
1954	12.66	11.98	12.65	11.60	12.27	11.31	11.05	12.92	13.38	13.52	11.49	13.38		11.99	14.17	17.79	15.28
1955	12.50	11.76	12.71	11.77	12.02	11.25	11.19	12.62	13.06	13.17	11.25	13.21		11.89	13.89	18.38	15.17
1956	12.11	11.62	12.30	11.56	11.96	11.18	10.98	12.40	12.68	13.12	10.93	13.03	12.47	11.55	13.31	17.41	14.38
1957	12.30	11.89	12.42	11.40	12.26	11.04	10.87	12.44	12.69	13.07	11.08	13.09	12.42	11.56	13.76	18.10	14.62
1958	12.21	11.56	12.03	11.18	11.60	11.04	10.79	12.09	12.35	12.75	10.98	12.84	12.29	11.38	13.55	17.33	14.08
1959	12.09	11.59	12.23	11.33	11.58	10.94	10.72	12.09	12.31	12.68	10.92	12.85	12.31	11.28	13.48	17.51	13.99
1960	12.01	11.55	12.26	11.13	11.20	10.83	10.70	11.76	12.00	12.57	10.81	12.43	12.05	11.04	13.09	16.43	13.49
1961	11.88	11.49	11.53	11.27	11.18	10.73	10.64	11.79	12.01	12.41	10.77	12.22	12.11	11.37	12.92	17.72	13.71
1962	12.04	11.48	12.09	11.06	10.93	10.64	10.47	11.70	11.79	12.32	10.65	12.08	11.84	11.16	12.81	16.55	13.14
1963	12.03	11.42	12.18	11.13	11.01	10.41	10.57	11.54	11.79	12.11	10.59	11.99	11.64	10.96	12.72	15.80	12.96
1964	12.19	11.66	11.87	10.96	10.79	10.64	10.67	11.70	11.80	12.33	10.79	11.94	11.76	11.05	12.55	15.87	12.91
1965	12.05	11.54	11.93	11.13	10.56	10.63	10.52	11.55	11.63	11.99	10.67	11.71	11.54	10.81	12.21	15.07	12.60
1966	11.94	11.46	11.92	10.85	10.69	10.43	10.37	11.54	11.68	11.88	10.65	11.76	11.53	10.77	12.21	14.93	12.52
1967	11.92	11.53	12.03	10.96	10.62	10.52	10.38	11.40	11.57	12.00	10.76	11.52	11.47	10.77	12.02	14.53	12.32
1968	11.69	11.30	11.92	11.13	10.45	10.42	10.36	11.16	11.50	11.77	10.57	11.47	11.25	10.68	11.84	14.52	12.06
1969	11.92	11.49	11.88	11.22	10.53	10.67	10.37	11.27	11.59	11.86	10.73	11.54	11.48	10.80	12.08	14.07	12.05
1970	11.85	11.44	11.93	11.18	10.46	10.25	10.54	11.32	11.40	11.84	10.64	11.48	11.41	10.63	11.79	14.39	11.74
1971	11.90	11.42	11.65	11.27	10.39	10.26	10.52	11.18	11.30	11.60	10.47	11.38	11.41	10.59	11.66	13.95	11.59
1972	11.70	11.34	11.85	11.11	10.27	10.19	10.41	11.19	11.34	11.57	10.42	11.45	11.32	10.53	11.61	12.91	11.41
1973	11.72	11.25	11.79	11.08	10.24	10.10	10.32	11.00	11.13	11.47	10.57	11.27	11.27	10.35	11.30	13.21	11.15

1974	11.77	11.22	11.57	10.85	10.33	10.09	10.36	10.94	11.02	11.25	10.46	11.23	11.17	10.36	11.08	12.51	11.01
1975	11.69	11.18	11.80	11.08	10.31	10.12	10.32	10.80	10.93	11.32	10.34	11.02	11.10	10.32	10.87	12.80	10.93
1976	11.44	10.96	11.86	10.87	10.28	10.12	10.19	10.78	10.81	11.06	10.36	10.75	10.97	10.11	10.77	12.38	10.74
1977	11.71	11.06	11.56	10.94	10.19	10.05	10.20	10.91	10.83	11.18	10.49	10.83	10.91	10.25	10.56	12.09	10.65
1978	11.62	11.14	11.66	10.96	10.05	10.06	10.16	10.74	10.68	11.12	10.39	10.51	10.81	10.10	10.55	11.77	10.64
1979	11.58	11.01	11.40	10.92	10.17	9.90	10.19	10.86	10.64	10.94	10.39	10.64	10.73	10.25	10.47	11.68	10.55
1980	11.51	11.00	11.42	11.04	9.87	9.90	10.09	10.71	10.58	10.91	10.34	10.56	10.63	10.10	10.41	11.59	10.32
1981	11.29	10.85	11.16	10.99	9.85	9.81	9.92	10.59	10.43	10.74	10.27	10.37	10.48	9.98	10.28	11.46	10.19
1982	11.17	10.80	11.42	11.08	10.00	9.98	10.05	10.58	10.47	10.83	10.24	10.42	10.45	9.96	10.26	11.38	10.21
1983	11.18	10.76	11.04	10.98	10.04	9.93	9.98	10.43	10.33	10.62	10.26	10.29	10.32	9.82	10.07	11.21	10.05
1984	11.35	10.78	10.91	11.02	9.96	9.94	9.92	10.36	10.33	10.66	10.19	10.33	10.29	9.96	10.12	11.05	10.01
1985	11.14	10.64	10.94	11.02	9.70	9.89	9.91	10.29	10.21	10.43	10.17	10.10	10.18	9.96	9.99	10.93	9.78
1986	10.97	10.65	10.77	11.22	9.79	10.24	9.84	10.23	10.18	10.33	10.15	10.09	10.11	9.81	9.97	10.99	9.88
1987	11.10	10.77	10.92	11.09	9.92	10.31	9.91	10.38	10.20	10.33	10.20	10.03	10.10	9.84	10.02	10.89	9.95
1988	11.04	10.68	11.00	11.18	9.88	10.24	9.81	10.36	10.20	10.41	10.00	9.90	10.08	9.85	9.92	10.73	9.88
1989	10.96	10.55	10.50	11.10	9.80	10.12	9.76	10.14	10.09	10.26	10.02	9.90	9.97	9.83	9.92	10.62	9.80
1990	10.96	10.60	10.73	10.99	9.84	10.14	9.76	10.19	10.00	10.37	9.99	9.89	9.88	9.73	9.80	10.40	9.76
1991	10.93	10.47	10.67	10.97	9.77	10.16	9.80	10.19	10.07	10.19	10.00	9.84	9.92	9.76	9.83	10.48	9.71
1992	10.95	10.46	10.47	10.84	9.75	9.87	9.68	10.19	10.03	10.19	10.01	9.84	9.86	9.71	9.84	10.39	9.70
1993	10.69	10.28	10.60	10.73	9.37	9.60	9.51	9.96	9.99	9.95	9.82	9.83	9.83	9.66	9.82	10.31	9.62
1994	10.90	10.35	10.49	10.79	9.49	9.73	9.60	10.11	10.02	9.92	9.86	9.84	9.79	9.65	9.73	10.31	9.58
1995	10.75	10.23	10.56	10.74	9.44	9.51	9.48	10.01	9.94	10.06	9.83	9.63	9.78	9.59	9.71	10.13	9.53
1996	10.76	10.23	10.28	10.75	9.29	9.59	9.43	9.86	9.83	9.87	9.85	9.58	9.70	9.35	9.78	10.05	9.47
1997	10.64	10.18	10.18	10.73	9.38	9.55	9.36	9.90	9.76	10.09	9.79	9.49	9.68	9.37	9.56	10.17	9.35
1998	10.63	10.12	10.11	10.69	9.38	9.51	9.37	9.94	9.72	9.86	9.70	9.41	9.59	9.21	9.47	10.00	9.19
1999	10.46	10.03	10.02	10.49	9.29	9.48	9.23	9.70	9.65	9.78	9.77	9.37	9.57	9.16	9.35	9.83	9.11
2000	10.57	10.08	10.03	10.55	9.33	9.48	9.30	9.77	9.63	9.87	9.75	9.38	9.56	9.23	9.36	9.82	9.18

2001	10.55	10.02	9.89	10.44	9.18	9.43	9.23	9.74	9.66	9.96	9.70	9.31	9.51	9.15	9.34	9.75	9.15
2002	10.45	9.90	9.92	10.26	9.06	9.39	9.11	9.58	9.58	9.68	9.67	9.19	9.46	9.11	9.25	9.67	9.03
2003	10.41	9.86	9.80	10.28	9.07	9.37	9.16	9.46	9.42	9.68	9.58	9.22	9.35	8.98	9.01	9.52	8.93
2004	10.35	9.87	10.06	10.20	9.29	9.40	9.19	9.47	9.59	9.68	9.57	9.21	9.38	8.99	9.17	9.54	8.95
2005	10.43	9.80	10.04	10.08	9.48	9.38	9.14	9.44	9.43	9.70	9.57	9.17	9.27	8.89	8.96	9.35	8.80
2006	10.39	9.88	9.99	10.04	9.20	9.16	9.08	9.50	9.51	9.51	9.42	8.99	9.24	9.00	8.97	9.31	8.85
2007	10.36	9.80	9.84	10.03	9.15	9.25	8.98	9.56	9.42	9.65	9.42	9.00	9.23	8.91	8.90	9.24	8.79
2008	10.32	9.73	9.91	9.99	9.12	9.21	8.94	9.46	9.43	9.65	9.38	8.92	9.13	8.78	8.87	9.16	8.73
2009	10.35	9.78	10.05	9.89	9.18	9.17	8.99	9.53	9.41	9.73	9.37	9.05	9.12	8.73	8.84	9.19	8.71
2010	10.32	9.71	10.05	9.81			8.89	9.46	9.37			9.00	9.12	8.76		9.03	
2011	10.36	9.70	9.64	9.74			8.84	9.51	9.34				9.16	8.73		9.02	
2012								9.29	9.20							8.81	

Appendix 5 Years of significant change in trend (joinpoints) and Annual Percentage Change (APC) for lifespan variation for each Western European country, males

Countries for Comparison		Joinpoint (95% CI)	Years	APC (95 % CI)
Scotland: United Kingdom (1950-2011)	Scotland		1950-1955	-1.25* (-1.8 to -0.7)
		1955 (1952 to 1958)	1955-1989	-0.45* (-0.5 to -0.4)
		1989 (1986 to 1993)	1989-2011	**0.05 (-0.0 to 0.1)**
	England and Wales		1950-1955	-1.23* (-1.7 to -0.8)
		1955 (1953 to 1958)	1955-1970	-0.20* (-0.3 to -0.1)
		1970 (1961 to 1975)	1970-1985	-0.55* (-0.6 to -0.5)
		1985 (1980 to 1999)	1985-2011	-0.24* (-0.3 to -0.2)
	Northern Ireland		1950-1956	-2.19* (-2.8 to -1.6)
		1956 (1954 to 1960)	1956-1974	-0.06 (-0.2 to 0.1)
		1974 (1971 to 1978)	1974-1997	-0.77* (-0.9 to -0.7)
		1997 (1992 to 2001)	1997-2011	**19* (0.0 to 0.4)**
Scotland: Northern Europe (1950-2009)	Scotland		1950-1955	-1.26* (-1.8 to -0.7)
		1955 (1952 to 1958)	1955-1990	-0.45* (-0.5 to -0.4)
		1990 (1987 to 1994)	1990-2009	**0.11*** (0.0 to 0.2)
	Denmark		1950-1960	-0.70* (-0.9 to -0.5)
		1960 (1952 to 1964)	1960-1989	-0.16* (-0.2 to -0.1)
		1989 (1957 to 1992)	1989-1993	-0.95 (-2.0 to 0.1)
		1993 (1985 to 2005)	1993-2009	-0.45* (-0.5 to -0.4)
	Finland		1950-1952	-4.64* (-7.9 to -1.3)
		1952 (1952 to 1954)	1952-1964	-0.81* (-1.0 to -0.6)
		1964 (1958 to 1970)	1964-2002	-0.39* (-0.4 to 0.4)
		2002 (1981 to 2007)	2002-2009	**0.08** (-0.4 to 0.5)
	Norway		1950-1984	-0.45* (-0.5 to -0.4)
		1984 (1954 to 1986)	1984-1988	**0.9** (-0.6 to 2.4)
		1988 (1986 to 1991)	1988-1993	-1.75* (-2.7 to -0.8)
		1993 (1991 to 1997)	1993-2009	-0.50* (-0.6 to -0.4)
	Sweden		1950-1962	-0.60* (-0.7 to -0.5)
		1962 (1956 to 1967)	1962-1972	**0.02** (-0.2 to 0.2)
		1972 (1970 to 1980)	1972-2009	-0.54* (-0.6 to -0.5)
Scotland: Central Europe (1956-	Scotland		1956-1989	-0.46* (-0.5 to -0.4)
		1989 (1987 to 1993)	1989-2010	**0.07** (-0.0 to 0.1)
	Austria		1956-1963	-1.58* (-1.9 to -1.2)

2010)		1963(1958 to 1968)	1963-1973	-0.22 (-0.5 to 0.0)
		1973 (1961 to 1976)	1973-1976	-1.28 (-3.9 to 1.4)
		1976 (1968 to 2008)	1976-2010	-0.55* (-0.6 to -0.5)
	West Germany		1956-1968	-0.83* (-0.9 to -0.7)
		1968 (1966 to 1969)	1968-1971	**0.62** (-0.9 to 2.1)
		1971 (1970 to 1973)	1971-1985	-0.88* (-1.0 to -0.8)
		1985 (1983 to 1988)	1985-2010	-0.41* (-0.4 to -0.4)
	Switzerland		1956-1985	-0.47* (-0.5 to -0.4)
		1985 (1958 to 1989)	1985-1992	**0.12** (-0.3 to 0.5)
		1992 (1978 to 1995)	1992-1997	-1.28* (-2.1 to -0.5)
		1997 (1989 to 2008)	1997-2010	-0.76* (-0.9 to -0.6)
Scotland: Southern Europe (1950-2009)	Scotland		1950-1955	-1.26* (-1.8 to -0.7)
		1955 (1952 to 1958)	1955-1990	-0.45* (-0.5 to -0.4)
		1990 (1987 to 1994)	1990-2009	0.11* (0.00 to 0.2)
	Italy		1950-1953	-2.09* (-3.3 to -0.8)
		1953 (1952 to 1958)	1953-1985	-0.94* (-1.0 to -0.9)
		1985 (1982 to 1988)	1985-1992	-0.08 (-0.4 to 0.5)
		1992 (1989 to 1996)	1992-2009	-0.86* (-1.0 to -0.8)
	Portugal		1950-1981	-1.37* (-1.4 to -1.3)
		1981 (1958 to 1986)	1981-1997	-0.49* (-0.7 to -0.3)
		1997 (1975 to 2007)	1997-2009	-0.95* (-1.2 to -0.7)
	Spain		1950-1954	-4.08* (-5.0 to -3.2)
		1954 (1952 to 1957)	1954-1978	-1.15* (-1.2 to -1.1)
		1978 (1974 to 1983)	1978-1992	0.06 (-0.1 to 0.2)
		1992 (1989 to 1996)	1992-2009	-0.73* (-0.8 to -0.6)
Scotland: Western Europe (1950-2009)	Scotland		1950-1955	-1.26* (-1.8 to -0.7)
		1955 (1952 to 1958)	1955-1990	-0.45* (-0.5 to -0.4)
		1990 (1987 to 1994)	1990-2009	**0.11*** (0.0 to 0.2)
	Belgium		1950-1952	-3.57* (-5.7 to -1.4)
		1952 (1952 to 1954)	1952-1960	-0.95* (-1.2 to -0.7)
		1960 (1955 to 1977)	1960-1983	-0.58* (-0.6 to -0.5)
		1983 (1974 to 1989)	1983-2009	-0.36* (-0.4 to -0.3)
	France		1950-1960	-1.25* (-1.4 to -1.1)
		1960 (1957 to 1962)	1960-1986	-0.36* (-0.4 to -0.3)
		1986 (1979 to 1990)	1986-1991	**0.05** (-0.6 to 0.7)
		1991 (1988 to 1994)	1991-2009	-0.59* (-0.6 to -0.5)
	Ireland		1950-1955	-2.29* (-3.0 to -1.6)
		1955 (1953 to 1961)	1955-1988	-0.65* (-0.7 to -0.6)
		1988 (1957 to 1995)	1988-2007	-0.27* (-0.4 to -0.2)

		2007 (1983 to 2007)	2007-2009	1.58 (-1.5 to 4.8)
	Netherlands		1950-1956	-1.03* (-1.3 to -0.7)
		1956 (1955 to 1959)	1956-1968	0.01 (-0.1 to 0.1)
		1968 (1965 to 1971)	1968-2009	-0.56* (-0.6 to -0.5)

Appendix 6 Years of significant change in trend (joinpoints) and Annual Percentage Change (APC) for lifespan variation for each Western European country, females

Countries for Comparison		Joinpoint (95% CI)	Years	APC (95% CI)
Scotland: United Kingdom (1950-2011)	Scotland		1950-1956	-1.94* (-2.3 to -1.6)
		1956 (1954 to 1958)	1956-1971	-0.20* (-0.3 to -0.1)
		1971 (1963 to 1981)	1971-2003	-0.40* (-0.4 to -0.4)
		2003 (1980 to 2009)	2003-2011	-0.08 (-0.3 to 0.2)
	Eng.& Wales		1950-1056	-1.28* (-1.6 to -0.9)
		1956 (1954 to 1959)	1956-1971	-0.16* (-0.3 to -0.1)
		1970 (1966 to 1975)	1970-2011	-0.41 (-0.4 to -0.4)
	Northern Ireland		1950-1958	-1.70* -2.1 to -1.3)
		1958 (1953 to 1961)	1958-1975	-0.17* (-0.3 to -0.0)
		1975 (1968 to 1981)	1975-2001	-0.63* (-0.7 to -0.6)
		2001 (1994 to 2005)	2001-2011	-0.06 (-0.4 to 0.2)
Scotland: Northern Europe (1950-2009)	Scotland		1950-1956	-1.91* (-2.3 to -1.5)
		1956 (1954 to 1957)	1956-1979	-0.23* (-0.3 to -0.2)
		1979 (1960 to 1981)	1979-1982	-1.00 (-3.2 to 1.2)
		1982 (1963 to 2007)	1982-2009	-0.34* (-0.4 to -0.3)
	Denmark		1950-1960	-0.83* (-1.0 to -0.6)
		1960 (1952 to 1965)	1960-1979	-0.09* (-0.2 to -0.0)
		1979 (1960 to 1992)	1979-1988	**0.20** (-0.1 to 0.5)
		1988 (1985 to 2007)	1988-2009	-0.55* (-0.6 to -0.5)
	Finland		1950-1953	-3.19* (-4.9 to -1.4)
		1953 (1952 to 1956)	1953-1965	-1.32* (-1.6 to -1.1)
		1965 (1962 to 1968)	1965-2009	-0.37 (-0.4 to -0.3)
	Norway		1950-1956	-1.73* (-2.3 to -1.2)
		1956 (1953 to 1963)	1956-1981	-0.46* (-0.5 to -0.4)
		1981 (1976 to 1985)	1981-1987	**0.55** (-0.2 to 1.3)
		1987 (1984 to 1990)	1987-2009	-0.51* (-0.6 to -0.4)
	Sweden		1950-1959	-0.86* (-1.0 to -0.7)
		1959 (1952 to 1963)	1959-1968	-0.36* (-0.6 to -0.2)
		1968 (1960 to 1977)	1968-1971	**0.37** (-1.5 to 2.2)
		1971 (1969 to 1997)	1971-2009	-0.42* (-0.4 to -0.4)
Scotland: Central Europe (1956-2010)	Scotland		1956-1979	-0.23* (-0.3 to-0.2)
		1979 (1965 to 1981)	1979-1982	-1.02 (-3.2 to 1.2)
		1982 (1980 to 2008)	1982-2010	-0.33* (-0.4 to -0.3)
	Austria		1956-1965	-1.30* (-1.5 to -1.1)

		1965 (1961 to 1969)	1965-1973	-0.41* (-0.7 to -0.1)
		1973 (1969 to 1976)	1973-1978	-1.34* (-2.1 to -0.6)
		1978 (1975 to 1986)	1978-2010	-0.55* (-0.6 to-0.5)
	West Germany		1956-1968	-0.83* (-0.9 to -0.8)
		1968 (1965 to 1969)	1968-1971	**0.39** (-0.8 to 1.6)
		1971 (1970 to 1974)	1971-1985	-0.84* (-0.9 to -0.8)
		1985 (1982 to 1988)	1985-2010	-0.45* (-0.5 to -0.4)
	Switzerland		1956-1976	-0.61* (-0.7 to -0.5)
		1976 (1958 to 1986)	1976-1994	-0.33* (-0.4 to -0.3)
		1994 (1975 to 1997)	1994-1998	-1.01 (-2.1 to 0.1)
		1998 (1986 to 2008)	1998-2010	-0.46* (-0.6 to -0.3)
Scotland: Southern Europe (1950-2009)	Scotland		1950-1956	-1.91* (-2.3 to -1.5)
		1956 (1954 to 1957)	1956-1979	-0.23* (-0.3 to -0.2)
		1979 (1960 to 1981)	1979-1982	-1.00 (-3.2 to 1.2)
		1982 (1963 to 2007)	1982-2009	-0.34* (-0.4 to -0.3)
	Italy		1950-1954	-2.66* (-3.5 to -1.8)
		1954 (1952 to 1957)	1954-1982	-1.17* (-1.2 to -1.1)
		1982 (1969 to 1985)	1982-1995	-0.33* (-0.5 to -0.2)
		1995 (1975 to 2002)	1995-2009	-0.72* (-0.9 to -0.6)
	Portugal		1950-1954	-2.68* (-3.9 to -1.4)
		1954 (1952 to 1964)	1954-1961	-1.16* (-1.8 to -0.5)
		1961 (1956 to 1980)	1961-1979	-2.05* (-2.2 to -1.9)
		1979 (1976 to 2007)	1979-2009	-0.79* (-0.9 to -0.7)
	Spain		1950-1954	-4.50* (-5.3 to -3.7)
		1954 (1952 to 1956)	1954-1974	-1.57* (-1.6 to -1.5)
		1974 (1955 to 1978)	1974-1981	-1.03* (-1.5 to -0.6)
		1981 (1975 to 2007)	1981-2009	-0.59* (-0.6 to -0.5)
Scotland: Western Europe (1950-2009)	Scotland		1950-1956	-1.91* (-2.3 to -1.5)
		1956 (1954 to 1957)	1956-1979	-0.23* (-0.3 to -0.2)
		1979 (1960 to 1981)	1979-1982	-1.00 (-3.2 to 1.2)
		1982 (1963 to 2007)	1982-2009	-0.34* (-0.4 to -0.3)
	Belgium		1950-193	-2.68* (-3.8 to -1.5)
		1953 (1952 to 1957)	1953-1961	-1.24* (-1.6 to -0.9)
		1961 (1958 to 1965)	1961-2005	-0.48* (-0.5 to -0.5)
		2005 (1967 to 2007)	2005-2009	**0.14** (-0.6 to 0.9)
	France		1950-1060	-1.77* (-1.9 to -1.6)

		1960 (1952 to 1962)	1960-1969	-0.43* (-0.6 to -0.2)
		1969 (1957 to 1984)	1969-1981	-0.81* (-0.9 to -0.7)
		1981 (1976 to 2006)	1981-2009	-0.40* (-0.4 to -0.4)
	Ireland		1950-1956	-2.53* (-3.0 to -2.1)
		1956 (1954 to 1959)	1956-1987	-0.73* (-0.8 to -0.7)
		1987 (1983 to 1995)	1987-2009	-0.36* (-0.4 to -0.3)
	Netherlands		1950-1953	-0.24 (-1.2 to 0.7)
		1953 (1952 to 1954)	1953-1956	-2.44* (-4.3 to -0.6)
		1956 (1955 to 1957)	1956-1986	-0.25* (-0.3 to -0.2)
		1986 (1969 to 2007)	1986-2009	-0.34* (-0.4 to -0.3)

Appendix 7 Full contributions from age 0 only, males (figure 43 and 44 show contributions truncated to 0.06 years)

shared ex (approx.)	Year achieved (*England*: Scotland)	contribution from age 0 at shared life expectancy (years)	contribution from age 0 to lifespan variation gap (years)
66	*1950*:1961	0.20	-0.16
67	*1953*:1971	0.57	-0.46
68	*1959*:1976	0.50	-0.41
69	*1971*:1981	0.47	-0.39
70	*1977*:1985	0.35	-0.29
71	*1981*:1990	0.25	-0.21
72	*1986*:1995	0.30	-0.25
73	*1990*:2000	0.23	-0.19
74	*1994*:2004	0.09	-0.08
75	*1999*:2008	0.11	-0.09
76	*2002*:2010	0.12	-0.10

The scale of the y axis in the decomposition figures was truncated at 0.06 years as contributions from mortality during the first year of life dominated the scale for the earliest years being compared. These are the full contributions made from the mortality rate difference during the first year of life.

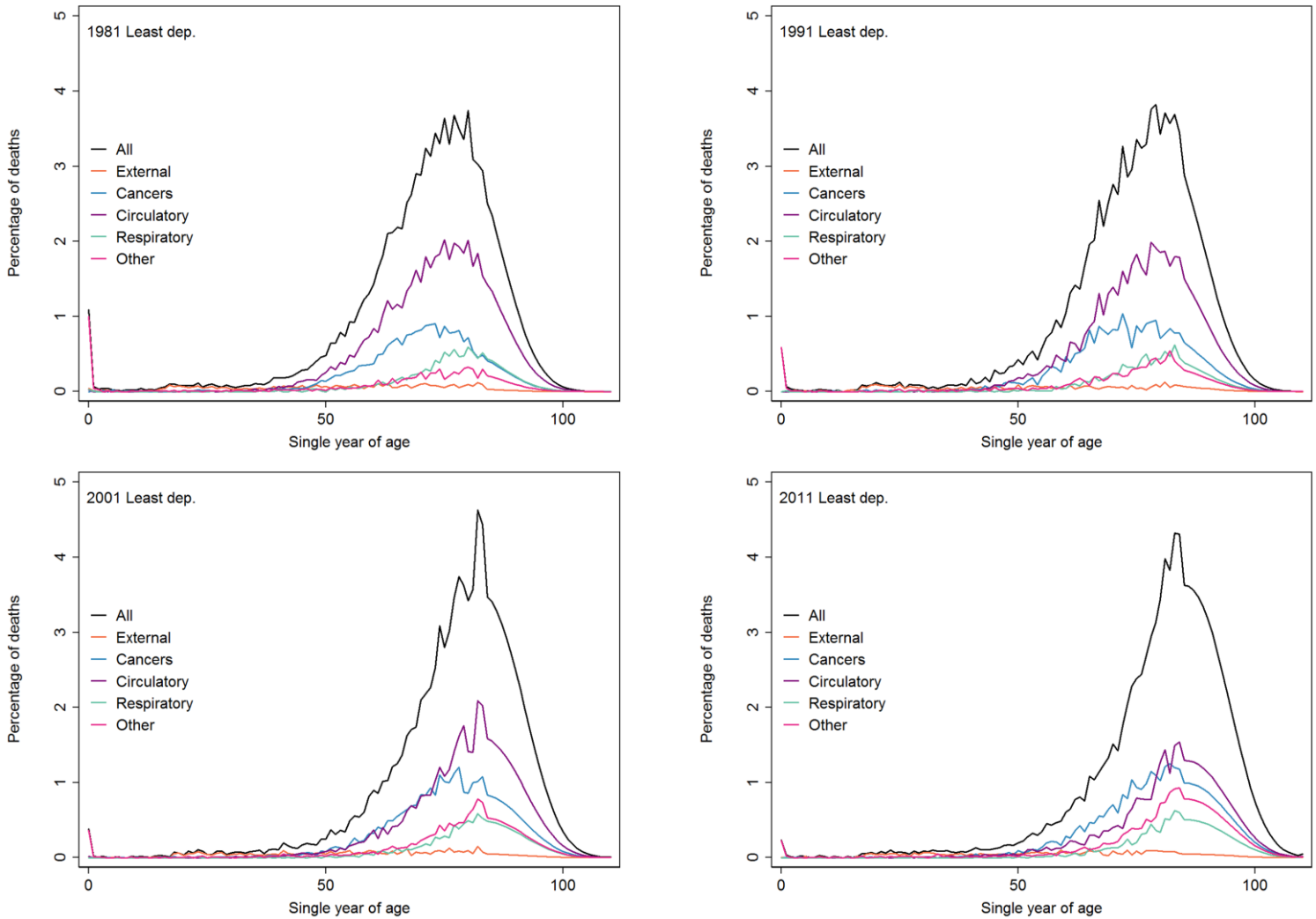
Appendix 8 Full contributions from age 0 only , females (figure 45 and 46 show contributions truncated to 0.06 years)

shared ex (approx.)	Year achieved (*England*: Scotland)	contribution from age 0 at shared life expectancy (years)	contribution from age 0 to lifespan variation gap (years)
71	*1950*:1956	-0.02*	0.02*
72	*1952*:1962	0.16	-0.11
73	*1955*:1967	0.10	-0.13
74	*1961*:1974	0.27	-0.22
75	*1968*:1980	0.35	-0.30
76	*1976*:1985	0.31	-0.26
77	*1982*:1992	0.25	-0.24
78	*1987*:1997	0.24	-0.20
79	*1993*:2004	0.10	-0.07
80	*1999*:2008	0.10	-0.09

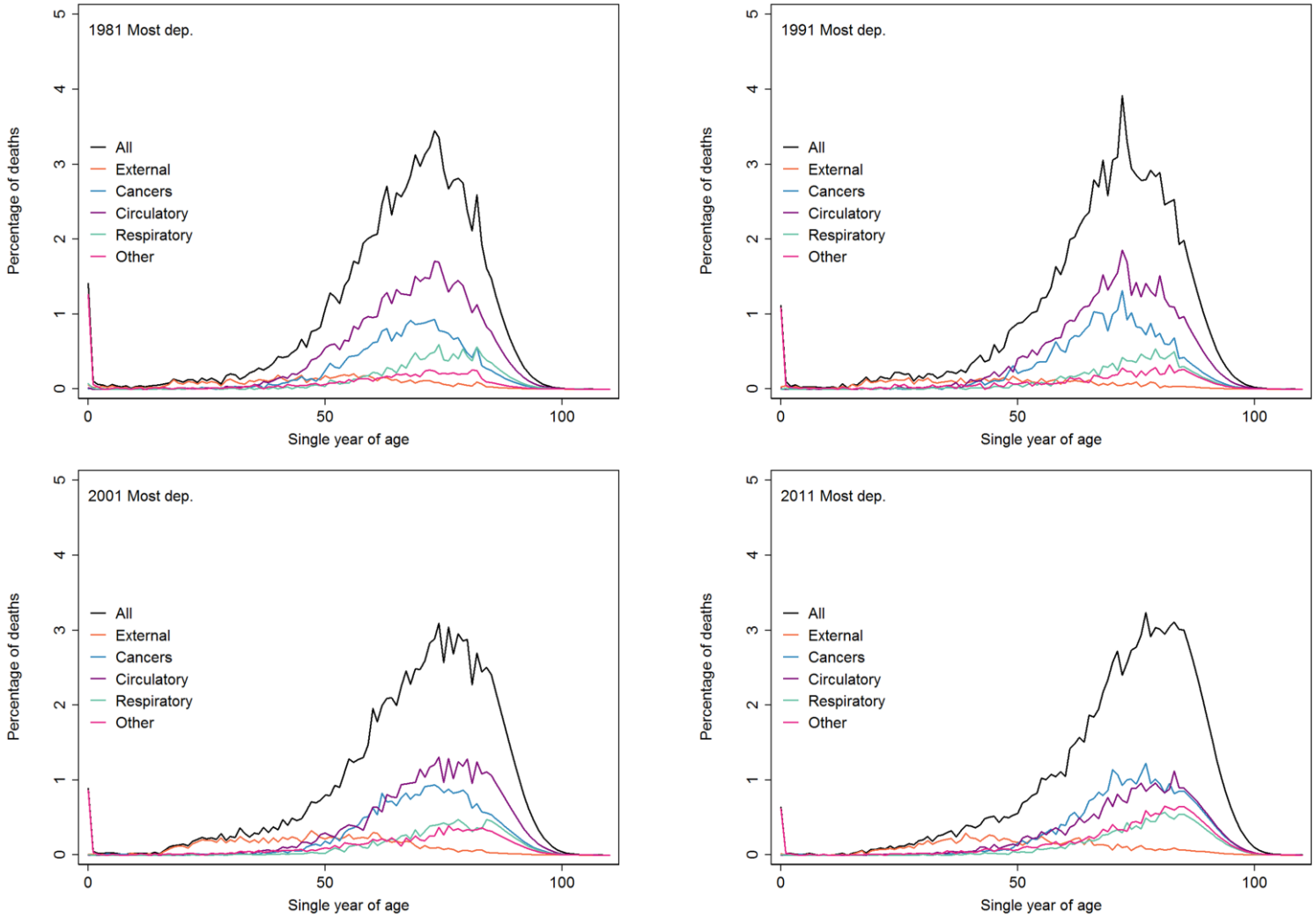
The scale of the y axis in the decomposition figures was truncated at 0.06 years as contributions from mortality during the first year of life dominated the scale for the earliest years being compared. These are the full contributions made from the mortality rate difference during the first year of life.

**Scotland had a very slightly higher infant mortality rate (0.02657) than England and Wales (0.02624) meaning the mortality difference at age 0 made a small, detrimental contribution to Scotland's lifespan variation.*

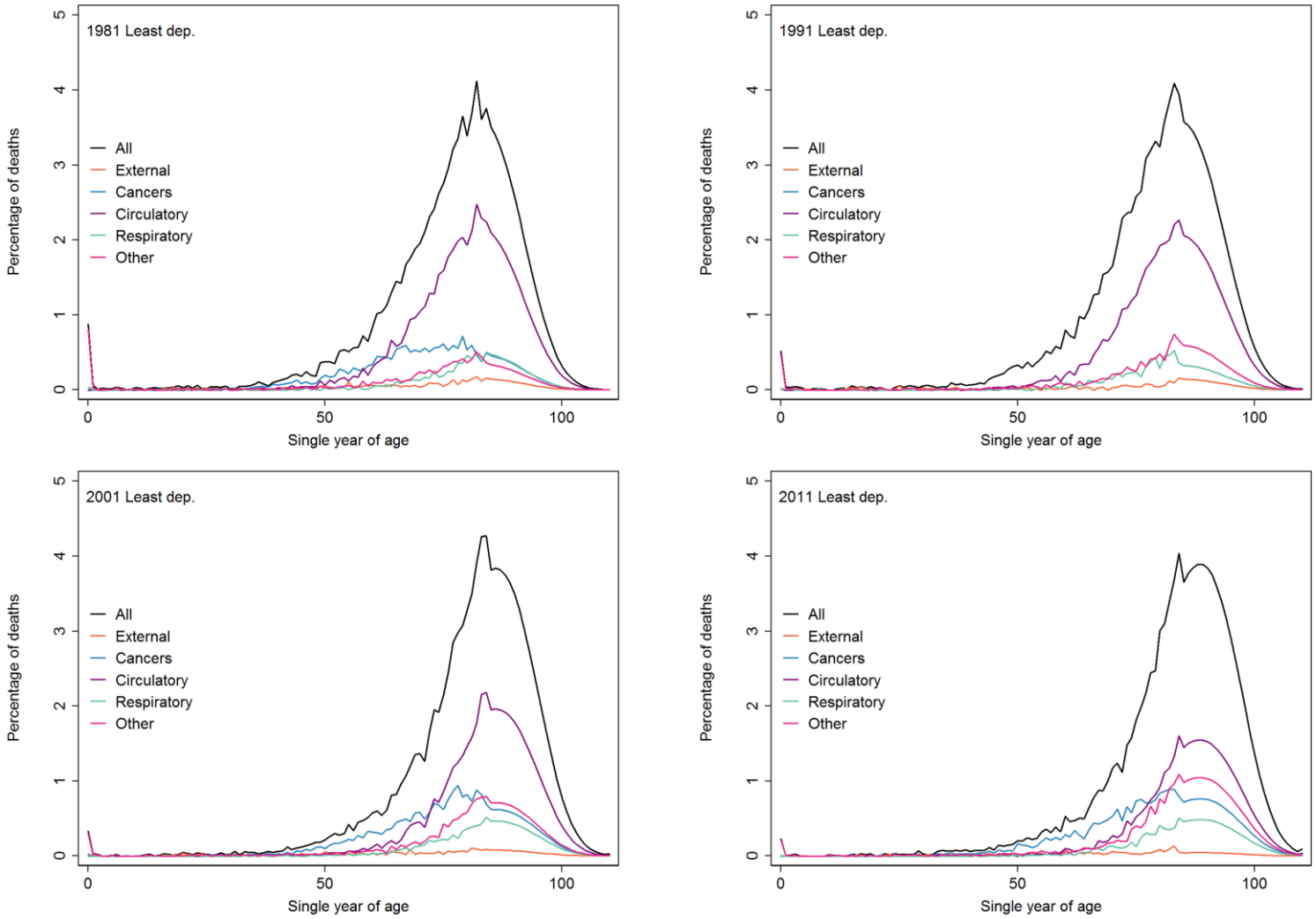
Appendix 9 Age distribution of death (0-110+) by cause of death and Census year, Males, Least deprived quintile



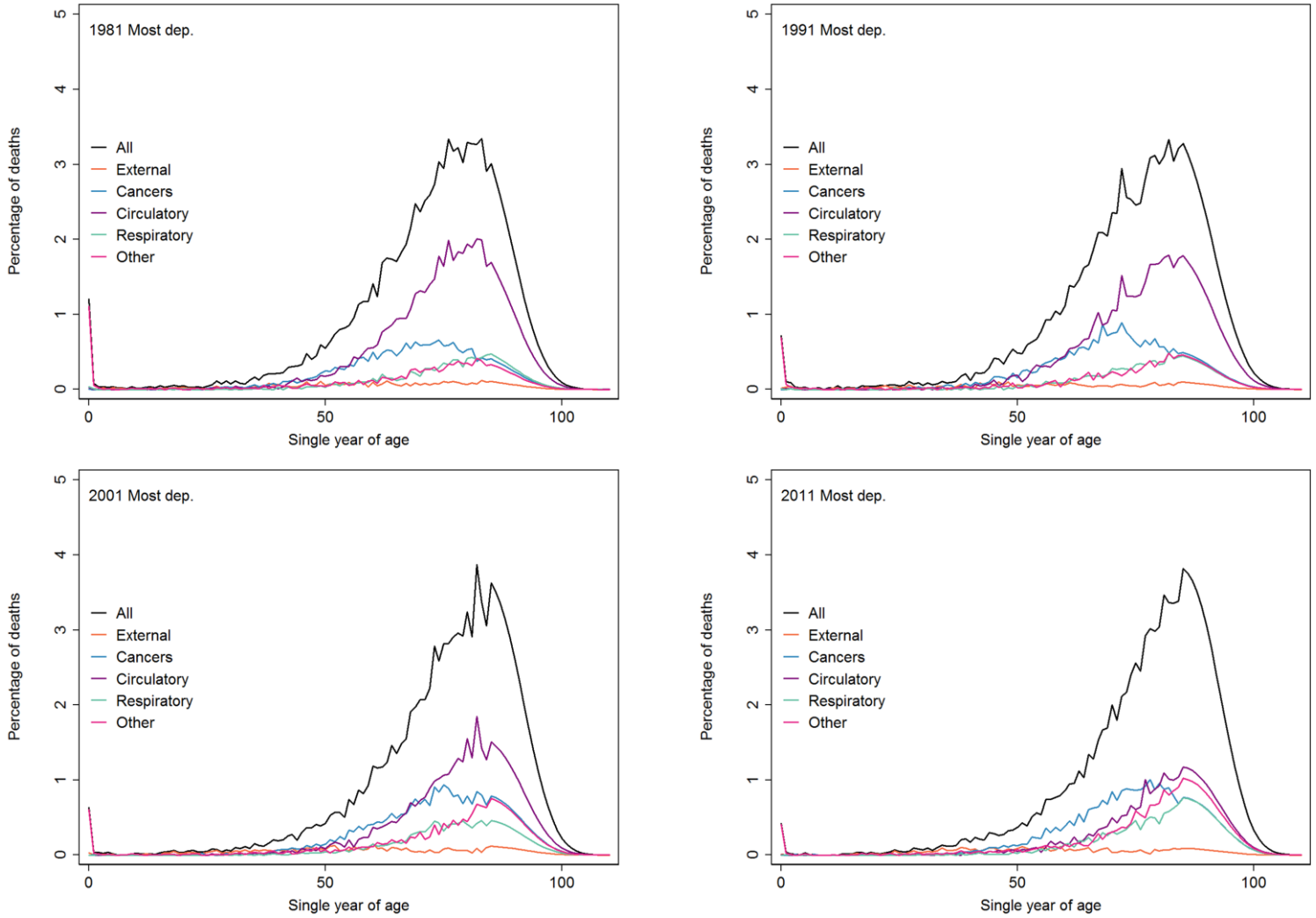
Appendix 10 Age distribution of death (0-110+) by cause of death and Census year, Males, Most deprived quintile



Appendix 11 Age distribution of death (0-110+) by cause of death and Census year, Females, Least deprived quintile



Appendix 12 Age distribution of death (0-110+) by cause of death and Census year, Females, Most deprived quintile



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