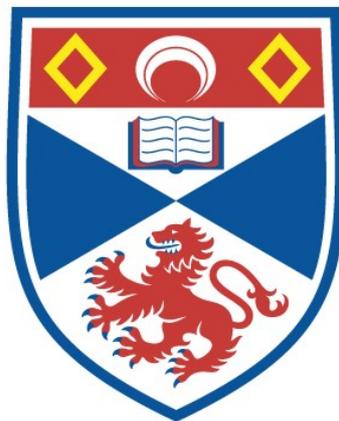


ETHNIC DIFFERENCES IN HEALTH IN SCOTLAND:
THE CONTRAST BETWEEN MORBIDITY AND MORTALITY

Genevieve Cezard

A Thesis Submitted for the Degree of PhD
at the
University of St Andrews



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Ethnic differences in health in Scotland: The contrast between morbidity and mortality

Genevieve Cezard



University of
St Andrews

This thesis is submitted in partial fulfilment for the degree of

Doctor of Philosophy (PhD)

at the University of St Andrews

September 2019

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ABSTRACT

In Scotland, most minority ethnic groups live longer than the White Scottish population but whether they report better health is unclear. Similarly, the international literature on ethnicity and health is divided between a strand that highlights the overall morbidity disadvantage in ethnic minorities and another strand that is puzzled by their mortality advantage. This thesis brings the two strands together by investigating whether ethnic patterns in morbidity and mortality align, based on a unique population source. The Scottish Health and Ethnicity Linkage Study links the Scottish Census 2001 to 12 years of hospitalisation and death records and provides a considerable sample size (4.6 million people) for this research. Therefore, this thesis makes a number of methodological contributions in addition to providing key empirical evidence of an ethnic morbidity-mortality paradox in Scotland. In particular, healthy life expectancy by sex and ethnicity is calculated for the first time in the UK using a direct method. Findings highlight the shortest healthy life expectancies in the Pakistani population contrasting with their mortality advantage. Hence, the Pakistani population experiences the highest number of years in poor health in Scotland. Indeed, the Pakistani population reports poorer health than the majority population which contrasts with their mortality advantage. This contrast persists beyond socio-economic circumstances and across migrant generations. Furthermore, using interaction analyses, this research demonstrates that reporting poorer health strongly predicts higher risks of mortality in minority ethnic groups but with greater strength for particular groups. Finally, findings show higher risks of (hospitalisation-based) multimorbidity in the Pakistani population which supports their morbidity disadvantage in Scotland. Diseases underlying this disadvantage include those related to the metabolic syndrome and respiratory disease. Policy makers should aim to improve the quality of life of the Pakistani population of Scotland while future research pinpoints the root causes of this morbidity-mortality paradox.

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ABBREVIATIONS

ADRN	Administrative Data Research Network
CHI	Community Health Index
CVD	Cardiovascular disease
DFLE	Disability-Free Life Expectancy
EHEMU	European Health Expectancy Monitoring Unit
EU	European Union
HE	Health Expectancy
HLE	Healthy Life Expectancy
LE	Life Expectancy
LLTI	Limiting Long Term Illness
NHS	National Health Service
NRS	National Records of Scotland
ONS-LS	Office for National Statistics Longitudinal Study
OR	Odd Ratio
PAC	Privacy Advisory Committee
PBPP	Public Benefit and Privacy Panel for health and social care
RR	Risk Ratio
SAH	Self-Assessed Health (includes SRH and LLTI)
SES	Socio-economic status
SHELS	Scottish Health and Ethnicity Linkage Study
SIMD	Scottish Index of Multiple Deprivation
SRH	Self-Reported Health
UK	United Kingdom
US	United States (of America)
WHO	World Health Organisation

CHAPTER 1

1. Introduction

1.1. Introduction

In many countries, there are academic and political concerns about how health experiences differ inter-nationally and sub-nationally (Bartley, 2016, Snowdon, 2010, Wilkinson and Pickett, 2010, Woodward and Kawachi, 2000). These concerns are founded, in the United Kingdom (UK) and elsewhere, on a philosophy of fairness and a vision to reduce health inequalities (Woodward and Kawachi, 2000). It is this philosophy that underpins the approach of comprehensive publicly funded healthcare in the UK, embodied in the principles of the National Health Service (NHS).

Despite principles of fairness and equality driving healthcare provision in the UK for half a century, considerable inequalities exist. The Black Report, published in 1980, was pivotal in raising these issues to the fore of political and research attention, and provided evidence of the relationship between 'social class' and health (Bartley, 2004). How socio-economic status (class) impacts on the health outcomes of individuals has been a policy and research focus for the last 40 years in the UK, and during this time, interest in understanding health inequalities has expanded to consider other axes of difference. One such axis that has received increasing attention is ethnicity.

Ethnic inequalities in health have been demonstrated internationally and the UK is not an exception. Early research in the UK portrayed minority ethnic groups as disadvantaged health-wise compared to the majority population (Blakemore and Boneham, 1994, Evandrou, 2000a, Harding and Balarajan, 2000). Analysis of data from the 1990s showed that, apart from the clear advantage seen in the Chinese population, most minority ethnic groups were more likely to report poorer health than the White majority (Becares, 2013, Evandrou, 2000a, Harding and Balarajan, 2000). However, patterns of ethnic differences in health are changing over time and this health disadvantage has become less evident for some of these ethnic groups in contemporary Britain (Becares, 2013, Darlington et al., 2015, Scottish Government, 2015). Hence, ethnic inequalities in health need to be continuously monitored. Importantly, different contexts can produce different patterns and consequently new clues for the understanding of health inequalities. For example, in Scotland, the mortality evidence shows an advantage in most ethnic groups compared to the majority population (Bhopal et al., 2018, Gruer et al., 2016) while morbidity evidence provide a somewhat different picture of ethnic inequalities in health

(Scottish Government, 2004, Scottish Government, 2015). These particularities of the Scottish context are a key element to test theories and to improve our understanding of processes underlying health inequalities.

Indeed, investigating the ethnic dimension of social inequality can augment understandings of the mechanisms of health inequalities; extant work has demonstrated, for example, how socio-economic inequalities and ethnic inequalities in health can be linked (Lorant and Bhopal, 2011, Nazroo, 2001, Nazroo, 2003). Some of the mechanisms underlying ethnic inequalities in health include socio-economic status, migrant health selection, acculturation, health behaviours, discrimination, and genetic/environment interaction processes (which are presented in chapter 2). However, further research is warranted to fully understand the contributions of these mechanisms in shaping health inequalities.

In epidemiology, researching ethnic variations in health is considered valuable since it has the potential to provide additional clues to the mechanisms involved in the aetiology of diseases (Chaturvedi, 2001, Bhopal, 2004). This stronger understanding of diseases could be fed into prevention, intervention, and changes in practices which could benefit the whole population. However, this work is not without its critics: some academics warn about the difficulties in defining and measuring the concept of ethnicity (see sections 2.1.2 and 3.3.4.2) and advise careful consideration of how to use ethnicity for aetiology determination (Bhopal, 1997, Senior and Bhopal, 1994). In particular, it is important to make a distinction between ethnicity and race, to acknowledge the concept of ethnicity as fluid and complex, to recognise the limitations associated with the ethnic classification process, and to include socio-economic status when researching the link between ethnicity and health. These guidelines are followed for this research.

In addition to epidemiological considerations, exploring ethnic inequalities in health in the UK is timely because of the diversity and changing nature of the country's population. In particular, the population of the UK is ageing as a result of the post-World War II demographic baby boom, followed by a fertility decline. The UK population is also becoming more ethnically diverse (Jivraj, 2012, Simpson, 2014a, Smith, 2013). In England and Wales, the White British population accounted for 80% of the population in 2011 compared to 87% in 2001 and non-white populations increased from 9% to 14% of the population between 2001 and 2011 (Jivraj, 2012). In Scotland, although the proportion of non-white minority groups accounted for only a small part of the population in 2001 (2%), this figure increased to 4% of the population in 2011.

The proportion of the White Scottish group in Scotland decreased from 88% to 84% between 2001 and 2011. The ethnic diversity of the UK is projected to continue increasing by mid-century (Rees et al., 2011, Rees et al., 2012).

Migration to the UK, which drives this diversity, is not new (Ahmad and Bradby, 2007). The term “migrants” used through this thesis refers to international immigrants. In the 19th century and the first half of the 20th century, migrants were mainly from Ireland or came as refugees from Russia, Germany and Poland (Hannemann and Kulu, 2015). However, most immigration happened during the post-World War II economic boom, predominantly from the Caribbean and India in the 1950-1960s, Pakistan and Bangladesh in the 1970-1980s and Hong-Kong in the 1980-1990s (Nazroo, 2004). By 1990, most migrants to the UK were from former dominions or colonies and migration from other European countries was low (Fassmann and Munz, 1992). Migration settlement patterns were tied to particular industries and regions (Ahmad and Bradby, 2007). Migrant settlement occurred primarily in England and migration from the New Commonwealth occurred to a lesser extent to Scotland (Anderson, 2016).

Post-1990, the origin countries of migrants to the UK increased in diversity (Smith, 2013). In the 1990s, the UK saw an increased number of migrants from countries experiencing conflicts and political unrest such as South Africa, Zimbabwe, Ghana, Iran, Iraq, Kosovo, Afghanistan, Somalia, Sri Lanka, Rwanda, Angola, Sierra Leone, Bosnia, Croatia, Serbia and Montenegro (Smith, 2013). Migration from European Union (EU) countries also increased, with the largest increase seen in Polish migrants arriving after 2004 when Poland joined the EU (Smith, 2013). With the exception of the Irish population, the primary migration groups have continued to grow in the UK due to continued immigration for family reunification, work or education but also due to natural growth (Simpson and Jivraj, 2015, Simpson, 2013). The migration history and natural growth of the UK thus drive its current ethnic mosaic. In 2011, the largest non-white minority ethnic groups were the Indian group followed by Pakistani, African, and Caribbean groups in England and Wales (Jivraj, 2012) while the Pakistani, Chinese and Indian groups represented the largest non-white minority ethnic groups in Scotland (Simpson, 2014a).

The increasing diversity of the UK, and the growth in the share of the non-White populations, has increased the relevance of ethnicity and ethnic inequalities for policy. In 2010, ethnicity (or race) became formalised as a protected characteristic in the Equality Act 2010 in the UK (Government Equalities Office, 2013). Since October 1st 2010, the Equality Act 2010 has legally protected people from discrimination, harassment and victimisation in the workplace and

the wider society. It replaced the Race Relations Act 1976 and the Race Relations Amendment Act 2000 in the UK. Following the Equality Act 2010, the Department of Health business plan in 2012-2013 aimed to improve health and reduce health inequalities for all people by their equality characteristics (including race/ethnicity). In this respect, identifying and understanding ethnic inequalities in health is key to reaching these policy targets. Indeed, the knowledge gained from researching ethnic inequalities in health can inform the decisions of policy makers. Informed policies can be developed and adapted to the needs of diverse minority groups and thus improve the quality of care and services provided.

This thesis explores ethnic inequalities in health in Scotland with a particular focus on the contrast between morbidity and mortality. The direction of this research stems from the realisation that international evidence on ethnic inequalities in health is divided between a strand focusing on morbidity and another strand puzzled by a mortality advantage (see chapter 2). The morbidity strand tends to show minority ethnic groups or migrants as disadvantaged compared to majority populations in their country of residence. In contrast, the mortality literature shows a mortality advantage in most migrants and some ethnic groups in specific contexts. What is striking is that both the morbidity disadvantage and the mortality advantage seem to apply simultaneously to certain ethnic groups in the Scottish context. This suggests that these ethnic groups live longer than the majority population but in poorer health. However, available evidence on morbidity and mortality patterns comes from different data and population sources. Hence, it has been difficult to compare morbidity and mortality directly. Internationally, evidence of a morbidity-mortality contrast is emerging in some migrant groups but this has had little formal investigation and these studies suffer from methodological limitations. This emerging morbidity-mortality contrast literature also raises the question of which measures of health are reliable. Some might argue that mortality prevails as a gold standard to measure population health due to its objective nature. Others might define health more broadly than the mere avoidance of death and consider different forms of disadvantage and discrimination which can lead to poorer health in ethnic minorities. This thesis argues that understanding morbidity and mortality together is essential if we are to further uncover the mechanisms of ethnic inequalities in health and improve planning for adapted care and services for all.

This research uses data linkage at the Scottish national level, bringing together ethnicity, morbidity and mortality data. The analysis of ethnic differences in both morbidity and mortality

is based on a unique population source and a considerable sample size of 4.6 million people who responded to the Scottish Census in 2001. Indicators of self-reported morbidity, doctor-diagnosed morbidity and mortality are used to research the morbidity-mortality contrast as well as the consistency of reported morbidity measures in predicting morbidity and mortality across ethnic groups. Hence, examining ethnic inequalities in health using various measures of morbidity and mortality also adds to the debate on the reliability of different health measures when researching ethnic health inequalities.

Using data linkage provides the opportunity to make both substantive and methodological contributions to research on the ethnic morbidity-mortality paradox. One methodological contribution is the calculation of healthy life expectancy estimates by ethnicity. For the first time, estimates are calculated based on a direct method and use both reported morbidity and mortality data from a single population source. Second, the analysis of morbidity and mortality linked together makes a crucial methodological contribution. Thanks to a very large sample size, the analysis and interpretation of interaction terms permits a robust study of the relationship between reported health and more objective measures of morbidity and mortality across ethnic groups. Substantive contributions also include bringing the morbidity and mortality strands together, allowing the investigation of whether a morbidity-mortality paradox exists in particular ethnic groups in Scotland and increasing our understanding of the relationship between subjective and objective health across ethnic groups.

1.2. Research aims and structure of the thesis

The limited existing evidence shows the need for further research on the health advantage or disadvantage experienced by minority ethnic groups in comparison to the majority population in Scotland. Understanding the experiences of different ethnic groups is particularly desirable in order to identify specific needs and ensure adapted care and services. Hence, this thesis seeks to examine the morbidity-mortality paradox in specific ethnic groups in Scotland by investigating:

- How patterns of reported morbidity by ethnicity compare to patterns of mortality by ethnicity based on the same population source.
- Whether mechanisms thought to shape ethnic inequalities in health contribute to explaining ethnic differences in reported morbidity.
- Whether reported morbidity relates to mortality consistently across ethnic groups.

- Whether using a doctor-diagnosed measure of health provides similar patterns of ethnic differences in morbidity as using reported morbidity.

The rest of this thesis is structured as follows. Chapter 2 defines the concepts used in this thesis. The literature is reviewed in relation to ethnic differences in morbidity and mortality. This leads to a discussion of the literature related to the contrast and the relationship between morbidity and mortality across ethnic groups. Key theories underlying ethnic differences in health are presented. Gaps are summarised and research questions outlined. Chapter 3 discusses the research design, data sources and methods appropriate for the investigation of the research questions. Chapter 4 explores ethnic differences in self-assessed health and healthy life expectancy and how these compare to mortality patterns by ethnicity. Chapter 5 investigates the relationship of self-assessed health with mortality across ethnic groups. Chapter 6 examines ethnic differences in multimorbidity and the subjective-objective morbidity relationship across ethnic groups. Finally, chapter 7 reflects on the core findings of this research, their contributions and implications and how they direct future research avenues.

CHAPTER 2

2. Literature review

Chapter 2 introduces some of the key concepts used in this thesis and reviews the evidence available regarding ethnic inequalities in health by distinguishing evidence related to morbidity from evidence related to mortality. Section 2.1 explains key concepts such as health and ethnicity. Section 2.2 draws on the empirical evidence available to shed light on what we know about ethnic variations in morbidity and mortality in the UK context. This review focuses on general indicators of health and the UK setting but is also enriched by evidence based on specific diseases and further international literature. The mortality and morbidity streams highlight different patterns of ethnic inequalities in health. Hence, a morbidity-mortality contrast is discussed. Contrasting morbidity and mortality evidence also questions how measures of reported morbidity relate to mortality. Section 2.3 describes the key mechanisms of ethnic differences in health that are investigated for this research. Finally, section 2.4 summarises the literature gaps and presents the research questions explored in this thesis.

2.1. Concepts

2.1.1. Health and health inequalities

For the purpose of this research project, health is considered in terms of both morbidity and mortality. Mortality refers to those who have died within a population and morbidity refers to those who are still alive in a diseased or unhealthy state. Both morbidity and mortality provide an indication of the population's general health and are widely used in official statistics.

Being in good health refers more generally to the state of being free of disease or illness considering someone's mental and physical condition. Beyond the presence or absence of diseases, the World Health Organisation (WHO) proposed a broad definition of health in 1948: "Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity." This thesis does not focus on a particular disease to research ethnic differences in health but rather looks at general health acknowledging that an individual's experience of health is not reduced to the presence or absence of a single disease. A range of self-declared and doctor-diagnosed indicators of general health align with this more holistic approach to health, some of which are used in this thesis. As measures of self-declared health, measures of self-assessed health (SAH) analysed include both self-reported health (SRH) and limiting long term illness (LLTI). Therefore, this thesis uses SAH as a collective term including

both SRH and LLTI. Mortality is also a marker of general health at the end of someone's life. The operationalisation of general health is detailed in chapter 3.

However, along with taking a more general approach to health, one needs to take into account the diversity of health experiences and to recognise that health can be socially constructed. Health is not only biological, it also varies across social groups. It is recognised that socio-economic status is a key determinant of health (Marmot and Wilkinson, 1999). There are also evident ethnic differences in health (reviewed in section 2.2). Indeed, social groups might have a set of characteristics, beliefs and exposures that influence their health. Many mechanisms are likely to be involved in shaping health inequalities by social group. The pathways by which socio-economic status and ethnicity influences health are further explained in section 2.3.

2.1.2. Concepts of ethnicity, race and intersectionality

2.1.2.1. Ethnicity

Ethnicity relates to identity. Etymologically, ethnicity is derived from the Greek term *ethnos/ethnikos* which means nation/national. In comparison to the *demos* (the people, democracy, state), *ethnos/ethnikos* means an ethnic group, people sharing a distinctive culture and ancestry. The contemporary use of the term ethnicity is in line with its Greek origin. Ethnicity is commonly associated with shared ancestry, culture, language, religion, tradition and history and ethnic groups are usually researched within a specific state (Meer, 2014, Peoples and Bailey, 2011).

Nonetheless, ethnicity is a fluid concept that changes over time and according to context (Bhopal, 2004, Simpson et al., 2016). Simpson et al. showed that 4% of the population who responded to the England and Wales censuses 2001 and 2011 changed their recorded ethnicity between 2001 and 2011 (Simpson et al., 2016). The authors explained how this change can be attributed at least in part to a change in individual's perception of ethnic identity.

Ethnic groups are social groups derived from real and imagined characteristics that shape ethnic identities. The set of characteristics taken into consideration in shaping ethnicity vary (Bhopal, 2004). Ethnicity is based on individual self-definition as well as the perception from others (Meer, 2014). This subjectivity leads to questions about how ethnicity can be accurately measured (see part 3.3.4.2 for the operationalisation of ethnicity). Barth's research has broken new grounds in the study of ethnic groups by focusing on ethnic boundaries and their

maintenance and presenting ethnic identity as an aspect of social organisation (Jenkins, 1994, Barth, 1969, Malešević, 2004, Meer, 2014). He argues that boundaries are constructed through internal self-awareness as well as delimitations from interaction with others (Barth, 1969). So group identities emerge by differentiating “us” from “them” (Eriksen, 1995, Eriksen, 2002). This perception renders ethnic boundaries subjective and changeable. Furthermore, through individual and collective perception, ethnicity is consequently influenced by social, economic and political contexts and is continuously reconstructed (Nagel, 2014).

To accommodate this complexity, Gabbert (Gabbert, 2006) suggests the following definition (p. 90), which is adopted in this thesis:

“Ethnicity refers to a phenomenon of social differentiation in which actors use cultural or phenotypic markers or symbols to distinguish themselves from others. It is a method of classifying people into categories that include individuals of both sexes and all age groups organized into several kin groups using a (socially constructed) common origin as its primary reference.”

2.1.2.2. Ethnicity and race

Concepts of ethnicity and race are often used interchangeably in the Anglo-American literature, not least in public health research (Bhopal, 2004). However, Blakemore and Boneham argue that it is important to differentiate ethnicity from race and that although ethnicity might be used as an euphemism for race, they are two distinctive concepts (Blakemore and Boneham, 1994). They consider race as a biological concept rather than a potentially broader concept. The biological concept of race refers to physical characteristics such as skin colour and facial attributes and consequently depends on visual perception. This visual perception can then lead to preconceived ideas and stereotypes related to physical attributes with the potential to trigger “a racist response”. In this context, Blakemore and Boneham therefore refer to racism as “biological reductionism” (Blakemore and Boneham, 1994). In this line of idea on the biological construct of race, the word “race” suggests biological variations between different races and implies genetic differences (Kaplan and Bennett, 2003). Historically, the genetic argument in relation to race advanced that different races were distinctive genetically. However, this argument is not supported by evidence in the genetic field because most of genetic variation is within population rather than between (race) groups (Chaturvedi, 2001, Kaplan and Bennett, 2003, Malik, 2009).

This line of arguments pushed some epidemiologists to suggest the omission of race in health research (Krieger, 2000, Krieger, 2005). However, as Krieger explains it (p. 212): “just because “race” is not a meaningful biological construct does not mean that “race”, per se, is a meaningless or “unscientific” category” (Krieger, 2000). Races as social groups or racialized social groups are constructed socio-historically with real consequences (Loveman, 1999). In his book “Strange Fruit: Why Both Sides Are Wrong in the Race Debate”, Malik explains that even though the biological concept of race is irrational and makes little sense on a genetic basis, both the process of racialization and racism remain real and impact on individual experiences (Malik, 2009). This thesis does not explore directly the effect of racism and discrimination on ethnic differences in health but it does not undermine it either. These theories are further developed in section 2.3.4 and discussed in relation to the findings in chapter 7.

Finally, ethnicity is a complex construct that encompasses biology but also considers culture and a range of social attributes. This complexity provides opportunities for the determination of aetiology through research on ethnic differences in diseases and therefore the use of ethnicity is invaluable (Chaturvedi, 2001, Bhopal, 1997).

2.1.2.3. Intersectionality

Intersectionality first emerged as a feminist demand to include women as subjects of research owing to their distinctive experience. This was criticised by Black feminists for ignoring intersections with other dimensions of identity (McCall, 2005, Meer, 2014). Intersectionality accounts for the multi-dimensional aspects of identity and the complexity of social life and questions the validity of discrete analytical categories. Some research might focus on the intersection of two or more typical identity-related characteristics such as age, sex, ethnicity, religion and social class. This type of research, especially when using a quantitative approach, still requires a process of classification and demarcation. Thus, McCall distinguishes three approaches to deal with intersectionality which aim to account for the social complexity and attain intelligibility: the ‘intra-categorical’ approach focusing on a neglected group at the intersection, the ‘anti-categorical’ approach deconstructing analytical categories and the ‘inter-categorical’ approach focusing on the relationship of inequalities while maintaining analytical categories without fixing them (McCall, 2005). These different approaches of exploring intersectionality offer tools which are adapted in this thesis, either using an intra-categorical approach focusing on a particular group at the intersection or a more global approach along the line of an inter-categorical approach.

In the context of ethnicity studies, other aspects of identity such as sex, age, religion, social class and disability can affect how ethnic health inequalities are shaped and hence, can be of value in refining our understanding of inequalities. Traditional epidemiological research on ethnicity tends to include multiple characteristics of identity but the analysis at the intersection is seldom developed. Increasingly, scholars advocate examining ethnic disparities in relation to other dimensions of social inequality as the way forward while recognising limitations and technical issues such as small sample sizes (Weber and Fore, 2007). The inclusion of intersectionality in health research has the potential to identify the needs of disadvantaged populations while possibly producing new theories and mechanisms (Bauer, 2014). Werbner argues that, in contrast with multiculturalism, which valorises multiple identities and inter-ethnic interactions into positive identities, intersectionality relates to negative forms of identity with the idea that multiple aspects of identity might lead to multiple forms of exclusion or discrimination and thus, disadvantage (Werbner, 2013). This thesis acknowledges the importance of exploring the intersection of ethnicity with other aspects of identity. Chapter 3 explains how intersectionality is included in the analysis and used in relation to the key underlying theories of ethnic inequalities in health presented in section 2.3.

2.2. Ethnicity and health: empirical evidence

2.2.1. Ethnicity and mortality

2.2.1.1. The history of mortality and ethnicity research in the UK

The literature focusing on mortality patterns by migrant or ethnic groups emerged in the UK in the 1980's (Balarajan et al., 1984, Marmot et al., 1984). During the first decades of research aiming to identify ethnic differences in mortality in the UK, studies used mainly country of birth as a proxy for ethnicity (Balarajan et al., 1984, Fischbacher et al., 2007, Harding and Balarajan, 2001a, Marmot et al., 1984, Maxwell and Harding, 1998, Smith et al., 2000, Wild and McKeigue, 1997, Wild et al., 2007, Wild et al., 2006). In these studies, numerators tended to be calculated from death records and denominators from census records separately (Fischbacher et al., 2007, Marmot et al., 1984, Maxwell and Harding, 1998, Smith et al., 2000, Wild and McKeigue, 1997, Wild et al., 2007, Wild et al., 2006). However, some studies attempted to use names and parents and grandparents' country of birth to distinguish migrants from expatriates born in the Indian subcontinent (Balarajan et al., 1984, Harding and Balarajan, 2001a, Harding, 2003) or to analyse migrants and descendants mortality patterns (Harding and Balarajan, 2001b, Harding et al., 1996).

Research was limited by the mortality data available. Indeed, UK death registers recorded country of birth but no ethnicity record was available (Harding and Balarajan, 2001a, Rees et al., 2009). While this is still the case in England and Wales, in Scotland, ethnicity recording on death records was introduced in 2012 (Christie, 2012). However, as any new data collected by registrars, their validity for monitoring and for analysing ethnic differences in mortality remains questionable and the collection process has to improve for this information to become usable (Dixon et al., 2018).

Due to the lack of national level data on mortality by ethnicity, Rees et al. published new approaches to calculate mortality and life expectancy estimates by ethnicity with the aim of using the resulting findings for population projections (Rees and Wohland, 2008, Rees et al., 2009). The authors developed two methods of estimations based on patterns of Limiting Long Term Illness (LLTI) by ethnicity and the geographical distribution of ethnic group for all local authorities in the UK. The Standardised Illness Ratio method relied on the hypothesis that LLTI is consistently associated with mortality across ethnic groups while the Geographical Weighted Model assumed that each ethnic group shared the same mortality level within local authorities. In the absence of mortality data by ethnicity at national level in the UK, these methods provided valuable information.

However, since Rees, Wohland and colleagues' advances in mortality patterns by ethnicity (Rees and Wohland, 2008, Rees et al., 2009, Wohland et al., 2016, Wohland et al., 2015), methodological progress were made through the use of linkage studies, linking death records to census data at the individual level (Bhopal et al., 2018, Gruer et al., 2016, Scott and Timæus, 2013, Wallace, 2016, Wallace and Kulu, 2015). Using the Office for National Statistics Longitudinal Study linking multiple censuses to mortality data for about 1% of the England and Wales population, the patterns of mortality by ethnicity were further investigated for both migrants and descendants (using self-reported ethnicity and country of birth from the census) (Scott and Timæus, 2013, Wallace, 2016). In Scotland, linking the Scottish census 2001 to death records at the individual level, the Scottish Health and Ethnicity Linkage Study (SHELS) explored mortality by ethnicity and nativity (Bhopal et al., 2018) and offered, for the first time in the UK, a direct calculation of life expectancy estimates by ethnicity (Gruer et al., 2016).

2.2.1.2. International evidence on mortality patterns in migrants and ethnic minorities: the mortality paradox

There has been an increasing body of evidence on ethnic differences in mortality with a particular interest in the mortality advantage experienced by particular ethnic or migrant groups compared to the majority population of the country they live in. For example, Hispanics in the US experience a mortality advantage despite lower socio-economic status compared to non-Hispanic Whites (Markides and Coreil, 1986, Markides and Eschbach, 2005). This phenomenon is considered as an epidemiologic paradox because one would expect those with higher level of deprivation to experience higher mortality. This often-called ‘Hispanic paradox’ has been studied in the US for many decades (Ellis, 1959, Ellis, 1962, Elo and Preston, 1997, Elo et al., 2004, Markides and Coreil, 1986, Markides and Eschbach, 2005) and has led to further research aiming to unravel the mystery (Abraído-Lanza et al., 2005, Abraido-Lanza et al., 1999, Jasso et al., 2004, Turra and Elo, 2008, Ullmann et al., 2011, Arias et al., 2010, Fenelon, 2013). Similarly in Belgium, older migrants had an all-cause mortality advantage compared to non-migrants regardless of their socio-economic status (Reus-Pons et al., 2016). This mortality advantage seems to apply to many ethnic or migrant groups in varying European countries (Anson, 2004, Bhopal et al., 2018, Bos et al., 2004, Brahimji, 1980, Deboosere and Gadeyne, 2005, Ikram et al., 2016, Khat and Darmon, 2003, Lehti et al., 2016, Marmot et al., 1984, Razum et al., 2000, Reus-Pons et al., 2016, Syse et al., 2018, Wallace and Kulu, 2014a) although it cannot be generalised and does not hold true for all minority groups in all countries (Bos et al., 2004, Ikram et al., 2016, Syse et al., 2018). Furthermore, the “migrant mortality advantage” has been shown to hold overall in a systematic review and meta-analysis recently published (Aldridge et al., 2018). Whether this mortality advantage applies to migrants only or extends to subsequent generations is an active debate discussed in section 2.3.2.

In the UK, the “migrant mortality advantage” varies by country of birth. For example, Wallace and Kulu found a higher all-cause mortality in those who were born in Scotland and Ireland compared to native born in England and Wales while most migrants from non-UK countries had lower all-cause mortality (Wallace and Kulu, 2015). In Scotland, most of the larger ethnic groups had a mortality advantage compared to the native White Scottish population (Bhopal et al., 2018, Gruer et al., 2016). The authors highlighted that observed ethnic differences in mortality can be shaped by how healthy the comparison group (usually the native population) is in their country of residence. Indeed, in Scotland, most ethnic groups had longer life expectancies compared to the White Scottish majority (Gruer et al., 2016). In males, Indian

followed by Pakistani males had the longest life expectancies. In females, Pakistani females had the longest life expectancy followed by Chinese and Indian females. In contrast, White Scottish and Any Mixed Background populations had the shortest life expectancies in Scotland.

Further research emphasized the need to explore the causes of death to distinguish the underlying diseases driving these mortality advantage or disadvantage in minority ethnic groups (Bos et al., 2004, Ikram et al., 2016, Lehti et al., 2016, Reus-Pons et al., 2016, Wallace and Kulu, 2015). In Europe, an excess mortality from infectious diseases and a lower mortality from cancers was generally found in minority ethnic groups compared to respective native populations (Bos, 2005, Ikram et al., 2016, Khlal and Guillot, 2017, Reus-Pons et al., 2016, Wallace and Kulu, 2015). There was mixed evidence in relation to mortality from cardiovascular disease (CVD). An overall lower CVD mortality was found in minorities with the clear exception of South Asians showing higher CVD mortality (Ikram et al., 2016, Wallace and Kulu, 2015).

In addition to the exploration of the causes of death, many hypotheses (see section 2.3 for theories of ethnic differences in health) have been investigated that can explain the mortality advantage in minority ethnic groups. This includes health selection processes such as the “healthy migrant effect” (Marmot et al., 1984, Ullmann et al., 2011) and the “salmon bias” (Pablos-Mendez, 1994, Turra and Elo, 2008, Vandenheede et al., 2015), protective health behaviours, cultural and family support mechanisms and acculturation processes (Abraído-Lanza et al., 2005, Harding, 2003, Harding, 2004). The potential for this advantage to be attributed to a data artefact was also tested (Arias et al., 2010, Kibele et al., 2008, Wallace and Kulu, 2014a). Despite the available evidence in the last few decades and especially in the US context regarding the Hispanic paradox, ethnic inequalities in mortality remain not fully understood and further evidence is required.

2.2.2. Ethnicity and morbidity

2.2.2.1. Ethnicity and general morbidity

Ethnic differences in general morbidity have been explored using self-assessed measures of health in many developed countries (Becares, 2013, Bombak and Bruce, 2012, Jylhä et al., 1998, Lindström et al., 2001, McDonald and Kennedy, 2004, McGee et al., 1999, Wiking et al., 2004, Wu and Schimmele, 2005). Minority ethnic groups were generally more likely to report poorer health than the majority population with some exceptions (Bombak and Bruce, 2012). Some evidence showed that poor outcomes for particular ethnic groups (of African origin) in the US context was not replicated for ethnic groups with similar origins in the Canadian context

highlighting the complexity of processes underlying ethnic health inequalities (Wu and Schimmele, 2005). Indeed, different contexts might yield different exposures, selection processes and population structures leading to various patterns of inequalities. As previously explained, ethnic differences in health within a country are also shaped by the health status of the majority population they are compared to (Gruer et al., 2016). Hence, research into ethnic differences in health is context specific. The following review of the literature surveys evidence of ethnic differences in self-assessed health (SAH) available in a broad UK context and then narrows the focus on available evidence in the specific context of Scotland.

Evidence of ethnic inequalities in SAH in England and Wales has increased in the last two decades (Becares, 2013, Darlington et al., 2015, Evandrou, 2000a, Evandrou et al., 2016, Harding and Balarajan, 2000, Karlsen and Nazroo, 2010, Mindell et al., 2014, Smith and Grundy, 2011, Smith et al., 2009) while Scottish based evidence has remained limited to official Scottish Government reports (Scottish Government, 2004, Scottish Government, 2015). A few publications used full census data in England and Wales and in Scotland (Scottish Government, 2004, Scottish Government, 2015, Becares, 2013). These studies provided a useful description of the general patterns of ethnic differences in SAH at the national level but were based on aggregated data derived from censuses. Findings based on aggregated data can be subject of the ecological fallacy, whereby interpretation of the nature of phenomenon at the individual level is deduced from the group individuals belong to (Piantadosi et al., 1988). Moreover, analyses based on aggregated data were, at best, age-standardised, did not add on further adjustment and consequently, could not explore the contribution of underlying theories of inequalities. In order to investigate explanations of ethnic inequalities in SAH, a more refined analysis based on individual level data would be desirable.

In England and Wales, a briefing by the Centre on Dynamics of Ethnicity (CoDE) explored trends in ethnic differences in Limiting Long Term Illness (LLTI) over time based on cross-sectional aggregated data of censuses 1991, 2001 and 2011 (Becares, 2013). Using age-standardised ratios of LLTI, the briefing found that the Chinese population consistently reported better health with half of the illness rates compared to the reference group (aggregated white group in 1991, White British group in 2001 and 2011). Other White and Black African populations reported better health in 2001 and 2011. In contrast, Pakistani and Bangladeshi groups reported higher illness rates in 1991 and 2001 and females of these groups carried on reporting poorer health up to 2011. In 2011, the highest illness rates was reported by those of White Gypsy or

Irish traveller backgrounds, newly created category in the census 2011. In 1991, Indian females and Black Caribbean males and females started with a disadvantage, a higher LLTI ratios compared to the White group, but their disadvantage faded away through to 2011 with LLTI ratios converging towards the reference group for the Black Caribbean population and starting to reverse for Indian females.

Further evidence in England and Wales used longitudinal and survey data at the individual level to explore the ethnic patterning of SAH, using either self-reported health (SRH) or LLTI or both (Darlington et al., 2015, Evandrou, 2000a, Evandrou et al., 2016, Harding and Balarajan, 2000, Mindell et al., 2014, Smith and Grundy, 2011, Smith et al., 2009). Overall, they found similar trends as in Becares' CoDE briefing (Becares, 2013). Smith and Grundy found a significant and persistent disadvantage in LLTI in the South Asian groups (Pakistani, Bangladeshi and Indian) compared to the White British majority between 1991 and 2001 (Smith and Grundy, 2011). Darlington et al. found higher rates of LLTI in a combined Pakistani and Bangladeshi group which declined up to 2005 at which stage no more difference in LLTI compared to the White British group was observed for the period 2005-2008 (Darlington et al., 2015). However, the authors found a persistent disadvantage in the combined Pakistani and Bangladeshi group from 1998 to 2011 when using SRH. Their research also supported an initial health disadvantage in Indian and Black minorities which disappeared through to 2011 when using SRH and which reversed in relation to illness rates from 2001 onwards. Using the Health Survey for England in 2003-2006, Mindell et al. analysed SAH by ethnicity with a fine ethnic granularity (Mindell et al., 2014). Their findings also supported poorer health in adults of Pakistani, Bangladeshi and Black Caribbean origins as well as in Indian women but only significantly so in Pakistani women when using LLTI rather than poor health as an indicator. The authors also found lower rates of illness in Black African and Chinese adults in 2003-2006 reinforcing previous evidence but an advantage in LLTI in Irish women which did not appear in the CoDE briefing.

In Scotland, two official reports published by the Scottish government provided information on ethnic inequalities in SAH (Scottish Government, 2004, Scottish Government, 2015). Based on the Scottish Census 2001, a report published in 2004 focused on the ethnic profile of the Scottish population in 2001 and described its health using SRH and LLTI (Scottish Government, 2004). The descriptive analysis used percentages of reporting poor health or a LLTI by ethnic group and for different age groups. Age stratification was justified by an increase of reporting poor health with age e.g. 10% of the Scottish population reported poor health in 2001

while 22% did so in those aged 60 years and above. However, the report had no sex stratification, no age standardisation nor direct comparison to the majority population and the analysis mostly ranked ethnic groups according to their percentage of reporting poor health or their percentage of reporting a LLTI. Overall the ranked results showed the highest percentages of reporting poor health in the Bangladeshi group for those aged 16-24 years, in Black Scottish and Other Black and Any Mixed Background groups in those aged 25-34 years and in the Pakistani group in those aged 35-59 years and those aged 60 years and above. In contrast, the Chinese population had the lowest percentage of reporting poor health in those aged 25-34 years and 35-59 years while Other White British and Bangladeshi groups had the best outcome in those aged 60 years and above. In 2001, 20% of the Scottish population reported a LLTI and 51% did so in those aged 60 years and above. Patterns of reporting a LLTI were similar to those observed in reporting poor health, with the exception of the African population showing better reported LLTI outcome in those aged 35-59 years and those aged 60 years and above.

Based on the Scottish census 2011, the Scottish Government published a new report in 2015 specifically focused on the patterns of SRH and LLTI by ethnic group (Scottish Government, 2015). Although no direct comparison to the White Scottish population was statistically tested, their analysis was stratified by sex and ranked ethnic groups according to their age-standardised ratios of SRH (bad or very bad) and LLTI. The report found that most minority ethnic groups reported better health than the White Scottish apart from a few exceptions. In 2011, the newly created category Gypsy/Travellers reported the poorest health. Worse health was also reported in Pakistani men and women, Any Mixed Background men and Bangladeshi women. In contrast, the Chinese, Polish, Other White, Other British, Indian, African and Other Asian groups reported consistently better health than the White Scottish in both men and women. Patterns of ethnic differences in health were broadly similar when using either SRH or LLTI.

In summary, despite evident methodological drawbacks as explained earlier, the Scottish evidence seems to point to a persistent disadvantage in the Pakistani and Any Mixed Background origin populations and a persistent advantage in the Chinese population which aligns with some of the patterns seen in the rest of the UK. However, further research is required in Scotland to confirm the identified patterns using a more refined analysis based on individual level data.

2.2.2.2. Ethnicity, general morbidity and ageing

As the UK population is ageing, majority and minority alike, one can wonder whether the ethnic patterning of health persists into older ages. Decades ago, Blakemore and Boneham reported evidence of poorer health and premature ageing in ethnic minority elders in the UK (Blakemore and Boneham, 1994). Whether this phenomenon persisted into more recent elderly populations needs further evidence. Indeed, most of the evidence on ethnicity and SAH focuses on working age population and patterns in later life remain under-researched. In the context of an increasing diverse ageing population, investigating inequalities at the older ages is required to adjust and develop appropriate care and resources.

A few studies have attempted to fill this gap. An initial study by Evandrou found higher odds of reporting poor health in a combined Pakistani and Bangladeshi group, in Indian, and in Black Caribbean groups as well as in Irish women in those aged 60 years and above in 1991-1996 (Evandrou, 2000a). Two decades later, using Understanding Society data in 2009-2011, Evandrou et al. revisited the patterns observed in those aged 60 years and above while accounting for time resident in the UK and socio-economic deprivation (Evandrou et al., 2016). The authors found significant adjusted odds of reporting poor health in Pakistani men and women compared to the White British groups. When using 'health limits' rather than SRH, there was higher adjusted odds in the Pakistani and Indian groups as well as in Bangladeshi men and Caribbean women. Finally, they showed graphically that, as the percentage of reporting poor health increase with age, so do the ethnic differential in reporting poor health, giving support to the hypothesis that ethnic inequalities in health widen with increasing age.

The CoDE briefing by Becares and the Scottish government report published in 2015 also presented findings for those aged 65 years and older (Becares, 2013, Scottish Government, 2015). Becares found that, in those aged 65 years and above in the England and Wales census 2011, White Gypsy/Travellers, Bangladeshi and Pakistani groups had the highest percentage of LLTI while Chinese had the lowest percentage (Becares, 2013). This was in line with findings for all age group combined for females and to some extent for males. In the Scottish census 2011, similar trends were also found in the elderly population as in the general population (Scottish Government, 2015). In those aged 65 years and above, Gypsy/Travellers had the worst health profile and Pakistani men and women, Bangladeshi women and Any Mixed Background men reported poorer health than the majority White Scottish. However, Indian women and White Polish men and women appeared to report poorer health when aged 65 years and above while

reporting a health advantage in younger age groups. These analyses, based on aggregated census data, could not explore the mechanisms involved in shaping these ethnic differences in health in older ages.

2.2.2.3. Disease-specific evidence

Ethnic inequalities in morbidity depend on the health indicator under investigation. Findings from general health indicators or from specific diseases might show a disadvantage in one case and an advantage in the other. Indeed, ethnic differences in general health may hide a high variability in relation to risk profile for different type of diseases. For example, Hispanics in the US tend to report poorer health and have higher risk of obesity and diabetes but in contrast lower risks of hypertension, stroke, coronary heart disease and major cancers (Hayward et al., 2014). Similarly, in the context of the UK, we know that South Asians have a higher risk of diabetes and renal disease (Dreyer et al., 2009, Forouhi et al., 2006, Hull et al., 2011, Sproston and Mindell, 2006) but a lower risk of cancer (Bhopal et al., 2012b).

In Scotland, disease-specific evidence is primarily based on the SHELS study (Walsh, 2017). For example, it showed that the Pakistani population was at higher risk of cardiovascular disease (Bansal et al., 2013, Bhopal et al., 2011), stroke (Bhopal et al., 2012a), asthma (Sheikh et al., 2016), all-cause and lower respiratory diseases (Bhopal et al., 2015, Simpson et al., 2015) but at lower risks of cancer including for specific cancers such as lung, colorectal, breast and prostate cancers (Bhopal et al., 2012b). In contrast, Chinese populations were at lower risks of most diseases explored within the SHELS study (Bansal et al., 2014, Bansal et al., 2013, Bhala et al., 2016, Bhopal et al., 2012a, Bhopal et al., 2015, Bhopal et al., 2011, Bhopal et al., 2012b, Bhopal et al., 2014, Sheikh et al., 2016, Simpson et al., 2015) with the exception of some gastrointestinal diseases such as peptic ulcer disease (Cezard et al., 2015). The Scottish literature of ethnic differences for particular diseases highlights that ethnic differences in health are disease-specific or health outcome dependent as well as varying by ethnic group beyond the simple foreign-born/native-born dichotomy.

2.2.3. The emergence of a morbidity-mortality paradox

A mortality advantage in a specific ethnic group does not necessary coincide with a reported morbidity advantage in this same group compared to the majority population of the country of residence. Evidence supporting this observation was initially seen in the Hispanic elderly population in the US showing a favourable mortality profile as well as poorer reported health compared to non-Hispanic Whites (Markides et al., 1997). This can lead to question the

validity of both the mortality advantage and the reported morbidity disadvantage or alternatively lead to the emergence of new theories and explanations on why some groups experiencing worse health might survive longer. A few research studies have published evidence of a morbidity disadvantage along with a mortality advantage in specific ethnic or migrant groups and contexts (De Grande et al., 2014, Deboosere and Gadeyne, 2005, Khlal and Guillot, 2017, Kouris - Blazos, 2002).

Kouris-Blazos was the first to refer to a “morbidity-mortality paradox” in relation to Greek migrants in Australia (Kouris - Blazos, 2002). Reviewing the literature, she found that Greek migrants in Australia had higher risks of CVD risk factors such as obesity, diabetes, hyperlipidaemia (high cholesterol levels), smoking, hypertension and sedentary lifestyle but also lower risks of CVD mortality and all-cause mortality. She hypothesised that the Mediterranean diet was a key protective factor of CVD and overall mortality in Greek Australians. Since then, two Belgium studies and one French review of the health and mortality of migrants have identified that the migrant mortality advantage did not fit with reported morbidity evidence (De Grande et al., 2014, Deboosere and Gadeyne, 2005, Khlal and Guillot, 2017). However, researching the morbidity-mortality contrast or ‘paradox’ was not the core aim of these publications. The aforementioned research in Australia, Belgium and France gathered evidence from different sources to identify a morbidity-mortality contrast in particular migrant groups compared to the majority population. The only research study showing evidence of a morbidity-mortality contrast using both morbidity and mortality data based on a unique population source is a recent study on the health of older Italian migrants compared to that of their native-born counterparts in Australia (Stanaway et al., 2019). With some similarities to the Greek Australians case, Stanaway et al. found that older Italian migrants aged 70 years and above were more likely to smoke and be overweight and had higher risks of diabetes which are risk factors for CVD. Higher morbidity was also found in relation to chronic pain, dementia and depressive symptoms in this population. In line with the health disadvantage observed for particular diseases and risk factors, older Italian migrants reported poorer self-rated health. However, they also had a 25% lower mortality risk compared to Australian-born when accounting for their lower SES. As lost-to-follow-up in this longitudinal study was minimal, this mortality advantage strikingly contrasted with their self-rated health and disease-specific profile.

The Scottish evidence in relation to mortality (section 2.2.1) and morbidity (section 2.2.2) shows a mortality advantage in most minority ethnic groups in Scotland while the morbidity

evidence shows more mixed results. As explained earlier, evidence in relation to reported morbidity by ethnicity in Scotland needs to be confirmed using more refined methods but, despite methodological limitations, the available morbidity evidence based on self-assessed health points to a disadvantage in the Pakistani population. This contrasts with the mortality findings from the SHELS linkage study which show one of the longest life expectancy in the Pakistani population in Scotland. Importantly, the above evidence on morbidity and mortality patterns by ethnicity came from different studies and sources. Hence, further research on ethnic differences in both morbidity and mortality would be stronger if based on the same population source.

Finally, an emerging literature in the UK shows that higher prevalence for a specific disease can coincide with a mortality advantage in a particular ethnic group diagnosed with the disease. For example, compared to the White majority population, South Asians are at higher risk of diabetes in the UK (Forouhi et al., 2006) but are at lower risk of all-cause mortality once diagnosed (Davis et al., 2014). South Asians with chronic kidney disease have also been shown to have better survival than white patients with chronic kidney disease in a UK diabetic population (Mathur et al., 2018). Similar evidence in Canada supported better survival in South Asians once diagnosed with diabetes (Khan et al., 2011) or once diagnosed with chronic kidney disease (Barbour et al., 2010) compared to white patients. In relation to myocardial infarction, a Scottish study found a higher risk of myocardial infarction incidence in the Pakistani population compared to the White Scottish majority but better survival after a first event (Bansal et al., 2013).

Both the literature presented on greater survival in diseased South Asians and the contrasted Scottish evidence in this section hints to a “morbidity-mortality paradox” in the Pakistani population in Scotland. To understand the discrepancy in outcomes between morbidity and mortality in this population, there is a need to confirm the little evidence on ethnic differences in morbidity and mortality available in the Scottish context.

2.2.4. Self-assessed health as indicator of morbidity and its relation with other measures of health

As explained in section 2.2.2.1, ethnic differences in general morbidity are often assessed using self-assessed health indicators. SAH indicators are indicators of health in the broader sense, in line with WHO holistic definition of health (see section 2.1.1). SAH has been shown to be associated with other measures of health such as measures of physical and mental

health, physician rating of health, health care usage and mortality (Cohen et al., 1995, Idler and Benyamini, 1997, Idler and Kasl, 1995, Larue et al., 1979, Miilunpalo et al., 1997, Mossey and Shapiro, 1982, Wannamethee and Shaper, 1991). The literature on the association between SAH and other measures of morbidity and mortality supports SAH as a valid measure of population health. In addition to the extensive evidence on SAH validity, a number of empirical research studies has also demonstrated its stability (Bailis et al., 2003, Fosse and Haas, 2009, Miilunpalo et al., 1997, Mossey and Shapiro, 1982, Power et al., 2000). Hence, SAH indicators are widely used and accepted as reliable measures of general health status.

In relation to ethnicity, a few initial studies have demonstrated a consistent association of SAH with more objective measures of morbidity, health services use and mortality across ethnic groups (Chandola and Jenkinson, 2000, McGee et al., 1999). This initial literature supports SAH as a valid measure of health to research ethnic inequalities in health. For example, a UK study by Chandola and Jenkinson found that the association between self-rated health and a range of more objective measures of morbidity (hypertension, stroke, heart disease, diabetes, GP visits, limiting health) did not significantly vary between ethnic groups (Chandola and Jenkinson, 2000). The authors concluded that self-assessed health was a valid measure of health across ethnic groups. The validation of SAH as a reliable measure of general health status across ethnic groups was a key contribution in the UK on which recent research relied on to justify further contributions (Rees et al., 2009, Darlington et al., 2015). For example, Rees and colleagues used the ethnic patterns in SAH to estimate mortality by ethnicity, relying on the evidence that SAH is a valid measure of health across ethnic groups and that SAH is strongly related to mortality (Rees et al., 2009, Wohland et al., 2015).

SAH is nevertheless contested to measure health similarly across different cultural groups (Bombak and Bruce, 2012, Franks et al., 2003, O'Reilly and Rosato, 2010, O'Reilly et al., 2005). Indeed, SAH is reported by the individual and as such it is deemed a subjective measure of health. Different groups might have different appraisals of what is considered good or bad health. There could be cultural differences in the meaning and reporting of health. In that case, SAH might not reflect objective health status similarly across ethnic groups. Chandola and Jenkinson found no evidence of a differential association between reported health and more objective measures of health across ethnic groups in the UK (Chandola and Jenkinson, 2000). However, their analysis was based on small sample size leading to wide confidence intervals in minority ethnic groups. This limitation points to the need to further confirm whether self-

reported measures of health are valid and meaningful measures of general health across ethnic groups. In addition, section 2.2.3 gathered initial evidence of contrasted ethnic patterns in SAH and in mortality in Scotland. In light of this SAH-mortality contrast for a specific ethnic group in Scotland, one can wonder whether SAH relates to mortality consistently across ethnic groups. The consistency of the SAH-mortality association across ethnic groups has been investigated in the US in two recent studies (Assari et al., 2016, Woo and Zajacova, 2016). The authors showed a stronger association of self-reported health with mortality in White compared to Black populations in the US. However, these studies remained limited in terms of fine ethnic categories to explore the consistency of the association across groups.

If SAH is used in research and clinical setting, there is a need to better understand its validity in different populations (Franks et al., 2003). The initial evidence of an ethnic SAH-mortality contrast in Scotland and the lack of research exploring whether SAH predicts objective health similarly across ethnic groups internationally, with a strong sample size and a fine ethnic granularity, point to the need for further research investigating the association between SAH and objective health across ethnic groups in the Scottish context.

2.3. Theories of ethnic differences in health

This section 2.3 introduces key theories underlying ethnic differentials in health outcomes. Various theories are proposed as potential explanatory factors for ethnic health inequalities in the literature. What determines ethnic differences in health is complex, multi-dimensional and is likely to be the results of intertwined theories including migration processes (migration effect, health selection, acculturation), environmental and social factors (socio-economic status, racism and discrimination, access to health care), cultural factors determining lifestyle and biological/genetic factors. In this section, theories of socio-economic status, acculturation and migrant generations are outlined and will be investigated in chapters 4, 5 and 6. In addition, health selection hypotheses are also introduced and will be drawn upon for the interpretation of our findings. Other theories are briefly summarised and will be reflected upon in the discussion chapter.

2.3.1. Socio-economic status

Socio-economic status (SES) is the combined economic and social status of an individual or group. SES encompasses different aspects of social position and circumstances which are important to distinguish (Bartley, 2004). Material deprivation or wealth is one of these aspects

which refers to the material assets one possesses. Another key element of SES is the concept of social class. The concept of class was initially studied through work related social structures but also includes aspects of social status and prestige (Bartley, 2004). Indeed, in his recent book on contemporary social classes, Savage explains that the status aspect is a key element of social class which includes three types of capital: economic, cultural and social (Savage, 2015).

A range of SES measures can inform on the material and social deprivation of an individual. For example, measures of material wealth can include income, household tenure, and car ownership. Social class can be studied using a combination of proxy measures such as occupation, education, and income but also more complex measures of status. Note that some socio-economic indicators might contribute to both social and material deprivation. For example, owning a house might play a role to confer a particular social status but would also be considered as material asset. In the UK, measures of area deprivation such as the index of multiple deprivation in England or the Scottish index of multiple deprivation in Scotland have been employed. A neighbourhood SES measure can also be a proxy for individual SES at both, material (neighbourhood assets such as access to services and green spaces) and social (community social prestige and norms) levels.

Low SES has long been associated with poorer health (Adler and Newman, 2002, Angell, 1993, Feinstein, 1993, Marmot and Wilkinson, 1999, Stringhini et al., 2017). A key landmark in the UK was the Black Report which emphasised on socio-economic inequalities in health (Townsend and Davidson, 1982, Holder, 2011, Nazroo, 1998). The direction of the relationship, whether low SES affects poor health or whether poor health affects low SES is debatable. Low SES can restrict access to better health resources and health education but alternatively an episode of sickness might lead to a drop in income via disengagement with the labour market and consequently poorer SES. Feinstein argued that the direction of the association depends on which aspect of SES is considered (Feinstein, 1993). When household wealth is used as a proxy for SES rather than household income, the direction of the association is most likely to be wealth affecting health rather than the opposite because wealth accumulates over time and will be less affected by an event of illness. Overall, the pathway in which the deterioration of health results in lower SES has been indicated as explaining a small portion of the SES-health association (Kneesebeck et al., 2003, Marmot and Nazroo, 2001). Most research assumes that SES impacts on health (Adler and Newman, 2002, Feinstein, 1993, Pampel et al., 2010, Winkleby et al., 1992, Nazroo, 2001) and the pathways by which SES impacts on health need to be understood. Indeed,

Angell explained that income, education and occupation do not affect health directly but are proxies for other determinants of health (Angell, 1993). In her book “Health inequalities”, Bartley describes the main models offered as pathways for socio-economic inequalities in health (Bartley, 2004). These includes behavioural and cultural explanations, psycho-social factors, material aspects, and life course circumstances. Some of the possible mechanisms involved in the SES-health pathway described by Bartley are summarised in the next four paragraphs.

The behavioural and cultural model focuses on the psychosocial and behavioural factors which influence health behaviours and consequently health outcomes. The selection hypothesis is one explanation offered in this type of model which proposes that a set of ‘personal characteristics’ could lead to both lower SES and worse health behaviours. For example, lower ‘human capital’ could lead to less resources to understand health promotion resulting in worse health behaviours. However, this type of selection argument was rejected by Bartley as it has been shown that everyone knows that smoking is bad for your health and the issue is more likely to lie in having the resources to invest in health and engage in behaviour change. Another explanation relates to the social and cultural environment individuals are exposed to, which influences their lifestyles (choice of leisure activities, books or food) and thus, their health behaviours and outcomes. The normative lifestyle of a social environment plays a role in maintaining distinctive social groups which influences health behaviours and consequently impacts on health inequalities.

In the psycho-social model, factors such as social support, the level of demand and control in the workplace, and the balance between efforts and rewards are likely to play a part in how SES affects health. It is hypothesised that low social support, lack of autonomy at work, and low efforts-rewards balance influence how people feel and their levels of stress which in turn produce a negative biological response. This biological response (often measured through individual inflammatory markers or allostatic load) is associated with an increased risk of worse health outcomes.

In the material model, income and assets have an influence on exposure to health hazards, both, in the home and work environments. For example, lower income might lead to difficulties in accessing good quality housing and, in turn, lower housing standard (damp, lack of heating) might increase the risk of infectious and respiratory diseases. In addition, a low-paid job might be strenuous and increase exposure to occupational hazards which would be detrimental for health.

Finally, to understand how SES influences health, many have sought explanations in a life course approach. Health disadvantage in later life has been related to an accumulation of SES and psychological disadvantage over the life course with potential origin in the social and emotional environment experienced in early childhood (Kendig et al., 2016, Marmot and Wilkinson, 1999, Crimmins et al., 2004). Jasso refers to the “health production function” to explain how health is an accumulation of “stock of health” with depreciations or investments to improve health over time which are influenced by various inputs including SES (Jasso et al., 2004).

Evidence of SES differences by ethnic group are undisputable. In the UK, ethnic differences in SES profile based on traditional SES measures from Survey and Census data are well documented and show a disadvantage in minority ethnic groups (Evandrou, 2000b, Holder, 2011, Nazroo, 1998, Nazroo, 2001). The England and Wales Census 2011 revealed SES differences by ethnicity; for example, higher levels of unemployment in non-White minorities compared to the White majority and especially so among non-White minority women (Potter-Collins, 2014). Some evidence points to Pakistani and Bangladeshi populations doing generally worse in the UK in terms of SES and deprivation (Evandrou, 2000a, Nazroo, 2004, Evandrou, 2000b, Holder, 2011, Nazroo, 2001). Second generation of migrants generally experience upward socio-economic mobility, at least in relation to educational level (Li and Heath, 2016). However, this positive generational shift exists alongside persistent unemployment among second generations (Li and Heath, 2016).

Many indicators of SES are household based. Some may be affected by the number of people in the household (e.g. household income) but for others, e.g. car availability, the large households of some ethnic minority groups may convey an advantage. For example, in considering the availability of a car to an older person, evidence suggests ethnic minority elders are in a better position than White British elders (Evandrou, 2000b, Kelaher et al., 2009). Ethnic minority elders may cohabit with grown-up children, who provide the car mobility. The size and structure of the household can influence the availability of household.

The SES measure employed when looking at ethnic differences in health is relevant in the sense that there are ethnic differences in economic priorities (Kelaher et al., 2009, Smith, 2000). For example, in contrast with the mainstream association of education with car and home ownership, Kehaler et al. found that Indian and Pakistani populations in the UK were more likely to own a car which was inversely associated with education and more likely to own a home at any level of education (Kelaher et al., 2009). Looking at people aged 60 years and above in Great

Britain, Evandrou found overall similar levels of education attainment by ethnic group but differences in occupation by ethnicity (Evandrou, 2000b). This might reflect a combination of educational attainment and the demands of the labour market at the time of migration. In addition, Nazroo explains that for the same occupational class, some minority ethnic groups have on average lower income than the majority ethnic group (Nazroo, 2006). These examples point to the non-equivalence of specific SES measures across ethnic groups. The potential non-equivalence of SES indicators across ethnic groups is an issue that might be more salient in the elderly. As previously explained, Evandrou's study found similar educational levels by ethnic group. However, she also showed that Indian elders were more likely to be home owners than White and other ethnic groups and also twice as likely as Black Caribbean elders to live in a household with a car (Evandrou, 2000b). These material asset differences could be the results of cultural and household composition differences. Finally, the level of income in minority elders is a complex issue as they might not have contributed enough time since migration to be entitled to a full pension (Blakemore, 1985, Evandrou, 2000b). Although multigenerational families can be seen as protective for elderly minorities, this might also come with a lack of control over financial resources (Blakemore, 1985) and household assets.

Many research studies attribute ethnic differences in health to SES differences either as the main contributor or as part of the explanation (Crimmins et al., 2004, Smith, 2000, Smith and Kington, 1997, Nazroo, 2001, Williams et al., 2016). To account for SES when looking at ethnic differences in health, research studies tend to adjust for a single or multiple measures of socio-economic status (Fischbacher et al., 2014, Harding and Balarajan, 2001a). Although some studies on the Black-White differential in health in the US show SES as the main contributor to ethnic differences in health (Smith and Kington, 1997, Smith, 2000, Hayward et al., 2000), most of the literature points to the fact that controlling for SES does not fully explain ethnic differences in health even after including multiple measures of SES to reduce residual SES confounding (Nazroo, 2001, Crimmins et al., 2004, Fischbacher et al., 2014).

Nazroo showed how traditional measures of SES such as occupational class and tenure made little difference to observed ethnic differences in a range of health outcomes and further showed more promising results when using a more tailored socio-economic indicator: "standard of living" (Nazroo, 2001). He explained that the disadvantage faced by minority ethnic groups has a multi-dimensional nature as they might experience more adversity and lower actual SES for the same level of measured SES compared to the White majority. The nature of the

disadvantage can be related to discrimination, inequality perception and geographical density. Consequently, accounting for SES in ethnic-health studies, even with the best measure, will never “be completely done” (Nazroo, 2001). In addition, the ability of SES to explain ethnic differences in health might depend on the combination of the SES measure(s) and the health indicator used and might be context-dependent. If ethnic differences in SES do not fully account for ethnic differences in health, this questions the ability of SES to explain ethnic differences in health and other explanations might come into play, including cultural and genetic factors (Nazroo, 2001). One could also wonder whether SES is associated with health similarly across ethnic groups and if this association follows a similar gradient. There is evidence both in the US and the UK that SES is better at predicting health in the White majority than in non-White minority groups (Bécares et al., 2012a, Crimmins et al., 2004, Fischbacher et al., 2014, Williams et al., 2016). In Scotland, Fischbacher et al. showed how different SES measures were inconsistently associated with CVD hospitalisation and death across ethnic groups (Fischbacher et al., 2014).

Finally, Williams et al. explored what is left to influence those ethnic disparities if SES does not fully account for the ethnic differential in health (Williams et al., 2016). Summarising the literature, they suggested four reasons: adversity through the life course in addition to current SES, non-equivalence of SES across ethnic groups, the burden of racism (exposure to both institutionalised and interpersonal discrimination) and exposure to psychosocial stressors (Williams et al., 2016). These four reasons can be considered in addition to low SES in potentially explaining worse health in minorities compared to the White majority. However, when non-White minorities fare better and experience better health outcomes than the White majority at comparatively lower level of SES, it casts doubt on the ability of low SES and the additional four reasons mentioned above to explain the health advantage seen in these ethnic groups. The “Hispanic paradox” (see section 2.2.1) is a good example which shows that low SES (disadvantage: low in Hispanics compared to Whites) cannot explain ethnic differences in health (advantage: better mortality in Hispanics than Whites). In that case, other mechanisms need to be considered.

2.3.2. Acculturation hypothesis and migrant generations

Acculturation refers to a culture change due to the contact of two distinct cultural groups (Berry, 1992). At the individual level, psychological changes can occur which include changes in values, attitudes and behaviours. This is referred to as behavioural shifts.

Acculturation stress may also appear through social and psychological problems during the acculturation process. Acculturation can be seen as unidimensional or bidimensional (Lara et al., 2005). The unidimensional model refers to an individual degree of acculturation into the new culture in a continuous fashion. The bidimensional model entails a combination of the old and new cultures to different degrees: assimilation (adopting the new culture), separation (keeping the original culture), integration (adopting both) and marginalisation (excluding both).

In the context of researching ethnic differences in health, acculturation is often considered as the process through which ethnic minorities tend to adopt the cultural traits and behaviours of the population of the receiving country. The acculturation hypothesis focuses predominantly on migrants and tends to use duration of residence as a proxy for exposure to the new culture of the country of residence. However, migrant generations are also often used to understand intergenerational processes of acculturation. The acculturation hypothesis posits that as length of stay increases in the receiving country, minorities will increasingly adopt the health behaviours and health risk profile of the native population. In this context, Abraido-Lanza et al. referred to the acculturation process as “the health behaviors and acculturation hypotheses” (Abraído-Lanza et al., 2005). Although ethnic minorities’ health status tends to decline with greater acculturation, this process can be associated with both positive and negative effects on health behaviours (Abraído-Lanza et al., 2005, Lara et al., 2005). Positive effects of acculturation include an increased level of physical activity (Abraído-Lanza et al., 2005), health education and promotion as well as an increased use of health care services (Lara et al., 2005). However, the acculturation process is often viewed in Western societies as detrimental for ethnic minorities. Indeed, if we assume an initial “healthy migrant effect” (see section 2.3.3) and healthy habits, it is hypothesised that, as they acculturate, ethnic minorities will tend to adopt unhealthy behaviours such as a bad diet, an increased prevalence of smoking and alcohol consumption.

The acculturation hypothesis is generally assessed in relation to duration of residence in the receiving country but also through looking at second and third generations as an extension of the potential disappearance of the cultural buffer. Although descendants of migrants might have inherited some of the norms and culture passed on by their ancestors, they are likely to be greatly acculturated to their country of residence. Hence, the effect of different levels of acculturation on health linked to different patterns of health behaviours can be studied using migrant generations.

The acculturation hypothesis has a temporal and spatial aspect. It focuses on individual health trajectories influenced over time by the fact of living in a particular space with people of a particular culture. This trajectory is usually explored for a particular minority ethnic group within the context of the receiving country. However, Jasso argues that the health trajectory of immigrants exposed to specific factors such as health behaviour, health environment and health care system in the receiving country matters in relation to the health trajectory of similar non-migrants in the sending countries to assess comparatively the process of acculturation (Jasso et al., 2004). However, most research explores the effect of acculturation through assessing whether the health behaviours and health status of the minorities converge towards that of the majority population in the receiving country.

Initial research by Ziegler et al. explored the effect of duration of residence in the “West” and degree of Western origin, as a proxy for Western lifestyles, on the risk of breast cancer in Asian-American women (Chinese, Japanese and Filipino women living in the US) (Ziegler et al., 1993). They found an 80% higher risk of breast cancer incidence in Asian women who lived more than 7 years compared to those who lived less than 7 years in the West (US). They also compared the risk of breast cancer in second, third and fourth generations to that of Asian immigrants and found an overall 60% higher risk of breast cancer incidence in Asian women born in the West compared to those born in the East (Asia). They found a gradient in risk as the number of parents and grandparents being born in the West increased, with higher breast cancer incidence than Whites for those with grandparents born in the West. In conclusion, this study supports increased risks of breast cancer with greater acculturation in Asian women living in the US.

In Western societies, an expected pattern of health associated with the acculturation process is a convergence of the health risk in a particular ethnic group towards the health level of the native population. When the health risk in minorities is low to start with compared to the native population and for a particular health outcome (e.g. cancer or mortality), it is expected to converge toward the health risk of the native population as duration of residence increase and over generations (Harding, 2003, Harding, 2004, Harding et al., 1996, Ziegler et al., 1993). However, some evidence points to higher risks in descendants compared to the native population. For example, when the risk for a particular health indicator is already high in a specific ethnic group compared to the native population, rather than observing a convergence toward the health level of the population, some of the literature points to a continued increased risk in subsequent generations (Harding and Balarajan, 2001b). This supports the idea that

greater acculturation in minorities living in Western societies leads to a decline in health status in both migrants and subsequent generations rather than a convergence.

A strand of the literature focuses on the effect of acculturation on mortality outcome. The focus on mortality is used here to shed light on a few key points. Initial evidence related to mortality as an outcome in the UK setting used both duration of residence in England and Wales (Harding, 2003, Harding, 2004) and migrant generations (Harding and Balarajan, 2001b, Harding et al., 1996). For example, looking at deaths from 1971 to 2000, Harding found that mortality from all causes and more specifically from cardiovascular disease and cancer increased with duration of residence in South Asian migrants (Harding, 2003). In contrast, she found no effect of duration of residence on mortality in Caribbean migrants, apart in the specific age group 45-54 years and for circulatory mortality (primarily stroke) (Harding, 2004). This points to varied effect of duration of residence on mortality for different ethnic groups in the UK. Further research in a UK setting found a high mortality risk in Irish migrants compared to the rest of the population in England and Wales (Harding and Balarajan, 2001b). This higher mortality risk was even more pronounced in Irish of second and third generations and adjustment for socio-economic status attenuated this higher mortality risk in immigrants only. This supports a greater health disadvantage in descendants. The literature in the European context provides mixed results in relation to the mortality outcomes of descendants when immigrants experience a mortality advantage to start with. In the Netherlands, Stirbu et al. found indications of convergence of lower cancer mortality rates towards the rates of the native Dutch population in immigrants as duration of residence increased and in second generations (Stirbu et al., 2006). For both first and second migrant generations, cancer mortality did not reach the level of the native Dutch population and remained lower. In Belgium, Vandenheede et al. found a mortality advantage in immigrants wearing off with length of stay and a mortality disadvantage in second generations disappearing when SES was controlled for, for both Western and non-Western second generations (Vandenheede et al., 2015). Similarly to the Belgium example, Wallace found immigrants to have a lower mortality in England and Wales while descendants had a higher mortality compared to the host population (Wallace, 2016). The mortality disadvantage observed in descendants disappeared when SES was accounted for. The mortality findings in descendants might reflect the higher deprivation level that subsequent generations are exposed to through their life course.

In summary, the convergence or decline in health with greater exposure to the country of residence seems to occur in both migrants and descendants. It can vary by ethnic group within a specific context. Evidence points to descendants having the greatest disadvantage which tends to disappear once SES is accounted for.

Finally, the direction of the effect of acculturation on health is likely to be shaped by the original level of risk when entering the country of residence for specific ethnic groups and for specific health outcomes comparatively to the native population. The rapidity of the acculturation process might also be ethnic-dependent. For example, Smith et al. showed that the level of obesity in non-White ethnic minorities, with the exception of the Black Caribbean group, converged towards the risk of the White majority in England with greater acculturation (Smith et al., 2012). Indian and Chinese second generations were more likely to be obese than respectively Indian and Chinese immigrants and to a greater extent than that in other minority ethnic groups (Smith et al., 2012).

2.3.3. Migrant health selection hypothesis

The health selection hypotheses relate to the health status of a migrant at the time of migration. Both in-migration to the new country of residence and out-migration from the country of settlement to the country of origin generate health selection hypotheses.

Ethnic inequalities in health are strongly linked to migration processes. The health selection hypotheses by definition focuses only on immigrants as a single homogeneous group rather than their descendants. Although the hypotheses focus on migrants, those processes are likely to differ by ethnic group due to varied cultures and beliefs in the benefits of migration. Different ethnic groups will also have different reasons for and timings of migration depending on the context in both their country of origin and country of destination. Jasso also outlines that the health selection effect of migrants might differ by the reason for migration (Jasso et al., 2004). For example, those migrating in old age might well do so attracted by access to better health care in the receiving country. Furthermore, refugees might experience bad health due to difficult experiences in their country of origin and during the process of migration. Hence, the relevance of the health selection hypotheses is likely to vary according to the reason for migration.

The first hypothesis focuses on the health status of migrants when moving from the country of origin to a new country of residence. The so-called “healthy migrant effect” (or

“healthy immigrant effect”) proposes that those who move are likely to be healthier than the ones they leave behind, as the process of migration requires a certain level of health and wealth (Jasso et al., 2004, Marmot et al., 1984). Two processes have been distinguished in the health selection of new migrants: individual self-selection and government health screening to enter the new country of residence (Jasso et al., 2004). The individual self-selection process relies on the assumption that “good health fosters mobility; ill health limits mobility” (Wallace and Kulu, 2014b). Hence, good health can encourage the decision to move. This assumption of selectivity can hold for both internal and international migrations and is thought to be positively associated with the distance of migration (Rubalcava et al., 2008). Health screening can happen prior to migration for potential migrants or at the time of entry into the receiving country. Although it might depend on the political context of the receiving country, health screening is likely to account for a very small part of the health selection process (Jasso et al., 2004). It is also worth noting that a receiving country selecting individuals for economic reasons based on qualifications and higher levels of education will indirectly select individuals with better health as higher education levels and socio-economic status are strongly associated with better health status. Those who migrate are more likely to have a set of characteristics that predispose them to better health in the long run including a determination and open mindedness to move beyond their familiar environment. This supports the idea of migrants being healthier than those they leave behind but not necessarily healthier than the population they come to live with in the country of settlement.

However, the concept of the “healthy migrant” has been extended to migrants being healthier generally and healthier than the population of the country they move to. Indeed, it can be argued that if the sending country has worse average health than the receiving country and the migrants have better health than the native population born in the receiving country, then migrants might have better health than those they have left behind in the sending country (Jasso et al., 2004). Wallace and Kulu refer to the health advantage of migrants over both those left behind in their country of origin and the residents of the receiving country as the “true healthy migrant effect” (Wallace and Kulu, 2014b).

Empirical evidence testing the “healthy migrant effect” (better health in migrants compared to their peers in their country of origin) is limited. Using standardised mortality ratios, initial work by Marmot et al. explored the mortality of migrants in England and Wales compared to that of their countries of birth (Marmot et al., 1984). The authors found better outcomes in

migrant men from Italy, Poland, the Indian subcontinent and the Caribbean compared to that of their respective countries of origin. The exception was that Irish migrants had a higher mortality level than those in Ireland. These ethnic differences in the health of different group of migrants and for this particular example of Irish migrants in the UK, was later attributed to a distance explanation arguing that a short migration distance makes the “healthy migrant effect” less likely as the cost of migration between the two countries is low (Jasso et al., 2004). Since Marmot et al., a few research studies have attempted to test the “healthy migrant effect”. Rubalcava et al. found weak support for the hypothesis with better health not necessarily predicting subsequent migration to the US in 15 to 29-year-old Mexican males and females (Rubalcava et al., 2008). Razum et al. found support for the “healthy migrant effect” in that the mortality of Turkish residents in Germany was low compared to that of Turkish residents in Ankara, in Turkey and also lower than that of Germans in Germany (Razum et al., 1998).

In response to the lack of health and migration data at an international level enabling researchers to follow mobile populations and their health status from the country of origin to the receiving country, a body of evidence has emerged testing the “healthy migrant effect” hypothesis within countries. Once international migration is excluded, the health effect of internal migration can be analysed between two geographical areas of the same country for example between England and Scotland within the UK, between Northern Sweden and Southern Sweden and between rural and urban areas in China and Indonesia (Andersson and Drefahl, 2017, Lu, 2008, Lu and Qin, 2014, Wallace and Kulu, 2014b). Evidence of a “healthy migrant effect” was found in adult migrants (aged 18-64 years) within the UK, both from Scotland to England (compared to those who stayed in Scotland) as well as from England to Scotland (compared to those who stayed in England) (Wallace and Kulu, 2014b). Similarly, in China, evidence showed a “healthy migrant effect” among rural migrants to urban areas compared to those who stayed in rural areas which included a gradient of the effect as distance increased (Lu and Qin, 2014). In the case of Sweden, however, there was no evidence of a “healthy migrant effect” in Northern Swedes of working age who moved to Southern Sweden compared to stayers (Andersson and Drefahl, 2017). Movers from Northern Sweden also showed a higher risk of mortality than stayers once education level was accounted for. Overall, the findings suggest that making a move within countries might require less of an advantage in health, economic status and resourcefulness generally.

The second theory relates to return migration in an unhealthy state. The “salmon bias” hypothesis is a key element of the unhealthy return migration theory. It proposes that ill health precipitates return migration to the country of origin with the idea of a wish to die at home or, if ill, a preference to be “at home”. The “salmon bias” terminology was proposed by Pablos-Mendez in 1994 based on the idea of the salmon run and the compulsion to go back to their birthplace and die (Pablos-Mendez, 1994). However, reasons other than ill health in older age can lead to unhealthy return migration. An unhealthier individual more generally might experience more difficulties in securing a job and the stability necessary to settle in the receiving country and could engage in return migration to find support and security back home. Health might not be the only reason for the inability to settle in the receiving country. An “unsuccessful migration” or negative experience of migration can leave scars. A negative experience of migration can be due to many factors such as language difficulties, culture and norm differences, a lack of transferability or recognition of skills, unemployment, and experiences of deprivation, discrimination, and racism. Those who engage in return migration due to unsuccessful settlement in the receiving country are likely to come back disillusioned, scarred and in worse health compared to the successful migrants who settled.

A few studies have tested the “salmon bias” hypothesis whereby ill health precipitates return migration to the country of origin when death is imminent. Turra and Elo tested this hypothesis using beneficiary data in the US (population aged 65 years and above) (Turra and Elo, 2008). The key advantage of beneficiary data is that individuals’ migration and health status can be tracked to the country people move to outside the US and the mortality outcome of migrants who engaged in return migration can be compared to those who remained in the US. The authors found a higher mortality in those who returned from the US compared to those who stayed in the US, in both foreign-born Hispanic Whites and, to a lesser extent, in foreign-born non-Hispanic Whites. Higher mortality was also found among recent returnees to their country of origin i.e. within a year of return migration, in line with the hypothesis of precipitated return migration due to deteriorating health. This supports the hypothesis of a “salmon bias” phenomenon in foreign-born elders.

Another US study aimed to test the “salmon bias” hypothesis in relation to morbidity. Ullmann et al. focused on the health of returnees from the US to Mexico and found a higher prevalence of mental health disorder, smoking and heart disease in returnees compared to stayers in the US. However, the authors could not distinguish whether the results were

attributable to the health effect of residing in the US (acculturation effect) or a negative health selection of Mexican migrants (salmon bias) who then returned to Mexico (Ullmann et al., 2011). This highlights the need to develop longitudinal studies in both sending and recipient countries. Further evidence on European Turkish migrants in the European context does not provide support for a “salmon bias” phenomenon. For example, Baykara-Krumme examined whether elderly Turkish migrants to Europe decide to live in Turkey (remigrants), stay in their European receiving country (immigrants) or both (transmigrants) (Baykara-Krumme, 2013). She found no evidence of a health selection process in the decision to stay, return or both for Turkish migrants aged 65 years and above. Similarly, Razum et al. conducted focus group sessions with returnees, Turkish male migrants from Germany to Turkey, and found varied reasons for return migration such as lack of economic success, emotional and value-oriented reasons. There was no indication of a return due to the desire to die at home although the returnees who did not ‘succeed’ in Germany were likely to be at higher risk of unhealthy return migration (Razum et al., 2005).

Due to limited international data to test health selection processes, a few studies have also tested the “salmon bias” in an internal migration context (Andersson and Drefahl, 2017, Lu, 2008, Lu and Qin, 2014, Wallace and Kulu, 2014b). Based on self-assessed health, Wallace and Kulu found no evidence of a “salmon bias” between Scotland and England which they attributed to possible similarities of language, culture and government as well as short distance of migration (Wallace and Kulu, 2014b). In contrast, studies in the Chinese context using self-reported health and in the Swedish context using mortality found evidence of an unhealthy return migration to the birth place (Andersson and Drefahl, 2017, Lu and Qin, 2014).

Under the “salmon bias” hypothesis, unhealthy people would emigrate and die abroad soon after. If emigrations and deaths abroad are not recorded in the receiving country, this creates a sample of “statistically immortal” migrants thus resulting in numerator and denominator biases and consequently, in an artificial mortality advantage in migrant populations. In an attempt to test whether the “salmon bias” could explain out the mortality advantage in Hispanics, Turra and Elo added the death data for both US residents and foreign-residents into a sensitivity analysis of ethnic differences in mortality (Turra and Elo, 2008). The mortality advantage of foreign-born Hispanics in the US was not explained when deaths abroad where included in the analysis. Some studies have also attempted to indirectly test the “salmon bias” effect by comparing ethnic groups to the native population within the receiving country

(Abraido-Lanza et al., 1999, Vandenheede et al., 2015). Although not assessing the health status of return migrants in their country of origin, this research provided indications that the mortality advantage seen in minorities was not or not fully the result of a salmon bias effect (Abraido-Lanza et al., 1999, Vandenheede et al., 2015). So far, the evidence of a “salmon bias” phenomenon that could explain a migrant mortality advantage is weak and requires further investigation.

In summary, both the “healthy migrant effect” and the unhealthy return migration including the “salmon bias” in later life have been offered as explanation for a migrant mortality advantage as it is expected that the healthiest come and the healthiest of the healthiest remain. However, current empirical evidence remains limited to conclude on the contribution of the health selection hypotheses in explaining the mortality patterns observed.

Finally, a few studies have ventured a third health selection hypothesis of migrants which cannot be offered as an explanation for the mortality advantage in migrants: a healthy return migration to the country of origin (Razum et al., 2005, Sander, 2007). This alternative theory proposes that ill and more frail migrants are less likely to engage in return migration due to their bad health restricting mobility and the availability and access to a good health care system in the receiving country (Razum et al., 2005). In this hypothesis, only the healthier and wealthier can afford the luxury to move back to their country of origin in later life and after a successful economic gain while in the receiving country. This supports that the “healthy migrant effect” holds whatever the direction of migration (from the country of origin to the receiving country or back to the country of origin) in line with the idea that healthiness fosters mobility.

2.3.4. Other theories

In addition to theories of socio-economic deprivation, acculturation and migrant health selection, other mechanisms might play a role in shaping ethnic differences in health. This section summarises a few additional theories of ethnic differences in health, not directly tested in this thesis, that are likely to influence the findings of this research.

Discrimination, racism and access to health care services

Discrimination and racism can occur at different levels and manifest in different ways. Two main types of discrimination can be distinguished: interpersonal and institutional (Karlsen and Nazroo, 2002). Interpersonal racism happens between individuals while institutionalised discrimination entails practices or policies emanating from organizational structures. Many

studies have looked at perceived discrimination overall and the process by which it might impact on health (Paradies, 2006, Pascoe and Smart Richman, 2009, Williams and Mohammed, 2009). They found that perceived discrimination affects both physical and mental health negatively. The pathway by which discrimination affects health is through increased level of physical and psychological stress (Pascoe and Smart Richman, 2009). For example, increased perception of discrimination increases physiological stress responses which in turn raise the likelihood of unhealthy behaviours resulting in worse health outcomes. Higher stress level might also affect cortisol level and allostatic load which is associated with varied health problems. Discrimination might also restrict the access to better job prospects and overall socio-economic status, resulting in less resources and higher stress level which in turns would affect health.

In relation to ethnicity, the relationship between perceived discrimination and health has been investigated and evidenced in many contexts (Borrell et al., 2015, Brondolo et al., 2011, Karlsen and Nazroo, 2002, Mays et al., 2007). However, evidence on whether the strength of the association is similar across ethnic groups is mixed (Brondolo et al., 2011, Karlsen and Nazroo, 2002). For example, a UK study tested the influence of reported experience of racism and perception of discrimination on health for all ethnic groups and found that both measures of discrimination independently predicted worse health outcomes for a range of health indicators (Karlsen and Nazroo, 2002). The authors also explored the effect of experience of racism and perception of discrimination on self-reported health for particular ethnic groups. They found poorer reported health in those who perceived most employers as racist in each ethnic group but only significantly so in Pakistani and Bangladeshi respondents. In relation to experience of racism, those who reported experiencing a physical attack were overall more likely to report poorer health but it was statistically significant in Indian populations only. The effect of these dimensions of discrimination on health were less conclusive in Caribbean and Chinese groups, possibly due to small sample sizes. This pointed to possible ethnic variations in the strength of the discrimination-health relationship. In contrast, a US study found significant association between perceived racism and self-reported health in each ethnic group (Asian, Black and Latino adults) with little evidence of a differential association across ethnic groups (Brondolo et al., 2011).

Finally, institutionalised discrimination can result in ethnic differences in access to healthcare (Nazroo, 2014). In Scotland (and the UK more generally), there is free universal healthcare provided by the National Health Service. Despite health services being 'free at the

point of use', access to care might remain unequal due to barriers such as language proficiency. Evidence from the Health Survey for England does not suggest unequal access to GP services for minority ethnic groups (Nazroo et al., 2009). However, available evidence both in England and Scotland suggests a more complex picture of unequal access operating at different level of healthcare and healthcare settings (Katikireddi et al., 2018, McFarland et al., 1989, Nazroo et al., 2009, Worth et al., 2009).

Health behaviour

Health behaviour refers to a behaviour that affects individual's physical health or that is perceived to affect health (Sutton, 2008). Four main health behaviours seen as key risk factors for specific diseases have been offered as explanation for ethnic differences in health: diet, physical activity, smoking and alcohol consumption (Berkman and Mullen, 1997, Winkleby and Cubbin, 2004). Diet and physical activity are linked to obesity, hypertension, diabetes and cardiovascular disease (Roberts and Barnard, 2005). Smoking is a strong risk factor for lung cancer (Furrukh, 2013) as well as cardiovascular disease and mortality even at older age (Mons et al., 2015, Müezziner et al., 2015). Alcohol consumption has been associated with elevated risk of many chronic diseases including specific cancers, cardiovascular disease and liver disease (Rehm, 2011, Shield et al., 2013). Assessing differences of these behaviours is complex due to the multiplicity of indicators and their varied dimensions of quality, frequency and quantity (Chartier and Caetano, 2009, Chowbey and Harrop, 2016, Fischbacher et al., 2004, Rao et al., 2015).

In the UK, health surveys provide useful information on health behaviours by ethnicity (Sproston and Mindell, 2006, Whybrow et al., 2012). For example, they showed that minority ethnic groups were more likely to follow the recommendation of consuming five or more portions of fruit and vegetable a day compared to the general population with higher proportions in Chinese and South Asian populations (Sproston and Mindell, 2006, Whybrow et al., 2012). In contrast, further research in the UK points to lower level of consumption of fruits and vegetables in South Asians with the highest level in Chinese followed by Afro-Caribbean (Chowbey and Harrop, 2016, Leung and Stanner, 2011). Lower level of recommended physical activity (at least 5 days of minimum 30 minutes of moderate to vigorous PA per week) were found in Chinese and South Asians and particularly so in Bangladeshi and Pakistani women in England (Sproston and Mindell, 2006). These findings were supported by a few smaller scale studies in the UK (Hayes et al., 2002, Fischbacher et al., 2004, Williams et al., 2010). In Scotland,

Pakistani respondents were the least likely to achieve the recommended physical activity levels and to participate in sport while Chinese respondents had the highest participation in sport (Whybrow et al., 2012). Cigarette smoking prevalence was higher in Irish and Black Caribbean as well as Pakistani and Bangladeshi men compared to the general population in England (Sproston and Mindell, 2006). With the exception of the Irish, most minority ethnic groups in England were less likely to drink alcohol and Pakistani and Bangladeshi populations had the highest proportion of non-drinkers (Sproston and Mindell, 2006). In Scotland, similar patterns of alcohol consumption by ethnicity were found however patterns of smoking by ethnicity differed (Whybrow et al., 2012). Indeed, Pakistani and Chinese respondents were the least likely to drink alcohol in Scotland but also the least likely to smoke. However, the sexes were aggregated in the Scottish report which might hide great variation in smoking prevalence between men and women from these ethnic groups.

As data sources on health behaviours by ethnicity and related health outcomes tend to be separate, very few studies have tested the direct relationship between ethnic differences in health behaviours and ethnic differences in health outcomes (Fenelon, 2013). Identifying the direct role of health behaviours on ethnic health inequalities remains relevant as it can inform interventions aiming to reduce the associated health risks. For example, a study by Fenelon confirmed a life expectancy advantage in Hispanics in the US as well as a lower burden of smoking especially among the foreign-born (Fenelon, 2013). US-born and foreign-born Mexican Americans had low smoking-attributable mortality in compared to Whites. Testing the contribution of smoking to the mortality advantage in Hispanics, the author found that smoking explained more than 60% of the Mexican-American mortality advantage over Whites (Fenelon, 2013). In Scotland, a pilot study linked health outcomes to risk factors including health behaviours (Douglas et al., 2015). They found that adjusting for smoking did not explain the high risks of CVD in Pakistani men compared to White Scottish men as they had somewhat lower smoking level. However, given the low prevalence of smoking in Pakistani women, adjusting for smoking increased their already high risks of CVD in comparison to White Scottish women. This later finding could have worrying implications if women of Pakistani origin were to acculturate and adopt the smoking patterns of the Scottish population.

Social networks

Social networks relate to social ties between individuals. Most research measures social network via the numbers of friends and relatives, marital status, membership in religious and

voluntary associations (Berkman et al., 2000). Social networks have long been associated with health (Berkman et al., 2000, Berkman and Syme, 1979, Christakis and Allison, 2006, Smith and Christakis, 2008). Research on social networks tends to emphasize how social support matters in relation to health especially how it acts as a stress-buffering i.e. protective of the negative effect of stress on health (Martire and Franks, 2014).

Initial research on ethnic differences in health has often described differences in social networks by ethnic group (Berkman and Mullen, 1997, Blakemore, 1985). In relations to minority ethnic groups, there is a preconceived idea that “they look after own” (Murray and Brown, 1998). Indeed, in relation to South Asian communities, it is thought that intergenerational families are supportive and thus, protective of worse health outcome. South Asians are less likely to live alone compared to their White counterparts in the UK (Donaldson, 1986, Evandrou, 2000b) but it does not mean they are safe from being or feeling isolated. In fact, Willis et al. found no indication of ethnic differences in instrumental support within the household in the UK except in the Indian group (Willis et al., 2013). Furthermore, Blakemore warns about the meaning of the frequency of interactions with relatives across ethnic groups and explains that the size of the social network as well as residing with family do not inform on the quality of relationships (Blakemore, 1985). A distinction needs to be made between social network and social support and their positive and negative effects on health as not all ties might be supportive. In her recent thesis on how social network relates to the use of mental health services in Pakistani women, Kapadia summarises the influence of social network into three aspects: perception of social support, the network size and the frequency of contact with relative and friends (Kapadia, 2015). She found that Pakistani women experience lower levels of social support compared to the white majority in the UK, although not necessarily in comparison to other minority ethnic groups. Furthermore, Pakistani and Indian women were more likely to be married than the other ethnic groups, but there were higher proportions of women from Black African, Indian, White Irish and White British ethnicity who felt their partners understood them and that they could rely on them if they had difficulties compared to women of Pakistani ethnicity.

A few studies have tested whether social network and support can explain ethnic inequalities in health. For example, using marital status as a proxy for a protective social marker, Maxwell and Harding showed that marital status did not explain ethnic differences in mortality in the UK (Maxwell and Harding, 1998). Kapadia found ethnic differences in mental health services use with Pakistani and Bangladeshi women being the least likely to use mental health

services in the UK (Kapadia, 2015). These ethnic differences in services use remained after accounting for social network characteristics (size, content, contact and support).

Genetic factors and interaction with the environment

There is a common misunderstanding that the genotype solely determines the phenotype but the phenotype is also influenced by the environment (Pearce et al., 2004). Similarly, genetics can determine health to some extent but available evidence points to its influence to be secondary in comparison to social and environmental determinants (Sankar et al., 2004). Its contribution to health inequalities overall is likely to be small (Sankar et al., 2004). In addition, most diseases related to genetic variability tend to be the results of a gene-environment interaction (Pearce et al., 2004). An example of gene-environment interaction relates to the fast increase of particular diseases in our societies. In 2008, the Centers for Diseases Control and Prevention (CDC) in the US reported a 90% increase of diabetes prevalence in one decade (from 4.8 per 1000 in 1995-1997 to 9.1 in 2005-2007) (Klonoff, 2009). Some people might be genetically predisposed to diabetes. However, at a population level, genetic propensity for a particular disease takes thousands of year to change. In contrast, lifestyle and exposure to obesogenic environments have changed rapidly in the last few decades. Hence, diabetes increase is likely due to a change in environmental exposures affecting primarily those that are more predisposed to the disease rather than a change in genetic predisposition at the population level. Genetics plays a role in the health risk for particular diseases but it is far from being the only determinant.

There is genetic heterogeneity within a population. When comparing two populations, this genetic diversity tends to overlap. Hence, it is commonly accepted that there is greater genetic variability within populations than between populations. In addition, health inequalities are primarily determined by social and environmental factors (Bartley, 2016). Therefore, very little genetic variation is expected to explain differences in health between ethnic groups. For particular diseases, there might be higher genetic propensity in particular ethnic groups. This higher genetic risk tends to be due to selection processes operating in interaction with the environment. A typical example is sickle cell anaemia. Sickle cell anaemia is seen in people living in Africa and those of African origin but not solely (Rees et al., 2010). For example, the gene can be found in all ethnic groups in the UK (National Institute for Health and Care Excellence, 2014). The genetic transmission of sickle cell traits was favoured in specific regions due to its protective effect against malaria (Rees et al., 2010). Consequently, the higher prevalence of sickle cell

anaemia in specific group of people is due to selective processes in response to environmental threats. It is also worth noting that sickle cell anaemia remains a rare disease in the UK (estimated prevalence of 18-22 cases per 100,000 population). Hence, ethnic differences in this particular disease have little capacity to contribute to overall ethnic differences in health.

2.4. Summary and research directions

Some gaps in the literature were identified in section 2.2. First, the review of available literature on ethnic differences in general measures of morbidity highlighted the need to understand ethnic inequalities in self-assessed health in Scotland with a finer analysis using data at the individual level. It also pointed to explore under-researched health inequalities in older minorities. The availability of individual level data and risk factor data also provides the opportunity to explore the mechanisms underlying the observed ethnic inequalities in reported health. Section 2.3 outlined key theories that have the potential to explain ethnic differences in health such as socio-economic status and acculturation theories. The contribution these theories make in explaining ethnic health inequalities is evaluated in this thesis.

Second, evidence in relation to mortality showed a mortality advantage in most minority ethnic groups in Scotland which contrasted with the available evidence in relation to their morbidity. Evidence of a morbidity-mortality contrast in particular ethnic or migrant groups in particular contexts is small in the international context and it mostly relies on morbidity data and mortality data gathered from different samples. Hence, there is space for a substantive contribution in researching the ethnic morbidity-mortality contrast in Scotland as well as a methodological contribution enabled by the use a unique population source.

Third, if ethnic patterns in morbidity and mortality differ and the contrast is real, one can wonder whether reported health relates to mortality similarly across ethnic groups. International research on the ethnic differential in the SAH-mortality association is restricted to a few US studies which points to a differential SAH-mortality association between blacks and whites but this has yet to be explored in the UK context.

Finally, to understand whether the contrast between reported morbidity and mortality arises from cultural differences in reporting health or whether a real morbidity-mortality paradox occurs in particular ethnic groups in Scotland, ethnic differences in health using a more objective measure of morbidity need to be investigated. As we age, health tend to be determined by more than one disease. Hence, studying ethnic differences in multimorbidity in

Scotland is timely and has not yet been explored in the UK setting. This investigation has the ability to inform about a more severe morbidity disadvantage in particular minority ethnic groups while providing evidence on whether a “morbidity-mortality paradox” is supported for specific ethnic groups in Scotland.

In summary, the purpose of this thesis is to contribute to understanding the ethnic morbidity-mortality paradox in Scotland by providing stronger evidence of ethnic differences in morbidity and by understanding the relationship between morbidity and mortality across ethnic groups. This thesis seeks to answer the following research questions:

1. How do patterns of reported morbidity by ethnicity compare to patterns of mortality by ethnicity based on the same population source?
2. Do mechanisms thought to shape ethnic inequalities in health, such as socio-economic status and migrant generations, contribute to explaining ethnic differences in reported morbidity?
3. Does reported morbidity relate to mortality consistently across ethnic groups?
4. Does using a doctor-diagnosed measure of health provide similar patterns of ethnic differences in morbidity as using reported morbidity? Does it support a morbidity-mortality paradox in specific ethnic groups in Scotland?

CHAPTER 3

3. Research design, data and methods

This thesis extends the research on ethnic inequalities in morbidity and mortality in the UK and contributes to the under-researched field of an ethnic specific morbidity-mortality paradox. The literature review of ethnic differences in health (chapter 2) identified a discrepancy whereby evidence of a reported morbidity disadvantage in specific ethnic minorities compared to the majority population contrasted with their mortality advantage. However, the evidence related to morbidity patterns and mortality patterns came from different data and population sources. This thesis responds to the substantive and methodological gaps in the literature by adopting cross-sectional and longitudinal approaches based on data linkages to interrogate the ethnic morbidity–mortality contrast.

This chapter explains the research design, data source and methods employed to investigate the research questions of this thesis. Section 3.1 explains the research design appropriate to explore them. Then, section 3.2 highlights the key Scottish data sources that are fit for purpose. Section 3.3 follows on to describe the data source used in this thesis and how the key concepts are operationalised. It also presents the socio-demographic profile of the population source. Finally, section 3.4 discusses the methods best-adapted for the investigation of each research question and introduces the refined research questions.

3.1. Research design and secondary data analysis

The research questions presented in section 2.4 are best-addressed by a cross-sectional and longitudinal research design and a quantitative approach. The aim to draw generalisable conclusions on outcomes across ethnic groups in Scotland necessitates a quantitative approach based on nationally representative data. Sufficient sample size is necessary to robustly analyse health outcomes across detailed ethnic categorisations. Collection of new data of this magnitude is not feasible or practical given the scope and aims of this PhD thesis, but it would also ignore the excellent and under-used secondary data on ethnicity and health that is available in the Scottish context.

Secondary data analysis is the analysis of data collected by another investigator or institution for another purpose. In public health, epidemiology, and social sciences of health, sources of secondary data usually come from information collected by governments such as censuses, surveys, national health data collected for administrative and monitoring purposes,

or from information collected by research institutions and researchers for other research purposes. Using secondary data analysis has the advantage of maximising the use of existing data with no extra time and resources allocated to data collection. However, because the data were collected for other purposes, they might not match perfectly with the intended research design. For example, the choice of variables and their categorisations might not fit adequately to the conceptualisation of the research. Therefore, the concepts tied to the research questions need to be operationalised according to the data collected from the most promising data sources.

3.2. Data selection

This section explains the rationale for the data source selected. It draws on the Scottish data sources able to provide data by ethnic group to investigate ethnic inequalities in morbidity and mortality from a unique population source and with sufficient sample size. The strength of the data sources is also evaluated according to the quality of the information collected and whether it contains reliable measures of both general morbidity and mortality.

In relation to the quality of the information collected, a key element is the reliability of the ethnicity variable, how it is collected and its fine granularity. Granularity of ethnic groups is required to ensure minimising the heterogeneous nature of aggregated ethnic groups. Analysing relatively homogenous groups in terms of culture and ancestry based on self-reported ethnicity will produce more meaningful results. Beside the availability of age and sex, other risk factor data such as socio-economic status and country of birth would be valuable to deepen the understanding of the identified ethnic inequalities in health.

In order to assess the ethnic morbidity-mortality contrast and the consistency of association between reported morbidity and more objective measures of health (mortality and morbidity) by ethnicity, the data source to be selected must contain a reliable measure of ethnicity, a measure of self-assessed health as well as mortality records and a more objective measure of morbidity. In Scotland, death records are held by National Records of Scotland (NRS). A data source linking the death records to the other key information can provide the data necessary for this research. As death is a relatively rare event in epidemiological terms and occurs in about 1% of the population every year in the UK, multiple years of death records will produce more powerful analysis of the SAH-mortality association by ethnicity.

Furthermore, a greater sample size of the data source and consequently of its ethnic minorities' absolute number makes possible a powerful analysis of the reported morbidity-mortality association by ethnicity. For example, Chandola and Jenkinson used the Health Survey for England 1991-1996 (6 surveys combined) and the Fourth National Survey of Ethnic Minorities (Ethmins4; 1993-1994) in order to investigate the association between self-rated health and a range of more objective measures of morbidity: hypertension, cardiovascular disease, diabetes, stroke, limiting health, number of visits to the doctor (Chandola and Jenkinson, 2000). Looking at the non-significant ethnicity and self-rated health interaction terms to explain the likelihood of each measure of morbidity, they concluded that the association between self-rated health and each measure of morbidity did not significantly vary between ethnic groups and that self-reported health was a valid measure of health across ethnic groups. They failed to address that their research was based on wide confidence intervals as a result of small ethnic group sample size which rendered difficult detecting differential associations by ethnicity. The so-called probability of making a type II error (failing to reject the null hypothesis i.e. detect differences in the population of interest) can only be minimised by greater sample size.

Finally, the contemporary nature of the data is desirable for better generalisation of the results. To allow for multiple years of mortality data and considering the restricted number of data sources with the above requirements, candidate sources are considered contemporary for this research if data were recorded within the last two decades.

With these criteria in mind, the search of the best data sources to address the research aims of this thesis can narrow down to a few existing data sources linked to mortality data: the Scottish Health and Ethnicity Linkage Study (SHELS), the Scottish Longitudinal Study (SLS) and the Scottish Health Survey. The following paragraphs explain how well-suited these data sources are to explore the specific aims of this thesis.

The *Scottish Health and Ethnicity Linkage Study* linked existing health data with a Community Health Index (CHI) to the Scottish Census 2001 (Bhopal et al., 2010). It holds data for 4.62 million people (*erratum in the initial papers which published a linkage of 4.65 instead of 4.615 million people*) who responded to the 2001 Scottish Census on 29 April 2001 and were linked to a CHI. Its ethnic sample size is ideal for our desired investigation. It has a considerable majority White Scottish sample with over 4 million people but also around 93 thousand people from non-white minority ethnic groups living in Scotland in 2001. Within non-white groups, Pakistani, Chinese and Indian minority groups had the biggest sample size with respectively over

28, 15 and 13 thousand people. In addition to self-reported ethnicity (14 categories) and two measures of reported health, the Scottish Census 2001 holds other socio-demographic and risk factor data such as age, sex, country of birth, religion, many indicators of individual, household and neighbourhood socio-economic status and broader geographical information such as health boards and an urban/rural indicator. More objective measures of general health are also available in the SHELS data. Within the SHELS phase 4 project, 12 years of hospitalisation and death records (May 2001 to April 2013) were linked to the Scottish Census 2001. It contains all information necessary to explore the reported health - objective health association and morbidity-mortality contrast by ethnicity at a quasi-national scale in Scotland. Risk factors data that are not available for the SHELS cohort includes information on health behaviours (diet, smoking, alcohol consumption, and physical activity), experience of discrimination, and history of migration.

The *Scottish Longitudinal Study* links 5% of the censuses 1991, 2001 and 2011 to health and mortality records through the CHI number. Similarly to SHELS, reported health and socio-demographic data are available from the census parts of SLS. Mortality and hospitalisation data can be available through linkage up to more recent years. However, its ethnic minority sample is limited. The SLS sample size is about 270 thousand people and 5 thousand people were from non-white minority ethnic groups in the census 2001 part of SLS (Hattersley and Boyle, 2008). This sample size is satisfactory enough for analysing ethnic differences in general morbidity however, it reduces the ability to analyse the health experience of ethnic minorities grouped into finer ethnic groups rather than aggregated. It also renders analysing ethnic differences in mortality difficult due to low mortality prevalence (about 1% per year) combined with small ethnic minority samples. Its longitudinal design is nevertheless an advantage.

The *Scottish Health Survey* has been valuable to provide information on mental health, dental health, smoking and alcohol consumption, diet, physical activity, obesity, cardiovascular disease and diabetes by equality group including ethnic group (Whybrow et al., 2012). It has been linked to Scottish hospital admissions and mortality data by the Information Services Division (ISD) for research purposes (<http://www.gov.scot/Topics/Statistics/Browse/Health/scottish-health-survey/Uses>).

However, it is limited by its sample size i.e. between 4 and 8 thousand respondents annually between 2008 and 2015, rendering its ethnic sample too small for the purpose of this research.

Although the research of this thesis narrows (geographically) down to Scotland, additional data sources with the potential to address similar research questions in a broader UK context, were reviewed. In summary, no better data source than SHELS was identified which would gather all the data necessary for this research and at a quasi-national level. The most promising data source was the *England and Wales Office for National Statistics Longitudinal Study (ONS-LS)*. Similarly to census linkage studies in Scotland, ONS-LS links 1% of censuses 1971, 1981, 1991, 2001 and 2011 to events data such as records of birth, death and migration. One drawback is that, apart from Cancer registrations, it is not linked to other more objective measures of general morbidity from either primary or secondary care data. Other sources such as *Understanding Society*, *Health Survey for England 2004* and *English Longitudinal Study of Ageing* are limited either by a lack of fine ethnicity recording, by the availability of both reported and more objective measure of general morbidity along with the recording of death or by their small ethnic minority sample size particularly in relation to analysing mortality as an outcome.

Finally, despite existing data sources linking all information necessary, the possibility of a new linkage was considered for this particular research. For example, the linkage of the Scottish Census 2011, holding SAH and ethnicity, to mortality and hospitalisation data would have the advantage of being more contemporary. However, this data linkage would provide its user with fewer years of mortality data. Due to practical reasons of time and resources, this avenue of a new linkage was put aside for this particular project in favour of the use of existing data sources.

In conclusion, the SHELS data possess a measure of self-reported ethnicity with a fine ethnic granularity, socio-demographic factors, and reported health variables linked to 12 years of morbidity and mortality data at an individual level. It has the best sample size in the Scottish context to explore the research questions presented in section 2.4.

3.3. SHELS

3.3.1. Data linkage and representativeness

The SHELS linkage was described in detail (Bhopal et al., 2010). In summary, SHELS linked the Scottish Census 2001 to the CHI using probability matching. The linkage rate was high with 94% of the census respondents linked to a CHI. It resulted in a look-up table with encrypted CHI and encrypted census numbers held at NRS. A second look-up table with CHI and encrypted CHI numbers was also held at ISD, National Health Service National Services Scotland. These look-up

tables were key to link any health and mortality records with a CHI to the Scottish Census 2001 while ensuring security and confidentiality of the data. As previously mentioned in section 3.2, the SHELS sample includes ethnicity, country of birth, demographic and socio-economic indicators as well as measures of reported health. Within the phase 4 of the SHELS project, the Scottish Census 2001 was anonymously linked to 12 years of hospitalisation and mortality records. This particular linkage of census data to hospitalisation and death at the national level is unique in Scotland. The resulting data source is exploited for the purpose of this research and provides the necessary data for 4.6 million people who responded to the census in 2001 (about 90% of the 5.1 million estimated population of Scotland in 2001). As shown in table 3.1, the ethnic distribution of the SHELS sample is representative of the ethnic distribution of the population of Scotland in 2001, based on the official Scottish Census 2001 statistics.

Table 3.1. Ethnic groups, Scotland, 2001: comparison of the ethnic distribution between the Census 2001 official release from NRS and the SHELS 2001 Census sample

Ethnicity	Census 2001 official release *		SHELS Census 2001 sample	
	Number	Percentage	Number	Percentage
White Scottish	4,459,000	88.1	4,088,120	88.6
Other White British	374,000	7.4	334,985	7.3
White Irish	49,000	1.0	43,505	0.9
Other White	78,000	1.5	65,655	1.4
Any Mixed Background	13,000	0.3	11,110	0.2
Indian	15,000	0.3	12,335	0.3
Pakistani	32,000	0.6	25,630	0.6
Bangladeshi	2,000	0.0	1,570	0.0
Other South Asian	6,000	0.1	4,945	0.1
Caribbean	2,000	0.0	1,485	0.0
African	5,000	0.1	3,905	0.1
Black Scottish, Other Black	1,000	0.0	940	0.0
Chinese	16,000	0.3	13,205	0.3
All Other Ethnic group	10,000	0.2	7,715	0.2
All people	5,062,000	100.0	4,615,105	100.0

* Source: National Records of Scotland, @ Crown copyright 2013

3.3.2. Data access, ethics and disclosure

The part of the SHELS study (phase 4) linking all-cause mortality and all-cause hospitalisation data to the Scottish Census 2001 to investigate ethnic inequalities in health was approved by the Multicentre Research Ethics Committee for Scotland (reference 11/MRE00/4) and the Privacy Advisory Committee (PAC) (reference 36/13). Further approval were obtained to use the SHELS data for this specific research project. Ethics approval was granted by the University Teaching and Research Ethics Committee on August 21st 2017 and an amendment to PAC approval was submitted to the Public Benefit and Privacy Panel for Health and Social Care (PBPP formerly PAC) and approved on November 9th 2017. This research project using SHELS was also submitted as an Administrative Data Research Network (ADRN) project (PROJ-208) in order to cover support and costs for PBPP application, safe haven use and disclosure review associated with the use of SHELS data. Approval was received from the ADRN panel on November 28th 2017.

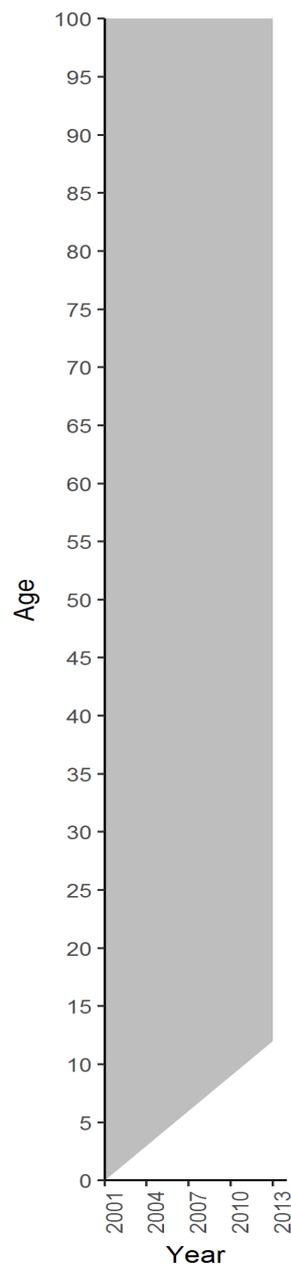
The results presented in this thesis had to go through a disclosure review process with clearance given by both ADRN and NRS. Following the NRS disclosure guidance for the SHELS project, all numerators and denominators are presented rounded to the nearest 5 but estimates were calculated from real number of events. Any results with a number of events of 5 or below were deemed disclosive and not released to researchers.

3.3.3. Study design

This research uses a retrospective cohort study design based on the SHELS cohort of 4.6 million people. Cohort studies are a suitable observational study design to analyse the effect of an exposure on an outcome (Euser et al., 2009). They have the advantage of providing the possibility to study multiple exposures and multiple outcomes from a single cohort but, due to the exposure being non-randomly allocated, it cannot establish causal effect (Euser et al., 2009). A retrospective cohort study also has the advantage of being time-efficient in comparison to a prospective approach. Figure 3.1 provides an illustration of the study design for this research. The grey area represent the population that is followed and analysed in this thesis. At the time of the census 2001, all age groups are included. The lexis diagram (figure 3.1) show an age range of 0-100 years as an indication for all age groups although a few individuals were over 100 years old at the time of the census in 2001. This cohort of individuals are linked to hospitalisation and mortality data from the time of the census up to April 2013. Hence, these individuals are followed over a period of 12 years. Data on Scotland exit and death was also linked to the SHELS

data in order to account for people who disappeared from the cohort at specific time points. No new entrant can be analysed by using this type of design. For example, any baby born in 2002 or any time after the census 2001 is not part of the SHELS cohort and cannot be included in the analysis. Similarly, any new entrant in Scotland after the time of the Scottish census 2001 cannot be part of the SHELS cohort.

Figure 3.1. Lexis diagram of the study design



3.3.4. Operationalisation of key concepts

3.3.4.1. Health

Chapter 2 highlighted the importance of considering someone's general health rather than focusing on a single disease. This section explains how general morbidity and mortality are operationalised for the purpose of this research project.

Measures of self-assessed health have the advantage of fitting with the WHO definition of health by including perception of health and well-being in addition to the state of being disease-free or not. As explained in section 2.2.4, SAH indicators are widely used and are deemed reliable measure of general health status with an extensive literature demonstrating their stability and validity. The two SAH measures recorded in the Scottish Census 2001 and used in this thesis are self-reported health (SRH) and limiting long term illness (LLTI). In the Scottish Census 2001, the question related to SRH was as follows: "Over the last twelve months would you say your health has on the whole been:" with the opportunity to answer "Good", "Fairly good" or "Not good". The question related to LLTI asked "Do you have any long-term illness, health problem or disability which limits your daily activities or the work you can do? Include problems which are due to old age." with the opportunity to answer "Yes" or "No".

Most research studies on ethnic inequalities in health focus on either mortality or a single disease. Recent evidence advocates going beyond the single-disease framework (Barnett et al., 2012, Salisbury, 2012, Starfield, 2006). The multimorbid state, defined as two or more disorders, has become the norm and especially so in the elderly population for whom multimorbidity affects more than half of the population (Glynn et al., 2011, Marengoni et al., 2011, Schiøtz et al., 2017). In Scotland, Barnett et al. similarly found that more than half of the patients were multimorbid by the age of 65 years (Barnett et al., 2012). Multimorbidity leads to worse quality of life and an increased use of health services (Glynn et al., 2011, Marengoni et al., 2011) and it is a key challenge to address by health services in order to avoid fragmentation of care and medical error. Multimorbidity prevalence increases in the very old, women and those with higher socio-economic deprivation (Barnett et al., 2012, Marengoni et al., 2011). As different ethnic groups face varied levels of deprivation (Nazroo, 2001) and minority ethnic groups are ageing, evidence is now required to quantify the magnitude of multimorbidity by ethnicity. This literature has emerged recently in the US showing ethnic differences in multimorbidity with trends varying by age and in multimorbidity combination (Johnson-Lawrence et al., 2017, Quinones et al., 2017, Rocca et al., 2014). In the UK context, evidence is,

to our knowledge, almost non-existent or remains focused on a single type of disease framework (Mathur et al., 2011). Using multimorbidity as an indicator of general health to monitor ethnic inequalities in health is a key step forward to assess the health state of minority ethnic populations and inform health care resourcing.

There is no standard on the best way to define and measure multimorbidity (Barnett et al., 2012, Diederichs et al., 2011, Xu et al., 2017). A recent systematic review and meta-analysis found that multimorbidity was associated with an increase in mortality in individuals aged 60 years and above, regardless of its operationalisation (Nunes et al., 2016). For the purpose of this research, multimorbidity is defined as the co-occurrence of diseases in a single individual and is operationalised as the presence of two or more diseases. The range of diseases from which to assess multimorbidity is identified through ICD10 diagnosis from hospital admission and uses the list of diseases from a commonly used measure of multimorbidity called the Charlson Index. The Charlson Index has been developed from hospitalisation data using a scoring system to select a list of chronic diseases based on their association with predicted 1-year mortality and was tested for its ability to predict 10-years mortality (Charlson et al., 1987).

The Charlson Index has being widely used to evaluate both the number and severity of co-occurrence of diseases (Huntley et al., 2012, Marengoni et al., 2011) and validated (Deyo et al., 1992, Huntley et al., 2012). Despite the strong evidence of high scores on the Charlson Index having a strong relationship with mortality (Huntley et al., 2012), it has been criticized for its lack of comprehensiveness and possible out-of-date prediction of mortality compared to more complex indicators recently developed such as the Multimorbidity-Weighted Index (Melissa and J Mukamal, 2017) or the M3 index score (Stanley and Sarfati, 2017). Notwithstanding critics, the Charlson index remains one of the most easy-to-implement indicators of multimorbidity. To overcome the issue related to the possible out-of-date scoring system, the number of morbidities was used to derive a simple indicator of multimorbidity rather than the score itself. In contrast to self-reported measures of health, our resulting multimorbidity indicator, based on the occurrence of two or more chronic diseases from the Charlson list, is doctor-diagnosed (derived from hospitalisation diagnosis) and can be considered as relatively objective. In the spectrum of general health indicators, it also reflects a more pronounced severity of diseased health status as it is based on diagnosis from hospitalisation data. The construction of the multimorbidity indicator is described in more details in chapter 6. The research of ethnic

differences in multimorbidity is supplemented by the exploration of the contribution of each condition of the Charlson Index list.

In section 2.2.1, the literature review showed that a strong body of evidence relating to ethnicity and health focuses on mortality. At the end of the spectrum, death is an objective fact. It remains a rare event in the population which increases in intensity with age. For example, in Scotland, NRS vital events statistics showed that there were about 58,000 deaths in 2015 (mortality rate of 1.1%), but around 95% of those deaths occurred to those aged 50 and above and 75% to those aged 70 and above. Deaths occurring in Scotland are recorded on the Scottish death registry and available in SHELS. 12 years of death records (between May 2001 and April 2013) are used in the analysis which provides enough events for a powerful analysis of ethnic differences in mortality and the relationship of SAH with mortality across ethnic groups.

3.3.4.2. Ethnicity

Initial research investigating ethnic inequalities in the UK was mainly based on the use of country of birth as a proxy for ethnicity. Drawbacks of using country of birth in place of ethnicity include its inability to distinguish different ethnic origins for those who were born in the same country. This lack of discriminatory power includes, for example, the limitation of classifying migrant's descendants as native-born or British born in India as foreign-born as well as different ethnic minorities with common geographical origin as a single group e.g. Hindustani and Creole residents in the Netherlands came from Suriname. Country of birth on its own lacks of specificity in accurately identifying ethnic background. Stronks et al. stated that country of birth (recorded on national registers) has the advantage of being objective and remaining stable over time and argued that it can be used in addition to the country of birth of parents and other measures of ethnicity, culture and language to better characterise ethnic groups (Stronks et al., 2009). Nevertheless, in health research and especially so in the UK, there has been a move towards the use of self-reported ethnicity (Bhopal, 2004). A turning point in the UK was the introduction of self-declared ethnicity in the 1991 Census (Aspinall, 2011).

As described in chapter 2, ethnicity is a fluid concept relating to belonging or perceptions of belonging in a social group sharing common culture, language, religion and/or ancestry. Its fluidity makes the concept of ethnicity difficult to operationalise in health research as most administrative data sources record ethnic groups using fixed categories defined by the data collector. In the UK, ethnic identities are usually self-reported with pre-specified ethnic categories and a space for 'other' ethnic identities to self-declare. Due to the complexity and

diversity of ethnic identities coupled with restricted ethnic categories to choose from, some individuals might not be able to identify themselves confidently into a pre-specified category. This is one reason explaining why the reporting of ethnicity changes over time but not the only one (Simpson, 2014b, Simpson et al., 2016). For example, of those who changed their ethnic classification between 2001 and 2011, Simpson et al. estimated that about a third to a half of these people did so due to an ambiguity of identity (Simpson et al., 2016). The changeable nature of self-reporting ethnicity with context and over time questions its reliability and use as a measure of ethnic identity in health research (Senior and Bhopal, 1994). In practice, a trade-off must be found between capturing ethnic diversity and using informative and reproducible ethnic groups for public health research (Aspinall, 2011). Changes in self-reported ethnicity remain small and have little effect on the analysis of ethnic differences in health because, in order to produce results with statistical confidence, there is a need to analyse ethnic groups with sufficient sample size to provide meaningful and consistent results. To achieve this, a balance between a fine ethnicity granularity and relatively homogeneous ethnic groups with sufficient sample size must be found.

The appropriateness of an ethnic group labelling is a constant debate as categories are not immutable and their relevance are time and space dependent (Agyemang et al., 2005, Bhopal, 2004, Bhopal and Donaldson, 1998, Bhopal et al., 1991). In the 1990's, Bhopal et al. pointed out to the inappropriate use of the term "Asian" in ethnicity and health research, its lack of precision and international applicability as it tended to be misused as a broader term to refer to people from the Indian subcontinent in the UK and to refer to people from East and South-East Asia in the US (Bhopal et al., 1991). Most recent research in the UK specifies labels in relation to people from South Asian origin as Pakistani, Indian and Bangladeshi. In relation to the majority group often referred to as White, European or Caucasian in the UK, Bhopal and Donaldson advised to use the terminology 'reference group' or 'comparison population' which avoids the expectations and associated norms related to the 'white population' (Bhopal and Donaldson, 1998). In regards to African origin populations, Agyemang et al. called for acknowledging the heterogeneity of African descent populations and offered in the debate of appropriate terminology the use of 'African origin' or 'African' as a prefix for a more specific ethnic label such as African Caribbean or African Surinamese (Agyemang et al., 2005).

The categorisation of ethnicity used in this thesis follows the 14 categories offered in the Scottish Census 2001: White Scottish, White Irish, Other White British, Other White, Any

Mixed Background, Indian, Pakistani, Bangladeshi, Other South Asian, Caribbean, African, Black Scottish or Other Black, Chinese and All other ethnic group. This categorisation overall aligns with the aforementioned recommendations on ethnic granularity, relative homogeneity of ethnic groups and appropriateness of labelling. In this thesis, results are presented for a disaggregated White group specifying and separating White Scottish and White Irish for example and using the majority population in Scotland, the White Scottish group as the reference population in the analysis. Furthermore, Chinese are considered as a separate category and South Asians are disaggregated into the Pakistani, Indian, Bangladeshi and Other South Asian groups. As much as possible, combining ethnic groups is avoided for this research as it can dilute strong effects seen in certain groups by mixing them with other ethnic groups. However, small sample size and disclosure issues might appear in relation to less prevalent health outcomes or for specific analyses. In cases of disclosure issue (5 events or less) in small minority ethnic groups, aggregation might be the solution to provide results for two small ethnic groups together rather than no result for these groups. On a few occasions, aggregation was done and justified. In these cases, the Bangladeshi group was combined with the Other South Asian group. People of Caribbean, Black African and Black Scottish and Other Black groups were also combined into one group, labelled 'African Origin', following Agyemang et al.'s recommendation on labelling.

A recent consensus on how ethnicity should be used in research on ethnicity and health showed concerns about considering ethnic groups as fixed and homogeneous (Mir et al., 2013) while identities are shaped by multiple factors. A way to minimise this ethnic category demarcation issue is to include multiple determinants of identities such as place of birth, religion, and language as well as other contextual social factors such as neighbourhood factors, discrimination, and migration experience (Bhopal, 2004, Mir et al., 2013). Including multiple aspects of identity when studying ethnic inequalities echoes with the concept of intersectionality presented in section 2.1.2.3 and is pursued in the analysis, especially in relation to country of birth.

3.3.4.3. Other socio-demographic factors

Socio-economic status

As explained in chapter 2 (section 2.3.1), there is a strong relationship between SES and health in the direction that higher socio-economic deprivation is associated with poorer health outcomes. Thus, if minority ethnic groups were to experience a health disadvantage, this could be related to their SES disadvantage. In other words, the relationship between ethnicity and

health could be modified (or explained) by SES. Which SES factors can be used to account for ethnic differences in health is a complex matter (Fischbacher et al., 2014, Kelaher et al., 2008, Kelaher et al., 2009). Using one SES indicator might also lead to residual SES confounding (the distortion that remains after controlling for available SES confounding factors) due to the inability of one measure to account for the full SES of an individual. Indeed, the literature review highlighted the need to use more than one measure of SES in ethnicity and health studies to account for the multi-faceted nature of deprivation faced by minority ethnic groups (Nazroo, 2001) and to avoid residual confounding. This thesis follows the approach of using multiple indicators of SES to account for socio-economic deprivation in the study of ethnic inequalities in health. The rationale for the chosen SES factors is based on available data, patterns of missingness, and reflecting upon evidence drawn from the literature review. As explained in chapter 2 (section 2.3.1), SES is multi-dimensional, reflecting material, cultural and social aspects. Ideally, the choice of SES measures would distinguish measures reflecting the material and the social class aspects of SES. However, in practice, most SES measures might represent both aspects of SES.

Table 3.2 shows the percentage of missing data by 10-year age group for each SES variable of interest in the SHELS data. The table presents no percentage when there was no missing data and a percentage is shown only when there was some missing data in a particular age group for a particular SES variable. First, one can notice that there was no missing data for the Scottish Index of Multiple Deprivation (SIMD). SIMD (2004) is an area-based measure of SES (Scottish Government, 2018). It was calculated for almost 7,000 data zones in Scotland and scores deprivation of each zone across seven domains: current income, employment, health, education, skills and training, housing, geographic access and crime. Each data zone covers an average population of 700-800 people.

For household tenure and car ownership, the percentage of missing data was exactly the same with about 1.5% of missing data overall. It occurred mainly in those aged over 80 years, most likely due to institutionalisation. Highest qualification which is the highest level of education one has achieved was only collected for those aged 16 to 74 years in the 2001 census. Hence, the highest qualification of the individual was 100% complete for those aged 16 to 74 years old and 100% missing for those aged 15 years and below as well as those aged 75 years and above. Overall, the highest qualification of an individual was missing for 26.2% of the SHELS cohort. The highest qualification measure was also available at the household level for each

individual. The percentage of missing data reduced to 0.1% in those aged 15 years and below and by more than half in the oldest respondent of the census. The overall percentage of missing data for the highest qualification of the household was 5%. Similarly, the National Statistics Socio-economic Classification (NS-SeC), a measure of occupation, was available for the individual and the household levels. NS-SeC for the individual was 100% missing in those aged below 15 and above 75 years and 45.5% missing overall. NS-SeC for the household had more information for the youngest and oldest census respondents than the individual NS-SeC measure but still had a high percentage of missing data with 17.5% of missing data overall.

Due to the nature of the SES measures and some of the age restriction applied during data collection, one can conclude that the observed patterns of missingness by age for household tenure, car ownership, highest qualification and NS-SeC at the individual and household levels were not missing at random. From this observation and as the analysis of this thesis focuses an overall measure of SES for all ages, the strategy to select SES variables for this research is to consider variables with the lowest percentages of missing data which are also relevant SES proxy across minority ethnic groups.

Table 3.2. Missing data for five SES variables available in the SHELS Scottish Census 2001 dataset by age group.

Age group	N	SIMD	Percentage of missing data				
			Household tenure & Car ownership	Highest qualification (individual)	Highest qualification (household)	NS-SeC (individual)	NS-SeC (household)
0-9	526445		0.1	100.0	0.1	100.0	5.3
10-19	579130		2.2	61.3	0.1	83.5	5.9
20-29	536855		1.7			15.9	7.0
30-39	711220		0.5			7.6	3.4
40-49	664835		0.4			9.4	3.8
50-59	600010		0.5			16.1	7.1
60-69	471935		0.7			60.2	41.7
70-79	348465		2.5	44.3	29.9	95.2	79.6
80-89	150135		10.6	100.0	73.8	100.0	81.3
90+	26075		32.8	100.0	55.5	100.0	67.5
All people	4,615,105	0.0	1.5	26.2	5.0	45.5	17.5

SIMD was the best variable in relation to completeness. SIMD was added as quantiles in the analysis as a proxy for exposure to neighbourhood deprivation. SIMD as an indicator of both neighbourhood assets and status is likely to impact on health. A UK-based study has shown that area deprivation predicted self-rated health more strongly in white British people than in minority ethnic groups (Bécares et al., 2012a). However, further research has also indicated that area deprivation is a good predictor of individual deprivation in minority ethnic groups (Baker et al., 2013). Although SIMD on its own might not be an ideal measure of individual socio-economic circumstances, it provides a more comprehensive picture of the economic and social deprivation faced by minority ethnic groups when combined with other SES measures.

In relation to individual and household SES measures, Kelaher et al. showed that education was effective to measure SES across ethnic groups but also that it would benefit from the addition of another measure of SES such as home ownership to reliably account for SES differences between ethnic groups (Kelaher et al., 2009). A measure of individual highest qualification when available complemented by the household highest qualification enabled the creation of a proxy SES measure referred to as combined individual and household education available for 95% of the SHELS cohort. The creation of this combined measure permitted minimisation of the number of missing data and increased the sample size in the analysis based on complete case while avoiding any age restriction. Household tenure had only 1.5% missing data and was deemed a good measure of wealth. Combining these three SES measures i.e. SIMD, combined education and household tenure was thought to provide a good proxy for SES incorporating measures of deprivation at the individual, household and area levels.

Other SES variables of interest were car ownership and occupation (NS-SeC). As previously explained in the literature review, car ownership might not be a good proxy for SES in the Pakistani and Indian populations, as it is inversely associated with education when age and sex are adjusted (Kelaher et al., 2009). Occupation at individual and household levels had high percentages of missing value and adding it to the analysis would reduce the sample size considerably particularly in the youngest and eldest groups. Hence, it was not deemed useful to include car ownership and occupation in this thesis's analysis. However, their addition to the three SES indicators selected was tested in a sensitivity analysis of SAH by ethnic group and is available in appendix 3.1. Adding either variable to the three SES proxies selected made no change to the interpretation of the findings.

In conclusion, three socio-economic indicators were selected to be used in the analysis: the Scottish Index for Multiple Deprivation, combined education, and household tenure. SIMD was used as quintiles. Combined education was categorised as no education, low education and high education and household tenure as own versus rent.

Migrant status and country of birth

Section 2.3.2.2 introduced the acculturation hypothesis whereby migrants' health behaviours and consequently health status tend to converge to that of the native population as they stay longer in their country of destination. Duration of residence was not available in the SHELS Census 2001 data but as previously explained in chapter 2, another way to investigate the processes of acculturation is to investigate the experience of migrants and their descendants. Combining country of birth and ethnicity can inform about migrant generations. The analysis used a proxy for migrant generation derived from the country of birth variable. A UK-birth variable was created as 1 if born in the UK (included England and Wales, Scotland and Northern Ireland) and 0 otherwise. UK-birth was combined with ethnicity to investigate the health outcomes of migrants and their descendants in relation to that of the White Scottish population born in the UK. Note that non-white ethnic groups who were born in the UK are considered as descendants and Other White British who were born in the UK are not for the obvious reason that British people are native of the UK.

However useful in the absence of information on the origin of the parents and grandparents for each individual, this combined measure of ethnicity/UK-birth, proxy for distinguishing migrants from descendants, is not perfect. For example, if descendants declare their ethnicity as part of the majority ethnic group rather than that of their ancestors, they cannot be counted as descendant with the measure employed. The identity shift of descendants towards the majority ethnic group might create some bias if the results by ethnicity/UK-birth are interpreted using a strict migrant/descendant dichotomy. Chapter 7 discusses the potential source of bias from the identity shift of descendants and how this might influence the findings of this thesis.

Ageing and old age

The concept of old age tends to be associated with chronological age. However, there are social and cultural influences which mean that using chronological age only informs of the ageing process of the average person (Stuart-Hamilton, 1991). Traditionally a 'floating threshold'

around 60-65 years has been used to delineate a group of older people more likely to experience signs of ageing as well as a change in social status intertwined with the retirement period (Stuart-Hamilton, 1991). The process of ageing is biological, psychological and social (Bromley, 1988). It occurs and accumulates over time. This accumulation can be positive such as an increase in wealth and wisdom but, in relation to health, despite a well-being increase in the post-retirement phase around 60-75, this accumulation tends to be associated with negative changes such as an increase in multimorbidity and a decline in physical and psychological health. Although this accumulation appears key to explore later life outcomes, it is also important to realise that the process of biological ageing and functional loss starts earlier in adulthood (Stuart-Hamilton, 1991) and that events such as retirement might buffer the accumulation process, at least for a short period (Marshall and Nazroo, 2016).

Most studies presented in section 2.2.2.2 used an age threshold of either 60 or 65 years to identify old age. However, an earlier age threshold was considered to investigate the health outcomes of younger versus older adults in this thesis, recognising as previously explained that health deterioration occurs earlier in life (Stuart-Hamilton, 1991). For example, the Survey of Health Ageing and Retirement in Europe (Börsch-Supan et al., 2013) as well as the English Longitudinal Study of Ageing (Stephens et al., 2012) have used an age cut-off of 50 years. Different ethnic groups might also age earlier (Blakemore and Boneham, 1994). Hence, the following age groups were used to distinguish younger adults from older adults: 16-49 years, 50 years and above.

3.3.5. Description of the SHELS cohort

Table 3.1 shows that the majority White Scottish accounted for 89% of the SHELS cohort followed by Other White British (7%), Other White (1%) and White Irish (1%). Only 2% of the population were from non-white minority ethnic groups. In absolute numbers, the largest non-white minority groups were the Pakistani population, followed by the Chinese and Indian populations.

The socio-demographic profile of the SHELS cohort has been published (Cezard et al., 2015, Fischbacher et al., 2014). White Scottish males were on average 38 years old while females were on average 41 years old. Other White British and Irish populations had an older age profile and non-white minority groups were on average at least 10 years younger than their white counterparts with people of Any Mixed Background origins showing the youngest age profile

(average age 21 years in males and 24 years in females). The White Scottish, Irish and Other British groups were likely to be born in the UK (95-99%) as well as Any Mixed Background group (75%). Around 60% of Pakistani and half of Indian populations were born in the UK which highlighted their well-settled nature in the UK. Of the people of Other White, Other South Asian, Chinese and African origin, 30% to 40% were born in the UK.

The socio-economic profile of the SHELS population is described through the proportions of those living in the least deprived SIMD quintile, those with higher level of the highest individual qualification/education attained and those living in an owned property. Just below 20% of the White Scottish population lived in the least deprived areas (quintile). In contrast, all other ethnic groups were more likely to live in the least deprived quintile. The highest proportions were seen in people of Indian (39%) and Chinese (39%) origins. The proportion of high level for the highest qualification one can attain was the lowest in the White Scottish population (24% in both males and females), similar to proportions observed in Pakistani males (26%) and females (23%). All other ethnic groups reported higher level of education. In males, about half of the Other White British, Other White, Indian, Other South Asian, and African origin groups reported high level of educational attainment while, in females, only the Other White females reached this proportion. In relation to household tenure, Indian and Pakistani populations were the most likely to be owners with respectively 73% and 76% living in an owned household. Between 65% and 71% of the White Scottish, other White British, White Irish groups lived in an owned household. In contrast, people of African origin were the least likely to live in an owned household (44%). In conclusion, in relation to their SES profile, minority ethnic groups in the SHELS cohort did not seem to be particularly disadvantaged compared to the White Scottish population. This is in line with a previous study which showed that there was no SES disadvantage in ethnic minorities relative to the majority population in Scotland contrasting with evidence from other settings like the US or England (Walsh, 2017). Comparing ethnic groups living in Scotland and in England in 2011, Walsh also showed that over 30% of Pakistani and Bangladeshi people lived in the most deprived decile in England while this proportion was below 10% in Scotland.

3.4. Statistical methods

This section offers an overview of the statistical methods appropriate to address the research questions of this thesis using the SHELS data. Following the structure of the empirical research chapters, the choice of the statistical methods fit to answer specific research questions is described. Statistical methods are presented in greater detail in each empirical chapter when necessary. Research questions are refined according to the methods chosen and available data. Finally, this section explains the choices made and the consistent approach in the way variables are used in statistical models.

3.4.1. Summary of statistical methods

Chapter 4 explores the following research questions:

1. How do patterns of reported morbidity by ethnicity compare to patterns of mortality by ethnicity based on the same population source?
2. Do mechanisms thought to shape ethnic inequalities in health, such as socio-economic status and migrant generations, contribute to explaining ethnic differences in reported morbidity?

These questions are addressed using two types of methods. As SHELS links SAH and mortality at the individual level, the first type of methods used in chapter 4 is based on the augmented life table method to calculate health expectancy indicators. Using the Sullivan method (Sullivan, 1971), it combines SAH and mortality information based on the same population source. In combination with life expectancy, this method has the advantage of informing on whether living longer comes with living longer healthily and thus, can also inform about a morbidity-mortality contrast. However, this method does not allow us to explore the contribution of key mechanisms of ethnic inequalities in health. Hence, a second type of method is used in chapter 4. Regression methods can be used from individual level data to explore ethnic differences in health. In other words, they can provide information on the relationship between ethnicity and health. Confounders can be added to the regression model and their effect in reducing inequalities assessed. Thus, regression analysis can also inform about whether other mechanisms such as SES plays a role in the ethnicity-health relationship. As a result, regression analysis is used to ascertain patterns of ethnic inequalities in reported morbidity (SAH) but also to explore what is the role of specific mechanisms in the ethnicity-health association. Chapter 4's discussion assesses whether ethnic differences in SAH align with previously published

mortality patterns (Bhopal et al., 2018) when confounders are accounted for. Both methods, life table and regression analysis, can be used to explore health inequalities in older ages.

The appropriate regression method to explore the relationship between ethnicity and health and its mechanisms needs to account for the type of variable used in the model. SAH can be treated as a binary outcome such as 'good health versus bad health' or 'at least one LLTI versus none'. Ethnicity is categorical. With a binary outcome, logistic regression and Poisson regression are commonly used in epidemiology. Logistic regression produces Odds Ratios (ORs) while Poisson regression produces Risk Ratios (RRs), sometimes also called Relative Risks. Both ORs and RRs allow us to compare the health of ethnic groups relative to the health of a reference group. RRs and ORs provide similar results in the case of rare events but when the outcome is more common, ORs tend to overestimate the risk differential (Grant, 2014, McNutt et al., 2003, Schmidt and Kohlmann, 2008, Zhang and Yu, 1998). Furthermore, ORs are often misinterpreted as risks (Grant, 2014). To transform ORs into RRs, a few methods have been proposed (Grant, 2014, Zhang and Yu, 1998) but also criticized (McNutt et al., 2003). An alternative is to use a statistical model which provide RRs directly. Some regression methods have been offered including Poisson regression which, with robust variances, produced correct point and interval estimates (Barros and Hirakata, 2003, Schmidt and Kohlmann, 2008). Because of a better representation of the risk differential and ease of interpretation in comparison to ORs (Grant, 2014, McNutt et al., 2003, Schmidt and Kohlmann, 2008), RRs were chosen over ORs and presented in this thesis. RRs were estimated directly using Poisson regression with robust variances. The link function in Poisson regression is the natural log and parameter estimates are on the log scale so estimates were back-transformed to get RRs. One assumption of Poisson regression is that the mean and variance are equal which can be violated. To account for potential issues of dispersion, robust variances were included as recommended by Barros and Hirakata (Barros and Hirakata, 2003).

Using SHELS data and the two types of methods described above, chapter 4 aims to answer the following refined questions:

- 1- What are the magnitude and direction of ethnic differences in health expectancies based on a direct method using individually linked data?
- 2- What are the magnitude and direction of ethnic differences in SAH based on individual level data?

- 3- To what extent can individual/neighbourhood socio-economic factors account for the ethnic differentials in SAH in Scotland?
- 4- Do patterns of ethnic differences in SAH differ by whether individuals were born in or outside the UK?
- 5- Do patterns of ethnic differences in SAH differ in older adults compared to younger adults?
- 6- Do patterns of ethnic differences in health expectancies differ from patterns of ethnic differences in life expectancy in Scotland (is there a morbidity-mortality paradox)?

Further details on the lifetable approach, the outcome variables and sets of models used in regression analysis are available in chapter 4.

Chapter 5 explores the following research question:

3. Does reported morbidity relate to mortality consistently across ethnic groups?

To investigate the relationship between SAH as recorded in the Scottish Census 2001 and the subsequent 12 years of mortality data available in SHELS, a survival approach is appropriate. The semi-parametric survival method, Cox regression, models time-to-event data. In chapter 5, it is used to ascertain whether SAH predicts mortality similarly across ethnic groups over a duration of 12 years. In this type of survival analysis, mortality takes the form of time to death and right censoring is done for any death, migration event, or the end of the 12 years period while controlling for age. Cox regression analysis does not make any assumption on the baseline hazards. However, it assumes that the hazards are proportional over time for the groups investigated (Bewick et al., 2004). Cumulative log-log plots, for each variable included in the analysis predicting time to death, were used to check whether these assumptions were met and are available in appendix 3.2. Cox regression produces Hazard Ratios (HRs). HRs allow us to assess ethnic differences in mortality by comparing the mortality risk of ethnic groups to the mortality of the ethnic group taken as the reference group.

Before assessing whether SAH predicts mortality consistently across ethnic groups, a first step to understand how SAH relates to mortality is to examine whether SAH is a predictor of mortality in each ethnic group. The analysis at this stage is stratified by ethnic group. Once investigated, the consistency of the SAH-mortality relationship among groups can be assessed using one model which includes an interaction term. The ethnicity*SAH interaction term entered

in the model (in addition to SAH and ethnicity) to predict mortality can provide information on whether a differential SAH-mortality association exists in a particular ethnic group in comparison to the ethnic group taken as the reference group. Potential confounders (variables that influence both exposure and outcome, e.g. both ethnicity and health, and that distort the association between the exposure and the outcome) can also be added to ascertain their effect on the observed differential association.

Hence, chapter 5 aims to answer the following refined research questions:

- 1- Does SAH predict subsequent mortality for each ethnic group in Scotland?
- 2- Is the SAH-mortality association consistent across ethnic groups in Scotland? In other words, does ethnicity mediate the SAH-mortality association?
- 3- Can socio-economic status differences explain any ethnic differential in the SAH-mortality association?
- 4- To what extent does the observed ethnic differential in the SAH-mortality association prevail in both migrants (born outside the UK) and their descendants (born in the UK)?

Finally, **chapter 6** explores the following research question:

4. Does using a doctor-diagnosed measure of health provide similar patterns of ethnic differences in morbidity as using reported morbidity? Does it support a morbidity-mortality paradox in specific ethnic groups in Scotland?

For this chapter, a multimorbidity indicator based on 12 years of hospital records is used. It is a binary outcome providing information on an individual's morbidity status (Yes/No). To explore ethnic differences in multimorbidity or any health outcome that is binary, using RRs, directly estimated from Poisson regression with robust variances, is a good approach as previously explained in relation to chapter 4 analysis on ethnic differences in SAH. Confounders can be added to the model and their contribution to the observed differences assessed. The discussion will interpret the multimorbidity patterns in chapter 6 in relation to the SAH patterns from chapter 4. Moreover, to determine the consistency of the SAH-multimorbidity association across ethnic groups, an ethnicity*SAH interaction term to predict multimorbidity can be added to the model in a similar fashion to the interaction analysis in chapter 5.

With the aforementioned methods and SHELS data, chapter 6 aims to answer the following refined research questions:

- 1- What are the magnitude and direction of ethnic differences in multimorbidity based on individual level hospitalisation data?
- 2- To what extent can individual/neighbourhood socio-economic factors account for the ethnic differentials in multimorbidity in Scotland?
- 3- Do patterns of ethnic differences in multimorbidity differ by whether individuals were born in or outside the UK?
- 4- Which comorbidities underlie the observed ethnic differences in multimorbidity?
- 5- Is the SAH-multimorbidity association consistent across ethnic groups in Scotland? In other words, does ethnicity mediate the SAH-multimorbidity association?

3.4.2. General modelling approach

The denominator was the 4.62 million people of the SHELS cohort. For the multimorbidity analyses, person-years (PY) were used as denominator over a 12 years period. PY denominators and survival analyses accounted for any information on death or migration available in SHELS. The analyses of this thesis mostly rely on regression analyses and RRs or HRs. The reference population was taken as the majority White Scottish population (RR=1 or HR=1) unless stated otherwise. For all analyses, 95% confidence intervals (CIs) were calculated and are presented. Significant results where confidence intervals did not overlap with the reference value (RR=1 or HR=1) were reported. All variables were added in the models as categorical variables. This section further describes how the socio-demographic variables were used in regression analyses.

Sex

There are many reports of differential ethnic patterns by sex. For example, analysing ethnic differences in mortality in the Netherlands, Bos found no differences in mortality between people of Indonesian origin and native Dutch but when males and females were analysed separately, there was a higher risk in Indonesian females and a lower risk in Indonesian males compared to their Dutch counterparts (Bos, 2005). To avoid missing out on differential ethnic patterns between males and females and respond to known sex differences in health risk profiles, it is preferable for our analyses to be stratified by sex. Sensitivity analysis presented in

appendix 3.3 showed a significant interaction between ethnicity and sex in predicting self-assessed health for particular ethnic groups supporting the usefulness of stratified analysis by sex. The large sample size of the SHELS data makes such a sex stratification possible and sensible.

Age

As known and explained in section 3.3.5, minority ethnic groups have a younger age profile than the White Scottish population. To account for different age profile between ethnic groups, it is important to adjust for it in each model. Hence, the base model of each set of regression models is a model adjusted for age. Sensitivity analysis adjusting for different forms of age are available in appendix 3.4. In summary, adjusting for age as a categorical variable in 1 year, 5 years or 10 years age bands made limited differences to the results of ethnic differences in SAH. Adjusting for age as a continuous variable gave somewhat different results but was ruled-out due to the non-linear relationship of age with the three key health outcomes of this thesis (self-assessed health, multimorbidity and mortality) as shown in appendix 3.5. Adjustment for age and age-squared gave results closer to adjusting for age as a unit category. Age was entered in the model as categorical due to its non-linear relationship with the health outcomes used in this research. Adjustment for age as a unit category was chosen because of its best fit to the data and because the size of the SHELS cohort (4.6 million people) was sufficiently large to use a precise measure of age for modelling purposes.

Some analyses also investigated whether ethnic differences in health persisted in older ages. The intersection between ethnicity and old age in shaping health inequalities was explored using models with an interaction term as well as stratified analysis by adult age groups.

SES and migrant generations

SES and UK-birth variables were added to the age-adjusted model in different ways. For example, to assess how SES or UK-birth influences ethnic differences in health, it was simply added into the model as it is but, to inform about a differential association between SES and health for specific ethnic groups, it was added as an interaction term with ethnicity. Furthermore, to explore ethnic differences in health by migrant generations, ethnicity was replaced by a combined measure of ethnicity and UK-birth in the model. The health of each ethnic group born in the UK as well as each ethnic group born outside the UK was compared to that of the White Scottish born in the UK, taken as reference. This type of intersectionality analysis was rendered possible due to the unique sample size of the SHELS cohort.

3.4.3. Statistical softwares

Data were analysed using SAS V9.4 (SAS Instituta Inc, Cary, North Carolina, USA). Cumulative log-log plots were also produced using SAS. Apart from appendix 3.5 where the figures came from Excel 2013, all other figures were created using R version 3.4.3 (R Core Team, 2017).

CHAPTER 4

4. Ethnic differences in health expectancies and self-assessed health in Scotland. How do they compare to ethnic mortality patterns?

4.1. Background and research questions

The literature review in chapter 2 has highlighted the need to identify and understand ethnic differences in general morbidity in Scotland. There is also a clear gap in the literature in determining the extent to which ethnic inequalities in morbidity diverge from mortality patterns. To bridge these gaps in knowledge, this chapter uses self-assessed health as a general measure of health to explore ethnic differences in general morbidity and assesses whether patterns follow the mortality advantage observed in ethnic minorities in Scotland (Bhopal et al., 2018, Gruer et al., 2016). As previously explained, the SHELS data is used and has the advantage of providing individual level data from the Scottish Census 2001. A refined analysis based on SHELS permits the exploration of explanations of ethnic differences in health based on a national level population sample in Scotland. The analysis of reported morbidity in this chapter draws from the same population which was used to draw evidence of ethnic differences in life expectancy (LE) and mortality in Scotland. This provides the opportunity to compare health expectancies (HE) to LE by ethnicity based on a single population source.

The research questions addressed in this chapter are as follows:

- 1- What are the magnitude and direction of ethnic differences in health expectancies based on a direct method using individually linked data?
- 2- What are the magnitude and direction of ethnic differences in SAH based on individual level data?
- 3- To what extent can individual/neighbourhood socio-economic factors account for the ethnic differentials in SAH in Scotland?
- 4- Do patterns of ethnic differences in SAH differ by whether individuals were born in or outside the UK?
- 5- Do patterns of ethnic differences in SAH differ in older adults compared to younger adults?
- 6- Do patterns of ethnic differences in health expectancies differ from patterns of ethnic differences in life expectancy in Scotland (is there a morbidity-mortality paradox)?

This chapter first presents ethnic differences in general health using two types of health indicator: health expectancies and self-assessed measures of health. Using the Sullivan method (Sullivan, 1971), health expectancies have the advantage of combining one self-assessed health measure with mortality into a single measure of general health. It enables the estimation of how long ethnic groups are expected to live in good health. However useful a description, combining health with mortality using the life table and Sullivan approach does not allow us to disentangle explanations of morbidity patterns by ethnicity. Consequently, SAH is also used on its own to investigate patterns of reported morbidity by ethnicity and to explore some of the mechanisms driving ethnic differences in health. The chapter assesses to what extent the observed patterns can be explained by socio-economic status. Whether patterns of SAH by ethnicity differ in old age and by whether individuals were born in the UK or not are also investigated. Finally, this chapter considers health expectancies by ethnicity as a useful tool for our purpose if contrasted with life expectancy estimates. This gives a first description of whether specific ethnic groups experience a morbidity-mortality paradox in Scotland i.e. live longer but not necessarily in good health.

As previously stressed, identifying and understanding ethnic differences in health matters in order to develop targeted health policies and interventions to reduce health inequalities. The literature of ethnic differences in the risk of specific diseases and initial evidence of ethnic differences in self-assessed health in Scotland (see section 2.2.2) provided an indication that ethnic differences in health expectancies and SAH would go in both directions compared to the majority White Scottish population. For example, Pakistani groups are expected to report worse health and have shorter health expectancies while Chinese populations are expected to report better health and have longer health expectancies compared to the White Scottish population.

Socio-economic differences between ethnic groups are often put forward as a key contributor to ethnic differences in health (see section 2.3.1). Socio-economic deprivation is linked to worse health outcomes. If minority ethnic groups have worse health outcomes, it could be argued that it is due to their relatively worse socio-economic profile compared to the majority population. Research on the relevance of adjusting for SES in ethnic health studies suggests the need to use more than one SES indicator to account for the multi-faceted experience of deprivation faced by minority ethnic groups (Fischbacher et al., 2014, Nazroo, 2001). In England and Wales, observed patterns of ethnic inequalities in SRH and LLTI were mostly reduced when

accounting for SES proxies using various combinations of educational level, occupation, social class, economic activity, income, car ownership, household tenure, household overcrowding and deprivation score (Darlington et al., 2015, Evandrou, 2000a, Evandrou et al., 2016, Harding and Balarajan, 2000, Mindell et al., 2014). Using multiple measures of SES, we hypothesise that lower SES in certain ethnic groups will explain their worse SAH outcomes or that higher SES will underlie better health outcomes.

A few studies have also looked into migrant generation differences in the ethnic patterning of SAH (Harding and Balarajan, 2000, Smith et al., 2009). Initial studies by Harding and Balarajan showed that the health disadvantage seen in immigrants persisted in the second generations in South Asian and Black Caribbean populations and worsened intergenerationally for Black Africans (Harding and Balarajan, 2000). More recent research by Smith et al. found that, despite a general upward socio-economic mobility in second generations compared to immigrants, there were similar high odds of reporting poor/fair health between first and second generations in Black Caribbean, Indian and Pakistani ethnic minorities. Assuming a positive health selection of migrants and a worsening of health with acculturation (see sections 2.3.2 and 2.3.3), better health is expected in immigrants while worse health is expected in descendants in comparison to the majority population.

As the population is ageing, investigating inequalities at the older ages is required to adjust and develop appropriate care and resources for the most vulnerable. However, the research on ethnicity and SAH seldom focuses on older ages. In addition, Evandrou et al. showed that as the percentage of reporting poor health increases with age, so do its ethnic differentials (Evandrou et al., 2016). Her findings of a stronger health disadvantage in older ages in specific ethnic groups in the UK support the idea of an accumulation of deprivation and discrimination over the life course which leads to worse health outcomes in specific ethnic groups. If we assume an accumulation of disadvantage over time, wider ethnic health inequalities are expected in older compared to younger age groups. However, it could also be argued that, as people get selected through survival and only the healthiest survive, ethnic inequalities in health might reduce in older ages.

Finally, knowledge of a “morbidity-mortality paradox” in specific minority ethnic groups in Scotland, living longer but in poorer health is key to plan for adapted care and services. The literature discussed in section 2.2.3 shows that the research of an ethnic morbidity-mortality

contrast remains limited internationally and methodologically. Initial evidence of a morbidity-mortality contrast appeared in Greek and Italian populations in Australia (Kouris - Blazos, 2002, Stanaway et al., 2019) and Mediterranean migrants in Belgium (Deboosere and Gadeyne, 2005) and France (Khlal and Guillot, 2017). However, the observed mortality patterns and morbidity patterns were mostly gathered from different sources and did not necessarily refer to the same individuals. Contrasting SAH and mortality patterns by ethnicity from the same population source will provide a strong indication of the likelihood of a morbidity-mortality paradox phenomenon in particular ethnic groups in Scotland. Furthermore, contrasting HE with LE estimates by ethnicity provides the opportunity to assess whether those who live longer, do so healthily and thus, informs on the morbidity-mortality contrast.

As well as addressing hitherto unanswered questions about ethnic differences in morbidity in Scotland and its contrast with ethnic differences in mortality, this chapter makes methodological contributions. Gathering data on health, mortality and ethnicity at the national level to calculate health expectancy by ethnicity is a challenge. In England and Wales, one study investigated health expectancies by ethnicity with HE calculations based on an indirect method of mortality estimation (Wohland et al., 2015). In the US and Europe, a few studies have calculated HE by ethnicity (Carnein et al., 2015, Hayward et al., 2014) or by migrant status (Reus-Pons et al., 2017) but they used different samples and sources to gather morbidity and mortality data. Thanks to the linkage of death records to census data at a national level available in the SHELS data, this is the first time that health expectancies by ethnicity have been calculated using a direct method and with morbidity and mortality data based on the same population source.

4.2. Data and methods

Section 3.3 explains how SHELS linked the Scottish Census 2001 to 12 years of hospitalisation and mortality records for 4.6 million people who responded to the Scottish census in 2001. In this chapter, the census part of the SHELS data is used which contains two SAH indicators (SRH and LLTI), self-declared ethnicity, country of birth, demographic and socio-economic indicators. The linked mortality part of SHELS is also used in combination to SAH to calculate two HE measures and to contrast mortality and SAH patterns using the same SHELS cohort. Ethnic differences in general health using additional health indicators originating from the linked hospitalisation data of SHELS will be explored in chapter 6.

The conceptualisation and operationalisation of health were detailed respectively in sections 2.1.1 and 3.3.4.1. Self-assessed measures of health alone and combined with mortality are used in this chapter to investigate ethnic inequalities in health. In line with previous research and the published Scottish reports on ethnicity and SAH (Evandrou, 2000a, Evandrou et al., 2016, Scottish Government, 2004, Scottish Government, 2015), the analysis of ethnic differences in SRH in this chapter models SRH as “Bad” versus “Fair/Good”. A sensitivity analysis using “Bad/Fair” versus “Good” was also done to understand the influence of the “Fair” category in shaping ethnic inequalities in SRH. Furthermore, SRH and LLTI are used to calculate the rate of good health which is included in the life tables in order to estimate respectively healthy life expectancy (HLE) and disability-free life expectancy (DFLE). The analysis of HLE by ethnicity dichotomised SRH similarly i.e. “Fair/Good” was used to calculate the rate of good health.

The concept of ethnicity was explained in section 2.1.2 and its operationalisation in section 3.3.4.2. The Scottish census 2001 data offer 14 ethnic categories for self-identification which were used for the SAH analysis. However, when SAH was combined with mortality to produce health expectancies, some ethnic groups were aggregated due to low number of deaths. The HE analysis shared the same constraint of low death events for some groups as the LE published results (Gruer et al., 2016). Consequently, ethnic groups followed the same aggregation process. The Bangladeshi group was combined with the Other South Asian group. The Caribbean, Black African and Black Scottish and Other Black groups were combined into one group, labelled ‘African Origin’. The results for the ‘All other ethnic group’ are not reported due to the heterogeneity of this category.

The operationalisation and categorisation of SES, UK-birth and old age were also explained in chapter 3 and apply in all empirical chapters. In summary, three SES proxies were used as measures of socio-economic deprivation combining information at the individual, household and neighbourhood level. The choice of SES indicators was also made to match the SES proxies used in the mortality publication using SHELS (Bhopal et al., 2018) which allows the comparison of the SAH and mortality patterns by ethnicity when SES is taken into account based on the same population source and the same SES indicators. The UK-birth variable was created as a dichotomous variable (1 if born in the UK and 0 otherwise) and used in combination with ethnicity to differentiate the risk of poorer health outcome between immigrants (ethnic groups who were born outside the UK) and their descendants (ethnic groups who were born in the UK) compared to the majority White Scottish population born in the UK. Ethnic differences in SAH

were assessed in older and younger adults to investigate whether inequalities remain, are greater or disappear into old age with an age cut off of 50 years old.

The modelling approach was explained in section 3.4. In this chapter the augmented life table and Poisson regression with RRs are used. The augmented life table methods and the sequence of regression models are detailed in the following paragraphs.

Life expectancy by ethnicity was previously published and its method explained (Gruer et al., 2016). It followed the Office of National Statistics recommendations in relation to life expectancy estimation for small populations using 3 years of data (Toson and Baker, 2003). It was based on 3 years of mortality data following the Scottish Census 2001 (May 2001 to April 2004), with the 4.6 million people of the SHELS cohort as the denominator in year 1 and adjusted denominators in years 2 and 3 accounting for any record of death or exit from Scotland. The Chiang II method (Chiang, 1984) and abridged life tables were used with 5-year age intervals and a category for new-borns aged 0, for young children aged 1 to 4 years old and for older people aged 85 years old and above. LE estimates at birth were calculated with their 95% confidence intervals by sex and ethnic group based on the SHELS cohort.

This chapter builds on this calculation of life expectancy at birth and the corresponding life table to calculate HLE and DFLE by sex and ethnic group using the Sullivan methods (Sullivan, 1971). The resulting augmented life tables incorporated the rate of good health calculated from SRH and LLTI to obtain respectively HLE and DFLE by sex and ethnic group. HE estimates were calculated following the practical guide developed by the European Health Expectancy Monitoring Unit (EHEMU) (Jagger et al., 2006). To provide a better picture of potential discrepancies between LE estimates and HLE/DFLE estimates, the number of years in poor health (LE subtracted by HLE) and number of years lived with disability (LE subtracted by DFLE) were calculated for each sex and ethnic group. If we rename these estimates as bad health expectancy (BHE), it is easy to note that HE and BHE adds to LE. Following the EHEMU guide, standard errors for HE and BHE accounted for the variance of the mortality rates and the corresponding 95% confidence intervals were calculated. The proportion of years lived in good health (HLE divided by LE) and the proportion of years lived without disability (DFLE divided by LE) are also provided as an indication of relative length of life lived in a healthy state. Finally, additionally to estimates at birth, this chapter also presents previously unpublished LE at 50 years of age based on the life

table used for the LE at birth by ethnic group in Scotland (Gruer et al., 2016) and HE at 50 years of age to assess whether inequalities persist into older ages.

In order to analyse ethnic differences in health, Risk Ratios (RRs) and their 95% confidence intervals were calculated using Poisson regression with robust variance. Arguments towards the use of Poisson regression rather than logistic regression and its relevance to this particular analysis are available in section 3.4.1 and include a better representation of the risk differential and ease of interpretation in comparison to odds ratios.

Ethnic differences in SRH (Bad health) and LLTI were analysed in separate models. A sensitivity analysis of SRH using “Bad/Fair health” instead of “Bad health” is available in appendix 4.1. RRs (95% CI) and associated p-values are reported for each ethnic group in comparison to the majority White Scottish population, used as reference (RR=1). As explained in section 3.4.2, the analysis was based on 4.6 million people who responded to the Scottish census 2001 as denominator. It was stratified by sex and the age-adjusted model was presented as the base model. In subsequent models, the three SES variables or the UK-birth variable were added as categorical variables to the age-adjusted model. To explore the differential relationship between each SES and SAH for different ethnic groups, models included each SES and its interaction with ethnicity. An interaction term UK-birth*ethnicity was similarly added to the model adjusted for UK-birth to assess the differential association between UK-birth and the outcome across ethnic groups. The influence of UK-birth on ethnic inequalities in health was also pursued through the analysis of SAH differences by a combined ethnicity and UK-birth variable. An interaction term of adult age groups and ethnicity was also used to investigate whether ethnic inequalities in SAH are consistent in older age groups. Additionally, to provide a better visualisation of the direction of potential divergence discovered from the interaction term, age-adjusted analysis were repeated (stratified) by adult age groups. Each set of model was based on complete cases i.e. when there was no missing for SES as other variables were complete. It meant that the results included all ages and was based on 95% of the original SHELS sample.

Note that only one SAH measure was presented for the analysis of ethnic differences in SAH at the intersection with UK-birth or with adult age groups. LLTI was preferred due to its higher prevalence compared to the report of Bad health. The higher prevalence of the LLTI outcome is likely to provide more reliable estimates when looking at the health experience of

ethnic minorities at the intersection with other characteristics. However results using either SRH or LLTI led to similar conclusions and SRH results are available in appendices.

4.3. Results

4.3.1. Ethnic differences in health expectancies in Scotland

Figure 4.1 shows HLE and DFLE estimates at birth with their 95% CI by ethnicity and sex. Precise estimates are also available in table 4.1 in column 4 for HLE at birth and column 5 for DFLE at birth. HLE at birth for the White Scottish population (taken as reference; dotted line in figure 4.1) was 67.4 years for males and 70.7 for females. DFLE at birth for the White Scottish population was 59.4 years for males and 61.9 for females. The expected number of years living disability-free was about 7 years shorter than the expected number of years living healthily in the reference population. White Scottish females had significantly (CIs do not overlap) longer HLE and DFLE than White Scottish males. It is worth noting that in all ethnic groups, females had longer (not necessarily significantly) HLE and DFLE at birth than their male counterparts with the exception of Indian and Pakistani females.

In males, HLE at birth was significantly longer in the Other White British, Other White, Indian and Chinese groups compared to the White Scottish population. Chinese males had the longest HLE at birth (73.1), almost 6 years over that of White Scottish males. In contrast, HLE at birth was significantly shorter in males of White Irish, Any Mixed Background and Pakistani origins compared to the reference group. In Females, HLE at birth was significantly longer in the Other White British, Other White and Chinese groups compared to the reference group. Pakistani females had the shortest HLE at birth (64.2 years), 6.5 years shorter than that of White Scottish females.

DFLE findings showed similar ethnic patterns to HLE findings in males i.e. there was significantly longer DFLE at birth in Other White British, Other White, Indian and Chinese males while shorter DFLE at birth in White Irish, Any Mixed Background and Pakistani males. In females, there was similar ethnic patterns in DFLE as in HLE with longer DFLE at birth in Other White British, Other White and Chinese females and shorter DFLE in Pakistani females. In addition to HLE patterns, DFLE results also showed significantly shorter DFLE at birth in Any Mixed Background and Indian females compared to the reference group.

Contrary to published LE estimates at birth where most minority ethnic groups were expected to live longer than the White Scottish majority (Gruer et al., 2016), ethnic patterns in HLE and DFLE showed differences going both ways. As expected the Chinese population experienced an advantage in health expectancies and the Pakistani population a disadvantage. Shorter health expectancies were also found in White Irish and Any Mixed Background males compared to White Scottish males. The use of DFLE (but not HLE) revealed a significant disadvantage (shorter disability-free life expectancy) in Any Mixed Background and Indian females compared to White Scottish females.

Figure 4.1. HLE and DFLE at birth (95% CI) by ethnicity and sex

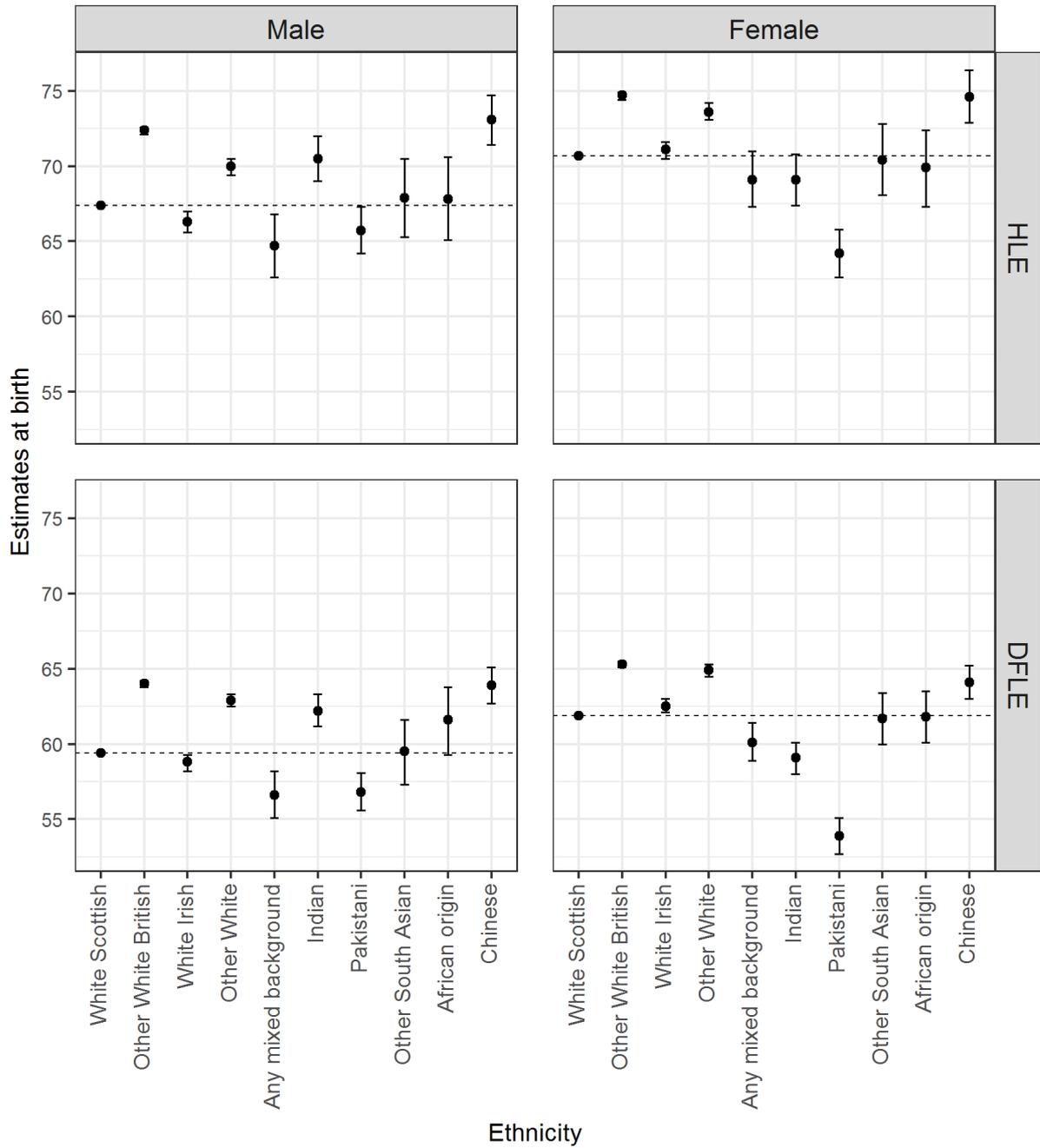


Table 4.1. Health expectancies (HLE and DFLE) and Life expectancy (LE)* at birth (95% CI) by ethnicity and sex in Scotland

Sex and ethnic groups	Population Census 2001	LE at birth* and 95% CI	HLE at birth and 95% CI	DFLE at birth and 95% CI	Number of years lived in poor health at birth and 95% CI	Number of years lived with limitations at birth and 95% CI	Proportion of life spent in good health at birth	Proportion of life spent without limitations at birth
MALES								
White Scottish	1,949,480	74.7 [74.6, 74.8]	67.4 [67.3, 67.5]	59.4 [59.3, 59.4]	7.3 [7.3, 7.4]	15.3 [15.3, 15.4]	90.2%	79.5%
Other White British	160,235	78.9 [78.6, 79.2]	72.4 [72.1, 72.6]	64.0 [63.8, 64.2]	6.5 [6.4, 6.6]	15.0 [14.8, 15.1]	91.7%	81.1%
White Irish	20,340	75.0 [74.0, 75.9]	66.3 [65.6, 67.0]	58.8 [58.2, 59.3]	8.7 [8.4, 9.0]	16.2 [15.7, 16.7]	88.4%	78.4%
Other White	29,945	77.2 [76.4, 78.1]	70.0 [69.4, 70.5]	62.9 [62.5, 63.3]	7.3 [6.9, 7.6]	14.4 [13.9, 14.9]	90.6%	81.4%
Any Mixed Background	5,310	73.0 [70.2, 75.8]	64.7 [62.6, 66.8]	56.6 [55.1, 58.2]	8.3 [7.3, 9.3]	16.4 [14.8, 18.0]	88.6%	77.6%
Indian	6,450	80.9 [78.4, 83.4]	70.5 [69.0, 72.0]	62.2 [61.2, 63.3]	10.4 [9.1, 11.8]	18.7 [16.9, 20.5]	87.1%	76.9%
Pakistani	12,930	79.3 [76.9, 81.6]	65.7 [64.2, 67.3]	56.8 [55.6, 58.1]	13.6 [12.4, 14.7]	22.5 [21.0, 23.9]	82.9%	71.7%
Other South Asian	3,550	76.2 [72.6, 79.7]	67.9 [65.3, 70.5]	59.5 [57.3, 61.6]	8.3 [6.9, 9.6]	16.7 [14.8, 18.6]	89.1%	78.1%
African origin	3,275	75.3 [71.6, 79.0]	67.8 [65.1, 70.6]	61.6 [59.3, 63.8]	7.5 [6.3, 8.7]	13.8 [12.0, 15.6]	90.0%	81.7%
Chinese	6,530	79.0 [76.5, 81.5]	73.1 [71.4, 74.7]	63.9 [62.7, 65.1]	5.9 [4.8, 7.0]	15.1 [13.4, 16.7]	92.5%	80.9%
FEMALES								
White Scottish	2,138,640	79.4 [79.3, 79.5]	70.7 [70.7, 70.8]	61.9 [61.8, 61.9]	8.7 [8.7, 8.7]	17.5 [17.5, 17.6]	89.1%	77.9%
Other White British	174,750	82.6 [82.3, 82.9]	74.7 [74.4, 74.9]	65.3 [65.1, 65.5]	7.9 [7.8, 8.0]	17.3 [17.1, 17.4]	90.4%	79.1%
White Irish	23,160	81.0 [80.2, 81.8]	71.1 [70.5, 71.6]	62.5 [62.1, 63.0]	9.9 [9.6, 10.3]	18.5 [18.0, 19.0]	87.7%	77.2%
Other White	35,710	82.0 [81.3, 82.8]	73.6 [73.1, 74.2]	64.9 [64.5, 65.3]	8.4 [8.1, 8.7]	17.2 [16.7, 17.6]	89.8%	79.1%
Any Mixed Background	5,800	79.3 [76.6, 82.0]	69.1 [67.3, 71.0]	60.1 [58.9, 61.4]	10.2 [9.1, 11.3]	19.2 [17.4, 20.9]	87.2%	75.8%
Indian	5,890	83.3 [80.7, 85.9]	69.1 [67.4, 70.8]	59.1 [58.0, 60.1]	14.2 [12.8, 15.6]	24.2 [22.3, 26.2]	83.0%	70.9%
Pakistani	12,700	84.6 [82.0, 87.3]	64.2 [62.6, 65.8]	53.9 [52.7, 55.1]	20.4 [18.7, 22.1]	30.7 [28.7, 32.7]	75.9%	63.7%
Other South Asian	2,965	81.1 [78.0, 84.3]	70.4 [68.1, 72.8]	61.7 [60.0, 63.4]	10.7 [9.4, 12.0]	19.5 [17.4, 21.5]	86.8%	76.0%
African origin	3,055	78.7 [75.1, 82.3]	69.9 [67.3, 72.4]	61.8 [60.1, 63.5]	8.9 [7.4, 10.3]	16.9 [14.6, 19.2]	88.8%	78.5%
Chinese	6,670	83.4 [81.1, 85.7]	74.6 [72.9, 76.4]	64.1 [63.0, 65.2]	8.8 [7.8, 9.8]	19.3 [17.6, 20.9]	89.5%	76.9%

* The published life expectancy at birth by ethnic group (Gruer et al., 2016) is included in column 3 to enable further calculation in combination with HLE and DFLE.

4.3.2. Ethnic differences in SAH in Scotland

Figure 4.2 shows age-adjusted RRs with their 95%CI for ethnic differences in SRH and in LLTI in 2001, stratified by sex (in separate panels). The top panels related to SRH present the RRs of reporting bad health versus good/fair health by ethnic group taking the White Scottish population as reference (RR=1; dotted line). Similarly, for the bottom panels related to LLTI, RRs of reporting a LLTI by ethnicity are presented. RRs (95% CI) and associated p-values are also reported in table 4.2 for more precise figures. Overall, 10% of the SHELS sample reported bad health and 20% reported a LLTI.

Our findings showed lower RRs of reporting bad health in the Other White British, Other White and Chinese groups and higher RRs of reporting bad health in the White Irish, Any Mixed Background and Pakistani groups compared to the White Scottish in both males and females. RRs of reporting bad health were also higher in Indian and Other South Asian females compared to the reference group. Pakistani females had the highest RR (1.84; 95% CI [1.72, 1.96]) with almost twice the risk of their White Scottish counterparts. Our findings showed overall consistent results when using SRH (Bad health) or LLTI as a measure of SAH by ethnicity. LLTI showed overall more conservative results (RR closer to the reference line). However, there was some additional differences revealed when using LLTI as measure of health. Indian males and African males and females had a lower risk of reporting a LLTI compared to the reference group while a reported advantage did not appear or not significantly when using SRH (Bad health).

Findings were in the expected directions. Analysing ethnic differences in SAH allowed us to appreciate patterns in more ethnic groups and with a better ethnic granularity than when using HLE/DFLE indicators, limited by small number of mortality events to produce reliable estimates. However, SAH findings were consistent with HLE/DFLE ethnic patterns in that the Other White British, Other White and Chinese groups reported a health advantage and the Any Mixed Background and Pakistani groups a health disadvantage compared to the White Scottish group. Compared to the White Scottish population, Indian males tended to report a health advantage while Indian females a health disadvantage.

Finally, the results of the sensitivity analysis of SRH using Bad/Fair health as outcome instead of Bad health (appendix 4.1) were overall consistent with previous findings using SRH (Bad health) but were closer to the reference value 1 (smaller differences). Contrasting patterns to highlight were that Chinese males and females had similar risk and Bangladeshi males higher

risk of reporting “Bad/Fair” health compared to the reference population. In effect, it meant that these ethnic groups had a higher propensity to report fair health which was strong enough to change the observed ethnic patterns in SRH. This questions the meaning of fair health for these populations and for the majority population and how this reporting relates to their actual health status.

Figure 4.2. Age-adjusted RRs (95% CI) of ethnic differences in SRH and LLTI by sex

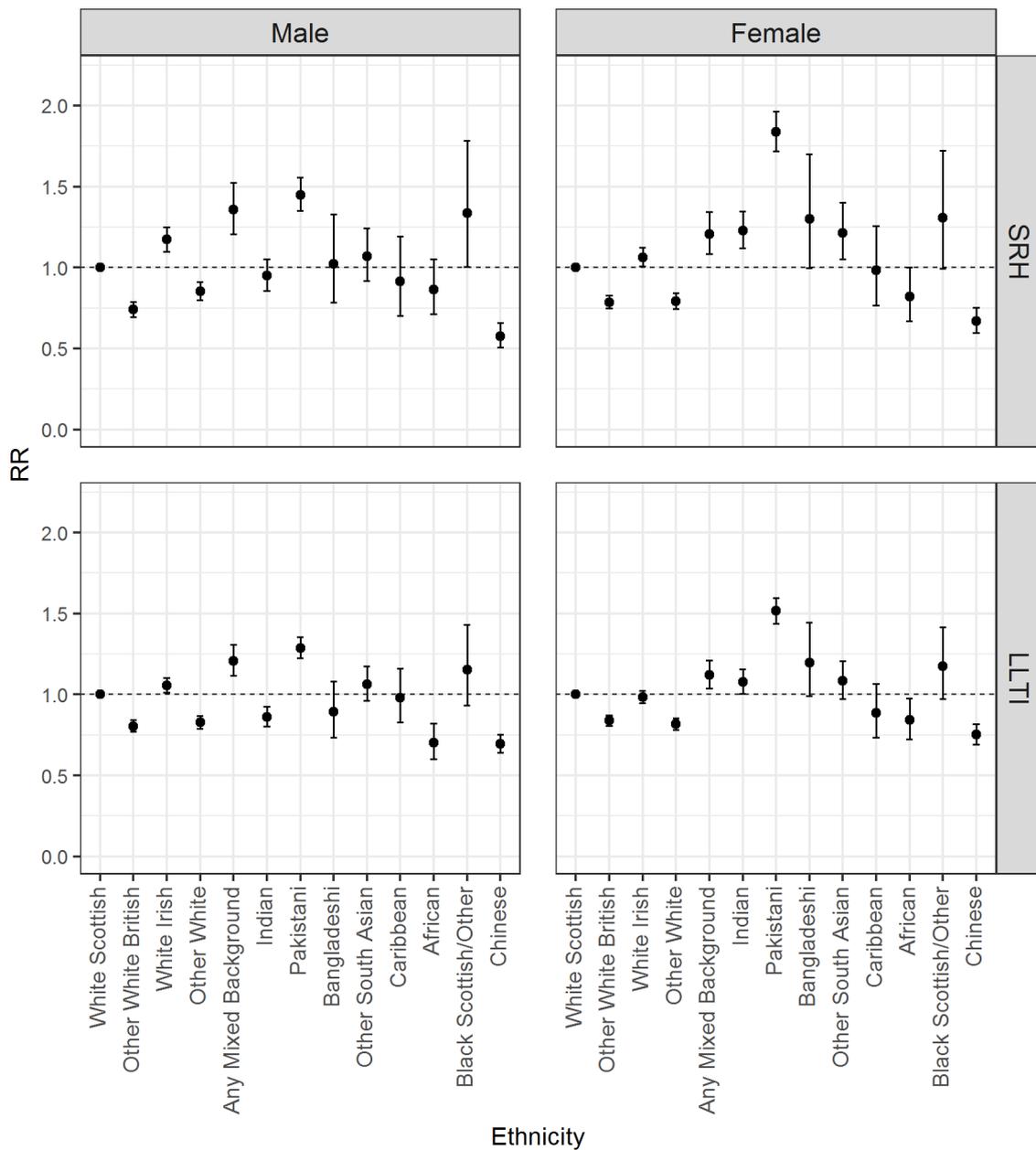


Table 4.2. RRs (95% CI) of reporting Bad health or a LLTI in 2001 by ethnicity, stratified by sex. Models are adjusted for age and for age and SES

Sex and ethnic groups	N	Reported Bad health	SRH (Bad health)				LLTI				
			Adjusted for Age		Adjusted for Age and SES		Adjusted for Age		Adjusted for Age and SES		
			RR (95% CI)	p-value	RR (95% CI)	p-value	Reported a LLTI	RR (95% CI)	p-value	RR (95% CI)	p-value
MALES											
White Scottish	1887415	170395	1		1		343095	1		1	
Other White British	153805	11645	0.74 (0.69, 0.79)	<.0001	1.00 (0.98, 1.03)	0.7164	24685	0.81 (0.77, 0.84)	<.0001	1.00 (0.98, 1.02)	0.8087
White Irish	19465	2565	1.17 (1.10, 1.25)	<.0001	1.12 (1.08, 1.17)	<.0001	4580	1.06 (1.01, 1.10)	0.0143	1.02 (0.99, 1.05)	0.2267
Other White	28835	1980	0.85 (0.80, 0.91)	<.0001	0.98 (0.94, 1.03)	0.4980	3800	0.83 (0.79, 0.87)	<.0001	0.91 (0.88, 0.95)	<.0001
Any Mixed Background	5260	310	1.36 (1.21, 1.52)	<.0001	1.31 (1.18, 1.46)	<.0001	615	1.21 (1.12, 1.31)	<.0001	1.19 (1.11, 1.28)	<.0001
Indian	6425	425	0.95 (0.86, 1.05)	0.3136	1.29 (1.18, 1.41)	<.0001	770	0.86 (0.80, 0.93)	<.0001	1.08 (1.01, 1.15)	0.0264
Pakistani	12905	1080	1.45 (1.35, 1.56)	<.0001	1.58 (1.49, 1.67)	<.0001	1980	1.29 (1.22, 1.36)	<.0001	1.37 (1.31, 1.43)	<.0001
Bangladeshi	860	50	1.02 (0.79, 1.33)	0.8699	1.03 (0.80, 1.32)	0.8263	90	0.89 (0.74, 1.08)	0.2441	0.90 (0.75, 1.08)	0.2646
Other South Asian	2670	180	1.07 (0.92, 1.24)	0.3939	1.07 (0.93, 1.22)	0.3634	355	1.06 (0.96, 1.18)	0.2226	1.07 (0.97, 1.17)	0.1714
Caribbean	700	50	0.92 (0.70, 1.19)	0.5093	0.93 (0.71, 1.20)	0.5541	100	0.98 (0.83, 1.16)	0.8181	0.99 (0.84, 1.17)	0.9124
African	2100	105	0.86 (0.71, 1.05)	0.1433	0.80 (0.67, 0.96)	0.0154	165	0.70 (0.60, 0.82)	<.0001	0.67 (0.58, 0.78)	<.0001
Black Scottish/Other	445	40	1.34 (1.00, 1.79)	0.0476	1.11 (0.84, 1.46)	0.4570	65	1.15 (0.93, 1.43)	0.1885	1.00 (0.82, 1.23)	0.9808
Chinese	6500	240	0.58 (0.51, 0.66)	<.0001	0.64 (0.57, 0.73)	<.0001	580	0.70 (0.64, 0.75)	<.0001	0.74 (0.69, 0.80)	<.0001
FEMALES											
White Scottish	1995375	197200	1		1		372375	1		1	
Other White British	162370	13690	0.79 (0.75, 0.83)	<.0001	1.01 (0.99, 1.03)	0.2714	26795	0.84 (0.81, 0.87)	<.0001	1.01 (1.00, 1.03)	0.1630
White Irish	21170	2835	1.06 (1.01, 1.12)	0.0268	1.10 (1.06, 1.14)	<.0001	5015	0.99 (0.95, 1.02)	0.4355	1.00 (0.98, 1.03)	0.8963
Other White	34200	2390	0.79 (0.75, 0.84)	<.0001	0.96 (0.93, 1.01)	0.0815	4460	0.82 (0.78, 0.86)	<.0001	0.95 (0.92, 0.98)	0.0004
Any Mixed Background	5675	345	1.21 (1.08, 1.34)	0.0006	1.20 (1.09, 1.32)	0.0002	605	1.12 (1.04, 1.21)	0.0041	1.11 (1.04, 1.19)	0.0030
Indian	5855	505	1.23 (1.12, 1.35)	<.0001	1.58 (1.45, 1.72)	<.0001	790	1.08 (1.00, 1.16)	0.0371	1.30 (1.22, 1.38)	<.0001
Pakistani	12690	1345	1.84 (1.72, 1.96)	<.0001	1.95 (1.85, 2.06)	<.0001	2005	1.52 (1.44, 1.60)	<.0001	1.59 (1.52, 1.66)	<.0001
Bangladeshi	700	50	1.30 (1.00, 1.70)	0.0524	1.33 (1.03, 1.72)	0.0295	85	1.20 (0.99, 1.45)	0.0606	1.21 (1.01, 1.46)	0.0411
Other South Asian	2205	175	1.21 (1.05, 1.40)	0.0078	1.21 (1.05, 1.39)	0.0068	280	1.08 (0.97, 1.21)	0.1540	1.07 (0.97, 1.19)	0.1909
Caribbean	755	60	0.98 (0.77, 1.26)	0.8836	1.14 (0.90, 1.45)	0.2700	95	0.89 (0.74, 1.07)	0.1981	1.00 (0.83, 1.19)	0.9528
African	1795	90	0.82 (0.67, 1.00)	0.0527	0.78 (0.64, 0.95)	0.0120	155	0.84 (0.73, 0.98)	0.0236	0.81 (0.70, 0.94)	0.0043
Black Scottish/Other	460	45	1.31 (0.99, 1.72)	0.0562	1.20 (0.92, 1.56)	0.1898	75	1.18 (0.97, 1.42)	0.0915	1.09 (0.90, 1.32)	0.3872
Chinese	6600	310	0.67 (0.60, 0.75)	<.0001	0.77 (0.69, 0.85)	<.0001	615	0.75 (0.69, 0.82)	<.0001	0.82 (0.76, 0.89)	<.0001

4.3.3. The contribution of SES in the ethnic patterning of SAH in Scotland

Figure 4.3 (and table 4.2) shows the RRs of SRH (bad health) or LLTI by ethnicity and stratified by sex, first adjusted for age in a base model (model in red) and then adjusted for age and SES (model in blue).

In contrast to the age-adjusted base model, the model adjusted for age and SES showed similar risk of reporting bad health in Other White British and Other White populations compared to the White Scottish population. The advantage in SRH previously observed in these populations prior to controlling for SES is no longer visible after controlling for SES suggesting that these ethnic differences in SRH can be explained by socio-economic differences between ethnic groups. However, the lower RRs of reporting bad health persisted in the Chinese population with a small convergence towards the reference once SES was accounted for. Furthermore, the disadvantage observed in the Any Mixed Background and Pakistani groups also persisted after adjustment for SES. The latter finding questions the ability of the three SES indicators used in this analysis in measuring deprivation consistently across groups. Alternatively, SES might not explain health inequalities in non-white minority ethnic groups. Finally, findings in the Indian population stand out. The Indian population had a relatively favourable socio-economic profile in Scotland in 2001. Similar age-adjusted risk of reporting bad health appeared significantly higher in Indian males and high age-adjusted RRs were even higher in Indian females when accounting for their favourable SES profile.

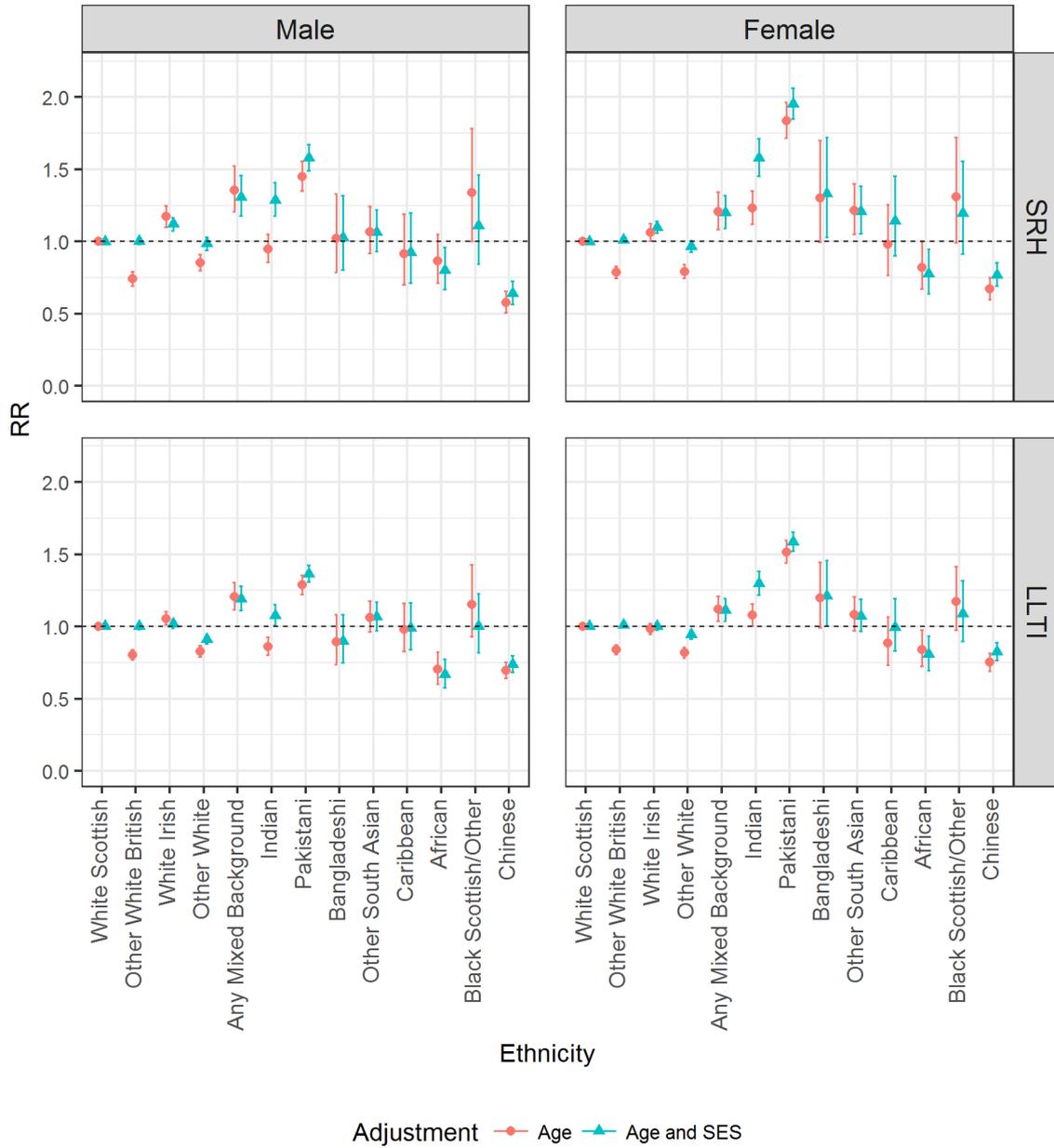
Overall, the LLTI results showed similar patterns once SES was accounted for. In white groups (Other White British, Irish and Other White), most RRs of reporting a LLTI were at the level of the White Scottish population when adjusted for SES. However, LLTI remained significantly lower in Other White males and females. This emphasises the SES effect in explaining or reducing ethnic inequalities in health in white ethnic groups. Similarly to SRH results, adjustment for SES had little effect on the observed ethnic differences in LLTI in non-white minority ethnic groups with the exception of higher RRs in the Indian populations.

In summary, the relative advantage observed in the Other White British and Other White groups mostly disappeared after SES adjustment, but SES adjustment had little effect on the patterns observed in other non-white minority ethnic groups. A relative disadvantage in SAH appeared in the Indian population when accounting for their favourable socio-economic profile.

They would have worse reported health if they had similar SES level as the majority White Scottish population.

A complementary analysis showed that the interaction of each SES indicator with ethnicity in predicting SAH was significant for specific SES indicators and specific ethnic groups (data not shown). For example, renting compared to owning (household tenure) was associated with a clear lower risk of reporting worse health outcomes in Indian, Pakistani and Chinese males and females compared to the White Scottish group and for both SRH and LLTI. Living in more deprived areas compared to least deprived areas (SIMD) was also associated with a lower risk of reporting worse health outcomes in the Pakistani population compared to the White Scottish population, in both males and females and for both SRH and LLTI. However, there was no evidence of consistent and significant interaction between the education indicator used in this analysis and ethnicity in predicting SAH. The interaction findings supports some differential SES-SAHA association for specific ethnic groups and certain SES measures.

Figure 4.3. RRs (95% CI) of ethnic differences in SRH and LLTI by sex, adjusted for age (red) and adjusted for age and SES (blue)



4.3.4. SAH patterns by ethnicity and UK-birth in Scotland

To explore the SAH patterns in relation to ethnicity and UK-birth, LLTI results are presented. As explained before, a similar analysis was done using SRH as a SAH measure, the corresponding results are made available in appendix 4.2.

Tables 4.3a&b show ethnic differences in reporting a LLTI by sex and for 3 sets of adjustment: adjusted for age, adjusted for age and UK-birth and adjusted for age, UK-birth and the interaction between UK-birth and ethnicity. The general patterns of UK-birth in predicting the risk of reporting worse health went in the expected direction. Being born outside the UK was associated with a lower risk of reporting worse health compared of being born in the UK. For example, in the model adjusted for age and UK-birth, the RRs (95% CI) of reporting a LLTI in those born outside the UK were 0.86 [0.84;0.89] for males and 0.88 [0.86;0.90] for females compared to those born in the UK taken as the reference (RR=1).

In relation to the ethnic patterns in LLTI, adjustment for UK-birth had a small effect. Compared to the age-adjusted base model, further adjusting for UK-birth had the effect to shift RRs of reporting a LLTI to higher values in most ethnic groups. It meant that if ethnic minorities were as likely to be born in the UK as the White Scottish, they would be more likely to report worse health. Despite this slight increased likelihood of reporting poorer health in ethnic minorities once the UK-birth distribution is accounted for, the ethnic patterning of reporting an advantage or a disadvantage in LLTI was similar in the model adjusted for age and in the model adjusted for age and UK-birth, with some exceptions. For example, Indian males had a lower risk of reporting a LLTI in the age-adjusted model but a similar risk compared to the White Scottish population once UK-birth was included in the model. Indian females had a similar risk of LLTI in the age-adjusted model but a higher risk of reporting a LLTI in the model including UK-birth.

In the last model including the interaction term between UK-birth and ethnicity, the results showed a significant interaction for specific ethnic groups. It meant that the strength of UK-birth in predicting the risk of reporting a LLTI differed for some ethnic groups compared to the White Scottish population. There was a significant interaction UK-birth*ethnicity in predicting LLTI for males and females of Other White, Any Mixed Background, and African origins as well as Caribbean females in the direction that, for these groups, being born outside the UK compared to born in the UK was associated with an even lower risk of reporting a LLTI than for the White Scottish population. In other words, the effect of UK-birth in predicting LLTI was

stronger for these ethnic groups. This could be translated into a wider the gap in reporting a LLTI between those born outside the UK and those born in the UK for those specific ethnic groups. For Other White British, White Irish and Pakistani males and females, there was a significant interaction term which went in the opposite direction. It meant that the strength of the UK-birth-LLTI relationship was weaker in these populations compared to their White Scottish counterparts.

To visualise and understand the direction of these differences in the UK-birth and LLTI association by ethnicity, figure 4.4 presents the RRs of reporting a LLTI by a combined ethnicity-UK-birth variable. All groups are compared to the White Scottish population who was born in the UK. The figure differentiates ethnic groups who were born in the UK (in black) from ethnic groups who were born outside the UK (in white). The age-adjusted results (top panels) showed that those who were born outside the UK are less likely to report a LLTI compared to the White Scottish population born in the UK, for all ethnic groups with a few exceptions. The risk of reporting a LLTI was significantly lower in those who were born outside the UK in males and females of White Scottish, Other White British, Other White, African and Chinese origins as well as in Indian males, Any Mixed Background females and Caribbean females compared to the reference group. However, Pakistani males and females who were born outside the UK had higher RRs of reporting a LLTI compared to the White Scottish population who was born in the UK. For those who were born in the UK, Other White British and Chinese males and females had lower risks of reporting a LLTI compared to the reference group. The age-adjusted RRs of reporting a LLTI were either similar or higher in all other ethnic groups born in the UK. Any Mixed Background males and females in particular had a significantly higher risk of reporting a LLTI compared to the reference group.

Further adjustment for SES (figure 4.4, bottom panels) completely removed the differences observed in the Other White British and White Irish groups, for both those who were born in and outside the UK and in the White Scottish group born outside the UK in comparison to the reference group. However, the age-adjusted patterns observed in the Other White and other non-white minority ethnic groups who were born in or outside the UK remained similar when the analysis adjusted for SES except for the Indian groups for which RRs were higher once their favourable SES profile was accounted for.

As expected, the results showed that being born outside the UK rather than born in the UK has a protective effect on reported health for most ethnic groups. This supports the healthy migrant effect hypothesis. Findings were also in line with an underlying acculturation process whereby health converges or worsens in descendants of migrants. However, the observed general patterns did not apply for all ethnic groups. The UK-birth and reported LLTI relationship differed by ethnic group with the most striking finding being that Pakistani born outside the UK reported the worst health while their descendants were less disadvantaged, even after SES adjustment. These findings in the Pakistani population challenge the healthy migrant effect and acculturation hypotheses as explanations for ethnic differences in health and direct to further avenues to be explored.

Figure 4.4. RRs (95% CI) of LLTI by ethnicity and UK-birth, stratified by sex, adjusted for age (top) and adjusted for age and SES (bottom)

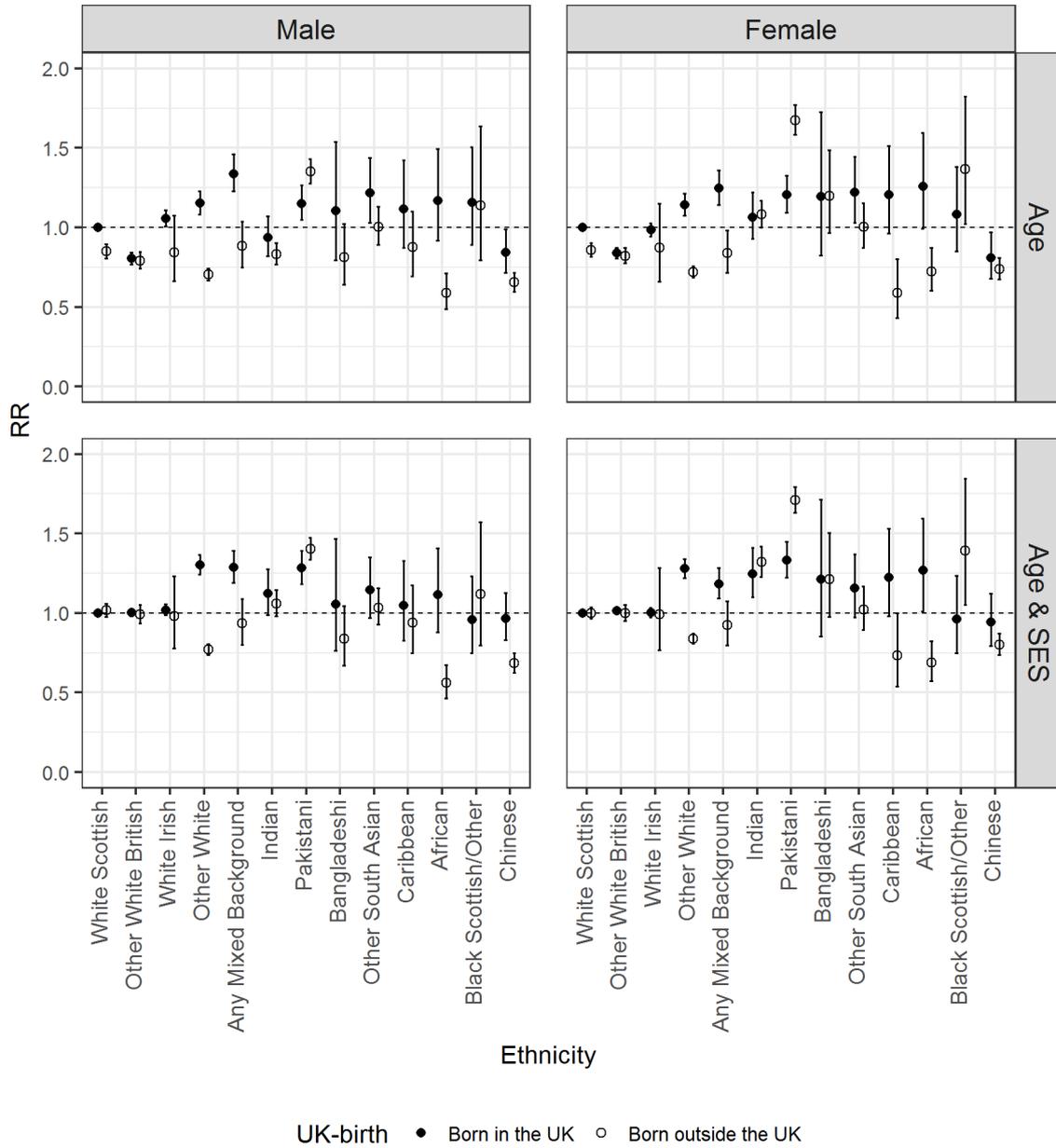


Table 4.3. RRs (95% CI) of reporting a LLTI in 2001 by ethnicity, stratified by sex. Models are adjusted for age, for age and UK-birth and for age, UK-birth and the interaction between UK-birth and ethnicity

a) MALES		Adjusted for Age		Adjusted for Age and UK-birth		Adjusted for Age, UK-birth, ethnicity*UK-birth		
	N	Reported a LLTI	RR (95% CI)	p-value	RR (95% CI)	p-value	RR (95% CI)	p-value
White Scottish	1887415	343095	1		1		1	
Other White British	153805	24685	0.81 (0.77, 0.84)	<.0001	0.81 (0.77, 0.85)	<.0001	0.80 (0.77, 0.84)	<.0001
White Irish	19465	4580	1.06 (1.01, 1.10)	0.0143	1.12 (1.08, 1.17)	<.0001	1.06 (1.01, 1.13)	0.0274
Other White	28835	3800	0.83 (0.79, 0.87)	<.0001	0.92 (0.88, 0.96)	0.0002	1.15 (1.08, 1.23)	<.0001
Any Mixed Background	5260	615	1.21 (1.12, 1.31)	<.0001	1.26 (1.16, 1.36)	<.0001	1.34 (1.23, 1.46)	<.0001
Indian	6425	770	0.86 (0.80, 0.93)	<.0001	0.96 (0.89, 1.03)	0.2276	0.94 (0.82, 1.07)	0.3522
Pakistani	12905	1980	1.29 (1.22, 1.36)	<.0001	1.42 (1.35, 1.49)	<.0001	1.15 (1.05, 1.26)	0.0041
Bangladeshi	860	90	0.89 (0.74, 1.08)	0.2441	0.99 (0.82, 1.20)	0.9170	1.11 (0.80, 1.54)	0.5442
Other South Asian	2670	355	1.06 (0.96, 1.18)	0.2226	1.18 (1.07, 1.30)	0.0010	1.21 (1.02, 1.43)	0.0296
Caribbean	700	100	0.98 (0.83, 1.16)	0.8181	1.06 (0.90, 1.26)	0.4919	1.11 (0.87, 1.42)	0.3836
African	2100	165	0.70 (0.60, 0.82)	<.0001	0.79 (0.68, 0.92)	0.0029	1.17 (0.92, 1.50)	0.2002
Black Scottish/Other	445	65	1.15 (0.93, 1.43)	0.1885	1.21 (0.97, 1.49)	0.0882	1.18 (0.91, 1.53)	0.2198
Chinese	6500	580	0.70 (0.64, 0.75)	<.0001	0.78 (0.72, 0.84)	<.0001	0.84 (0.72, 0.99)	0.0367
Born outside UK vs UK-born					0.86 (0.84, 0.89)	<.0001	0.86 (0.81, 0.90)	<.0001
White Scottish*Born outside UK							1	
Other White British*Born outside UK							1.16 (1.06, 1.26)	0.0007
White Irish*Born outside UK							1.14 (1.05, 1.25)	0.0025
Other White*Born outside UK							0.71 (0.65, 0.78)	<.0001
Any Mixed Background*Born outside UK							0.77 (0.64, 0.92)	0.0048
Indian*Born outside UK							1.04 (0.88, 1.22)	0.6689
Pakistani*Born outside UK							1.37 (1.23, 1.54)	<.0001
Bangladeshi*Born outside UK							0.85 (0.57, 1.28)	0.4445
Other South Asian*Born outside UK							0.98 (0.79, 1.20)	0.8164
Caribbean*Born outside UK							0.92 (0.65, 1.28)	0.6111
African*Born outside UK							0.59 (0.43, 0.80)	0.0007
Black Scottish/Other*Born outside UK							1.09 (0.70, 1.71)	0.7065
Chinese*Born outside UK							0.91 (0.75, 1.09)	0.3037

b) FEMALES

	N	Reported a LLTI	Adjusted for Age		Adjusted for Age and UK- birth		Adjusted for Age, UK-birth and ethnicity*UK-birth	
			RR (95% CI)	p-value	RR (95% CI)	p-value	RR (95% CI)	p-value
White Scottish	1995375	372375	1		1		1	
Other White British	162370	26795	0.84 (0.81, 0.87)	<.0001	0.84 (0.81, 0.88)	<.0001	0.84 (0.81, 0.87)	<.0001
White Irish	21170	5015	0.99 (0.95, 1.02)	0.4355	1.05 (1.01, 1.09)	0.0098	1.02 (0.97, 1.07)	0.5014
Other White	34200	4460	0.82 (0.78, 0.86)	<.0001	0.90 (0.86, 0.94)	<.0001	1.14 (1.07, 1.21)	<.0001
Any Mixed Background	5675	605	1.12 (1.04, 1.21)	0.0041	1.17 (1.08, 1.26)	0.0001	1.25 (1.15, 1.36)	<.0001
Indian	5855	790	1.08 (1.00, 1.16)	0.0371	1.18 (1.10, 1.27)	<.0001	1.06 (0.93, 1.22)	0.3780
Pakistani	12690	2005	1.52 (1.44, 1.60)	<.0001	1.65 (1.56, 1.74)	<.0001	1.21 (1.10, 1.33)	0.0001
Bangladeshi	700	85	1.20 (0.99, 1.45)	0.0606	1.31 (1.08, 1.58)	0.0058	1.19 (0.83, 1.73)	0.3479
Other South Asian	2205	280	1.08 (0.97, 1.21)	0.1540	1.17 (1.05, 1.31)	0.0042	1.22 (1.03, 1.45)	0.0203
Caribbean	755	95	0.89 (0.74, 1.07)	0.1981	0.94 (0.78, 1.14)	0.5395	1.21 (0.96, 1.52)	0.1024
African	1795	155	0.84 (0.73, 0.98)	0.0236	0.93 (0.80, 1.08)	0.3319	1.26 (1.00, 1.60)	0.0537
Black Scottish/Other	460	75	1.18 (0.97, 1.42)	0.0915	1.22 (1.01, 1.48)	0.0363	1.09 (0.86, 1.39)	0.4719
Chinese	6600	615	0.75 (0.69, 0.82)	<.0001	0.84 (0.77, 0.91)	<.0001	0.81 (0.68, 0.97)	0.0226
Born outside UK vs UK-born					0.88 (0.86, 0.90)	<.0001	0.87 (0.83, 0.91)	<.0001
White Scottish*Born outside UK							1	
Other White British*Born outside UK							1.13 (1.04, 1.22)	0.0025
White Irish*Born outside UK							1.08 (1.00, 1.17)	0.0445
Other White*Born outside UK							0.73 (0.67, 0.79)	<.0001
Any Mixed Background*Born outside UK							0.77 (0.65, 0.93)	0.0055
Indian*Born outside UK							1.17 (1.00, 1.38)	0.0522
Pakistani*Born outside UK							1.60 (1.43, 1.79)	<.0001
Bangladeshi*Born outside UK							1.16 (0.76, 1.77)	0.5018
Other South Asian*Born outside UK							0.95 (0.76, 1.18)	0.6365
Caribbean*Born outside UK							0.56 (0.38, 0.83)	0.0033
African*Born outside UK							0.66 (0.49, 0.89)	0.0069
Black Scottish/Other*Born outside UK							1.42 (0.97, 2.07)	0.0717
Chinese*Born outside UK							1.05 (0.86, 1.29)	0.6306

4.3.5. The patterns of ethnic differences in SAH in older adults

As previously explained, the exploration of ethnic differences in SAH and whether it is differentiated in younger or older adults focused on the results using LLTI. However, SRH (bad health) results are made available in appendix 4.3 and provide similar findings.

A preliminary analysis (data not shown) of the interaction between adult age groups (16-49 years versus 50 years and over) and ethnicity in predicting the risk of reporting a LLTI found significant interaction for specific ethnic groups. As expected, being younger (16-49) rather than older (50+) was associated with a lower risk of reporting a LLTI. However, this association differed for some ethnic groups. The gap between younger and older adults in reporting a LLTI was significantly wider in males and females of White Irish, Other White, Indian, Pakistani and Chinese origins compared to their White Scottish counterparts.

Stratified analysis by adult age group provide the opportunity to visualise and understand the direction of ethnic differences in LLTI for the younger and the older adults. Figure 4.5 shows the age-adjusted RRs of reporting a LLTI by ethnicity, stratified by sex (different panels) and adult age groups (different colours). Analysis was done for each adult age group separately i.e. for those aged 16-49 years (model in red) and for those aged 50 years and over (model in blue). Precise estimates are presented in table 4.4.

In younger adults (16-49), results showed a clear advantage in reporting a LLTI in Other White British, Other White, African and Chinese males and females as well as in Indian males and White Irish females. Any Mixed Background and Pakistani males and females had a higher risk of reporting a LLTI compared to their White Scottish counterparts. In older adults (50+), a significant advantage in reporting a LLTI was also observed in Other White British and Other White males and females compared older reference population. In contrast to the patterns observed in the analysis for the younger adults, the likelihood of reporting a LLTI in older adults converge towards similar level in Indian males, White Irish females, African females and Chinese males and females. This supports a diminution or even disappearance of ethnic health inequalities in older ages. However, findings diverged for some ethnic groups. High RRs of reporting a LLTI were observed in older adults in Pakistani males and females, Bangladeshi females and Indian females while these patterns were either smaller (in Pakistani males and females), non-significant (in Bangladeshi females) or unapparent (in Indian females, no reported

disadvantage) in those aged 16 to 49 years. The findings in these South Asian groups support a widening of ethnic inequalities in health in older ages.

In summary, whether ethnic inequalities in health reduce or widen in older ages appeared to be ethnic specific. Generally, we found that ethnic differences in health reduced in older ages and converged towards the level of reported health of the majority population for those groups who reported better health than the White Scottish group at younger ages. However, for a few ethnic groups (Pakistani males and females, Bangladeshi females and Indian females), a clear disadvantage and greater differences appeared in older ages. This patterns would be expected under an accumulation of disadvantage over time and warrant further investigation.

Figure 4.5. Age-adjusted RRs (95% CI) of LLTI by ethnicity, stratified by sex and adults age groups

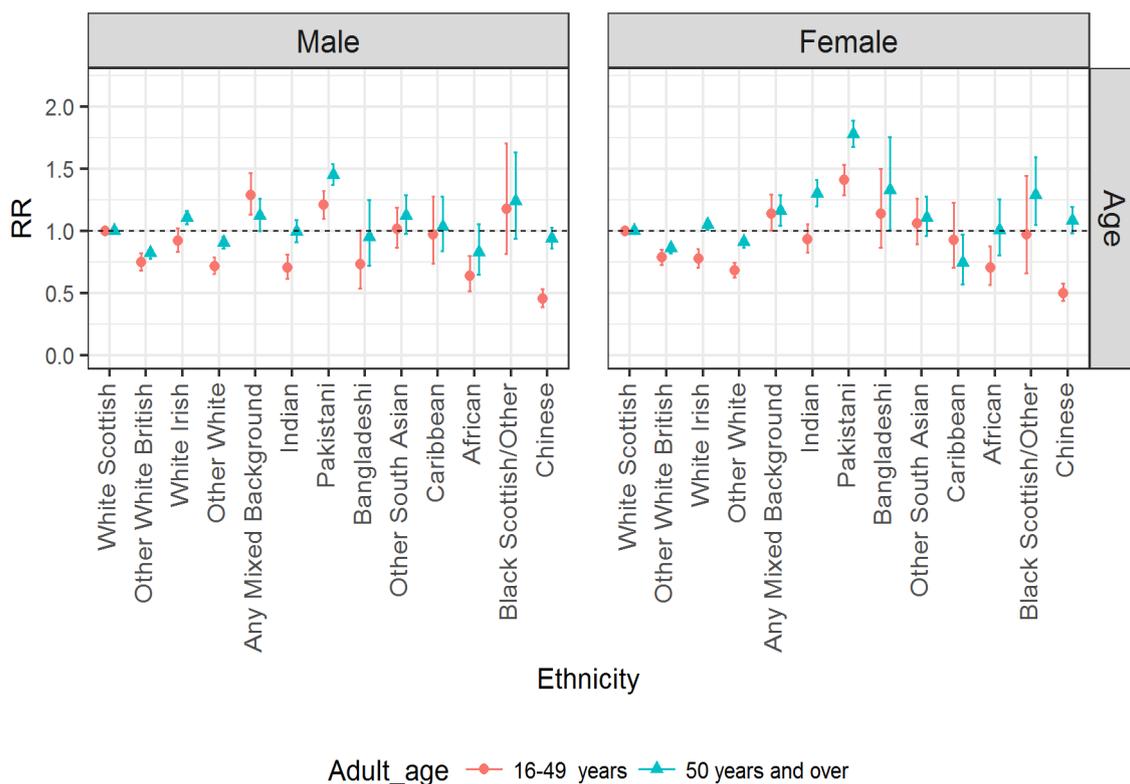


Table 4.4. RRs (95% CI) of reporting a LLTI in 2001 by ethnicity, stratified by sex and by age: 16-49 years old, above 50 years. Models are adjusted for age

Sex and ethnic groups	People aged 16-49				People aged 50 and over			
	Reported a LLTI	N	Age-adjusted RR (95% CI)	p-value	Reported a LLTI	N	Age-adjusted RR (95% CI)	p-value
MALES								
White Scottish	100100	900465	1		221275	573365	1	
Other White British	6985	81195	0.75 (0.68, 0.82)	<.0001	16730	54705	0.82 (0.78, 0.87)	<.0001
White Irish	1090	10405	0.92 (0.84, 1.02)	0.1313	3420	7750	1.11 (1.06, 1.16)	<.0001
Other White	1325	17295	0.72 (0.65, 0.79)	<.0001	2265	6405	0.91 (0.86, 0.96)	0.0002
Any Mixed Background	260	2210	1.29 (1.13, 1.47)	0.0001	200	475	1.12 (1.00, 1.26)	0.0471
Indian	275	3780	0.71 (0.62, 0.81)	<.0001	425	1145	0.99 (0.91, 1.09)	0.8922
Pakistani	870	7025	1.21 (1.10, 1.32)	<.0001	865	1550	1.45 (1.37, 1.54)	<.0001
Bangladeshi	35	495	0.73 (0.54, 1.00)	0.0524	40	110	0.95 (0.72, 1.25)	0.7203
Other South Asian	170	1515	1.02 (0.87, 1.19)	0.8547	145	380	1.12 (0.98, 1.29)	0.0982
Caribbean	50	450	0.97 (0.74, 1.28)	0.8342	50	120	1.04 (0.84, 1.28)	0.7506
African	95	1410	0.64 (0.51, 0.80)	<.0001	50	200	0.83 (0.65, 1.05)	0.1246
Black Scottish/Other	25	215	1.18 (0.82, 1.70)	0.3806	35	75	1.24 (0.94, 1.63)	0.1262
Chinese	185	3940	0.46 (0.39, 0.53)	<.0001	325	975	0.94 (0.86, 1.03)	0.1604
FEMALES								
White Scottish	108505	958980	1		248955	641180	1	
Other White British	8060	89380	0.79 (0.73, 0.85)	<.0001	18125	56115	0.86 (0.82, 0.90)	<.0001
White Irish	925	10570	0.78 (0.71, 0.85)	<.0001	4040	9425	1.05 (1.01, 1.09)	0.0191
Other White	1540	21375	0.68 (0.63, 0.74)	<.0001	2750	7890	0.91 (0.87, 0.96)	0.0002
Any Mixed Background	285	2660	1.14 (1.00, 1.29)	0.0445	235	560	1.16 (1.04, 1.29)	0.0057
Indian	340	3540	0.93 (0.83, 1.05)	0.2641	410	890	1.30 (1.20, 1.41)	<.0001
Pakistani	1010	7185	1.41 (1.29, 1.54)	<.0001	805	1290	1.78 (1.68, 1.89)	<.0001
Bangladeshi	45	400	1.14 (0.86, 1.50)	0.3538	35	70	1.33 (1.00, 1.76)	0.0466
Other South Asian	135	1255	1.06 (0.89, 1.26)	0.5033	120	305	1.11 (0.96, 1.28)	0.1579
Caribbean	50	475	0.93 (0.70, 1.23)	0.6008	40	145	0.75 (0.57, 0.97)	0.0310
African	85	1170	0.71 (0.57, 0.88)	0.0017	50	160	1.01 (0.81, 1.26)	0.9580
Black Scottish/Other	25	235	0.97 (0.66, 1.44)	0.8966	45	100	1.29 (1.05, 1.59)	0.0170
Chinese	220	4210	0.50 (0.44, 0.58)	<.0001	345	915	1.08 (0.98, 1.19)	0.1080

4.3.6. The contrasted patterns of ethnic differences in HLE/DFLE and LE in Scotland

Table 4.1 contrasts health expectancies at birth by ethnicity with life expectancy at birth by ethnicity. As explained in section 4.3.1, ethnic patterns in HLE and DFLE showed differences going both ways which contrasts with published LE estimates at birth where most minority ethnic groups showed an advantage, longer LE at birth, compared to the White Scottish majority (Gruer et al., 2016). Results at 50 years are also available in appendix 4.4.

Table 4.1 presents previously published LE at birth by ethnicity in Scotland (column 3). In males, the Indian (80.9), Pakistani (79.3) followed by the Chinese (79.0) and Other White British (78.9) groups had the longest LE at birth while the Any Mixed Background (73.0) and White Scottish (74.7) groups had the shortest. However, in terms of health expectancies (columns 4 and 5), the longest HLE and DFLE were found in Chinese and Other White British males and the lowest in the Any Mixed Background and Pakistani males. Albeit less marked than their LE advantage, Indian males had significantly longer HLE and DFLE compared to White Scottish males. Hence, a consistent advantage in LE and HLE/DFLE was observed in Other White British, Chinese and Indian males and a consistent disadvantage in Any Mixed Background males. Only Pakistani males broke this pattern, with one of the longest LE at birth combined with the shortest HLE (65.7) and DFLE (56.8) at birth. Similarly in females, the Pakistani (84.6), Chinese (83.4), Indian (83.3) and Other White British (82.6) groups had the longest LE at birth while Any Mixed Background (79.3), White Scottish (79.4) and African origin (78.7) females had the shortest. In line with LE results, a significant advantage in HLE and DFLE at birth appeared in the Other White British and Chinese groups. However, in contrast to their longest LE at birth, Indian and Pakistani females had the shortest HLE and DFLE.

Table 4.1 also presents a descriptive quantification of the differences between HLE/DFLE and LE through the estimated number of years lived in poor health (LE minus HLE) or with disability (LE minus DFLE) as well as through the proportion of years lived in good health (HLE divided by LE) or without disability (DFLE divided by LE). Figure 4.6 shows the number of years in poor health or with limitations by ethnicity and sex.

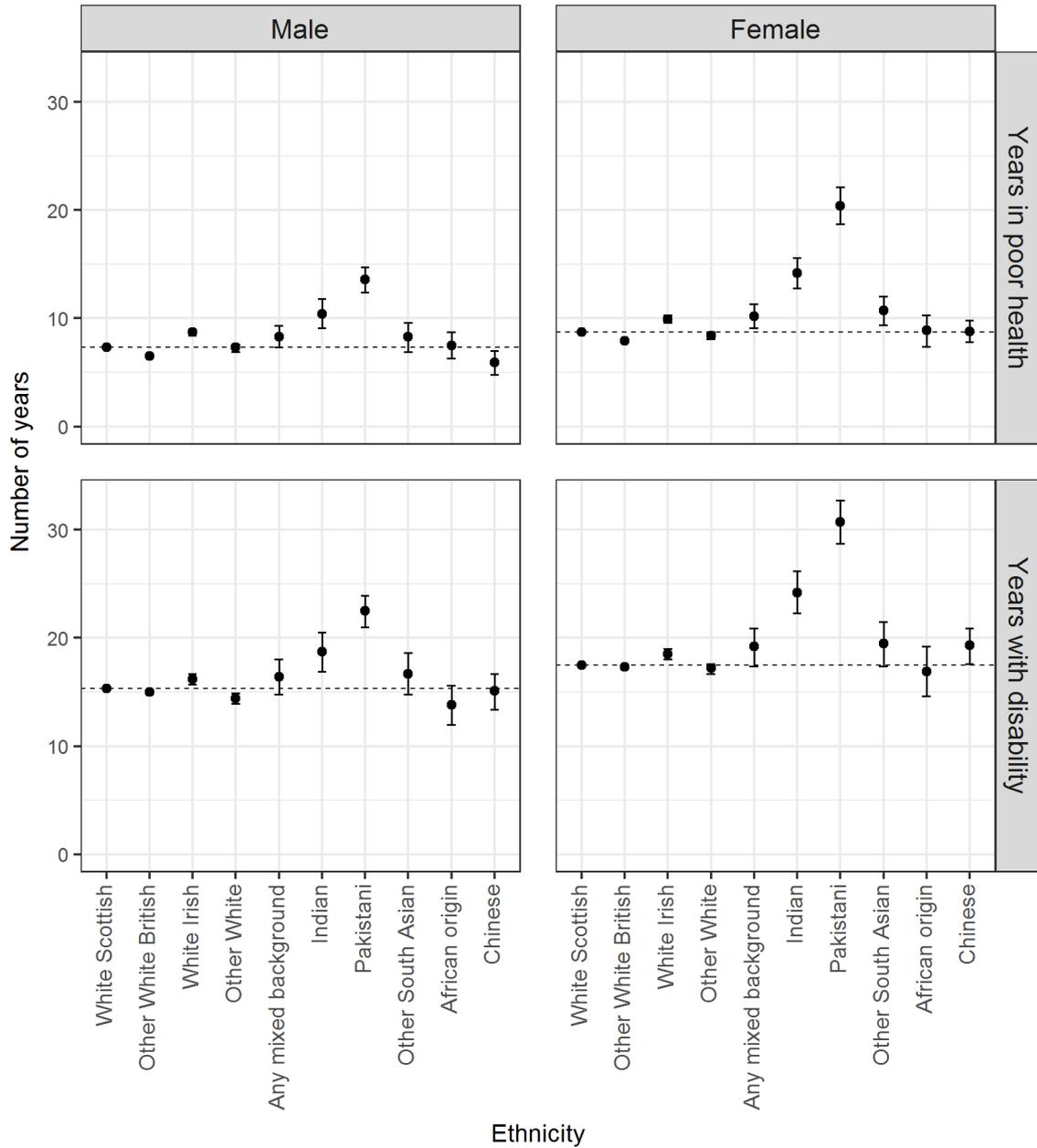
In males, the number of years lived in poor health was 7.3 years in the White Scottish population and ranged from 5.9 years in Chinese males to 13.6 years in Pakistani males. In females, the number of years lived in poor health was 8.7 years in White Scottish females and ranged from 8.4 years in Other White females to 20.4 years in Pakistani females. The number of

years lived in poor health was greater in females than in males in all ethnic groups. Most ethnic groups had around 6-9 years lived in poor health in males and 8-11 years in females with the clear exception of the Indian (10.4 years in males, 14.2 years in females) and Pakistani (13.6 years in males, 20.4 years in females) groups. Results in relation to years lived with limitations showed similar patterns of higher number of years in unhealthy state in the Indian (18.7 years in males, 24.2 years in females) and Pakistani populations (22.5 years in males, 30.7 years in females). Most ethnic groups had around 14-17 years lived with limitations in males and 17-20 years in females. Looking at the same problem through the proportion of life spent in good health or without limitations provided similar conclusions. The lowest percentages of life spent healthily was found in Indian and Pakistani populations. The most striking disadvantage was seen in Pakistani females with 75.9% of life spent in good health and 63.7% of life spent without limitations compared to respectively 89.1% and 77.9% in White Scottish females.

Consistently with results at birth, results at 50 years (appendix 4.4) showed longer LE at 50 years in Indian and Pakistani populations, but once health expectancies were considered, a disadvantage was revealed in Indian and Pakistani populations and particularly so in females. At 50 years, Pakistani females had the highest estimated number of years in unhealthy state with 16.6 years in poor health and 25.0 years with limitations compared to respectively 6.3 years and 13.7 years in White Scottish females. Results at 50 years also showed longer LE at 50 in Chinese females (34.5) compared to White Scottish females (30.9) but similar DFLE at 50 years (17.2 versus 17.1). Thus, the difference between LE and DFLE and the resulting years with limitations shows a disadvantage in Chinese females (17.3 years) compared to White Scottish females (13.7 years). This disadvantage in Chinese females seen in results at 50 years did not particularly appear from the estimates at birth. This aligns with the lack of health advantage found in older Chinese females while younger Chinese females are more likely to report better health than the reference population (figure 4.5).

In summary, the findings highlight a clear contrast between the LE advantage and the corresponding HLE and DFLE patterns in the Pakistani and Indian populations. The number of years lived in poor health and with limitations were greater in females than males. If considered with ethnicity, results were concerning in the Indian and Pakistani populations and particularly so in females. The findings showed the worse outcome in Pakistani Females (20.4 years lived in poor health, 30.7 years with limitations) in contrast with White Scottish females (8.7 years lived in poor health, 17.5 years with limitations).

Figure 4.6. Number of years in poor health or with limitations by ethnicity and sex



4.4. Discussion and conclusion

4.4.1. Summary of findings

In this chapter, ethnic differences in health expectancies and self-assessed health in Scotland were identified in both directions. Compared to the White Scottish group, an advantage was observed in males and females of Other White British, Other White and Chinese origins as well as in Indian males while Any Mixed Background and Pakistani males and females had a clear health disadvantage. Potential underlying mechanisms of the health differences observed were investigated. Findings supported socio-economic deprivation as an explanatory factor of ethnic health differences between the white populations of Scotland. Taking account of their favourable SES profile, the Indian population had a reported health disadvantage compared to the White Scottish population. However, SES adjustment had little effect on the ethnic patterns observed in other non-white minority ethnic groups relative to the majority population. Overall, being born outside the UK rather than in the UK was protective of worse reported health in most ethnic groups. These findings supported the healthy migrant effect hypothesis as well as theories of acculturation and health convergence in descendants. However, regardless of being born in or outside the UK, the Pakistani population reported worse health outcomes compared to the White Scottish population born in the UK, with the Pakistani population born outside the UK having the worst reported health profile. This empirical evidence in the Pakistani population challenges the healthy migrant effect as underlying mechanism of ethnic differences in health. Findings also showed that the reported health advantage observed in many younger ethnic minority adults, was either smaller or unapparent in older ethnic minority adults when compared to their respective White Scottish counterparts. It supported a reduction of ethnic differences in SAH in older ages for most ethnic groups. However, increased SAH differences and disadvantage were observed in older Pakistani males and females and Indian females.

Finally, findings highlighted that ethnic patterns of health expectancies did not necessarily match the previously published life expectancy patterns by ethnicity in Scotland (Gruer et al., 2016). For example, Pakistani females had the longest LE at birth but also the shortest HLE and DFLE at birth. When discrepancies between life expectancy and health expectancies were quantified through the number of years lived in bad health or with disability,

a clear disadvantage was revealed in Indian and Pakistani populations and especially in females, living longer but in poorer health.

4.4.2. Findings in relation to current evidence

This chapter has shown ethnic differences in HE in Scotland. Previously, HE figures have been produced in Scotland by sex, Scottish council areas, health boards and area-level deprivation (Scottish Government, 2019). However, monitoring progress in tackling health inequalities requires the investigation of various dimensions of social inequalities. No measure of healthy life expectancy by disability, ethnicity, origin, religion or individual socio-economic circumstances is available in the Scottish context. Hence, this study is the first to provide HLE and DFLE estimates by ethnicity in Scotland and contributes to bridging the gap in quantifying health inequalities between social subgroups in Scotland.

Furthermore, a systematic literature review of studies investigating HE inequalities in older ages found that HE measures were used to compare the health of different subpopulations using variables such as gender, social class, race/ethnicity and health behaviours but evidence related to the study of HE differences by ethnic or race groups came uniquely from the US context (Pongiglione et al., 2015). In addition to studies identified in the review, a few recent European studies have explored HE by ethnic or migrant groups (Carnein et al., 2015, Reus-Pons et al., 2017, Wohland et al., 2015). Thus, evidence of ethnic inequalities in HE remains limited worldwide. Moreover, most of this body of research used the Sullivan method. This method requires cross-sectional data which can combine morbidity and mortality from different sources. Indeed, available studies of HE by ethnicity gathered data on morbidity and mortality from different data and population sources. Mortality data came from official life tables or death registry while health data came primarily from surveys in both the US and European studies.

In this study, death registry and census data were used to gather data on mortality and morbidity respectively but the individual linkage between the two data sources permitted the calculation of HE by ethnicity based on a unique population source to be obtained. Indeed, a major strength of this study is that it was based on the national sample size of SHELS linking reported morbidity to mortality data at an individual level. Therefore, the calculation of health expectancy by ethnicity was based on a direct method, gathering data from the same cohort of individuals and at a national level. Analysis was done on 10 ethnic groups in Scotland with a fine ethnic granularity. However, due to low number of deaths, smaller ethnic groups were

aggregated into Other South Asian and African origin groups. No difference in HE were found for these aggregated ethnic groups in comparison to the White Scottish population, potentially hiding divergent HE patterns for their subpopulations.

This chapter has also shown ethnic differences in SAH in Scotland in 2001, in line with the HE results. The SAH results bolster previous findings published in official Scottish reports (Scottish Government, 2004, Scottish Government, 2015) and confirmed higher risks of reporting bad health or LLTI in the Pakistani populations and lower risks in the Chinese populations compared to the White Scottish groups. Despite different reference populations and contexts, the Scottish results also echoed the CoDE briefing by Becares which found that the Chinese group reported better health with half of the illness rates of the White British population and that the Pakistani group reported higher LLTI rates compared to the majority population in England and Wales in 2001 (Becares, 2013). Scottish findings contrast with the international literature which depicts minority ethnic groups as generally more likely to report poorer health than the majority population (Bombak and Bruce, 2012). Indeed, the picture in Scotland was different with only Any Mixed Background and Pakistani populations significantly reporting worse health than the majority White Scottish population. Clear patterns of advantageous reported health were demonstrated in Other White British, Other White and Chinese populations in Scotland. Different contexts might entail different exposures and population structure leading to various patterns of inequalities. As previously stressed in chapter 2, ethnic differences in health within a country are also driven by the health status of the majority population they are compared to and that of the White Scottish majority in Scotland might play a key role in the inequality patterns observed.

In the Scottish context, lower proportions of high educational attainment were observed in the White Scottish group which highlights a SES disadvantage in comparison to other ethnic groups (see section 3.3.5). This educational disadvantage in the White Scottish population of Scotland could be the result of the contrast with educational level due to educational selection in migrants and to the willingness to achieve good educational attainment in their descendants. The findings of this study aimed to account for the SES disadvantage faced by minority ethnic groups in order to understand their poorer health. However, in this context, adjusting ethnic differences in health for SES can account for the SES disadvantage in both majority and minority alike and helped us to understand whether a health disadvantage in the White Scottish population can be explained by lower SES. For example, the Other White British

population showed a better SES profile than the White Scottish population and adjusting for SES, using three SES measures, fully accounted for the SAH differences observed between the Other White British and White Scottish groups. In line with previous studies in England and Wales using various combinations of SES measures (Darlington et al., 2015, Evandrou, 2000a, Evandrou et al., 2016, Harding and Balarajan, 2000, Mindell et al., 2014), this finding in the white groups, persistent across different SAH outcomes and in stratified analysis, supported the hypothesis that ethnic health inequalities can be explained by SES.

Nevertheless, the contribution of SES in explaining differences observed in non-white minority ethnic groups was not clear cut. In the Indian population, adjusting for SES showed a previously unobserved SAH disadvantage. This phenomenon was also found in a study of ethnic differences in SAH using the Health Survey for England (Darlington et al., 2015). The authors referred to an 'ethnic penalty' in the Indian population i.e. penalisation due to their ethnicity which might determine their poorer health beyond the benefits of their favourable SES profile. Furthermore, SES had little effect on reducing the observed SAH disadvantage in the Pakistani population, contrasting with findings from England and Wales (Mindell et al., 2014). SES adjustment also failed to explain the SAH advantage identified in the Chinese population. In Scotland, there were indications that SES failed to account for ethnic inequalities in various health outcomes (Bhala et al., 2016, Bhopal et al., 2015, Cezard et al., 2015, Fischbacher et al., 2014, Sheikh et al., 2016, Simpson et al., 2015) even when multiple measures of SES were used as a more comprehensive SES proxy (Fischbacher et al., 2014). In other studies based on SHELS, adjusting for SES in the analysis of ethnic differences in health mostly reduced the differences observed between the Other White British group and the White Scottish majority but appeared to lead to variable to no effect in the health risk of other ethnic groups in comparison to the White Scottish majority (Cezard et al., 2015, Fischbacher et al., 2014, Simpson et al., 2015) which is in line with the findings of this chapter.

Various reasons can be proposed for why SES fails to account for ethnic differences in health. First, the SES proxies used to capture the SES disadvantage experienced by the diverse ethnic groups of Scotland might not be reliable. This study used a limited number of SES proxies. As hinted by Nazroo (Nazroo, 2001), it is possible that the SES proxies accounted for in this research cannot fully capture the deprivation status and social disadvantage faced by ethnic minorities. Hence, adjustment for SES in this analysis might not account for the full social deprivation picture between ethnic groups. Another possible explanation of the inability for SES

adjustment to explain ethnic health inequalities might lie in that analyses account for SES measures in a cross-sectional way while experience of deprivation and social disadvantage occurs over the life course. There is a need to understand the accumulative process of deprivation embodied in minority and majority ethnic groups from early life into older ages that shapes their health condition later in life. However, accessing this type of data poses issues as it involves potentially cross-country and life-long follow-up of individuals. The feasibility of collecting data on experiences and potential processes occurring over time in various minority ethnic groups composed of multiple migrant cohorts and their descendants is also challenging. Finally, the possibility that SES might not be such a strong determinant of ethnic inequalities in health in Scotland could lie in the different SES profiles of its ethnic majority and minorities in comparison to similar ethnic counterparts in England and Wales (Walsh, 2017). In conclusion, SES is unlikely to provide the whole answer to ethnic differences in health in Scotland, especially in relation to non-white minority ethnic groups. Hence, there is a need to acknowledge the contribution of other mechanisms as drivers of ethnic differences in health in Scotland.

The findings of this chapter, disentangling the reported health of minority ethnic groups by whether born in the UK or not, provided a new empirical insight into the Scottish ethnic health patterns. Of note was the higher risk of reporting worse health in the Pakistani populations, both those who were born in and outside the UK. These results align with previous findings of persistent SAH disadvantage across generations in the Pakistani population in England and Wales (Harding and Balarajan, 2000, Smith et al., 2009). In addition, accounting for SES did not explain the intergenerational continuities of disadvantage in Pakistani migrants and descendants. Particularly striking was the finding of the worst reported health in Pakistani migrants, beyond the reported health level of their descendants. This challenges the role of the healthy migrant effect as the underlying mechanism of ethnic health inequalities. Alternatively, it could also question the meaning of SAH in this particular population, how their reporting of health might match their actual health status and whether the way health is reported might be influenced differently in migrants and descendants.

In line with previous research on ethnic differences in health in older ages in the UK (Becares, 2013, Evandrou, 2000a, Evandrou et al., 2016), higher risks of poor health were found in older Pakistani males and females and Indian females in comparison to the majority population in Scotland. However, in contrast to the CoDE briefing by Becares (Becares, 2013), there was no Chinese advantage in SAH in older adults in Scotland. While findings about the

Pakistani population support a widening of health inequalities in older ages, findings in the Chinese population and other ethnic groups (who mostly reported a health advantage in younger ages) tended to support the opposite phenomenon i.e. a reduction of inequalities in older adults. Alternatively, the same findings could also be interpreted as a worsening of health in all minority ethnic groups in older ages compared to the majority, either losing their health advantage or increasing their health disadvantage as they get older.

A phenomenon of reduction of health inequalities in older ages is plausible if we assume that a survival selection operates, there could be a convergence of health inequalities toward the same average level of health for all ethnic groups in older ages. However, why this would operate differently in South Asian populations in particular would need to be elucidated. One argument could be linked to greater exposure to discrimination and deprivation over the life course in this particular ethnic group. It could also be argued that migration selection plays differentially for certain ethnic groups, age groups and migrant cohorts. For example, reasons for migration to Scotland in younger Chinese adults such as studying or working might be different from the reasons for migration at the time when older Chinese populations migrated to Scotland and thus, might explain why the younger Chinese adult population showed a health advantage while the older Chinese population do not. Indeed, older Chinese come from an immigration stream in the 1950s who originated in Hong Kong and who primarily worked in Chinese restaurants and takeaways (Song, 2015, Young and George, 1993). However, many of their children entered higher education and moved up the social ladder into mainstream professionals jobs (Song, 2015). The younger generation consists as well of students from mainland China, some of whom return to China or take up opportunities in other countries (Shen, 2005). Finally, deprivation and life experiences prior migration as well as longer exposure to discrimination in the destination country in older adults might also play a role in determining their health in later life differentially than younger adults of the same ethnic group.

In summary, the availability of individual level data has permitted further exploration of health inequalities and its determinants. The use of risk ratios provided a fine understanding of the magnitude of the differences between groups. With an exceptional national level sample size in Scotland, the analysis provided insight into inequalities with a strong granularity of ethnicity and some of the ethnic-specific underlying processes which reinforces the need to avoid amalgamating ethnic groups. Indeed, the determinants of ethnic inequalities in health explored in this chapter showed ethnic-specific patterns which suggests that further

mechanisms contribute to the observed differences. However, information on other potential mechanisms, such as discrimination or health behaviours, was not available in the SHELS data to further disentangle explanations. The number of years in poor health in the Pakistani population could also be linked to genetic problems. For example, the Born in Bradford study showed higher levels of consanguineous marriages in the Pakistani community, which is linked to genetic risk of congenital anomaly in children (Bhopal et al., 2013). However, this should be balanced with other protective effects of consanguineous relationship on health (Bhopal et al., 2013).

Finally, findings supports a morbidity-mortality paradox in the Pakistani and Indian populations in Scotland who lived longer but relatively shorter healthy life and consequently had more years in an unhealthy state. This is the first time that evidence of a morbidity-mortality discrepancy phenomenon in particular ethnic groups is demonstrated at the country level, based on a unique population source. To gauge further the extent of the phenomenon, recent evidence of ethnic differences in mortality in Scotland showed a mortality advantage in Pakistani and Indian males and females which persisted after adjustment for SES (Bhopal et al., 2018) and this chapter also demonstrated a SAH disadvantage in those same ethnic groups once SES was accounted for, using the same SES proxies. In addition, Bhopal et al. also showed a mortality advantage in the Pakistani populations who were both born in and outside the UK (Bhopal et al., 2018) while the findings of this chapter highlighted worse reported health in the same Pakistani populations who were born in and outside the UK. This points to a morbidity-mortality paradox which goes beyond SES adjustment and in both Pakistani migrants and their descendants. Previous research on a morbidity-mortality gap in specific ethnic groups gathered evidence from different sources and population samples or were based on small sample size (Deboosere and Gadeyne, 2005, Khlal and Guillot, 2017, Kouris-Blazos, 2002, Stanaway et al., 2018). Whether the morbidity-mortality paradox phenomenon in ethnic minorities is real can be questioned the same way as it has been in relation to the morbidity-survival paradox observed in women compared to men (Arber and Cooper, 1999, Case and Paxson, 2005, Idler and Benyamini, 1997, Kulminski et al., 2008, Oksuzyan et al., 2018, Oksuzyan et al., 2008, Rieker and Bird, 2005, Van Oyen et al., 2013, Verbrugge, 1982).

The observed mortality advantage in most ethnic groups in Scotland could be due to a data artefact, the effect of selective moves on health (e.g. unhealthy return migration) and unrecorded emigration and death. For example, migrants, when getting older and sick, could

decide to migrate back to their country of origin to finish their lives 'at home' with their relatives. This unhealthy return migration phenomenon, often referred to as the "salmon bias", if combined with unrecorded emigrations and deaths, could bias mortality estimates by producing a mortality advantage where there is none. However, a salmon bias hypothesis seems unlikely in the Pakistani and Indian populations in Scotland. In the SHELS cohort, around 60% of the Pakistani and half of the Indian populations were born in the UK, highlighting the well-settled nature of these populations. Furthermore, these ethnic groups have a strong sense of national belonging in the UK. Indeed, more than half of people from ethnic minorities in the UK describe their national identity as some form of UK identity with the highest proportions seen in South Asian and Black Caribbean populations (up to 84% in the Pakistani population) (Jivraj and Simpson, 2015). National Health Service in Scotland also offers health care services that are 'free at the point of use'. Hence, if South Asians living in Scotland are well-settled and can access free health care services when becoming ill, they are likely to stay to benefit from the health care they need. If most of their family and descendants are also settled in the UK, it would reduce even more their likelihood of returning to their country of origin in older ages. Little is known about the prevalence of the salmon bias in the UK and its contribution to the observed mortality advantage in ethnic minorities. One recent study showed that there was an unhealthy return migration phenomenon for specific ethnic groups in the UK but this phenomenon was not strong enough to explain the mortality advantage observed in South Asian populations in the UK (Wallace and Kulu, 2018).

The reliability of the reported morbidity data can be challenged. One could argue that there can be language barriers or cultural differences in the reporting and meaning of health. In that case, we could assume that SAH as a subjective measure of health might not accurately reflect the objective health status of minority ethnic groups. Hence, this reporting bias could result in biased morbidity estimates and possibly lower-than-expected HE estimates. Based on the Health Survey for England, Chandola and Jenkinson showed that worse reported health was associated with greater morbidity in all ethnic groups and for a range of more objective measures of health (Chandola and Jenkinson, 2000). Their analysis also supported no differential association between reported health and more objective morbidity across ethnic groups. Their findings supported a strong and consistent association between subjective morbidity and more objective morbidity across ethnic groups. However, on account of the present findings, further research is warranted to support claims of the consistency of the subjective-objective health link between ethnic groups in the Scottish context.

If we assume that a salmon bias cannot explain the mortality advantage in the Pakistani and Indian populations in Scotland and that their reported morbidity reflects their objective health status, we could argue that there is a real morbidity-mortality paradox (i.e. living longer but in poorer health) in these populations. Higher morbidity in certain groups could be due to differences in access and quality of care. Although, evidence from the Health Survey for England does not suggest unequal access to GP services for minority ethnic groups (Nazroo et al., 2009), available evidence both in England and Scotland suggests a more complex picture of potential unequal access operating at different level of healthcare and healthcare settings (Katikireddi et al., 2018, McFarland et al., 1989, Nazroo et al., 2009, Worth et al., 2009). Morbidity inequalities are less likely to be solely due to an ethnic differential in health services engagement. Nevertheless, this is an area for policy to consider.

To explain the morbidity-mortality contrast, the sex morbidity-mortality paradox literature offers additional alternatives. Some groups could suffer from specific conditions that contribute to reporting higher morbidity as well as have lower risk of mortality (Case and Paxson, 2005). In the case of the Pakistani population in the UK, they have a particular disease profile including higher risk of metabolic syndrome related diseases such as diabetes, renal disease and cardiovascular disease (Bansal et al., 2013, Bhopal et al., 2011, Dreyer et al., 2009, Forouhi et al., 2006, Hull et al., 2011, Sproston and Mindell, 2006), higher risk of respiratory disease such as asthma (Sheikh et al., 2016) and lower risk of cancer (Bhopal et al., 2012b) in comparison to their white counterparts. This disease profile (particularly their higher risk of CVD) does not fully fit with the expected set that contributes to higher morbidity and lower mortality. However, an emerging literature in the UK (see section 2.2.3) shows that these populations, once diagnosed with either diabetes, renal disease or CVD survive longer than their white counterparts diagnosed with the same disease (Bansal et al., 2013, Davis et al., 2014, Mathur et al., 2018). In relation to the sex morbidity-survival paradox, Oksuzyan and colleagues highlight that potential mechanisms can be complex and multifactorial including biological, social and psychological (Oksuzyan et al., 2018, Oksuzyan et al., 2008). Understanding mechanisms will require further research along these avenues.

4.4.3. Conclusion and opportunities for future research

This chapter has shown strong ethnic differences in SAH and health expectancies, contrasted with mortality. Ethnic differences in SES explained the differences in SAH observed between white groups but contributed little to the patterns observed in non-white minority

groups. It is an important message for policy makers that there is space for improvement in the White Scottish population socio-economic circumstances and improving their socio-economic conditions might reduce the health gap observed between white ethnic groups in Scotland. Alternatively, there might also be a brain drain effect in the White Scottish population of Scotland whereby well-educated White Scottish people leave Scotland for better employment opportunities elsewhere. Hence, the Scottish Government should ensure the development of strategies to keep skilled and well-educated White Scottish individuals in Scotland. The reported health disadvantage in Indian population once SES was accounted for is also concerning and requires further investigation for underlying processes. However, findings showed that SES factors are not the unique explanation to ethnic differences in SAH in Scotland and further mechanisms need to be investigated. The analysis looking into the reported health of ethnic groups who were born in or outside the UK pinpointed a particular disadvantage in the Pakistani population, for both migrants and their descendants. Furthermore, a SAH disadvantage was also shown to be stronger in older ages in the Pakistani population as well as in Indian females. Although age is not a modifiable risk factors, exploring the differential patterns experienced by various age cohort is essential in order to target prevention and develop the right support for each generation. Finally, a morbidity-mortality discrepancy was observed in the Pakistani and Indian populations with Pakistani females showing the worst outcomes in terms of years in unhealthy state. Additionally, in older populations, the emergent disadvantage in terms of years in unhealthy state in Chinese females should not be disregarded.

Policy and future research should pay particular attention to the health and experiences of the most vulnerable groups such as Pakistani and Indian populations and especially older women. After accounting for SES and UK-birth, there were persistent SAH inequalities in Scotland. This highlights the need to further research mechanisms in order to disentangle the explanations of remaining inequalities in different groups. Whether worse health behaviour, discrimination, accumulated deprivation, higher allostatic load or other processes underlie the higher risk of reporting poor health in the well settled Pakistani population of Scotland need to be unravelled, especially so in the older age groups as this finding contrast with a their previously reported advantage in mortality in Scotland (Gruer et al., 2016). This chapter showed a morbidity-mortality contrast in the Pakistani population of Scotland which goes beyond SES and migrant generations. This contrast needs to be understood to better address the health need of Scotland diverse population. Testing the meaning of subjective health across ethnic groups, the reliability of mortality data and the contribution of health selection hypothesis would be a useful

first step to strengthen the evidence of the morbidity-mortality paradox in Scotland. To understand and characterise the paradox further, other research avenues include the investigation of the disease profile and the trajectory of health of Pakistani people.

In the next chapters, the contrast between reported health and mortality is further explored. Despite findings of this chapter being based on the same SHELS sample, it is possible that those who report poor health and those who are in age bands with higher risks of dying might relate to different people/cohorts. The Pakistani population remain a younger population in Scotland. Issues of potential time lag between the time of reported health and subsequent mortality also need to be addressed. Chapter 5 links SAH to mortality at the individual level and addresses the need to determine whether SAH relates to mortality similarly across ethnic groups. Chapter 6 also provides further pieces of the puzzle in relation to the morbidity-mortality paradox by looking into ethnic differences in more objective measures of health i.e. multimorbidity based on hospitalisation data.

CHAPTER 5

5. Ethnic differences in the association between self-assessed health and mortality in Scotland

5.1. Background and research questions

Chapter 4 explored ethnic differences in self-assessed health in Scotland in 2001 and compared the observed patterns to the mortality evidence based on individuals of the same SHELS cohort in Scotland. An evident discrepancy in outcome between SAH and mortality patterns emerged for particular ethnic groups in Scotland. Internationally, a limited number of studies have gathered evidence showing a morbidity-mortality contrast in specific ethnic groups (De Grande et al., 2014, Deboosere and Gadeyne, 2005, Khlata and Guillot, 2017, Kouris-Blazos, 2002, Stanaway et al., 2018, Vang et al., 2017). In these studies, the observed mortality patterns and morbidity patterns were mostly gathered from different sources and did not necessarily refer to the same individuals. Chapter 4 strengthened the evidence of a morbidity-mortality contrast in the Pakistani population in Scotland by analysing morbidity and mortality from a unique population source with a national level sample size.

However, even based on a single population source, a time lag and cohort effect might drive the observed morbidity-mortality discrepancy and should not be ruled out. A time lag effect might occur if a morbidity disadvantage observed in a particular population is reflected in related mortality a few decades later. Hence, the contrasting morbidity-mortality patterns can be the results of the cross-sectional study of different cohorts of migrants or minority ethnic groups. In Scotland, the reported morbidity disadvantage in the Pakistani population presented in chapter 4 might indeed depict a phenomenon driven by a specific cohort not yet in age of high mortality risk. In contrast, the separate analysis of mortality (Bhopal et al., 2018) showing an advantage in the Pakistani population might reflect the patterns of older and healthier cohorts of migrants. Hence, the morbidity disadvantage and the mortality advantage seen in the Pakistani population might reflect the health experience of separate sets of people. These are significant issues that need to be addressed in order to confirm a morbidity-mortality paradox. However, due to separate population sources, previous research was unable to explore these considerations. A first step to assess whether the morbidity-mortality contrast occurs in the same people is to link the morbidity and mortality data for each individual and examine how morbidity relates to mortality in each group.

In this chapter, SAH is used as an indicator of general morbidity and is linked to mortality at the individual level to investigate the SAH-mortality relationship in each ethnic group. SAH and mortality are brought together rather than analysed separately or from different sources. By looking at the SAH-mortality association, this chapter engages with another strand of the literature on the validation of SAH as a reliable measure of health in population subgroups and the relationship of SAH with more objective measures of health.

The research questions addressed in this chapter are as follows:

- 1- Does SAH predict subsequent mortality for each ethnic group in Scotland?
- 2- Is the SAH-mortality association consistent across ethnic groups in Scotland? In other words, does ethnicity mediate the SAH-mortality association?
- 3- Can socio-economic status differences explain any ethnic differential in the SAH-mortality association?
- 4- To what extent does the observed ethnic differential in the SAH-mortality association prevail in both migrants (born outside the UK) and their descendants (born in the UK)?

The use of SAH measures is widespread due to their ease of collection, accessibility and validity in estimating the health of populations. As specified in chapter 2, there is a strong body of literature validating SAH as a reliable measure of health. This validation was done by showing that SAH is strongly associated with more objective measures of health such as measures of physical and mental health, physician rating of health, health care usage and mortality (Cohen et al., 1995, DeSalvo et al., 2006, Idler and Benyamini, 1997, Idler and Kasl, 1995, Larue et al., 1979, Miilunpalo et al., 1997, Mossey and Shapiro, 1982, Wannamethee and Shaper, 1991). Despite consistent findings in general populations, whether SAH predicts or is associated with other measures of health equally well across specific subpopulations is debated. For example, initial work explored whether sex could mediate the relationship between self-reported health and mortality. Findings were inconsistent as some showed no sex differences in this association (Jylhä et al., 1998, Singh-Manoux et al., 2007b) while others showed evidence of greater predictiveness of SRH for mortality risk in men compared to women (Assari, 2016, Deeg and Kriegsman, 2003, DeSalvo et al., 2006) or in women compared to men (Benyamini et al., 2003). Similarly, conflicting findings were found according to different SES groups (Burström and Fredlund, 2001, Dowd and Zajacova, 2007, Franks et al., 2003, Huisman et al., 2007, McFadden et al., 2009, Singh-Manoux et al., 2007a).

In relation to ethnicity, evidence of the validity of SAH as a measure of health and the consistency of its association with other measures of health is limited. The research available primarily focuses on the self-reported health indicator. In relation to the SAH-mortality relationship by ethnicity, most of the available literature come from the US. Initial research by McGee et al. showed that SRH was consistently associated with mortality in each of the 5 ethnic groups investigated (McGee et al., 1999). By analysing the SAH-mortality relationship for each ethnic group in Scotland, this chapter provides further evidence of the predictiveness of SAH for mortality for a wide range of ethnic groups in the Scottish context. However, looking at the SAH-mortality for each ethnic group does not inform on whether SAH predicts mortality similarly across ethnic groups.

The US study by McGee et al. showed a stronger SRH-mortality association in White populations than in other ethnic groups (McGee et al., 1999) but the investigators did not explore this ethnic differential further. The ethnic differential in SRH-mortality association has been formally investigated in the US using interaction analysis (between SRH and ethnicity in predicting mortality) only in more recent years. Two studies showed a stronger SRH-mortality association in White than in Black populations in the US (Assari et al., 2016, Woo and Zajacova, 2016). However, the meaning of ethnicity and health vary across countries making problematic the transfer of the US findings to the Scottish context and further investigation is warranted.

In the UK, a key contribution on the relationship of SAH with other measures of health was made by Chandola and Jenkinson (Chandola and Jenkinson, 2000). They showed that SRH was consistently associated with objective measures of morbidity (hypertension, stroke, heart disease, diabetes, GP visits, limiting health) and health service use across ethnic groups in the UK. As the authors could not detect differences in the association between SRH and their chosen objective health measures across ethnic groups (confidence intervals overlapped), they claimed SRH to be a valid measure of health across ethnic groups. However, their analysis was based on small samples for minority groups and had wide confidence intervals pointing to a potential type II error where the absence of significant differences does not prove there are none. Hence, there is a need to further research the differential association of SAH with other measures of health across ethnic groups in the UK.

Assessing whether the SAH-mortality relationship is similar across ethnic groups, based on a national sample of the population in Scotland and using interaction analysis, will make a

unique contribution in the UK context and will permit us to gauge the mediating role of ethnicity in the SAH-mortality relationship. If the ethnic morbidity-mortality paradox as reported in chapter 4 is real, we can hypothesize that an ethnic differential in the SAH-mortality relationship will occur in groups which appear to live longer while reporting poorer health. In that case, we expect the SAH-mortality association based on individual level linkage to differ in the Pakistani and Indian populations in comparison to the White Scottish population.

As explained earlier, evidence of the association between SAH and subsequent mortality has been explored in relation to different subgroups such as by sex (Assari, 2016, Benyamini et al., 2003, Deeg and Kriegsman, 2003, DeSalvo et al., 2006, Jylhä et al., 1998, Singh-Manoux et al., 2008, Singh-Manoux et al., 2007b) and by SES (Burström and Fredlund, 2001, Dowd and Zajacova, 2007, Franks et al., 2003, Huisman et al., 2007, McFadden et al., 2009, Singh-Manoux et al., 2007a). Evidence of whether SAH relates to mortality similarly for different SES groups is conflicting. Using a range of SES indicators such as education, occupation and income, a UK study supported SRH as a stronger predictor of mortality in those with a lower socio-economic profile and a weaker SRH-mortality relationship in the most socio-economically advantaged (Singh-Manoux et al., 2007a). In contrast other studies pointed to a stronger SRH-mortality association in those with the most favourable socio-economic status (Burström and Fredlund, 2001, Dowd and Zajacova, 2007, Franks et al., 2003) or to no differential association (McFadden et al., 2009). If there is a differential SAH-mortality association by SES and if ethnic groups experience different level of SES, the ethnic differential observed in the SAH-mortality association could be explained by SES differences between ethnic groups. This chapter determines whether SES mediates the SAH-mortality relationship and whether it can explain any ethnic differential in the SAH-mortality association.

Finally, this thesis is interested in disentangling ethnic differences with the additional lens of whether ethnic minorities were born in or outside the UK in order to identify the differential experience of migrants and their descendants. A study analysing the SRH-mortality association in Latinos living in the US found that SRH was strongly associated with mortality in those US-born but this association was weak in recent migrants (less than 10 years) (Finch et al., 2002). These findings supported that the SAH-mortality association holds weakly in recent migrants and that greater acculturation underlies greater strength of the SAH-mortality association. In migrants, a healthy migrant effect might be protective of death but not necessarily of minor ailments. Minor health issues would lead to reporting poorer health but not

necessarily lead to higher risk of death. This could explain a smaller explanatory power of SAH for mortality in migrants. In descendants, acculturation processes could underlie a convergence in the predictiveness of SRH for mortality towards the degree of predictiveness seen in the majority population. These acculturation processes could operate at many levels. For example, the strength of the SAH-mortality association could be influenced by converging health behaviours and health profile of descendants towards that of the majority population but also by cultural differences in the meaning and reporting of SAH across migrant generations. Hence, the strength of the SAH-mortality association is expected to be similar in ethnic groups who were born in the UK compared to the majority population.

5.2. Data and methods

As explained in chapter 3, SHELS is particularly fit to analyse the relationship between SAH and other objective measures of health by ethnicity. Additionally, SHELS provides a unique data source to analyse interactions with exceptional sample size in the Scottish context. In this chapter, the SHELS census-mortality linked data is used to investigate the association between SAH as reported in the Scottish census 2001, and subsequent mortality (May 2001-April 2013).

The exploration of the association between subjective health and mortality across ethnic groups was based on three indicators of health. All-cause mortality over 12 years was used as the key outcome of this chapter in the form of time to death. SAH measures (SRH and LLTI) as reported in the Scottish census 2001 were used as measures of subjective morbidity and included in separate models as binary variables. As previously dichotomised in chapter 4, self-reported health was categorised as reporting bad health (1) versus reporting good or fair health (0) and LLTI as reporting at least one limiting long term illness (1) versus none (0).

Ethnicity has been described earlier in chapter 2 (concept) and chapter 3 (operationalisation). In this chapter, ethnicity followed the same categorisation as in the SAH analysis by ethnicity in chapter 4. The following 13 ethnic categories were used: White Scottish, White Irish, Other White British, Other White, Any Mixed Background, Indian, Pakistani, Bangladeshi, Other South Asian, Caribbean, African, Black Scottish/Other and Chinese.

The literature points to the role of SES as a potential confounder in the differential SAH-mortality association by ethnicity. The body of evidence on whether SAH predicts mortality similarly across varied socio-economic groups provided conflicting results but overall found a

differential SAH-mortality association by socio-economic groups with either a weaker or stronger SAH-mortality association among individuals of lower social class. Similarly and consistently with chapter 4, this chapter used three SES proxies: SIMD, household tenure and the combined individual and household measure of highest qualification.

An initial analysis reported in appendix 5.1 showed a significant interaction of each SES indicator with SAH in predicting subsequent mortality. Our findings in the Scottish context supports the evidence of a significant SAH-mortality differential association between individuals of different socio-economic status. The interaction terms showed a weaker SAH-mortality association in those with lower SES compared to those with higher SES. A differential SAH-mortality association by ethnic group could then be the results of underlying SES differences by ethnic group. Hence, this initial analysis by SES group presented in appendices justifies adjusting for the three SES indicators in the core analysis of this chapter in order to explore whether the potential ethnic variations in the SAH-mortality association can be explained by underlying SES differences between ethnic groups.

The literature, as well as chapter 4 in relation to the Scottish context, has shown evidence of varied health status between migrants and their descendants in both SAH and mortality. Using the same categorisation as in chapter 4, UK-birth was used to assess whether it could explain some of the ethnic differential in the subjective health-mortality association. Analyses were also stratified by a combined ethnicity and UK-birth variable to pinpoint whether the effect could be identified in both ethnic groups who were born outside the UK and those who were born in the UK in comparison with the majority White Scottish population who was born in the UK.

To analyse whether SAH can predict subsequent mortality (SAH-Mortality association) for each ethnic group and similarly across ethnic groups, Hazard Ratios and their 95% confidence intervals were calculated using Cox regression survival analysis. Mortality data were available over 12 years and the associated death date available in month and year was used to derive time to death in months. The population at risk was the 4.62 million people who responded to the Scottish census 2001 in the SHELS cohort and analysis was censored for death, emigration records acquired from the NHS data or the end of the period of interest (April 2013). As explained in chapter 3, Cox models assume that the effect of the covariates included in the model on the hazard (mortality risk here) is the same over time. Proportional hazard

assumptions were checked graphically for each of the covariates by inspecting visually whether the cumulative hazards were parallel in the complimentary log log plots (appendix 3.2). Additionally, sensitivity analyses were done using mortality over a 5 years period instead of 12 years. This resulted in smaller number of deaths in each ethnic group and less robust findings for smaller ethnic minority groups.

The SAH-mortality association analyses used both SRH and LLTI as measure of SAH to predict mortality in separate models. All analyses were stratified by sex and adjusted for age. The first type of analysis explored the SAH-mortality association for each ethnic group so one HR of SAH predicting time to death is presented for each model (one model per SAH indicator, per ethnic group and per sex). The second type of analysis included SAH, ethnicity and their interaction ethnicity*SAH into the model predicting time to death. This interaction analysis allows us to distinguish any ethnic differential in the SAH-mortality association and whether SAH predicts mortality similarly, more strongly or more weakly for specific ethnic groups. The best health outcome was taken as reference for each SAH variable in these interaction analyses to facilitate interpretation. Due to the greater prevalence of reporting a LLTI rather than bad health, analyses including an ethnicity*SAH interaction term primarily focused in the LLTI-mortality association for which estimates are expected to be more robust. However, the SRH-mortality association results are available in appendices and discussed when relevant. The addition of each interaction term into the model including SAH and ethnicity was tested using likelihood ratio tests. To explore the contribution of SES in explaining the potential ethnic differential in SAH-mortality association, models were additionally adjusted for SES. Finally, in a last set of age-adjusted models, the differential association was also explored once ethnicity was broken down into those who were born in the UK and those who were born outside the UK.

5.3. Results

5.3.1. SAH-mortality association for each ethnic group

Figure 5.1 (and table 5.1) shows the age-adjusted risk of death (HRs and 95% CIs) of SRH (bad health versus good/fair health) or LLTI (LLTI versus none) stratified by ethnic group and sex. Each estimate in the figure represents one model using one SAH measure as predictor, for one ethnic group and one sex. The top panels show the risk of subsequent death (2001-2013) associated with reporting bad health versus reporting good/fair health in 2001. Results for Bangladeshi females were not shown due to disclosure issues when interacting SRH (bad health)

with mortality events. The bottom panels show the risk of subsequent death (2001-2013) associated with reporting at least one LLTI versus none in 2001. The reference line shows the mortality risk for those who reported either good/fair health or no LLTI for each ethnic and sex group.

The SRH-mortality association results showed that reporting bad health in 2001 versus good/fair health was associated with respectively a 2.5 and 2.3 higher HRs of subsequent mortality in White Scottish males and females. The HRs of reporting bad health gravitated around 2.1-2.6 in white ethnic males and females showing a consistent higher risk of death in those reporting poorer health in each white ethnic group. Findings showed more variability in non-white minority ethnic groups with wide confidence intervals particularly in groups with small sample size. HRs were closer to the reference value and non-significant for Any Mixed Background males, Caribbean males and females and Black Scottish or Other Black males and females. In other words, there was no significant SRH-mortality association for these particular ethnic groups.

Results on the LLTI-mortality association showed similar high risks of mortality in White Scottish males and females (HRs around 2.3 in males and 2.2 in females) when reporting at least one LLTI versus none. In white ethnic groups, HRs ranged from 2.0 to 2.5 showing a certain consistency in how LLTI predicts higher mortality risk in white groups. In the Pakistani and Indian populations, reporting at least one LLTI versus none appeared more strongly associated with a higher risk of mortality (HRs of 3.1-3.2 in males and 3.6-3.8 in females). In contrast, a non-significant LLTI-mortality association appeared in Any Mixed Background males, Caribbean females and Black Scottish or Other Black females. HRs were closer to 1 (reference), showing clearly that LLTI predicts mortality weakly in these ethnic groups. No significant LLTI-mortality association was found in Bangladeshi males and females but these groups had small sample size and large CIs which could explain non-significant results.

A sensitivity analysis of the SAH-mortality association for each ethnic group used 5 years of mortality data rather than 12 years (appendix 5.2). Results provided similar patterns in white, Indian, Pakistani and Chinese populations and tended to be disclosive or to produce wide CIs for other minority ethnic groups. The sensitivity analysis using 5 years of mortality data showed a different pattern in Any Mixed Background males: the SAH-mortality association for this group was significant and in similar range to that of the white groups.

In summary, results supported that reporting poor health or reporting a LLTI predicts a higher risk of mortality in ethnic minority subgroups. There was however some exceptions in smaller ethnic minority groups where the predictiveness of SRH and LLTI for the risk of subsequent mortality was weaker and non-significant. Results specific to the LLTI-mortality association showed a strong ability of LLTI in predicting mortality for Pakistani and Indian populations, particularly in females.

Figure 5.1. Age-adjusted HRs (mortality risk) and 95% CIs of SRH (bad health versus good/fair health) and LLTI (LLTI versus no) for each ethnic group and sex

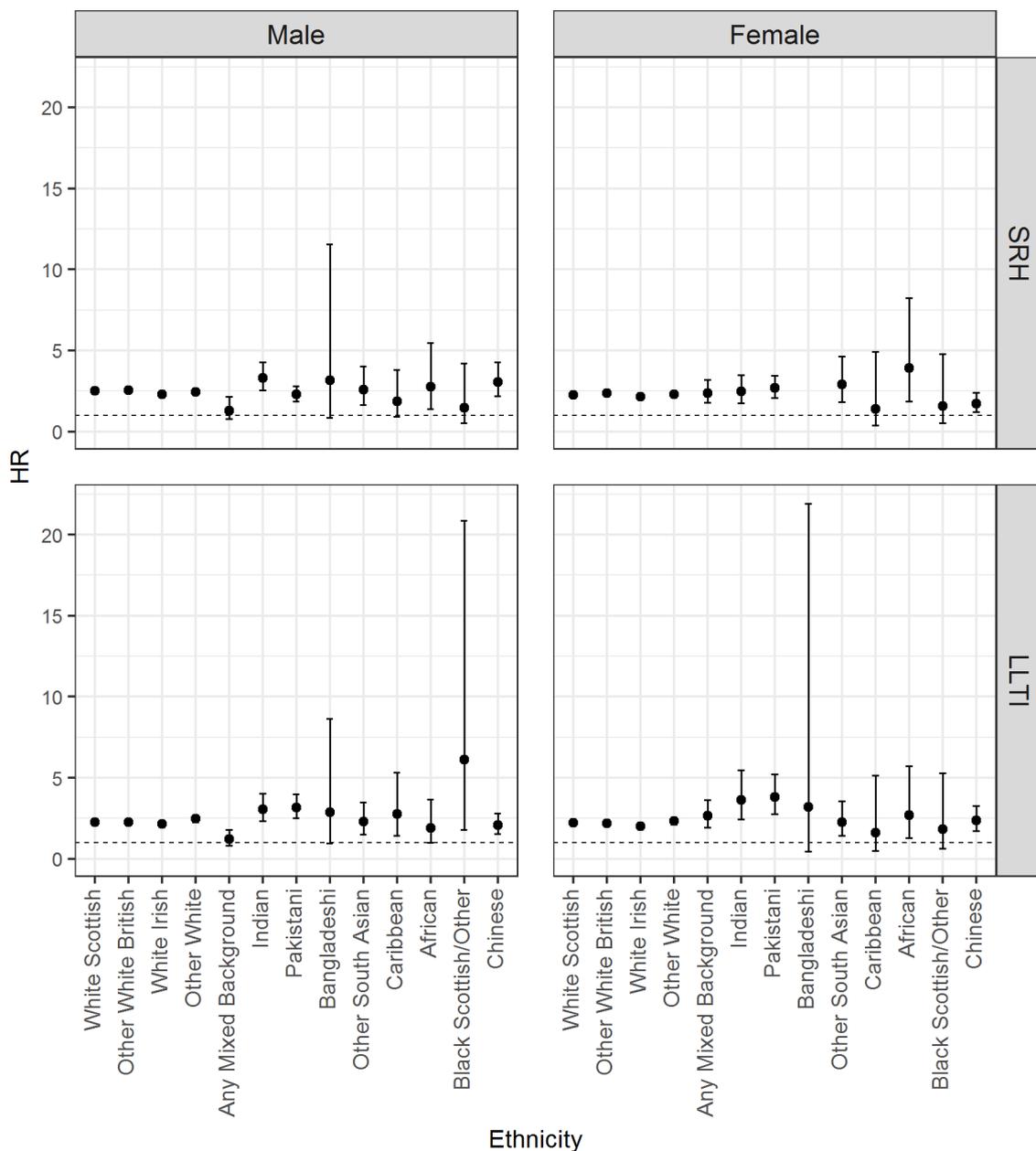


Table 5.1. HRs (mortality risk) and 95% CI of reporting Bad health or a LLTI in 2001 stratified by ethnicity and by sex. Models are adjusted for age at baseline

Sex and ethnic groups	People in 2001	Deaths 2001-13	SRH (<i>Bad health versus Good/Fair</i>)			LLTI (<i>ref=no LLTI</i>)		
			Reported Bad health and died	HR (95% CI)	p-value	Reported LLTI and died	HR (95% CI)	p-value
MALES								
White Scottish	1949480	251755	77025	2.51 (2.49, 2.53)	<0.0001	147610	2.27 (2.25, 2.29)	<0.0001
Other White British	160235	17855	4945	2.56 (2.48, 2.65)	<0.0001	10180	2.25 (2.18, 2.32)	<0.0001
White Irish	20340	3420	1245	2.31 (2.15, 2.48)	<0.0001	2180	2.16 (2.01, 2.32)	<0.0001
Other White	29945	2785	975	2.45 (2.26, 2.65)	<0.0001	1765	2.47 (2.27, 2.69)	<0.0001
Any Mixed Background	5310	240	75	1.29 (0.78, 2.15)	0.3272	140	1.22 (0.83, 1.79)	0.3077
Indian	6450	280	110	3.29 (2.54, 4.28)	<0.0001	170	3.07 (2.34, 4.04)	<0.0001
Pakistani	12930	450	190	2.28 (1.86, 2.80)	<0.0001	305	3.17 (2.53, 3.99)	<0.0001
Bangladeshi	860	25	10	3.17 (0.87, 11.6)	0.0797	15	2.89 (0.97, 8.65)	0.0578
Other South Asian	2685	125	40	2.57 (1.63, 4.04)	<0.0001	65	2.29 (1.51, 3.48)	0.0001
Caribbean	710	55	15	1.86 (0.91, 3.81)	0.0893	30	2.77 (1.44, 5.34)	0.0023
African	2105	65	15	2.75 (1.38, 5.48)	0.0040	20	1.92 (1.00, 3.67)	0.0495
Black Scottish or Other Black	460	40	10	1.48 (0.52, 4.22)	0.4615	25	6.11 (1.79, 20.9)	0.0038
Chinese	6530	215	55	3.05 (2.17, 4.28)	<0.0001	105	2.07 (1.53, 2.81)	<0.0001
FEMALES								
White Scottish	2138640	285440	94120	2.26 (2.24, 2.28)	<0.0001	185730	2.21 (2.19, 2.23)	<0.0001
Other White British	174750	19310	6125	2.36 (2.28, 2.43)	<0.0001	12530	2.19 (2.12, 2.26)	<0.0001
White Irish	23160	3895	1530	2.14 (2.01, 2.29)	<0.0001	2680	2.01 (1.87, 2.16)	<0.0001
Other White	35710	2670	940	2.31 (2.13, 2.51)	<0.0001	1750	2.33 (2.13, 2.54)	<0.0001
Any Mixed Background	5800	230	80	2.38 (1.78, 3.19)	<0.0001	150	2.66 (1.95, 3.63)	<0.0001
Indian	5890	175	85	2.49 (1.77, 3.48)	<0.0001	135	3.64 (2.43, 5.46)	<0.0001
Pakistani	12700	295	165	2.68 (2.08, 3.46)	<0.0001	225	3.81 (2.78, 5.22)	<0.0001
Bangladeshi	705	15	.			10	3.19 (0.47, 21.9)	0.2373
Other South Asian	2255	120	40	2.90 (1.81, 4.65)	<0.0001	75	2.27 (1.44, 3.56)	0.0004
Caribbean	775	35	10	1.38 (0.39, 4.91)	0.6197	25	1.63 (0.51, 5.16)	0.4072
African	1800	45	15	3.92 (1.86, 8.25)	0.0003	20	2.71 (1.29, 5.72)	0.0086
Black Scottish or Other Black	480	40	15	1.57 (0.52, 4.80)	0.4266	25	1.83 (0.63, 5.31)	0.2685
Chinese	6670	210	55	1.72 (1.23, 2.39)	0.0015	125	2.37 (1.73, 3.26)	<0.0001

Table 5.2. HR (mortality risk) and 95% CI by ethnicity, stratified by sex. Models are adjusted for age at baseline, and subsequently for LLTI (No versus Yes), the interaction between LLTI and ethnicity and SES

a) Males

	Deaths 2001-13	People 2001	Model 1		Model 2		Model 3		Model 4	
			Adjusted for age		Adjusted for age and LLTI		Adjusted for age, LLTI and ethnicity*LLTI(0)		Adjusted for age, LLTI, ethnicity*LLTI(0) and SES	
			HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value
White Scottish	251755	1949480	1		1		1		1	
Other White British	17855	160235	0.76 (0.75, 0.77)	<.0001	0.78 (0.77, 0.80)	<.0001	0.81 (0.79, 0.82)	<.0001	0.90 (0.87, 0.92)	<.0001
White Irish	3420	20340	1.01 (0.98, 1.05)	0.5353	0.98 (0.95, 1.01)	0.2067	0.97 (0.93, 1.02)	0.2206	0.91 (0.87, 0.95)	0.0001
Other White	2785	29945	0.82 (0.79, 0.86)	<.0001	0.83 (0.80, 0.86)	<.0001	0.88 (0.84, 0.92)	<.0001	0.93 (0.88, 0.99)	0.0157
Any Mixed Background	240	5310	1.16 (1.02, 1.31)	0.0240	1.13 (1.00, 1.28)	0.0541	1.20 (1.01, 1.41)	0.0340	1.12 (0.93, 1.35)	0.2194
Indian	280	6450	0.63 (0.56, 0.71)	<.0001	0.62 (0.55, 0.70)	<.0001	0.69 (0.59, 0.80)	<.0001	0.84 (0.72, 0.98)	0.0305
Pakistani	450	12930	0.68 (0.62, 0.75)	<.0001	0.62 (0.56, 0.68)	<.0001	0.69 (0.62, 0.77)	<.0001	0.73 (0.66, 0.82)	0.0000
Bangladeshi	25	860	0.55 (0.36, 0.82)	0.0038	0.55 (0.37, 0.83)	0.0045	0.65 (0.38, 1.13)	0.1258	0.66 (0.37, 1.16)	0.1494
Other South Asian	125	2685	0.94 (0.79, 1.12)	0.5093	0.90 (0.76, 1.07)	0.2423	0.89 (0.70, 1.13)	0.3201	0.92 (0.71, 1.19)	0.5151
Caribbean	55	710	0.93 (0.72, 1.21)	0.6109	0.95 (0.73, 1.23)	0.6902	0.98 (0.69, 1.39)	0.9030	1.06 (0.72, 1.55)	0.7812
African	65	2105	0.94 (0.74, 1.20)	0.6294	0.99 (0.77, 1.27)	0.9288	0.91 (0.58, 1.43)	0.6849	0.83 (0.53, 1.31)	0.4259
Black Scottish or Other Black	40	460	0.94 (0.69, 1.29)	0.7231	0.97 (0.71, 1.33)	0.8536	1.01 (0.67, 1.52)	0.9533	0.77 (0.46, 1.31)	0.3372
Chinese	215	6530	0.56 (0.49, 0.64)	<.0001	0.57 (0.49, 0.65)	<.0001	0.57 (0.47, 0.69)	<.0001	0.54 (0.44, 0.67)	<.0001
LLTI (No versus Yes)					0.44 (0.44, 0.44)	<.0001	0.44 (0.44, 0.45)	<.0001	0.47 (0.47, 0.48)	<.0001
Other White British * LLTI							0.94 (0.91, 0.96)	<.0001	0.92 (0.88, 0.95)	<.0001
White Irish * LLTI							1.01 (0.95, 1.09)	0.6953	1.05 (0.97, 1.14)	0.2136
Other White * LLTI							0.86 (0.79, 0.93)	0.0001	0.79 (0.72, 0.87)	0.0000
Any Mixed Background * LLTI							0.88 (0.68, 1.14)	0.3273	0.89 (0.67, 1.18)	0.4103
Indian * LLTI							0.80 (0.63, 1.01)	0.0621	0.74 (0.58, 0.95)	0.0194

Pakistani * LLTI	0.74 (0.60, 0.90)	0.0026	0.75 (0.61, 0.92)	0.0048
Bangladeshi * LLTI	0.70 (0.31, 1.60)	0.4009	0.76 (0.33, 1.76)	0.5210
Other South Asian * LLTI	1.04 (0.73, 1.47)	0.8355	0.99 (0.69, 1.43)	0.9505
Caribbean * LLTI	0.93 (0.55, 1.58)	0.7955	0.85 (0.48, 1.50)	0.5671
African * LLTI	1.12 (0.66, 1.92)	0.6713	1.16 (0.68, 2.01)	0.5847
Black Scottish/Other Black * LLTI	0.91 (0.48, 1.71)	0.7609	0.96 (0.43, 2.16)	0.9233
Chinese * LLTI	0.98 (0.75, 1.28)	0.8601	1.07 (0.80, 1.42)	0.6464
SIMD (1 vs 5-least deprived)			1.34 (1.32, 1.36)	<.0001
SIMD (2 vs 5-least deprived)			1.23 (1.21, 1.25)	<.0001
SIMD (3 vs 5-least deprived)			1.16 (1.14, 1.18)	<.0001
SIMD (4 vs 5-least deprived)			1.06 (1.04, 1.07)	<.0001
Household tenure (own vs. rent)			1.54 (1.53, 1.56)	<.0001
Highest qualification (higher vs. no)			0.86 (0.85, 0.87)	<.0001
Highest qualification (lower vs. no)			0.92 (0.91, 0.93)	<.0001

b) Females

	Deaths 2001-13	People 2001	Model 1		Model 2		Model 3		Model 4	
			Adjusted for age		Adjusted for age and LLTI		Adjusted for age, LLTI and ethnicity*LLTI(0)		Adjusted for age, LLTI, ethnicity*LLTI(0) and SES	
			HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value
White Scottish	285440	2138640	1		1		1		1	
Other White British	19310	174750	0.80 (0.79, 0.81)	<.0001	0.81 (0.80, 0.82)	<.0001	0.83 (0.81, 0.84)	<.0001	0.88 (0.86, 0.90)	<.0001
White Irish	3895	23160	0.92 (0.89, 0.95)	<.0001	0.89 (0.86, 0.92)	<.0001	0.88 (0.85, 0.91)	<.0001	0.83 (0.79, 0.87)	<.0001
Other White	2670	35710	0.79 (0.76, 0.82)	<.0001	0.79 (0.76, 0.82)	<.0001	0.82 (0.78, 0.86)	<.0001	0.86 (0.81, 0.92)	<.0001
Any Mixed Background	230	5800	0.95 (0.83, 1.08)	0.4249	0.93 (0.82, 1.06)	0.2623	0.98 (0.84, 1.15)	0.8125	0.85 (0.69, 1.04)	0.1081
Indian	175	5890	0.65 (0.56, 0.76)	<.0001	0.59 (0.51, 0.68)	<.0001	0.66 (0.56, 0.78)	<.0001	0.69 (0.57, 0.83)	0.0001
Pakistani	295	12700	0.78 (0.69, 0.87)	<.0001	0.66 (0.59, 0.74)	<.0001	0.72 (0.63, 0.82)	<.0001	0.75 (0.65, 0.85)	<.0001
Bangladeshi	15	705	0.87 (0.54, 1.41)	0.5790	0.85 (0.53, 1.37)	0.5152	0.82 (0.43, 1.57)	0.5474	0.59 (0.27, 1.31)	0.1964
Other South Asian	120	2255	1.08 (0.90, 1.29)	0.4044	1.05 (0.87, 1.25)	0.6140	1.03 (0.82, 1.29)	0.8087	1.08 (0.81, 1.45)	0.5844
Caribbean	35	775	0.71 (0.52, 0.98)	0.0389	0.76 (0.55, 1.05)	0.0929	0.90 (0.60, 1.36)	0.6229	0.69 (0.38, 1.25)	0.2184
African	45	1800	1.13 (0.85, 1.50)	0.4061	1.16 (0.87, 1.54)	0.3179	1.23 (0.81, 1.87)	0.3306	1.01 (0.64, 1.61)	0.9599
Black Scottish or Other Black	40	480	1.11 (0.82, 1.51)	0.4908	1.02 (0.75, 1.38)	0.9211	0.88 (0.60, 1.29)	0.5150	0.74 (0.44, 1.22)	0.2360
Chinese	210	6670	0.69 (0.60, 0.78)	<.0001	0.68 (0.59, 0.77)	<.0001	0.71 (0.60, 0.85)	0.0001	0.73 (0.59, 0.89)	0.0016
LLTI (No versus Yes)					0.45 (0.45, 0.46)	<.0001	0.45 (0.45, 0.46)	<.0001	0.44 (0.44, 0.45)	<.0001
Other White British * LLTI							0.95 (0.92, 0.98)	0.0004	0.93 (0.89, 0.96)	0.0001
White Irish * LLTI							1.05 (0.98, 1.12)	0.1908	1.05 (0.96, 1.14)	0.2730
Other White * LLTI							0.90 (0.84, 0.98)	0.0146	0.90 (0.81, 0.99)	0.0292
Any Mixed Background * LLTI							0.86 (0.65, 1.13)	0.2723	0.91 (0.64, 1.28)	0.5765
Indian * LLTI							0.65 (0.46, 0.93)	0.0168	0.74 (0.52, 1.06)	0.0999
Pakistani * LLTI							0.73 (0.56, 0.95)	0.0198	0.77 (0.59, 1.00)	0.0476
Bangladeshi * LLTI							1.10 (0.42, 2.84)	0.8506	1.41 (0.47, 4.18)	0.5402
Other South Asian * LLTI							1.05 (0.72, 1.53)	0.7957	0.84 (0.53, 1.33)	0.4575

Caribbean * LLTI	0.67 (0.34, 1.29)	0.2289	1.00 (0.45, 2.24)	0.9920
African * LLTI	0.89 (0.50, 1.58)	0.6936	1.08 (0.58, 2.00)	0.8144
Black Scottish/Other Black * LLTI	1.58 (0.83, 2.97)	0.1610	1.28 (0.56, 2.93)	0.5568
Chinese * LLTI	0.89 (0.68, 1.17)	0.4141	0.93 (0.69, 1.25)	0.6132
SIMD (1 vs 5-least deprived)			1.18 (1.17, 1.20)	<.0001
SIMD (2 vs 5-least deprived)			1.13 (1.11, 1.15)	<.0001
SIMD (3 vs 5-least deprived)			1.11 (1.09, 1.12)	<.0001
SIMD (4 vs 5-least deprived)			1.06 (1.04, 1.07)	<.0001
Household tenure (own vs. rent)			1.54 (1.53, 1.56)	<.0001
Highest qualification (higher vs. no)			0.88 (0.87, 0.89)	<.0001
Highest qualification (lower vs. no)			0.89 (0.88, 0.90)	<.0001

5.3.2. Ethnic differential in the SAH-mortality association

As explained in chapter 4, there is a higher prevalence of reporting a LLTI than there is of reporting bad health. Using LLTI rather than SRH provides an analysis relying on a greater numerator when looking at the intersection between SAH and mortality and is preferred in interaction analysis. Hence, the results presented in this section focus on the LLTI-mortality relationship but the SRH-mortality association results are made available in appendices 5.3a&b and its specific findings, divergent from the LLTI-mortality association results, highlighted.

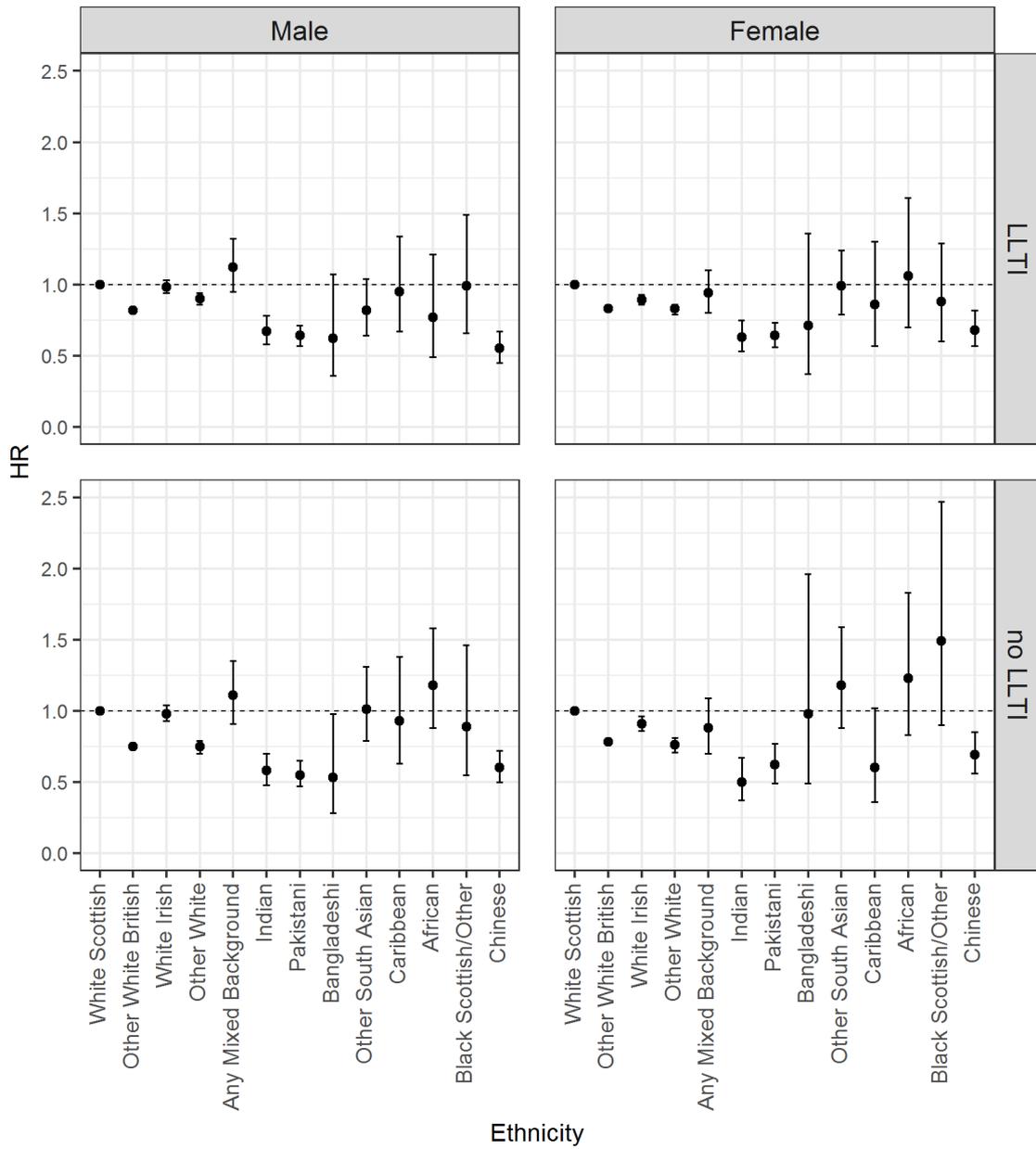
Tables 5.2a&b show the age-adjusted risk of death by ethnic group for males and females (model 1). The White Scottish population was taken as reference (HR=1). Additional adjustment were made step by step in subsequent models: model 2 additionally adjusted for LLTI, model 3 for the interaction between LLTI and ethnicity and model 4 for SES.

In model 1, results showed an age-adjusted mortality advantage (HR<1) in most ethnic minority groups compared to the White Scottish majority in both males and females. The mortality advantage was statistically significant (confidence interval did not include 1) in Other White British, Other White, Indian, Pakistani and Chinese males and females as well as Bangladeshi males, White Irish females and Caribbean females compared to their White Scottish counterparts. This was in line with previous findings by Bhopal et al. based on the same SHELS cohort and looking at ethnic differences in mortality using Poisson regression (Bhopal et al., 2018). However, our findings using survival analysis showed a higher risk of death in Any Mixed Background males which was not detected in previously published analysis using Poisson regression (Bhopal et al., 2018).

Adjusting for LLTI in model 2 did not change the mortality advantage observed in most minority ethnic groups in the age-adjusted model. For Any Mixed Background males, their mortality disadvantage was reduced to non-significant level when LLTI was accounted for. The HRs of reporting no LLTI versus at least one LLTI was low (HRs [95% CI] of 0.44 [0.44; 0.44] in males and 0.45 [0.45; 0.46] in females) supporting the expected relationship that reporting no health issue or limitation is associated with better mortality outcome. Similar findings were found with SRH adjustment (appendix 5.3). However, adjustment for SRH had no attenuation effect on the higher risk of death observed in Any Mixed Background males.

Figure 5.2 shows the age-adjusted risk of mortality by ethnicity stratified by LLTI and sex. For the ethnic groups reporting at least one LLTI in 2001 (top panels), a mortality advantage was evident in most minority ethnic groups and significant in Other White British, Other White, Indian, Pakistani and Chinese groups as well as White Irish females compared to the White Scottish population. In those reporting no LLTI in 2001, the 12-year mortality risk was also significantly lower in Other White British, Other White, Indian, Pakistani and Chinese groups as well as White Irish females but patterns were more varied and with larger CIs in smaller minority ethnic groups. In summary, the general patterns of a mortality advantage in most minority ethnic groups in Scotland remain consistent in the largest ethnic groups and regardless of reporting a LLTI or not.

Figure 5.2. Age-adjusted HRs (mortality risk) and 95% CIs by ethnicity, stratified by sex and LLTI (LLTI versus no)



This section is particularly interested in the interaction term of ethnicity*LLTI included in model 3. The addition of the interaction term in the model had little effect on the estimates related to ethnicity and related to LLTI. Model 3 shows a mortality advantage in most ethnic minority groups and a mortality disadvantage in Any Mixed Background males. Reporting no LLTI in 2001 remained protective of a higher risk of mortality in the next 12 years. However, findings showed a significant ethnicity*LLTI interaction term in predicting mortality for specific ethnic groups: Other White British, Other White and Pakistani males and females as well as Indian females. Tables 5.2a&b show that these particular ethnic groups had a mortality advantage compared to the White Scottish population and that reporting no LLTI is associated with lower risk of mortality. For these groups with a significant interaction term in predicting mortality, findings indicated that reporting no LLTI was associated with an even lower risk of mortality compared to the White Scottish population. In other words, results showed a stronger LLTI-mortality association in males and females of Other White British, Other White and Pakistani origins and females of Indian origins compared to the White Scottish population. In contrast, findings using SRH to investigate the SAH-mortality association (appendices 5.3a&b) showed significant interaction terms for the Other White British population only and additionally for the White Irish males in the opposite direction.

To assess the overall significance of the interaction terms, likelihood ratio tests were performed testing the addition of the interaction terms ethnicity*SAH into the models already including SAH and ethnicity. Significance was considered for a p-value below the 0.05 level. For the models including LLTI, the addition of the interaction term was significant for both males and females. However, for the models including SRH, the addition of the interaction term was significant for females and borderline non-significant for males (p-value = 0.0640). Overall, this supports an ethnic differential in the LLTI-mortality association and to a lesser extent in the SRH-mortality association.

The sensitivity analysis of the ethnic differential in the LLTI-mortality association using 5 years of mortality data found similar patterns to the analysis based on 12 years of mortality data (data not shown). Other White British, Other white, Indian, Pakistani and Chinese populations as well as Bangladeshi males and Irish females had a mortality advantage compared to the White Scottish population. A differential LLTI-mortality association was shown in the Other White British and Other White groups but this was no longer significant in the Indian and Pakistani groups when the death outcome was reduced to 5 years of data.

In Summary, there was a mortality advantage in most minority ethnic groups in Scotland compared to the White Scottish population. Additionally, reporting no LLTI versus at least one LLTI was predictive of a lower risk of mortality. For some ethnic groups, the interaction analysis showed a differential SAH-mortality association compared to the reference group. Males and females of Other White British, Other White, Pakistani origins and females of Indian origin had a stronger LLTI-mortality association whereby reporting no LLTI was more strongly associated with lower risk of mortality than in the majority population.

5.3.3. The role of SES in the ethnic SAH-mortality association differential observed

Tables 5.2a&b (model 4) show the interaction model further adjusted for SES. Adjustment for SES moderately reduced the mortality advantage observed in most groups except for the White Irish males and females and Chinese males for whom the risk of mortality changed to slightly lower values. The mortality advantage observed in specific groups remained significant and SES adjustment made overall little difference to the ethnic patterning of mortality.

The ethnicity*LLTI interaction in predicting mortality remained significant in Other White British, Other White and Pakistani males and females after SES adjustment. Similarly, in appendices 5.3a&b, the significant ethnicity*SRH interaction remained after SES adjustment in Other White British males and females and White Irish males. The effect of SES adjustment was apparent on the ethnicity*LLTI interaction term only for Indian populations. In females, the interaction with LLTI was non-significant when SES was accounted for despite the estimate still showing a lower risk of mortality in Indian females reporting no LLTI compared to the reference group. In males, the opposite was observed where a non-significant LLTI*mortality interaction term prior to SES adjustment was significant once SES was included in the model; it showed an even lower risk of mortality in Indian males who reported no LLTI compared to the reference population.

In Summary, SES adjustment reduced some of the mortality advantage observed in ethnic minority groups in Scotland. However, SES could not explain the ethnic differential in the LLTI-mortality association and a differential LLTI-mortality association remained in Other White British, Other White and Pakistani populations. SAH predicts mortality differently for these groups compared to the White Scottish population, regardless of their socio-economic circumstances.

5.3.4. SAH-mortality association by ethnicity and UK-birth

Table 5.3 shows the results of the interaction analysis between LLTI and a combined UK-birth-ethnicity variable in predicting subsequent mortality. It informs on whether the differential SAH-mortality association persists in ethnic groups who were born in the UK as well as those who were born outside the UK in comparison the White Scottish population who was born in the UK. The table presents one model for males and one model for females.

Results show significant interaction terms in the Other White British population who was born in the UK but the interaction terms were non-significant in those who were born outside the UK. Hence, the differential LLTI-mortality association holds in the Other White British populations who were born in but not outside the UK. In Indian females, a differential association was found for both those who were born in and outside the UK. A significant interaction term was also found in the Other White populations, for males who were born in the UK and for males and females who were born outside the UK, and in the Pakistani populations, for males and females who were born in the UK and for males who were born outside the UK. In Summary, the differential LLTI-mortality association holds in the Other White British population who was born in the UK and overall in Other White males, Pakistani males and Indian females regardless of migrant generations.

Table 5.3. HRs (mortality risk) and 95% CI by ethnicity and UK-birth, stratified by sex. Models are adjusted for age at baseline, LLTI and the interaction between LLTI and UK-birth/ethnicity

	Deaths 2001-13	People 2001	Adjusted for age, LLTI and UK-birth/ethnicity*LLTI(0)			
			males		females	
			HR (95% CI)	p-value	HR (95% CI)	p-value
Born inside the UK - White Scottish	249760	1931645	1		1	.
Born inside the UK - Other White British	17025	152775	0.81 (0.79, 0.82)	<.0001	0.83 (0.81, 0.84)	<.0001
Born inside the UK - White Irish	3390	20010	0.97 (0.93, 1.02)	0.2214	0.88 (0.85, 0.91)	<.0001
Born inside the UK - Other White	660	9145	0.96 (0.87, 1.05)	0.3452	0.86 (0.79, 0.95)	0.0021
Born inside the UK - Any Mixed Background	200	4035	1.26 (1.05, 1.52)	0.0126	1.09 (0.92, 1.29)	0.3059
Born inside the UK - Indian	40	3120	1.09 (0.69, 1.73)	0.7198	1.46 (0.97, 2.20)	0.0696
Born inside the UK - Pakistani	65	7505	1.18 (0.80, 1.74)	0.4117	1.54 (1.05, 2.24)	0.0256
Born inside the UK - Bangladeshi	15	370	1.22 (0.55, 2.72)	0.6250	1.54 (0.64, 3.70)	0.3329
Born inside the UK - Other South Asian	50	1010	1.03 (0.70, 1.52)	0.8655	1.19 (0.90, 1.58)	0.2306
Born inside the UK - Caribbean	20	425	1.07 (0.57, 1.99)	0.8338	1.08 (0.65, 1.79)	0.7634
Born inside the UK - African	20	545	1.40 (0.70, 2.81)	0.3377	1.37 (0.76, 2.47)	0.2984
Born inside the UK - Black Scottish or Other Black	25	340	1.10 (0.66, 1.83)	0.7115	1.02 (0.67, 1.56)	0.9094
Born inside the UK - Chinese	35	2505	0.76 (0.48, 1.21)	0.2535	1.22 (0.74, 2.03)	0.4340
Born outside the UK - White Scottish	2000	17835	0.84 (0.79, 0.89)	<.0001	0.79 (0.75, 0.83)	<.0001
Born outside the UK - Other White British	825	7460	0.82 (0.75, 0.89)	<.0001	0.78 (0.72, 0.85)	<.0001
Born outside the UK - White Irish	30	330	0.85 (0.55, 1.31)	0.4541	0.68 (0.42, 1.12)	0.1298
Born outside the UK - Other White	2125	20800	0.86 (0.81, 0.90)	<.0001	0.80 (0.76, 0.85)	<.0001
Born outside the UK - Any Mixed Background	40	1275	0.96 (0.65, 1.42)	0.8392	0.54 (0.33, 0.86)	0.0103
Born outside the UK - Indian	240	3325	0.66 (0.56, 0.77)	<.0001	0.59 (0.49, 0.71)	<.0001
Born outside the UK - Pakistani	385	5425	0.66 (0.59, 0.75)	<.0001	0.67 (0.58, 0.77)	<.0001
Born outside the UK - Bangladeshi	10	495	0.47 (0.22, 0.98)	0.0440	0.51 (0.19, 1.37)	0.1836

Born outside the UK - Other South Asian	75	1675	0.81 (0.60, 1.10)	0.1779	0.82 (0.56, 1.20)	0.3122
Born outside the UK - Caribbean	35	290	0.94 (0.61, 1.44)	0.7713	0.69 (0.34, 1.37)	0.2873
Born outside the UK - African	40	1560	0.72 (0.40, 1.31)	0.2857	1.11 (0.62, 2.01)	0.7198
Born outside the UK - Black Scottish or Other Black	10	120	0.88 (0.44, 1.75)	0.7111	0.49 (0.18, 1.31)	0.1561
Born outside the UK - Chinese	180	4025	0.54 (0.44, 0.67)	<.0001	0.67 (0.56, 0.81)	<.0001
LLTI			0.44 (0.44, 0.45)	<.0001	0.45 (0.45, 0.46)	<.0001
Born inside the UK - Other White British * LLTI			0.94 (0.91, 0.97)	<.0001	0.95 (0.92, 0.98)	0.0006
Born inside the UK - White Irish * LLTI			1.02 (0.95, 1.09)	0.5959	1.05 (0.98, 1.12)	0.2031
Born inside the UK - Other White * LLTI			0.76 (0.65, 0.90)	0.0010	1.00 (0.85, 1.17)	0.9996
Born inside the UK - Any Mixed Background * LLTI			1.03 (0.78, 1.36)	0.8429	0.85 (0.63, 1.15)	0.2907
Born inside the UK - Indian * LLTI			0.56 (0.30, 1.05)	0.0698	0.43 (0.22, 0.83)	0.0118
Born inside the UK - Pakistani * LLTI			0.46 (0.28, 0.76)	0.0024	0.38 (0.22, 0.66)	0.0005
Born inside the UK - Bangladeshi * LLTI			1.14 (0.38, 3.40)	0.8117	1.34 (0.41, 4.40)	0.6274
Born inside the UK - Other South Asian * LLTI			1.54 (0.89, 2.65)	0.1194	1.35 (0.85, 2.14)	0.2024
Born inside the UK - Caribbean * LLTI			0.93 (0.38, 2.29)	0.8765	1.00 (0.44, 2.28)	0.9934
Born inside the UK - African * LLTI			1.43 (0.60, 3.41)	0.4203	0.88 (0.32, 2.37)	0.7929
Born inside the UK - Black Scottish/Other Black * LLTI			0.84 (0.39, 1.79)	0.6484	1.54 (0.79, 3.01)	0.2056
Born inside the UK - Chinese * LLTI			0.85 (0.43, 1.67)	0.6360	0.89 (0.45, 1.76)	0.7353
Born outside the UK - White Scottish * LLTI			1.02 (0.93, 1.11)	0.7018	1.14 (1.05, 1.24)	0.0024
Born outside the UK - Other White British * LLTI			0.91 (0.80, 1.05)	0.2045	0.96 (0.84, 1.10)	0.5411
Born outside the UK - White Irish * LLTI			0.70 (0.34, 1.43)	0.3259	1.38 (0.64, 2.98)	0.4094
Born outside the UK - Other White * LLTI			0.89 (0.81, 0.97)	0.0083	0.88 (0.80, 0.97)	0.0068
Born outside the UK - Any Mixed Background * LLTI			0.54 (0.29, 1.02)	0.0558	1.22 (0.63, 2.34)	0.5589
Born outside the UK - Indian * LLTI			0.81 (0.62, 1.05)	0.1139	0.63 (0.42, 0.96)	0.0318
Born outside the UK - Pakistani * LLTI			0.74 (0.59, 0.93)	0.0102	0.75 (0.55, 1.02)	0.0711
Born outside the UK - Bangladeshi * LLTI			0.38 (0.10, 1.48)	0.1644	0.65 (0.12, 3.52)	0.6131
Born outside the UK - Other South Asian * LLTI			0.86 (0.54, 1.35)	0.5013	0.81 (0.43, 1.52)	0.5071
Born outside the UK - Caribbean * LLTI			0.93 (0.48, 1.78)	0.8189	0.49 (0.16, 1.49)	0.2056

Born outside the UK - African * LLTI	1.15 (0.58, 2.29)	0.6953	0.96 (0.46, 2.01)	0.9095
Born outside the UK - Black Scottish or Other Black * LLTI	1.02 (0.31, 3.39)	0.9726	1.04 (0.12, 9.26)	0.9751
Born outside the UK - Chinese * LLTI	1.00 (0.75, 1.35)	0.9784	0.85 (0.63, 1.15)	0.2962

5.4. Discussion and conclusion

5.4.1. Summary of findings

This chapter has shown a strong relationship between SAH (both SRH and LLTI) and subsequent mortality for most ethnic groups in Scotland. For a few smaller ethnic groups, reporting worse health was not significantly associated with a higher risk of mortality. Interaction analysis highlighted a differential LLTI-mortality association for the Other White British, Other White and Pakistani populations in comparison to the White Scottish population which was not explained by SES differences. Findings were less clear in the Indian populations for whom the LLTI-mortality association fluctuates with/without SES adjustment and differently for males and females. For these ethnic groups with differential associations, analysis by UK-birth showed that the differential LLTI-mortality association persisted in those who were born in the UK as well as for some of these groups who were born outside the UK. The SRH-mortality association results showed significant ethnicity*SRH interaction term for fewer ethnic groups than in the LLTI-mortality association analysis.

5.4.2. Findings in relation to current evidence

In line with McGee et al. (McGee et al., 1999), the results of this chapter showed SRH as a strong predictor of mortality risk in most ethnic groups in Scotland. While most research focuses on the self-rated health measure, the predictiveness of LLTI for the risk of mortality was also investigated in this chapter and provided further insight on a broader SAH-mortality association beyond the use of the usual SRH indicator. Findings showed a consistent relationship between SAH, both SRH and LLTI, in predicting the risk of mortality with similar range of strength in the majority White Scottish and other white groups. Results for non-white ethnic minority groups showed variable strength of the SAH-mortality relationship.

The significant interaction between SAH and specific ethnic groups in predicting mortality risk in Scotland supported ethnicity as a mediator of the SAH-mortality association. Although with a particular set of ethnic groups specific of the Scottish context, our finding echoes US studies which found a differential SRH-mortality association between white populations and other ethnic groups (Assari et al., 2016, Woo and Zajacova, 2016). No research apart from this chapter has explored the differential SAH-mortality association by ethnicity in the UK context. However one study approached our investigation. In Northern Ireland, O'Reilly

and Rosato explored the differential SRH-mortality association using denominational groups as a proxy for 'ancestry' rather than ethnicity. They found a differential SRH and mortality association with Protestants having a higher risk of mortality than Catholics for a given level of SRH. The greatest differences were observed between Catholics and Presbyterians, the latter taken as a marker of Scottish ancestry. Similarly, the findings of this chapter pointed to a greater predictiveness of SRH for mortality in the Other White British population compared to the White Scottish population.

Extending comparison of this chapter's findings to research using other measures of objective health, the only other study in the UK looking at a differential SRH-objective health association by ethnic group was by Chandola and Jenkinson (Chandola and Jenkinson, 2000). While they showed a consistent association between SRH and other objective measures of morbidity across ethnic groups, the present findings highlighted a differential SAH-mortality association for specific ethnic groups. Using SRH rather than LLTI highlighted fewer ethnic groups with a differential SAH-mortality association. This could be due to SRH predicting mortality more consistently across ethnic groups than LLTI; this would then be more in line with Chandola and Jenkinson findings. However, in comparison to the LLTI-mortality association, the more consistent SRH-mortality association across groups could also be the result of the smaller prevalence of bad health, reducing the ability of the interaction analysis with SRH to detect a significant interaction. If that is the case, these findings of an ethnic differential in the SAH-mortality association in Scotland could be considered as contrasting with earlier UK findings on the subjective-objective health association across ethnic groups. However, Chandola and Jenkinson used objective morbidity to validate SRH as a reliable measure of health whereas this chapter focuses on mortality. As shown in chapter 4, certain ethnic groups might live longer in poorer health compared to the majority population. In that case, self-assessed health might well predict a certain severity of morbidity in these groups without being a consistent predictor of mortality in comparison to the reference group. Specific ethnic groups could experience certain type of diseases which are reflected in their rating of health but are not necessarily particularly deadly.

In summary, in the context of a dearth of evidence on the ethnic differential in the SAH-mortality association in the UK context and internationally, this chapter makes a key contribution thanks to data linkage and a unique data source. The sample size of the SHELS data (4.6 million people) also brings evident strength to the study of interactions with ethnicity.

However, despite a considerable sample size, the number of deaths remained limited, even in a 12 year period, for some of the smallest ethnic groups and led to disclosure issues in some cases (number of events below 6).

Knowing about the limited evidence highlighting a differential SAH-mortality association across ethnic groups, research about its explanations is expectedly scarce (Assari et al., 2016, Woo and Zajacova, 2016). Woo and Zajacova looked at the contribution of SES and immigrant status in explaining this differential association (Woo and Zajacova, 2016). They found a slight attenuation of the ethnicity*SRH interaction terms in predicting mortality for non-Hispanic Blacks when three proxies of SES, such as education, poverty and employment, were adjusted for and for Hispanics when immigrant status - US-born, recent migrants (less than 10 years), migrants for more than 10 years - was accounted for. Despite this attenuation, the interaction terms remained significant and the differential SRH-mortality association by ethnic group remained unexplained. Similarly, in this chapter, SES had little effect in explaining the differential SAH-mortality association identified in specific ethnic groups. With acculturation, it was expected to find similar SAH-mortality association in minority ethnic groups who were born in the UK compared to the majority population of Scotland. However, the addition of the UK-birth dimension made little difference to the results of ethnic differential associations and did not provide support for this hypothesis. Hence, further mechanisms of the ethnic differential in the SAH-mortality association need to be explored.

Along the lines of investigation required to understand the morbidity-mortality paradox, one might find explanations for the SAH-mortality differential association across ethnic groups. For example, one could question the reliability of the mortality data collected. Indeed, if a salmon bias phenomenon and unrecorded death were to occur in the Pakistani population, this would create an artificial divergent SAH-mortality association for this group. However, a salmon bias and recording bias is very unlikely in the Other White British population who also showed a differential SAH-mortality association compared to the White Scottish population and for whom out-migration to the rest of the UK and death are considered reliable in the SHELS data.

The form and meaning of SAH has also been proposed as a potential reason for the SAH-mortality association differential. Previous research used SRH in different forms (nominal, dichotomous, continuous) to assess whether it would alter their significant findings but results of an ethnic differential in SRH-mortality association were consistent whichever form of SRH was

used (Assari et al., 2016). A common explanation put forward in relation to the SAH-mortality differential findings is linked to the subjective nature of SAH and to potential cultural differences in the assessment and reporting of health. Indeed, the ethnic differential association could arise from underlying cultural differences in the meaning and reporting of health for the ethnic groups concerned. However, it does remain difficult to test the direct influence of cultural differences on SAH and whether it affects the SAH relationship with mortality for different ethnic groups. Measures of cultural values were previously used to test their association with SRH in regard to both within- and between-country differences (Roudijk et al., 2017). A way forward could be to include cultural values in survey data with a large ethnic minority sample in order to understand the relationship between these cultural values and SAH across ethnic groups.

Finally, another explanation of the differential findings could be an actual morbidity-mortality paradox or at least differential morbidity and mortality patterns due to various combination of diseases more or less likely to lead to death. For example, Assari et al. looked at whether chronic conditions could mediate the SRH-mortality association in specific ethnic groups in the US. They found a persistent ethnic differential after adjustment for chronic conditions and particularly so as chronic conditions seemed to explain the SRH-mortality association in Blacks but not in Whites (Assari et al., 2016). Further research should consider the type of diseases driving the differential predictiveness of SAH for mortality in specific groups.

5.4.3. Conclusion and opportunities for future research

There are ethnic differences in the SAH-mortality relationship in Scotland. As expected, the Pakistani population had a significantly different SAH-mortality association compared to the White Scottish population. They were less likely to die at a given level of SAH compared to the White Scottish population. This is in line with the contrasting morbidity-mortality patterns highlighted in chapter 4 where the Pakistani population would live longer despite reporting poorer health. Their differential SAH-mortality association coupled with their contrasted SAH and mortality patterns compared to the majority White Scottish population support a morbidity-mortality paradox in the Pakistani population. By looking at how SAH relates to mortality across groups, the literature strand on SAH validation as a reliable measure of health can be informative for the morbidity-mortality discourse.

However, many avenues are still to be explored to understand why a discrepancy in outcome and differential association between SAH and mortality appears for certain ethnic

groups in Scotland. Following the identification of an ethnic differential in the SAH-mortality association, a few explanations were investigated. However, SES and UK-birth could not explain the ethnic variations in the predictive strength of SAH for mortality. Further research need to disentangle why SAH might predict mortality differently for specific ethnic groups. Future research avenues might gain from following the lines of investigation required to understand the morbidity-mortality paradox in the Pakistani population in Scotland.

As previously stressed, this study was based on a considerable sample size. Nevertheless, non-white minority ethnic groups accounted for about 2% of the population in Scotland in 2001 and the population of Scotland was estimated at 5.1 million people in 2001. As explained in chapter 1, the absolute size of Scotland ethnic minority groups has grown between 2001 and 2011. Ideally, this analysis should be replicated using the full Scottish census 2011 linked to mortality data. Data from other national settings with greater ethnic diversity could also help researchers to confirm the mediating effect of ethnicity on the SAH-mortality association in different contexts. This type of research should also bring additional opportunities to test the mechanisms involved in the ethnic differential SAH-mortality association.

In the meantime, a way to assess the cultural influence on the findings of this chapter would be to bring a more objective measure of morbidity into the picture. This would allow the exploration of whether health self-assessment reflects actual morbidity status across ethnic groups. Chapter 6 investigates ethnic differences in multimorbidity in Scotland which will provide indications on the likelihood of cultural differences in the reporting of health as well as additional clues on a morbidity-mortality paradox in the Pakistani population.

CHAPTER 6

6. Ethnic differences in multimorbidity and in the SAH-multimorbidity association in Scotland

6.1. Background and research questions

Chapter 4 identified discrepancies in outcomes between self-assessed health and mortality for specific ethnic groups in Scotland. For example, the Pakistani population experienced a mortality advantage and longer life expectancy while reporting poorer health and having a shorter healthy life expectancy compared to the White Scottish population. The morbidity-mortality contrast appeared in members of the Pakistani population who were born outside the UK and those who were born in the UK. SAH and mortality findings were derived from the same national-level population sample, the SHELS cohort. Although based on the same cohort, ethnic differences in SAH and mortality were based on separate analysis with patterns that could reflect phenomena disproportionately affecting different age cohorts. For example, a morbidity disadvantage could be affecting younger cohorts while a mortality advantage could be the result of healthier older cohorts. Hence, chapter 5 analysed the SAH-mortality contrast in greater depth by using linking SAH and mortality at the individual level. Chapter 5 findings confirmed a differential association between SAH and mortality for specific ethnic groups compared to the White Scottish majority. Reporting poorer health was associated with a higher risk of mortality in most ethnic groups in Scotland but not consistently across ethnic groups. This ethnic differential in the SAH-mortality association was seen in the Pakistani population for whom reporting no LLTI conferred an even lower risk of mortality compared to the White Scottish population reporting no LLTI. In summary, chapters 4 and 5 supported the claim that SAH does not relate to mortality to the same degree across ethnic groups and particularly so in the Pakistani population showing a morbidity disadvantage and a mortality advantage.

These findings could underlie a real morbidity-mortality paradox whereby certain groups live longer but in poorer health. However, SAH indicators are self-declared measures of morbidity and are, consequently, deemed subjective. The subjectiveness of SAH measures could explain why findings point to both contrasting morbidity-mortality patterns and a differential SAH-mortality association in the Pakistani population. It could be that there are cultural and ethnic differences in the meaning and reporting of health which do not reflect objective health (morbidity and mortality) similarly across ethnic groups. If we hypothesize that the Pakistani

population reports comparatively poorer health than other ethnic groups at the same level of objective morbidity, this could explain why the Pakistani population reports poorer health while living longer. This chapter uses a more objective measure of morbidity and thus, engages with this debate on whether previous findings arose due to a discrepancy between subjective health and objective health (and possible cultural differences in the meaning and reporting of health) or whether the differential patterns are due to differences in morbidity and mortality (morbidity-mortality paradox).

A measure of multimorbidity was created from the main clinical diagnosis of 12 years of hospitalisation data, reflecting a certain severity of objective morbidity. Using the same SHELS cohort as in previous chapters, ethnic differences in multimorbidity were assessed and potential mechanisms explored. Furthermore, whether subjective morbidity (SAH) relates to objective morbidity (multimorbidity) similarly across ethnic groups was investigated with the aim of assessing the existence of a morbidity-mortality paradox in the Pakistani population in Scotland.

The research questions addressed in this chapter are as follows:

- 1- What are the magnitude and direction of ethnic differences in multimorbidity based on individual level hospitalisation data?
- 2- To what extent can individual/neighbourhood socio-economic factors account for the ethnic differentials in multimorbidity in Scotland?
- 3- Do patterns of ethnic differences in multimorbidity differ by whether individuals were born in or outside the UK?
- 4- Which comorbidities underlie the observed ethnic differences in multimorbidity?
- 5- Is the SAH-multimorbidity association consistent across ethnic groups in Scotland? In other words, does ethnicity mediate the SAH-multimorbidity association?

Research in health inequalities should use more objective measures of health in complement to subjective health to characterise and understand inequalities. This thesis also researches health differences while keeping a holistic approach to health. A recent strand of health research advocates the need to assess health more globally and to go beyond the focus on a single disease (Barnett et al., 2012, Salisbury, 2012, Starfield, 2006). Therefore, multimorbidity based on clinical diagnosis was chosen as a more objective measure of health for this chapter. Indeed, the health of individuals tends to be determined by more than one disease, especially as we age (Barnett et al., 2012, Marengoni et al., 2011). For example, Barnett et al.

found that more than half of those aged 65 years and above were multimorbid (two or more morbidities) in a Scottish study using primary care data (Barnett et al., 2012). Furthermore, the authors showed that, in absolute numbers, more people were multimorbid below the age of 65 years than above that age, making the case that multimorbidity affects more than the elderly. In this context, multimorbidity has become the “norm rather than the exception” (Barnett et al., 2012, Salisbury, 2012) and UK projections show that it is expected to rise in the next 20 years particularly for complex multimorbidity (4+ diseases) (Kingston et al., 2018). Thus, there is a need to recognise the importance of the co-occurrence of diseases and explore its social determinants.

Multimorbidity captures a certain severity of morbidity which has been shown to be higher in older ages, women and those with higher levels of socio-economic deprivation (Barnett et al., 2012, Marengoni et al., 2011, Schiøtz et al., 2017). However, more evidence on the determinants of multimorbidity is required to better grasp which pathways are leading to the co-occurrence of diseases. This evidence on determinants would provide a better understanding of how to age well without chronic disease which would in turn lead to better global quality of life. Consequently, researching ethnic differences in multimorbidity can add value to available evidence by providing a more in-depth understanding of population health. So far, research into ethnic inequalities in multimorbidity remains scarce and is limited to a few US studies and a cardiovascular-related multimorbidity UK study (Johnson-Lawrence et al., 2017, Mathur et al., 2011, Quinones et al., 2017, Rocca et al., 2014).

The literature review in chapter 2 gathered evidence of ethnic differences in specific diseases in Scotland which was mostly based on the SHELS cohort. Overall, a Pakistani disadvantage for many specific diseases was found in comparison to the White Scottish population (Bansal et al., 2014, Bansal et al., 2013, Bhala et al., 2016, Bhopal et al., 2012a, Bhopal et al., 2015, Bhopal et al., 2011, Bhopal et al., 2014, Cezard et al., 2015, Sheikh et al., 2016, Simpson et al., 2015). Additionally, chapter 4 demonstrated that the Pakistani population is more likely to report poorer health than the White Scottish population. Therefore, it seems likely that this disadvantage will be reflected in a measure of multimorbidity based on hospitalisation data. We expect to find a higher risk of multimorbidity in the Pakistani population of Scotland in line with their reported morbidity disadvantage and disease-specific evidence. In other words, we hypothesize that the subjective health patterns observed in chapter 4 reflect objective morbidity status for this particular group.

Section 2.3.1 explained that poorer SES can lead to poorer health outcomes. Similarly, research in the social determinants of multimorbidity found that lower SES such as higher area deprivation or lower educational level increased the likelihood of multimorbidity (Barnett et al., 2012, Johnson-Lawrence et al., 2017, Marengoni et al., 2011, Schiøtz et al., 2017). As different ethnic groups have different socio-economic circumstances, SES could underlie ethnic differences in multimorbidity. Using multiple SES indicators, this chapter explores the contribution of SES in explaining ethnic differences in multimorbidity.

Furthermore, a few recent studies explored multimorbidity differences by migrant status (Diaz et al., 2015, Lenzi et al., 2016). Lower multimorbidity rates in migrants compared to native-born were found in Norway and in Italy. This goes along with the “healthy migrant effect” hypothesis whereby migrants tend to be selected and healthier than their peers left in the country of origin but also, in certain circumstances, healthier than the people they come to join in their destination country. In line with previous evidence, we expect a lower risk of multimorbidity in those who were born outside the UK rather than those who were born in the UK. This would match the overall effect of UK-birth on SAH found in chapter 4 where those who were born outside the UK were less likely to report poorer health. However, this UK-birth effect on the reporting of health did not hold true for all ethnic groups. For example, both the Pakistani individuals who were born outside the UK and those who were born in the UK reported worse health compared to the reference group, with those who were born outside the UK having the worst health profile. If subjective health reflects objective health, we might expect higher multimorbidity in members of the Pakistani population who were born outside the UK.

Finally, Chandola and Jenkinson showed that reported health was strongly associated with a range of more objective measures of health in the UK with no significant differences across ethnic groups (Chandola and Jenkinson, 2000). They found no significant interaction between ethnicity and reported health in their association with other measures of objective health. In contrast, chapter 5 showed that reported health was associated with mortality in each ethnic group in Scotland but not similarly across ethnic groups (significant interaction). If subjective morbidity reflects objective morbidity, we would expect as in previous evidence by Chandola and Jenkinson that there would be no significant interaction between ethnicity and SAH in predicting subsequent multimorbidity.

A higher risk of multimorbidity and the lack of a significant interaction between ethnicity and SAH in predicting multimorbidity, if observed in the Pakistani population of Scotland, would support a morbidity-mortality paradox in this particular ethnic group relative to the majority White Scottish population.

6.2. Data and methods

In chapters 4 and 5, the reported health variables available from the Scottish Census 2001 available in the SHELS data and its link to 12 years of mortality data were used. SHELS also provides a linked dataset between the Scottish census 2001 and the following 12 years of hospitalisation data. The hospitalisation data came from NHS National Services Scotland under the name of “SMR01- General Acute Inpatients/Day Cases”. Hospitalisation data in SMR01 generally provide 6 diagnosis codes for an admission/discharge which can be used to look at comorbidities. However, for sensitivity reasons and to ensure the privacy and the confidentiality of the data, the whole range of diagnosis available in SMR01 data for a 12 years period could not be shared with SHELS. Under the Privacy Advisory Committee of NHS National Services Scotland (PAC – number 36/13), hospitalisation data with the main (first) diagnosis only were provided for linkage to the Scottish census 2001 within the SHELS project. This chapter uses 12 years of hospitalisation data (May 2001-April 2013) and its recorded main diagnosis to identify multimorbidity. SMR01 records from 2001 to 2013 used the 10th revision of the International Classification of Diseases (ICD-10 codes).

As explained in chapter 3, there is no standard way to measure multimorbidity. Evidence points to multimorbidity being strongly associated with mortality regardless of its operationalisation (Nunes et al., 2016). The Charlson index, an indicator of multimorbidity, has been widely used and validated worldwide (Deyo et al., 1992, Marengoni et al., 2011) and fits with the purpose of this chapter as it was created from hospitalisation data for its strong relationship with mortality (Charlson et al., 1987). The Charlson index of multimorbidity uses the following 17 comorbidities: Myocardial infarction, Congestive heart failure, Peripheral vascular disease, Cerebrovascular disease, Dementia, Chronic pulmonary disease, Connective tissue disease, Ulcer disease, Mild liver disease, Diabetes, Hemiplegia, Moderate or severe renal disease, Diabetes with end organ damage, Cancer, Moderate or severe liver disease, Metastatic cancer and HIV related.

The 17 comorbidities of the Charlson index were identified using ICD-10 codes and following Quan et al. coding algorithms (Quan et al., 2005). The list of codes used to identify each comorbidity was double-checked with the Information Services Division of NHS National Services Scotland for accuracy and is available in table 6.1. Once identified for each individual, the comorbidities of the Charlson index were added to get the number of comorbidities over 12 years. The number of comorbidities was then used to create the key indicator of this chapter: a binary multimorbid indicator with the category 1 (Yes) if the individual had at least 2 comorbidities from the list of 17 during the 12 years period or 0 (No) if not. It allowed us to identify 119,580 individuals (about 3% of the 4.6 million people of the SHELS cohort) deemed multimorbid based on hospitalisation data and the Charlson comorbidities.

This chapter also investigates the relationship between subjective morbidity and more objective morbidity. SAH was used as a measure of subjective health and followed the same operationalisation and categorisation as specified in chapters 4 and 5.

The operationalisation of multimorbidity described previously led to a small prevalence of people identified as multimorbid (3%). Consequently, low number of outcome and disclosure issues ($N < 6$) appeared in minority ethnic groups. To comply with the Statistical Disclosure Control guidance and ensure the confidentiality and security of individual data, some ethnic groups had to be aggregated prior results could go through disclosure review and released to researchers. This chapter uses ethnicity as operationalised in chapter 4 for the calculation of health expectancy indicators. The Bangladeshi group was combined with the Other South Asian group. The Caribbean, Black African and Black Scottish and Other Black groups were combined into the 'African Origin' group. The results for the 'All other ethnic group' were not reported due to their heterogeneity. Therefore, results were presented for 10 ethnic groups: White Scottish, White Irish, Other White British, Other White, Any Mixed Background, Indian, Pakistani, Other South Asian, African Origin and Chinese.

This chapter follows the categorisation of SES and UK-birth as previously described in chapter 3 and further specified in chapters 4 and 5. As explained before, evidence shows that higher deprivation is associated with higher multimorbidity (Barnett et al., 2012, Johnson-Lawrence et al., 2017, Marengoni et al., 2011, Schiøtz et al., 2017). Hence, ethnic differences in SES could underlie ethnic differences in multimorbidity. Consistently with previous empirical chapters, this chapter accounts for SES differences using three SES proxies: SIMD, household tenure and the combined individual and household measure of highest qualification. Evidence

also shows differences in multimorbidity according to migrant status (Diaz et al., 2015, Lenzi et al., 2016). Consequently, the contribution of being born in the UK or outside the UK to ethnic differences in multimorbidity is also investigated.

Poisson regression was used in order to analyse ethnic differences in multimorbidity as well as the ethnic differential in the SAH-multimorbidity association. RRs (95% CI) were used to compare the risk of multimorbidity in each ethnic group relative to the level of multimorbidity in the reference group (RR=1). The first set of models looks at ethnic differences in multimorbidity. The baseline model was adjusted for age. In the next model, SES was adjusted for. Finally, UK-birth was accounted for by adjusting for it as well as combined with ethnicity to evaluate the risk of multimorbidity in ethnic groups both born in the UK and outside the UK. A second set of models presented age-adjusted RRs by ethnicity for each of the 17 comorbidities of the Charlson index in order to understand which type of diseases contribute to the overall multimorbidity patterns observed. Finally, the last set of models focused on the predictiveness of SAH for subsequent multimorbidity. The ethnic differential in the SAH-multimorbidity association was assessed through the inclusion of an ethnicity*SAH interaction term in addition to ethnicity and SAH variables in predicting multimorbidity. As in chapter 5, due to the reporting a LLTI being more prevalent than the reporting of bad health, estimates of the model including an ethnicity*SAH interaction term using LLTI as a SAH measure rather than SRH are expected to be more robust. Hence, results are presented for the ethnic differential observed in the LLTI-multimorbidity association. However, results of the same type of model using SRH as a measure of SAH are reflected upon and available in appendices.

Table 6.1. List of ICD-10 codes published by Quan et al. (2015) used to identify the 17 morbidities of the Charlson Index

Morbidity from the Charlson Index	Codes published by Quan et al. (2015)
Myocardial infarction	I21, I22, I252
Congestive heart failure	I099, I110, I130, I132, I255, I420, I425, I426, I427, I428, I429, I43, I50, P290
Peripheral vascular disease	I70, I71, I731, I738, I739, I771, I790, I792, K551, K558, K559, Z958, Z959
Cerebrovascular disease	G45, G46, H340, I60, I61, I62, I63, I64, I65, I66, I67, I68, I69
Dementia	F00, F01, F02, F03, F051, G30, G311
Chronic pulmonary disease	I278, I279, J40, J41, J42, J43, J44, J45, J46, J47, J60, J61, J62, J63, J64, J65, J66, J67, J684, J701, J703
Connective tissue disease	M05, M06, M315, M32, M33, M34, M351, M353, M360
Ulcer disease	K25, K26, K27, K28
Mild liver disease	B18, K700, K701, K702, K703, K709, K713, K714, K715, K717, K73, K74, K760, K762, K763, K764, K768, K769, Z944
Diabetes	E100, E101, E106, E108, E109, E110, E111, E116, E118, E119, E120, E121, E126, E128, E129, E130, E131, E136, E138, E139, E140, E141, E146, E148, E149
Hemiplegia	G041, G114, G801, G802, G81, G82, G830, G831, G832, G833, G834, G839
Moderate or severe renal disease	I120, I131, N032, N033, N034, N035, N036, N037, N052, N053, N054, N055, N056, N057, N18, N19, N250, Z490, Z491, Z492, Z940, Z992
Diabetes with end organ damage	E102, E103, E104, E105, E107, E112, E113, E114, E115, E117, E122, E123, E124, E125, E127, E132, E133, E134, E135, E137, E142, E143, E144, E145, E147
Cancer	C00, C01, C02, C03, C04, C05, C06, C07, C08, C09, C10, C11, C12, C13, C14, C15, C16, C17, C18, C19, C20, C21, C22, C23, C24, C25, C26, C30, C31, C32, C33, C34, C37, C38, C39, C40, C41, C43, C45, C46, C47, C48, C49, C50, C51, C52, C53, C54, C55, C56, C57, C58, C60, C61, C62, C63, C64, C65, C66, C67, C68, C69, C70, C71, C72, C73, C74, C75, C76, C81, C82, C83, C84, C85, C88, C90, C91, C92, C93, C94, C95, C96, C97
Moderate or severe liver disease	I850, I859, I864, I982, K704, K711, K721, K729, K765, K766, K767
Metastatic cancer	C77, C78, C79, C80
HIV related	B20, B21, B22, B24

6.3. Results

6.3.1. Ethnic differences in multimorbidity in Scotland

Figure 6.1 shows the RRs (95% CI) of multimorbidity by ethnicity adjusted for age in red, with separate panels for males (left panel) and for females (right panel). Exact values for RRs (95% CI) and corresponding p-values are reported in table 6.2.

Findings showed lower RRs of multimorbidity in males and females of Other White British, Other White and Chinese origins compared to their White Scottish counterparts. In contrast, higher RRs were found in Pakistani males and females compared to the White Scottish groups.

Ethnic differences in multimorbidity align with patterns found in regards to SRH and LLTI in chapter 4 where the Other White British, Other White and Chinese populations reported a health advantage while the Pakistani population reported a health disadvantage compared to the White Scottish population. However, observed patterns of worse health reported by Any Mixed Background males and females and Indian females in chapter 4 are not seen when using multimorbidity as a health indicator. In contrast, results showed a similar risk of multimorbidity compared to that of the White Scottish population in these groups.

6.3.2. The role of SES in the ethnic patterning of multimorbidity

R Rs (95% CI) of multimorbidity by ethnicity adjusted for age and additionally for SES are presented in figure 6.1 in blue. RR (95% CI) exact values and associated p-values are available in the last two columns in table 6.2.

The models adjusted for age and SES showed similar trends to the models adjusted for age i.e. lower risks of multimorbidity in males and females of Other White British, Other White and Chinese origins and higher risks in males and females of Pakistani origin compared to the White Scottish groups. However lower once SES was accounted for, the risk of multimorbidity in the Other White British and Other White groups converged towards the level of risk in the White Scottish population. This observed convergence of multimorbidity in minority white groups towards the level of the White Scottish population once accounting for SES, reached non-significant level in Other White females only. These patterns suggest that SES (through three proxies) partly explains ethnic differences in multimorbidity between the white groups in Scotland. For the Pakistani and Chinese populations, SES adjustment had minimal impact on the

risk of multimorbidity. Overall, adjustment for SES had little effect on the risk of multimorbidity in other minority ethnic groups with the exception of Indian males. In the latter, similar age-adjusted RRs of multimorbidity were relatively higher when accounting for their favourable socio-economic profile compared to the White Scottish population.

Patterns of SES adjustment and the ability of SES to explain ethnic differences in health presented in this section mostly align with previous findings using SAH in chapter 4. In white groups, SES fully explained the health differences observed when using SAH and partly explained these differences when using multimorbidity as a measure of health. Whether using SAH or multimorbidity as a health outcome, Indian males had similar RRs of poor health compared to White Scottish males in age-adjusted models but higher risks of poorer health was observed when accounting for their favourable socio-economic profile. Using either SAH or multimorbidity as a health outcome, there was a consistent inability of SES in explaining ethnic differences in health in other non-white minority ethnic groups compared to the reference group.

Figure 6.1. RRs (95% CI) of ethnic differences in multimorbidity by sex, adjusted for age (red) and adjusted for age and SES (blue)

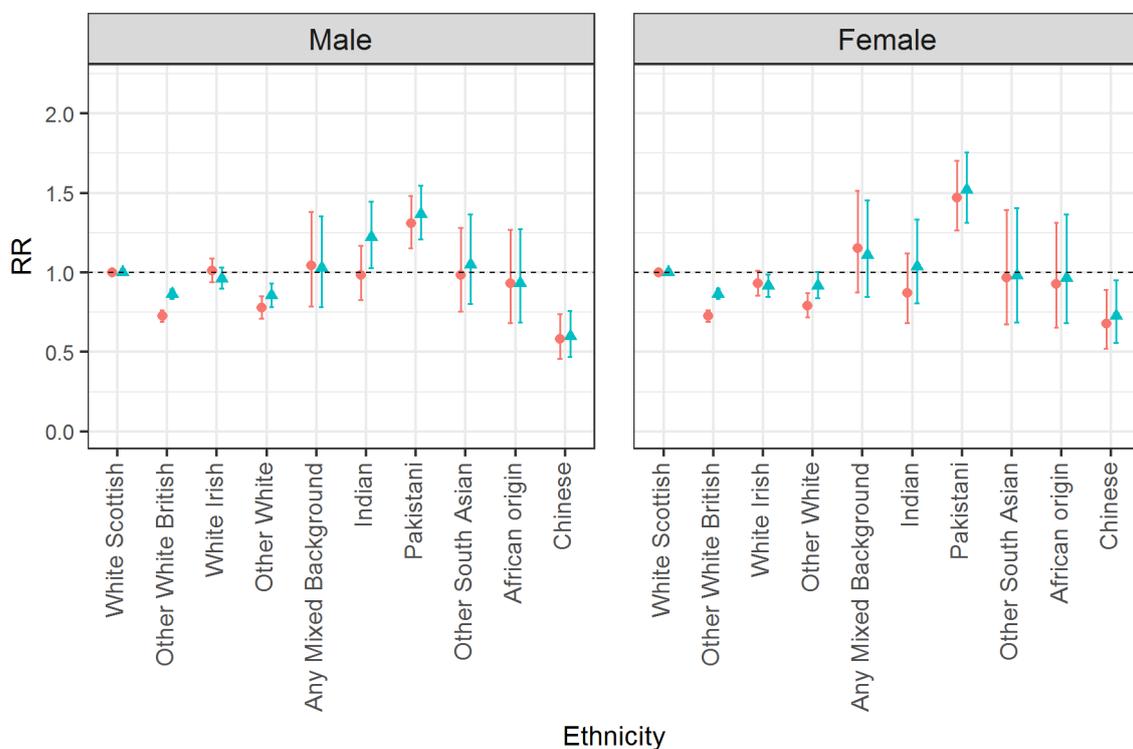


Table 6.2. RRs (95% CI) of multimorbidity by ethnicity, stratified by sex. Models are adjusted for age and adjusted for age and SES

Sex and ethnic groups	PY	Multi-morbidity	Adjusted for Age		Adjusted for Age and SES	
			RR (95% CI)	p-value	RR (95% CI)	p-value
MALES						
White Scottish	21179755	58895	1		1	
Other White British	1571080	3845	0.73 (0.69, 0.76)	<.0001	0.87 (0.84, 0.90)	<.0001
White Irish	202190	790	1.01 (0.94, 1.09)	0.7688	0.96 (0.90, 1.03)	0.2852
Other White	278515	520	0.78 (0.71, 0.85)	<.0001	0.86 (0.79, 0.93)	0.0003
Any Mixed Background	56265	50	1.04 (0.79, 1.38)	0.7649	1.03 (0.78, 1.35)	0.8413
Indian	65945	120	0.98 (0.83, 1.17)	0.8459	1.22 (1.03, 1.45)	0.0220
Pakistani	146430	245	1.31 (1.15, 1.48)	<.0001	1.37 (1.21, 1.55)	<.0001
Other South Asian	35500	50	0.98 (0.75, 1.28)	0.9038	1.05 (0.81, 1.36)	0.7300
African origin	32160	40	0.93 (0.68, 1.27)	0.6488	0.93 (0.69, 1.27)	0.6622
Chinese	68685	65	0.58 (0.46, 0.74)	<.0001	0.60 (0.47, 0.76)	<.0001
FEMALES						
White Scottish	22581190	50290	1		1	
Other White British	1644435	3010	0.73 (0.69, 0.76)	<.0001	0.87 (0.84, 0.90)	<.0001
White Irish	216905	675	0.93 (0.85, 1.01)	0.0900	0.92 (0.85, 0.99)	0.0264
Other White	319915	505	0.79 (0.72, 0.87)	<.0001	0.92 (0.84, 1.00)	0.0570
Any Mixed Background	59970	55	1.15 (0.88, 1.52)	0.3102	1.11 (0.85, 1.45)	0.4437
Indian	59925	65	0.87 (0.68, 1.12)	0.2858	1.04 (0.81, 1.33)	0.7646
Pakistani	143940	185	1.47 (1.27, 1.70)	<.0001	1.52 (1.31, 1.76)	<.0001
Other South Asian	28610	30	0.97 (0.68, 1.39)	0.8657	0.98 (0.69, 1.41)	0.9192
African origin	28590	30	0.93 (0.66, 1.31)	0.6720	0.96 (0.68, 1.36)	0.8359
Chinese	68010	55	0.68 (0.52, 0.89)	0.0050	0.73 (0.56, 0.95)	0.0195

6.3.3. Multimorbidity differences by ethnicity and UK-birth in Scotland

Tables 6.3a&b show the RRs of multimorbidity by ethnicity adjusted for age (model 1) and additionally for UK-birth (model 2) and for the interaction between UK-birth and ethnicity (model 3). Note that model 1 results were presented in table 6.2 and reported in section 6.3.1. They were reused in tables 6.3a&b to understand the effect of further adjustment.

Compared to the age-adjusted model (model 1), further adjusting for UK-birth (model 2) raised the RRs of multimorbidity slightly upward for Pakistani males and females and Chinese females. If these groups were more likely to be born in the UK, their risk of multimorbidity would see a small increase. Overall there were minor changes and adjustment of UK-birth had a minimal effect with ethnic patterns remaining very similar. Furthermore, the RRs (95% CI) of being born outside the UK in predicting multimorbidity were 0.95 (0.89, 1.01) for males and 0.92 (0.86, 0.98) for females (model 2). Results showed no to borderline significant effect of UK-birth on the risk of multimorbidity. Unlike in chapter 4, the protective effect of being born outside the UK on health is not fully supported when using multimorbidity rather than SAH as a health indicator. This could be due to a smaller prevalence of the outcome (3% of the SHELS cohort with hospitalisation-based multimorbidity against 10% reporting bad health and 20% reporting a LLTI).

The last model in tables 6.3a&b shows the model including the interaction term between UK-birth and ethnicity in predicting multimorbidity (model 3). Results showed a significant interaction UK-birth*ethnicity in predicting multimorbidity for Other White British and White Irish males compared to White Scottish males. No differential association of UK-birth and multimorbidity was revealed in the interaction analysis (model 3) for females.

In summary, UK-birth did not appear to be associated with the risk of multimorbidity in males in Scotland. However, there was a differential association of UK-birth with multimorbidity for Other White British and White Irish males compared to the reference group. In females, being born outside the UK was somewhat protective of a higher risk of multimorbidity and there was no significant differences in the ability of UK-birth to predict multimorbidity in any particular ethnic group.

Figure 6.2 shows the RRs of multimorbidity by ethnicity and UK-birth. Results were first adjusted for age (top panels) and then adjusted for age and SES (bottom panels), for males (left panels) and females (right panels). In these figures, the reference population is the White

Scottish population who was born in the UK. The age-adjusted results showed no uniform pattern of the effect of being born outside the UK on the risk of multimorbidity across ethnic groups. The multimorbidity advantage seen in Other White British males and females in section 6.3.1 appears to hold in both those who were born outside the UK and those who were born in the UK. In the Other White populations, there is a multimorbidity advantage for males regardless of them being born in the UK or not but for females, the advantage is only seen in those who were born outside the UK. UK-born Other White females had similar risk of multimorbidity compared to UK-born White Scottish females. Age-adjusted results showed a multimorbidity advantage in Chinese males and females who were born outside the UK and a disadvantage in Pakistani males and females who were born outside the UK in line with chapter 4 patterns using SAH as an outcome. In the other non-white minority ethnic groups who were born in the UK, fewer people were multimorbid. Consequently, results were either non-significant due to wide confidence intervals or disclosive (N < 6 for Chinese females).

In the models adjusted for SES, the multimorbidity advantage seen in males and females of Other White British origin who were born outside the UK was no longer significant to the extent that males had similar multimorbidity level to that of UK-born White Scottish males. SES adjustment had no clear effect on ethnic differences in multimorbidity for other ethnic groups who were born in or outside the UK.

In summary, there was no clear-cut association between UK-birth and the risk of multimorbidity. Overall, being born outside the UK did not confer a particularly lower risk of multimorbidity than being born in the UK. Likewise, multimorbidity patterns in those who were born in the UK were not uniform across ethnic groups. There was a higher risk of multimorbidity in Pakistani males and females who were born outside the UK in line with the patterns observed in chapter 4.

Figure 6.2. RRs (95% CI) of multimorbidity by ethnicity and UK-birth, stratified by sex, adjusted for age (top) and adjusted for age and SES (bottom)

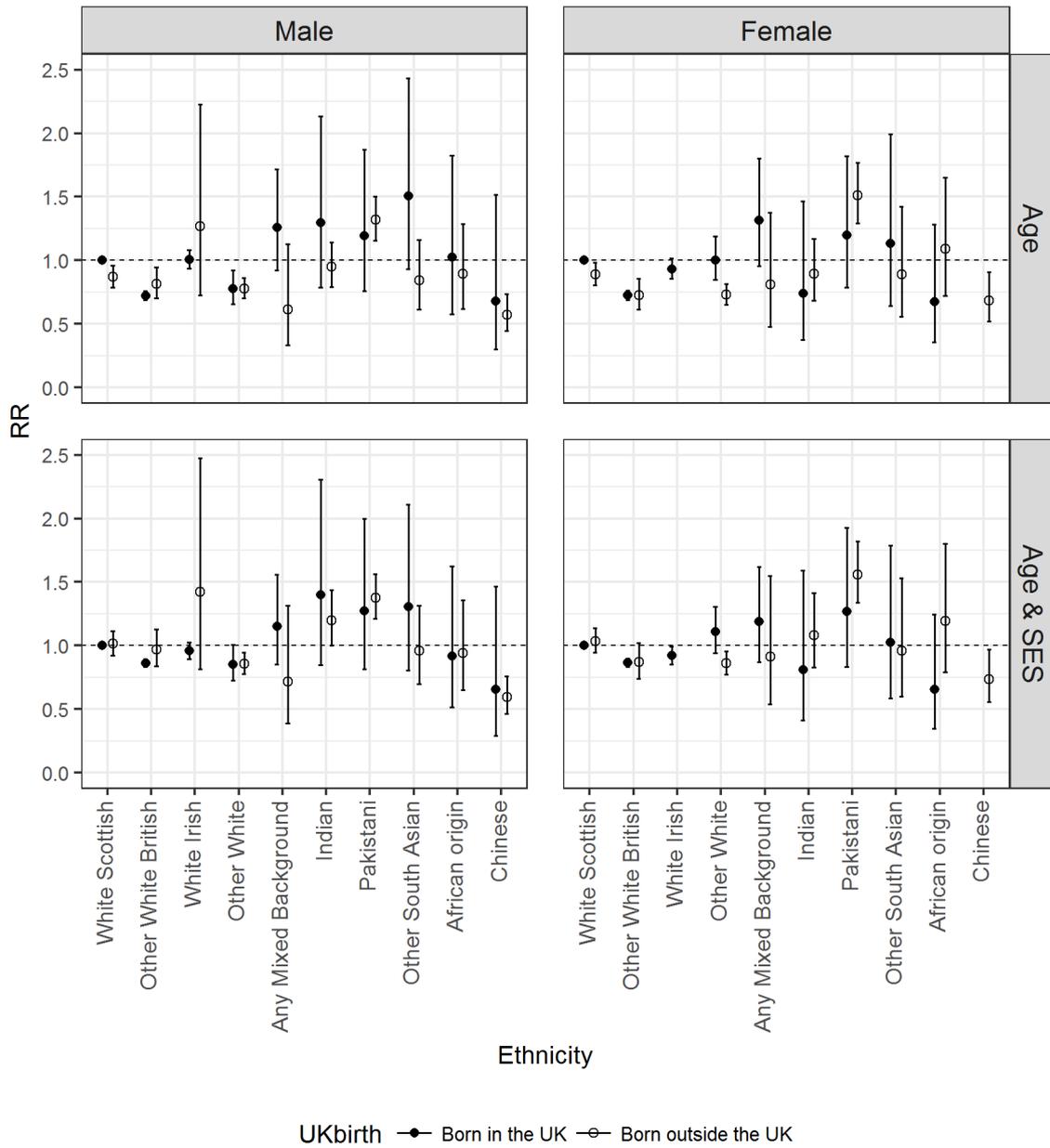


Table 6.3. RRs (95% CI) of multimorbidity by ethnicity, stratified by sex. Models are adjusted for age (model 1), for age and UK-birth (model 2) and for age, UK-birth and the interaction between UK-birth and ethnicity (model 3)

a) Males

Sex and ethnic groups	PY	Multi-morbidity	Model 1		Model 2		Model 3	
			Adjusted for Age		Adjusted for Age and UK-birth		Adjusted for Age, UK-birth and ethnicity*UK-birth	
			RR (95% CI)	p-value	RR (95% CI)	p-value	RR (95% CI)	p-value
White Scottish	21179755	58895	1		1		1	
Other White British	1571080	3845	0.73 (0.69, 0.76)	<.0001	0.73 (0.69, 0.77)	<.0001	0.72 (0.69, 0.76)	<.0001
White Irish	202190	790	1.01 (0.94, 1.09)	0.7688	1.04 (0.96, 1.12)	0.3349	0.98 (0.89, 1.09)	0.7540
Other White	278515	520	0.78 (0.71, 0.85)	<.0001	0.81 (0.74, 0.89)	<.0001	0.78 (0.66, 0.92)	0.0032
Any Mixed Background	56265	50	1.04 (0.79, 1.38)	0.7649	1.06 (0.80, 1.41)	0.6685	1.22 (0.88, 1.68)	0.2322
Indian	65945	120	0.98 (0.83, 1.17)	0.8459	1.03 (0.86, 1.24)	0.7192	1.30 (0.79, 2.14)	0.3071
Pakistani	146430	245	1.31 (1.15, 1.48)	<.0001	1.38 (1.20, 1.58)	<.0001	1.19 (0.76, 1.87)	0.4458
Other South Asian	35500	50	0.98 (0.75, 1.28)	0.9038	1.03 (0.78, 1.35)	0.8420	1.51 (0.93, 2.44)	0.0925
African origin	32160	40	0.93 (0.68, 1.27)	0.6488	0.97 (0.71, 1.33)	0.8416	1.05 (0.59, 1.86)	0.8726
Chinese	68685	65	0.58 (0.46, 0.74)	<.0001	0.61 (0.48, 0.78)	<.0001	0.68 (0.30, 1.52)	0.3440
Born outside UK vs UK-born					0.95 (0.89, 1.01)	0.0759	0.86 (0.78, 0.94)	0.0015
White Scottish*Born outside UK							1	
Other White British*Born outside UK							1.32 (1.11, 1.58)	0.0021
White Irish*Born outside UK							1.23 (1.04, 1.45)	0.0148
Other White*Born outside UK							1.17 (0.94, 1.45)	0.1578
Any Mixed Background*Born outside UK							0.68 (0.36, 1.31)	0.2485
Indian*Born outside UK							0.86 (0.50, 1.47)	0.5676
Pakistani*Born outside UK							1.29 (0.80, 2.08)	0.2947
Other South Asian*Born outside UK							0.65 (0.37, 1.17)	0.1518
African origin*Born outside UK							0.99 (0.50, 1.96)	0.9665
Chinese*Born outside UK							0.99 (0.42, 2.31)	0.9754

b) Females

Sex and ethnic groups	PY	Multi-morbidity	Model 1		Model 2		Model 3	
			Adjusted for Age		Adjusted for Age and UK-birth		Adjusted for Age, UK-birth and ethnicity*UK-birth	
			RR (95% CI)	p-value	RR (95% CI)	p-value	RR (95% CI)	p-value
White Scottish	22581190	50290	1		1		1	
Other White British	1644435	3010	0.73 (0.69, 0.76)	<.0001	0.73 (0.69, 0.77)	<.0001	0.73 (0.69, 0.76)	<.0001
White Irish	216905	675	0.93 (0.85, 1.01)	0.0900	0.97 (0.89, 1.07)	0.5636	0.95 (0.84, 1.07)	0.4156
Other White	319915	505	0.79 (0.72, 0.87)	<.0001	0.85 (0.76, 0.94)	0.0023	0.98 (0.83, 1.16)	0.8246
Any Mixed Background	59970	55	1.15 (0.88, 1.52)	0.3102	1.18 (0.90, 1.56)	0.2275	1.29 (0.94, 1.78)	0.1197
Indian	59925	65	0.87 (0.68, 1.12)	0.2858	0.94 (0.73, 1.21)	0.6360	0.66 (0.32, 1.38)	0.2676
Pakistani	143940	185	1.47 (1.27, 1.70)	<.0001	1.58 (1.35, 1.85)	<.0001	1.20 (0.79, 1.83)	0.3916
Other South Asian	28610	30	0.97 (0.68, 1.39)	0.8657	1.03 (0.71, 1.48)	0.8863	1.13 (0.64, 1.99)	0.6687
African origin	28590	30	0.93 (0.66, 1.31)	0.6720	0.98 (0.69, 1.38)	0.8940	0.68 (0.36, 1.29)	0.2349
Chinese	68010	55	0.68 (0.52, 0.89)	0.0050	0.74 (0.56, 0.97)	0.0296	0.62 (0.24, 1.62)	0.3239
Born outside UK vs UK-born					0.92 (0.86, 0.98)	0.0111	0.90 (0.81, 0.99)	0.0299
White Scottish*Born outside UK							1	
Other White British*Born outside UK							1.13 (0.94, 1.36)	0.2031
White Irish*Born outside UK							1.07 (0.89, 1.29)	0.4900
Other White*Born outside UK							0.84 (0.67, 1.05)	0.1182
Any Mixed Background*Born outside UK							0.75 (0.41, 1.37)	0.3485
Indian*Born outside UK							1.53 (0.70, 3.38)	0.2873
Pakistani*Born outside UK							1.40 (0.89, 2.21)	0.1482
Other South Asian*Born outside UK							0.88 (0.42, 1.84)	0.7267
African origin*Born outside UK							1.79 (0.83, 3.86)	0.1371
Chinese*Born outside UK							1.24 (0.45, 3.40)	0.6752

6.3.4. Ethnic differences in the 17 comorbidities of the Charlson index

Figures 6.3.a-b-c-d-e-f-g display the RRs (95% CI) of hospitalisation for each of the 17 comorbidities of the Charlson index by ethnicity and stratified by sex. Appendix 6.1 provides the RRs (95% CI) values and their associated p-values for the figures presented in this section. Even over a 12 years period, there was low number of hospitalisations in minority ethnic groups for a range of conditions creating disclosure issues or wide confidence intervals.

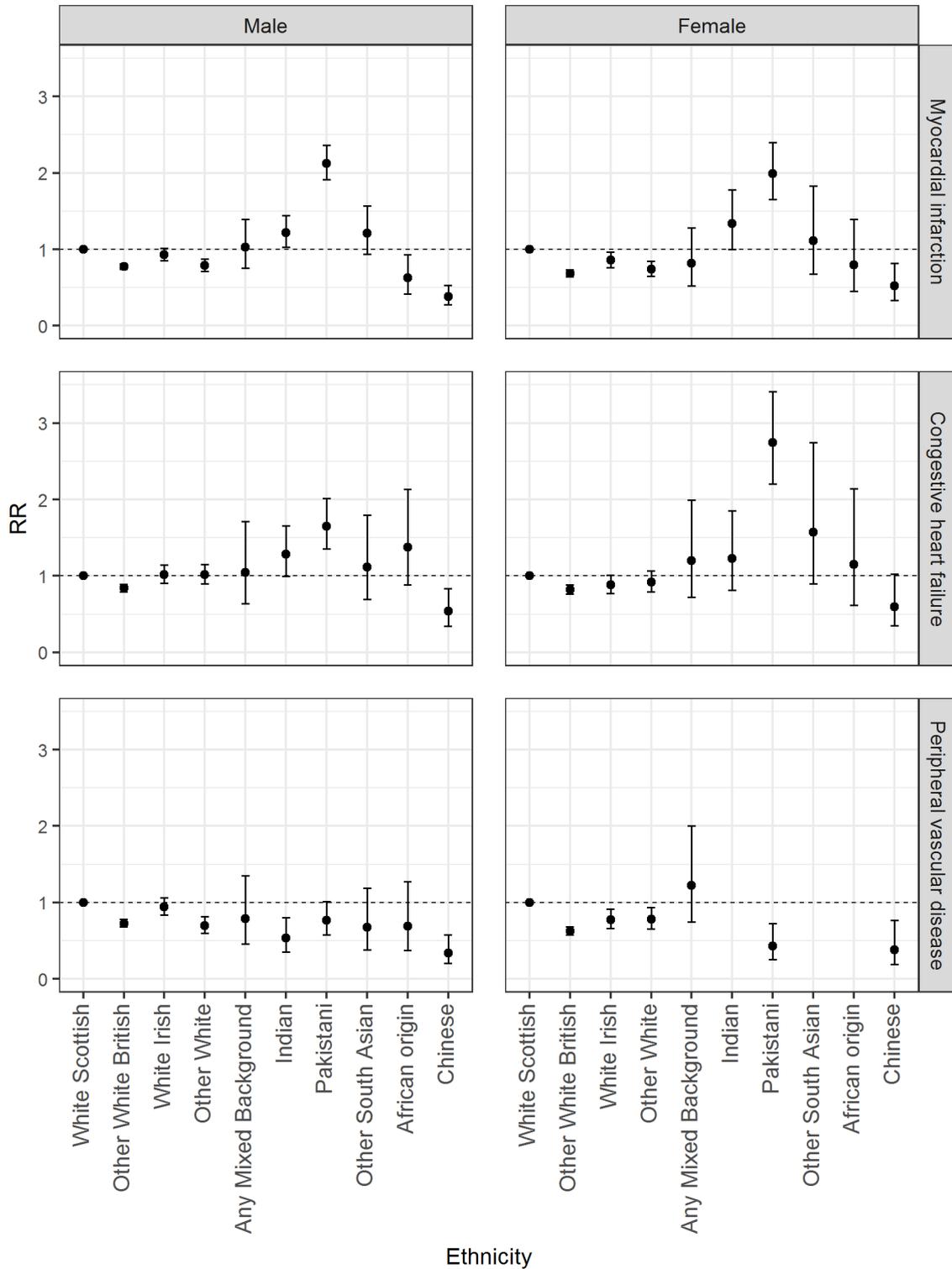
As demonstrated in previous findings of this chapter (sections 6.3.1 to 6.3.3), Pakistani males and females are at particular high risk of multimorbidity in Scotland. Rather than looking at ethnic differences for 17 comorbidities for all ethnic groups, this section focuses on the conditions underlying the disadvantage observed in the Pakistani population. The aim is to gain a global view of the diseases that are more prevalent in the Pakistani population based on hospitalisation data.

Compared to the White Scottish population, Pakistani males and females had higher risks of hospitalisation due to myocardial infarction, congestive heart failure and cerebrovascular disease (stroke) but lower risks of hospitalisation due to peripheral vascular disease. Pakistani males and females had similar risks of hospitalisation for hemiplegia compared to the White Scottish population. Chronic pulmonary disease hospitalisations were more likely in Pakistani males and females as well as hospitalisation due to connective tissue disease in Pakistani females compared to the reference group. Compared to White Scottish males, Pakistani males had a lower risk of hospitalisation due to ulcer disease. The RRs of hospitalisations due to mild liver disease were significantly higher in Pakistani males and females but non-significantly higher for the RRs of hospitalisation due to moderate to severe liver disease. Hospitalisations due to diabetes, diabetes with end organ damage and moderate or severe renal disease were more likely in the Pakistani group compared to the White Scottish group. Results showed lower relative risks of hospitalisation due to cancer and metastatic cancer in Pakistani males and females. Hospitalisations due to dementia were almost non-existent in the Pakistani population as those due to HIV.

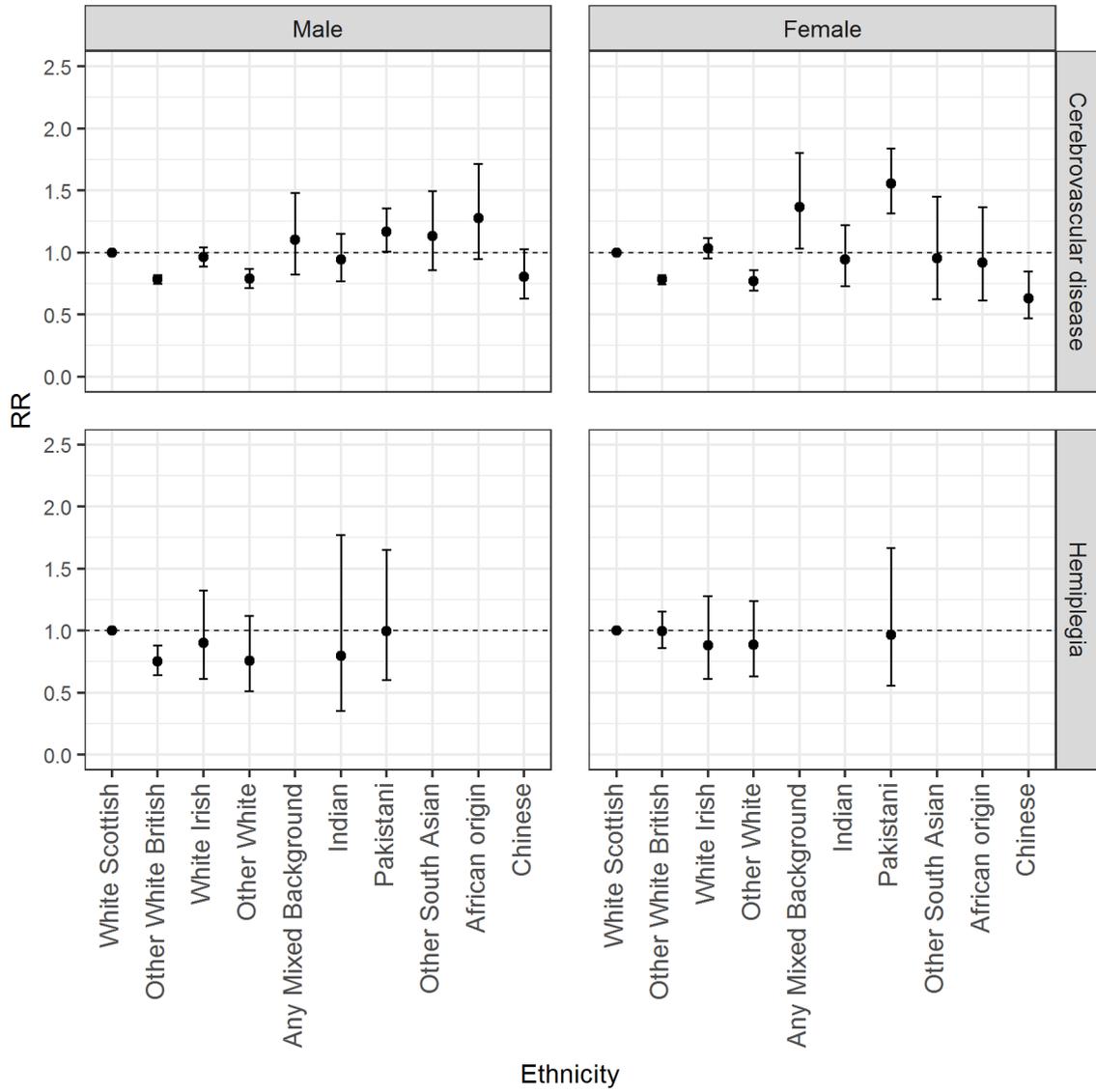
In summary, results showed that the Pakistani population was particularly more likely than the White Scottish population to be hospitalised for cardiovascular disease, stroke, diabetes, renal disease and respiratory disease while showing 30% to 60% lower risk of cancer and virtually no hospitalisation for dementia.

Figure 6.3. Age-adjusted RRs (95% CI) of ethnic differences for each comorbidity included in the Charlson Index, stratified by sex

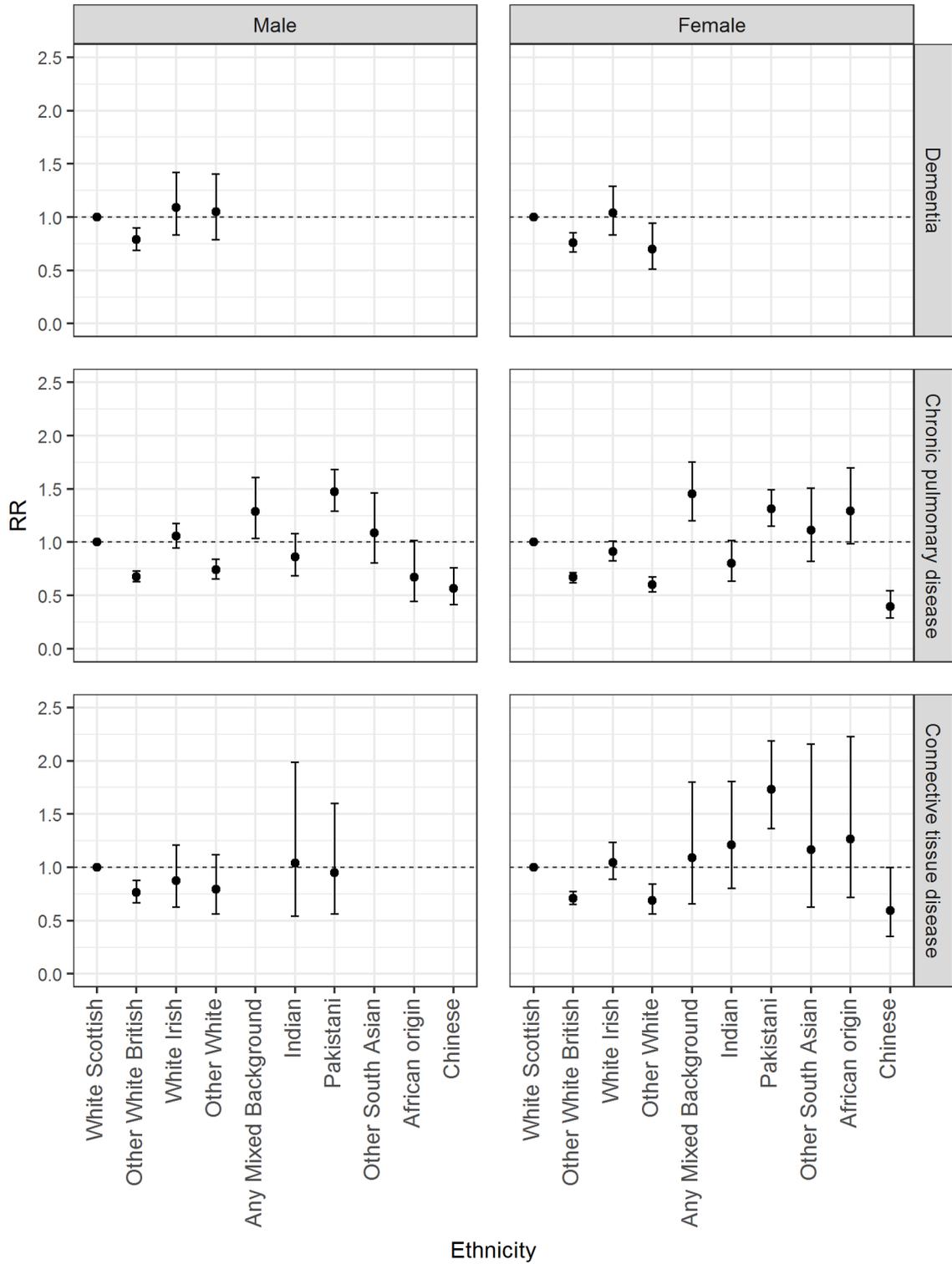
a) Myocardial infarction, congestive heart failure and perivascular vascular disease



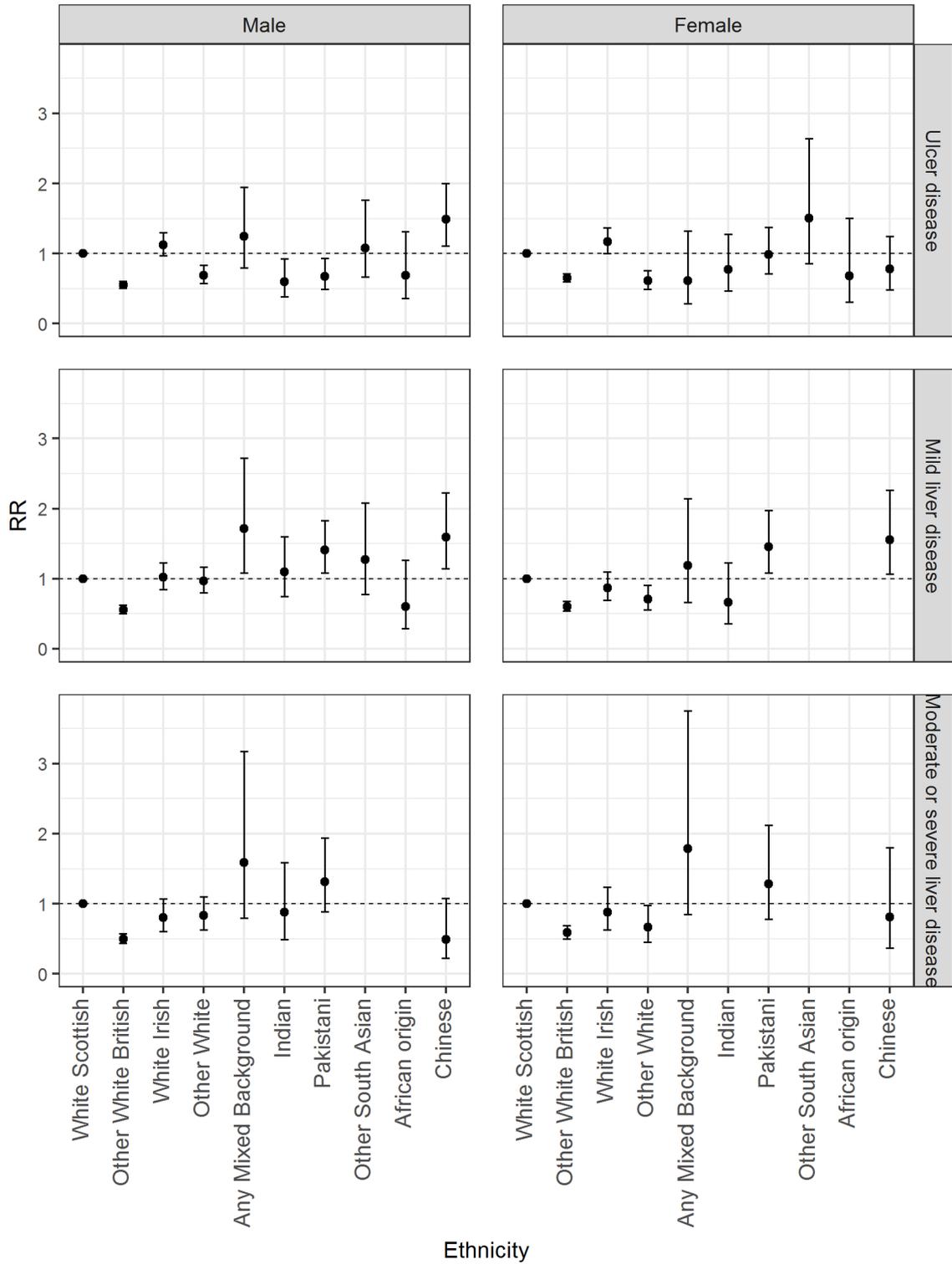
b) Cerebrovascular disease and hemiplegia



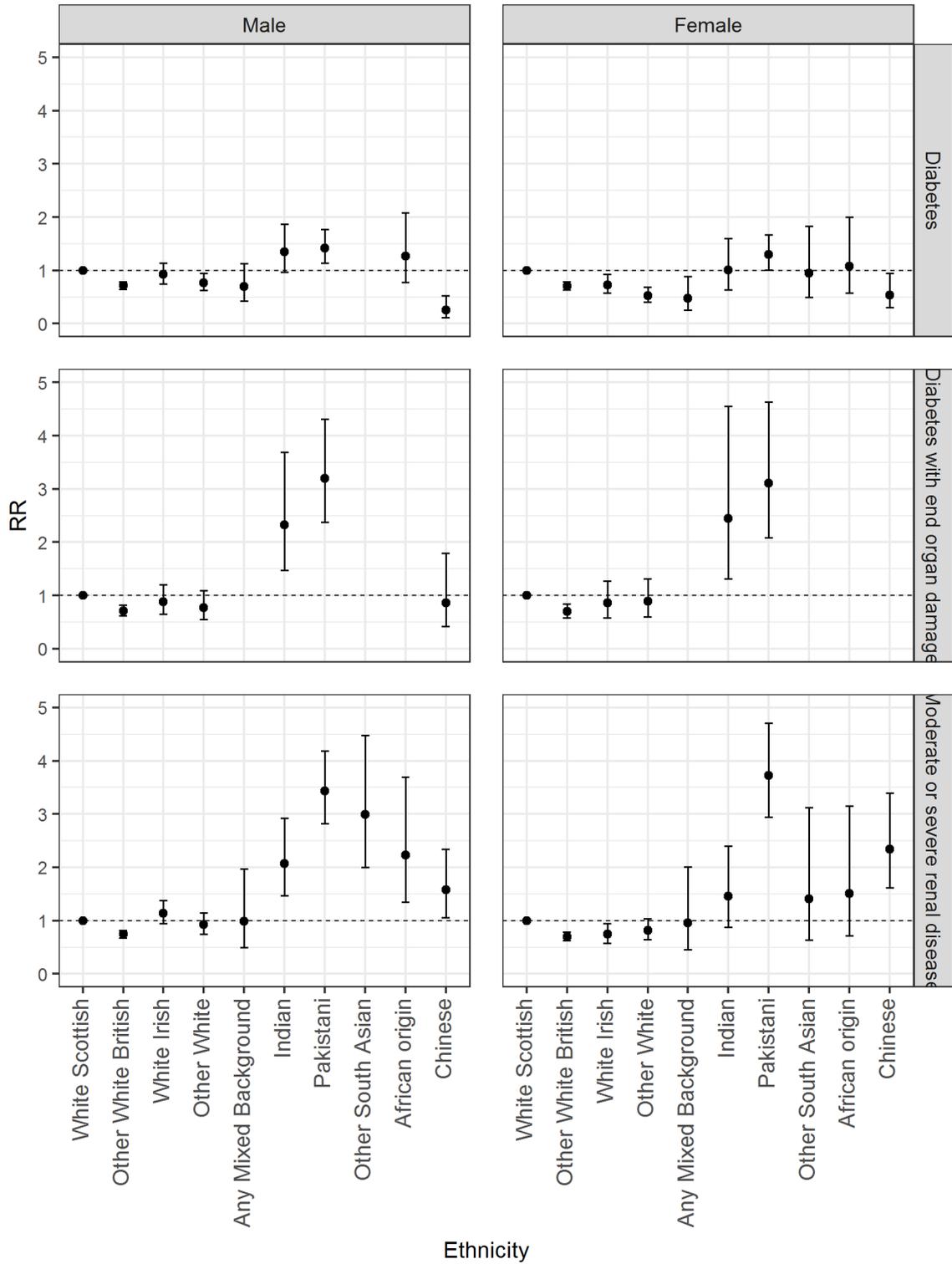
c) Dementia, chronic pulmonary disease and congestive tissue disease



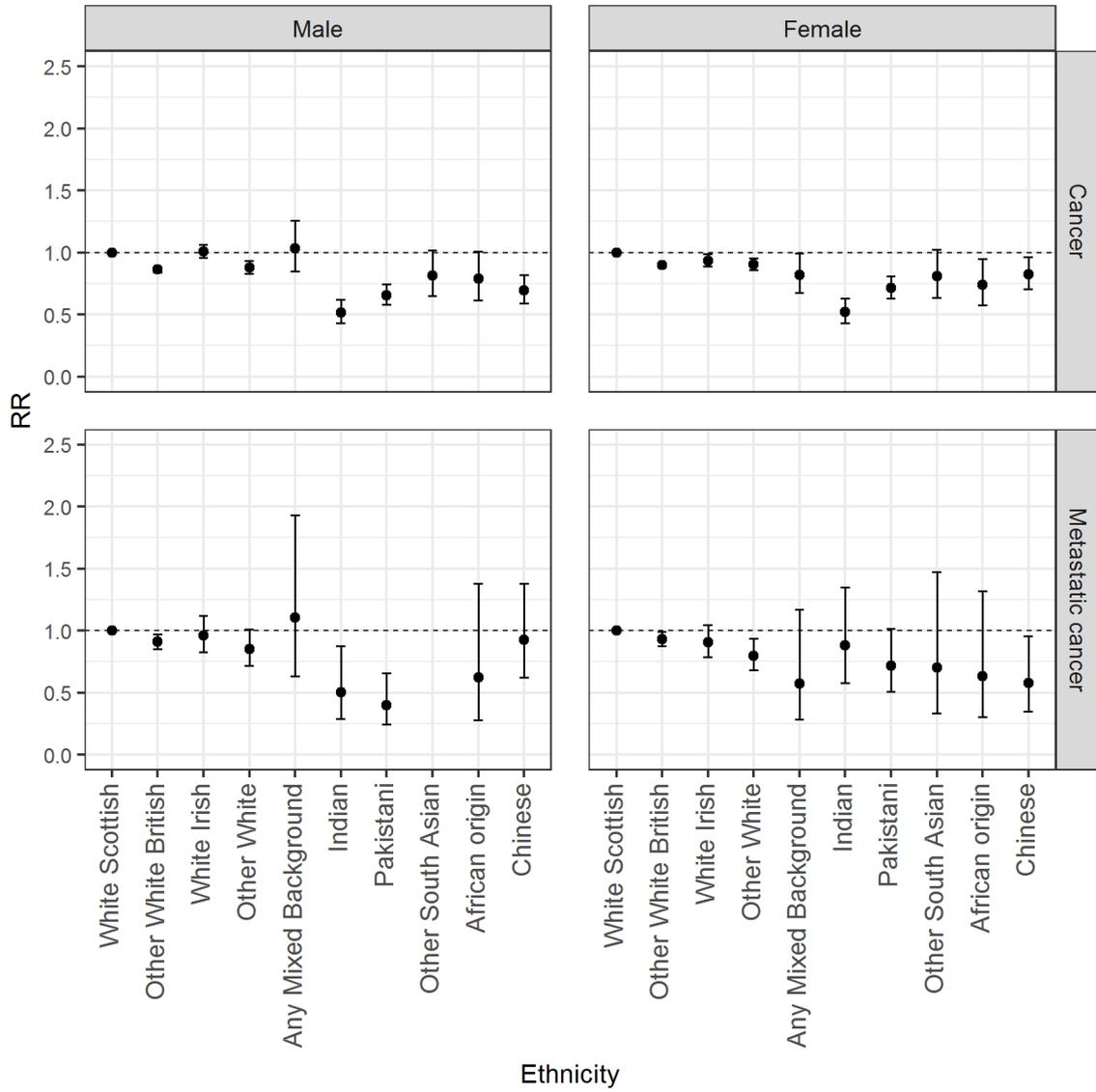
d) Ulcer disease, mild liver disease and moderate or severe liver disease



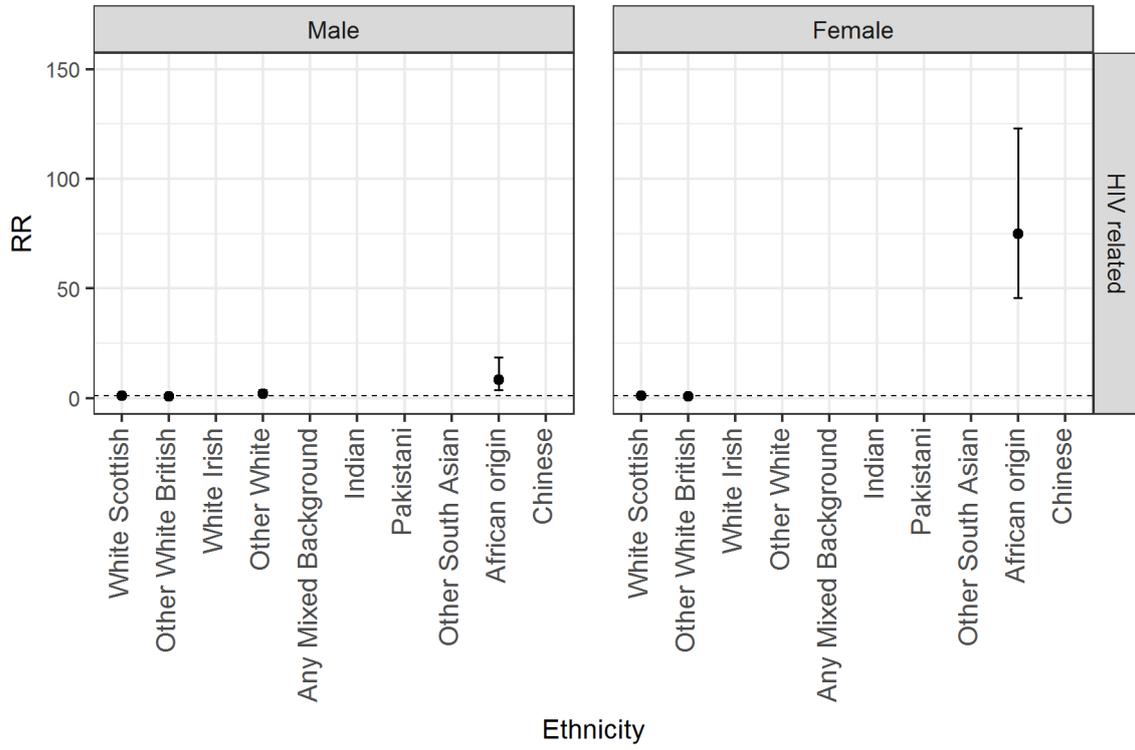
e) Diabetes, diabetes with end organ damage and moderate or severe renal disease



f) Cancer and metastatic cancer



g) HIV related



6.3.5. The association between SAH and multimorbidity by ethnicity

Tables 6.4a&b show the age-adjusted risk of multimorbidity by ethnic group for males and females (model 1). Model 1 findings were reported in section 6.3.1 and are reused in tables 6.4a&b to build on with additional adjustment. Models presented in this section were additionally adjusted for LLTI (model 2), for the interaction between LLTI and ethnicity (model 3) and for SES (model 4).

This section aims to investigate the ethnic differential in the SAH-multimorbidity association. As previously explained, it focuses on the results using LLTI as a measure of SAH but findings using SRH are available in appendix 6.2 and reflected upon.

Adjusting for LLTI (model 2) did not change the multimorbidity advantage observed in males and females of Other White British, Other White and Chinese origins. However, the high risks of multimorbidity observed in Pakistani males and females were partly reduced when accounting for LLTI. Reporting no LLTI versus at least one LLTI was associated with lower risk of multimorbidity (RRs [95% CI]: 0.40 [0.39; 0.41] in males and 0.37 [0.36; 0.39] in females) supporting the expected relationship that reporting no limitation or chronic condition is associated with lower multimorbidity and related hospitalisations.

Adding an interaction LLTI*ethnicity term into the model (model 3) allowed us to detect whether the LLTI-multimorbidity association was consistent across ethnic groups. Findings showed a significant interaction in predicting subsequent multimorbidity in Other White and Any Mixed Background males and in Indian, Pakistani and Chinese females. Once SES was accounted for (model 4), the differential LLTI-multimorbidity association remained significant in these ethnic groups.

The interaction term was in the same direction as previously seen in chapter 5 i.e. for these groups, if they reported no LLTI, they were even less likely to have subsequent hospitalisation-based multimorbidity than their White Scottish counterparts. In other words, the strength of the SAH-multimorbidity association was greater in these groups compared to that in the White Scottish population.

Tables in appendix 6.2 (using SRH) showed no significant SRH*ethnicity interaction in predicting multimorbidity in females. In males, the interaction term was significant for the Any Mixed Background group in the same direction as previously described in relation to LLTI. Any

Mixed Background males who did not report bad health were even less likely to be multimorbid based on hospitalisation data compared to White Scottish males.

As in chapter 5, the overall significance of the interaction terms was assessed by using likelihood ratio tests, testing the addition of the interaction terms ethnicity*SAH into the models already including SAH and ethnicity. For the models including LLTI, the addition of the interaction term was significant for females (p -value = 0.0006) but not significant for males (p -value = 0.2376). For the models including SRH, the addition of the interaction term was neither significant for males nor females. Overall, this provides little support for an ethnic differential in the SAH-multimorbidity association with the exception of the LLTI-multimorbidity association being somewhat stronger for non-white minority ethnic females in Scotland.

Table 6.4. RRs (95% CI) of multimorbidity by ethnicity, stratified by sex. Models are adjusted for age (model 1), and subsequently for LLTI (model 2), the interaction between LLTI and ethnicity (model 3) and SES (model 4)

a) Males

Ethnicity	PY	Multi-morbidity	Model 1		Model 2		Model 3		Model 4	
			RR (95% CI)	p-value						
White Scottish	21179755	58895	1		1		1		1	
Other White British	1571080	3845	0.73 (0.69, 0.76)	<.0001	0.77 (0.74, 0.80)	<.0001	0.79 (0.75, 0.83)	<.0001	0.90 (0.86, 0.94)	<.0001
White Irish	202190	790	1.01 (0.94, 1.09)	0.7688	0.97 (0.91, 1.04)	0.4193	0.98 (0.90, 1.07)	0.7028	0.95 (0.88, 1.04)	0.2682
Other White	278515	520	0.78 (0.71, 0.85)	<.0001	0.80 (0.73, 0.87)	<.0001	0.87 (0.78, 0.97)	0.0151	0.94 (0.84, 1.05)	0.2389
Any Mixed Background	56265	50	1.04 (0.79, 1.38)	0.7649	1.00 (0.77, 1.31)	0.9955	1.25 (0.91, 1.73)	0.1638	1.24 (0.90, 1.70)	0.1843
Indian	65945	120	0.98 (0.83, 1.17)	0.8459	0.98 (0.82, 1.17)	0.8158	1.00 (0.80, 1.26)	0.9829	1.19 (0.94, 1.50)	0.1410
Pakistani	146430	245	1.31 (1.15, 1.48)	<.0001	1.14 (1.01, 1.30)	0.0372	1.13 (0.97, 1.33)	0.1196	1.22 (1.04, 1.43)	0.0145
Other South Asian	35500	50	0.98 (0.75, 1.28)	0.9038	0.96 (0.73, 1.25)	0.7383	1.16 (0.84, 1.62)	0.3714	1.25 (0.90, 1.73)	0.1913
African origin	32160	40	0.93 (0.68, 1.27)	0.6488	0.95 (0.70, 1.28)	0.7278	0.95 (0.61, 1.48)	0.8288	0.95 (0.61, 1.48)	0.8115
Chinese	68685	65	0.58 (0.46, 0.74)	<.0001	0.60 (0.47, 0.76)	<.0001	0.51 (0.35, 0.74)	0.0003	0.53 (0.37, 0.77)	0.0008
LLTI (No versus Yes)					0.40 (0.39, 0.41)	<.0001	0.41 (0.39, 0.42)	<.0001	0.46 (0.44, 0.47)	<.0001
Other White British * LLTI							0.94 (0.87, 1.02)	0.1152	0.93 (0.87, 0.99)	0.0296
White Irish * LLTI							0.97 (0.84, 1.12)	0.6868	0.98 (0.86, 1.13)	0.8052
Other White * LLTI							0.81 (0.68, 0.97)	0.0216	0.81 (0.68, 0.96)	0.0156
Any Mixed Background * LLTI							0.54 (0.30, 0.97)	0.0388	0.55 (0.31, 0.99)	0.0461
Indian * LLTI							0.95 (0.67, 1.35)	0.7737	0.92 (0.65, 1.31)	0.6515
Pakistani * LLTI							1.03 (0.79, 1.33)	0.8518	0.97 (0.75, 1.26)	0.8346
Other South Asian * LLTI							0.62 (0.36, 1.09)	0.0984	0.61 (0.35, 1.07)	0.0853
African origin * LLTI							0.99 (0.54, 1.82)	0.9719	1.01 (0.55, 1.86)	0.9753

Chinese * LLTI	1.32 (0.81, 2.16)	0.2596	1.28 (0.78, 2.08)	0.3245
SIMD (1 vs 5-least deprived)			1.39 (1.34, 1.45)	<.0001
SIMD (2 vs 5-least deprived)			1.25 (1.20, 1.30)	<.0001
SIMD (3 vs 5-least deprived)			1.17 (1.12, 1.21)	<.0001
SIMD (4 vs 5-least deprived)			1.07 (1.03, 1.11)	0.0011
Household tenure (own vs. rent)			1.23 (1.20, 1.27)	<.0001
Highest qualification (higher vs. no)			0.81 (0.79, 0.84)	<.0001
Highest qualification (lower vs. no)			0.93 (0.90, 0.95)	<.0001

b) Females

Ethnicity			Model 1		Model 2		Model 3		Model 4	
			Adjusted for age		Adjusted for age and LLTI		Adjusted for age, LLTI and ethnicity*LLTI(0)		Adjusted for age, LLTI, ethnicity*LLTI(0) and SES	
			RR (95% CI)	p-value	RR (95% CI)	p-value	RR (95% CI)	p-value	RR (95% CI)	p-value
White Scottish	22581190	50290	1		1		1		1	
Other White British	1644435	3010	0.73 (0.69, 0.76)	<.0001	0.76 (0.73, 0.79)	<.0001	0.76 (0.72, 0.81)	<.0001	0.87 (0.82, 0.92)	<.0001
White Irish	216905	675	0.93 (0.85, 1.01)	0.0900	0.91 (0.84, 0.98)	0.0145	0.88 (0.80, 0.98)	0.0137	0.88 (0.79, 0.97)	0.0086
Other White	319915	505	0.79 (0.72, 0.87)	<.0001	0.81 (0.75, 0.89)	<.0001	0.88 (0.78, 0.99)	0.0279	0.98 (0.87, 1.09)	0.6757
Any Mixed Background	59970	55	1.15 (0.88, 1.52)	0.3102	1.08 (0.84, 1.40)	0.5470	1.17 (0.84, 1.63)	0.3397	1.14 (0.82, 1.59)	0.4228
Indian	59925	65	0.87 (0.68, 1.12)	0.2858	0.78 (0.62, 0.99)	0.0447	0.95 (0.72, 1.25)	0.7008	1.11 (0.84, 1.47)	0.4704
Pakistani	143940	185	1.47 (1.27, 1.70)	<.0001	1.19 (1.03, 1.36)	0.0147	1.34 (1.15, 1.57)	0.0002	1.45 (1.25, 1.70)	<.0001
Other South Asian	28610	30	0.97 (0.68, 1.39)	0.8657	0.93 (0.65, 1.33)	0.6912	0.98 (0.61, 1.55)	0.9140	1.00 (0.63, 1.60)	0.9877
African origin	28590	30	0.93 (0.66, 1.31)	0.6720	0.92 (0.65, 1.31)	0.6458	0.78 (0.46, 1.33)	0.3656	0.83 (0.49, 1.41)	0.4897
Chinese	68010	55	0.68 (0.52, 0.89)	0.0050	0.68 (0.53, 0.87)	0.0024	0.85 (0.62, 1.17)	0.3113	0.91 (0.66, 1.26)	0.5730
LLTI (No versus Yes)					0.37 (0.36, 0.39)	<.0001	0.38 (0.36, 0.39)	<.0001	0.43 (0.41, 0.44)	<.0001
Other White British * LLTI							0.99 (0.92, 1.08)	0.8618	0.98 (0.90, 1.06)	0.5387
White Irish * LLTI							1.08 (0.92, 1.26)	0.3650	1.08 (0.93, 1.26)	0.3253
Other White * LLTI							0.85 (0.71, 1.01)	0.0680	0.85 (0.71, 1.01)	0.0610
Any Mixed Background * LLTI							0.82 (0.48, 1.39)	0.4575	0.85 (0.50, 1.44)	0.5395
Indian * LLTI							0.55 (0.32, 0.95)	0.0318	0.53 (0.31, 0.91)	0.0222
Pakistani * LLTI							0.64 (0.45, 0.90)	0.0092	0.59 (0.42, 0.83)	0.0023
Other South Asian * LLTI							0.90 (0.44, 1.85)	0.7819	0.90 (0.44, 1.83)	0.7650
African origin * LLTI							1.35 (0.67, 2.72)	0.0576	1.31 (0.65, 2.64)	0.4555
Chinese * LLTI							0.61 (0.36, 1.02)	0.0003	0.59 (0.35, 0.98)	0.0425
SIMD (1 vs 5-least deprived)									1.48 (1.42, 1.54)	<.0001
SIMD (2 vs 5-least deprived)									1.29 (1.24, 1.34)	<.0001

SIMD (3 vs 5-least deprived)	1.22 (1.17, 1.27)	<.0001
SIMD (4 vs 5-least deprived)	1.09 (1.04, 1.13)	0.0001
Household tenure (own vs. rent)	1.22 (1.19, 1.26)	<.0001
Highest qualification (higher vs. no)	0.80 (0.77, 0.82)	<.0001
Highest qualification (lower vs. no)	0.88 (0.86, 0.91)	<.0001

6.4. Discussion and conclusion

6.4.1. Summary of findings

Based on hospitalisation data, our findings showed ethnic differences in multimorbidity in Scotland. In line with the SAH patterns by ethnicity presented in chapter 4, an advantage was observed in the Other White British, Other White and Chinese groups while the Pakistani population appeared to have a multimorbidity disadvantage compared to the White Scottish population. In contrast to chapter 4 findings that showed a disadvantage in reported health in the Any Mixed Background groups compared to the reference population, we found no apparent disadvantage in hospitalisation-based multimorbidity in these groups.

With some variability, socio-economic status and UK-birth overall failed to explain the multimorbidity patterns by ethnicity. Adjustment for SES showed a convergence towards the multimorbidity level of the White Scottish population in white groups (Other White British and Other White). In addition and in line with the chapter 4 findings, adjusting for the three measures of SES used in this thesis failed to explain ethnic differences in multimorbidity for other non-white minority ethnic groups. In Indian males only, adjusting for their favourable SES profile led to a significantly higher relative risk of multimorbidity compared to the reference population. UK-birth did not significantly predict our hospitalisation-based multimorbidity outcome. In analysis stratified by UK-birth, Pakistani males and females who were born outside the UK were at higher risks of multimorbidity. Numbers of events some of the UK-born non-white minority ethnic groups were too small to detect significant differences.

The Pakistani disadvantage in multimorbidity was characterised by higher levels of hospitalisations due to cardiovascular disease, stroke, diabetes, renal disease and respiratory disease compared to the White Scottish population. In contrast, Pakistanis were less likely to be hospitalised from cancer and barely had any hospitalisation due to dementia.

Finally, we found a strong association between LLTI as declared in 2001 and subsequent multimorbidity based on 12 years of hospitalisation data (2001-2013). This association was significant in that reporting better health was associated with a lower risk of hospitalisation-based multimorbidity. Support for an ethnic differential in the SAH-multimorbidity association was limited. Females from some of the non-white minority ethnic groups showed a stronger LLTI-multimorbidity association than White Scottish females.

6.4.2. Findings in relation to current evidence

This chapter identifies for the first time ethnic differences in multimorbidity in Scotland. Despite a few recent US studies (Bobo et al., 2016, Johnson-Lawrence et al., 2017, Quinones et al., 2017, Rocca et al., 2014, St Sauver et al., 2015), relatively little is known about ethnic differences in multimorbidity. For example, a group of US studies has shown higher risks of multimorbidity in Blacks and lower risks in Asians compared to Whites (Bobo et al., 2016, Johnson-Lawrence et al., 2017, Rocca et al., 2014, St Sauver et al., 2015) but the ethnic categories in these studies remained very broad and only one study looked into explanations of observed ethnic differences (Johnson-Lawrence et al., 2017). This gap in knowledge is particularly striking in the contexts of the UK and the rest of Europe (Diaz et al., 2015, Lenzi et al., 2016, Mathur et al., 2011). Two European studies explored multimorbidity by nativity (Diaz et al., 2015, Lenzi et al., 2016) and one UK study looked at ethnic differences in cardiovascular-related multimorbidity (Mathur et al., 2011). Consequently, our findings with great ethnic granularity of higher risks of hospitalisation-based multimorbidity in the Pakistani group and lower risks in Other White British, Other White and Chinese groups compared to the majority ethnic group in Scotland makes a unique contribution to understanding ethnic differences in multimorbidity in the international context.

In relation to explanations, one study in the US looked at the effect of education on ethnic differences in multimorbidity (Johnson-Lawrence et al., 2017). The authors found that education reduced the observed Black/White gap in multimorbidity with a smaller but persistent disadvantage in Black populations compared to Whites. As explained in previous chapters, using one measure of SES might not be enough to adequately reflect the multifaceted nature of socio-economic deprivation. This thesis uses two measures of SES in addition to education to explore the contribution of SES in explaining ethnic differences in health. Nevertheless, SES could only explain some of the differences in multimorbidity observed between the white groups in Scotland and had little effect on the multimorbidity advantage or disadvantage observed in non-white minority ethnic groups.

The effect of UK-birth on the ethnic differences observed in this chapter was also explored. We found a no to weak association between UK-birth and multimorbidity and little effect of UK-birth adjustment on ethnic differences in multimorbidity. Our findings contrast with those of previous studies showing lower risks of multimorbidity in foreign-born compared to native-born populations (Diaz et al., 2015, Johnson-Lawrence et al., 2017, Lenzi et al., 2016).

Indeed, we expected those who were born outside the UK to fare better in terms of multimorbidity than the majority population and those who were born in the UK, in line with a healthy migrant effect hypothesis. In chapter 4, we found a significant association between SAH and UK-birth and a clear protective effect of being born outside the UK in the risk of reporting poorer health which supported a healthy migrant effect. Why such a nativity-multimorbidity association does not appear in Scotland could be due to the type of data used to create our multimorbidity indicator (hospitalisation versus survey or primary care data) or that the healthy migrant effect might not apply when we study more severe measures of morbidity. Further research should explore how the creation of the multimorbidity indicator and the type of data used affect the nativity-health association. Johnson-Lawrence et al. adjusted for nativity (foreign-born versus US-born) in their analysis of ethnic differences in multimorbidity but adjustment was made in combination with other factors (Johnson-Lawrence et al., 2017). Consequently, how nativity contributes to explaining observed ethnic differences in multimorbidity could not be assessed on its own in their study.

The Pakistani disadvantage in multimorbidity found in this study was characterised by hospitalisations due to a list of specific diseases. One UK study looked at ethnicity and differences in multimorbidity prevalence (Mathur et al., 2011). The authors focused on cardiovascular multimorbidity rather than overall multimorbidity but their findings bring support to the results of this research. Based on primary care data and a large population sample from east London, the authors found a higher prevalence of cardiovascular multimorbidity in South Asians compared to their reference group (Whites). Cardiovascular multimorbidity was based on diagnostic codes of hypertension, ischaemic heart disease, heart failure, stroke and diabetes. Hence, their findings echo the higher risk of multimorbidity found in this study in the Pakistani population compared to the White Scottish population. The Pakistani disadvantage was characterised by a higher risk of hospitalisation due to cardiovascular disease, stroke, diabetes and renal disease but also in our case, respiratory disease. In addition, the findings of this chapter have the advantage of being able to disaggregate the South Asian groups into Indian, Pakistani and Other South Asians, ethnic groups which have been shown in this thesis and elsewhere to be epidemiologically distinct. Research in the field of multimorbidity has recently investigated clusters of diseases (Schäfer et al., 2014, Schäfer et al., 2010, Xu et al., 2017). Three key patterns of specific disease combinations were identified: cardiovascular and metabolic diseases, mental health related problems and musculoskeletal disorders (Schäfer et al., 2010, Xu et al., 2017). The list of diseases underlying the multimorbidity disadvantage in the

Pakistani population fits with the recently identified cardiovascular and metabolic diseases cluster. However this classification does not include respiratory disease which contributes to the overall health condition of Pakistani population in Scotland.

Our findings on the Pakistani population also fit with previous research on ethnic differences in specific diseases in the UK. For example, Pakistani populations and more generally South Asian populations have a higher prevalence of diabetes and renal disease in the UK (Dreyer et al., 2009, Forouhi et al., 2006, Hull et al., 2011, Sproston and Mindell, 2006). In Scotland, evidence based on the SHELS study showed that the Pakistani population was at higher risks of cardiovascular disease (Bansal et al., 2013, Bhopal et al., 2011), stroke (Bhopal et al., 2012a), asthma (Sheikh et al., 2016), all-cause and lower respiratory diseases (Bhopal et al., 2015, Simpson et al., 2015). Low cancer incidence was also found in the Pakistani population in Scotland including for specific cancers such as lung, colorectal, breast and prostate cancers (Bhopal et al., 2012b). These cancer morbidity patterns align with cause-specific mortality evidence in the UK where those who were born in Pakistan were less likely to die from cancer compared to native-born individuals (Smith et al., 2000, Wallace and Kulu, 2015, Wild et al., 2006). However, evidence in relation to cardiovascular disease and stroke mortality among those who were born in Pakistan is conflicting, with earlier studies showing a disadvantage in this population (Smith et al., 2000, Wild et al., 2007) while more recent evidence found no disadvantage (Wallace and Kulu, 2015) in comparison to those born in England and Wales. This later research in England and Wales found an all-cause mortality advantage in those born in Pakistan compared to native-born individuals (Wallace and Kulu, 2015) which echoes the recent Scottish evidence of an all-cause mortality advantage in the Pakistani population compared to the White Scottish population (Bhopal et al., 2018, Gruer et al., 2016). The question of why higher risks of hospitalisation due to the metabolic cluster type of diseases do not seem to lead to higher mortality in the Pakistani population in Scotland remains unresolved and need further exploration. The likelihood and understanding of this morbidity-mortality contrast will be further discussed in chapter 7.

Finally, this chapter explores the relationship between SAH and multimorbidity with the aim of understanding how subjective health predicts more objective measures of morbidity. To our knowledge, no other study has explored the relationship between SAH and subsequent multimorbidity across ethnic groups. Moreover, research in general population tend to focus on how multiple conditions predict the rating of health (Mavaddat et al., 2014, Perruccio et al.,

2012) rather than the other way around. Hence, our findings contribute to the literature on the predictiveness of SAH for objective morbidity using a new measure of general morbidity as outcome. We found that SAH predicted subsequent hospitalisation-based multimorbidity across ethnic groups with a differential association for some ethnic groups. This differential association was almost inexistent when using SRH as SAH measure which supported that SRH predicts multimorbidity consistently across ethnic groups, in line with the study by Chandola and Jenkinson (Chandola and Jenkinson, 2000). When using LLTI as a measure of SAH, some ethnicity*SAH interaction terms were significant in predicting multimorbidity for some ethnic groups. As explained in chapter 5, this could be due to the higher prevalence of LLTI compared to SRH which permits the analysis to be powerful enough to detect significant interactions when using LLTI but not when using SRH. In females, this differential association was significant in females of Indian, Pakistani and Chinese origins. In Any Mixed Background males, findings of a differential SAH-multimorbidity association were consistent when using LLTI and SRH. This differential association was consistent with the fact that Any Mixed Background males showed a reported health disadvantage and similar multimorbidity level compared to White Scottish males. Their differential patterns could be due to their relatively younger profile in Scotland reporting a relative health disadvantage in younger ages that has not yet been formalised into severe morbidity leading to hospitalisation later in life.

Nevertheless, the observed differential association for specific ethnic groups could be more generally the result of cultural differences in the reporting of health which in turn could lead to predict hospitalisation-based multimorbidity differently for these groups. However, caution is required with this interpretation as other reasons might underlie the ethnic differential in SAH-multimorbidity association. Due to the nature of our multimorbidity measure, there is a possibility that a differential association reflects selection effect into hospitalisation whereby particular ethnic groups would be more or less likely to be hospitalised for the same condition and at the same level of disease severity compared to the reference ethnic group.

A differential selection into hospitalisation in the Pakistani population compared to the White Scottish population could also underlie the higher hospitalisation-based multimorbidity found in this population compared to the reference population. It is possible that some minority groups might be more likely to reach secondary care rather than primary care for similar conditions and disease severity compared to the majority, either due to different cultural preferences and norms in seeking care or due to lack of effective treatment in primary care

settings leading to hospitalisation. In Scotland, Katikireddi et al. explored ethnic differences in the likelihood of being hospitalised due to lack of effective primary care delivery (Katikireddi et al., 2018). The authors found that the Pakistani population had higher risks of avoidable hospital admissions and unplanned readmissions in Scotland. Their finding suggests that the reason for higher rates of hospitalisations in the Pakistani population of Scotland might be due to the lack of effective primary care delivery. These results should be interpreted with caution as the definition of avoidable hospitalisation relies on a list of diseases which might not be culturally sensitive nor reflect the severity of disease which might lead to hospitalisation rather than primary care treatment. In addition, English proficiency could also be a barrier to differential access to care. For example, the Pakistani population could be reluctant to consult their GPs because of poor English and the embarrassment of needing to take a relative along with good English or request translation services. This could lead to delayed presentation with symptoms. Finally, Scottish evidence points to physical multimorbidity being a strong predictor of unplanned and preventable admissions to hospital (Payne et al., 2013). Hence, higher levels of hospitalisations and related multimorbidity in the Pakistani population might well reflect their actual morbidity level. Looking at multimorbidity using primary care data would be the obvious way forward to further support the morbidity disadvantage observed in the Pakistani population of Scotland.

6.4.3. Conclusion and opportunities for future research

This chapter has shown ethnic differences in multimorbidity in Scotland along the lines of the subjective health patterns observed in chapter 4. SES and UK-birth contributed little into the observed differences. Results were particularly worrying for the Pakistani population who was more likely to be hospitalised for multiple diseases compared to the White Scottish population. This chapter offered some clues in relation to which diseases underlie this disadvantage but what are the underlying mechanisms of the cluster of diseases driving the disadvantage in the Pakistani population has yet to be understood. Assuming that the mortality advantage in the Pakistani population is real, our findings on multimorbidity and related diseases support a morbidity-mortality paradox in the Pakistani population in Scotland. This morbidity disadvantage combined with a mortality advantage means many years of life in unhealthy state and poor quality of life.

As SES and UK-birth had little influence on our findings, research should aim to disentangle how these ethnic inequalities in multimorbidity are produced. Multimorbidity

results from an accumulation of health conditions over time. Thus, a life course approach would be useful, as it has been shown that childhood hardship and mid-adulthood earnings predicts greater multimorbidity (additional number of chronic conditions) later in life (Tucker-Seeley et al., 2011). Additionally, as context, history of migration and exposure vary across countries, future research needs to assess multimorbidity inequalities and associated needs in various ethnically diverse contexts.

Finally, multimorbidity was measured based on hospitalisation data and for a particular set of diseases. As acknowledged earlier, selection into hospitalisation cannot fully be ruled out as an underlying mechanism of the differences observed in this chapter. However, our findings were in line with the ethnic patterns observed in chapter 4 which brought support to the relevance of the ethnic differences observed in our hospitalisation-based multimorbidity measure. Furthermore, the key diseases underlying the excess multimorbidity found in the Pakistani population in Scotland were in line with disease-specific evidence available in Scotland and the UK more broadly, adding support to the relevance of our findings. To confirm our findings in the Scottish context, further investigation should seek to use primary care data in order to avoid the issue of selection into hospitalisation and assess ethnic differences in (possibly a less severe measure of) multimorbidity. Further qualitative work should complement quantitative research by providing additional in-depth information on the likelihood of differential access to primary and secondary care for different ethnic groups.

CHAPTER 7

7. Conclusion

This thesis has investigated the contrast between morbidity and mortality for ethnic groups in Scotland. Driven by the broad aim of improving understandings of ethnic inequalities in health and their underlying mechanisms, this research has tackled tensions in the extant literature that have identified a morbidity disadvantage combined, paradoxically, with a mortality advantage, for some ethnic groups. This was possible through use of a unique population source that links, for the first time, information on morbidity and mortality for the same individuals, with sufficient population size to enable a robust analysis by ethnic groups. Data linkage permitted the gathering of ethnicity, socio-demographic indicators and a range of health measures. Data on health included subjective and more objective measures of health as well as measures of morbidity and mortality. Hence, the richness of the health data provided the opportunity for a detailed investigation of a morbidity-mortality contrast in specific ethnic groups in Scotland. Thus, this thesis has explored:

- How patterns of reported morbidity by ethnicity compare to patterns of mortality by ethnicity based on the same population source.
- Whether mechanisms thought to shape ethnic inequalities in health contribute to explaining ethnic differences in reported morbidity.
- Whether reported morbidity relates to mortality consistently across ethnic groups.
- Whether using a doctor-diagnosed measure of health provides similar patterns of ethnic differences in morbidity as using reported morbidity.

This last chapter provides a summary of the findings (section 7.1), discusses methodological contributions (sections 7.2) and theoretical contributions along with the interpretation of the findings (section 7.3), recommends future research avenues (section 7.4) and finishes with concluding remarks (section 7.5).

7.1. Summary of findings

Chapter 4 showed ethnic differences in self-assessed health in 2001 in Scotland. Males and females from Other White British, Other White, and Chinese origins and Indian males reported a health advantage compared to the White Scottish group, while the Any Mixed Background and Pakistani groups reported a disadvantage.

Chapter 4 aimed to investigate *whether mechanisms thought to shape ethnic inequalities in health contribute to explaining ethnic differences in reported morbidity*.

The findings showed that socio-economic circumstances explained the differences in SAH observed between the white groups but not those observed in non-white minority ethnic groups. Accounting for being born in or outside the UK had little effect on the patterns of ethnic differences in SAH observed. Nevertheless, being born outside the UK was overall protective and associated with reporting better health, supporting the healthy migrant effect hypothesis. However, the findings in the Pakistani population diverged from this pattern as Pakistani people who were born outside the UK showed worse health than the majority population and to a greater extent than Pakistani people who were born in the UK.

In older ages, differences overall reduced to the level of reported health of the majority population which support theories of a convergence of inequalities in older ages. However, Pakistani males and females and Indian females had greater SAH inequalities in older ages, somewhat supporting theories of an accumulation of disadvantage and increased ethnic inequalities in older ages.

This thesis also aimed to explore *how patterns of reported morbidity by ethnicity compare to patterns of mortality by ethnicity based on the same population source*.

The findings showed overall consistent patterns of ethnic differences in reported morbidity and ethnic difference in mortality based on the SHELS cohort. However, SAH patterns diverged from mortality patterns for particular ethnic groups. Through the use of healthy life expectancy and life expectancy, a striking morbidity-mortality contrast appeared in the Pakistani population, with this group having among the longest life expectancies in Scotland along with the shortest healthy life expectancies and thus, the longest expected life in poor health.

The contrasted SAH-mortality findings in the Pakistani population of Scotland bring into question the ability of SAH to predict mortality consistently across ethnic groups in the Scottish context. It is possible that the morbidity and mortality findings presented in chapter 4 reflect the patterns of particular age groups and cohorts within the Pakistani population. For example, there could be a mortality advantage in Pakistani elders and a reported morbidity disadvantage in the Pakistani working age population which affects the overall morbidity and mortality patterns and does not directly indicate whether poor health is related to mortality at the

individual level. Hence, it was found necessary to analyse the health trajectory of each individual and link SAH to mortality at the individual level to assess the SAH-mortality relationship across ethnic groups, and this was the focus of chapter 5.

Chapter 5 tested *whether reported morbidity relates to mortality consistently across ethnic groups*.

A first analysis demonstrated that poorer health reported in 2001 predicted higher risk of mortality over the next 12 years for most ethnic groups in Scotland. Non-significant SAH-mortality associations were found for small minority ethnic groups as a likely result of small sample size. The exploration of ethnic differences in mortality stratified by whether reporting a LLTI or not showed a mortality advantage in most minority ethnic groups in line with the general mortality advantage findings. In addition, accounting for reporting a LLTI in the mortality analysis had little effect on the observed mortality patterns by ethnicity. However, a differential SAH-mortality association was found in large ethnic groups such as Other White British, Other White and Pakistani groups in comparison to the SAH-mortality association seen in the White Scottish group. The results showed that SAH predicted mortality for these groups but with a greater strength of association i.e. reporting better health was associated with even lower mortality risk compared to the majority population. SES differences in the SAH-mortality association could not explain this ethnic differential. The mortality advantage found in the Pakistani population compared to the majority population regardless of their declared LLTI was in line with the SAH-mortality contrast in the Pakistani population presented in chapter 4.

In light of the findings of chapters 4 and 5, one can question the validity of SAH to measure the health status of individuals similarly across ethnic groups. To assess if the morbidity disadvantage seen in the Pakistani population relates to the subjective nature of SAH or a general morbidity disadvantage, ethnic differences in health need to be investigated using a more objective measure of morbidity. This was the focus of chapter 6.

In chapter 6, this thesis investigated *whether using a doctor-diagnosed measure of health provides similar patterns of ethnic differences in morbidity as using reported morbidity*.

The findings showed ethnic differences in doctor-diagnosed multimorbidity (based on 12 years of hospitalisations) in Scotland. A multimorbidity advantage was observed in the Other White British, Other White and Chinese groups while the Pakistani group had a disadvantage

compared to the White Scottish population. These findings aligned with the patterns of morbidity found using SAH as a measure of health. To better understand the multimorbidity disadvantage seen in the Pakistani population, chapter 6 also explored ethnic differences in the risk of hospitalisation from each of the diseases used to create the multimorbidity indicator. The Pakistani population showed a higher risk of hospitalisation due to diabetes, renal disease, cardiovascular disease and respiratory disease and a lower risk of hospitalisation due to cancer. Hospitalisation for dementia was almost non-existent in the Pakistani population. These results supported a morbidity disadvantage in the Pakistani population of Scotland, driven by a particular set of diseases, primarily linked to the metabolic syndrome. This was in line with known ethnic differences in the disease-specific literature. Hence, the hospitalisation-based multimorbidity patterns by ethnicity provide additional support to the existence of a morbidity-mortality paradox in the Pakistani population of Scotland whereby Pakistani people live longer but in poorer health. When assessing the relationship between SAH and multimorbidity, the results showed a differential SAH-multimorbidity association in a few ethnic groups. This suggests that the indicator used to assess ethnic inequalities in morbidity matters at least for some groups and that various measures of morbidity might provide information on different aspects of health.

7.2. Methodological contributions

SHELS is an exceptional data source for researching ethnic differences in health in Scotland. Its considerable sample size at the national-level in Scotland (4.6 million people) adds strength to the robustness of the findings and permitted analyses with greater estimate precision and ethnic granularity than has previously been achieved. The population of Scotland was estimated to be 5.1 million people in Scotland in 2001 and linkage biases within the SHELS study might exist. However, the analysis compared the health of minority ethnic groups to the health of the White Scottish population mostly using regression analysis techniques which allow this research to account for multiple socio-demographic individual characteristics. Hence, our results based on a high proportion of the population (90% of the estimated population of Scotland in 2001) are likely to be overall generalizable to the ethnically diverse population of Scotland. Furthermore, the availability of the data at the individual level was key to identifying differences and associated explanations in a more refined way than has been possible before (Scottish Government, 2004, Scottish Government, 2015). The large sample size of the SHELS

data combined with its individual linkage between census data and 12 years of hospitalisation and mortality data permitted this thesis to make a number of methodological contributions.

First, this research offers for the first time health expectancy estimates by ethnicity using a direct method and data based on a unique population source. A few studies have provided health expectancy estimates by ethnic or migrant groups but their calculations were either based on an indirect method involving mortality estimation (Rees et al., 2009, Wohland et al., 2015) or on a direct method involving data from different population sources (Carnein et al., 2015, Hayward et al., 2014, Reus-Pons et al., 2017).

Second, despite minority ethnic groups comprising a small proportion of the population in Scotland, analyses intersecting ethnicity with other individual characteristics and interaction analyses were rendered possible thanks to the large sample size of the data source. This permitted the exploration of the health experience of minority ethnic groups who were born in and outside the UK, highlighting diverging patterns in migrants and descendants and differential associations between UK-birth and health for particular ethnic groups in Scotland. Analysis was also refined to explore health inequalities in younger and older populations of ethnic groups, offering clues about the mechanisms of ethnic health inequalities in older ages.

Third, using interaction analysis combined with data linkage enabled the analysis of the individual link between more subjective health such as self-assessed health and more objective health such as doctor-diagnosed multimorbidity and mortality, and to assess the likelihood of an ethnic differential in the SAH-mortality and SAH-multimorbidity associations. Previous research in the UK lacked large samples to confidently identify an ethnic differential in the association between reported health and other measures of health (Chandola and Jenkinson, 2000). So far, the SAH-mortality association across ethnic groups has been explored in a couple of studies in the US (Assari et al., 2016, Woo and Zajacova, 2016) but no other study has yet explored it in the European context.

Finally, the multimorbidity analysis was based on 12 years of hospitalisation data linked to the Scottish Census 2001. This individual linkage was determinant in exploring, for the first time in Europe, ethnic differences in multimorbidity. Previous studies were limited to the US setting (Bobo et al., 2016, Johnson-Lawrence et al., 2017, Quinones et al., 2017, Rocca et al., 2014, St Sauver et al., 2015) or a specific type of disease and wide ethnic groupings in the UK (Mathur et al., 2011). However, as the proportion and accuracy of ethnicity recording improves on primary and secondary care data (Information Services Division, 2014, Mathur et al., 2013),

further methodological advances are to be expected in the exploration of multimorbidity by ethnicity in the UK.

7.3. Interpretation of the findings and theoretical contributions

This thesis provides empirical evidence as summarised in section 7.1. It is crucial to appreciate how this empirical evidence contributes to the theories hypothesised as potential explanations for ethnic inequalities in health. In Scotland, our findings of ethnic inequalities in health show an advantage in some ethnic groups and a disadvantage in other groups compared to the White Scottish population. Different ethnic groups have different health experiences for a combination of reasons including different migration history, selection processes, socio-economic status, exposure to discrimination, health behaviours, and biological factors.

For this project, the contribution of SES in explaining ethnic differences in health was explored. Overall, minority ethnic groups in Scotland showed a better or similar socio-economic status compared to the White Scottish population. This aligns with the idea that migrants to Scotland are socio-economically selected (Walsh, 2017). Results showed that the health advantage observed in some of the minority white groups in Scotland in comparison to the White Scottish group was fully explained by SES in the SAH analysis and partly explained by SES in the multimorbidity analysis. Thus, SES plays a key role in explaining ethnic differences in health at least between the white groups. However, accounting for SES had little impact on the differences observed in non-white minority ethnic groups. As previously suggested (Nazroo, 2001), this could mean that the SES proxies chosen for this analysis lack sensitivity to fully capture the socio-economic level of minority ethnic groups in Scotland. Material aspects are likely to be captured similarly across ethnic groups but SES proxies might not reflect social status consistently across ethnic groups. Alternatively, other mechanisms might be at play which buffer the effect of SES on health for ethnic minorities. Our findings in relation to the contribution of SES in explaining ethnic differences in health provide some answers but overall direct the research for explanations to other theories.

This thesis also investigated the influence of UK-birth on ethnic inequalities in health. UK-birth was used as a proxy to distinguish migrants from descendants and the majority population. Theories of acculturation and health selection hypotheses, such as the healthy migrant effect, could drive the patterns observed. Accounting for UK-birth had little impact on the patterns of ethnic differences in morbidity observed in Scotland suggesting that being born

in the UK (or not) fails to explain ethnic differences in health and that other mechanisms are involved. Even though accounting for compositional differences in the share of UK-born and non UK-born did not explain ethnic differences in health, processes of health selection and acculturation might still operate to some extent. Indeed, the findings showed that being born outside the UK rather than being born in the UK was overall a strong predictor of reporting better health in the SAH analysis. These findings would still hold in the case of an identity shift in descendants towards reporting their ethnicity as the majority ethnicity because both descendants and the majority ethnic group were born in the UK. Hence, findings support a protective effect of being born outside of the UK on reported health.

This health protective effect of being born outside rather than in the UK found in the SAH analysis aligned with the assumption that migrants are healthier than descendants and majority populations. Although the health of migrants is not compared to that of their peers in the country of origin, the SAH analysis in chapter 4 indirectly supports the “healthy migrant effect” hypothesis and the idea that migrants are selected through their health. In the stratified analysis by adult age groups, the health advantage found in many ethnic groups in younger ages was no longer apparent in those aged 50 years old and over. Indeed, migrants selected through their health at the time of migration are likely to be younger and equipped with the necessary resources to cope with the processes of migration and settlement in the country of destination. However, this selection at arrival is likely to fade off as migrants stay in the country of destination, for multiple reasons. Previous European evidence showed little evidence for a healthy migrant effect (Solé-Auró and Crimmins, 2008). Indeed, the authors of this European research found that migrants aged 50 years and over reported poorer health than native-born populations in most European countries investigated. This is to some extent consistent with the results of chapter 4 suggesting that, as a result of the healthy migrant effect operating, a health advantage might only be seen in younger rather than older migrants.

Furthermore, in the multimorbidity analysis, UK-birth was a weak predictor of the risk of multimorbidity and only in females. The discovery that being born outside the UK rather than in the UK would predict better reported health but not better multimorbidity outcomes might be surprising. However, it is important to remember that although multimorbidity affects more than the elderly, it does primarily affects older people rather than younger one (Barnett et al., 2012). This might explain why findings do not show a protective effect of being born outside the UK on multimorbidity as a healthy migrant effect might be seen in the younger migrants and

fade off in older migrants. This findings point to the conclusion that the mechanisms involved in ethnic inequalities in health depend on different aspects and measures of health investigated as the outcome.

Some of the findings of this research further challenge the healthy migrant effect hypothesis. The stratified analysis by ethnicity and UK-birth showed that Pakistani migrants were in worse health than the White Scottish population and to a greater extent than the Pakistani individuals who were born in the UK. The finding that Pakistani migrants were not healthier than the majority population in Scotland does not support a healthy migrant effect. Interestingly, previous findings in England and Wales showed a persistent SAH disadvantage across migrant generations in the Pakistani population (Harding and Balarajan, 2000, Smith et al., 2009). In these studies, Pakistani migrants did not appear to be healthier either. However, no data are available to know whether Pakistani migrants who moved to the UK were healthier than the Pakistani people who remained in their country of origin. A healthy migrant effect could still operate in the Pakistani population even though they have poorer health than the majority population in the country of destination. Assuming Pakistani migrants might be selected through their health at the time of arrival in the UK, they might also have been exposed to migratory processes on their way to the UK as well as discrimination in their country of residence which might have influenced their health and their reporting of health negatively and to a greater extent than other ethnic groups.

The acculturation hypothesis was explained in chapter 2. The empirical evidence related to the role of acculturation on health was divided, showing both a convergence of the health of descendants towards that of the majority population as well as an emerging or increasing health disadvantage in descendants (Harding and Balarajan, 2001b, Stirbu et al., 2006, Vandenheede et al., 2015, Wallace, 2016). In chapter 4, the stratified analysis by both ethnic and UK-birth showed an overall SAH disadvantage in minority ethnic groups who were born in the UK compared to the native-born White Scottish population. In the case of the Pakistani population, this disadvantage in those who were born in the UK appeared to be less strong than that of Pakistani migrants suggesting a convergence towards the levels of reported health of the White Scottish population. However, overall patterns suggested a disadvantage in descendants which could not be explained by their socio-economic circumstances. In the multimorbidity analysis, this UK-birth and ethnicity stratified analysis showed no uniform patterns suggesting again different mechanisms at play underlying inequalities for different aspects of health.

Traditionally, reasons for a morbidity disadvantage in a particular ethnic group compared to the majority population would be attributed to exposure to greater deprivation, discrimination, poorer health behaviours and worse access to health care services. In contrast, a health advantage, like the mortality advantage, has been thought to be the result of the health selection of migrants, protective health behaviours and community support. In this thesis, Pakistani populations showed both a morbidity disadvantage and a mortality advantage compared to the white Scottish population. The traditional way of hypothesizing explanations for the health disadvantage or advantage seen in minority ethnic groups needs to cater for the extra consideration that health inequalities are health-outcome dependent. Thus, explanations are likely to be more complex than usually hypothesised. For example, most minority ethnic groups show a mortality advantage compared to the White Scottish population in Scotland. These ethnic groups also have similar to better socio-economic status than the majority population. Hence, if we were to assume that SES explains the mortality advantage seen in most minority ethnic groups in Scotland, one can wonder how the same level of SES could also explain the morbidity disadvantage seen in some of these same groups. As a result, understanding any ethnic morbidity-mortality paradox provides a key to greater understanding of the mechanisms underlying ethnic inequalities in health.

This thesis supports a morbidity-mortality paradox in the Pakistani population of Scotland. It showed that Pakistani males and females live longer but in poorer health compared to the White Scottish population in Scotland. The link between reported morbidity and mortality was strong in the Pakistani population of Scotland but regardless of reporting an advantage or a disadvantage in health, they had a mortality advantage compared to the White Scottish population. Understanding this discrepancy between morbidity and mortality is necessary to advance the theories of ethnic health inequalities. Some findings of this thesis help us to understand the Pakistani morbidity-mortality in Scotland. First, the multimorbidity evidence based on hospitalisation data in this thesis showed a morbidity disadvantage in Pakistani populations which supported the morbidity disadvantage found using reported health indicators. Hence, the Pakistani disadvantage in reported health is more likely due to a real morbidity disadvantage than cultural differences in the meaning and reporting of health. Second, this morbidity disadvantage was characterised by a particular disease profile with the greatest risk of hospitalisation due to cardiovascular disease, stroke, diabetes, renal disease, and respiratory disease. The Pakistani population also had a lower risk of hospitalisation due to cancer compared to the majority population. One can notice that, within the morbidity disadvantage observed in

the Pakistani population, inequalities are disease or outcome-dependent and thus, explanations need to account for this variation in outcome.

Building on the empirical evidence provided by this thesis, the likely mechanisms that drive the discrepancy between morbidity and mortality in the Pakistani population can be hypothesised. The analysis has already shown that SES could not explain the patterns observed in the Pakistani group, notably as Pakistani people share relatively similar SES profiles with the White Scottish population in Scotland. This thesis has also demonstrated that both Pakistani migrants and descendants report poorer health and have an advantage in mortality. Hence, the paradox applies across migrant generations in the Pakistani population. Therefore, explanations go beyond the theories tested in this thesis.

One hypothesis is that the morbidity disadvantage in the Pakistani population in Scotland could be due to greater exposure to personal and institutionalised discrimination. Indeed, perceived discrimination has been linked to worse health in the Pakistani population of the UK (Karlsen and Nazroo, 2002). Discrimination is hypothesised to affect health through raised levels of physical and biological stress (Pascoe and Smart Richman, 2009). These higher levels of stress could lead to worse health behaviours and negative biological response which would in turn impact negatively on the health status of individuals. In addition, discrimination in relation to access to services might lead to differential care and thus greater morbidity and severity of disease in the long run. This exposure to discrimination at the individual and institutional level could drive worse morbidity. However, whether this would influence morbidity but not lead to worse mortality is questionable. Therefore, some other mechanisms are likely to protect the Pakistani population from the potential harmful effect of discrimination on their risk of mortality.

A strand of the literature on ethnicity and health aims to understand why South Asians are more likely to have diabetes and cardiovascular disease than their white counterparts (Bhopal, 2013, Kakde et al., 2017, Shah and Kanaya, 2014, Sniderman et al., 2007, Wells, 2008). Some suggest that this is related to their adipose tissue distribution and a differential metabolic activity (Bhopal, 2013, Sniderman et al., 2007, Wells, 2008). Further, this differential adiposity distribution and activity might have been induced by the living environment either as a strategy of 'thrifty' or as a response to infectious diseases (Neel, 1962, Wells, 2008). Hence, the higher risk for the diseases of the metabolic syndrome in the Pakistani population of Scotland could be

biological and environmentally-induced. This propensity to the diseases related to the metabolic syndrome is thought to increase in a food-abundant environment such as contemporary Britain. However, and as previously explained in relation to the discrimination hypothesis, one can wonder why these biological environmentally-induced processes lead to a morbidity disadvantage for these particular diseases but do not lead to worse mortality from these same diseases. Indeed, evidence gathered in chapter 2 showed that South Asians diagnosed with diabetes, renal disease or cardiovascular disease are more likely to survive compared to their white counterparts (Bansal et al., 2013, Barbour et al., 2010, Davis et al., 2014, Khan et al., 2011, Mathur et al., 2018).

A slightly convoluted explanation is that the morbidity disadvantage seen in the Pakistani population could be the results of their greater survival combined with their exposure to discrimination and propensity for specific diseases. If one lives longer, this could increase both the length of time spent with a specific set of diseases but also the chances of developing new health issues over time. This hypothesis, echoing ideas of morbidity expansion, could explain the greater morbidity disadvantage seen in older Pakistani males and females in Scotland. However, with survival selection, we would expect the healthiest to survive. Could the process of survival selection be different in the Pakistani population? The results of longer survival could be that a greater proportion of older Pakistani people are left to live longer in poor health.

Relatedly, we could question why there is such a survival advantage in the Pakistani population of Scotland. A first explanation to the mortality advantage in particular ethnic groups is that there is no mortality advantage and that what we see from the data is not a real phenomenon. Processes of negative health selection have been introduced in chapter 2. Indeed, if an unhealthy return migration and salmon bias were to occur in the Pakistani population of Scotland and this was not recorded in our emigration and mortality data, this would create 'statistically immortal' Pakistani people in the data and consequently contribute to an artificial mortality advantage in this population. However, as previously explained in chapter 4, the Pakistani population is well-settled in the UK and have access to free health care services, thus rendering unlikely their return to Pakistan in the event of illness. Nevertheless, previous evidence in the UK has shown that a salmon bias seems to occur in this population but to an extent too small to explain the mortality advantage observed (Wallace and Kulu, 2018).

If we consider that unrecorded out-migration and subsequent mortality are unlikely to explain the mortality patterns observed in this thesis, one explanation of the mortality advantage seen in the Pakistani population compared to the White Scottish population might lie in their protective health behaviours. Fenelon has shown that the smaller burden of smoking among Hispanics in the US explains a great part of the mortality advantage evidenced in this population (Fenelon, 2013). In the UK, evidence shows that the Pakistani population is less likely to drink alcohol and that females, in particular, are unlikely to smoke (Sproston and Mindell, 2006, Whybrow et al., 2012). This health behaviour profile fits with the lower risk of cancer shown in chapter 6. However, these protective health behaviours do not fit with the higher risk of diabetes, renal disease, cardiovascular disease and respiratory disease seen in the Pakistani population of Scotland. The greater propensity of South Asians for the diseases linked to the metabolic syndrome was previously discussed in this section. Health behaviours might be protective in the long run towards fewer consequences of these diseases and consequently greater survival. However, these health behaviours might not protect against the onset of this metabolic type of diseases, which are probably due to combined biological and environmental pressures.

Social and community support has also been seen as protective of worse health in ethnic minorities but, as explained in chapter 2, evidence for the social network of minority ethnic groups being supportive is not clear-cut in ethnic minorities in the UK and experience of isolation has been reported (Kapadia, 2015, Willis et al., 2013). Similarly, ethnic density is thought to be protective of worse health for minority ethnic groups (Pickett and Wilkinson, 2008) but evidence of the protective effect of own and mixed ethnic density effect on health and health behaviours is mixed in the UK (Bécares et al., 2012b, Bécares et al., 2009, Bécares et al., 2011, Das-Munshi et al., 2010, Feng et al., 2017, Uphoff et al., 2016).

Finally, the healthy migrant effect implies that migrants are selected through their health. Healthier migrants are also likely to be selected for personal traits that makes them more likely to succeed in the process of migration and settlement. If we assume that these specific traits are strongly linked to their better health at arrival, these same traits and resourcefulness characteristics necessary for a successful migration might also give migrants a greater resilience to live and cope with a particular set of diseases. As an extension of the healthy migrant effect, I propose that there is a 'resilient migrant effect' whereby migrants are more likely to overcome health issues due to selective personality traits that also make them more likely to succeed in

the process of migration and settlement in a new country. If such a resilience hypothesis is real, this could explain in part why the Pakistani population lives longer despite experiencing higher morbidity for a particular set of diseases. Anson offers an alternative idea along the same lines (p.192): “it is this hope which may make tolerable otherwise intolerable conditions, and thus reduce the mortality risks” in migrant populations (Anson, 2004). The Pakistani population living in Scotland could well have a set of cultural protective health behaviours along with particular traits which make them more likely to keep on going despite adverse health circumstances.

7.4. Opportunities for future research

As explained in chapter 3, SHELS was the best available data source to investigate the ethnic morbidity-mortality paradox in the Scottish context but the use of other data linkages will permit researchers to further confirm and understand this phenomenon in the UK. ONS-LS data is one data source which was identified as promising for this type of investigation even though it lacks data on more objective measures of morbidity. Further linkages of morbidity and mortality data to the Scottish censuses 2011 or 2021 will provide a better sense of the contemporary nature of the paradox in Scotland and whether this phenomenon persists over time as younger cohorts of the Pakistani population grow older. The effect of unrecorded emigration on mortality outcomes could also be accounted for as previously demonstrated by Wallace and Kulu using ONS-LS data (Wallace and Kulu, 2014a)

Initial evidence of a morbidity-mortality contrast in specific groups in Belgium, France, Australia and the US (Deboosere and Gadeyne, 2005, Hayward et al., 2014, Khlal and Guillot, 2017, Kouris - Blazos, 2002, Markides et al., 1997, Stanaway et al., 2019) needs to be further researched to understand how generalised this morbidity-mortality paradox is in migrants and minority ethnic groups. Hence, the democratisation of linkages of health data to other administrative databases such as censuses or population registers in different countries (e.g. Belgium, Switzerland, or Sweden) is timely and offers further opportunities to test the ethnic morbidity-mortality paradox in other contexts.

In relation to explanations, this thesis found little support for the SES, acculturation and healthy migrant effect hypotheses in explaining the observed ethnic differences in morbidity in Scotland. Ideally, further research should gather the necessary data to test the theories likely to underlie the morbidity-mortality paradox as discussed in section 7.3. For example, data sources should include information on discrimination, exposure to stress, biomarkers and various health

outcomes to assess whether there are differences between the discrimination-morbidity and the discrimination-mortality relationships for particular ethnic groups. Personality traits could also provide information on a resilient migrant phenomenon and how this is related to morbidity trajectories and mortality.

To understand the higher risks of poor health and multimorbidity in the Pakistani population of Scotland, primary and secondary care data in combination would be also useful. Qualitative and quantitative research should aim to disentangle the ethnic differential in access to care that might contribute to the hospitalisation-based multimorbidity patterns observed in this thesis. Whether primary and secondary care are utilised similarly for different ethnic groups and for the same conditions would be a first step to further explore the extent of ethnic inequalities in multimorbidity. For example, people with a particular disease could be identified through census or disease registry information and their health trajectories and use of health services followed to capture differences in health care usage.

Further work is also required to verify the accuracy of the mortality data for minority ethnic groups. One avenue to consider in order to follow people across countries in older ages is to investigate pension data and whether people move back to their country of origin and die abroad. This type of investigation has been done in France (unpublished) to explore the likelihood of the salmon bias and to assess whether an unhealthy return migration could explain the mortality advantage seen in some migrant groups in France. Investigating pension data along these lines would be possible in the UK context assuming access to the Department for Work and Pensions data can be granted.

An alternative avenue to provide clues on the extent of emigration and unhealthy return migration would be to link the SHELS cohort used in this thesis to the Scottish census 2011. This would permit the exploration of whether people disappeared between censuses and whether remaining or not in Scotland is differentially predicted by the reported health status declared in the Scottish census 2001. Census linkage between Scotland and England and Wales would also provide the ability to deepen our understanding of migration processes and how this is linked to health.

This thesis used cross-sectional and longitudinal approaches to explore the morbidity-mortality contrast in particular ethnic groups compared to the majority population. This contrast calls for future research to investigate how ethnic inequalities are shaped over time, how

morbidity occurs and then contributes to the mortality outcome. This would give clues on how a discrepancy in outcome might occur. Along with this idea of following the health trajectories of individuals, future research should also aim to adopt a life course approach to understand how individual trajectories contribute to different health experiences and ultimately how they influence the creation of health inequalities between different ethnicities. Ideally, people would be followed from birth with a prospective approach in order to give researchers the opportunity to capture various life events and exposure to difficult circumstances from early on in life; such a study would also need a long follow-up to capture health issues and outcomes happening later in life. However, gathering data for a long time period for different cohorts of migrant groups who have lived in different countries is a tremendous challenge.

Finally, chapter 5 showed that accounting for reported health did not change the observed patterns of ethnic differences in mortality. In other words, reported health did not mediate the relationship between ethnicity and mortality. Similarly, chapter 6 showed that overall reported health did not mediate the relationship between ethnicity and multimorbidity. These findings point to the non-equivalence of health indicators. Future research should consider how the health outcome investigated fits within a gradient of health, what level of severity is measured and what aspect of health is considered.

7.5. Concluding remarks

This thesis has shown the importance of bridging the morbidity and the mortality strands of scholarship in order to advance understandings of ethnic inequalities in health. In Scotland, gathering morbidity and mortality data permitted the identification of a morbidity-mortality contrast in the Pakistani population whereby they live longer but in poorer health than the majority population. Although some of the literature on the health of migrants and ethnic groups hints at such a phenomenon, this is the first time a morbidity-mortality paradox has been demonstrated in a particular ethnic group and from a unique data source. This research was made possible by the use of data linkage of various health data to the Scottish Census 2001. This research demonstrates the usefulness of data linkage and access to national level population data. Governments should contribute to the facilitation of access to key data sources for researchers to continue understanding health inequalities across different social groups so the knowledge gained can be fed into action plans aiming to improve the health of their population.

The morbidity-mortality contrast found in the Pakistani population was striking. For example, Pakistani females had the longest life expectancy at birth in Scotland (Gruer et al., 2016) but also the longest number of years in poor health (20.4 years versus 8.7 years in White Scottish females). The morbidity disadvantage seen in the Pakistani population using self-assessed measures of health was confirmed by the multimorbidity findings presented in this thesis. Pakistani males and females were more likely to be multimorbid based on hospitalisation data than White Scottish males and females. The diseases underlying this multimorbidity disadvantage were mainly related to the metabolic syndrome. Furthermore, this morbidity disadvantage applied to both Pakistani migrants and descendants but to a greater extent in Pakistani migrants. Pakistanis aged 50 years and above were particularly disadvantaged in terms of reported health compared to their White Scottish counterparts.

The morbidity-mortality contrast was very clear in the Pakistani population in Scotland but it also appeared to some extent in the Indian population and particularly so in females. The Indian population of Scotland was also shown to have an advantageous socioeconomic profile but once this was taken into account, they showed a marked morbidity disadvantage compared to the White Scottish population. Findings for the other ethnic groups did not provide an indication of contrasting morbidity-mortality patterns. In the Other White British, Other White and Chinese groups, advantageous reporting of health and multimorbidity risks aligned with their mortality patterns. In the white groups, their reported health advantage compared to the White Scottish population disappeared when accounting for their advantageous socio-economic profile. In the Chinese group, their morbidity advantage persisted after accounting for their socio-economic circumstances and across migrant generations. Hence, different ethnic groups experience different health profiles and for different reasons.

One issue of the findings of this thesis is that the morbidity-mortality paradox brings the focus to a particular ethnic group i.e. the Pakistani population. A potential consequence of the focus on this particular group is the racialization of ethnicity and Pakistani population rather than considering the reasons underlying the disadvantage faced by this population. The question of why the Pakistani population lives longer but in poorer health needs to be understood so that it contributes to our understanding of the meaning of ethnicity in researching health inequalities, and how useful ethnicity is for understanding health inequalities.

Reasons for the discrepancy in outcome has been discussed in section 7.3 and further work is required to understand the mechanisms underlying the morbidity-mortality contrast.

This opens up a range of future avenues to research this phenomenon as explained in section 7.4. Both qualitative and quantitative methodologies have the ability to provide a deeper understanding of the phenomenon. However, in relation to quantitative research, advancing knowledge will only be possible through access to new linkages and key data sources.

Finally, the morbidity disadvantage seen in the Pakistani population in Scotland and to some extent in the Indian population is particularly worrying as members of these ethnic groups are likely to spend many years of life in poorer health than their contemporaries of other ethnic groups. This has the potential to impact on health expenditures at local levels. The current NHS Scotland Resource Allocation Committee formula includes poverty and deprivation as key elements to allocate funds to each health board. It is constructed from smaller geographical units for better precision in predicting needs and also accounts for higher relative needs of the very old and very young. In light of the current findings, it appears essential to also accommodate for the health needs of different ethnic groups in Scotland. The current formula would benefit from an update on this aspect. More generally, policy makers should aim to improve the quality of life of these populations in Scotland. Whether the morbidity findings reflect differential access to care needs to be addressed. Policy makers should ensure that fair and culturally-adapted care is provided in primary and secondary care settings while the root causes of the morbidity-mortality paradox are further pinpointed. Monitoring the delivery of care provided, through the onset and progression of the diseases particularly prevalent in these populations, will give the National Health Service the tools to ensure fair and equal access to care and the opportunity to reach policy equality targets.

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APPENDICES

Chapter 3 appendices

Appendix 3.1. Sensitivity analysis including car ownership and occupation for SES adjustment in the model exploring ethnic differences in self-reported health

a- Sensitivity analysis adding car ownership

The numerators, denominators and models are based on complete case analysis i.e. when the 4 SES indicators (household tenure, combined education, SIMD and **car ownership**) are not missing.

Sex and ethnic groups	N	Reported Bad health	Adjusted for Age and 3 SES (household tenure, combined education, SIMD)		Adjusted for Age and 3 SES + car ownership	
			RR (95% CI)	p-value	RR (95% CI)	p-value
MALES						
White Scottish	1838305	163755	1		1	
Other White British	147675	11165	1.02 (0.99, 1.04)	0.2223	1.04 (1.01, 1.06)	0.0029
White Irish	18750	2465	1.13 (1.09, 1.18)	<.0001	1.10 (1.06, 1.15)	<.0001
Other White	26890	1825	0.99 (0.95, 1.04)	0.7739	0.99 (0.94, 1.03)	0.5399
Any Mixed Background	5090	300	1.34 (1.20, 1.49)	<.0001	1.29 (1.15, 1.43)	<.0001
Indian	6075	405	1.30 (1.19, 1.43)	<.0001	1.27 (1.16, 1.39)	<.0001
Pakistani	12420	1060	1.60 (1.51, 1.70)	<.0001	1.66 (1.56, 1.76)	<.0001
Bangladeshi	825	50	1.04 (0.80, 1.33)	0.7913	1.00 (0.77, 1.29)	0.9805
Other South Asian	2550	175	1.08 (0.94, 1.24)	0.2636	1.05 (0.91, 1.21)	0.5156
Caribbean	655	45	0.96 (0.73, 1.26)	0.7620	0.93 (0.71, 1.22)	0.5932
African	1940	90	0.75 (0.62, 0.92)	0.0044	0.72 (0.59, 0.87)	0.0009
Black Scottish/Other	430	40	1.14 (0.86, 1.51)	0.3557	1.08 (0.82, 1.43)	0.5682
Chinese	6030	230	0.65 (0.58, 0.74)	<.0001	0.65 (0.58, 0.74)	<.0001
FEMALES						
White Scottish	1941805	185880	1		1	
Other White British	155560	12840	1.02 (1.00, 1.04)	0.0327	1.04 (1.01, 1.06)	0.0009
White Irish	20245	2675	1.11 (1.07, 1.15)	<.0001	1.10 (1.06, 1.14)	<.0001
Other White	32170	2240	0.99 (0.95, 1.03)	0.7082	0.99 (0.95, 1.03)	0.5543
Any Mixed Background	5480	340	1.22 (1.11, 1.34)	<.0001	1.19 (1.08, 1.31)	0.0003
Indian	5550	485	1.61 (1.48, 1.74)	<.0001	1.60 (1.47, 1.74)	<.0001
Pakistani	12250	1310	1.95 (1.85, 2.06)	<.0001	2.00 (1.89, 2.10)	<.0001
Bangladeshi	680	50	1.41 (1.10, 1.81)	0.0074	1.39 (1.08, 1.79)	0.0097
Other South Asian	2140	175	1.23 (1.07, 1.41)	0.0029	1.21 (1.06, 1.39)	0.0055
Caribbean	720	60	1.20 (0.94, 1.52)	0.1454	1.19 (0.93, 1.51)	0.1607
African	1700	85	0.81 (0.66, 0.99)	0.0376	0.79 (0.65, 0.96)	0.0189
Black Scottish/Other	445	45	1.24 (0.96, 1.61)	0.1050	1.23 (0.95, 1.60)	0.1193
Chinese	6165	295	0.78 (0.70, 0.87)	<.0001	0.78 (0.70, 0.87)	<.0001

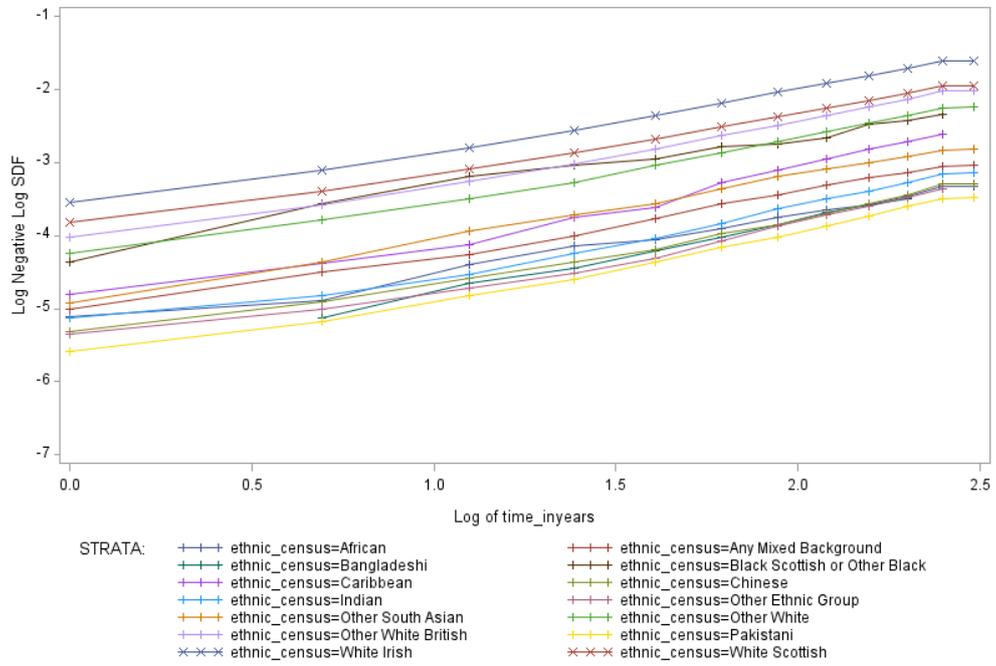
b- Sensitivity analysis adding occupation

The numerators, denominators and models are based on complete case analysis i.e. when the 4 SES indicators (household tenure, combined education, SIMD and **occupation (NS-SeC)**) are not missing.

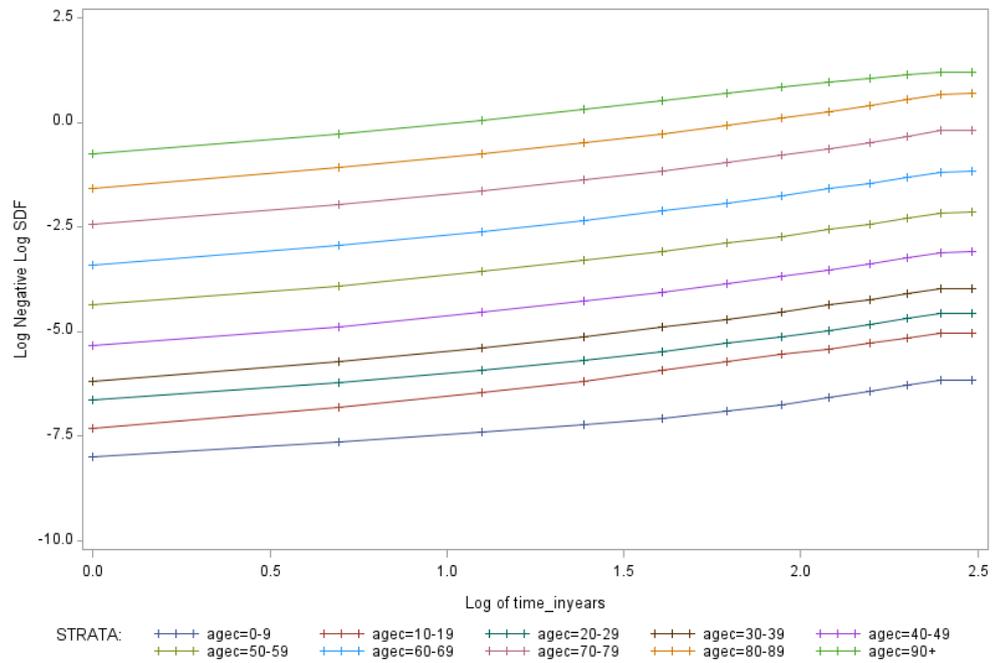
Sex and ethnic groups	N	Reported Bad health	Adjusted for Age and 3 SES (household tenure, combined education, SIMD)		Adjusted for Age and 3 SES + occupation (NS-SeC)	
			RR (95% CI)	p-value	RR (95% CI)	p-value
MALES						
White Scottish	1670510	114615	1		1	
Other White British	134135	8250	1.01 (0.99, 1.04)	0.2933	1.02 (0.99, 1.05)	0.1290
White Irish	16130	1650	1.13 (1.08, 1.18)	<.0001	1.13 (1.08, 1.18)	<.0001
Other White	24915	1390	0.96 (0.91, 1.02)	0.1570	0.97 (0.93, 1.03)	0.3244
Any Mixed Background	4635	230	1.40 (1.24, 1.59)	<.0001	1.39 (1.23, 1.57)	<.0001
Indian	5925	360	1.34 (1.22, 1.48)	<.0001	1.33 (1.21, 1.46)	<.0001
Pakistani	12565	1000	1.70 (1.61, 1.80)	<.0001	1.64 (1.55, 1.74)	<.0001
Bangladeshi	810	45	1.12 (0.87, 1.46)	0.3827	1.11 (0.86, 1.44)	0.4202
Other South Asian	2365	155	1.22 (1.05, 1.42)	0.0082	1.17 (1.01, 1.36)	0.0425
Caribbean	640	35	0.92 (0.68, 1.24)	0.5703	0.90 (0.66, 1.21)	0.4735
African	1790	85	0.89 (0.73, 1.09)	0.2782	0.89 (0.73, 1.09)	0.2457
Black Scottish/Other	400	30	1.28 (0.95, 1.72)	0.1040	1.28 (0.96, 1.72)	0.0963
Chinese	5815	215	0.72 (0.63, 0.82)	<.0001	0.70 (0.62, 0.80)	<.0001
FEMALES						
White Scottish	1705845	133550	1		1	
Other White British	136705	9720	1.02 (0.99, 1.04)	0.1591	1.02 (1.00, 1.04)	0.1148
White Irish	16825	1845	1.10 (1.05, 1.15)	<.0001	1.09 (1.05, 1.14)	<.0001
Other White	29095	1680	0.94 (0.89, 0.98)	0.0059	0.94 (0.89, 0.98)	0.0065
Any Mixed Background	4930	250	1.25 (1.11, 1.40)	0.0001	1.23 (1.09, 1.38)	0.0005
Indian	5465	455	1.66 (1.52, 1.80)	<.0001	1.54 (1.41, 1.68)	<.0001
Pakistani	12355	1285	2.06 (1.96, 2.17)	<.0001	1.75 (1.66, 1.85)	<.0001
Bangladeshi	665	50	1.45 (1.12, 1.89)	0.0054	1.28 (0.98, 1.66)	0.0686
Other South Asian	1915	150	1.43 (1.24, 1.66)	<.0001	1.31 (1.13, 1.52)	0.0003
Caribbean	675	50	1.20 (0.92, 1.56)	0.1908	1.19 (0.92, 1.56)	0.1897
African	1515	70	0.83 (0.67, 1.04)	0.0998	0.80 (0.64, 0.99)	0.0414
Black Scottish/Other	410	35	1.19 (0.88, 1.62)	0.2658	1.16 (0.85, 1.58)	0.3521
Chinese	5840	265	0.81 (0.73, 0.91)	0.0004	0.77 (0.69, 0.87)	<.0001

Appendix 3.2. Cumulative log-log plots for each variable included in Cox model to check that the proportional hazard assumptions are met

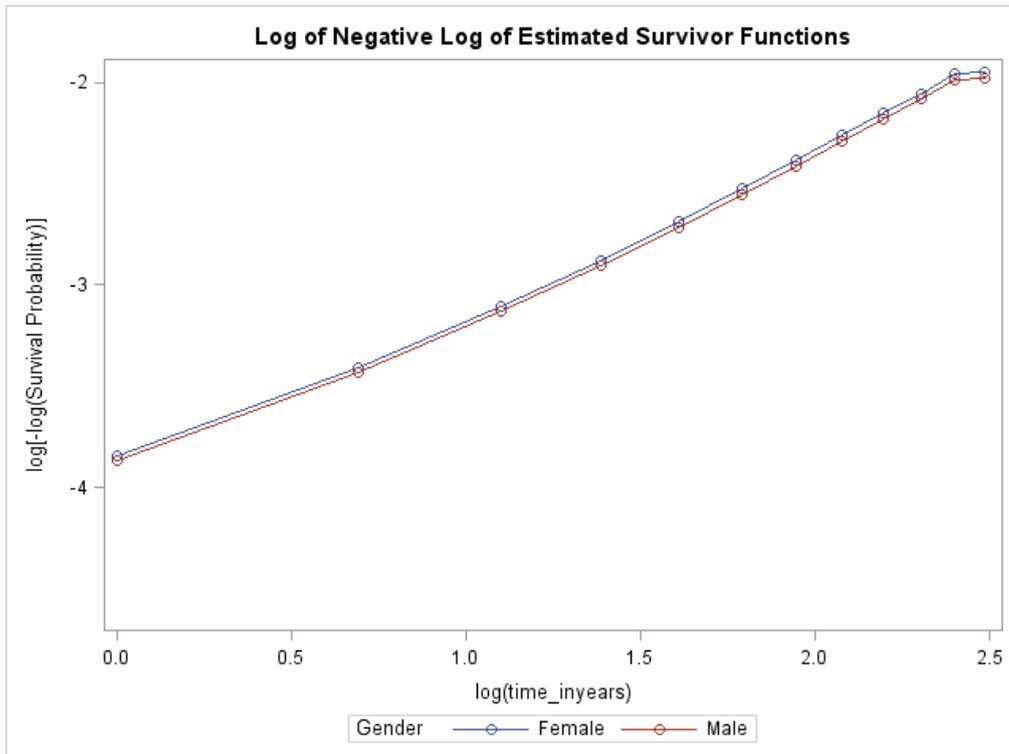
a- Ethnicity



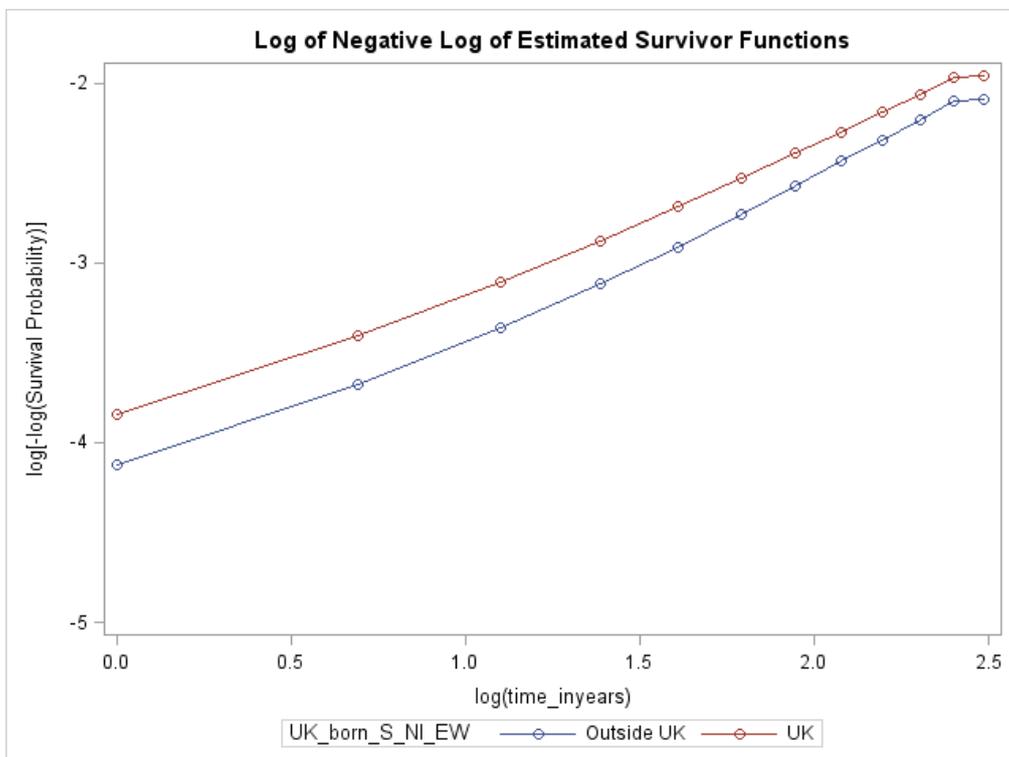
b- Age



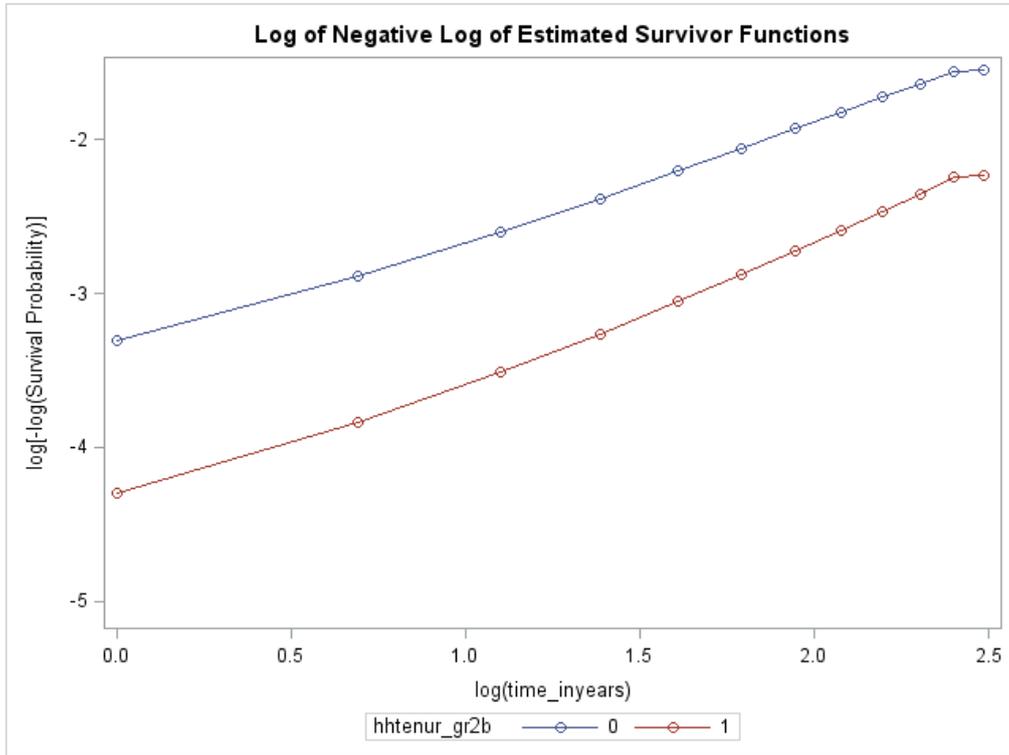
c- Sex



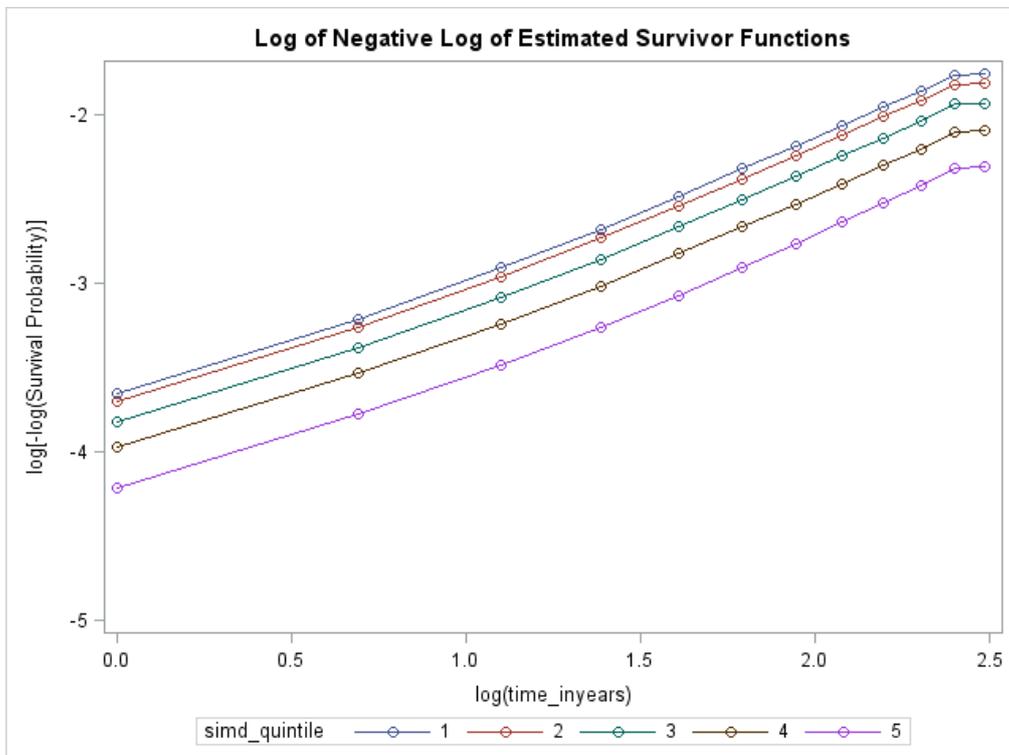
d- UK-birth



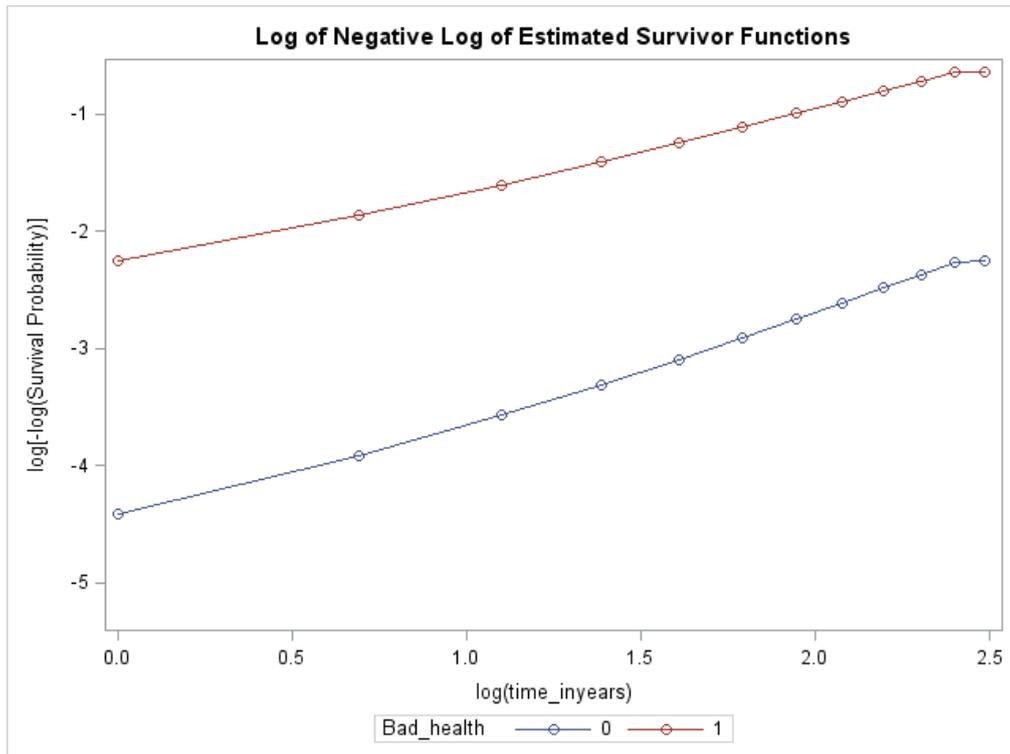
e- Household tenure



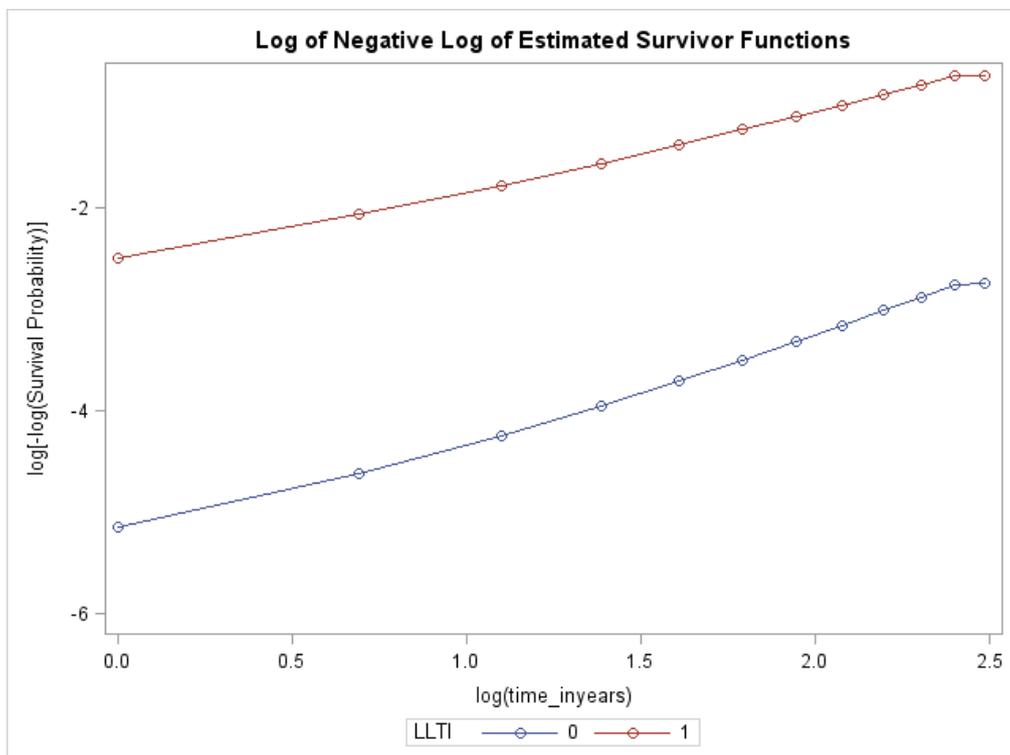
f- SIMD



g- SRH (bad health)



h- LLTI



Appendix 3.3. Sensitivity analysis testing the interaction between ethnicity and sex in the model exploring ethnic differences in self-reported health

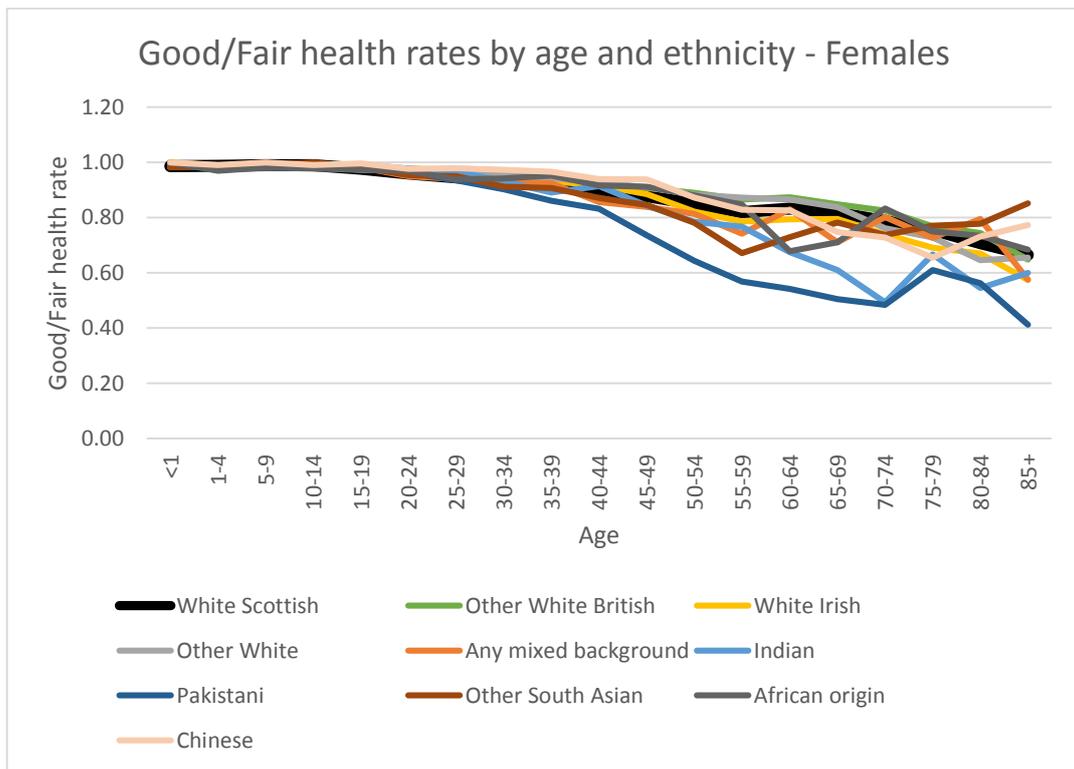
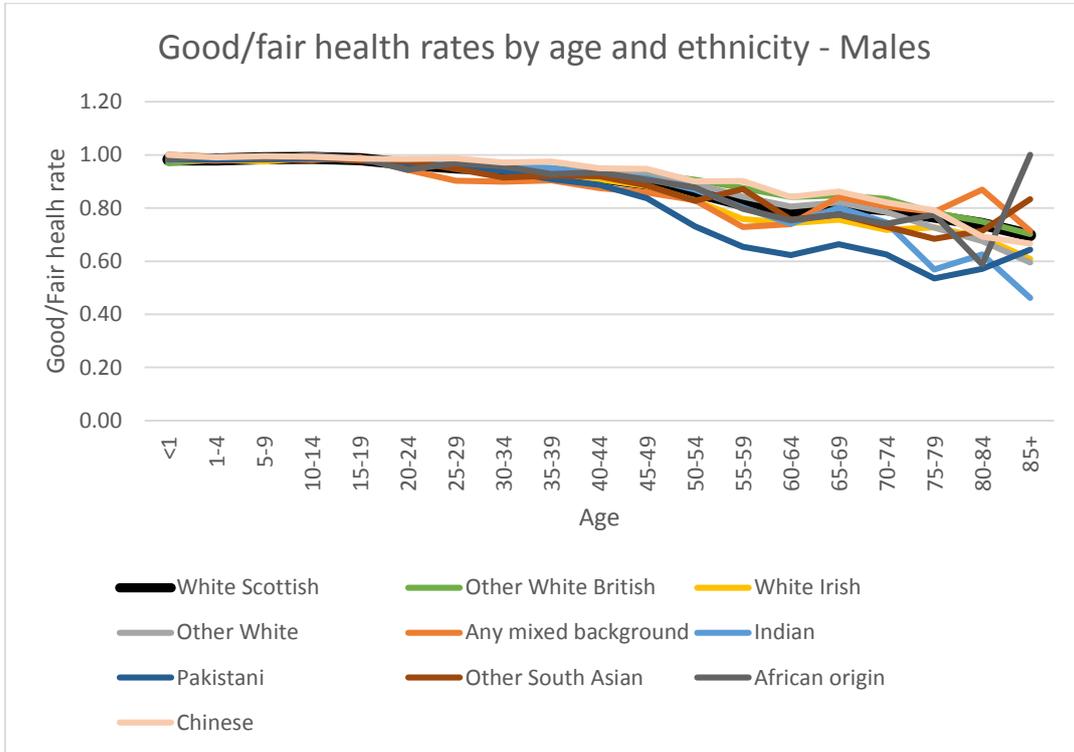
	N	Reported Bad health	Adjusted for Age, sex and ethnicity*sex interaction	
			RR (95% CI)	p-value
White Scottish	367595	3882790	1	
Other White British	25335	316175	0.74 (0.69, 0.79)	<.0001
White Irish	5400	40635	1.17 (1.10, 1.25)	<.0001
Other White	4370	63030	0.85 (0.80, 0.91)	<.0001
Any Mixed Background	655	10930	1.36 (1.21, 1.52)	<.0001
Indian	930	12280	0.95 (0.86, 1.05)	0.2297
Pakistani	2425	25595	1.45 (1.35, 1.56)	<.0001
Bangladeshi	105	1565	1.02 (0.79, 1.33)	0.9813
Other South Asian	355	4875	1.07 (0.92, 1.24)	0.5487
Caribbean	110	1455	0.92 (0.70, 1.19)	0.3888
African	195	3895	0.86 (0.71, 1.05)	0.0711
Black Scottish/Other	85	905	1.34 (1.00, 1.79)	0.0747
Chinese	550	13100	0.58 (0.51, 0.66)	<.0001
Sex (Female versus Male)			1.04 (0.98, 1.10)	0.1994
Other White British* female			1.07 (0.99, 1.16)	0.0946
White Irish* female			0.91 (0.84, 0.99)	0.0310
Other White* female			0.96 (0.88, 1.05)	0.3892
Any Mixed Background* female			0.91 (0.77, 1.06)	0.2262
Indian* female			1.33 (1.16, 1.52)	<.0001
Pakistani* female			1.30 (1.18, 1.43)	<.0001
Bangladeshi* female			1.32 (0.91, 1.92)	0.1417
Other South Asian* female			1.18 (0.96, 1.45)	0.1176
Caribbean* female			1.12 (0.78, 1.61)	0.5309
African* female			1.01 (0.76, 1.33)	0.9614
Black Scottish/Other* female			1.01 (0.68, 1.51)	0.9492
Chinese* female			1.21 (1.02, 1.43)	0.0302

Appendix 3.4. Sensitivity analysis adjusting for different form of age in the model exploring ethnic differences in self-reported health

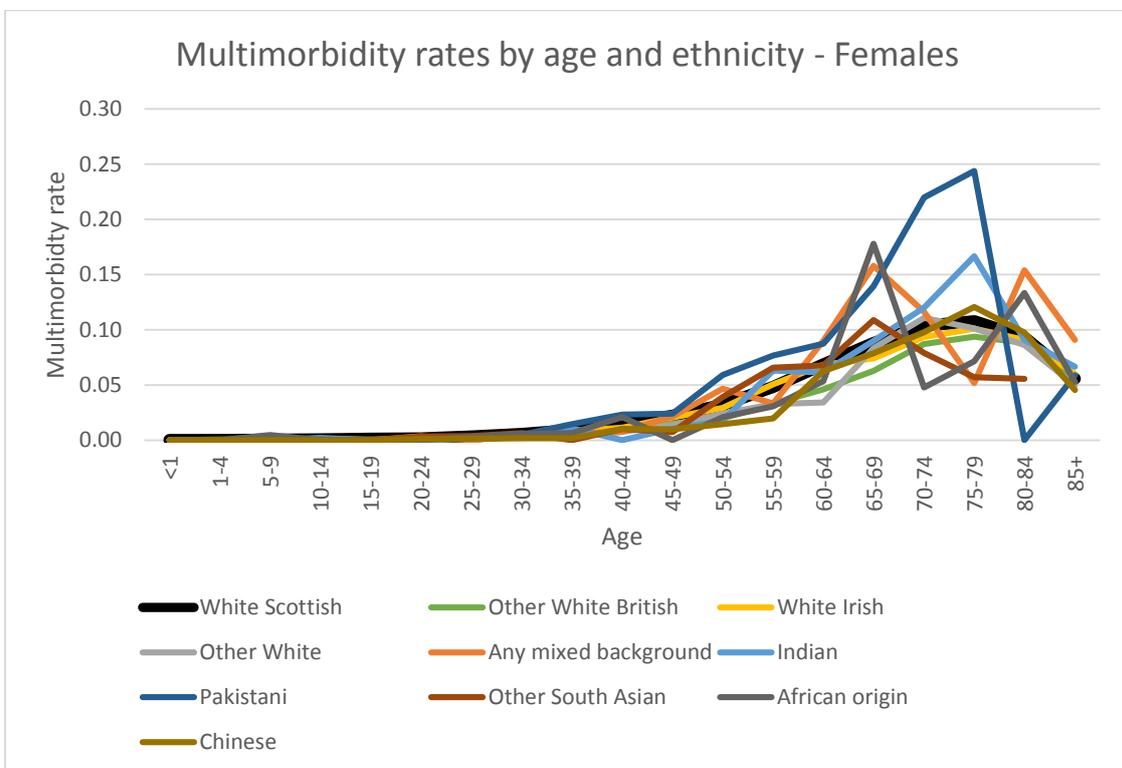
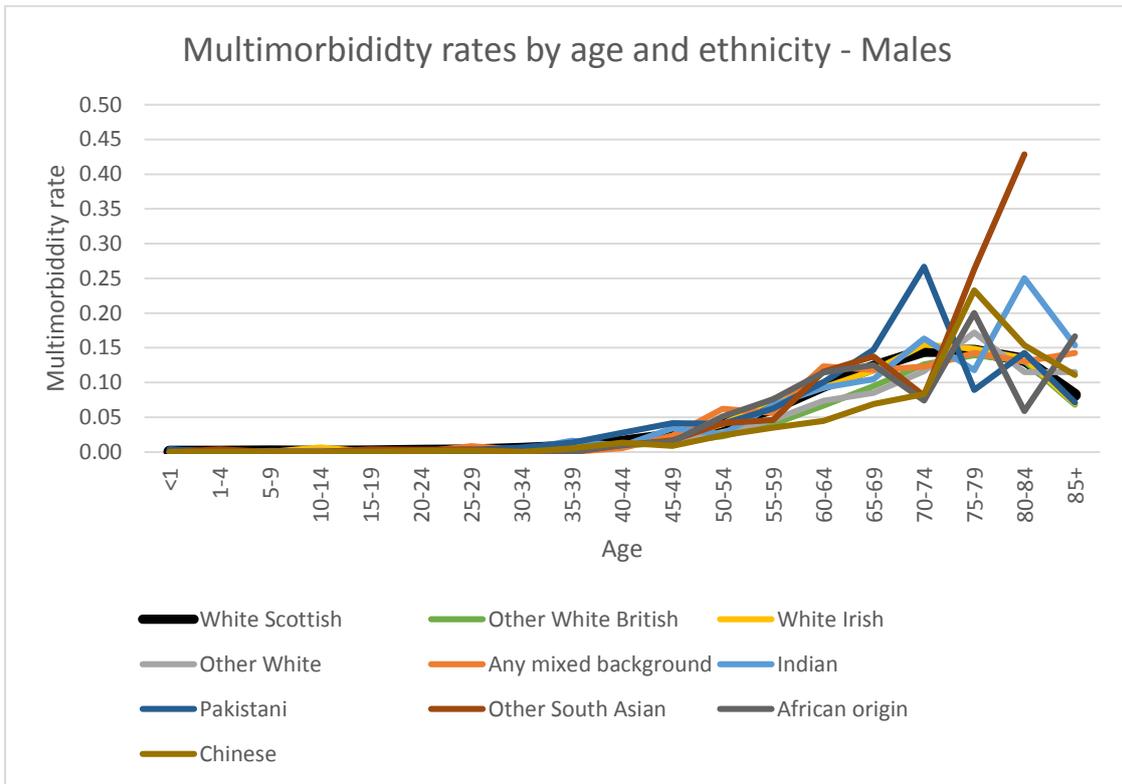
Sex and ethnic groups	N	Reported Bad health	Age categorical (1 year age band)	Age categorical (5 years age band)	Age categorical (10 years age band)	Age continuous	Age continuous and age-squared
MALES							
White Scottish	1887415	170395	1	1	1	1	1
Other White British	153805	11645	0.74 (0.69, 0.79)	0.74 (0.64, 0.85)	0.74 (0.61, 0.90)	0.76 (0.71, 0.81)	0.74 (0.70, 0.79)
White Irish	19465	2565	1.17 (1.10, 1.25)	1.17 (1.04, 1.33)	1.18 (0.97, 1.43)	1.18 (1.11, 1.26)	1.18 (1.10, 1.26)
Other White	28835	1980	0.85 (0.80, 0.91)	0.86 (0.76, 0.97)	0.86 (0.73, 1.01)	0.85 (0.79, 0.91)	0.86 (0.80, 0.92)
Any Mixed Background	5260	310	1.36 (1.21, 1.52)	1.35 (1.16, 1.58)	1.34 (1.10, 1.62)	1.25 (1.11, 1.41)	1.36 (1.21, 1.53)
Indian	6425	425	0.95 (0.86, 1.05)	0.95 (0.82, 1.10)	0.95 (0.78, 1.15)	0.96 (0.86, 1.06)	0.96 (0.86, 1.06)
Pakistani	12905	1080	1.45 (1.35, 1.56)	1.45 (1.29, 1.63)	1.44 (1.23, 1.69)	1.43 (1.33, 1.54)	1.45 (1.35, 1.56)
Bangladeshi	860	50	1.02 (0.79, 1.33)	1.02 (0.76, 1.36)	1.01 (0.74, 1.37)	1.00 (0.77, 1.30)	1.02 (0.78, 1.32)
Other South Asian	2670	180	1.07 (0.92, 1.24)	1.06 (0.87, 1.30)	1.06 (0.83, 1.35)	1.07 (0.92, 1.25)	1.06 (0.91, 1.23)
Caribbean	700	50	0.92 (0.70, 1.19)	0.91 (0.69, 1.20)	0.92 (0.69, 1.23)	0.91 (0.70, 1.19)	0.90 (0.69, 1.18)
African	2100	105	0.86 (0.71, 1.05)	0.86 (0.66, 1.12)	0.85 (0.61, 1.18)	0.88 (0.72, 1.07)	0.86 (0.71, 1.04)
Black Scottish/Other	445	40	1.34 (1.00, 1.79)	1.35 (0.99, 1.84)	1.34 (0.94, 1.90)	1.29 (0.96, 1.73)	1.34 (1.00, 1.78)
Chinese	6500	240	0.58 (0.51, 0.66)	0.58 (0.49, 0.68)	0.57 (0.47, 0.69)	0.57 (0.50, 0.65)	0.57 (0.50, 0.65)
FEMALES							
White Scottish	1995375	197200	1	1	1	1	1
Other White British	162370	13690	0.79 (0.75, 0.83)	0.79 (0.70, 0.88)	0.79 (0.68, 0.92)	0.81 (0.77, 0.85)	0.80 (0.76, 0.84)
White Irish	21170	2835	1.06 (1.01, 1.12)	1.07 (0.96, 1.18)	1.07 (0.91, 1.26)	1.08 (1.02, 1.14)	1.08 (1.02, 1.14)
Other White	34200	2390	0.79 (0.75, 0.84)	0.79 (0.71, 0.89)	0.79 (0.68, 0.92)	0.81 (0.76, 0.86)	0.81 (0.76, 0.86)
Any Mixed Background	5675	345	1.21 (1.08, 1.34)	1.20 (1.04, 1.39)	1.19 (1.00, 1.42)	1.12 (1.00, 1.25)	1.20 (1.07, 1.33)
Indian	5855	505	1.23 (1.12, 1.35)	1.23 (1.06, 1.42)	1.22 (1.02, 1.47)	1.25 (1.14, 1.38)	1.26 (1.15, 1.38)
Pakistani	12690	1345	1.84 (1.72, 1.96)	1.83 (1.63, 2.06)	1.81 (1.56, 2.12)	1.80 (1.67, 1.93)	1.85 (1.73, 1.98)
Bangladeshi	700	50	1.30 (1.00, 1.70)	1.30 (0.98, 1.73)	1.28 (0.95, 1.72)	1.28 (0.98, 1.67)	1.34 (1.03, 1.74)
Other South Asian	2205	175	1.21 (1.05, 1.40)	1.21 (1.02, 1.44)	1.20 (0.99, 1.47)	1.21 (1.05, 1.40)	1.23 (1.07, 1.42)
Caribbean	755	60	0.98 (0.77, 1.26)	0.98 (0.75, 1.29)	0.98 (0.73, 1.34)	1.02 (0.80, 1.31)	1.01 (0.79, 1.29)
African	1795	90	0.82 (0.67, 1.00)	0.81 (0.65, 1.02)	0.81 (0.62, 1.05)	0.83 (0.68, 1.02)	0.84 (0.69, 1.03)
Black Scottish/Other	460	45	1.31 (0.99, 1.72)	1.30 (0.97, 1.75)	1.29 (0.94, 1.78)	1.30 (0.98, 1.71)	1.31 (0.99, 1.72)
Chinese	6600	310	0.67 (0.60, 0.75)	0.67 (0.57, 0.78)	0.67 (0.55, 0.81)	0.68 (0.61, 0.77)	0.68 (0.61, 0.77)

Appendix 3.5. The age distribution of self-assessed health, multimorbidity and mortality by ethnicity and sex

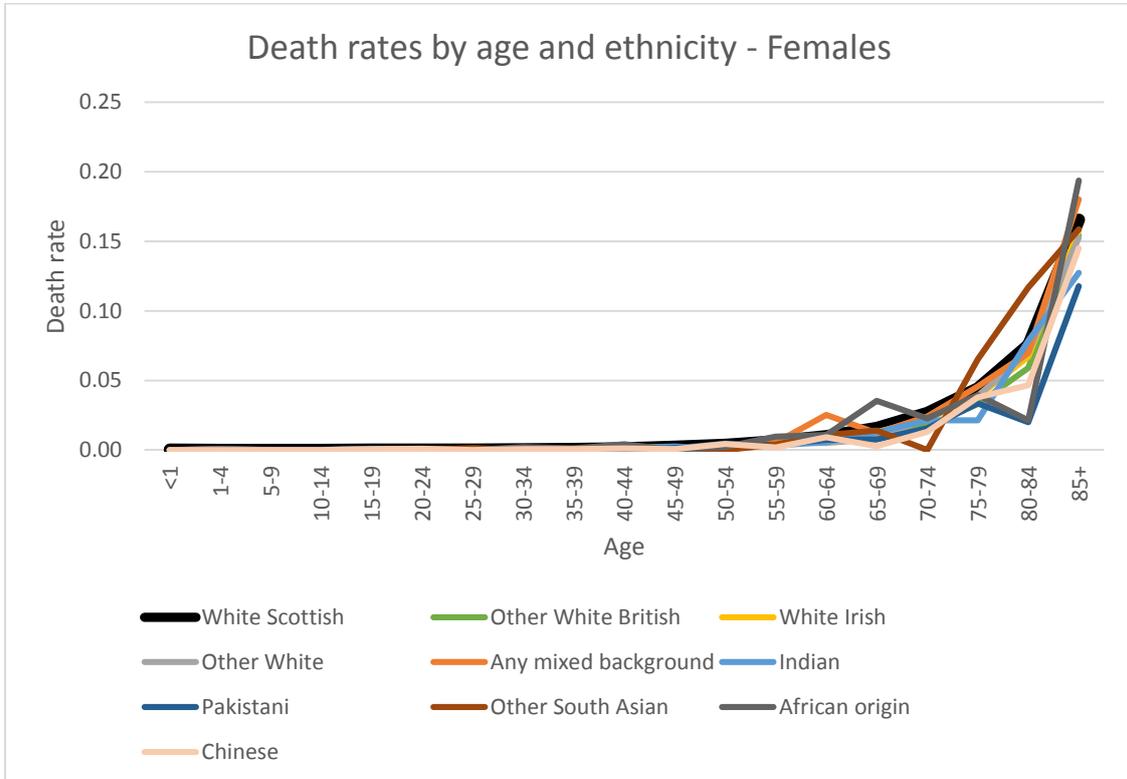
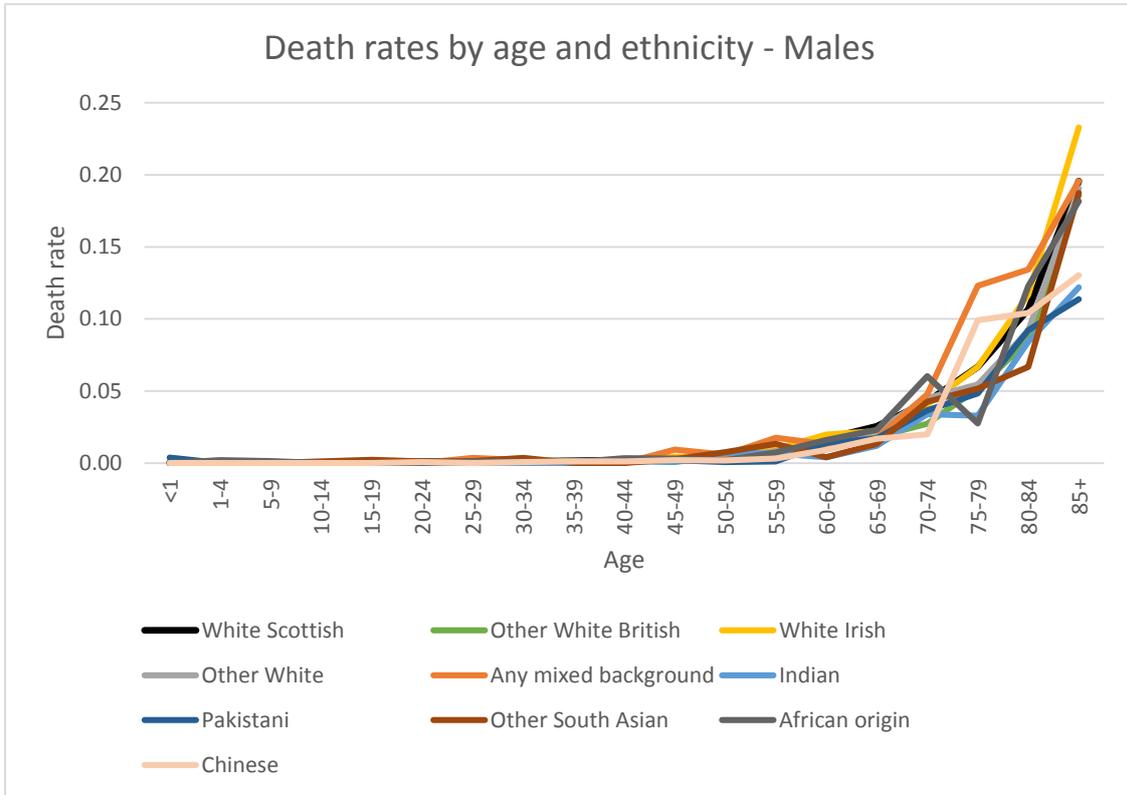
a- Report of good/fair health (SAH) in the SHELS census 2001 data by age and ethnicity, for males and females.



b- Multimorbidity rates (May 2001- April 2013) by age and ethnicity, for males and females.



c- Mortality rates (May 2001- April 2013) by age and ethnicity, for males and females.



Chapter 4 appendices

Appendix 4.1- Risk Ratios (RR) and 95% confidence intervals (CI) of reporting Bad/Fair health in 2001 by ethnicity, stratified by sex. Models are adjusted for age and adjusted for age and SES

Sex and ethnic groups	N	Reported Bad/Fair health	Adjusted for Age		Adjusted for Age and SES	
			RR (95% CI)	p-value	RR (95% CI)	p-value
MALES						
White Scottish	1887415	531000	1		1	
Other White British	153805	39655	0.84 (0.81, 0.87)	<.0001	1.00 (0.98, 1.01)	0.4434
White Irish	19465	6770	1.04 (1.01, 1.08)	0.0113	1.03 (1.01, 1.06)	0.0017
Other White	28835	6710	0.89 (0.86, 0.92)	<.0001	0.97 (0.95, 0.99)	0.0110
Any Mixed Background	5260	1060	1.16 (1.10, 1.23)	<.0001	1.17 (1.11, 1.23)	<.0001
Indian	6425	1570	1.04 (0.99, 1.09)	0.1658	1.24 (1.18, 1.29)	<.0001
Pakistani	12905	3770	1.41 (1.36, 1.46)	<.0001	1.46 (1.42, 1.50)	<.0001
Bangladeshi	860	255	1.40 (1.27, 1.54)	<.0001	1.41 (1.28, 1.55)	<.0001
Other South Asian	2670	625	1.05 (0.98, 1.13)	0.1483	1.07 (1.00, 1.14)	0.0615
Caribbean	700	155	0.88 (0.77, 1.01)	0.0587	0.89 (0.78, 1.01)	0.0757
African	2100	380	0.85 (0.77, 0.94)	0.0014	0.83 (0.76, 0.91)	<.0001
Black Scottish/Other	445	115	1.19 (1.02, 1.37)	0.0240	1.08 (0.93, 1.24)	0.3209
Chinese	6500	1460	1.01 (0.96, 1.06)	0.8014	1.07 (1.02, 1.12)	0.0049
FEMALES						
White Scottish	1995375	652820	1		1	
Other White British	162370	49385	0.87 (0.85, 0.90)	<.0001	1.01 (1.00, 1.02)	0.0917
White Irish	21170	7995	0.96 (0.93, 0.99)	0.0072	0.99 (0.97, 1.01)	0.4559
Other White	34200	9095	0.86 (0.84, 0.89)	<.0001	0.98 (0.95, 1.00)	0.0209
Any Mixed Background	5675	1295	1.09 (1.03, 1.15)	0.0036	1.11 (1.06, 1.16)	<.0001
Indian	5855	1765	1.14 (1.09, 1.20)	<.0001	1.32 (1.27, 1.38)	<.0001
Pakistani	12690	4275	1.45 (1.40, 1.51)	<.0001	1.50 (1.46, 1.53)	<.0001
Bangladeshi	700	240	1.51 (1.36, 1.67)	<.0001	1.52 (1.37, 1.68)	<.0001
Other South Asian	2205	610	1.10 (1.03, 1.18)	0.0065	1.11 (1.04, 1.19)	0.0017
Caribbean	755	220	1.00 (0.90, 1.12)	0.9697	1.09 (0.98, 1.21)	0.1046
African	1795	370	0.86 (0.78, 0.94)	0.0008	0.84 (0.77, 0.92)	0.0001
Black Scottish/Other	460	135	1.09 (0.96, 1.25)	0.1889	1.04 (0.91, 1.18)	0.5495
Chinese	6600	1685	0.97 (0.92, 1.02)	0.1796	1.05 (1.00, 1.09)	0.0327

Appendix 4.2a- Risk Ratios (RR) and 95% confidence intervals (CI) of reporting bad health in 2001 by ethnicity, for males. Models are adjusted for age, for age and UK-birth and for age, UK-birth and the interaction between UK-birth and ethnicity

	N	Reported Bad health	Adjusted for Age		Adjusted for Age and UK- birth		Adjusted for Age, UK-birth and ethnicity*UK-birth	
			RR (95% CI)	p-value	RR (95% CI)	p-value	RR (95% CI)	p-value
White Scottish	1887415	170395	1		1		1	
Other White British	153805	11645	0.74 (0.69, 0.79)	<.0001	0.74 (0.70, 0.79)	<.0001	0.74 (0.69, 0.79)	<.0001
White Irish	19465	2565	1.17 (1.10, 1.25)	<.0001	1.27 (1.19, 1.35)	<.0001	1.15 (1.06, 1.24)	0.0005
Other White	28835	1980	0.85 (0.80, 0.91)	<.0001	0.98 (0.92, 1.05)	0.5629	1.21 (1.11, 1.33)	<.0001
Any Mixed Background	5260	310	1.36 (1.21, 1.52)	<.0001	1.44 (1.28, 1.61)	<.0001	1.52 (1.34, 1.73)	<.0001
Indian	6425	425	0.95 (0.86, 1.05)	0.3136	1.10 (0.99, 1.22)	0.0682	1.02 (0.84, 1.25)	0.8223
Pakistani	12905	1080	1.45 (1.35, 1.56)	<.0001	1.67 (1.55, 1.79)	<.0001	1.19 (1.04, 1.37)	0.0140
Bangladeshi	860	50	1.02 (0.79, 1.33)	0.8699	1.19 (0.91, 1.55)	0.2013	1.19 (0.71, 2.00)	0.5094
Other South Asian	2670	180	1.07 (0.92, 1.24)	0.3939	1.24 (1.06, 1.44)	0.0059	1.26 (0.97, 1.65)	0.0886
Caribbean	700	50	0.92 (0.70, 1.19)	0.5093	1.02 (0.78, 1.32)	0.9090	1.01 (0.68, 1.48)	0.9803
African	2100	105	0.86 (0.71, 1.05)	0.1433	1.01 (0.83, 1.23)	0.8999	1.60 (1.18, 2.17)	0.0024
Black Scottish/Other	445	40	1.34 (1.00, 1.79)	0.0476	1.42 (1.07, 1.90)	0.0168	1.27 (0.89, 1.83)	0.1927
Chinese	6500	240	0.58 (0.51, 0.66)	<.0001	0.68 (0.59, 0.77)	<.0001	0.81 (0.62, 1.06)	0.1277
Born outside UK vs UK-born					0.82 (0.79, 0.86)	0.0001	0.76 (0.70, 0.82)	<.0001
White Scottish*Born outside UK							1	
Other White British*Born outside UK							1.18 (1.03, 1.36)	0.0145
White Irish*Born outside UK							1.38 (1.21, 1.57)	<.0001
Other White*Born outside UK							0.78 (0.69, 0.89)	0.0002
Any Mixed Background*Born outside UK							0.86 (0.66, 1.12)	0.2672
Indian*Born outside UK							1.19 (0.94, 1.51)	0.1452
Pakistani*Born outside UK							1.70 (1.44, 2.01)	<.0001
Bangladeshi*Born outside UK							1.08 (0.59, 1.97)	0.8097
Other South Asian*Born outside UK							1.05 (0.76, 1.45)	0.7629
Caribbean*Born outside UK							1.10 (0.65, 1.88)	0.7175
African*Born outside UK							0.58 (0.40, 0.86)	0.0062
Black Scottish/Other*Born outside UK							1.51 (0.83, 2.73)	0.1743
Chinese*Born outside UK							0.86 (0.63, 1.17)	0.3253

Appendix 4.2b- Risk Ratios (RR) and 95% confidence intervals (CI) of reporting bad health in 2001 by ethnicity, for females. Models are adjusted for age, for age and UK-birth and for age, UK-birth and the interaction between UK-birth and ethnicity

	N	Reported Bad health	Adjusted for Age		Adjusted for Age and UK- birth		Adjusted for Age, UK-birth and ethnicity*UK-birth	
			RR (95% CI)	p-value	RR (95% CI)	p-value	RR (95% CI)	p-value
White Scottish	1995375	197200	1		1		1	
Other White British	162370	13690	0.79 (0.75, 0.83)	<.0001	0.79 (0.75, 0.83)	<.0001	0.79 (0.74, 0.83)	<.0001
White Irish	21170	2835	1.06 (1.01, 1.12)	0.0268	1.14 (1.08, 1.21)	<.0001	1.10 (1.02, 1.18)	0.0081
Other White	34200	2390	0.79 (0.75, 0.84)	<.0001	0.89 (0.83, 0.94)	<.0001	1.14 (1.05, 1.24)	0.0023
Any Mixed Background	5675	345	1.21 (1.08, 1.34)	0.0006	1.26 (1.14, 1.41)	<.0001	1.41 (1.25, 1.59)	<.0001
Indian	5855	505	1.23 (1.12, 1.35)	<.0001	1.37 (1.24, 1.50)	<.0001	1.16 (0.98, 1.38)	0.0850
Pakistani	12690	1345	1.84 (1.72, 1.96)	<.0001	2.03 (1.90, 2.17)	<.0001	1.46 (1.29, 1.65)	<.0001
Bangladeshi	700	50	1.30 (1.00, 1.70)	0.0524	1.45 (1.11, 1.89)	0.0068	1.53 (0.95, 2.47)	0.0796
Other South Asian	2205	175	1.21 (1.05, 1.40)	0.0078	1.34 (1.16, 1.54)	<.0001	1.37 (1.09, 1.71)	0.0064
Caribbean	755	60	0.98 (0.77, 1.26)	0.8836	1.06 (0.83, 1.35)	0.6582	1.47 (1.11, 1.95)	0.0071
African	1795	90	0.82 (0.67, 1.00)	0.0527	0.92 (0.75, 1.13)	0.4262	1.28 (0.90, 1.80)	0.1667
Black Scottish/Other	460	45	1.31 (0.99, 1.72)	0.0562	1.37 (1.04, 1.81)	0.0248	1.10 (0.76, 1.59)	0.6218
Chinese	6600	310	0.67 (0.60, 0.75)	<.0001	0.76 (0.68, 0.85)	<.0001	0.57 (0.42, 0.78)	0.0005
Born outside UK vs UK-born					0.86 (0.83, 0.90)	<.0001	0.81 (0.76, 0.87)	<.0001
White Scottish*Born outside UK							1	
Other White British*Born outside UK							1.24 (1.11, 1.38)	0.0002
White Irish*Born outside UK							1.15 (1.03, 1.29)	0.0138
Other White*Born outside UK							0.74 (0.66, 0.83)	<.0001
Any Mixed Background*Born outside UK							0.69 (0.53, 0.90)	0.0055
Indian*Born outside UK							1.32 (1.08, 1.63)	0.0078
Pakistani*Born outside UK							1.69 (1.46, 1.96)	<.0001
Bangladeshi*Born outside UK							0.97 (0.55, 1.73)	0.9192
Other South Asian*Born outside UK							1.02 (0.76, 1.37)	0.8845
Caribbean*Born outside UK							0.45 (0.26, 0.77)	0.0037
African*Born outside UK							0.68 (0.45, 1.04)	0.0754
Black Scottish/Other*Born outside UK							1.92 (1.10, 3.34)	0.0219
Chinese*Born outside UK							1.47 (1.05, 2.06)	0.0254

Appendix 4.3- Age-adjusted Risk Ratios (RR) and 95% confidence intervals (CI) of reporting bad health in 2001 by ethnicity, stratified by sex and age: 16-49 years old, above 50 years

Sex and ethnic groups	People aged 16-49				People aged 50 and over			
	Reported Bad health	N	Age-adjusted RR (95% CI)	p-value	Reported Bad health	N	Age-adjusted RR (95% CI)	p-value
MALES								
White Scottish	55870	900465	1		109050	573365	1	
Other White British	3740	81195	0.71 (0.64, 0.79)	<.0001	7655	54705	0.75 (0.69, 0.81)	<.0001
White Irish	675	10405	1.01 (0.89, 1.15)	0.8408	1870	7750	1.24 (1.16, 1.34)	<.0001
Other White	780	17295	0.76 (0.68, 0.86)	<.0001	1160	6405	0.94 (0.86, 1.02)	0.1253
Any Mixed Background	165	2210	1.56 (1.33, 1.84)	<.0001	100	475	1.12 (0.93, 1.35)	0.2390
Indian	160	3780	0.76 (0.64, 0.91)	0.0024	245	1145	1.14 (1.00, 1.29)	0.0510
Pakistani	485	7025	1.24 (1.10, 1.39)	0.0003	530	1550	1.79 (1.64, 1.96)	<.0001
Bangladeshi	30	495	1.17 (0.83, 1.64)	0.3675	20	110	0.95 (0.62, 1.46)	0.8147
Other South Asian	100	1515	1.10 (0.89, 1.36)	0.3887	75	380	1.11 (0.89, 1.39)	0.3538
Caribbean	20	450	0.80 (0.53, 1.21)	0.2884	25	120	1.11 (0.79, 1.57)	0.5476
African	75	1410	0.89 (0.70, 1.14)	0.3551	25	200	0.81 (0.56, 1.15)	0.2387
Black Scottish/Other	15	215	1.35 (0.86, 2.12)	0.1944	20	75	1.45 (0.99, 2.12)	0.0546
Chinese	105	3940	0.48 (0.40, 0.59)	<.0001	125	975	0.71 (0.60, 0.84)	<.0001
FEMALES								
White Scottish	70570	958980	1		122240	641180	1	
Other White British	5035	89380	0.75 (0.69, 0.82)	<.0001	8490	56115	0.80 (0.75, 0.86)	<.0001
White Irish	615	10570	0.79 (0.71, 0.88)	<.0001	2205	9425	1.18 (1.11, 1.25)	<.0001
Other White	1010	21375	0.69 (0.63, 0.76)	<.0001	1325	7890	0.88 (0.82, 0.96)	0.0020
Any Mixed Background	185	2660	1.17 (1.00, 1.37)	0.0442	125	560	1.24 (1.06, 1.45)	0.0088
Indian	225	3540	0.96 (0.84, 1.11)	0.6034	265	890	1.63 (1.44, 1.84)	<.0001
Pakistani	710	7185	1.54 (1.40, 1.70)	<.0001	555	1290	2.43 (2.24, 2.64)	<.0001
Bangladeshi	30	400	1.22 (0.85, 1.74)	0.2873	20	70	1.40 (0.91, 2.17)	0.1277
Other South Asian	100	1255	1.18 (0.96, 1.44)	0.1159	75	305	1.36 (1.11, 1.66)	0.0025
Caribbean	35	475	1.07 (0.78, 1.48)	0.6675	20	145	0.80 (0.54, 1.20)	0.2825
African	50	1170	0.65 (0.50, 0.86)	0.0020	30	160	1.10 (0.79, 1.53)	0.5884
Black Scottish/Other	20	235	1.19 (0.76, 1.86)	0.4385	25	100	1.34 (0.93, 1.94)	0.1183
Chinese	135	4210	0.48 (0.40, 0.57)	<.0001	165	915	1.02 (0.88, 1.18)	0.8204

Appendix 4.4. Health expectancies (HLE and DFLE) and Life expectancy (LE)* at 50 (95% CI) by ethnicity and sex in Scotland

Sex and ethnic groups	Population Census 2001 age 50 and over	LE at age 50* and 95% CI	HLE at age 50 and 95% CI	DFLE at age 50 and 95% CI	Number of years lived in poor health at age 50 and 95% CI	Number of years lived with limitations at age 50 and 95% CI	Proportion of life spent in good health at age 50	Proportion of life spent without limitations at age 50
MALES								
White Scottish	635105	27.1 [27.0, 27.1]	21.6 [21.6, 21.7]	15.5 [15.4, 15.5]	5.4 [5.4, 5.5]	11.6 [11.5, 11.6]	79.9%	57.2%
Other White British	61120	30.3 [30.0, 30.5]	25.3 [25.1, 25.5]	18.5 [18.4, 18.7]	5.0 [4.9, 5.1]	11.7 [11.6, 11.9]	83.5%	61.2%
White Irish	8625	26.9 [26.3, 27.6]	20.2 [19.8, 20.6]	14.3 [14.0, 14.6]	6.7 [6.5, 7.0]	12.6 [12.2, 13.1]	75.0%	53.1%
Other White	7485	28.3 [27.5, 29.0]	22.6 [22.1, 23.1]	16.9 [16.5, 17.3]	5.7 [5.4, 6.0]	11.4 [10.9, 11.8]	79.8%	59.8%
Any Mixed Background	520	25.6 [23.1, 28.1]	20.2 [18.4, 22.0]	14.0 [12.6, 15.3]	5.4 [4.4, 6.3]	11.6 [10.1, 13.1]	78.9%	54.6%
Indian	1165	31.9 [29.4, 34.3]	23.0 [21.6, 24.5]	16.3 [15.3, 17.3]	8.8 [7.5, 10.2]	15.6 [13.8, 17.3]	72.3%	51.2%
Pakistani	1570	31.5 [29.2, 33.7]	20.1 [18.7, 21.6]	13.6 [12.4, 14.7]	11.3 [10.2, 12.5]	17.9 [16.4, 19.4]	64.0%	43.1%
Other South Asian	505	29.1 [25.8, 32.4]	22.7 [20.4, 25.1]	15.9 [14.0, 17.8]	6.4 [5.0, 7.7]	13.2 [11.3, 15.1]	78.1%	54.6%
African origin	425	28.1 [24.7, 31.5]	22.2 [19.7, 24.6]	17.1 [15.1, 19.0]	5.9 [4.7, 7.2]	11.0 [9.2, 12.8]	78.9%	60.8%
Chinese	1000	30.2 [27.8, 32.7]	25.2 [23.6, 26.9]	17.1 [16.0, 18.2]	5.0 [3.9, 6.1]	13.1 [11.4, 14.8]	79.9%	57.2%
FEMALES								
White Scottish	784190	30.9 [30.8, 30.9]	24.5 [24.5, 24.6]	17.1 [17.1, 17.2]	6.3 [6.3, 6.4]	13.7 [13.7, 13.8]	79.4%	55.6%
Other White British	68485	33.5 [33.3, 33.8]	27.5 [27.3, 27.7]	19.5 [19.3, 19.6]	6.0 [5.9, 6.2]	14.1 [13.9, 14.2]	82.0%	58.0%
White Irish	11415	32.2 [31.5, 32.8]	24.1 [23.7, 24.5]	16.8 [16.5, 17.0]	8.1 [7.8, 8.3]	15.4 [15.0, 15.8]	74.9%	52.1%
Other White	9395	33.1 [32.5, 33.8]	26.4 [26.0, 26.9]	18.8 [18.5, 19.1]	6.7 [6.4, 7.0]	14.3 [13.9, 14.8]	79.8%	56.8%
Any Mixed Background	680	30.1 [27.5, 32.7]	22.9 [21.1, 24.6]	15.5 [14.3, 16.7]	7.2 [6.1, 8.3]	14.6 [12.8, 16.3]	76.1%	51.5%
Indian	925	34.2 [31.7, 36.8]	22.3 [20.7, 24.0]	13.6 [12.6, 14.5]	11.9 [10.5, 13.3]	20.7 [18.7, 22.6]	65.2%	39.6%
Pakistani	1305	35.8 [33.2, 38.4]	19.2 [17.7, 20.8]	10.8 [9.7, 12.0]	16.6 [14.8, 18.3]	25.0 [23.0, 27.0]	53.8%	30.2%
Other South Asian	425	31.9 [28.8, 34.9]	24.0 [21.7, 26.3]	16.9 [15.3, 18.5]	7.8 [6.6, 9.1]	15.0 [13.0, 16.9]	75.4%	53.1%
African origin	445	30.2 [26.7, 33.8]	23.4 [20.9, 25.8]	16.6 [15.0, 18.2]	6.8 [5.4, 8.3]	13.6 [11.3, 16.0]	77.4%	54.9%
Chinese	985	34.5 [32.3, 36.8]	26.8 [25.2, 28.4]	17.2 [16.2, 18.2]	7.7 [6.7, 8.7]	17.3 [15.7, 18.9]	77.6%	49.9%

* The life expectancy at 50 years by ethnic group was not previously published but was taken from the life table used to calculate LE at birth (Gruer et al., 2016). It is included in column 3 and used in combination with HLE and DFLE for further calculation.

Chapter 5 appendices

Appendix 5.1. HRs (95% CI) of time to death (2001-2013) for one socio-economic indicator, one self-assessed health indicator reported in 2001 and their interaction. Models are adjusted for age, stratified by sex, reported separately for each self-assessed health indicator (self-reported health, Limiting Long Term Illness) and in separate tables for each socio-economic indicators (household tenure, highest qualification level, SIMD)

a. Household Tenure

Variables	Model with Household tenure*SRH interaction				Model with Household tenure *LLTI interaction			
	Males		Females		Males		Females	
	HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value
Household tenure								
Own versus rent	0.73 (0.72, 0.74)	<.0001	0.80 (0.79, 0.81)	<.0001	0.66 (0.65, 0.66)	<.0001	0.70 (0.70, 0.71)	<.0001
Self-reported health (SRH)								
Good/Fair versus Bad	0.49 (0.48, 0.49)	<.0001	0.53 (0.52, 0.53)	<.0001				
Interaction Household tenure*SRH								
Own * Good/Fair health	0.80 (0.79, 0.81)	<.0001	0.76 (0.75, 0.78)	<.0001				
Limiting Long term Illness (LLTI)								
No LLTI versus LLTI					0.52 (0.51, 0.52)	<.0001	0.51 (0.51, 0.52)	<.0001
Interaction Household tenure*LLTI								
Rent * no LLTI					0.88 (0.87, 0.89)	<.0001	0.89 (0.88, 0.90)	<.0001

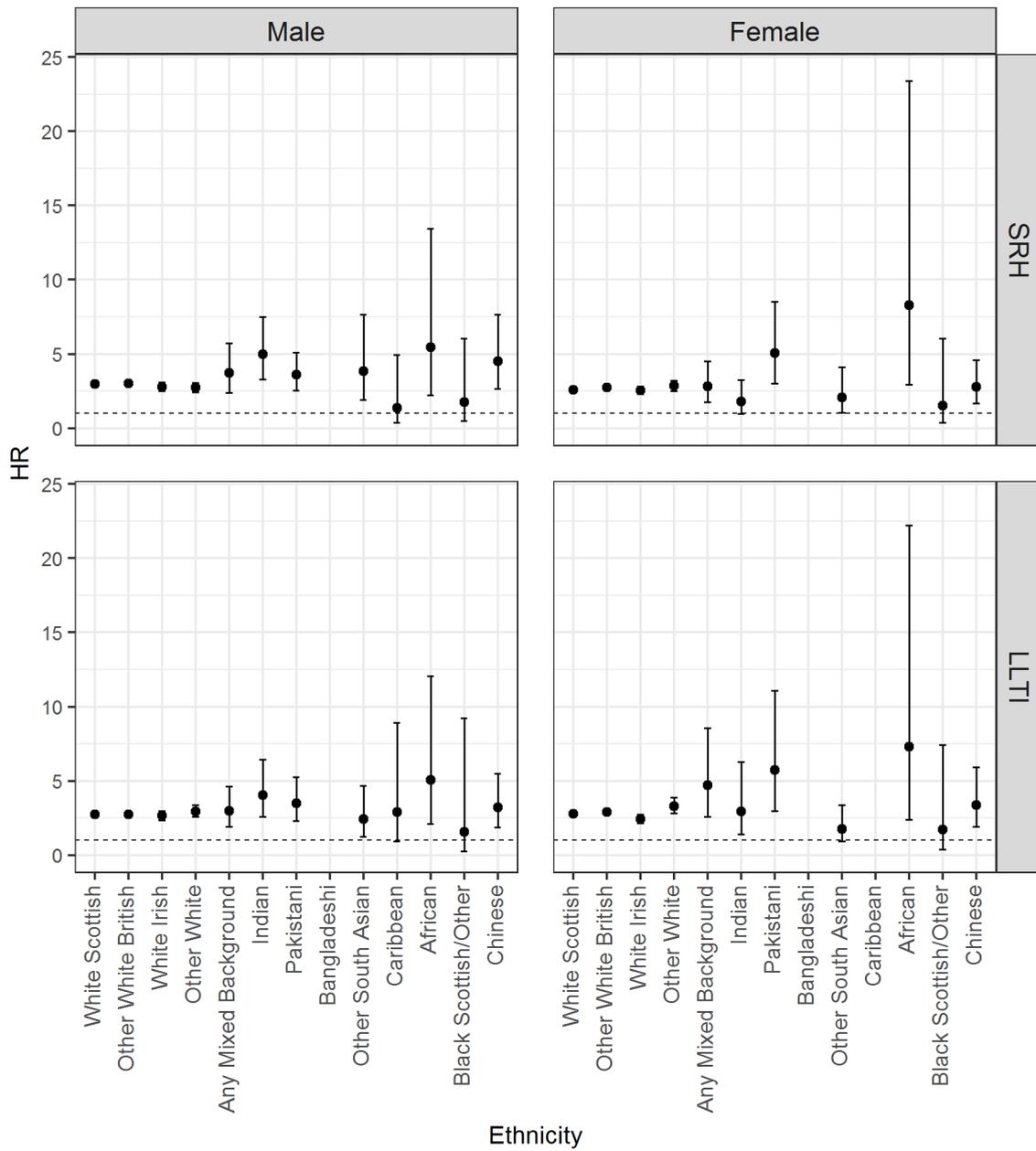
b. Highest level of qualification (combined individual and household)

Variables	Model with Qualification level *SRH interaction				Model with Qualification level *LLTI interaction			
	Males		Females		Males		Females	
	HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value
Highest level of qualification								
Lower level versus No qualification	0.96 (0.94, 0.98)	0.0001	0.89 (0.87, 0.91)	<.0001	0.90 (0.89, 0.92)	<.0001	0.88 (0.87, 0.89)	<.0001
Higher level versus No qualification	0.93 (0.91, 0.98)	<.0001	0.86 (0.84, 0.88)	<.0001	0.83 (0.82, 0.84)	<.0001	0.87 (0.83, 0.88)	<.0001
Self-reported health (SRH)								
Good/Fair versus Bad	0.42 (0.42, 0.43)	<.0001	0.43 (0.42, 0.43)	<.0001				
Interaction Qualification level*SRH								
Lower level * Good/Fair health	0.80 (0.78, 0.82)	<.0001	0.84 (0.82, 0.86)	<.0001				
Higher level * Good/Fair health	0.67 (0.66, 0.69)	<.0001	0.81 (0.79, 0.83)	<.0001				
Limiting Long term Illness (LLTI)								
No LLTI versus LLTI					0.47 (0.46, 0.47)	<.0001	0.43 (0.43, 0.44)	<.0001
Interaction Qualification level*LLTI								
Lower level * no LLTI					0.81 (0.79, 0.83)	<.0001	0.79 (0.77, 0.81)	<.0001
Higher level * no LLTI					0.70 (0.69, 0.72)	<.0001	0.69 (0.67, 0.71)	<.0001

c. Scottish Index for Multiple Deprivation (SIMD)

Variables	Model with SIMD*SRH interaction				Model with SIMD *LLTI interaction			
	Males		Females		Males		Females	
	HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value
SIMD								
Quintile 2 versus 1 (most deprived)	0.90 (0.88, 0.92)	<.0001	0.93 (0.91, 0.94)	<.0001	0.87 (0.86, 0.88)	<.0001	0.91 (0.90, 0.92)	<.0001
Quintile 3 versus 1 (most deprived)	0.87 (0.85, 0.89)	<.0001	0.92 (0.90, 0.94)	<.0001	0.81 (0.80, 0.82)	<.0001	0.88 (0.86, 0.89)	<.0001
Quintile 4 versus 1 (most deprived)	0.81 (0.79, 0.82)	<.0001	0.92 (0.90, 0.93)	<.0001	0.73 (0.72, 0.74)	<.0001	0.84 (0.83, 0.85)	<.0001
Quintile 5 versus 1 (most deprived)	0.78 (0.76, 0.80)	<.0001	0.87 (0.85, 0.89)	<.0001	0.68 (0.67, 0.69)	<.0001	0.78 (0.77, 0.79)	<.0001
Self-reported health (SRH)								
Good/Fair versus Bad	0.49 (0.48, 0.49)	<.0001	0.51 (0.50, 0.52)	<.0001				
Interaction Qualification level*SRH								
Quintile 2 * Good/Fair health	0.93 (0.91, 0.95)	<.0001	0.95 (0.93, 0.97)	<.0001				
Quintile 3 * Good/Fair health	0.85 (0.83, 0.87)	<.0001	0.88 (0.86, 0.90)	<.0001				
Quintile 4 * Good/Fair health	0.78 (0.76, 0.80)	<.0001	0.79 (0.77, 0.81)	<.0001				
Quintile 5 * Good/Fair health	0.70 (0.68, 0.72)	<.0001	0.74 (0.72, 0.76)	<.0001				
Limiting long term illness (LLTI)								
No LLTI versus LLTI					0.53 (0.52, 0.53)	<.0001	0.53 (0.52, 0.54)	<.0001
Interaction Qualification level*LLTI								
Quintile 2 * no LLTI					0.95 (0.92, 0.97)	<.0001	0.95 (0.93, 0.97)	<.0001
Quintile 3 * no LLTI					0.88 (0.86, 0.90)	<.0001	0.88 (0.86, 0.90)	<.0001
Quintile 4 * no LLTI					0.82 (0.80, 0.84)	<.0001	0.80 (0.78, 0.82)	<.0001
Quintile 5 * no LLTI					0.76 (0.74, 0.78)	<.0001	0.76 (0.74, 0.77)	<.0001

Appendix 5.2. Age-adjusted HRs (mortality risk) and 95% CIs of SRH (bad health versus good/fair health) and LLTI (LLTI versus no) for each ethnic group and sex – sensitivity analysis using 5 years mortality (2001-2006)



Appendix 5.3.a. HRs (mortality risk) and 95% CIs by ethnicity, for males. Models are adjusted for age at baseline, and subsequently for SRH (good/fair versus bad health), the interaction between SRH and ethnicity and SES

	Deaths 2001-13	People 2001	Adjusted for age		Adjusted for age and SRH		Adjusted for age, SRH and ethnicity*SRH(0)		Adjusted for age, SRH, ethnicity*SRH(0) and SES	
			HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value
White Scottish	251755	1949480	1		1		1		1	
Other White British	17855	160235	0.76 (0.75, 0.77)	<.0001	0.78 (0.77, 0.79)	<.0001	0.81 (0.78, 0.83)	<.0001	0.91 (0.88, 0.94)	<.0001
White Irish	3420	20340	1.01 (0.98, 1.05)	0.5353	0.96 (0.93, 0.99)	0.0179	0.92 (0.87, 0.97)	0.0029	0.86 (0.81, 0.92)	<.0001
Other White	2785	29945	0.82 (0.79, 0.86)	<.0001	0.80 (0.77, 0.83)	<.0001	0.80 (0.75, 0.85)	<.0001	0.88 (0.82, 0.95)	0.0008
Any Mixed Background	240	5310	1.16 (1.02, 1.31)	0.0240	1.16 (1.02, 1.31)	0.0230	1.33 (1.06, 1.67)	0.0123	1.30 (1.02, 1.66)	0.0315
Indian	280	6450	0.63 (0.56, 0.71)	<.0001	0.60 (0.53, 0.68)	<.0001	0.66 (0.55, 0.80)	<.0001	0.84 (0.69, 1.01)	0.0636
Pakistani	450	12930	0.68 (0.62, 0.75)	<.0001	0.60 (0.54, 0.65)	<.0001	0.56 (0.49, 0.65)	<.0001	0.61 (0.53, 0.71)	<.0001
Bangladeshi	25	860	0.55 (0.36, 0.82)	0.0038	0.53 (0.35, 0.79)	0.0020	0.42 (0.19, 0.95)	0.0360	0.41 (0.19, 0.92)	0.0302
Other South Asian	125	2685	0.94 (0.79, 1.12)	0.5093	0.88 (0.74, 1.05)	0.1552	0.73 (0.53, 1.00)	0.0531	0.83 (0.60, 1.16)	0.2788
Caribbean	55	710	0.93 (0.72, 1.21)	0.6109	0.91 (0.70, 1.18)	0.4632	0.78 (0.48, 1.27)	0.3214	0.81 (0.47, 1.39)	0.4392
African	65	2105	0.94 (0.74, 1.20)	0.6294	0.95 (0.74, 1.22)	0.6936	1.07 (0.66, 1.75)	0.7771	1.01 (0.62, 1.65)	0.9727
Black Scottish or Other Black	40	460	0.94 (0.69, 1.29)	0.7231	0.98 (0.71, 1.33)	0.8760	1.14 (0.65, 2.01)	0.6452	0.71 (0.34, 1.49)	0.3665
Chinese	215	6530	0.56 (0.49, 0.64)	<.0001	0.58 (0.51, 0.67)	<.0001	0.70 (0.54, 0.92)	0.0096	0.71 (0.54, 0.95)	0.0207
SRH (good/fair versus bad)					0.40 (0.39, 0.40)	<.0001	0.40 (0.39, 0.40)	<.0001	0.44 (0.43, 0.44)	<.0001
Other White British * SRH							0.96 (0.93, 0.99)	0.0158	0.92 (0.89, 0.96)	<.0001
White Irish * SRH							1.07 (1.00, 1.15)	0.0478	1.09 (1.01, 1.18)	0.0273
Other White * SRH							1.00 (0.92, 1.08)	0.9478	0.91 (0.83, 1.00)	0.0432
Any Mixed Background * SRH							0.82 (0.62, 1.07)	0.1500	0.78 (0.58, 1.05)	0.0968
Indian * SRH							0.85 (0.67, 1.08)	0.1957	0.79 (0.62, 1.01)	0.0567
Pakistani * SRH							1.11 (0.92, 1.34)	0.2655	1.11 (0.92, 1.34)	0.2898
Bangladeshi * SRH							1.35 (0.53, 3.43)	0.5266	1.49 (0.58, 3.82)	0.4022

Other South Asian * SRH	1.32 (0.90, 1.93)	0.1486	1.13 (0.76, 1.68)	0.5471
Caribbean * SRH	1.24 (0.70, 2.22)	0.4637	1.25 (0.66, 2.37)	0.4955
African * SRH	0.85 (0.48, 1.51)	0.5839	0.86 (0.49, 1.53)	0.6084
Black Scottish/Oth Black * SRH	0.80 (0.41, 1.58)	0.5245	1.12 (0.46, 2.69)	0.8051
Chinese * SRH	0.78 (0.58, 1.07)	0.1222	0.77 (0.55, 1.07)	0.1132
SIMD (1 vs 5-least deprived)			1.34 (1.32, 1.36)	<.0001
SIMD (2 vs 5-least deprived)			1.24 (1.23, 1.26)	<.0001
SIMD (3 vs 5-least deprived)			1.18 (1.16, 1.19)	<.0001
SIMD (4 vs 5-least deprived)			1.07 (1.05, 1.08)	<.0001
Household tenure (own vs. rent)			1.55 (1.53, 1.56)	<.0001
Highest qualification (higher vs. no)			0.85 (0.84, 0.86)	<.0001
Highest qualification (lower vs. no)			0.92 (0.91, 0.93)	<.0001

Appendix 5.3.b. HRs (mortality risk) and 95% CIs by ethnicity, for females. Models are adjusted for age at baseline, and subsequently for SRH (good/fair versus bad health), the interaction between SRH and ethnicity and SES

	Deaths 2001-13	People 2001	Adjusted for age		Adjusted for age and SRH		Adjusted for age, SRH and ethnicity*SRH(0)		Adjusted for age, SRH, ethnicity*SRH(0) and SES	
			HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value	HR (95% CI)	p-value
White Scottish	285440	2138640	1		1		1		1	
Other White British	19310	174750	0.80 (0.79, 0.81)	<.0001	0.81 (0.80, 0.83)	<.0001	0.85 (0.83, 0.88)	<.0001	0.92 (0.89, 0.95)	<.0001
White Irish	3895	23160	0.92 (0.89, 0.95)	<.0001	0.87 (0.85, 0.90)	<.0001	0.85 (0.81, 0.90)	<.0001	0.83 (0.78, 0.88)	<.0001
Other White	2670	35710	0.79 (0.76, 0.82)	<.0001	0.78 (0.75, 0.81)	<.0001	0.81 (0.75, 0.86)	<.0001	0.83 (0.77, 0.90)	<.0001
Any Mixed Background	230	5800	0.95 (0.83, 1.08)	0.4249	0.92 (0.81, 1.05)	0.2109	0.94 (0.75, 1.16)	0.5446	0.79 (0.60, 1.04)	0.0892
Indian	175	5890	0.65 (0.56, 0.76)	<.0001	0.58 (0.50, 0.67)	<.0001	0.60 (0.48, 0.74)	<.0001	0.64 (0.51, 0.81)	0.0001
Pakistani	295	12700	0.78 (0.69, 0.87)	<.0001	0.64 (0.57, 0.71)	<.0001	0.65 (0.56, 0.76)	<.0001	0.70 (0.60, 0.81)	0.0000
Bangladeshi	15	705	0.87 (0.54, 1.41)	0.5790	0.84 (0.52, 1.36)	0.4828	0.74 (0.31, 1.77)	0.4937	0.52 (0.17, 1.60)	0.2523
Other South Asian	120	2255	1.08 (0.90, 1.29)	0.4044	1.07 (0.90, 1.29)	0.4422	1.10 (0.80, 1.50)	0.5689	1.07 (0.74, 1.54)	0.7099
Caribbean	35	775	0.71 (0.52, 0.98)	0.0389	0.73 (0.53, 1.00)	0.0533	0.70 (0.38, 1.30)	0.2571	0.73 (0.37, 1.46)	0.3754
African	45	1800	1.13 (0.85, 1.50)	0.4061	1.15 (0.86, 1.53)	0.3489	1.51 (0.91, 2.51)	0.1093	1.25 (0.73, 2.16)	0.4166
Black Scottish or Other Black	40	480	1.11 (0.82, 1.51)	0.4908	0.99 (0.73, 1.34)	0.9419	0.65 (0.37, 1.11)	0.1140	0.88 (0.44, 1.77)	0.7242
Chinese	210	6670	0.69 (0.60, 0.78)	<.0001	0.67 (0.59, 0.77)	<.0001	0.57 (0.44, 0.74)	<.0001	0.56 (0.41, 0.74)	0.0001
SRH (good/fair versus bad)					0.44 (0.44, 0.44)	<.0001	0.44 (0.44, 0.45)	<.0001	0.44 (0.44, 0.45)	<.0001
Other White British * SRH							0.94 (0.91, 0.97)	<.0001	0.90 (0.87, 0.94)	<.0001
White Irish * SRH							1.04 (0.98, 1.11)	0.2020	1.00 (0.92, 1.08)	0.9811
Other White * SRH							0.95 (0.88, 1.03)	0.2525	0.98 (0.89, 1.08)	0.6925
Any Mixed Background * SRH							0.98 (0.75, 1.28)	0.8632	1.06 (0.75, 1.49)	0.7368
Indian * SRH							0.94 (0.70, 1.26)	0.6780	0.95 (0.69, 1.30)	0.7484
Pakistani * SRH							0.95 (0.75, 1.19)	0.6333	0.94 (0.75, 1.19)	0.6184
Bangladeshi * SRH							1.22 (0.43, 3.46)	0.7093	1.52 (0.42, 5.51)	0.5276

Other South Asian * SRH	0.97 (0.66, 1.42)	0.8760	0.88 (0.55, 1.39)	0.5833
Caribbean * SRH	1.06 (0.51, 2.18)	0.8800	0.83 (0.35, 1.93)	0.6613
African * SRH	0.68 (0.37, 1.26)	0.2189	0.77 (0.40, 1.49)	0.4454
Black Scottish/Oth Black * SRH	2.04 (1.06, 3.94)	0.0339	0.97 (0.41, 2.26)	0.9352
Chinese * SRH	1.26 (0.93, 1.70)	0.1411	1.37 (0.97, 1.92)	0.0700
SIMD (1 vs 5-least deprived)			1.18 (1.17, 1.20)	<.0001
SIMD (2 vs 5-least deprived)			1.13 (1.11, 1.15)	<.0001
SIMD (3 vs 5-least deprived)			1.11 (1.09, 1.12)	<.0001
SIMD (4 vs 5-least deprived)			1.06 (1.04, 1.07)	<.0001
Household tenure (own vs. rent)			1.54 (1.53, 1.56)	<.0001
Highest qualification (higher vs. no)			0.88 (0.87, 0.89)	<.0001
Highest qualification (lower vs. no)			0.89 (0.88, 0.90)	<.0001

Chapter 6 appendices

Appendix 6.1- Risk Ratios (RR) and 95% confidence intervals (CI) of each comorbidity of the Charlson index by ethnicity and stratified by sex.

Models are adjusted for age

Sex and ethnic groups	PY	Myocardial infarction		Congestive heart failure		Peripheral vascular disease		Cerebrovascular disease	
		Number of events	Age-adjusted RR (95% CI)	Number of events	Age-adjusted RR (95% CI)	Number of events	Age-adjusted RR (95% CI)	Number of events	Age-adjusted RR (95% CI)
MALES									
White Scottish	21179755	43970	1	20935	1	20740	1	44320	1
Other White British	1571080	3125	0.78 (0.74, 0.81)	1575	0.84 (0.79, 0.89)	1365	0.73 (0.68, 0.78)	3135	0.78 (0.75, 0.82)
White Irish	202190	530	0.93 (0.85, 1.01)	295	1.02 (0.91, 1.15)	260	0.94 (0.84, 1.06)	570	0.96 (0.89, 1.04)
Other White	278515	410	0.79 (0.71, 0.87)	255	1.02 (0.90, 1.15)	160	0.70 (0.60, 0.82)	410	0.79 (0.72, 0.87)
Any Mixed Background	56265	40	1.03 (0.76, 1.39)	15	1.05 (0.64, 1.72)	15	0.79 (0.46, 1.35)	40	1.10 (0.82, 1.48)
Indian	65945	120	1.22 (1.03, 1.44)	55	1.28 (1.00, 1.65)	25	0.53 (0.35, 0.80)	90	0.94 (0.77, 1.15)
Pakistani	146430	330	2.12 (1.91, 2.36)	100	1.65 (1.35, 2.01)	50	0.77 (0.58, 1.01)	165	1.17 (1.01, 1.36)
Other South Asian	35500	55	1.21 (0.93, 1.57)	20	1.11 (0.69, 1.80)	10	0.68 (0.39, 1.19)	45	1.13 (0.86, 1.50)
African origin	32160	25	0.63 (0.42, 0.93)	20	1.37 (0.89, 2.13)	10	0.69 (0.38, 1.28)	40	1.28 (0.95, 1.71)
Chinese	68685	35	0.39 (0.28, 0.53)	20	0.54 (0.35, 0.84)	15	0.34 (0.20, 0.58)	65	0.80 (0.63, 1.03)
FEMALES									
White Scottish	22581190	23775	1	15860	1	13380	1	40600	1
Other White British	1644435	1345	0.68 (0.64, 0.73)	1060	0.82 (0.76, 0.88)	690	0.63 (0.58, 0.68)	2620	0.78 (0.75, 0.82)
White Irish	216905	300	0.86 (0.76, 0.97)	225	0.89 (0.78, 1.01)	155	0.78 (0.66, 0.91)	635	1.03 (0.95, 1.12)
Other White	319915	225	0.74 (0.65, 0.85)	185	0.92 (0.80, 1.07)	130	0.79 (0.66, 0.94)	400	0.77 (0.69, 0.86)
Any Mixed Background	59970	20	0.82 (0.53, 1.28)	15	1.20 (0.72, 1.99)	15	1.23 (0.75, 2.00)	50	1.36 (1.03, 1.81)
Indian	59925	45	1.34 (1.00, 1.78)	25	1.23 (0.81, 1.85)	.	.	55	0.94 (0.73, 1.22)
Pakistani	143940	110	1.99 (1.65, 2.40)	85	2.74 (2.21, 3.41)	15	0.43 (0.25, 0.73)	150	1.56 (1.32, 1.84)
Other South Asian	28610	15	1.11 (0.68, 1.83)	10	1.57 (0.90, 2.75)	.	.	20	0.95 (0.63, 1.45)
African origin	28590	10	0.79 (0.45, 1.39)	10	1.15 (0.62, 2.14)	.	.	25	0.92 (0.62, 1.37)
Chinese	68010	20	0.52 (0.34, 0.82)	15	0.60 (0.35, 1.03)	10	0.39 (0.19, 0.77)	40	0.63 (0.47, 0.85)

Continued...

Sex and ethnic groups	PY	Dementia		Chronic pulmonary disease		Connective tissue disease		Ulcer disease	
		Number of events	Age-adjusted RR (95% CI)	Number of events	Age-adjusted RR (95% CI)	Number of events	Age-adjusted RR (95% CI)	Number of events	Age-adjusted RR (95% CI)
MALES									
White Scottish	21179755	3325	1	34910	1	3590	1	13235	1
Other White British	1571080	230	0.79 (0.69, 0.90)	1910	0.68 (0.63, 0.73)	250	0.77 (0.67, 0.88)	650	0.55 (0.51, 0.60)
White Irish	202190	55	1.09 (0.84, 1.42)	430	1.06 (0.95, 1.18)	40	0.87 (0.63, 1.21)	185	1.12 (0.97, 1.30)
Other White	278515	45	1.05 (0.79, 1.40)	300	0.74 (0.66, 0.84)	35	0.79 (0.56, 1.12)	110	0.69 (0.57, 0.83)
Any Mixed Background	56265	.		80	1.29 (1.04, 1.61)	.		20	1.25 (0.80, 1.95)
Indian	65945	.		70	0.86 (0.69, 1.08)	10	1.04 (0.54, 1.99)	20	0.60 (0.38, 0.93)
Pakistani	146430	.		240	1.48 (1.29, 1.68)	15	0.95 (0.56, 1.60)	35	0.68 (0.49, 0.93)
Other South Asian	35500	.		45	1.09 (0.81, 1.47)	.		15	1.08 (0.66, 1.76)
African origin	32160	.		20	0.67 (0.45, 1.02)	.		10	0.69 (0.36, 1.32)
Chinese	68685	.		45	0.56 (0.42, 0.76)	.		45	1.49 (1.11, 2.00)
FEMALES									
White Scottish	22581190	4525	1	45065	1	10865	1	10845	1
Other White British	1644435	280	0.76 (0.67, 0.85)	2360	0.67 (0.62, 0.72)	650	0.71 (0.65, 0.77)	585	0.65 (0.60, 0.72)
White Irish	216905	85	1.04 (0.84, 1.29)	520	0.91 (0.83, 1.01)	145	1.05 (0.89, 1.23)	170	1.17 (1.00, 1.37)
Other White	319915	40	0.70 (0.52, 0.95)	355	0.60 (0.54, 0.68)	100	0.69 (0.56, 0.84)	90	0.61 (0.50, 0.76)
Any Mixed Background	59970	.		105	1.45 (1.21, 1.75)	15	1.09 (0.66, 1.80)	10	0.61 (0.28, 1.32)
Indian	59925	.		70	0.80 (0.64, 1.02)	25	1.21 (0.81, 1.81)	15	0.77 (0.47, 1.28)
Pakistani	143940	.		230	1.31 (1.15, 1.50)	65	1.73 (1.37, 2.19)	35	0.99 (0.71, 1.38)
Other South Asian	28610	.		40	1.11 (0.82, 1.51)	10	1.17 (0.63, 2.16)	10	1.50 (0.86, 2.64)
African origin	28590	.		50	1.30 (0.99, 1.70)	10	1.27 (0.72, 2.23)	10	0.68 (0.31, 1.51)
Chinese	68010	.		40	0.40 (0.29, 0.54)	15	0.60 (0.36, 1.00)	15	0.78 (0.49, 1.24)

Continued...

Sex and ethnic groups	PY	Mild liver disease		Diabetes		Hemiplegia		Moderate or severe renal disease	
		Number of events	Age-adjusted RR (95% CI)	Number of events	Age-adjusted RR (95% CI)	Number of events	Age-adjusted RR (95% CI)	Number of events	Age-adjusted RR (95% CI)
MALES									
White Scottish	21179755	9545	1	9565	1	2860	1	7615	1
Other White British	1571080	490	0.56 (0.50, 0.62)	515	0.71 (0.65, 0.79)	175	0.75 (0.64, 0.88)	500	0.74 (0.67, 0.82)
White Irish	202190	120	1.02 (0.85, 1.23)	90	0.92 (0.75, 1.14)	30	0.90 (0.61, 1.32)	110	1.14 (0.95, 1.37)
Other White	278515	115	0.96 (0.80, 1.16)	90	0.77 (0.63, 0.95)	25	0.76 (0.51, 1.12)	85	0.93 (0.75, 1.15)
Any Mixed Background	56265	20	1.72 (1.08, 2.72)	15	0.70 (0.43, 1.13)	.	.	10	0.99 (0.50, 1.97)
Indian	65945	25	1.09 (0.75, 1.60)	35	1.35 (0.97, 1.87)	10	0.80 (0.36, 1.77)	35	2.07 (1.47, 2.92)
Pakistani	146430	60	1.41 (1.08, 1.83)	85	1.42 (1.13, 1.77)	15	1.00 (0.60, 1.65)	100	3.44 (2.83, 4.19)
Other South Asian	35500	15	1.27 (0.78, 2.08)	25	2.99 (2.00, 4.48)
African origin	32160	10	0.60 (0.29, 1.27)	15	1.27 (0.78, 2.08)	.	.	15	2.23 (1.34, 3.69)
Chinese	68685	40	1.59 (1.14, 2.22)	10	0.25 (0.12, 0.53)	.	.	25	1.58 (1.06, 2.34)
FEMALES									
White Scottish	22581190	7075	1	8490	1	2720	1	6170	1
Other White British	1644435	370	0.60 (0.54, 0.68)	430	0.71 (0.64, 0.78)	210	1.00 (0.86, 1.16)	350	0.70 (0.63, 0.78)
White Irish	216905	75	0.87 (0.69, 1.10)	65	0.73 (0.57, 0.93)	30	0.88 (0.61, 1.28)	65	0.74 (0.58, 0.95)
Other White	319915	70	0.71 (0.56, 0.90)	60	0.53 (0.41, 0.69)	35	0.89 (0.63, 1.24)	65	0.82 (0.64, 1.04)
Any Mixed Background	59970	10	1.19 (0.66, 2.14)	10	0.48 (0.26, 0.89)	.	.	10	0.96 (0.46, 2.01)
Indian	59925	10	0.66 (0.36, 1.23)	20	1.01 (0.64, 1.60)	.	.	15	1.45 (0.88, 2.40)
Pakistani	143940	40	1.46 (1.08, 1.97)	60	1.30 (1.01, 1.67)	15	0.97 (0.56, 1.67)	70	3.72 (2.94, 4.71)
Other South Asian	28610	.	.	10	0.95 (0.50, 1.83)	.	.	10	1.41 (0.63, 3.13)
African origin	28590	.	.	10	1.08 (0.58, 2.00)	.	.	10	1.51 (0.72, 3.15)
Chinese	68010	25	1.55 (1.06, 2.26)	10	0.54 (0.31, 0.95)	.	.	25	2.35 (1.62, 3.39)

Continued...

Sex and ethnic groups	PY	Diabetes with end organ damage		Cancer		Moderate or severe liver disease		Metastatic cancer	
		Number of events	Age-adjusted RR (95% CI)	Number of events	Age-adjusted RR (95% CI)	Number of events	Age-adjusted RR (95% CI)	Number of events	Age-adjusted RR (95% CI)
MALES									
White Scottish	21179755	3640	1	101795	1	5010	1	13095	1
Other White British	1571080	235	0.71 (0.62, 0.82)	7890	0.86 (0.84, 0.88)	235	0.5 (0.43, 0.58)	1080	0.91 (0.85, 0.97)
White Irish	202190	40	0.89 (0.65, 1.2)	1340	1.01 (0.96, 1.06)	50	0.81 (0.61, 1.07)	165	0.96 (0.83, 1.12)
Other White	278515	35	0.77 (0.55, 1.09)	1005	0.88 (0.83, 0.93)	50	0.83 (0.63, 1.1)	125	0.85 (0.72, 1.01)
Any Mixed Background	56265	.		95	1.03 (0.85, 1.26)	10	1.59 (0.79, 3.18)	10	1.11 (0.63, 1.93)
Indian	65945	20	2.33 (1.47, 3.69)	115	0.52 (0.43, 0.62)	10	0.88 (0.49, 1.59)	15	0.5 (0.29, 0.88)
Pakistani	146430	45	3.2 (2.38, 4.31)	225	0.66 (0.58, 0.74)	30	1.31 (0.88, 1.94)	15	0.4 (0.24, 0.66)
Other South Asian	35500	.		75	0.81 (0.65, 1.02)	.		.	
African origin	32160	.		60	0.79 (0.62, 1.01)	.		10	0.62 (0.28, 1.38)
Chinese	68685	10	0.86 (0.42, 1.79)	135	0.7 (0.59, 0.82)	10	0.49 (0.22, 1.08)	25	0.93 (0.62, 1.38)
FEMALES									
White Scottish	22581190	2190	1	108650	1	3180	1	15325	1
Other White British	1644435	125	0.7 (0.58, 0.84)	8200	0.9 (0.88, 0.92)	160	0.59 (0.5, 0.69)	1200	0.93 (0.88, 0.99)
White Irish	216905	25	0.86 (0.58, 1.27)	1370	0.94 (0.89, 0.99)	35	0.88 (0.63, 1.24)	190	0.91 (0.79, 1.05)
Other White	319915	25	0.89 (0.61, 1.31)	1270	0.91 (0.86, 0.95)	30	0.66 (0.45, 0.98)	155	0.8 (0.68, 0.94)
Any Mixed Background	59970	.		100	0.82 (0.67, 0.99)	10	1.78 (0.85, 3.75)	10	0.57 (0.28, 1.17)
Indian	59925	10	2.45 (1.32, 4.56)	95	0.52 (0.43, 0.63)	.		20	0.88 (0.58, 1.35)
Pakistani	143940	25	3.11 (2.09, 4.63)	230	0.71 (0.63, 0.81)	15	1.28 (0.78, 2.12)	30	0.72 (0.51, 1.02)
Other South Asian	28610	.		60	0.81 (0.64, 1.02)	.		10	0.7 (0.34, 1.47)
African origin	28590	.		60	0.74 (0.58, 0.95)	.		10	0.63 (0.3, 1.32)
Chinese	68010	.		170	0.82 (0.7, 0.96)	10	0.81 (0.37, 1.8)	15	0.58 (0.35, 0.96)

Continued...

Sex and ethnic groups	PY	HIV related	
		Number of events	Age-adjusted RR (95% CI)
MALES			
White Scottish	21179755	415	1
Other White British	1571080	30	0.84 (0.57, 1.22)
White Irish	202190	.	
Other White	278515	15	2.06 (1.16, 3.68)
Any Mixed Background	56265	.	
Indian	65945	.	
Pakistani	146430	.	
Other South Asian	35500	.	
African origin	32160	10	8.30 (3.69, 18.68)
Chinese	68685	.	
FEMALES			
White Scottish	22581190	165	1
Other White British	1644435	10	0.74 (0.37, 1.47)
White Irish	216905	.	
Other White	319915	.	
Any Mixed Background	59970	.	
Indian	59925	.	
Pakistani	143940	.	
Other South Asian	28610	.	
African origin	28590	15	74.85 (45.56, 122.96)
Chinese	68010	.	

Appendix 6.2.a. Risk Ratios (RR) and 95% confidence intervals (CI) of multimorbidity by ethnicity, for males. Models are adjusted for age (model 1), and subsequently for SRH (Bad health - No versus Yes) (model 2), the interaction between SRH and ethnicity (model 3) and SES (model 4)

Ethnicity	PY	Multi-morbidity	Model 1		Model 2		Model 3		Model 4	
			Adjusted for age		Adjusted for age and SRH		Adjusted for age, SRH and ethnicity* SRH (0)		Adjusted for age, SRH, ethnicity* SRH (0) and SES	
			RR (95% CI)	p-value	RR (95% CI)	p-value	RR (95% CI)	p-value	RR (95% CI)	p-value
White Scottish	21179755	58895	1		1		1		1	
Other White British	1571080	3845	0.73 (0.69, 0.76)	<.0001	0.76 (0.73, 0.79)	<.0001	0.79 (0.74, 0.84)	<.0001	0.90 (0.84, 0.95)	0.0004
White Irish	202190	790	1.01 (0.94, 1.09)	0.7688	0.96 (0.89, 1.02)	0.1907	0.92 (0.82, 1.03)	0.1327	0.90 (0.80, 1.01)	0.0609
Other White	278515	520	0.78 (0.71, 0.85)	<.0001	0.78 (0.71, 0.85)	<.0001	0.86 (0.74, 0.99)	0.0355	0.92 (0.80, 1.07)	0.2823
Any Mixed Background	56265	50	1.04 (0.79, 1.38)	0.7649	1.02 (0.79, 1.33)	0.8635	1.50 (1.03, 2.21)	0.0372	1.51 (1.03, 2.20)	0.0338
Indian	65945	120	0.98 (0.83, 1.17)	0.8459	0.95 (0.80, 1.13)	0.5426	0.87 (0.65, 1.18)	0.3777	1.06 (0.78, 1.43)	0.7243
Pakistani	146430	245	1.31 (1.15, 1.48)	<.0001	1.13 (0.99, 1.28)	0.0695	0.99 (0.81, 1.20)	0.8795	1.08 (0.88, 1.32)	0.4615
Other South Asian	35500	50	0.98 (0.75, 1.28)	0.9038	0.95 (0.72, 1.24)	0.6823	0.99 (0.64, 1.55)	0.9789	1.05 (0.67, 1.63)	0.8450
African origin	32160	40	0.93 (0.68, 1.27)	0.6488	0.92 (0.68, 1.24)	0.5774	0.85 (0.48, 1.50)	0.5715	0.86 (0.49, 1.54)	0.6200
Chinese	68685	65	0.58 (0.46, 0.74)	<.0001	0.62 (0.48, 0.79)	<.0001	0.53 (0.31, 0.90)	0.0179	0.57 (0.33, 0.96)	0.0338
SRH- Bad health (No versus Yes)					0.38 (0.37, 0.39)	<.0001	0.38 (0.37, 0.39)	<.0001	0.43 (0.42, 0.44)	<.0001
Other White British * SRH							0.95 (0.88, 1.03)	0.2053	0.95 (0.88, 1.02)	0.1620
White Irish * SRH							1.07 (0.93, 1.23)	0.3812	1.06 (0.92, 1.22)	0.4064
Other White * SRH							0.86 (0.72, 1.03)	0.1074	0.86 (0.72, 1.03)	0.0974
Any Mixed Background * SRH							0.53 (0.31, 0.90)	0.0194	0.53 (0.31, 0.90)	0.0179
Indian * SRH							1.13 (0.78, 1.64)	0.5122	1.10 (0.76, 1.59)	0.6249
Pakistani * SRH							1.28 (0.99, 1.65)	0.0627	1.20 (0.93, 1.56)	0.1665
Other South Asian * SRH							0.93 (0.53, 1.62)	0.7857	0.94 (0.54, 1.64)	0.8213
African origin * SRH							1.12 (0.57, 2.19)	0.7495	1.11 (0.56, 2.19)	0.7599
Chinese * SRH							1.22 (0.67, 2.20)	0.5211	1.15 (0.63, 2.07)	0.6543

SIMD (1 vs 5-least deprived)	1.41 (1.35, 1.47)	<.0001
SIMD (2 vs 5-least deprived)	1.28 (1.22, 1.33)	<.0001
SIMD (3 vs 5-least deprived)	1.19 (1.14, 1.24)	<.0001
SIMD (4 vs 5-least deprived)	1.08 (1.03, 1.13)	0.0012
Household tenure (own vs. rent)	1.24 (1.21, 1.27)	<.0001
Highest qualification (higher vs. no)	0.80 (0.78, 0.83)	<.0001
Highest qualification (lower vs. no)	0.92 (0.90, 0.95)	<.0001

Appendix 6.2.b. Risk Ratios (RR) and 95% confidence intervals (CI) of multimorbidity by ethnicity, for females. Models are adjusted for age (model 1), and subsequently for SRH (Bad health - No versus Yes) (model 2), the interaction between SRH and ethnicity (model 3) and SES (model 4)

Ethnicity			Model 1		Model 2		Model 3		Model 4	
			Adjusted for age		Adjusted for age and SRH		Adjusted for age, SRH and ethnicity* SRH (0)		Adjusted for age, SRH, ethnicity* SRH (0) and SES	
			RR (95% CI)	p-value	RR (95% CI)	p-value	RR (95% CI)	p-value	RR (95% CI)	p-value
White Scottish	22581190	50290	1		1		1		1	
Other White British	1644435	3010	0.73 (0.69, 0.76)	<.0001	0.76 (0.73, 0.79)	<.0001	0.79 (0.73, 0.84)	<.0001	0.90 (0.84, 0.96)	0.0022
White Irish	216905	675	0.93 (0.85, 1.01)	0.0900	0.89 (0.82, 0.96)	0.0023	0.86 (0.76, 0.97)	0.0108	0.86 (0.76, 0.97)	0.0146
Other White	319915	505	0.79 (0.72, 0.87)	<.0001	0.80 (0.74, 0.88)	<.0001	0.78 (0.67, 0.91)	0.0012	0.86 (0.74, 1.00)	0.0521
Any Mixed Background	59970	55	1.15 (0.88, 1.52)	0.3102	1.08 (0.84, 1.40)	0.5331	1.25 (0.85, 1.82)	0.2570	1.20 (0.82, 1.76)	0.3512
Indian	59925	65	0.87 (0.68, 1.12)	0.2858	0.75 (0.59, 0.95)	0.0191	0.77 (0.55, 1.09)	0.1383	0.93 (0.66, 1.32)	0.6970
Pakistani	143940	185	1.47 (1.27, 1.70)	<.0001	1.14 (0.99, 1.31)	0.0633	1.11 (0.92, 1.33)	0.2795	1.23 (1.03, 1.48)	0.0240
Other South Asian	28610	30	0.97 (0.68, 1.39)	0.8657	0.89 (0.63, 1.27)	0.5330	0.92 (0.54, 1.57)	0.7524	0.99 (0.58, 1.69)	0.9647
African origin	28590	30	0.93 (0.66, 1.31)	0.6720	0.92 (0.65, 1.30)	0.6152	0.99 (0.56, 1.76)	0.9717	1.03 (0.58, 1.83)	0.9314
Chinese	68010	55	0.68 (0.52, 0.89)	0.0050	0.68 (0.53, 0.87)	0.0026	0.68 (0.43, 1.08)	0.0991	0.74 (0.47, 1.16)	0.1914
SRH- Bad health (No versus Yes)					0.34 (0.33, 0.35)	<.0001	0.34 (0.33, 0.36)	<.0001	0.39 (0.38, 0.41)	<.0001
Other White British * SRH							0.95 (0.87, 1.04)	0.2709	0.94 (0.87, 1.03)	0.1668
White Irish * SRH							1.06 (0.91, 1.24)	0.4670	1.05 (0.90, 1.22)	0.5578
Other White * SRH							1.05 (0.87, 1.26)	0.6263	1.06 (0.88, 1.27)	0.5684
Any Mixed Background * SRH							0.79 (0.48, 1.32)	0.3662	0.82 (0.49, 1.36)	0.4398
Indian * SRH							0.95 (0.59, 1.52)	0.8165	0.89 (0.55, 1.43)	0.6233
Pakistani * SRH							1.07 (0.81, 1.42)	0.6167	0.98 (0.74, 1.29)	0.8707
Other South Asian * SRH							0.96 (0.47, 1.95)	0.9030	0.90 (0.44, 1.84)	0.7710
African origin * SRH							0.89 (0.43, 1.83)	0.7462	0.89 (0.43, 1.83)	0.7459
Chinese * SRH							0.99 (0.57, 1.71)	0.9781	0.96 (0.56, 1.65)	0.8727
SIMD (1 vs 5-least deprived)									1.48 (1.42, 1.55)	<.0001

SIMD (2 vs 5-least deprived)	1.31 (1.26, 1.37)	<.0001
SIMD (3 vs 5-least deprived)	1.24 (1.19, 1.30)	<.0001
SIMD (4 vs 5-least deprived)	1.09 (1.05, 1.15)	<.0001
Household tenure (own vs. rent)	1.24 (1.20, 1.27)	<.0001
Highest qualification (higher vs. no)	0.80 (0.77, 0.83)	<.0001
Highest qualification (lower vs. no)	0.88 (0.86, 0.91)	<.0001