History, politics and vulnerability: explaining excess mortality in Scotland and Glasgow

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Preface

Scotland experiences high levels of 'excess' mortality: that is, higher mortality *over and above* that explained by the country's socioeconomic profile. Compared with England & Wales, and adjusting for differences in poverty and deprivation (the main causes of poor health in any society), 5,000 more people die every year in Scotland than should be the case.

This excess plays a major role in explaining why Scotland has both the lowest life expectancy, and the widest mortality inequalities, in Western Europe. Although usually expressed in statistical terms (such as standardised rates or ratios or expected years of life), behind such summary epidemiological expressions lie genuine human tragedies: individual stories of shortened, wasted lives, pain, sickness, early death and grief, affecting individual men, women and children, their families, friends and communities.

This report seeks to summarise all the research that has been undertaken into this phenomenon, with the aim of achieving a greater understanding of its most likely underlying causes and, therefore, the most appropriate responses. As the report makes clear, however, such responses need to be entwined with ever more urgent actions to address the key drivers of overall poor health in the country – poverty and deprivation – and to seek to narrow the widening gaps in income, power, wealth and, therefore, health in Scottish society.

The conclusions of the report, including a list of policy recommendations, have been endorsed by a wide range of experts in public health and other disciplines, who are listed below. Together with these signatories, we implore action on the part of both national and local government to address the many issues highlighted in this research.

Signatories

The principal findings of this report, including the set of resulting policy recommendations, have been endorsed by the following individuals:

- Professor Nick Bailey, Professor of Urban Studies, University of Glasgow
- Professor Clare Bambra, Professor of Public Health Geography, Durham University
- Professor Sir Harry Burns, Professor of Global Public Health, University of Strathclyde
- Colin Cox, Former Deputy Director of Public Health, Manchester
- Dr Peter Craig, Senior Research Fellow, MRC/CSO Social & Public Health Sciences Unit, University of Glasgow
- Fiona Crawford, Consultant in Public Health, NHS Greater Glasgow & Clyde/Glasgow Centre for Population Health
- Professor Mike Danson, Professor of Enterprise Policy, Heriot-Watt University
- Professor Sir Tom Devine, Emeritus Chair of Scottish History and Palaeography, The University of Edinburgh
- Professor Danny Dorling, Professor of Geography, University of Oxford
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- Dr Andrew Fraser, Director of Public Health Science, NHS Health Scotland
- Dr Lisa Garnham, Public Health Research Specialist, Glasgow Centre for Population Health
- Professor Phil Hanlon, Honorary Senior Research Fellow (formerly Professor of Public Health), University of Glasgow
- Professor Annette Hastings, Professor of Urban Studies, University of Glasgow
- Dr S. Vittal Katikireddi, Senior Clinical Research Fellow, MRC/CSO Social & Public Health Sciences Unit, University of Glasgow
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- Professor Mhairi Mackenzie, Professor of Public Policy, University of Glasgow
- Professor Alison McCallum, Director of Public Health and Health Policy, NHS Lothian, and Chair of Scottish Directors of Public Health
- Dr Jennifer McLean, Public Health Programme Manager, Glasgow Centre for Population Health
- Professor Rich Mitchell, Professor of Health and Environment, University of Glasgow, and Head of Public Health & Co-Director of the Centre for Research on Environment, Society and Health (CRESH)

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- Dr Michael Smith, Associate Medical Director for Mental Health, NHS Greater Glasgow and Clyde
- Dr Katherine Trebeck, Senior Researcher, Oxfam GB Global Research Team, and Honorary Professor, University of the West of Scotland
- Dr David Taylor-Robinson, Senior Clinical Lecturer in Public Health, University of Liverpool
- Professor Jim Tomlinson, Professor of Economic and Social History, University of Glasgow
- Bruce Whyte, Public Health Programme Manager, Glasgow Centre for Population Health
- Dr Andrea Williamson, GP and Director of Community Based Education (Strategy and Innovation), School of Medicine, University of Glasgow

Executive summary

The poor health profiles of Scotland, and especially that of its largest city, Glasgow, are well known. Much of this is explained by recent experiences of deindustrialisation, deprivation and poverty: the latter are the root causes of poor health in all societies, not just Scotland. However, in addition, high levels of *excess* mortality – that is, higher mortality *over and above that* explained by differences in socioeconomic deprivation – have been observed for Scotland compared with England & Wales, as well as for Glasgow compared with similar post-industrial UK cities such as Liverpool, Manchester and Belfast.

The scale of this excess is considerable. It accounts for approximately 5,000 extra, 'unexplained', deaths per year in Scotland, and makes a substantial contribution to the other principal mortality 'phenomena' associated with Scotland in recent times: the lowest, and most slowly improving, life expectancy in Western Europe; the widest mortality inequalities in Western Europe; and the persistently high rates of mortality among those of younger working ages. After adjustment for differences in deprivation, premature mortality (<65 years) in Scotland is 20% higher than in England & Wales (10% higher for deaths at all ages); similarly, the excess for Glasgow compared with Liverpool, Manchester and Belfast has been shown to be approximately 30% for premature mortality, and around 15% for deaths at all ages.

The key features of this excess are:

- it is observed in all parts of Scotland compared with the rest of Great Britain, but is greatest in and around the post-industrial West Central Scotland (WCS) conurbation and, in particular, Glasgow
- it is increasing over time
- it is seen across all adult age groups, but is highest among those of working age (especially younger working age)
- it is observed across all social classes, although for premature mortality, it is more pronounced in comparisons of the poorest populations
- it is observed for a broad range of causes of death, although with important distinctions between excess *premature* mortality (particularly influenced by higher rates of death from alcohol, drugs and suicide) and excess mortality *at all ages* (driven particularly by higher numbers of deaths from cancer, heart disease and stroke)
- and given the relationship between socioeconomic factors and health behaviours, the excess persists even after statistical adjustment for differences in behaviours such as smoking, physical activity, diet etc.

A great many potential explanations have been proposed to explain this extremely complex phenomenon. A previous report published in 2011 summarised and assessed a range of potential explanations, and attempted a synthesis of the most likely causes. That synthesis, however, was hindered by a lack of available evidence for many of the proposed theories. Since then a considerable amount of further research has been carried out, including a number of new projects undertaken in support of the *new* synthesis of evidence which is the focus of this report. The ultimate aim of this new work is to provide a much greater understanding of the causes of, and therefore the most appropriate responses to, Scotland's and Glasgow's high levels of excess mortality.

A total of 40 potential explanations for Scottish excess mortality have been examined, based on an assessment of evidence that has been gathered over many years. On the basis of these assessments, two explanatory models have been developed: one for Glasgow (based on comparison with Liverpool and Manchester – both having been shown to be excellent comparator cities), and one for Scotland (based on comparison with England & Wales).

Both models are 'anchored' in important contextual knowledge. This includes the importance of key exposures for adverse population health in terms of poverty, deprivation and deindustrialisation. These sit alongside, and are related to, UK economic and social policies since the late 1970s which have resulted in a widening of inequalities across the UK in terms of both socioeconomic and – as a consequence – health characteristics. As part of that process, post-industrial, deprived cities such as Glasgow, Liverpool and Manchester are placed at the 'wrong' end of that spectrum of inequality, exhibiting the highest rates of both poverty and mortality. However, over the same decades, two further, less easily explained, outcomes have been observed. The first is that differences in poverty and deprivation no longer explain the mortality gap between Scotland and the rest of Britain. Second, there has been a similarly unexplained divergence in mortality between Glasgow and the two English comparator cities. The explanatory models in the report are, therefore, focused upon identifying the factors (so-called 'effect modifiers') which are likely to have brought about these additional adverse outcomes.

Key to **the explanatory model for Glasgow** is that the city, over time, was made **more vulnerable** to the particular socioeconomic and political exposures mentioned above. The concept of vulnerability has been shown to be important in understanding differences in health between populations (and across different sections of populations). For Glasgow, the heightened vulnerability has been generated by a series of historical processes which have cumulatively impacted on the city's population. These include:

- The lagged effect of high historical levels of deprivation: although analyses of historical income and employment based measures of deprivation show few differences between Glasgow, Liverpool and Manchester over many decades, compared with these English cities, Glasgow (alongside other Scottish areas) endured notably higher levels of deprivation, as evidenced by overcrowding, from at least the middle of the 20th century. This may represent an underlying vulnerability.
- A further level of vulnerability resulted from Scottish Office regional policy from the later 1950s, including the socially selective New Town programme. Policy was aimed at relocating both industry and a section of the population (generally younger, skilled workers, in employment, and often with families) to New Towns and other growth areas across central Scotland, away from what had been designated a 'declining' city, as part of a wider regional 'modernisation' agenda focused on attracting lighter industries. These other areas became the key priority in terms of investment, and this policy was extended and expedited over the ensuing decades

despite awareness of the negative consequences (both socioeconomic and also ultimately health-related) for Glasgow.

- Closely related to this evolving regional policy agenda, the nature (and scale) of urban change experienced within Glasgow in the post-war period (1945-1980) was different to that in the comparator cities. This is relevant to population health in terms of social determinants such as housing, living conditions and social and community networks. Glasgow differed from the comparator cities in terms of: larger-scale slum clearances and demolitions; larger within-city (poor quality) peripheral council house estates; greater emphasis on high-rise development; and crucially, much lower per capita investment in housing repairs and maintenance of the public housing stock.
- Differences in local government responses to UK government economic policy in the 1980s also had impacts. Research suggests that in Glasgow, local responses, in their early prioritisation of inner-city gentrification and commercial development, potentially exacerbated the damaging impacts of UK policy on what was already a vulnerable population. In the other cities, however, responses were more likely to have mitigated these damaging impacts, either by slowing them (Manchester) or by mobilising local opposition against them (Liverpool). In the latter case, the city-level response fostered widespread participation and politicisation of the Liverpool public and, as a consequence, local government gave greater priority at an important stage to dealing with important social issues (e.g. addressing poverty, building new council housing and public amenities) than was the case in Glasgow. Thus, differences in responses brought about protective factors in the comparator populations relative to Glasgow.
- A further resulting protective factor (related to these historical processes of politicisation, participation and associated factors such as strengthening of community ties) is higher levels of what is often referred to as **social capital** (or social fabric) in Liverpool as compared with Glasgow.
- More speculatively, the research suggests that other protective factors may be operating in Manchester e.g. in terms of the city having a greater level of ethnic diversity (and the healthy migrant effects with which that is likely to be associated).
- Alongside, and entwined with, the 1980s processes highlighted above, the vulnerability of the Scottish (including Glaswegian) population was potentially enhanced by the negative impact of the so-called 'democratic deficit' of that period, characterised by feelings of despondency, disempowerment, and lack of sense of control (recognised 'psychosocial' risk factors with links to adverse health outcomes).
- A further major component of the model (although one that is more a core determinant of health rather than an 'effect modifier') is the inadequate measurement of poverty and deprivation: that is, that despite many different measures of deprivation and socioeconomic circumstances having been used in

analyses of excess mortality to date, these measures fail to capture sufficiently differences in the complex, multi-dimensional, 'lived reality' of deprivation and poverty in Scotland, and especially in Glasgow, compared with elsewhere in Great Britain and the UK. It seems likely that aspects of the vulnerability-inducing historical processes described above are highly relevant to this.

- It is likely that unmeasured aspects of deprivation potentially also include a more **negative physical environment** (specifically in relation to levels of vacant and derelict land), as well as aspects of **educational attainment** (although the contribution of the latter in particular is small).
- The synthesis also points to a number of **smaller**, **additional factors**, the individual impacts of which are likely to be very small, but which cumulatively may be relevant to particular aspects of population health.

The **<u>explanatory model for Scotland</u>** as a whole is made up of various components, including:

- the model for Glasgow in its entirety, given the extent to which that impacts on the national level of excess mortality.
- particular elements of the Glasgow model which are also highly (in some cases more) relevant to Scotland as a whole. These include: the inadequate measurement of deprivation; the lagged effects of deprivation (in particular higher levels of overcrowding historically); and key vulnerabilities, including the so-called democratic deficit, as well as other aspects of Scottish Office regional economic policy in the post-war period which not only had a detrimental effect on Glasgow, but failed to deliver anticipated benefits elsewhere in the country.
- Additional factors including a more profound experience of deindustrialisation compared with England & Wales, and some differences in (potentially culturally-influenced) 'downstream' health determinants such as diet.

Implications for policy

A key point emphasised throughout the report, and elsewhere, is that **economic policies matter for population health**. In response to the evidence presented in the report, therefore, a number of recommendations for policy (in particular economic policy) are listed. These emphasise the need to address three issues simultaneously:

- to protect against key exposures (e.g. poverty, deprivation) which impact detrimentally across the whole UK (but especially in places like Glasgow, Liverpool and Manchester)
- to address the *existing* consequences of Glasgow's vulnerability
- and further, to mitigate against the effects of *future* vulnerabilities which are likely to emerge from UK government changes to social security and reduced public spending.

The important factors which emerge from this analysis – **poverty and deprivation**, and **exacerbated inequality** linked to current, past and future **vulnerabilities** – are intractably entwined. Thus the policy recommendations in the report seek to address all these issues in unison, including – specifically – the need to narrow inequalities in income and wealth in order to narrow inequalities in health in Scottish society. The recommendations are drawn from different sources: some follow directly from specific research findings; some reflect existing evidence of appropriate responses to issues highlighted in the report; and others have been proposed by others with expertise in the relevant policy areas. They are listed under four headings:

- National (Scottish) economic and social policy. Given all the evidence that economic policies have profound implications for population health, the report urges that all opportunities available within Scotland are taken to redistribute income and wealth across Scottish society. Specific measures relating to ownership of capital, income and corporate taxation, wealth and asset taxation, 'fair work' (including adequate wage levels), industrial policy, social security, addressing the cost of living, and 'poverty-proofing' Scottish Government policies are all set out.
- Housing and the physical environment. These include recommendations in relation to: expanding the social housing building programme; extending the Scottish Housing Quality Standard; targeting cold and damp housing and fuel poverty; strengthening the impact of the Place Standard for Scotland; and improving greenspace access and quality in deprived areas.
- 3. Local government actions. These include: the need to recognise, and act upon at the highest levels of local government the impact of local decision making on population health; the role of local government in redistributing resources towards areas of greater need; a review of the boundaries and/or the funding allocation system for local government; a 'poverty proofing' approach to local government policy-making; further actions to narrow inequalities at the local level; and specific to Glasgow (and a number of other local authorities), consideration should be given as to how to maximise the potential of the recent 'City Deals' investment to mitigate against the effects of vulnerability in the population.
- 4. **Understanding deprivation**: specifically, that there is an urgent need to prioritise further research on the true nature and experience of deprivation in Scotland that does not seem to be captured by existing data and measurements.

A number of weaknesses associated with the synthesis, and resulting explanatory models, are acknowledged in the report. These principally relate to the fact that assessment of some hypotheses is still hindered by a lack of robust evidence and data. A greater number of these 'unknowns' relate to comparisons of Scotland with England & Wales, meaning that there is less certainty around whether and how far some of the important vulnerabilities highlighted in the Glasgow model also apply to areas that lie outside the West Central Scotland conurbation. Despite these, and other weaknesses, however, we believe the report has helped to further our understanding of the underlying causes of Scotland's and Glasgow's excess mortality. What is important now is that there is an appropriate response to that

evidence in order to improve the health of Scotland's population. This must be done alongside, and entwined with, ever more urgent actions to address the key drivers of overall poor health in the country – poverty and deprivation – and to seek to narrow the widening gap in wealth and, therefore, health in Scottish society.

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Report overview

As the title states, this report seeks to synthesise all the evidence for, and the likely causes of, Scottish excess mortality. In doing so, it seeks to develop appropriate responses for policy-makers in Scotland.

The structure of the report is as follows:

- **Part One** explains exactly what we mean by the term 'excess mortality', describing its many complex components
- **Parts Two and Three** detail, respectively, the precise aims of, and methodologies employed in, the research described in this report
- **Part Four** briefly presents important contextual information regarding excess mortality in Scotland
- Part Five (the main part of the report) presents two explanatory models based on an in-depth assessment of a wealth of evidence – for the high levels of excess mortality seen in both Scotland and its largest city, Glasgow
- **Part Six** discusses those models in more detail, including an assessment of the extent to which they explain key features of excess mortality, and the strengths and weaknesses associated with the approach taken
- **Part Seven** outlines the implications for policy
- **Part Eight** presents overall conclusions and recommendations for future research.

The many individual hypotheses that have been proposed to explain excess mortality in Glasgow and Scotland, the assessment of which lies at the heart of this synthesis report, are outlined in detail in **Appendix A**.

1. Introduction

1.1 What do we mean by excess mortality?

Scotland's poor health status relative to the rest of the UK and other parts of Europe has been well documented^{1,2}, as, especially, has that of its largest city, Glasgow³⁻⁶. As is discussed in more detail in Part Four of this report, this is evidenced by Scotland having the lowest, and most slowly improving, life expectancy in Western Europe, and – related to this – the widest social inequalities in health in Western Europe⁷⁻⁹.

Given its importance as the fundamental driver of poor health in *any* population, socioeconomic deprivation (linked to the impact of post-industrial decline) has, correctly, been the focus of traditional explanations for the high rates of Scottish mortality¹⁰⁻¹³. Levels of poverty have, historically, been higher in Scotland compared with elsewhere in Great Britain, and the high levels of deprivation in and around Glasgow in particular are well established^{3-6,14-17}. Indeed across Eastern and Western Europe, mortality rates tend to be highest in the poorest, and deindustrialised, regions: Glasgow and Scotland are not alone in that regard¹⁸⁻²⁰.

However, despite the importance of this explanation, a plethora of research evidence suggests it is not sufficient to explain the *particularly* poor health profile of Scotland as a whole, nor that of Glasgow and its surrounding post-industrial region in particular. Mortality rates in Scotland are substantially higher than in England and Wales *even after taking account of* differences in levels of deprivation among the respective populations²¹⁻²⁵. This is true of both the resident population, and of those who have moved to elsewhere in the UK^{22,23,26}, and has been shown when taking into account differences in *area* (neighbourhood) measures of poverty/deprivation, as well as *individual* socioeconomic status (e.g. social class, educational attainment). This higher mortality also persists after further adjustment for differences between countries in a range of well-established behavioural (e.g. smoking status, alcohol consumption, diet, physical activity) and biological (e.g. body mass index, blood pressure) risk factors.

Although this unexplained excess level of mortality has been shown to exist in all parts of Scotland compared with the rest of Great Britain, it is highest in and around Glasgow^{3,21,27,28}. Mortality in West Central Scotland (WCS)ⁱ is higher, and improving more slowly, than in all comparably deindustrialised regions in Europe: these include regions in the UK such as the Welsh ex-coalfields areas and Merseyside, areas with similar levels of poverty and economic histories as WCS in recent decades^{18,19}. Most strikingly, research published in 2010 showed the deprivation profiles of Glasgow, Liverpool and Manchester to be very similar: yet despite this, and after adjusting for any remaining differences in deprivation between the cities, premature mortality (<65 years) in Glasgow was shown to be 30% higher than in the English

ⁱ WCS is an approximate, descriptive term, rather than an officially used administrative geography. However, in previous population-based studies^{4,18}, it has been defined by 11 local authority areas: East Ayrshire, East Dunbartonshire, East Renfrewshire, Glasgow City, Inverclyde, North Ayrshire, North Lanarkshire, Renfrewshire, South Ayrshire, South Lanarkshire, and West Dunbartonshire.

cities, with deaths at all ages around 15% higher^{29,30 ii}. Near-identical results were obtained from a similarly in-depth comparison of deprivation and mortality between Glasgow and Belfast³¹.

As these figures show, the scale of the excess is striking. The city analyses showed that even when compared with Liverpool and Manchester – cities with the lowest life expectancy in England – Glasgow experienced an additional 4,500 deaths over the five year period examined (2003-2007). The most recent analyses of Scotland compared with England & Wales showed that once deprivation had been accounted for, an average of approximately 5,000 extra deaths in Scotland occurred *every year* between 2010 and 2012. Similar figures have been shown in comparisons of the West Central Scotland conurbation with other deindustrialised areas of Europe such as Northern Ireland and parts of Eastern Germany.

Importantly, the excess mortality is increasing over time. In 1981, after adjustment for differences in deprivation, all-cause mortality in Scotland was approximately 4% higher than in England & Wales. Three decades later, the excess had more than doubled to 10% (20% for deaths under 65 years) (Figure 1)^{25 iii}. There is clear evidence of a similar widening of the excess observed in Glasgow compared with Liverpool and Manchester since the mid to late 1970s. As stated above, mortality rates in WCS are improving more slowly than in other, similarly disadvantaged, post-industrial European regions.

Figure 1: Relative difference in mortality rates between Scotland and England & Wales, all ages and age <65 years, 1981-2011 (Source: Schofield *et al.*, 2016) (note different scales on the y axis of each chart).



ⁱⁱ As is explained later in the report, Liverpool and Manchester are, for a number of historical, socioeconomic and demographic reasons, excellent comparator cities for analysis of excess mortality in Glasgow.

^{III} Figure 1 shows the *relative* differences between the mortality rates of Scotland and England & Wales (i.e. the difference in rates expressed as a percentage). Between 1981 and 2011, overall mortality rates decreased in both Scotland and England & Wales. However, they did so to a slightly greater degree in England & Wales than in Scotland. After adjustment for age and sex only, the *absolute* difference in rates decreased (from 18.2 to 15.0), but – as shown with the green bars in the figure – the relative difference increased steadily from 11% to 15%. After further adjustment for deprivation, the *absolute* difference increased from 6.8 to 10.1 between c.1981 and c.2011 (with the biggest increase between 1981 (6.8) and 1991 (10.2)), but the relative difference (the percentage excess shown by the purple bars in the figure) increased from 4% higher in c.1981 to 10% higher in c.2011. Both absolute and relative differences are important, but Scottish excess mortality tends to be defined in terms of relative differences.

The excess is observed in analysis of a wide range of different causes of death. For example, in 2010-12, the excess in Scotland was, respectively, 11%, 16% and 24% for deaths from all cancers combined, ischaemic heart disease and stroke, but as high as 54% for alcohol-related causes, 74% for suicide and almost 250% for drug-related deaths. Similar differences were noted in comparisons of Glasgow with Liverpool and Manchester in the mid-2000s: for example, after adjusting for age, sex and deprivation, approximately 12% higher mortality for deaths from heart disease and stroke, and from all cancers, but almost 70% higher for suicide, and 2.3 and 2.5 times higher for alcohol- and drug-related deaths respectively (Figure 2).

Figure 2: Cause-specific excess mortality, Glasgow relative to Liverpool & Manchester (Source: Walsh *et al.*, 2010).



This mortality excess is a complex phenomenon. Not only is it observed in analyses of different causes of death, it is seen across gender, adult age groups, and social strata (as evidenced by people living in affluent versus deprived areas). This notwithstanding, the mortality excess is greatest among those of working age (especially younger working ages) and those living in the poorest neighbourhoods. There is also a distinction in this regard between premature mortality (defined here as deaths <65 years) and deaths at all ages. For example, the city-based analyses showed that for the latter (deaths at all ages) the 15% higher mortality observed in Glasgow was distributed fairly evenly across all deprivation deciles, with the greatest contribution (in terms of causes of death) from cancers, heart disease and stroke. For premature mortality, however, the excess was much higher in comparisons of those living in the more, rather than less, deprived areas (particularly men), and was driven in particular by higher rates of death from alcohol, drugs and suicide.

1.2 What's the focus: Glasgow or Scotland?

The answer to this question is: both. Part of the reason for this relates to population size and geographical scale. In a small country of only five million people, Glasgow (as defined by its local authority boundary) accounts for 11% of the total population of Scotland; the wider WCS conurbation contains over 40%. Thus what is observed in and around Glasgow impacts on national figures. In health terms, the same negative characteristics associated with Glasgow (e.g. poor, and more slowly improving, mortality rates compared with elsewhere in the UK) apply also to all other parts of Scotland, but in a less pronounced form. This is also true of excess mortality: as stated above, this has been shown for all parts of Scotland compared with elsewhere in Britain, but is greatest in and around the country's largest city. It is unlikely, therefore, that the underlying causes of Scotland's and Glasgow's excess levels of mortality are entirely dissimilar or unrelated, albeit that some important factors specific to, or impacting in a heightened fashion upon, the city may be implicated.

Furthermore, many of the important contextual factors that will be described later in the report (deprivation, deindustrialisation, widening of inequalities in society in recent decades) have affected many parts of Scotland, not just Glasgow. Thus, it is right that both Glasgow and Scotland are the focus for the various population-based investigations that are discussed within this report.

It is also important to note that in this report we focus principally on comparisons of Scotland and Scottish areas (Glasgow, WCS) with *other parts of the UK*. We do *not* include comparisons of Glasgow with other parts of Scotland. Some research has sought to investigate excess mortality in Glasgow (or a so-called 'Glasgow effect') by comparing the city with Scotland as a whole, or with other parts of the country^{28,32,33}. However, as excess mortality has been defined in terms of comparison with England & Wales, and as that excess has been shown to be ubiquitous in Scotland, such comparisons *within* Scotland represent a different epidemiological issue.

Finally, although the focus of this research is Scotland and Glasgow, as evidence of this type of excess mortality has also been demonstrated outside Scotland (e.g. in comparison of northern English cities and regions³⁴⁻³⁶), explanations for the excess are likely to be relevant to research elsewhere.

1.3 What's the focus: mortality or morbidity?

As the title of the report suggests, the focus is on mortality. The reason for this mainly relates to the issue of measurement. Morbidity in whole populations can be assessed principally in two ways: from disease registers, and from self-reported measures (e.g. in population surveys). For comparisons across different countries, the former are limited by issues related to comparability (e.g. of different systems, recording methods) and availability. The latter are also subject to difficulties. On the one hand, some studies have shown that self-reported measures of general health status can be good predictors of subsequent mortality³⁷⁻³⁹. On the other hand, however, other analyses have pointed to important demographic, socioeconomic and cultural factors which can influence self-assessment of health⁴⁰⁻⁴⁴. At the population level, disparities between measures of self-

assessed health and mortality have been shown internationally^{45,46} including, importantly, within the UK. A number of analyses have shown that Scottish populations tend to underreport levels of self-assessed morbidity compared with other parts of the UK, and compared with actual levels of illness reflected in relatively higher rates of mortality⁴⁷⁻⁴⁹. Mortality, therefore, is a 'better' health outcome in this sense – although clearly it will fail to capture many other, highly important, aspects of population health (e.g. aspects of mental health and wellbeing, or particular diseases unrelated to death).

1.4 Synthesising the evidence

The key features of Scottish excess mortality described above are summarised in Box 1. From the many analyses undertaken to date, it seems clear that the complexity of this phenomenon will require an equally complex and multifactorial explanation. A great many such potential explanations have been proposed to explain the excess seen in Scotland compared with England & Wales, and, more particularly, in Glasgow compared with Liverpool and Manchester. Work carried out in 2010 (published in a joint report by the Glasgow Centre for Population Health and NHS Health Scotland in 2011⁵⁰ and a journal paper in 2012⁵¹) sought to summarise and assess the principal hypotheses that had been suggested by that point. No fewer than 17 explanations were included, with a synthesis of the most likely causes attempted. That synthesis, however, was hindered by a lack of available evidence for many of those potential explanations. Since publication, however, a considerable amount of further research in this area has been undertaken, partly in relation to quantifying the excess itself, but especially in relation to examining the evidence relating to these, and other, suggested hypotheses 5^{2-69} . There is a clear need, therefore, to update the previous synthesis based on all this new knowledge, with the ultimate aspiration of providing a much greater understanding of the causes of Scotland's excess mortality. This is the aim of this report.

Box 1. The key features of Scottish excess mortality.

- Mortality is considerably higher in Scotland relative to England & Wales, and in Glasgow relative to Liverpool and Manchester, after accounting for differences in neighbourhood deprivation and individual socioeconomic status (SES) of the populations.
- After adjustment for deprivation/SES, premature mortality rates are 20% higher in Scotland, compared with England & Wales, and 30% higher in Glasgow compared with Liverpool and Manchester. The equivalent figures for mortality at all ages are 10% and 15% respectively.
- This excess is observed in all parts of Scotland compared with the rest of Great Britain, but is greatest in and around the West Central Scotland (WCS) conurbation and, in particular, Glasgow.
- The excess is increasing over time.

- The excess is seen across all adult age groups, but is highest among those of working age (15-64 years), and especially younger working age (15-44 years) (although in absolute terms, deaths at younger ages obviously account for a smaller proportion of the total).
- The excess is observed across all deprivation groups/social classes, although for premature mortality, it is more pronounced in comparisons of the poorest/most deprived populations.
- The excess is observed for a broad range of causes of death.
- There are differences in the contribution of particular causes of death to the excess observed at all ages (driven particularly by higher numbers of deaths from cancer, heart disease and stroke) compared with the excess observed for deaths among working ages (over half of which relate to deaths from alcohol, drugs, suicide and violence).
- Given the relationship between socioeconomic factors (e.g. deprivation) and health behaviours, the excess persists even after statistical adjustment for differences in behaviours such as smoking, physical activity, diet etc.

2. Aims and research questions

The overall aim of this report is to provide an updated and expanded synthesis of the evidence for, and the most likely causes of, excess mortality in Scotland and Glasgow, and an assessment of the best policy responses.

To achieve this overall aim, the following research questions have been addressed, all of which build upon, and further expand, the excess mortality synthesis published in $2011/12^{50,51}$:

- 1. What hypotheses have been suggested to explain excess mortality in Glasgow and Scotland?
- 2. What is the most up-to-date evidence base for those potential explanations?
- 3. On the basis of that evidence, which hypothesised causes are most likely to play a role in the excess in (a) Glasgow and (b) Scotland?
- 4. Can the most relevant explanations be drawn together to produce convincing, cohesive, evidence-based, explanatory models for all, or most of, the observed features of excess mortality in both Scotland and Glasgow?
- 5. Do the models help to explain some of the key Scottish mortality phenomena that have been observed since the middle of the 20th century? (These are discussed in more detail in Part Four of the report.)
- 6. What are the implications of, and most appropriate policy responses to, the causes of excess mortality in Scotland?
- 7. Is there consensus across public health and other relevant disciplines regarding the proposed underlying causes and responses?

The methods employed to answer these questions are described in the next section of the report.

3. Methods

Details of the methods employed in this research are presented under each of the research questions listed in the previous section.

3.1 Research question 1: What hypotheses have been suggested to explain excess mortality in Glasgow and Scotland?

As mentioned in the introduction, many potential explanations for Scottish excess mortality have been proposed. These have been suggested via books⁷⁰, peer reviewed journals^{71 76}, official government reports^{77,78}, invited commentaries⁷⁹, personal communications, and in discussion at numerous events where evidence of Scottish excess mortality has been presented or discussed by the authors. A list of all these suggestions was compiled. As also stated earlier, 17 such hypotheses were the focus of the 2011/12 synthesis^{50,51}; since then, many more have been added. However to ensure no other, potentially relevant, suggested explanations were omitted, a systematic review of (a) all proposed explanations for Scottish excess mortality between otherwise comparable high-income populations outside Scotland was carried out in 2015⁸⁰.

3.2 Research questions 2 and 3:

- What is the most up-to-date evidence base for those explanations?
- On the basis of that evidence, which hypotheses are most likely to play a role in the excess in (a) Glasgow and (b) Scotland?

Overall, each hypothesis was assessed in terms of: a) whether there was evidence in the research literature of causal links to health outcomes; and b) (if such an association was found) whether relevant data showed differences in the 'exposure' for Scottish populations compared with those in England & Wales.

However, there were a number of different stages associated with this process. First, where gaps in the evidence base were identified for *potentially* relevant and plausible hypotheses, new data were collected (e.g. from a population survey of Glasgow, Liverpool and Manchester) and/or new research projects were undertaken or commissioned (e.g. for topics such as the nature and scale of urban change (see Appendix A33), employment and the labour market (Appendix A12), diet (Appendix A17), early years experiences (Appendix A10), political influences and vulnerability (Appendix A28)). Thus a range of qualitative and quantitative research methodologies have been employed in increasing the available evidence. However, as is acknowledged and discussed in more detail in Part Six, given the sheer number of hypotheses that have been identified, and the complexity involved in assessing the relevant evidence base for many of them, not all gaps could be adequately filled and not all issues could be fully addressed. In such a complex area, many questions remain. Recommendations for future research to resolve some of these issues are listed in Part Eight of the report.

Second, the validity of each hypothesis was assessed individually using the Bradford Hill criteria for causality⁸¹. Although by no means exempt from criticism⁸², this has been shown to be a useful manner of assessing causality when using observational as opposed to

experimental data, and was used in the 2011/12 synthesis research^{50,51}. All nine of Bradford Hill's criteria were used in the assessment^{iv}, but three in particular were given greater prominence in the assessment:

- 1. Strength of association i.e. from the literature, what is the magnitude of the association between the 'exposure' and the 'outcome'? To use a simple example, in assessing the hypothesis that excess mortality is caused in part by higher smoking prevalence in Scotland (see Appendix A20), the 'exposure' was smoking, and the 'outcome' was mortality
- 2. *Temporality* i.e. does the exposure precede the outcome? In the above example, is there evidence that changes in smoking prevalence have been shown to result in subsequent changes in mortality patterns among populations?
- 3. *Consistency* i.e. has the association between exposure and outcome been shown consistently across different settings (in different populations, by different research groups etc), thereby strengthening the likelihood of there being a causal association?

The first two criteria have been identified as being among the most useful in the assessment of 'upstream' (or macro-level) causes of adverse health outcomes⁸³, while the third has been highlighted by other commentators as particularly important⁸⁴.

If the above assessments suggested a causal association was likely, the third stage involved examination of relevant data to ascertain whether there were differences in the exposure between Scotland and England & Wales, and/or between Glasgow and Liverpool/Manchester. Specifically, we sought to answer two questions:

- 1. Is the hypothesised causal factor worse or more in evidence in Scotland/Glasgow?
- 2. Assuming a difference in exposure, would this have occurred prior to the outcome (i.e. the emergence of excess mortality in Scotland/Glasgow)?

Identification of any potentially relevant factors from the above *individual* assessments led to the next stage (involving *multiple* assessments) described below.

3.3 Research questions 4-7:

• Can the most relevant explanations be drawn together to produce convincing, cohesive, evidence-based, explanatory models for all, or most of, the observed features of excess mortality in both Scotland and Glasgow?

^{iv} These are: **strength of association** (i.e. between exposure and outcome); **consistency** (the extent to which an association between exposure and outcome has been observed in different contexts); **specificity** (repeated observations of an association between a specific outcome and exposure support causality); **temporality** (causality is more likely if exposure precedes outcome); **biological gradient** (the extent to which a 'dose response' is evident in the association between exposure and outcome); **plausibility** (is the hypothesised relationship between exposure and outcome biologically plausible?); **coherence** (the hypothesised relationship fits with the existing knowledge base and does not require a fundamental rethink of science); **experiment** (the likelihood of causality is strengthened by any supportive experimental/quasi-experimental evidence); and **analogy** (where a similar exposure has caused a similar outcome)⁸¹.

- Do the models help to explain some of the key Scottish mortality phenomena that have been observed since the middle of the 20th century?
- What are the implications of, and most appropriate policy responses to, the causes of excess mortality in Scotland?
- Is there consensus across public health and other relevant disciplines regarding the proposed underlying causes and responses?

As aspects of the methodology employed to answer each of these research questions overlap to a degree, they are summarised here together.

It was recognised (and indeed this was an explicit criticism of the previous synthesis) that the individual assessment of each hypothesis described above could be viewed as a somewhat reductionist approach. Thus, in the work described here, each hypothesis deemed likely to be relevant was further assessed in terms of links to other key health exposures and risk factors, as well as to other hypotheses. A so-called 'dialectical' approach was employed whereby a series of causal chains was created, and the authors used logic and argument to decide whether the inclusion or exclusion of particular factors was likely to improve or weaken the 'fit' and explanatory power of the assembled hypothesised causes in relation to the associated outcomes (i.e. the different facets of excess mortality). In this way, two initial explanatory models were created, one for Glasgow and one for Scotland. These were presented, and discussed, at a 'workshop' in June 2015 in which key figures from public health and other relevant disciplines participated, and at which the same 'dialectical' approach was used, with participants asked to assess, and where required, make amendments to the models. 'Key informant' participation was also used to agree sets of policy recommendations in response to the presented models. The full list of participants is included in Appendix C.

The models were tested, first by the authors, and then later by the wider group of workshop participants, to assess whether (and to what extent) they were likely to explain the main features of excess mortality (e.g. higher mortality across all social classes but greater premature excess mortality among the poorest, a widening excess since the early 1980s etc), and the key Scottish mortality phenomena (e.g. slower rate of improvement in mortality over time compared with other European countries, widest inequalities in mortality in Western Europe, increases in mortality in younger working ages – these are discussed in more detail in the next section of the report).

The results of the processes described above are presented in Part Five (the explanatory models for Scotland and Glasgow), Appendix A (assessment of all individual hypotheses) and Appendix B (summary table of those assessments). Prior to that, however, the next section of the report includes a brief discussion of a number of factors which provide important context for the discussion of excess mortality in Scotland.

4. Context: Scottish mortality phenomena and the importance of social, economic and historical influences

The key features of excess mortality were described in Part One (and summarised in Box 1 in at the end of that section of the report). However, these features cannot be examined in isolation from important contextual information: we cannot seek an explanation for the excess without first setting it in the context of what we already know about mortality in Scotland in recent decades, and what we know about its social, economic and historical influences.

4.1 Scottish mortality phenomena

There are a number of features of the health of the Scottish population in recent times that are directly relevant to the issue of excess mortality.

(i) Change in relative health status since the middle of the 20th century

Life expectancy (a useful proxy for population health) is lower in Scotland than in any other Western European country. This is true for both males and females. However, this has not always been the case: in the middle of the 20th century, Scottish life expectancy was similar to, or better than, a number of other European countries. Since then, however, Scotland's health status has, in relative terms, deteriorated: while in absolute terms life expectancy has improved over time, it has done so more slowly than in any other Western European country^{1,2}. This slower rate of improvement means that if these trends continue, life expectancy will soon be lower in Scotland than in a number of Eastern European countries as well. As stated in the introduction to the report, more slowly increasing life expectancy has been observed not just in comparison with other countries, but also when comparing the parts of Scotland that have experienced profound levels of deindustrialisation (an underlying cause of poverty and, therefore, poor health) with other, similarly deindustrialised, regions across Eastern and Western Europe¹⁸⁻²⁰. This is also true of comparisons of Glasgow with other post-industrial UK cities^{29,31}. The excess, and the increasing nature of it, plays a major role in this.

Some of these trends are shown in Figure 3.





Relatively slower rate of improvement in life expectancy for: Scotland (males and females) compared with other W. European countries (top left); Scotland (males and females) compared with England & Wales (top right); West Central Scotland (females) compared with other post-industrial European regions (bottom left); Glasgow compared with other UK cities (bottom right).

(ii) Period effects

The 2011/12 synthesis^{50,51} explored this relative decline in Scottish life expectancy within two distinct periods: 1950-1980, and 1980 onwards. It showed that the slower rate of decline in the first period was principally driven by higher levels of mortality from cardiovascular disease (including stroke), respiratory disease and all cancers combined. Alongside continued higher mortality from these chronic diseases, Scottish mortality in the second period was characterised by relatively higher rates of deaths from alcohol, drugs, suicide and violence, especially among those of younger working ages (discussed further below). Recently an increasing prevalence of obesity and the emergence of inequalities in obesity have been observed⁸⁵. In Part Six of this report we test the extent to which the models put forward to explain the high levels of Scottish excess mortality provide an understanding of past and emergent trends for three separate periods: 1950-1980; 1980-2010; and 2010 onwards.

(iii) Wide inequalities in health

An important component of Scotland's poor health status is that the country exhibits extremely wide socioeconomic inequalities in health compared with elsewhere in Europe. This is true both at the national level (in comparisons of mortality by individual

socioeconomic status^{7,8}) and at the regional level (in spatial comparisons of mortality across similarly deindustrialised regions). Analyses have shown clear evidence of widening inequalities in mortality in Scotland in recent decades. These highlighted the contribution of particular causes of death to overall inequalities within particular age groups: inequalities were greatest among those of working age and, in particular, younger working ages. In the latter case, this was attributable to differences between deprived and non-deprived areas for deaths from alcohol-related causes, drug misuse, suicide and violence i.e. those causes of death that make the greatest contribution to Scottish excess premature mortality.

(iv) Working-age mortality

Clearly related to these mortality inequalities, Scotland's poor health status relative to other parts of Europe has also been shown to have been influenced by particularly high mortality among those of working age^{2,86}. Again, this has been demonstrated not just at the national level, but also in regional comparisons of West Central Scotland (WCS) with other comparably deindustrialised regions of Europe. The latter analyses showed particularly high rates of death among younger working ages (15-44 years) in WCS, driven by high numbers of deaths from alcohol-related causes, drug misuse, suicide and violence - that is, the causes (highlighted above) shown to be associated with the widest inequalities in mortality in Scotland. Analyses have also shown increasing rates of all-cause mortality in WCS for this age group over the course of the 1990s: this was in stark contrast to decreasing rates recorded in the other European regions analysed. This upward trend has also been shown for Scotland as a whole: mortality rates among 15-44 year-olds increased in absolute terms between the early 1980s and the early 2000s, driven by increases in those same causes of death (alcohol, drugs, suicide and violence)^{2,87}. Although mortality rates for this age group have since fallen, they are currently no lower than they were 35 years ago - in contrast to the trends observed in other Western European countries.

Excess mortality is entwined with all four of the above phenomena in terms of its scale, its increasing nature, and the age groups and causes of death with which it is most associated. From what was presented in Part One, it is clear that excess mortality in Scotland and Glasgow is a deeply complex phenomenon. However, the search for the equally complex explanation needs to be anchored in the vast amount of existing public health knowledge and evidence that has been garnered over very many years and in different parts of the globe.

4.2 The importance of existing knowledge

Social determinants

In seeking explanations for the excess, we are, fundamentally, trying to understand why health differs between places. Many years of research, evidence building, debate and consideration have led to a sophisticated understanding of what creates or diminishes the health of populations. Debate continues but, over time, an appreciation has emerged that health determinants are multiple and interwoven and impact across different life stages. Implicit in this understanding is the impact of wider economic, social and environmental factors on an individual's health status (the 'social' model, or understanding, of health). Many socio-ecological models of health determinants have been proposed to illustrate this, all reflecting a broadly similar general understanding of the wider social and environmental influences, and the many links between them. One well-known example, the Dahlgren & Whitehead model^{88,89}, is included here for illustration: however, many more have been proposed and debated⁹⁰⁻¹⁰⁴. The model is presented in Figure 4 and shows various 'layers' of influences on an individual's health: thus, while age, gender, hereditary factors and lifestyle choices are clearly more proximal to one's health status, many of these are in turn influenced and governed by social networks and relations, and then by broader living and working conditions, which in turn are influenced by 'macro' socioeconomic, cultural and environmental factors.

Figure 4: Dahlgren & Whitehead's model of the principal determinants of health (Source: Dahlgren and Whitehead, 1993).



Importantly, this model was first presented in the context of describing the impact of *social inequalities* on health, with an accompanying discussion of the social gradient associated with the main determinants; and although there is much academic debate regarding the precise causes of health inequalities¹⁰⁵⁻¹⁰⁷, most observers (including the World Health Organization and others) recognise that the key drivers of health inequalities are socioeconomic¹⁰⁸⁻¹¹⁰. Given the link between social circumstances and health, inequalities in income and broader socioeconomic conditions across society manifest themselves as inequalities in health status. This is discussed further below.

The political economy

Implicit within models such as Dahlgren & Whitehead's, but not always explicitly highlighted, are the effects of the political and economic systems within societies – the underlying 'political economy'. Socioeconomic, living and working conditions are clearly influenced by the economic and social policies in place in any country. The importance of the underlying political economy for health and, in particular, health inequalities, has been the focus for many writers ^{111- 115}. Examples of this impact include the positive effects of governments favouring redistribution and more generous welfare state provision¹¹⁶, and the negative effects of neoliberal regimes¹¹⁷⁻¹¹⁹ (including, for example: the direct effects of unemployment¹²⁰⁻¹²², welfare reform¹²³, and low income^{124,125}; the widening of inequalities in health driven by widening of inequalities in income^{126,127}; and the 'psychosocial' effects on adversely affected groups related to loss of status, disempowerment, low self-esteem and isolation etc^{128,129}). Political economy, therefore, is an important component of our understanding of the social determinants of health. Its role is shown more explicitly in Krieger's model of 'disease distribution, population health, and health inequities' presented in Figure 5 below, and, as shall become apparent, is fundamental to our understanding of the causes of excess mortality.

Figure 5: A heuristic diagram for guiding ecosocial analyses of disease distribution, population health, and health inequities (Source: Krieger, 2011).



4.3 The importance of historical context

Alongside – and intertwining with – existing knowledge, history is also important in terms of understanding the context for excess mortality. As stated, of all the determinants of population health and health inequalities between social groups, the most important are socioeconomic: thus the socioeconomic history of Scotland and the rest of the UK, and within that, of Glasgow, Liverpool and Manchester (and the wider regions of WCS and NW England), is of paramount importance.

Widening income inequalities and the 'spatial polarisation' of the UK

The widening socioeconomic inequalities in health that have been observed within Scotland in recent decades took place in the context of widening socioeconomic inequality in health (and other aspects of society) across the UK as a whole from the late 1970s onwards, a phenomenon created by UK government policies implemented over the period. Figure 6 shows the dramatic increase in income inequality that occurred in that time, part of a process described as the 'spatial polarisation of the UK', whereby disadvantaged areas (including large parts of Glasgow, Liverpool and Manchester) became relatively more disadvantaged in contrast to other areas which became 'disproportionately wealthier'¹³⁰. The importance of income inequalities as a fundamental driver of health inequalities is further reflected in the accompanying chart (Figure 7) which shows, over a much longer time period, the parallel trends in income inequality (here using a different measure to that shown in Figure 6) and mortality inequality (here the gap in mortality between areas of Britain).

Figure 6: Trends in income inequality (Source: charted from data from the Institute of Fiscal Studies¹³¹).





Figure 7: Trends in income inequality and mortality inequality (Source: charted from data from Thomas *et al.*, 2010¹³² and Dorling, 2011¹³³).

In the same time frame as that shown in Figure 7, the histories of Glasgow, Liverpool and Manchester are equally important to our understanding of the context for city-level excess mortality. All three cities had previously experienced rapid, industrial revolution led, expansion, both literally, in terms of population size and city boundaries, and figuratively in terms of economic wealth and importance. Each also experienced the side effects of that process which included populations subjected to appalling living conditions, poverty and poor health, with comparably high mortality relative to the rest of Britain. Following that, the latter half of the 20th century saw a process of continual and accelerating deindustrialisation (Figure 8), associated high levels of poverty and deprivation (Figure 9), and relative decline within that context of widening inequalities in the UK^{134, 135- 140}.

Figure 8: Trends in industrial employment (Source: Walsh, 2014 (from data from University of Portsmouth/Great Britain Historical GIS Project^v)).



Figure 9: Levels of 'core poverty' in British cities (Source: Walsh, 2014 (from data from Dorling *et al*, 2007)).



There is a need, therefore, to combine our knowledge of the determinants of health and health inequalities, with the knowledge of the implications of historical socioeconomic

^v See <u>www.visionofbritain.org.uk</u>⁵⁵⁸.

trends, linked to an understanding of underlying political economy, to help develop an understanding of the most likely set of explanations for excess mortality in Scotland and Glasgow. These are set out in the next section of the report.

5. Developing explanatory models for Scotland and Glasgow

5.1 Introduction

The explanatory models that are presented here are based on the assessment of a large number of hypotheses that have been proposed as explanations, or partial explanations, for Scotland's and Glasgow's high levels of excess mortality, as well as others identified from a systematic review of similar 'excesses' observed in comparisons of other countries. In total, 40 proposed hypotheses were examined, individually and collectively.

In the hope of both enhancing the readability of this report, and also to focus attention on *the overall synthesis* of the relevant explanatory factors (rather than the many other proposed explanations which are less relevant), the assessments of the many hypotheses are not presented here, but instead have been placed in Appendix A. On the basis of those assessments, both individual and collective, and of all the evidence contained within them, we here present two explanatory models which seek to integrate and synthesise the most likely factors contributing to the excess mortality phenomena.

Reflecting the discussion in the previous section, we begin the model with the important contextual factors that were described:

- The importance of **knowledge and evidence** of what determines good or bad health in any society.
- The key **exposures** in terms of poverty, deprivation, deindustrialisation and UK economic and social policies (political economy).
- A range of key health outcomes relatively poorer, and more slowly improving, health in Scotland, wide inequalities in socioeconomic circumstances and, by consequence, in health across Scotland and the UK, with post-industrial, deprived, cities such as Glasgow, Liverpool and Manchester at the 'wrong' end of that spectrum of inequality, exhibiting the highest rates of both poverty and mortality.

All these factors are all shown in Figure 10. However, that figure also presents two further outcomes: nationally, the fact that differences in poverty and deprivation no longer explain the mortality gap between Scotland and the rest of Britain, and – related to this – the similar unexplained divergence between Glasgow and the two English comparator cities.

In epidemiology, if a statistical relationship between an 'exposure' (e.g. a risk factor such as smoking) and an outcome (e.g. lung cancer) differs because of, and according to the values of, a third variable, the latter is known as an 'effect modifier'. The unknown 'effect modifiers' that have influenced the divergence between Glasgow and the two English cities, and the relationship between deprivation and mortality in comparing Scotland with England & Wales, are what lie at the heart of this synthesis.





The explanatory models presented below, therefore, are centred primarily around what the most likely effect modifiers are, with that judgement based in turn on existing knowledge, historical context and the assessment of over 40 suggested contributory explanations. There are two models, one for Glasgow, and one for Scotland: as already explained, however, these overlap to a large degree, given the influence that levels of population health in Glasgow and the surrounding area have on national outcomes, and the relevance of some aspects of the policy approach, and other exposures, to other parts of Scotland.

NB: it is important to note that the descriptions of the models' components that are presented here are, in general, brief summaries of the more in-depth assessments that are presented in Appendix A. This approach has been to taken in an attempt to make the material as accessible and readable as possible.

5.2 Developing an explanatory model for Glasgow

As stated in the methods section of this report, the Glasgow model is based on explicit comparison with Liverpool and Manchester. However, as is discussed in more detail later in the report, a number of the factors included in the model are relevant not just to Glasgow, but also apply to the wider surrounding area.

The final explanatory model is included as a fold-out diagram later in this report (see Figure 26 on page 61). In the following pages, that model is assembled 'piece by piece'.

There are many components of this model. The first, the key 'effect modifier', is the notion of **vulnerability** (Appendix A28).

GREATER VULNERABILITY

On the basis of all the evidence viewed to date, it is argued that Glasgow's population has been made *more vulnerable* than the comparator populations of Liverpool and Manchester to the exposures highlighted in Figure 10 above.

Vulnerability (discussed in more detail in Appendix A28 and elsewhere¹⁴¹) is a concept that has been used in the field of 'disaster mitigation', in terms of – for example – 'natural' disasters being 'socially determined'¹⁴²⁻¹⁴⁷, but has also been applied more recently to public health by a number of commentators including Galea and colleagues¹⁴⁸⁻¹⁵². They propose an understanding of population health in terms of a relationship, or balance, between 'underlying vulnerabilities' (e.g. poverty, income distribution) on the one hand, and what they call 'capacities' (described as 'salutary resources' – in other words protective factors) on the other – and how populations, shaped by this balance, then respond to particular 'stressors' (e.g. economic processes such as rapid deindustrialisation or recession) and 'protective events' (e.g. economic or social investments).

In one sense this is not a radically different view of population health from that outlined by many others in the field (and illustrated in some of the models of health determinants discussed in the preceding section of this report). However, it is an extremely useful framework within which to view and understand the issue of Scottish excess mortality, especially as some of Galea and colleagues' work in this area has explicitly focused on populations with shared vulnerabilities, but varying levels of health. As they point out:

"there might be tremendous insight into the health of populations gained by studying why populations that share underlying vulnerabilities, such as poverty, often have quite different health outcomes".

The relevance to the comparison of Glasgow with Liverpool and Manchester is obvious.

This framework has been shown to be a highly useful way of understanding lagged or lifecourse impacts, hidden social factors, and important protective factors – and the importance of the latter to understanding Glasgow's excess mortality shall become apparent. Furthermore, a particularly important aspect is that, according to Galea and colleagues, vulnerabilities are seen to impact at the population level: vulnerabilities impact across whole communities, including on those within those communities not themselves specifically exhibiting the vulnerabilities which mark the population as a whole. In other words, when we identify vulnerabilities, and combinations of vulnerabilities, which characterise a population at a specific scale, then all sections of the population are liable to be affected, albeit not necessarily in the same ways or to the same degree. This is highly relevant to the evidence of excess mortality in Glasgow (and Scotland) being observed across the whole social spectrum, not just among the poorest in society.

What factors have made Glasgow more vulnerable to the economic and political exposures included within our model? The evidence suggests that a number of different historical events and processes are likely to have contributed.
The first of these, representing an underlying vulnerability for Glasgow's population (and thus an effect modifier, but also a core determinant of health), is the **lagged effects of high historical levels of deprivation** (Appendix A25).

LAGGED EFFECTS OF DEPRIVATION (OVERCROWDING)

Although current levels of deprivation may be similar across the cities (and do not statistically explain the higher mortality in Glasgow), aspects of Glasgow's mortality profile might be explained by socioeconomic conditions experienced by the Scottish population in previous years. This hypothesis is not supported by analyses of historical income and employment based measures (e.g. 'core poverty', 'breadline poverty', social class, unemployment): as Appendix A25 shows, there have been few differences between the cities for these measures going back almost 70 years. However, the hypothesis is supported by the fact that Glasgow (and, with relevance to the explanatory model for Scotland presented later in this report, other Scottish cities as well) has experienced notably higher levels of overcrowding compared with the English cities back to at least the middle of the 20th century. Such levels of overcrowding could be a marker of historical deprivation and/or a direct causal pathway to poor health from exposure to inadequate housing. Indeed, it can be argued that the stark differences in overcrowding levels in the post-war era placed Glasgow's population (and that of other parts of Scotland) at a disadvantage in terms of the potential to benefit from the opportunities for health improvement which were then emerging from the creation of the welfare state. Figures 11-14 compare, using different measures, levels of overcrowding in Glasgow, Liverpool and Manchester: between 1981 and 2001 (Figure 11); in 1971 (Figure 12); and in 1951 (alongside data for other British cities) (Figures 13 and 14)^{vi}. All show a dramatically different picture of overcrowding in Glasgow compared with the English cities^{vii}.

^{vi} As discussed in Appendix A25, this (the lagged effects of overcrowding) is a complex issue. Overcrowding is included within the Carstairs index of deprivation and the latter has been used in many analyses of excess mortality i.e. where a high excess has been observed *after adjustment for* this important measure. However, the time-specific element is important here e.g. those analyses took account of contemporary, not past, levels of overcrowding. For analysis around the 2011 Census, therefore, overcrowding would not have attenuated the excess because it was not higher in Scotland in that period; however it had been higher in at least the previous six decades.

^{vii} It should be noted that levels of population loss in the period 1951-1981 were very similar in the three cities: thus, this did not play a part in Glasgow's relatively higher levels of overcrowding at this time.



Figure 11: Trends in overcrowding (Source: Walsh, 2014 (from census data)).



Figure 12: Distribution of overcrowded households in 1971 (Source: Walsh, 2014 (from census data))^{viii}.

^{viii} Note that the data presented in Figures 11-14 may, to a degree, overestimate the precise levels of overcrowding in Glasgow (and other Scottish cities) relative to Liverpool and Manchester in the period 1951-71. This is because of potential definitional differences between the Scottish and English censuses relating to the calculation of the number of rooms in a household. However, it is difficult to adjust for these potential differences with any accuracy; furthermore, sensitivity analyses have shown that any such crude adjustment would not change the overall picture of significantly higher levels of overcrowding in Glasgow relative to the other English cities.



Figure 13: Overcrowding (persons per room), 1951 (Source: Taulbut *et al*, 2016).



Figure 14: Overcrowding (rooms per dwelling), 1951 (Source: Taulbut et al, 2016)

Figure 15 provides a useful photographic reminder of what this type of overcrowding – and associated living conditions – meant for people living in particular parts of Glasgow at the

time (c.1971). Clearly, similar photographs could be shown for the comparator cities: however, the point is that these conditions were much more prevalent in Glasgow^{ix}.

Figure 15: Living conditions in parts of Glasgow, c.1971 (Source: Nick Hedges/Shelter Scotland (© Shelter 2015))^x.



Thus, higher levels of overcrowding in Glasgow represent a potential underlying vulnerability for the city's population. The second component of the model incorporates two sets of historical processes, both linked, and both entirely related to the adverse living conditions experienced by many Glaswegians referred to above. They are described here under the overarching title of **social, economic & physical change in the post-war decades (1945-1979)**. The first of these sub-components is **Scottish Office regional economic policy** in the same post-war period, in particular with regard to the **socially selective New Town programme.**

^{ix} It is also worth reflecting that the housing conditions shown in Figure 15 existed, despite many years' activity to improve housing quality. This perhaps emphasises the scale of the challenge facing the city in the post-war decades.

^x These images – reproduced with permission of Shelter Scotland – are Nick Hedges' photographs which were taken for Shelter between 1969 and 1972 (see

<u>http://www.shelterscotland.org/lifeworthliving</u> for more details). Clockwise from top left: Mother living with her children in an overcrowded single end tenement flat Glasgow 1971; Family living in an overcrowded tenement flat Glasgow 1971; Children playing in a Gorbals tenement courtyard 1970; Mother takes her baby inside her condemned tenement block Gorbals 1970.

SOCIAL, ECONOMIC & PHYSICAL CHANGE IN THE POST-WAR DECADES:

SCOTTISH OFFICE REGIONAL ECONOMIC POLICY, INCLUDING SOCIALLY SELECTIVE NEW TOWNS POLICY (OUTWARD MIGRATION)

As outlined in Appendix A28 (and in greater detail elsewhere¹⁵³) new research, based on newly accessed and extensive government archive material, sheds further light on aspects of policy which are likely to have contributed to a greater vulnerability among Glasgow's population.

This is a detailed and complex story and only a brief summary of some of the more pertinent elements are included in this section of the report. The research highlights Scottish Office recognition of the severe challenges faced by Glasgow in the post-war period, in particular in terms of the deep-rooted health, housing and economic problems referred to above. The Clyde Valley Regional Plan of 1946^{xi, 154} set out to address these issues through the development of a number of nearby New Towns (East Kilbride, Cumbernauld, Houston, Bishopton), to which population and industry were to be dispersed. Initially, Glasgow was at the heart of this plan. However, there were delays in its implementation, and when it was returned to in the late 1950s, there was a very different focus: the economic plan no longer concentrated on the economic, housing and health needs of the city, but instead prioritised economic 'modernisation' to achieve growth (based on the development of newer, lighter industries) primarily away from Glasgow^{xii}. Thus, further New Towns were designated (more distant from Glasgow: Livingston, Irvine and Glenrothes) to receive both population and industrial 'overspill' from the city and the plan required the *selective removal of the city's* population on a mass scale. Thus sections of the population – generally younger, skilled workers, in employment, and often with families - were relocated to New Towns and other overspill settlements. This policy was referred to parliamentary discussions of the mid-1960s as 'skimming the cream of Glasgow'. Glasgow itself was at this stage officially designated as 'declining' and these other areas were henceforth to be the priority, not just for economic investment, but also for wider investment in infrastructure and amenities.

All this was different to what happened in Liverpool. For example, Figure 16 shows that by the time of the 1981 Census, the social composition of Cumbernauld and East Kilbride was quite different to that of Glasgow (with a much lower percentage of people of 'low' social

^{xi} The Clyde Valley Regional Plan is also known as the Abercrombie Plan, and is perhaps best known in the context of the competing visions for the city between that plan and the 'Bruce Plan'. The latter (properly known as the 1945 Glasgow Development Plan) argued for comprehensive redevelopment of the City of Glasgow and the rehousing of the existing population (c. 1m) at much higher densities within city boundaries. In contrast, the Abercrombie Plan, argued for large-scale dispersal, including the rehousing of 250,000-300,000 Glaswegians outside the city, especially in New Towns. NB This particular context (Bruce versus Abercrombie) is a different, earlier, issue to that discussed here, which concerns the adoption (and the effects of the adoption) of aspects of the plan from the late 1950s onwards.

^{xii} The economic plan for Central Scotland had its roots, therefore, in the Clyde Valley Regional Plan. However, the different focus from the late 1950s onwards on prioritising economic growth away from the city was formalised in, first, the 1961 *Report of the Committee of Inquiry into the Scottish Economy* (often referred to as The Toothill Report), produced by the Scottish Council (Development and Industry)¹⁵⁵, and then in the 1963 White Paper, *Central Scotland: A Plan for Development and Growth*¹⁵⁶.

class (social class IV and V)), whereas that was not the case for Liverpool compared with Skelmersdale and Runcorn, the two New Towns built to deal with that city's overspill ^{xiii}. Ongoing research suggests that this is indicative of some significant differences in the timing, conception, resourcing and implementation of regional policy in NW England compared with Central Scotland¹⁵⁷.



Figure 16: Adult population of social class IV and V (Source: Taulbut et al, 2016).

Importantly, this policy (the socially selective 'redeployment' of sections of Glasgow's population as part of a broader plan of 'modernisation' based on development and growth away from Glasgow) was pursued through the 1960s and 1970s despite growing concern about, and awareness of, the consequences – both socioeconomic and health related – for the city. For example, the new research quotes Labour MP Hugh Brown in the mid-1960s expressing (in the House of Commons) his concern about 'overspill':

"it is true that today we are getting rid of some of our best tenants and are leaving ourselves with this gap, and we are losing the capacity for leadership in the very communities which are creating the social problems [emphasis added]"

Similarly, a review of overspill policy within the Scottish Office in 1971 (entitled, significantly, 'The Glasgow Crisis') noted that:

"Glasgow is in a socially... [and] economically dangerous position. The position is becoming worse because, although the rate of population reduction... is acceptable, *the manner of it is*

^{xiii} The New Towns programme is much less relevant in relation to Manchester. Plans were made to expand Warrington (1968) and create a Central Lancashire New Town (1971) to accommodate the city's overspill, but these ambitions were scaled back dramatically in 1977 and much of the planned new housing was never built¹⁶⁵.

destined within a decade or so to produce a seriously unbalanced population with a very high proportion of the old, the very poor and the almost unemployable... the above factors amount to a very powerful case for drastic action to reverse present trends within the city. [But] there is an immediate question as to how much room exists for manoeuvre [emphasis added]"

Thus it was recognised within the Scottish Office that these policies were having very serious consequences for the remaining residents of Glasgow, but they were pursued nevertheless (even when explicitly challenged by the Secretary of State for Scotland in the later 1970s) – arguably increasing the vulnerability of the city's population in the process.

With regard to these regional policies, it is also worth noting that Appendix A32 shows that, proportionally, levels of deindustrialisation (measured by the change in levels of industrial employment) in Glasgow, Liverpool and Manchester over the course of the 20th century were remarkably similar. Thus, they are unlikely to have impacted directly on levels of excess mortality in Glasgow^{xiv}. However, the evidence above suggests: first, that the *nature* of earlier deindustrialisation (1960s-70s) was different in Glasgow in terms of the regional policy which deliberately designated the city's staple industries as 'declining' and pursued development away from the city; and second (and as argued by some commentators¹⁵⁸), that the effects of later (1980s onwards) deindustrialisation in Glasgow may have been compounded by the change in population composition which resulted from the New Town policies described above. This included the city having relatively fewer people of younger working age, and relatively more people classed as unskilled.

As outlined in a different Appendix (A26), the available evidence also suggests that, in general terms, it is unlikely that population migration in later decades has contributed significantly to the issue of excess mortality in Glasgow and Scotland. Analyses have shown that migration did not impact on the widening inequalities in mortality observed in Scotland between 1981 and 2001, nor on Glasgow's relatively higher, and worsening, mortality rates in the same period. Other research has also confirmed Scottish migrants elsewhere in the UK tend to display a mortality pattern very similar to that of the non-emigrating population. Despite this, however, it seems highly unlikely that this *particular* form of socially-selective population movement from Glasgow to its New Towns in the preceding decades of the 1950s-1970s would have been without consequences. Thus **outward migration** (in this sense only) can be added to the explanatory model.

To what extent did this particular vulnerability affect only the city of Glasgow, or instead the wider West Central Scotland conurbation? As is discussed in more detail in the next section of the report (Part Six), although the immediate impact was greatest in Glasgow, the evidence suggests that in the longer term the wider region was affected by a broader vulnerability because the entire economic plan for Central Scotland, of which the socially selective New Town programme was a major component, ultimately failed to deliver the intended benefits for the other parts of the region¹⁵⁹⁻¹⁶⁴.

^{xiv} For example (and as shown in more detail in Appendix A32), between 1931 and 2001 levels of industrial employment decreased by approximately 83% in both Glasgow and Liverpool, and 86% in Manchester.

The second sub-component of **social, economic & physical change in the post-war decades**, one which is closely related to the policy agenda described above, relates to **the nature (and scale) of urban change** experienced in Glasgow within that post-war period. This concerns how local government responded to the challenges facing the city in this period. This response was considerably influenced by the wider regional economic policy which had 'deprioritised' Glasgow (see Appendix A28). Importantly, it resulted in Glasgow experiencing aspects of urban change in a different manner and, in part, on a greater scale than was the case in the comparator cities. The nature of these changes is likely to have impacted negatively on the health of sections of the population.

SOCIAL, ECONOMIC & PHYSICAL CHANGE IN THE POST-WAR DECADES:

LOCAL GOVERNMENT RESPONSES: NATURE AND SCALE OF URBAN CHANGE 1950s-1980s

As discussed in more detail in Appendix A33, in common with a number of other UK cities, Glasgow, Liverpool and Manchester experienced large-scale urban change in the post-war period (c. 1945-1980). This was characterised by slum clearance and the relocation of communities to public housing estates, overspill developments in surrounding areas, highrise flats and New Towns. It therefore had the potential to influence population health in several ways, especially through the important social determinants of housing, living conditions and social and community networks. New research has confirmed that although there were similarities in the experiences of Glasgow, Liverpool and Manchester – not least in terms of the severe housing shortages faced by each at the end of the second world war, and the types of policies adopted to address such challenges – there were also a number of potentially important differences¹⁶⁵. These differences included:

- Much higher overcrowding levels in Glasgow (as already shown in Figures 13 and 14 above).
- A greater scale of slum clearances and demolitions in Glasgow than in both English cities (especially Liverpool) (Figure 17). In part this obviously reflects the greater housing challenges faced by Glasgow at the time, but there is also an associated potentially negative impact in terms of the break-up of existing communities and dismantling of social networks.
- More building of within-city (poor quality) peripheral council house estates in Glasgow. Figure 18 shows that although the two English cities also built large estates within the city boundaries, Glasgow built more of them, housing (and impacting on the lives of) a greater number of people.
- A much greater emphasis on high-rise development in Glasgow. As Figure 19 shows, Glasgow built proportionately more blocks of 6+ storeys, but a great many more with 20+ storeys. This is potentially relevant because of the known links between high-rise living and negative impacts on mental health¹⁶⁶⁻¹⁶⁸.
- Crucially, the greater scale of within-city council house building was, however, accompanied by much lower investment in housing repairs and maintenance. Figure 20 (local authority spend per property on repairs, supervision and maintenance) shows how Glasgow compared badly in this respect particularly in the earlier period analysed. This chimes with descriptions in a number of social histories of the many,

and profound, problems associated with living in poor quality damp housing in the city, and the perceived lack of attention given to the problem by the authorities¹⁶⁹.



Figure 17: Housing demolitions 1955-1985 (Source: Taulbut et al., 2016)

Figure 18: Size of post-war housing estates c. 1980 (Source: Taulbut et al., 2016)





Figure 19: High-rise dwellings 1945-1975 (Source: Taulbut et al., 2016)

Figure 20: Local authority repairs & maintenance housing expenditure (Source: Taulbut *et al.*, 2016)



Our model so far suggests that in Glasgow vulnerability to detrimental exposures was enhanced by a series of historical processes: overcrowded housing conditions in the postwar period, the scale and nature of change brought about in the city in attempts to address those conditions, and related economic and social planning which included 'skimming the cream' of Glasgow's population by moving them – alongside employers – away from the city. To this process we can add **differences in later city-level responses to UK government policy in the 1980s**. As is described below (and in Appendix A28 and elsewhere¹⁷⁰), the evidence suggests that in relation to Glasgow, this is likely to have exacerbated the damaging impacts of national-level policies (what Galea *et al.* would describe as 'stressors' impacting on pre-existing 'vulnerabilities'¹⁵²). In Manchester, and particularly Liverpool, on the other hand, city-level responses were different, and seem likely to have offset some of those damaging impacts, fostering 'capacities for coping' and bringing about 'protective events' and experiences.

LOCAL GOVERNMENT RESPONSES 1980s

The impact of the newly elected (in 1979) Conservative UK government's policies on population health – in terms of widening inequalities in income (and other, related, socioeconomic characteristics) and, therefore, ultimately health status across the UK – was discussed in Part Four of this report. Specific policies which particularly affected cities like Glasgow, Liverpool, and Manchester in this period included accelerated deindustrialisation resulting in increased unemployment, sharp reductions in financial support for council housing, and broader policies which impacted adversely on local government finance, trade union organisation, and on individuals in receipt of social security payments. The key point in relation to this synthesis is how local government and other agencies responded at the time to the challenges presented by those UK policies at city level. A brief summary of the material described in more detail within Appendix A28 is that the differences in those responses were most pronounced in relation to the cities' approaches to urban regeneration and in important features of, and development of, local democracy.

In Manchester, the city authorities resisted co-operation with the Conservative government until 1987 when, faced with the third consecutive Westminster electoral victory for the Party, it reversed its previous policy of non-co-operation, and set out to facilitate a model of urban renewal and regeneration which was agreeable to central government^{140,171,172}.

Local politics in Liverpool in the 1980s was characterised by the emergence, following years of (principally) Liberal control, of a Labour-controlled authority – and in particular by the rise of the so-called 'Militant' group within that ruling Labour Party, and its (and, by association, the city's) subsequent overt confrontation with the UK Conservative government of the time. Described in great detail by various historians and political commentators^{137, 170, 173, 174}, the relevance of this to this synthesis is that the council's actions, in rejecting UK government cuts, capping rates and rent increases at a time of economic hardship (the recession in the 1980s – an additional economic 'stressor' in terms of our model of vulnerability and population health), and committing itself to its own large-scale programme of council house building and regeneration, conferred *protective effects* on the city's population. Importantly, the Council's response in Liverpool also entailed considerable mobilisation and political participation among the city's residents. This was a vibrant process, and had further impacts in terms of local government, as a consequence, prioritising and responding to some of the important issues of the day for the majority of

working class Liverpudlians: addressing poverty and providing new, good quality, affordable council housing and wider public services and amenities.

This contrasts considerably with the situation in Glasgow at the time. Although Labour was also the ruling party in the city, it had been the dominant political force over such a long and sustained period of time (in power from the early 1930s, with only brief periods out of office between 1948-51, 1968-71 and 1977-1980) that it was very much more 'the establishment'. Notwithstanding this Labour majority throughout the 1980s, Glasgow's local government rejected the pathway of overt confrontation with central government and took a rather more conciliatory approach. This did not amount to compliance, for the Council, like other local authorities, was willing to breach spending guidelines, and also employed other measures, including 'creative accounting'^{xv}, to try to protect jobs and services^{173,175}. However, this form of defence of the city did not involve the popular mobilisation and participation seen in Liverpool, and from a social determinants of health perspective concerned with issues of power, control and alienation, this is an important difference.

It should be stressed that there were a number of agencies involved in the policy direction which was taken in Glasgow in this period, in particular the City of Glasgow District Council, the larger Strathclyde Regional Council^{xvi}, and the Scottish Development Agency (SDA). The latter was established in the mid-1970s in response to growing concerns around economic development in the country. It should further be emphasised that the policy direction adopted at city level reflected two issues in particular: first, the exceptionally difficult circumstances facing the city at the time in light of the maintenance of the main geographical priorities for development and growth – i.e. away from Glasgow – established by the Scottish Office in the early 1960s (discussed earlier in this section of the report); and second, the election of the Conservative Government to Westminster in 1979. A consequence of the latter was the reorientation of the activities of the SDA. The agency was heavily involved in the development of the city at the time and its reorientation reflected the neoliberal approach to economic and social policy favoured by the Conservative government. However, a considerable amount of research has highlighted ways in which Glasgow District Council itself came to take the lead within the city in this period on the crucial issue of urban regeneration in particular^{173,176-179}.

Commentators and researchers have also described how the Council in this period actively experimented and innovated with neoliberal policy measures guided by the maxim (perceived as agreed by key – although not necessarily all – civic and business leaders of the

^{xv} 'Creative accounting' is a well-known euphemism to describe accounting practices which seek to evade or to 'work around' specific accounting rules, while being seen to adhere to those rules. During the 1980s, local government officers across a number of cities demonstrated great ingenuity in identifying and exploiting loopholes to enable them to maintain jobs and services in the face of central government cuts and the imposition of spending limits and cash controls.

^{xvi} There were two tiers of local government in operation in this period: Strathclyde Regional Council, which covered not only Glasgow but 18 other sub-regional districts, and Glasgow District Council. The larger regional council had responsibility for areas such as education, social work, policing and transport, while the district council's functions included housing, refuse collection, museums and libraries. The two-tier system was in operation between 1975 and 1996, when it was reorganised into the current organisation of 32 local Councils.

time) that "what's good for business is good for Glasgow". These were seen as quite "astonishing" developments in such a "solidly Labour City"^{173,176,xvii}, and were soon to lead to the identification of Glasgow as a so-called 'dual city' with 'dual urban policy': on the one hand high budget, high profile retail and property development in the city centre led by (what has been referred to as) a "growth coalition" in which the city council and the SDA played lead roles; but on the other hand much lower resourced and very limited mitigation and management of poverty, and an intensifying social crisis in the city's poorer areas, principally in the peripheral estates¹⁷⁷⁻¹⁸⁴. Thus, in contrast to Liverpool especially, the priority given to poverty and the housing and living conditions in the poorer parts of the city during the 1980s was quite limited. As one example of these contrasting responses by the cities' authorities, Figure 21 shows the considerable difference in new public housing provision between the cities in that decade.



Figure 21: Public sector housing completions 1980-1987 (Source: Taulbut et al., 2016).

It is important to emphasise that we are not suggesting that Liverpool Council in the 1980s was an optimum model of local government. However, from a social determinants of health perspective, and in light of the vulnerability model of population health outlined earlier, the differences in the historical experience of the cities – the "markedly different postures" struck by the cities' respective local governments in relation to the central government policy agenda of the 1980s – are highly pertinent to the issue of excess mortality. Indeed,

^{xvii} It should be pointed out that (as already indicated and as discussed in Appendix A28) these steps were taken for important reasons: an attempt in the later 1970s to make Glasgow part of the main, wider economic plan for Scotland was roundly defeated in the Scottish Office¹⁵³. Thus, the city authorities were arguably attempting to find a vision and a plan for the city because no such vision or plan was included within the main national government policy.

this contrast between the cities in terms of the effects on the local population can be neatly captured in the words of different commentators at the time. In Liverpool:

"Labour's radical rhetoric *struck a chord with despondent voters*. Support for the council reflected *a groundswell of popular opinion against the government* [emphasis added]."¹⁷³

"There is no doubt at all that the politics of the financial crisis *electrified the people* and *alerted them to its problems* in a way that was simply never there before. Everyone knew about it and *everyone had an opinion* [emphasis added]."

Whereas in Glasgow:

"The peripheral areas of Glasgow are to some extent *politically disarmed*. Nor is there necessarily a serious danger of social disorder, as geographically isolated, alienated youth would have nothing to attack but their neighbours [emphasis added]."

The new research conducted recently summarises these differences in the following way:

"In Liverpool the actions of the Council in the mid-1980s were, for all the controversy associated with them, genuinely popular and apparently invigorating; even for those who disagreed with them, there was a meaningful discussion about the needs of the city, the damage being done by central government and how best to address all of that. In Glasgow, however, there was little scope for that, and in fact there seems to have been an on-going process of managing and manipulating communities in ways which compounded their problems and led, perhaps, to even more damaging outcomes – breaking down fragile bonds of community and turning frustration into something rather more dangerous."

These important aspects – participation, politicisation, community bonds, collective organisation – link very clearly to the concept of '**social capital**': this is discussed further below as a key component of the model. However, it can also be argued that another aspect of the 1980s (extending into the 1990s) further increased Glasgow's (and Scotland's) vulnerability alongside the other issues already presented. This was the so-called '**democratic deficit**'.

DEMOCRATIC DEFICIT

This was a key component of the 2011/12 synthesis report^{50,51} which argued that an aspect of the heightened vulnerability of Glasgow (and Scotland) to the effects of the UK government economic policies of the 1980s to mid-1990s was the fact that those policies were implemented by governments that were being ever more emphatically rejected in Westminster elections by the Scottish electorate (including, in particular, constituencies in WCS and Glasgow). This perceived imposition of 'alien' policies on Scotland by a distant UK government led to feelings of despondency, disempowerment, and lack of sense of control^{164,185-187} – the latter being recognised 'psychosocial' risk factors with known links to adverse health outcomes^{108,126,128}.

As will be discussed later, what emerges from this explanatory model in relation to vulnerability is a sense of the cumulative effects of a range of factors, some of them closely

interrelated. Thus, not simply the issue of democratic deficit, but that alongside: features of local government response to UK government policies; Scottish Office regional policies in the previous decade; highly problematic changes to the city landscape in the post-war era; and at the root of those changes, the distinct challenges related to housing conditions in the city at the end of the second world war. Combined, these had the cumulative potential to increase vulnerability in the city: and in comparing Glasgow with the English cities, Glasgow's relative position was made worse by apparent capacities and protective responses evident in Liverpool and Manchester. The first of these 'capacities' discussed here is **social capital**.

SOCIAL CAPITAL

As described in more detail in Appendix A36, the term 'social capital' is often used in relation to the idea of social connectedness and the value of social networks (a popular definition is: the "features of social organization such as networks, norms, and social trust that facilitate coordination and co-operation for mutual benefit"¹⁸⁸), and such connectedness and networks have been shown to be associated with population health outcomes (including allcause mortality) in a large number of studies. The term itself can be seen as controversial, given its roots in Chicago School economics and the manner in which it was promoted by the World Bank¹⁸⁹. In terms of its importance and relevance to community development and health, it could perhaps be less contentiously termed 'positive social connectedness' and 'community support'. However, the concept itself is not new, with some commentators having highlighted its wider roots in 19th century sociology^{190,191}. Whatever the label used, there is a wealth of evidence linking the concept to many aspects of population health, with some of the causal pathways proposed involving political engagement linked to 'social participation'. There is also a wealth of evidence demonstrating that Liverpool, for a city of its type, has surprisingly high levels of social capital^{xviii,xix}. As described in Appendix A36, the topic was included in a population survey of Glasgow, Liverpool and Manchester, and this showed clear differences between Glasgow and Liverpool in particular in terms of higher levels of – for example – neighbourhood trust, 'reciprocity' (e.g. looking out for, and after, friends and neighbours) and volunteering (a component of social participation) in the English city. Using volunteering as one example, Figure 22 shows that although this activity is generally more common among middle classes (this is true across the UK)^{192,193}, it is notably higher in Manchester, and even more so and across the whole social spectrum in Liverpool, compared with Glasgow.

^{xviii} Despite the controversial nature of the term 'social capital' (alluded to above), for simplicity the report will continue to use this terms as a well-recognised description of the concept.

^{XIX} Many of the indicators used to measure social capital are highly socially patterned. Previous analyses have shown that Liverpool exhibits higher levels of social capital than its socioeconomic profile (i.e. with high levels of deprivation relative to other cities in England) would predict.



Figure 22: Volunteering rates by city and social class (Source: Walsh et al., 2013)^{xx}

The same survey also asked questions about views on the UK government of the time (the survey was carried out in 2011 when the Conservative–Liberal Democrat coalition was in power) as well as – among those who had lived through the 1980s – whether people had taken part in demonstrations in relation to government policies in the latter decade. As Figures 23 and 24 clearly show, the Liverpool sample was much more politicised in those regards, and all the relevant data from the survey seem to suggest a lasting legacy from the politicisation and participation of Liverpool's population in the 1980s. Other research has shown similar findings. For example, analyses of suicide across Britain between 1980 and 2000 highlighted much lower than expected rates in Liverpool (contrasting with higher than expected rates in Glasgow), with the authors suggesting protective factors relating to greater social integration (and possibly religion – see Appendix A36) might be operating¹⁹⁴. Similarly, recent qualitative research in the three cities suggested that Liverpool stood out in terms of its 'strong sense of social solidarity', described as a key part of the city's identity and culture¹⁹⁵.

^{xx} The chart shows the percentage of respondents in each city broken down by social class (defined by the census measure of 'social grade'). The black lines represent the values of 95% confidence intervals.







Figure 24: Participation in anti-government demonstrations (Source: Walsh et al., 2013)

As Figure 22 above suggests, and Appendix A36 discusses in more detail, many of the differences in aspects of 'social capital' between Glasgow and Liverpool are seen in comparison of the middle classes, not just those living in the poorest parts of the city. Alongside the previous discussion of vulnerability affecting different social classes, this is

also potentially relevant to the fact that one of the key components of excess mortality in Glasgow is that it is observed across the whole social spectrum.

The assessment of the evidence suggests, therefore, that among Liverpool's population, aspects of social capital, potentially derived at least in part from the political history of the city, may have fostered – in Galea *et al.*'s terms – 'capacities' for coping with the damaging impacts of 'stressful events'. We add this to the model – and also suggest that there may be other **protective effects** at work in relation to Manchester.

PROTECTIVE EFFECTS:

LIVERPOOL - SOCIAL FABRIC, POLITICISED AND PARTICIPATIVE POPULATION

MANCHESTER – ETHNIC DIVERSITY

Two potential protective factors (capacities) for Manchester have been identified from the research to date. First, the city's more ethnically diverse population may offer protective effects in terms of population health. As Appendix A13 describes, some researchers have suggested that higher numbers of ethnic minority groups may be an explanatory factor for lower than expected mortality among some more deprived UK populations. It is possible – although further research would be required to confirm this – that this plays a part in explaining the lower mortality in Manchester (a city where, in 2011, 33% of the population was classed as being from an ethnic minority) compared with Glasgow (where the equivalent figure was 12%^{xxi}). A further protective factor for Manchester was proposed in the results of qualitative research in the three cities published in 2015. This suggested that Manchester's culture is one that has adjusted better to the transformation (common to all three cities) from industrial to post-industrial society: the city had experienced a "cultural adaptation to more mobile lifestyles well suited to the changing nature of employment opportunity in a post-industrial economy". This was shown to particularly relate to the city's more affluent residents, which is again potentially relevant to the issue of excess mortality having been observed across different social classes. However, as these findings were based on a relatively small qualitative study, we should be cautious in attempting to estimate their impact.

A further major component of the explanatory model for Glasgow's excess mortality relates less to effect modification, and more to the key determinants of health ('knowledge' in Figure 10 in Part Four) – although, that said, it is likely that some aspects of enhanced vulnerability discussed above are relevant to it. On the basis of the assessment of all the available evidence – and given the importance of poverty in terms of understanding population health – we suggest that **artefact** plays a part. By this we mean the **inadequate measurement of poverty and deprivation** in the studies carried out to date. We argue here that currently used measures of deprivation fail to capture important differences in the complex 'lived reality' of deprivation and poverty in Scotland, and especially in Glasgow, compared with elsewhere in Great Britain and the UK.

^{xxi} The equivalent figure for Liverpool is very similar to that of Glasgow – 11%.

INADEQUATE MEASUREMENT OF DEPRIVATION

Appendix A3 summarises the main arguments for and against this hypothesis. Briefly, the arguments against are: that excess mortality has been observed in comparison of non-deprived populations as well as deprived^{21,24,25,29,31}; that Glasgow, Liverpool and Manchester (and surrounding areas) have been shown to be similar not just in terms of single measures such as income deprivation (the measure used in the published comparative analyses of deprivation and mortality in the three cities, and which was shown be an excellent proxy for multiple deprivation as currently measured in both Scotland and England^{29,30}), but also in terms of a range of other measures of poverty (e.g. unemployment, 'breadline poverty', social class) and related social characteristics (e.g. lone parenthood, rates of teenage pregnancy); and that an excess level of mortality in Scotland and Glasgow has been shown no matter the measure, nor the geographical unit of calculation, that has been used^{21-27,29,31,56}.

However, a number of arguments can be made in support of this hypothesis.

Reflecting the importance of existing knowledge to the model (as highlighted in Figure 10), there is a wealth of epidemiological evidence that demonstrates the importance of poverty and socioeconomic deprivation in explaining differences in health outcomes between populations^{3,105,107-109,120,121,196,197}. The explanatory power of these factors has been proven in countless research projects. It seems entirely unlikely that this principle would not equally apply to comparisons within the UK.

Research has also highlighted the complex nature of poverty and deprivation: it encompasses many diverse and overlapping dimensions^{196, 198}. It is equally unlikely that any routine administrative indicators (e.g. from the census or social security systems – even when included within more recent measures of multiple deprivation) can fully capture those many different facets. (Reflecting this complexity of deprivation, it has also been shown that increasing the range of socioeconomic measures used in analyses tends to explain more of the variation or inequality in the health outcomes¹⁹⁹, suggesting that a more comprehensive and multidimensional set of measures of poverty may actually explain a greater proportion of the excess mortality).

Related to the above, the scale of excess mortality in Scotland and Glasgow has been shown to vary depending on which indicator of deprivation or socioeconomic status (SES) has been used: this sensitivity further emphasises the importance of how poverty is measured.

With regard to this last point, the particular measure of deprivation used in the analyses of Glasgow, Liverpool and Manchester was based on recipients of social-security benefits, and as such, is subject to considerable weaknesses: individuals are either in receipt of such benefits or they are not, and there is no scale associated with lesser or greater need^{xxii}.

^{xxii} The three-city analyses referenced earlier²⁹ were based on comparisons of mortality and areabased deprivation. The latter was measured by 'income deprivation' derived from UK Department for Work & Pensions (DWP) data, and defined as the percentage of the population in receipt of key low

Part Four of the report highlighted the major changes that have taken place in UK society since the early 1980s in terms of a dramatic widening of socioeconomic inequalities. A considerable amount of research undertaken over the period has highlighted the 'social exclusion' and marginalisation of sections of the population^{6,130,133,200-202}: thus there will have been changes in the experience of relative deprivation over that period that will not have been captured by the routine indicators used in analyses over time. This is relevant to the argument that the Carstairs measure of deprivation (used in a number of analyses of excess mortality^{21,25}), which at the start of the 1980s explained much of the difference in mortality between Scotland and other parts of Britain, no longer adequately captures differences in poverty between those populations^{50,51}. Furthermore, the mortality profile of Glasgow in the decades since the 1980s has been characterised - particularly in the city's most disadvantaged communities – by relatively higher rates of death from more sociallydetermined causes: alcohol, drugs and suicide (see Figure 2 in the Introduction) i.e. what might be described as the 'diseases of despair' associated with people living with, and attempting (or failing) to cope with, extremely difficult circumstances. The complexity of and changes in - these aspects of relative poverty, and the associated 'lived experiences' of those who have suffered it, currently (and perhaps inevitably) lie beyond measurement by routine administrative recording systems. It also seems likely (although further research would be required to ascertain this) that aspects of the vulnerability-inducing historical processes described earlier in this section of the report are highly relevant to this.

Finally, the argument that there are likely to be additional, unmeasured, aspects associated with living in deprivation that are more prevalent among the Glasgow population compared with those living in the English cities is further reinforced by the fact that although few differences between the cities have been observed in relation to many aspects of poverty, we know – as discussed above – that levels of overcrowding have been higher in Glasgow (and in Scotland as a whole) since at least the end of WWII; and that although these declined subsequently, marked differences remained. This suggests that there may have been (and may still be) 'residual confounding' i.e. aspects of socioeconomic conditions and status that are inadequately captured by the current measures relating to income, unemployment or social class alone. This obviously also links to the **lagged effects of poverty and deprivation** discussed earlier.

An additional, potentially unmeasured, difference in living conditions between the cities may relate to the **physical environment** (Appendix A31).

MORE ADVERSE PHYSICAL ENVIRONMENT

Data limitations mean we have to be cautious in our assessment of this hypothesis, but it appears likely that a higher percentage of the population of Glasgow lives in proximity to areas which have potentially negative environmental characteristics compared with those in Liverpool and Manchester. For example, 6.8% of Glasgow's land is classed as being vacant or derelict, more than the equivalent figure for Liverpool (5.4%) and over three times the figure

income related social security ('welfare') benefits, as well as children dependent on adult recipients of those benefits.

for Manchester (2.2%)^{203,204,xxiii}. Within Glasgow, a remarkable 60% of the entire population currently live within 500m of such vacant or derelict land²⁰⁵. Other analyses suggest similar differences, particularly between Glasgow and Liverpool^{xxiv}. These are relevant because of the considerable amount of research evidence linking aspects of the physical environment to population health, including mental health²⁰⁷⁻²¹². Some differences in the physical environment to another component of the model discussed earlier, **the nature (and scale) of urban change** experienced in the city (i.e. in relation to the high prevalence of 'brownfield' sites and derelict land in Glasgow).

It is possible that other aspects of disadvantage that have not been adequately captured can, to a degree, be glimpsed in analyses of **educational attainment** (Appendix A11).

EDUCATIONAL ATTAINMENT

Although Scotland and Glasgow compare well in terms of levels of tertiary level education, in recent decades higher percentages of adults in Scotland compared with England, and of Glasgow compared in particular with Manchester but also with Liverpool, have been recorded as having no educational qualifications at all. For example, comparisons of the cities at the last census (2011), showed that the percentage of adults (aged 16+ years) with no educational qualifications was 32% in Glasgow compared with 29% in Liverpool and 23% in Manchester (Figure 25)^{213,214}.

^{xxiii} As described in Appendix A31, it should be noted that these figures are derived from different sources and a number of caveats apply. However, the definitions on which the Scottish and English data are based are broadly comparable.

^{xxiv} For example, analysis of the European Environment Agency's (EEA) Urban Atlas data²⁰⁶ (aerial photography based land-use maps from 2005-08) suggests that a higher percentage of land within Glasgow City is classed as 'land without current use' compared with Liverpool, and this is also true in comparison of the most deprived neighbourhoods in both cities.



Figure 25: Adults with no educational qualifications (Source: 2011 Census).

Education is an important social determinant of health, known to impact on health status by means of interactions with other important determinants such as employment and income (and it is a measure, therefore, that is often employed in analyses of socioeconomic inequalities of health)^{108,215-220}. However, its additional relevance to the model here is that most of the evidence regarding school-based educational attainment suggests that, at a population level, differences in attainment largely reflect differences in socioeconomic background, including key features of a child's home environment, rather than necessarily differences in education systems or school performance^{133,221-224,xxv}. A review of education systems within the four countries of the UK argued that: 'education systems interact with their contexts; differences in systems reflect and sustain differences in social relations'²²⁵. Indeed, an independent review of the Scottish school education system in 2007 also emphasised the importance of the social context (especially the socioeconomic context) in assessing performance:

"Little of the variation in student achievement in Scotland is associated with the ways in which schools differ. Most of it is connected with how children differ. Who you are in

^{xxv} As the sociologist Bernstein stated in the 1970s: 'education cannot compensate for society'²²³, and sociologists generally have explained variation in educational attainment between social classes in terms of three forms of so-called 'capital' (all overlapping): economic capital, cultural capital (related to particular cultural practices such as reading, and associated with levels of parental education) and social capital. The latter is discussed in detail elsewhere in this report, but with regard to education, it relates to links between families, schools and communities²²¹. This is obviously not to say, however, that education systems and schools are not important. Recent research estimated that 20% of variation in educational progress was explained by schools²²⁴ and recent reviews have highlighted, for example, the importance of teacher quality in explaining some outcomes^{221, 222}.

Scotland is far more important than what school you attend... Socio-economic status is the most important difference between individuals."²²⁶

Thus it is possible that differences in levels of educational attainment between Glasgow, Liverpool and Manchester may reflect differences in socioeconomic background not captured by indicators such as income deprivation, and others used in analyses of excess mortality in Scotland. The fact that – as shown in Appendix A11 – the greatest differences in attainment (in terms of the population having no qualifications) are seen in comparison of the more, rather than less, deprived areas of the cities is potentially highly relevant to this.

That said, it is likely that the impact of differences in educational qualifications on excess mortality is fairly small: when included in statistical modelling analyses at the national level (i.e. Scotland relative to England & Wales, rather than Glasgow relative to Liverpool and Manchester), the excess was reduced from 10% higher mortality (after adjustment for age, gender and deprivation) to 8.7% higher (after further adjustment for differences in educational qualifications^{xxvi})²²⁷.

OTHER SMALL EFFECTS

The final component of the explanatory model covers a number of **additional factors**, the individual impacts of which are likely to be very small, but which cumulatively may be relevant to particular aspects of population health. One example only is offered here: as Appendix A40 describes in greater detail, the weight of evidence suggests there may be a link between areas of 'soft' drinking (tap) water (specifically in relation to lower levels of magnesium, rather than calcium) and rates of cardiovascular disease²²⁸. Cardiovascular mortality is higher (after adjustment for differences in deprivation), and magnesium levels are lower, in Scotland compared with England & Wales, and in Glasgow compared with Liverpool and Manchester^{25,29,229-231}. Importantly, however, one of the major UK studies that have investigated the issue concluded that any levels of increased risk were probably not large enough to warrant any kind of intervention²³². It is likely that there are other, small differences between the cities for which the associated risk is small, but that cumulatively there may be an effect. Vitamin D deficiency among the elderly is another possible example^{xxvii}.

^{xxvi} Excess after adjustment for age, gender, Carstairs area deprivation: 10.0% (95% CIs 9.45-10.63); after further adjustment for area-based measure of educational attainment (defined as no educational qualifications among those of working age, grouped into deciles): 8.7% (95% CIs (7.90-9.07)

^{xxvii} As Appendix A6 explains, research to date suggests that Vitamin D deficiency is unlikely to play a major role in Scotland's high levels of excess mortality. Although there is research evidence (albeit often disputed) showing an association between Vitamin D deficiency and all-cause mortality, it relates principally to mortality among the elderly – and as described elsewhere in this report, excess mortality in Scotland is driven mainly by higher rates of mortality among those of working age. However, given that excess mortality has been observed across all adult age groups, it is at least plausible that Vitamin D deficiency contributes to the excess among the elderly Scottish population. Although the evidence remains disputed (and indeed, one review has suggested Vitamin D deficiency may actually represent a *consequence* of disease rather than cause), randomised control trials (RCTs) are currently underway which will provide much higher quality evidence of the link between Vitamin D status and population health (see Appendix A6).

The final proposed explanatory model for Glasgow is shown in full below. This suggests that the principal underlying causes of excess mortality in Glasgow relate to:

- the lagged effects of historically high levels of deprivation (principally overcrowding), representing an underlying vulnerability
- further vulnerability to political and economic exposures from a range of 'effect modifiers' emerging from differences in the processes of regional and city change in the post-war decades, and then from further differences in city and regional-level responses to the impacts of the post-1979 central government policy agenda
- the fact that some of these responses had impacts (negative in Glasgow, positive in Liverpool) on social capital, and that other capacities (offering protection or mitigation) exist for Manchester, thereby further disadvantaging Glasgow in relative terms
- an inadequate measurement of the lived experience of poverty and deprivation, with the likelihood that aspects of the vulnerability-inducing historical processes described above are relevant to this
- potentially, that some of the unmeasured aspects of deprivation in Glasgow include a more adverse physical environment and differences in educational attainment
- that there may be a range of other factors with smaller impacts which, cumulatively, may add to the excess.

The particular causal pathways associated with the model are important to understand, and are discussed further in the next section of the report. Before that, however, we need to build a similar explanatory model for excess mortality in Scotland, not just Glasgow.



Figure 26: Explanatory diagram for excess mortality in Glasgow.

5.3 Developing an explanatory model for Scotland

All the research that has gone into developing the explanatory model for Glasgow is also relevant to the model for Scotland. This is because of the impact that population health in Glasgow, and in the wider West Central Scotland conurbation, has on national outcomes: with 11% of Scotland's population resident in the city, and more than 40% resident in that wider conurbation, this is simply a consequence – an important consequence – of scale.

However, to that Glasgow model, and based on all the evidence assessed within this programme of research (all summarised within Appendix A), we would make a number of key alterations.

First, the lagged effects of deprivation and poverty (Appendix A25) should be given even greater emphasis in the all-Scotland model. The city-level analyses showed that among various poverty and deprivation related indicators, only overcrowding had historically been higher in Glasgow than in the comparator English cities (with few differences visible from examination of income or employment based measures)^{xxviii}. However, at the national level, analyses have suggested that Scotland has been materially more deprived than England over many decades. For example, Figure 27 shows trends in the four components of the Carstairs & Morris index of deprivation (although a less clear picture emerges when different measures of poverty are used^{xxix}). Furthermore, the relationship between historical deprivation and Scottish mortality is clearly complex, given the fact that excess mortality has increased over the period in which relative deprivation has decreased. Nonetheless, the weight of evidence suggests that Scotland as a whole experienced higher levels of economic hardship in the 20th century than was the case in England as a whole, and that that is likely to have influenced more recent trends in mortality. However, as with the other components of the models, it is impossible to quantify the impact on health. This limitation is discussed later in the report.

^{xxviii} However, it should be noted that levels of car ownership have also been shown to be lower in Glasgow than in Liverpool and Manchester. When used as a proxy for income/deprivation in comparative analyses, this measure attenuated the level of excess mortality in Glasgow compared with the two English cities (although a significant excess remained)⁵⁶. However, some of the results of those analyses were surprising and inconsistent, and a number of commentators have questioned the usefulness and appropriateness of this indicator as a measure of deprivation²³³⁻²³⁷.

^{xxix} For example, analyses of relative poverty/low income households at different time periods have shown Scotland to be similar to Great Britain^{202,313} and the same is true of analyses UK longitudinal cohort data⁵².



Figure 27: Components of Carstairs index, 1981-2011, Scotland and England & Wales (Source: Schofield *et al.*, 2016).

Second, the impact of **deindustrialisation** has been greater in Scotland than in England. As explained in Appendix A32, analyses of industrial employment data over the course of the 20th century suggest rates of deindustrialisation in Glasgow, Liverpool and Manchester were very similar; however the equivalent figures for Scotland as a whole were much higher than they were for England^{xxx}. It is likely that aspects of this difference relate to post-war regional policy issues described earlier. Although the effect of this on comparisons of mortality between the countries is likely to be confounded by the associated higher levels of deprivation in Scotland over the period, there may still be a residual effect on health status.

Third, **Scottish Office regional economic policy in the post-war decades** (a key component of the Glasgow model) is also relevant to Scotland as a whole (and to Central Scotland in particular – thus to the majority of the Scottish population), although in a different way. This is because the evidence suggests that not only did the economic plans have a detrimental effect on Glasgow, they also failed to deliver the anticipated benefits – 'modernisation', economic growth and wider human welfare – elsewhere in the country ¹⁵⁹⁻¹⁶⁴. The economic plan was very much an 'assumed normative' (i.e. it was assumed to be the correct course of action despite a lack of any supporting evidence)^{159,160}, one which was implemented with a high degree of commitment and co-ordination, and neither the form of the plan nor the manner of its implementation seem to have been replicated in other parts of the UK and Europe^{159,160,163,238}. However, the policies neglected what were Scotland's established areas of industrial strength and achievement (which were designated as 'declining'), and

^{xxx} As shown in Appendix A32, analyses of historic census data between 1931 and 2001 show that by 2001 the rate of deindustrialisation (the loss of industrial jobs expressed in relation to the size of the industrial employment base in 1931) was 47% in Scotland compared with 30% in England. The equivalent figures for the cities were 83% (Glasgow and Liverpool) and 86% (Manchester).

contributed to the loss of a far larger number of jobs than they managed to create in the new industries. Many of those that were created through inward investment proved to be low-skill and situated in 'branch plants' which were themselves highly vulnerable to turning economic tides and the decisions of corporations located elsewhere. Nonetheless, having set out on this path with a high degree of conviction and enormous public expenditure, government was to remain essentially committed to it for decades to come – arguably until the collapse in the microelectronics sector between 2001 and 2003 provoked an embrace of the 'cities agenda' by the then Scottish Executive²³⁹. In this sense, it is not just Glasgow, but Scotland much more widely which can be seen to have been, and still be, adversely affected by the regional policy adopted in Scotland in the late 1950s and early 1960s, and implemented with force and sustained commitment in the decades to come. As Devine has recorded, in the period immediately after 1979, it was not just that "the great staples of the Victorian economy... virtually all crumbled with astonishing swiftness... Even many of the regional policy successes of the post-war years succumbed". That is to say: even the 'successes' of regional policy were to prove vulnerable. Overall, then, the regional policy aspect of the explanation proffered in relation to Glasgow seems to have a clear relevance to the lagging health outcomes across Scotland more widely.

Fourth, the Scotland model is hindered by a lack of evidence relating to potentially important 'effect modifiers' that are included within the Glasgow model. Differences in aspects of the **physical environment** (e.g. the likelihood of living in proximity to negative landscapes such as vacant and derelict land) that were shown for Glasgow compared with Liverpool and Manchester may not apply to all comparisons of Scottish and English urban areas. Furthermore, differences in the urban-rural mix of Scotland and England make interpretation of the likely impact of this measure even more difficult at the national level. Another, arguably more important omission – given its importance to the Glasgow model – is research evidence relating to the **responses of local government** in other parts of Scotland in the critical periods identified above (e.g. in the post-war period and the 1980s). This clearly lends itself to future research (recommendations for further work are presented in Part Eight of the report).

Fifth, **social capital** is likely to be less relevant to national comparisons. As stated in Appendix A36, there has been very little research into differences in social capital at the national level, and what little evidence there is does not suggest Scotland is particularly 'worse' than England in this regard²⁴⁰. The city level research suggests it is very much about Liverpool being 'better', rather than Glasgow being particularly 'worse' in this sense.

Finally, there are differences in some more **'downstream' health behaviours** that are evident at the national level (but not in comparison of Glasgow with Liverpool and Manchester) which may be relevant. For example, national survey data suggest that levels of **obesity** among the middle classes in Scotland are higher than in England (Appendix A27)^{241, 242}. The relevance of this to Scottish excess mortality is questionable, given that obesity-related measures have been included in statistical modelling analyses and did not attenuate the high level of excess mortality observed in the Scottish sample compared with the English sample. However, it is possible that there may be particular interactions between class, income and obesity that might warrant further investigation. New analyses have also

suggested there are potentially relevant differences in some aspects of **diet** between Scotland and England (but not between the cities, nor indeed in comparison of Scotland with North West England) (Appendix A17)²⁴³. This may suggest that particular cultural influences on diet may play a role in explaining aspects of the excess at the national level. However, limited information on diet has been included in statistical modelling of excess mortality for Scotland relative to England and has been shown to reduce, but not in any way eradicate, the excess^{24 xxxi}. Similar results have been obtained from analyses of **smoking**: as smoking prevalence is higher in Scotland²⁴⁴ it reduces the excess to a degree, but does not remove it. Thus, these differences in health behaviours are relevant and important: however, at best, they only partially contribute towards explaining the excess mortality, and indeed, as discussed elsewhere, in many cases they raise a number of questions regarding underlying factors (the 'causes of the causes'^{3,245-247}).

In summary, the explanatory model for Scotland can be seen as being made up of various components:

- the model for Glasgow (and its wider area) in its entirety, given the extent to which that impacts on the national level of excess mortality
- particular elements of the Glasgow model which are also highly (in some cases more) relevant for Scotland as a whole. These include:
 - o the inadequate measurement of deprivation
 - the lagged effects of poverty and deprivation
 - key vulnerabilities these include the so-called democratic deficit and (related to the above), historical high rates of overcrowding in other Scottish towns and cities, as well economic policy in the post-war period which not only had a detrimental effect on Glasgow, but failed to deliver anticipated benefits elsewhere in the country; however, it is clear there are a number of areas that require further research to clarify any potential contribution (e.g. the scale and nature of urban change in other cities; local government responses to UK policies in the 1980s).

In the next section of the report, both these explanatory models are discussed in more detail. This includes testing the causal chains suggested by the models in terms of the key features of excess mortality, and appraising the strengths and weaknesses of the approach taken in developing the models.

^{xxxi} As stated in Appendix A17, statistical modelling analyses, using the Scottish Health Survey and the Health Survey for England, showed that excess level of mortality among the Scottish sample reduced from approximately 40% higher (after adjustment for age and gender only (Hazard Ratio (HR) 1.40 (95% CI 1.34 – 1.47))) to approximately 31% higher (HR 1.31 (95% CI 1.22, 1.44)) after adjustment for diet. Mortality for cardiovascular disease and cancer was approximately 51% and 39% higher respectively among Scottish respondents after adjustment for diet. It should also be noted, however, that in these analyses 'diet' was measured only in terms of consumption of fruit and vegetables, which is clearly an extremely limited definition²⁴.

6. Discussion

6.1 Summary of the models

As summarised at the end of the previous section, both the Glasgow and Scotland explanatory models are based on a number of different components, all of which, we suggest, via particular pathways, cumulatively contribute to the high levels of Scottish excess mortality.

Of the 40 hypotheses that have been summarised and assessed within Appendix A, the final models include key 'upstream' (macro-level) explanations such as political influences and socioeconomic elements, 'midstream' factors like social capital, as well as 'downstream' aspects: alcohol and drugs (included in the models as important outcomes) as well as (somewhat more speculatively) issues relating to diet and obesity. As Appendix A shows, assessments of relevant evidence suggest a wide range of other issues such as differences in health and social services (Appendix A15), 'sense of coherence' (A35), individual values (A24), social mobility (A37), climate (A5-A7), and many others, are unlikely to contribute to the excess. As discussed further below, however, the influence of some factors remains unknown because of a lack of robust, high quality, evidence and data.

Chief among the contributory components of the models are the inadequate measurement of socioeconomic deprivation and greater vulnerability, the latter acting as an 'effect modifier' which, for the Scots, has altered the relationship between key socioeconomic exposures and health outcomes. These factors, acting via well-understood mechanisms linking socioeconomic circumstances, stress and associated detrimental health behaviours, present plausible causal pathways that would explain the range of outcomes associated with the excess; and in the case of Glasgow these factors are entwined with important protective factors (e.g. better social integration) for the comparator cities, resulting in Glasgow's relative position being made worse still. To be sure of this, however, the models were tested explicitly in relation to the extent to which they explain the principal features of excess mortality and – linked to that – aspects of the key Scottish mortality phenomena described in Part Four. This is discussed below.

6.2 Testing the models

Key features of Scottish excess mortality

The most relevant features of excess mortality were outlined in Part One of this report, and summarised in bullet format in Box 1. Here we revisit five key features to consider the extent to which they are adequately explained by the models. As will become apparent, all five overlap to a considerable degree.

 The excess is observed in all parts of Scotland, but is greatest in and around Glasgow. Two separate models, one for Glasgow, and one for Scotland, have been created to explicitly address the issue of a Glasgow-specific and a wider Scottish excess mortality. Furthermore, and as stated in the previous section, given the fact that more than 40% of the Scottish population are resident in Glasgow and the wider West Central Scotland (WCS) conurbation, the entire Glasgow model is relevant to national outcomes. In both cases, unmeasured aspects of deprivation allied to a greater vulnerability (and manifested via key health behaviours and other risk factors for chronic diseases) provide the most likely explanatory power. As acknowledged in the previous section of the report (and discussed further below), however, there is a greater number of 'unknowns' associated with the Scottish model, for example in relation to local government responses in other parts of Scotland at key historical periods. The extent to which the model explains excess mortality in Edinburgh, for example, is obviously less clear than in Glasgow, for which a greater amount of supporting evidence has been amassed (reflecting a greater research focus resulting from the city exhibiting the highest levels of the excess). However, the uncaptured 'lived reality' of deprivation (including associated lagged effects) alongside other key vulnerabilities such as the 'democratic deficit', unsuccessful regional economic planning in the post-war period, and a range of other smaller influences, are likely to provide at least partial explanations for the excess mortality observed in Edinburgh and other parts of Scotland that lie outside the WCS conurbation.

Another important consideration is the extent to which the Glasgow model is only relevant to the city itself, or instead to the wider area (Greater Glasgow or the WCS conurbation). Although a number of components are clearly relevant to the wider area, others are less so. In relation to the key historical vulnerabilities, research on the impact of Scottish Office regional policy on Glasgow is, as explained in the previous section of the report, relevant to the wider conurbation because of its failure to deliver economic benefits to those populations outside Glasgow (as well as impacting detrimentally on the city itself). On the other hand, research on the differences in the processes of urban change across the three cities in the post-war period focused principally on the city itself, not the wider region. Similarly, the conclusions relating to local government responses to UK economic policies in the 1980s relate only to Glasgow. As is discussed elsewhere in this section of the report, local government responses elsewhere in the WCS conurbation have not been studied as part of this research. In summary, there are some aspects of the excess across Scotland which are well explained by the model, but a significant amount that remains unknown.

2. The excess is observed among all social classes, but – in the case of premature mortality – is greatest in comparison of those living in the poorest neighbourhoods. In terms of the high levels of excess premature mortality among more deprived populations (linked to high rates of death from, in particular, alcohol, drugs and suicide), the key components of the model are all relevant and provide an explanatory framework based on much of our existing knowledge of the determinants of health (i.e. in which socioeconomic factors, including the lagged effects of poverty and deprivation, play key roles). Thus, the effects of poverty and deprivation, other negative impacts of deindustrialisation (e.g. de-skilling , role redefinition), the psychosocial impacts of marginalisation and social exclusion – all factors which are common to many populations in a Britain that has been characterised by significantly widening inequality over the past 35 years – have been made worse in Glasgow (and Scotland) by existing vulnerabilities (brought about a by a series of historical factors), feelings of powerlessness, and other 'modifying' factors. This has led to relatively greater stress, worse mental and physical

health, compensated for – in some cases – by greater reliance on alcohol and drugs related 'coping mechanisms', resulting in yet worse health outcomes. In the case of the cities, the position of Glasgow's more disadvantaged population has been made worse relative to Liverpool's in particular because the vulnerability of Glasgow has operated alongside a protective 'capacity' for Liverpool in terms of the latter's greater social fabric and cohesion.

Relatively higher mortality in Glasgow and Scotland among *those of higher social class* compared with the rest of Britain is best explained by the evidence of vulnerability – as the key 'effect modifier' – affecting *all* social classes in society, as suggested in Part Five and in Appendix A28. Thus, in the case of Glasgow in particular, it would seem that the city's population as a whole has been rendered more vulnerable to the key economic and political exposures, resulting in higher mortality across the social scale. In addition, the relative position of Glasgow's middle classes has been made worse because some of the capacities (protective effects) of the comparator cities have related particularly to those of higher socioeconomic status (SES): differences in some aspects of social capital between Liverpool and Glasgow (trust, reciprocity, volunteering) have been shown to be greatest in comparisons of those of higher SES^{57,66,134}; and the qualitative research in the cities suggested Manchester's better cultural adaptation to being a post-industrial centre was most apparent among more affluent residents (albeit that this finding was based on a small study).

- 3. The excess is observed for a wide range of causes of death (with, in the case of premature mortality, a particularly high contribution from deaths from alcohol, drugs, suicide and violence). This overlaps considerably with the second feature discussed above (excess mortality across all social classes). The links between poverty (and the model focuses both on unmeasured aspects of, and on the lagged effects of, poverty and deprivation) and early death from alcohol, drugs, suicide and violence are well evidenced. Socioeconomic deprivation is also a key risk factor for the other common causes of death that have been highlighted in analyses of excess mortality e.g. cardiovascular disease and many cancers. Again, the models suggest causal pathways (outlined above) linking those socioeconomic exposures, associated health behaviours, and a greater vulnerability to their effects brought about by effect modification relating to key historical factors.
- 4. The excess is increasing over time. On one level, this is a difficult feature of excess mortality to comprehend. As Scotland has become relatively less deprived compared with England & Wales in recent decades (at least in terms of how deprivation is routinely measured), its mortality profile has become relatively worse, not better. However, the models (and the evidence on which they are based, presented in more detail in Appendix A) do suggest plausible explanations for this. Principally, the suggestion is that combinations of particular vulnerabilities have 'held back' the Scottish and Glasgow populations relative to other populations. These have occurred as 'sweeps', one following on from the other, and arguably allowing no time for recovery. This is all reflected in slower rates of improvement in mortality compared with elsewhere in the UK. For the later period it links directly to a greater vulnerability to UK economic policies

which, in combination with other factors, saw an increase (in absolute, not just relative, terms) in mortality among those aged 15-44 years over almost 20 years from the early 1980s (a key historical period in the models). As described elsewhere in this report (including within this section), this was driven by increases in mortality from alcohol, drugs, suicide and violence. Although this increase was not observed in most other Western European countries, it was observed in other regions of the UK such as Merseyside and the South Wales coalfields that were also exposed to the (closely interconnecting) factors of deindustrialisation, deprivation and UK economic policy; however, the rates and increases were generally not on the same scale as in Glasgow and West Central Scotland. Indeed, absolute increases in Glasgow – but not anywhere else in the UK. These vulnerabilities, alongside the inadequate measurement of, and delayed effects of, poverty and deprivation, and following on directly from previous vulnerabilities, represent a plausible explanation for the relative increase, albeit one in which period-specific contributions are difficult to quantify.

It is worth highlighting that the report has not explicitly considered the possible relevance and impact of the devolution of powers to the Scottish Parliament since 1999. On the one hand, given that excess mortality increased between 2001 and 2011, it could be argued that this is a noteworthy omission. On the other hand, there is little evidence currently available to suggest that the excess is a consequence of factors coming into play in the relatively short term. Given the time lags generally associated with many aspects of mortality, the available evidence strongly points to the importance of key vulnerabilities, processes and events over a longer period, and involving interactions at different scales (local, regional, national and international). The current excess, therefore, is principally a contemporary manifestation of the consequences of those historical vulnerabilities.

Whether, and to what extent, factors which are attributable to the process of devolution and the use of devolved powers since 1999 might impact on Scottish mortality rates (positively or negatively) is a question which is likely to be considered by future researchers. The economic powers devolved to the Scottish Parliament have thus far been limited, but their use may well prove to be a relevant consideration as time progresses. Some devolved policy areas such as health and social services (see Appendix A15) do not appear to have played a major part in the excess. However, others such as urban policy and planning, housing, local government funding and, indeed, decisions regarding the distribution of funding across Scotland, are highly pertinent to the social determinants of health: as such, they will require particular consideration in the future. Moreover, as further powers are accrued by the Scottish Parliament, it is possible that features of devolution will become increasingly more relevant to Scottish mortality levels.

All that said, action to reduce excess mortality is currently required at all levels of government – including that of the Scottish Government. This is discussed further in the next section of the report.

5. The excess is observed among all adult age groups, with the highest excess among younger working ages. This clearly overlaps with much of the discussion above. However, there are also important time and age elements which need to be explicitly addressed. These relate to the age groups most affected by key historical factors which the models suggest have rendered the Scottish populations more vulnerable, and also to the age groups for which the lagged effects of deprivation would be most relevant. With regard to the latter, higher levels of overcrowding have been evident for Scotland and Glasgow (compared with elsewhere in Britain) for most of the post-war period, although the gap has narrowed considerably - indeed the 2011 Census showed that, for the first time, overcrowding in Scotland was no longer higher than in England. The age profile of those born into, or already experiencing, difficult, overcrowded, conditions is consistent with the age profile of those contributing to the excess in later periods. For example, those born in 1951 would have been 40 years old by 1991 (when Scottish excess mortality was about 7% (10% for premature mortality)) and 60 years old by 2011 (by which point the excess was 10% for all ages and 20% for <65 years). The key historical periods associated with vulnerabilities in the models were in the period 1950s-1980s, and 1980s onwards. This is consistent with the age groups which contribute most to the excess. From the most recent national analyses, the greatest relative differences between Scotland and England & Wales around 2011 were for the 15-44 age band, i.e. relating to those born between 1967 and 1996. This period aligns with the key periods highlighted in the models.

Table 1 below summarises the age profiles of the population in relation to key historical vulnerabilities (in particular those affecting Glasgow) and exposures and age-specific excess mortality.

It has also been suggested that an element of epigenetics^{xxxii} could play a role in the excess among those of younger ages whereby offspring of those affected by the key historical vulnerabilities could themselves be more at risk of future morbidity and mortality. However, as explained in Appendix A14, the supporting evidence for this is very limited, and therefore we do not propose it as a component of the models.

^{xxxii} As discussed in Appendix A14, epigenetic changes refer to the damage that can occur to individual genes due to environmental exposures (defined broadly to include aspects such as poverty) and which can confer an increased risk of disease and death. It has been suggested that such exposures might occur either during an individual's life-course, or in the previous generation with the genetic changes being transferred to the subsequent generation.

Table 1: Age and vulnerabilities associated with excess mortali	ty.
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Year of birth	Associated vulnerability	Age at <u>1981 (</u> all-age excess: 4%; premature excess: 6%)	Age at <u>1991</u> (all-age excess: 7%; premature excess: 10%)	Age at <u>2001</u> (all-age excess: 7%; premature excess: 15%)	Age at <u>2011</u> (all-age excess: 10%; premature excess: 20%)
1951	High levels of overcrowding; nature and scale of urban change; New Town selective migration ^{xxxiii}	30 years	40 years	50 years	60 years
1961	Nature and scale of urban change; New Town selective migration; high levels of overcrowding	20 years	30 years	40 years	50 years
1971	Nature and scale of urban change; New Town selective migration; high levels of overcrowding	<15 years	20 years	30 years	40 years
1981	Local response to UK government policy; high levels of overcrowding	<15 years	<15 years	20 years	30 years
1991	Local response to UK government policy; high levels of overcrowding	-	<15 years	<15 years	20 years

Scottish mortality phenomena

As described earlier in this report, excess mortality plays a major role in the main Scottish mortality phenomena that were outlined in Part Four: the slower improvement in mortality compared with elsewhere in the UK and Europe; Scotland exhibiting the widest inequalities in mortality in Western Europe; and the high (and non-reduction over 30 years) mortality rates among 15-44 year-olds compared with elsewhere in Europe. Thus, in arguing above

^{xoxiii} Note that the peak years for urban change (as defined earlier in the report) and selective population movement were in the later decades, rather than the 1950s. However, the groundwork for later changes (in terms of the start of the demolition and clearance programme, the building of the new peripheral estates, initial movement to East Kilbride) took place in this decade.
that the models explain the key features of excess mortality, we also argue that aspects of these key phenomena are also explained. However, this was also explicitly checked by additional testing of the models. The brevity of this section is explained by the obvious duplication resulting from both sets of testing.

In terms of *the overall slower rate of improvement in mortality* relative to other countries, the discussion of the extent to which the models explain the increasing nature of the excess is entirely relevant; and a greater vulnerability from a number of historical processes resulting in a slower rate of improvement in general, including increases in aspects of mortality (alcohol, drugs, suicide) closely linked to deprivation (both current and inadequately measured, and historical), appears to be a highly plausible explanation for both.

Overlapping with the discussion of the most affected age groups above (including that presented in Table 1), this explanation 'fits' with both the 1950-1980 and 1980-2010 periods. Looking forward from 2010 onwards, it is also consistent with emergent problems in terms of ever increasing rates of obesity, and potential further waves of vulnerability relating to social security cuts ('welfare reform'), and associated widening of socioeconomic inequalities across UK society.

As discussed earlier, there is clear evidence that Scotland exhibits *extremely wide inequalities in mortality* compared with other Western European countries⁷⁻⁹. We also know what the particular age- and cause-related characteristics of those inequalities are i.e. highest in younger working ages, and related to alcohol, drugs, suicide and violence. These latter characteristics have been discussed above, and therefore we can conclude that the model also provides a plausible explanation for this feature of Scottish population health. This is also true of the *high and non-declining rates in mortality in those aged 15-44 years*, for which the models appear to provide plausible explanations.

In summary, the models do appear to account for the principal features of excess mortality, and they are also consistent with the mortality trends that have been observed in Scotland since the middle of the 20th century.

6.3 Strengths and weaknesses

There are a number of key strengths associated with this work. First, the considerable number of hypotheses included within the work, and the manner in which they were identified (including from a systematic review of all proposed explanations for differences in mortality between high income countries) means that this aspect of the research has been extremely comprehensive. Second, and as a consequence of the first point, the scale of the project, including a large number of research projects to assess hypotheses which previously had an absence of evidence, has been another advantage. The fact that the weaknesses of the previous (2011/12) synthesis^{xxxiv} have been addressed is another strength, while other

^{xxxiv} These were principally that: it was reductionist to only evaluate the hypotheses for causality individually, and instead an assessment of potential interactions between hypothesised causes was required; that there was insufficient attempt to explain the patterning of mortality across social groups (i.e. in relation to excess mortality impacting across all deprivation groups (not just those living

aspects of the methodology, including the use of Bradford Hill criteria for causality, have ensured that an appropriately robust approach has been applied to the research. The 'framing' of the effect modifiers around the evidence-based notion of vulnerability added a highly useful dimension to the work. Finally, the testing, and subsequent modification, of the models with key informants from public health and a range of other relevant disciplines, has resulted not only in improved explanatory models but also – and importantly – a broad consensus among key individuals regarding the most likely drivers of excess mortality. This itself, given the many years of debate around this issue, is a helpful step forward.

There are, however, also a number of weaknesses associated with the work presented in this report. First and foremost, the assessment of some of the topics within Appendix A was severely hindered by a lack of robust evidence and data. This was true of, for example, subjects such as diet (Appendix A17), housing quality (Appendix A21), the labour market (Appendix A12), aspects of alienation ('anomie') (Appendix A2) and some features of the physical environment (Appendix A31). Although for some of these topics, specific projects were commissioned both to explore the evidence base and to search for relevant comparative data, in many cases such data were simply not available, and in other cases time constraints meant that not all potentially relevant data could be accessed and analysed. For some topics, comparative data were available for Glasgow (compared with Liverpool and Manchester) or Scotland (compared with England & Wales) but not both, thus hindering the effectiveness of that assessment. A greater number of these 'unknowns' related to comparisons of Scotland with England & Wales, meaning that there is less certainty around how far some of the important vulnerabilities highlighted in the Glasgow model also apply to areas that lie outside the West Central Scotland conurbation. Edinburgh, Aberdeen and Dundee are good examples, given both their geographical position and the high rates of excess mortality that have been observed in comparison with England & Wales. All of these are important weaknesses in a study of this type, and are, therefore, reflected in the list of recommended future research (Part Eight).

The second important weakness of the research is the inability to quantify the impact of each component of the model on the level of excess mortality. Given the nature of the research, and the evidence and data upon which it is based, it is simply not possible to estimate such effects. This weakens the research, but it is a weakness that cannot be overcome.

As stated in the methods section, the use of the Bradford Hill criteria for causality has been criticised by some, with – for example – each criterion on its own deemed insufficient to prove causality (and other commentators have since argued for a greater focus on the multifactorial aspects of disease development²⁴⁸). Similarly, although we have proposed the use of the vulnerability 'framework' as a core strength of the work, the application (and development) of this theory to public health is relatively recent, and work to develop and strengthen it is still ongoing.

in the poorest neighbourhoods); and that there was a lack of clarity around the assessment of the impact of lagged and historical effects⁷⁹.

Despite all these issues, however, we would argue that the core strengths of this work substantially outweigh the weaknesses, and the accumulated evidence has helped provide a much improved insight into the likely causes of excess mortality in Scotland than has previously been available. The penultimate section of the report outlines what the implications and responses to that evidence are.

7. Implications and responses

As summarised in Part Six, the principal contributory components of the two models presented in Part Five are an inadequate measurement of deprivation, alongside a number of important historical processes and developments which, in combination, have rendered Glasgow and Scotland more vulnerable to key socioeconomic and political exposures. A range of sub-topics are part of that broader explanation. In light of that, therefore, what are the most appropriate policy responses to address these issues?

From research to policy

In assessing responses for Glasgow (and the wider area) in particular – the city and area with the highest levels of excess mortality in Scotland – it is important to reflect on the 'context' diagram in Part Four (Figure 10). That emphasised two important points:

- Particular parts of Britain (including Glasgow, Liverpool and Manchester) have been subjected to the same historical, socioeconomic and political negative exposures: high levels of deindustrialisation and associated poverty and deprivation, and UK economic policies which entrenched the cities at the bottom end of a widening spectrum of inequality in the UK. It is no coincidence that in the early decades of the 20th century premature mortality rates were (a) higher in these cities compared with elsewhere in Great Britain but (b) similar in all three cities.
- 2. Processes that rendered Glasgow's population more vulnerable to those exposures resulted in the Scottish city falling behind the English areas in mortality terms in the latter half of the 20th century.

On top of this, there is also a need to consider further, future, vulnerabilities which are likely to emerge from the effects of UK government changes to social security, as well as other government policy on social protection, and reduced public spending.

Thus, policy responses must address three overlapping issues simultaneously:

- to protect against key exposures (e.g. poverty, deprivation) which impact detrimentally across the whole UK (but especially in places like Glasgow, Liverpool and Manchester);
- 2. to address the accumulated consequences of Glasgow's greater vulnerability and the impact of exposures over time;
- 3. to mitigate against the effects of future vulnerabilities linked to current UK government policy.

The important factors which emerge from this analysis – poverty and deprivation, and exacerbated inequality linked to current, past and future vulnerabilities – are intractably entwined. Thus the policy recommendations presented here (made in agreement with all the representatives of public health, and other disciplines, who are signatories to this report) are not set out separately in relation to the three issues above. Instead they are presented together, grouped under four thematic headings: national economic policy (including, specifically, the need to redistribute income and wealth to narrow economic and, by extension, health inequalities in society); housing and the physical environment; additional

actions in relation to local government and partner organisations; and understanding deprivation. As will become apparent, there are also clear overlaps between these headings.

The recommendations have been drawn from different sources: some follow directly from specific research findings; some reflect existing evidence of appropriate responses to issues highlighted in the report; and others have been proposed by others with expertise in the relevant policy areas.

National economic policies

A key point emphasised throughout this report, and elsewhere, is that **economic policies matter for population health**. Widening inequalities in health are a consequence of more general widening inequalities across society, most notably measured in terms of income inequalities. Although the most important fiscal policy levers still remain under Westminster control, it is of paramount importance that **all opportunities available within Scotland are taken to redistribute income and wealth** across Scottish society. These opportunities include those presented by the devolution of new powers listed in the 2016 Scotland Act²⁴⁹. Specific policy recommendations aimed at achieving this are listed below. It should be noted that some of these echo recommendations made by other organisations, including some included within a recent NHS Health Scotland Income Briefing²⁵⁰, as well as policies (highlighted below) which are the subject of proposals by the Joseph Rowntree Foundation (JRF)²⁵¹.

- Economic strategy: making the reduction of income and wealth inequalities the central objective of economic policy is important. It is increasingly recognised that more equal distribution of income and wealth leads to wealthier, healthier, more resilient and democratic economies (even amongst bodies previously advocating a growth-first approach such as the Organisation for Economic Cooperation and Development (OECD)²⁵² and the International Monetary Fund (IMF)²⁵³). Aside from all the other societal benefits this would bring, placing the reduction of income inequalities at the heart of policy would help the Scottish Government to achieve its stated aims of achieving 'inclusive growth'²⁵⁴ and reducing poverty and inequality²⁵⁵.
- Ownership: policies which reduce inequalities in the ownership of capital (e.g. land, housing and shares) are likely to contribute to greater equality of incomes. Models of co-operative ownership (e.g. of companies or land) also have potential to enhance equity.
- 3. **Income and corporate taxation:** increased tax take and greater progressivity in taxation (i.e. a steeper marginal taxation as incomes increase and a fairer alternative to the council tax) would reduce income inequalities and provide greater resources for redistribution and public services.
- 4. Wealth and asset taxation: there is a need to identify ways of redistributing assets more fairly (e.g. land) and taxing the proceeds of wealth (e.g. through a more progressive Land & Buildings Transactions Tax).

- 5. 'Fair work' and wages: support the vision set out in the Fair Work Framework^{256,257}, to ensure that all work in Scotland offers security, fulfilment and respect. This would include the introduction of a Scottish living wage at a level which exceeds that proposed by the UK government, and which would reduce the ratio between the highest and lowest earners. Greater income security (e.g. by providing a guarantee of hours for those who wish them) is another important component of wages policy. Removing barriers to worker organisation and ownership may also ensure that there is a rebalancing of power between the owners of wealth and those who work to create it (e.g. through greater collective bargaining). Although employment law remains a reserved matter, the Scottish Parliament has influence over public sector pay and the supply chain of the public sector.
- 6. Industrial policy: diversify the economy to foster more resilience to change (e.g. reduce reliance on the financial services sector and oil industry) and provide support for those sectors which produce high quality and well-paid jobs in areas of greatest need.
- 7. Social security: the social security system must ensure that all in society have sufficient income, and provide the basis from which people can develop their skills and provide for the needs of their families. This would involve increased levels of protection and less conditionality, such as would be the case with a Citizen's Income²⁵⁸. It will be important to use all opportunities offered by the partial devolution of benefits in the Scotland Act 2016, and to build on existing mitigation (e.g. on housing benefit changes), to protect geographical, equality group, and socioeconomic populations at greatest risk. If possible, this should include reversing the effects of UK government cuts and reforms (e.g. to tax credits, incapacity benefits, housing benefit and child benefits), thereby ensuring the provision of a more effective 'safety net' for the most vulnerable in society. In addition, there may be opportunities to change the administration and culture of (aspects) of the system to one that is centred around the needs of claimants.
- 8. Addressing the costs of living: reducing costs which impact most on the poorest groups (including childcare, housing, heating, transport and food) relative to income is an important component in a strategy to reduce poverty and inequality. In particular:
 - a. The **creation of an 'anti-poverty childcare system'** is important. This is a specific proposal by the JRF, and is based on flexible, year-round child care provision (as already exists for school-age education), professionally qualified staff earning salaries comparable with those working in schools, and an affordable fee system which includes free access to childcare for those on low incomes. Evidence cited in the JRF proposals suggest such a transformation in pre-school childcare would be cost effective in the long run as later savings (e.g. from reduced social security payments, higher pay,

lower costs to criminal justice systems) would exceed the investment many times over. More importantly, such a transformation would have major benefits across all Scottish society, potentially conferring protection against future vulnerabilities.

- b. Reduction of the 'poverty premium' (i.e. the proportionally higher costs of goods and services faced by those living in poverty). New measures in relation to this issue have been proposed by the JRF. Specific recommendations include: changes to the role and remit of competition authorities and regulatory bodies (e.g. strategies to identify, monitor and reduce disadvantage among those in poverty); establishment of innovation funds to encourage third sector and not-for-profit companies to develop products appropriate to the needs of those on low incomes (e.g. specialist insurance policies); enabling interventions such as 'collective switching' in the energy market; facilitating 'transactional banking' based on partnerships between banks and third sector organisations; enabling access to small loans (e.g. through developments to the social finance market or by provision of 'micro-finance' in the banking sector); and various initiatives to improve energy efficiency (and thereby reduce costs).
- c. Alongside the establishment of a living wage, the implementation of the JRF's proposal for a 'living rent', whereby social housing rental costs would be directly linked to local earnings, would make housing costs across the country fairer, and lower the cost of living for some sections of the population.
- d. The cost of **public transport** is significant for those living in poverty. This is particularly the case for those living in Glasgow's peripheral estates and Scotland's rural areas, where amenities are few and journeys to higher amenity locations are long, expensive and can involve a number of stages. While transport services may be better managed locally, free or subsidised transport for those on low incomes could significantly improve accessibility to education, employment and services, particularly for those living in more isolated locations.
- As others have previously recommended (e.g. Oxfam Scotland²⁵⁹, The Poverty Alliance²⁶⁰), the Scottish Government should adopt a 'poverty proofing' approach to all policies and major spending decisions.
- Related to this, and in recognition of the unique health challenges facing Scotland, the Scottish Government should adopt the World Health Organization's principle of 'Health in all policies'²⁶¹ in order to more explicitly address issues relating to the social determinants of health and health inequalities in Scotland.

Housing and the physical environment

Another key aspect of vulnerability identified in the report relates to the physical environment – particularly (and specifically in relation to Glasgow) the availability of good quality housing and the distribution of this housing within the city among social groups. Partly as a result of the substantial deindustrialisation experienced in Glasgow, and of the need to demolish substandard housing, there remains a large quantity of derelict land in the city. Policy to improve housing in the city and the physical environment are therefore important, and could include:

- 11. A substantial **expansion of the social housing building programme** of high quality, low rent, sustainable, social housing. The JRF sees the expansion of affordable housing as being a priority call upon new borrowing and bond issuing powers afforded to the Scottish Parliament.
- **12.** An extension of the Scottish Housing Quality Standard²⁶² to the private rented sector and tied housing.
- 13. Targeting cold and damp housing and people who struggle to afford fuel by implementing affordable heating, ventilation and quality energy efficiency measures in all housing both new and existing properties (without the need to apply for grants) with a focus on private rented and owner occupied sectors.
- 14. Related to the above, ensuring **maintenance and repair funding for social housing** is at an adequate level, and is protected from any impact of cuts to public services.
- 15. Strengthening the impact of the Place Standard for Scotland^{263,xxxv} by: providing ongoing support for its development and delivery; making it a 'material consideration' in the spatial planning system for private and public sector development; and investing in support for communities from deprived areas to use it.
- 16. Improving greenspace access and quality in deprived areas by: providing access to good quality greenspace within 300m of the home for all; addressing current inequalities in greenspace quality; and supporting engagement in outdoor activities (including spaces for all to support intergenerational mixing and spaces to play that challenge children and allow for risk taking).
- 17. Improving neighbourhood maintenance according to need and deprivation levels by ensuring that CPPs, Local Authorities and Scottish Government work together to identify mechanisms to support the ongoing maintenance of streets and open, green, and public spaces, and ensure that environmental incivilities,

^{xxxv} The Place Standard has been developed by Scottish Government Architecture & Place, NHS Health Scotland and Architecture & Design Scotland. Its purpose is "to support the delivery of high quality places in Scotland and to maximise the potential of the physical and social environment in supporting health, wellbeing and a high quality of life".

crime and anti-social behaviour do not act as disincentives to their use and enjoyment.

18. **Improving road safety for pedestrians and cyclists** by establishing 20mph zones, area-wide traffic calming schemes, and segregation of pedestrians, cyclists and traffic, as the norm for residential and urban areas.

Additional actions in relation to local government and partner organisations xxxvi

A number of the above recommendations relate to local, as well as national, government. Further, specific, recommendations relating to local government (including Glasgow in particular) and partner organisations include:

- 19. The need to recognise, understand and act upon at the highest levels of local government the impact of local decision making on population health. This includes the need to understand the need for and protective benefits of strong civic leadership in times of political and economic difficulty.
- 20. The need to avoid repeating historical mistakes and ensure that we create, and keep together, viable and supportive communities, and build further affordable public sector housing.
- 21. Local government also has a part to play in **distributing income**, with progressive use of proceeds from a **fairer system of local taxation** (e.g. in redistributing resources towards areas of greater need).
- 22. There is also an argument that current local authority boundaries prevent adequate redistribution across the country. Given the controversial manner in which the boundaries were created in the 1990s^{264,265}, **the boundaries and/or the funding allocation system for local government should be reviewed** with the explicit objective of ascertaining whether any potential changes could more effectively facilitate resource distribution across Scotland.
- 23. As with national government, a **'poverty proofing'** approach to local government (and partner organisation) policy-making should be adopted, alongside the WHO's **'health in all policies'**.
- 24. Related to the above, an approach to local policy-making should be adopted which explicitly embraces previously identified **actions to narrow inequalities at the local level**²⁶⁶. The latter include: implementation of the living wage at a level and coverage recommended by the Living Wage Foundation^{XXXVII} (and in the case of Glasgow City Council in particular this should include the various 'arm's-length external organisations' (ALEOS)); adoption of 20mph speed limits across the whole city (mentioned above); and taking 'health first' approach to tackling worklessness.

^{xxxvi} 'Partner organisations' include Health & Social Care Partnerships and Community Planning Partnerships.

xxxvii See: <u>http://www.livingwage.org.uk/what-living-wage</u>

25. In the case of Glasgow City and the other local authorities within the Glasgow and Clyde Valley region^{xxxviii}, consideration should be given as to how to maximise the potential of **City Deals**²⁶⁷ investment to help mitigate against the effects of vulnerability in the population (e.g. through capital investment in social housing or the creation of sustainable high quality employment).

Understanding deprivation

Finally, there are a number of aspects of the excess mortality phenomena that we do not understand sufficiently, and there are, therefore, a number of research priorities which flow from this work. A full list of such priorities are listed in the next section of the report. However, one in particular is highlighted here, given its particular importance to the explanatory models presented in the report and its links to the issue vulnerability discussed above:

26. There is an urgent need to prioritise **further research on the true nature of deprivation** in Scotland that is not captured by existing data and measurements. The evidence included within this report strongly suggests that the 'lived reality' of living in socially and materially deprived circumstances in Scotland differs from elsewhere in Britain, and it is imperative that new research, perhaps based on ethnographic methodologies and involving a comparative approach, is undertaken to better understand those differences, and to formulate appropriate policy responses.

Overall conclusions, alongside additional recommendations for further research, resulting from all the work presented in this report are included in the next, and final, section.

^{xoxviii} As defined by the UK government's 'City Deal' report²⁶⁷, these are: East Dunbartonshire; East Renfrewshire; Glasgow City; Inverclyde; North Lanarkshire; Renfrewshire; South Lanarkshire; and West Dunbartonshire.

8. Conclusions and recommendations for future research.

This report has summarised the results of a considerable amount of research from across a range of disciplines into the reasons why Scotland's population health profile, compared with that of our near neighbours in England & Wales, has, in relative terms, worsened over the past 30-40 years, even when differences in poverty and deprivation – the main drivers of poor health in any society – have been taken into account.

A large number of key representatives of the public health community in Scotland, and representatives of other important disciplines, are now – on the basis of the material included within this report – in broad agreement both with what the main causes of this excess mortality are, and what, therefore, the responses to that now must be.

As we have emphasised in a number of places in this report (including in Part Seven), we do not suggest that our models are perfect, nor that a number of important questions, and gaps in our knowledge, do not remain. Indeed Box 2 below lists a considerable number of areas for future research, reflecting where the evidence base is lacking. However, in 'stepping back' from the intricate detail and complexities of individual research projects and assessment of the merits of individual hypotheses, and in adopting instead an 'overview' of what all the research evidence, in its interlinked totality, indicates, we believe we have considerably furthered our understanding of the causes of Scotland's and Glasgow's excess mortality. What is important now is that there is an appropriate response to that evidence to improve the health of Scotland's population. This must be done alongside, and entwined with, ever more urgent actions to address the key drivers of overall poor health in the country – poverty and deprivation – and to seek to narrow the widening gap in wealth and, therefore, health in Scottish society. Box 2. Recommendations for future research

- As stated in the previous section, research should be prioritised to fully understand **unmeasured differences in living in deprived circumstances** in Glasgow and Scotland compared with elsewhere in Britain. This may best be achieved through comparative ethnographic methodologies, although other qualitative approaches could be considered.
- Early years: the importance of early years' experiences for later adult outcomes is well understood. However, robust comparative data for Scotland and England are lacking, and the data that are available do not allow the study of links between relevant exposures (e.g. childhood adversity and complex trauma), modifiers (parent-child attachment) and adult health outcomes. To rectify this, new research is needed to collect accurate, unbiased, measures of such exposures (alongside a range of potential confounding variables) and to undertake analyses based on an understanding of the correct periods of risk exposure and outcome.
- Research into the historical vulnerability of the Scottish population should explore **local government responses** in the key periods highlighted in the models (1950s-1980s, and 1980s onwards) in areas outside the WCS conurbation.
- Employment & labour market: potential differences in aspects of employment and the labour market (e.g. workplace environment, work quality, remuneration, precariousness) between Glasgow, Liverpool and Manchester were the focus of recent research²⁶⁸. However, the scope of that work should be extended to analysis of these, and other related issues, using various datasets identified by the recent work, and by extending the analyses to Scotland compared with England & Wales.
- The complex interactions between poverty, health outcomes, **ethnic diversity** and excess mortality should be explored.
- Related to the above suggestion, the (tentatively) proposed protective factors in Manchester should be the focus for further research. This relates both to the issue of greater ethnic diversity, but also to the suggestion that the city's culture is one that has adjusted better to the transformation from industrial to post-industrial society (in particular in terms of what might underlie that different adjustment).
- Further modelling analyses of Scottish and English health survey data and mortality should explore interactions between social class and emerging health problems such as **obesity**, as well as class, income and **educational attainment**.
- Health and social services research: differences in the scope and quality of health and social services (e.g. drug addictions services) between Scotland and England, and their impact on differences in health outcomes, would be another useful area of further research.
- A recent review of **housing quality** and allocation between Scotland and England was limited in its scope because of time and resource restrictions. New research, focusing on

potential differences in current and historical housing quality and their potential impacts on population health, is required to fill that gap.

- As stated earlier, further research on the role of the **physical environment** as a component of deprivation is required. More generally, research into differences in the physical environment between Scotland and England (focusing particularly on urban areas), based on the collection of new comparative data on land use and quality, and linked to existing evidence of associations between environment and health, would be extremely helpful.
- Exploratory research into how best to measure of **alienation ('anomie')** among Scottish and comparator populations would be helpful.
- **Diet**: a better understanding of differences in diet between Scotland and England, and between Glasgow, Liverpool and Manchester, alongside quantification of the impact of those differences on all-cause and cause-specific mortality, should be undertaken. Although new research has been recently published, it was again limited in its scope for reasons of time and finance, and it would be helpful to fill gaps in the evidence base that were identified by that research.
- Further exploration of the '**fundamental causes**' theory of health inequalities^{109,110} and how the causes of inequalities are evolving over time would be important, particularly in terms of looking forward to new, emergent, aspects of health inequalities.
- Valuable lessons for policy may be derived from examination of **other deindustrialising and vulnerable populations** beyond Europe (e.g. 'rust-belt' areas of the US).
- Comparative analyses of trends in mortality across peripheral estates, outer estates and New Towns in Scotland and NW England would be potentially informative.

Appendices

Appendix A. Assessments of hypotheses.

- A1 Air pollution
- A2 Anomie
- A3 Artefact: inadequate measurement of poverty and deprivation
- A4 (Culture)
- A5 Climate: rainfall
- A6 Climate: vitamin D deficiency
- A7 Climate: winter deaths
- A8 Culture of dependency
- A9 Culture of substance misuse
- A10 Early years: family, gender relations and parenting differences
- A11 Educational attainment
- A12 Employment/ labour market
- A13 Ethnicity
- A14 Genetics
- A15 Health & social services
- A16 Health behaviours alcohol
- A17 Health behaviours diet
- A18 Health behaviours drug misuse
- A19 Health behaviours physical activity
- A20 Health behaviours smoking
- A21 Housing quality and provision
- A22 Impacts of the World Wars
- A23 Income inequalities
- A24 Individual values
- A25 Lagged effects of poverty and deprivation

A26 Migration

A27 Obesity

- A28 Political influences and vulnerability
- A29 Premature and low birthweight babies
- A30 Quality of external physical environment: land contamination
- A31 Quality of external physical environment: vacant & derelict land
- A32 Scale of deindustrialisation
- A33 Scale and nature of post-war urban change
- A34 Sectarianism
- A35 Sense of coherence
- A36 Social capital
- A37 Social mobility
- A38 Spatial patterning of deprivation
- A39 Terminations of pregnancy
- A40 Water hardness

A1 Air pollution

Description of hypothesis

Greater direct exposure to air pollution has been proposed as a cause of excess mortality in the Scottish populations.

Rationale

If historical or contemporary air pollution exposure has been/is relatively worse for the Scottish populations then it is possible that this could explain the excess mortality given the wide range of negative health impacts it causes.²⁶⁹ In particular, chronic conditions such as cardiovascular disease, respiratory conditions and a wide range of cancers are associated with higher exposure²⁷⁰⁻²⁷².

Links to other hypotheses

Exposure to air pollution is linked to a wide range of other factors including: (the type and position of) housing; employment (e.g. nearby industry type); and health behaviours such as smoking²⁷³.

Evidence overview

Both short-term and long-term exposures to air pollution are relevant to health, as are different forms of pollution. Indoor air pollution is heavily influenced by the form of heating system in place (e.g. solid fuel fires with inadequate ventilation), whether there are smokers within the household, the ventilation and drying facilities available and used, and the prevalent outdoor air pollution²⁷³⁻²⁷⁵. Outdoor air pollution is influenced by the co-location of polluting industries, housing using polluting fuels (such as coal), transport density and dominant fuel type used (e.g. diesel), and the prevalent wind direction. Air pollution can also take a variety of forms including particulates (e.g. PM10s), chemical toxins (e.g. sulphur dioxide) and biological agents such as viruses.

Air pollution is known to be harmful to health. Around 80% of deaths linked to air pollution are due to ischaemic heart disease and strokes, with almost all the rest due to chronic lung disease, respiratory infections and lung cancer. The most important pollutants are: particulate matter (PM), ozone (O₃); nitrogen dioxide (NO₂); and sulphur dioxide (SO₂)²⁷⁶. The association between exposure to air pollution (particulate and nitric oxide) and respiratory disease has been confirmed within Clydeside²⁷⁷.

Some specific air pollution episodes have been noted to have been particularly severe in Scotland (e.g. smog in winter 2001), but these have not been prolonged (in recent times) nor have they uniquely impacted on Scottish populations²⁷⁸.

Recent exposure to particulate air pollution (for mean anthropogenic PM $_{2.5}$ µg⁻³) has been modelled for local authority areas across the UK. The exposure estimates for Scotland overall (at 6.8), and for Glasgow in particular (at 8.3) were lower than for England (at 9.9), and for Liverpool (at 9.6) and Manchester (at 10.4), leading to lower estimates for Scotland and Glasgow for the attributable fraction of deaths^{279,280}. The similar, or slightly lower, air

pollution estimates are observed back to 2003 when the time trends began.²⁸¹ However, there is a lack of historical data and evidence comparing the exposure of Scottish populations with others across the range of potential pollutants. Given the known impacts of air pollution from factors such as heavy industry and coal burning (e.g. from power generation, steelmaking and heavy engineering) which were much more prevalent historically,²⁸² it is possible that historical differential exposures may be partially responsible for some aspects of the excess.

Note that there are some aspects of industrial air pollution exposure which impacted most on workers and their families (such as to asbestos) that are known to have been high in Glasgow and are responsible for a number of deaths from lung cancer and asbestosis²⁸³.

Conclusion

The available data suggest that Scotland and Glasgow are not exposed to higher air pollution than the comparison populations, but historical data are not available prior to 2003. Greater exposure to air pollution is causally associated with some of the important causes of excess mortality such as IHD, stroke, respiratory disease and lung cancer, and Scotland and Glasgow did have a concentration of industry historically that could plausibly have generated high exposure to air pollution; however, it is unknown whether or not this would have been worse than elsewhere. It is, therefore, uncertain whether or not air pollution may have historically been partially responsible for aspects of the excess mortality phenomena, or whether lagged impacts may be important for older adults. However, contemporary exposure to air pollution does not make a contribution to Scottish excess mortality.

Assessment of evidence of causality

1)	General	assessment	of likelihood	of causality i	n terms of Bra	dford Hill criteria.
-,				or caucancy r		

Bradford Hill's criteria for	Assessment ^{xxxix}	Comments
causality		
Strength of	Y	
association		
Temporality	Y	
Consistency	Y	
Specificity	Y	Air pollution is a known negative influence on
Biological gradient	Y	substantial burden of disease.
Plausibility	Y	
Coherence	Y	
Experiment	Υ	
Analogy	Υ	

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor	Ν	Exposure to air pollution
worse in Scotland?		is better in Scotland than
		in England &Wales,
Assuming a difference in exposure,	U	although little time trend
would this have occurred prior to		
		data were available.
the outcome?		

^{xxxix} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments	
Is the hypothesised causal factor worse in Glasgow?	N	Exposure to air pollution is better in Glasgow than in Liverpool and	
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	Manchester, although little time trend data were available.	

A2 Anomie

Description of hypothesis

It has been suggested that sections of Scotland's – and in particular Glasgow's – population may be affected by a form of 'anomie' (or 'boundlessness and alienation'), and this has a detrimental impact on health outcomes.

It should be noted that the use of 'anomie' can suggest two rather different hypotheses: anomie, as proposed and defined by Durkheim in the 1800s; and the term's later adoption (and, arguably, misuse) by a number of commentators in relation to 'underclass' and 'culture of poverty' theories. Both these uses of anomie discussed here.

Rationale

Anomie was introduced as a concept in the 19th century by the French sociologist Emile Durkheim to describe the breakdown of social and moral norms that follow periods of economic and social change^{284,285}. Durkheim argued that such change can bring about less regulated, less integrated societies in which previous social norms no longer apply and no longer control the behaviour of individuals. As a result, 'anomie' leads to increasing levels of crime and 'deviant behaviour'. Durkheim focused on suicide as one manifestation of a more generalised set of self-destructive behaviours. Anomie has also been used to explain how the socioeconomic disruption precipitated by the collapse of the USSR led directly to deteriorating health in ex-Communist countries (most notably Russia) from the early 1990s onwards²⁸⁶. The latter analysis focused on the reduction in life expectancy, the widening of health inequalities between regions and the striking impact of substance misuse, especially alcohol-related harm, among men – all factors which have their parallels in Glasgow^{4,5}, a city that in recent decades has experienced the economic and social transformation from an industrial to post-industrial, service-sector based, economy^{4,18,20,287}.

When considered as a potential explanation for Scotland's excess mortality in the 2011 synthesis report, the concept was described under a more general heading of a 'culture of boundlessness and alienation'. This was specifically to distinguish it from the 'underclass' theory²⁸⁸⁻²⁹¹ with which anomie has become associated, and which has been attacked for 'demonising' the poor. The hypothesis arguably has parallels with the 'culture of poverty' thesis proposed by Murray and others, which proposes that poverty is less the cause of social problems, but instead that social problems result from cultures endemic within the poorest groups in society²⁹².

Thus, there are two distinct causal pathways, relating to the two uses of the notion of anomie, that link the concept to health outcomes. In the first, poverty (brought about by economic change) leads to a breakdown in, or abandonment of, societal 'norms', resulting in self-destructive behaviours (e.g. substance misuse, suicide) which impact on mortality. In the second, however, the poorest in society are instead blamed for having an inherently negative culture which includes health damaging behaviours which, in turn, leads directly to higher mortality.

Links to other hypotheses

As discussed further below, there is an overlap between the notion of anomie and Antonovsky's 'sense of coherence'. It also links to aspects of the 'individual values' thesis (in particular psychological outlook), social capital (in terms of social connections), and behaviours such as alcohol and drug misuse. The original Durkheim concept also overlaps with the deindustrialisation and political influences hypotheses, while the 'culture of poverty'/ 'underclass' version of anomie links closely with the idea of a 'dependency culture'.

Evidence overview

A) Durkheim's concept of anomie

As detailed elsewhere in this report, there is a large contribution to Scottish excess mortality (in particular, premature excess mortality) from 'self-destructive' causes – suicide, violence, alcohol and drugs. These are also highly socially patterned, with much higher mortality from these causes among the poorest in society. This probably explains the emergence of this hypothesis in the first place.

No comparable population level data on the concept of anomie have been identified for Scotland and England. Given this, it was included in the three-city (Glasgow, Liverpool and Manchester) population survey undertaken in 2011 (and described elsewhere in this report). Different survey questions were employed to measure the concept. Antonovsky's Sense of Coherence (SoC) overall scale (also discussed elsewhere in this report) was deemed directly relevant, as were some of the individual questions that make up the scale, in particular those within the 'meaningfulness' sub-scale^{xi}. In addition the *conformity* value of Schwartz's Human Values scale (also discussed elsewhere in this report) was also deemed relevant to the hypothesis: with anomie defined as the breakdown in, or lack of, social values or norms (resulting potentially in greater risk-taking and self-destructive behaviours), the *conformity* value of Schwartz's scale instead captures respondents' perceptions of the importance of such social norms^{xli}.

Overall, there was no evidence from the survey of any differences between Glasgow and the two English cities that might support the hypothesis. As discussed in Appendix A35, the survey analyses showed both the overall SoC scale and its 'meaningfulness' sub-scale to have significantly higher mean scores among the Glasgow sample compared with the English

^{x1} As described in Appendix A35, Antonovsky's measure of 'sense of coherence' comprises three components: comprehensibility, manageability and meaningfulness (of life). The individual questions which make up the latter part, and which are especially relevant to the notion of anomie, relate to: whether or not the respondent cares about what goes on around them; whether their life has any clear goals or purpose; whether daily activities are a source of pleasure and satisfaction, or whether instead a source of pain and boredom; whether or not there is meaning in the things the respondent does in their daily life.

^{xli} The 'conformity' value is derived from responses to the following two statements: (1) He/she believes that people should do what they're told. He/she thinks people should follow rules at all times, even when no-one is watching; (2) It is important to him/her always to behave properly. He/she wants to avoid doing anything people would say is wrong.

samples, and this was the case in the vast majority of stratified analyses (age, gender, social class and so on). Analyses of the individual questions generally showed a similar pattern. Similarly, analyses of Schwartz's 'conformity' value from the survey showed it to be significantly *more* associated (not less) with the Glasgow sample than with those in Liverpool and Manchester. The greatest differences were in comparisons of the least deprived areas, with no significant differences shown in comparisons of those in the most deprived areas. Similar results were obtained from analyses by social class rather than area-based deprivation.

Thus, survey data do not support the hypothesis of Durkheim's concept of 'anomie' affecting Glasgow's population (and this reinforces the negative assessment of the hypothesis made in the 2011 synthesis report). Clearly, however, the extent to which such data accurately measure the notion of anomie, whether *any* scale could accurately capture it^{xiii}, and whether a population survey of the type employed in 2011 would adequately represent the section of the population theoretically at most risk of being affected by it, is very much open to question (although other surveys, which are subject to similar response biases, still show a high level of excess in mortality).

B) Underclass/culture of poverty theories

As stated in the 2011 synthesis report, there is conflicting evidence around whether any kind of distinctive subculture exists in parts of the UK: the debate concerns whether the most disadvantaged sections of the population simply lack material resources to participate in societal norms and cultural activities, or whether instead they are associated with a different set of values and behavioural norms compared with the rest of society. As discussed elsewhere in this report, social polarisation in the UK increased dramatically from the 1980s onwards³⁰³, but there is no evidence to suggest that this generated a distinctive culture³⁰⁴ – instead the evidence suggests that behaviours have been shared across society, but are socially patterned^{289,305}. There is a clear overlap with debates within public health on the causes of health inequalities, and the weight of public health evidence across the globe (including the World Health Organization and many others) tends to support the neomaterialist theory i.e. that the main drivers of health inequalities are economic (rather than behavioural) and structural (rather than individual); there is little evidence to support a cultural-behavioural theory^{107, 306- 310}. Several aspects of the evidence base are relevant here. Studies which test whether socioeconomic factors in early life determine subsequent health and social outcomes or vice versa using longitudinal data convincingly show it is socioeconomic factors that are most important^{107,311}. Furthermore, other longitudinal studies show that changes in the socioeconomic patterning of health behaviours such as

^{xlii} Other methods have been used in attempts to measure the concept of anomie. Perhaps the best known is the five-item 'Srole' scale²⁹³ developed in the 1950s. This includes questions on: perceived futility of engaging with public officials; the need to 'live for today' and not worry about tomorrow; perception that the 'lot of the average man' is 'getting worse' rather than better; that the future holds a bleak outlook; the lack of being able to rely on people for support. The scale has, however, been criticised as being less a measure of anomie and more of hopelessness and despair. Others include the Dean Alienation scale²⁹⁴ developed in the 1960s, as well as various other alienationrelated scales (e.g. Nettler²⁹⁵, Middleton²⁹⁶, and Streuning & Richardson²⁹⁷), many of which also overlap with aspects of SoC, especially its *meaningfulness* component.

smoking do not result in changes in mortality inequalities (again evidencing that socioeconomic factors are the most important and that the pathways linking socioeconomic status and health outcomes can change over time^{107,312}). Finally, a high quality systematic review showed that changes in the socio-political situation have been found to be the most important factor in explaining increased health inequalities, rather than cultural factors.

Conclusion

The available evidence suggests it is unlikely that Durkheim's notion of anomie plays a major part in Scotland's and Glasgow's high levels of excess mortality. However, there are many limitations and caveats associated with the data on which this assessment is made. The weight of public health evidence does not support the notion of 'anomie' in relation to an underclass theory/culture of poverty, as used by US commentators such as Murray^{290,292}.

Assessment of evidence of causality

A) Durkheim's concept of anomie

Bradford Hill's	Assessment ^{xliii}	Comments
criteria for		
causality		
Strength of	Y	
association		
Temporality	U	
Consistency	Y	
Specificity	Y	Durkheim's original work, and more recent descriptions of events in places like the former USSR, suggest that
Biological	U	forms of anomie may be associated with mortality
gradient		outcomes. The extent to which this has been a
Plausibility	Y	
Coherence	Y	
Experiment	U	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

^{xlii} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	U	There are no data available to compare Scotland with England &
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	Wales.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments	
Is the hypothesised causal factor worse in Glasgow?	Ν	The limited data available to examine aspects of anomie suggest no	
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	anomie suggest no difference in Glasgow (or even a lower prevalence) than in Liverpool or Manchester.	

B) Underclass/culture of poverty theories

Bradford Hill's criteria for causality	Assessment ^{xliv}	Comments
Strength of association	N/A	
Temporality	N/A	
Consistency	N/A	There is little evidence for such a culture existing which
Specificity	N/A	makes assessment using the Bradford Hill criteria
Biological gradient	N/A	politicised and contested, but there is little evidence to support the existence of such cultures or evidence to
Plausibility	N/A	support such a culture as a cause of poverty or ill-health.
Coherence	N/A	
Experiment	N/A	
Analogy	N/A	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor	U	We have not examined
worse in Scotland?		data to compare
		measures at the Scotland
Assuming a difference in exposure,	Y	level. Given the lack of
would this have accurred prior to		
would this have occurred prior to		evidence for the theory.
the outcome?		endence for the theory,
the outcome?		however, it is an unlikely
		contributor.

^{xliv} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	N	For those markers of dependency that the advocates argue for there
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	is little difference between the cities. However, these markers did increase at the time of the emergence of the excess.

A3 Artefact: inadequate measurement of poverty and deprivation

Description of hypothesis

The hypothesis here is that higher mortality in Scotland/Glasgow, compared with the rest of the UK, is in fact entirely explained by higher levels of deprivation and poverty, but that existing measures of the latter fail to capture important differences between Scottish and other UK populations. Excess mortality is therefore an artefact created by inadequate measurement.

Rationale

There is a wealth of epidemiological evidence, amassed over many years and across different parts of the world, of the importance of poverty and socioeconomic deprivation in explaining adverse health outcomes^{3,105,107-109,120,121,196,197}. It is argued, therefore, that currently used measures in the UK simply fail to capture differences in the 'lived reality' of deprivation and poverty in Scotland, and especially in Glasgow.

Links to other hypotheses

This is closely related to the 'lagged effects of deprivation' hypothesis, and is also highly relevant to others such as: the spatial patterning of deprivation; housing quality and allocation; the scale and nature of urban change; deindustrialisation; quality of physical environment.

Evidence overview

On the one hand, a number of arguments can be made against this hypothesis. First and foremost, high levels of excess mortality have been observed in comparison of non-deprived populations as well as deprived^{21,24,25,29,31}, and this is an important characteristic of Scottish excess mortality. Second, 'relative poverty' is an internationally recognised and accepted measure: analyses have shown that such poverty is not higher in Scotland compared with the other nations of the UK, and this has been the case consistently for a number of years³¹³. At the city level, Glasgow, Liverpool and Manchester (and surrounding areas) have been shown to be similar not just in terms of single measures such as income deprivation, but also in terms of measures of absolute poverty, relative poverty, child poverty, as well as a broad number of related social characteristics (e.g. lone parenthood, rates of teenage pregnancies)^{20,30}. Indeed for some of the latter measures, rates in Glasgow have been shown to be lower than in the English cities. Importantly, an excess level of mortality has been shown for Scotland compared with England & Wales, and for Glasgow compared with other UK cities, no matter the measure, nor the geographical unit of calculation, that has been used. Nationally, an excess for all Scotland has been shown using area-based measures such as Carstairs and 'employment deprivation'^{21,25}, and using individual measures of socioeconomic status such as social class, educational attainment, housing tenure and car access^{22,24}. City comparisons have also shown an excess based on different measures (e.g. income deprivation and car ownership)^{29,56}.

However, there are arguments in favour of this hypothesis.

As emphasised by a considerable amount of research, poverty and deprivation are extremely complex constructs. They encompass many diverse and overlapping dimensions^{196,198}. It is, therefore, extremely unlikely that any routine administrative indicators (e.g. from the census or social security systems – even when included within more recent measures of multiple deprivation) can fully capture those many different facets.

The complex nature of deprivation is further emphasised by the major changes – discussed elsewhere in this report – that have taken place across UK society since the early 1980s in terms of a dramatic widening of socioeconomic inequalities, and resulting 'social exclusion' and marginalisation of sections of the population^{6,130,133,200-202}: thus there will have been changes in the experience of relative deprivation over that period that will not have been captured by the routine indicators used in analyses over time. Indeed, the mortality profile of Glasgow in the decades since the 1980s has been characterised – particularly in the city's most disadvantaged communities – by relatively higher rates of death from more socially-determined causes: alcohol, drugs and suicide (see Figure 2 in Part One of the report) i.e. what might be described as the 'diseases of despair' associated with people living with, and attempting (or failing) to cope with, difficult circumstances. It can be argued that the complexity of – and changes in – these aspects of relative poverty, and the associated 'lived experiences' of those who have suffered it, lie beyond measurement by routine administrative recording systems.

Related to these changes, it is notable that at the start of the 1980s, much of Scotland's higher mortality (compared with England & Wales) was explained by higher levels of deprivation (as measured by the Carstairs & Morris index, calculated for Scottish postcode sectors and English electoral wards^{xiv}). That was no longer the case by 1991 and 2001, and even less so by 2011: as was suggested in the 2011 synthesis, that is likely to reflect the fact that that index is now out of date and no longer adequately captures differences in poverty between Scottish and English populations (given that it is based on a combination of pragmatism (using data available from the census) and measures designed to capture the lived reality during the mid-20th century (when overcrowding and car ownership were more sensitive measures of that experience)). In comparative analyses, the use of employment deprivation^{xlvi}, calculated for smaller geographical units^{xlvii}, reduced the excess considerably in comparison of those living in the most deprived areas (from 15.1% using Carstairs to 9.7% using employment deprivation). The use of employment deprivation in those analyses did not, however, reduce the overall levels of Scottish excess mortality: however, like income deprivation, this is a measure based on recipients of social security benefits, and as such, subject to considerable weaknesses. Individuals are either in receipt of such benefits or they

^{xiv} The Carstairs & Morris deprivation score is based on four standardised census variables: adult male unemployment; lack of car ownership; low social class; overcrowding.

^{xlvi} This was the used in the Scottish Index of Multiple Deprivation (SIMD) 2012: derived from UK Department of Work & Pensions (DWP) data, it is calculated as the percentage of the working age population who are receipt of a number of employment-related social security benefits. These combine unemployment related benefits (e.g. Jobseekers' Allowance) and sickness-related benefits (Incapacity Benefit, Severe Disability Allowance).

^{xivii} These were Lower Super Output Areas (LSOAs) for England & Wales, and an equivalently-size bespoke geographical unit created for Scotland, based on amalgamated pairs of 'datazones'. The average population size in both England & Wales, and Scotland, was approximately 1,600 people²⁵.

are not: there is no scale associated with lesser or greater need. Thus, there is likely to be a 'ceiling effect'. This has been pointed out in relation to the 'three city' analyses of deprivation and mortality.

Although an excess exists no matter the measure of deprivation/SES used, the level of excess has varied considerably, which again poses questions regarding the sensitivity of the measures used. The excess observed in Glasgow compared with other UK cities was reduced considerably when car ownership (a component of the Carstairs index) was used rather than income deprivation and the whole Carstairs index^{27,56}, albeit that questions have been raised regarding the suitability of car ownership as a proxy measure of income and deprivation²³³⁻²³⁷. It is also known that increasing the range of socioeconomic measures used in analyses tends to explain more of the variation or inequality in the health outcomes – suggesting that a more comprehensive and multidimensional set of measures of poverty may actually explain a greater proportion of the mortality phenomena.

There are some aspects of deprivation (particularly overcrowding) which have been notably worse in Glasgow and Scotland since the post-war period. Thus although employment and income related measures have shown little differences between the cities of Glasgow, Liverpool and Manchester, other aspects of life may have been – and continue to be – quite different, overcrowding being just one (more easily measured) example.

The 2011 synthesis considered many of the issues discussed here (increased insensitivity of the Carstairs index over time; size of geographical units of analysis; failure to capture the 'reality' of deprivation using routine data) and some of the further research questions posed have since been answered^{25,56}. In addition the report discussed a further potential artefactual component: the underestimation of the size of the Scottish population denominator used in analyses, an issue also alluded to by Dorling³¹⁴. However, as the level of excess mortality was lower around the time period most affected by any potential undercount (around the time of the 1991 Census) than it is now, this seems a less likely explanation. It is rendered even less likely by the fact that comparably high levels of excess mortality have been shown in analyses of individual level data (i.e. individuals linked to death records)^{22,24} rather than solely area based data.

Conclusion

It is possible that part of the excess mortality in Scotland and Glasgow is due to inadequate measurement of the lived reality of poverty and deprivation, although the only differences that are evidenced relate to overcrowding (and, more contentiously, car ownership).

Assessment of evidence of causality

Bradford Hill's criteria for causality	Assessment ^{xiviii}	Comments
Strength of association	Y	
Temporality	Y	
Consistency	Y	By definition, inadequate measures of poverty and
Specificity	Y	deprivation do not lend themselves to quantification and
Biological gradient	Y	testing. There is evidence that multiple measures of deprivation are more helpful, and that existing measures have become dated and may not reflect the lived
Plausibility	Y	experience and depth of poverty within the population.
Coherence	Y	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland? Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	Where multiple measures of SES are available, the excess decreases, suggesting that more and better measures would explain a larger proportion of the total excess. Some proxies of lived experience, such as overcrowding, display large differences.

^{xlviii} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow? Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	Where multiple measures of SES are available, the excess decreases, suggesting that more and better measures would explain a larger proportion of the total excess. Some proxies of lived experience, such as overcrowding, display large differences.

A4 Differences in culture

NB This hypothesis – that there are differences in culture between Scottish and English populations which impact on differences in health status – has not been assessed and summarised in the same manner as the other hypotheses included within this Appendix. This is because it is not a single, specific hypothesis, but is rather an 'overarching' theory relevant to a number of other 'cultural' theories that have been systematically assessed in this report. These other cultural theories include:

- individual values (psychological outlook (e.g. optimism, aspiration/achievement, meaningfulness of life), self-efficacy, hedonism, time and risk 'preferences', individualism, materialism, consumerism) (Appendix A24)
- a culture of substance misuse (Appendix A9)
- sectarianism (Appendix A34)
- social capital (Appendix A36)
- behavioural hypotheses (e.g. alcohol (Appendix A16), drugs (Appendix A18), diet (Appendix A17) and physical activity (Appendix A19)).

However, in addition to the individual assessments carried out for these many culturerelated hypotheses, a specific literature search was also undertaken which sought specific mentions of culture or cultural aspects in relation to differences in mortality and other health outcomes between Scotland and England. No relevant studies were identified.

It should also be noted that, as discussed in the main part of the report, a qualitative research project (published in 2015) was undertaken in Glasgow, Liverpool and Manchester, which looked at some of the above hypotheses (psychological outlook, social capital) and others (family life, social mobility, anomie) from a specifically cultural perspective. More broadly, the research investigated whether 'the causes of Glasgow's poorer health profile are located in the culture of the city'. The findings emphasised that 'culture is dynamic and ever-changing with residents of all three cities responding and adapting to changes in the economic and social underpinnings of their lives in particular places'. The research highlighted potential differences between the cities in terms of economic aspects, the 'wider welfare landscape', and in relation to community and mutual support, but argued that Glasgow's experience may actually not be distinctive or surprising, but rather that Liverpool and Manchester may have protective characteristics. For Liverpool this was the 'strong sense of social solidarity' ('a core component of city identity and culture'), a characteristic also suggested by the social capital research discussed elsewhere in this report. For Manchester, it was suggested that the city's culture is one that has adjusted better to the transformation (common to all three cities) from industrial to post-industrial society: the city had experienced a "cultural adaptation to more mobile lifestyles well suited to the changing nature of employment opportunity in a post-industrial economy". This aspect of the city's culture was "reflected in its more cosmopolitan character"; the latter also echoes the suggestion (outlined elsewhere in the report) that Manchester's more ethnically diverse population may also offer protective effects in terms of health.

A5 Climate: rainfall

Description of hypothesis

Related to other climatic hypotheses, it has been suggested that Scotland (and Glasgow/West Central Scotland in particular) experiences higher levels of rainfall than other parts of the UK, and that this has a detrimental effect on health, in particular mental health.

Rationale

It has been assumed both that Scotland and Glasgow experience more rainfall, and that there is evidence of links between weather (including seasonality) and health status.

Links to other hypotheses

Other climate-related hypotheses (winter deaths; vitamin D deficiency) and physical activity.

Evidence overview

a) Does Scotland/Glasgow experience more rainfall?

Data from the Met Office (formerly the Meteorological Office, the UK's weather service) averaged over 20 years (1981-2000) show that Scotland has, on average, experienced more rainfall (around 1,570mm total per year, and approximately 188 days per year with at least 1mm rainfall) than the UK as a whole (equivalent figures of approximately 1,370mm, and 156 days)³¹⁵. Mean rainfall data are also higher for Glasgow compared with the UK cities of Liverpool, Manchester and Belfast.

b) Does rainfall have a negative effect on the mental health of populations?

A considerable amount of research has been undertaken in relation to seasonality and health outcomes – and in particular mental health outcomes. Research into Seasonal Affective Disorder (SAD)³¹⁶ is arguably the best known, although other outcomes such as depression, mood, schizophrenia and suicidal behaviour also feature prominently in the literature. Overall, however, the evidence has generally been mixed and often contradictory. Furthermore, despite the assumption that any observed seasonal variation in mental health outcomes relates directly to weather conditions, there is in fact little evidence that this is the case. Indeed, with regard to rainfall in particular, most evidence does not support any such link.

Research has been undertaken examining the link between rainfall (usually as one component of many climatic conditions) and outcomes of: suicide^{317,318,} and attempted suicide^{319,320}; depression^{321,322}, including specifically winter depression³²³; mood³²⁴; affective disorder³²⁵; hospital admissions for psychosis³²⁶; and panic attacks and anxiety³²⁷. Only two of the above studies suggested a meaningful association between rainfall and the outcome in question. Bulbena *et al.* demonstrated a correlation between levels of rainfall and panic attacks (the latter recorded by emergency attendance at a general hospital), but the correlation was negative – i.e. higher rainfall predicted fewer, not more, panic attacks. In contrast, Geltzer *et al.*'s study showed a strong correlation between mean and total levels of

precipitation and attempted suicide by carbon monoxide poisoning. However, this study was small (n=264), and is contradicted by findings from other studies e.g. Chiu and Ajdacic-Gross *et al.*.

There appears to be slightly more evidence of the link between health outcomes and other aspects of seasonality such as daylight hours, sunshine and atmospheric pressure. As stated, however, that evidence is often inconsistent. Furthermore, and with particular regard to mood and depression (key components of the hypothesis), Huibers *et al.*, in a study of a near 15,000-strong sample of the general population in the Netherlands, concluded that "contrary to popular belief, weather conditions and sad mood or depression do not seem to be associated". The authors back up this statement by referring to findings of a review by Watson in 2000³²⁸, and by stating that "strong empirical evidence for such an association is lacking".

Research has also demonstrated that there is no evidence of any effects of rainfall on heart disease outcomes³²⁹; however, an association has been shown between rain and levels of physical activity³³⁰.

Conclusion

The weight of research evidence does not support the hypothesis that there is an association between levels of rainfall and adverse effects on population health. Thus, although Scotland, and West Central Scotland/Glasgow in particular, experience higher levels of rainfall than the relevant comparator populations, this is unlikely to make any contribution to the high levels of Scottish excess mortality.

Assessment of evidence of causality

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

Bradford Hill's criteria	Assessment ^{xlix}	Comments
for causality		
Strength of association	N	
Temporality	N	
Consistency	N	
Specificity	N	The available evidence does not suggest that there
Biological gradient	N	is a negative impact of higher levels of rainfall.
Plausibility	Y	
Coherence	Y	
Experiment	N	
Analogy	Y	

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	Y	Rainfall has been consistently higher in Scotland over the relevant time period.
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	

^{xlix} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.
Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	Y	Rainfall has been consistently higher in Glasgow over the relevant
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	time period.

A6 Climate: vitamin D deficiency

Description of hypothesis

It has been proposed that higher mortality in Scotland (and Glasgow/West Central Scotland in particular) compared with the rest of the UK is in part the result of lower levels of sunshine, resulting in a deficiency of vitamin D among the population.

Rationale

Vitamin D deficiency has been linked to a number of morbidities³³¹, as well as to mortality, and in the northern hemisphere, northern, compared with southern, climates are characterised by relatively less sunlight (the means by which vitamin D is produced naturally by the skin).

Links to other hypotheses

Other climatic hypotheses (winter deaths, rainfall); diet; physical activity.

Evidence overview

Scotland receives less sunlight than England, and Glasgow receives less than both Liverpool and Manchester³³². Vitamin D levels are known to be lower in Scotland than in England³³³. Despite this, however, for a variety of reasons it seems unlikely that vitamin D deficiency plays a major role in the emergence of Scotland's and Glasgow's excess mortality.

First, although Glasgow does receive less sunlight than Liverpool and Manchester, the city receives slightly more than Belfast, another similar post-industrial city in the UK^{1,332}. Despite this, and as discussed elsewhere in this report, high levels of excess mortality (on a par with those shown in comparison with Liverpool and Manchester) have been shown for Glasgow relative to Belfast: 27% higher mortality for premature deaths, and 18% higher for deaths at all ages (after adjustment for differences in neighbourhood deprivation). Second, as has been shown, the excess levels of premature mortality in Scotland and Glasgow are driven by higher numbers of deaths from causes related to alcohol, drugs, suicide and violence, most of which are clearly not directly attributable to vitamin D deficiency. Third, a systematic review of the link between vitamin D deficiency and all-cause deaths was published in 2013, and although this suggested that there was an independent association between the two (despite quite limited and, in cases, problematic evidence^{li}), it related mainly to deaths among older age groups: as described elsewhere in this report, excess mortality in Glasgow and Scotland is highest among those of working age. Furthermore, a more recent (2014) systematic review which examined a wider set of health outcomes and broader set of studies suggested that low levels of vitamin D may in fact be a symptom of disease, rather

¹ Met Office data show that the average annual number of hours of sunshine for weather stations located close to Glasgow, Liverpool, Manchester and Belfast respectively between 1981 and 2010 was 1,265, 1,566, 1,373 and 1,247.

^{II} Very few studies were identified which examined premature mortality (deaths <65 years). Furthermore, a number of the studies did not adjust for potential confounders such as SES, increasing the risk of residual confounding in the relationship between vitamin D deficiency and mortality.

than a cause^{lii,334}. However, the evidence remains disputed, and randomised control trials (RCTs) are currently underway which will provide much higher quality evidence of the link between vitamin D status and population health^{335, liii}.

Conclusion

It appears unlikely that vitamin D deficiency makes a major contribution to Scottish excess mortality. As discussed, this relates principally to: the age profile most associated with excess mortality; the causes of death for which the greatest relative differences have been observed; and uncertainty around whether vitamin D deficiency is causal, or instead is just a marker of ill-health. The comparisons with Northern Ireland suggest that it may not be critical even if it is causal. However, that said, while we still await results from the randomised control trials, it would be wrong to *entirely* dismiss the role of vitamin D in contributing to Scotland's excess mortality. It is theoretically possible that it may make a partial contribution, for example in relation to higher mortality rates among the elderly. Furthermore, new evidence in this area is emerging constantly – for example, a recent paper showing a link between genetically low vitamin D with all-cause and cancer-related mortality³³⁶. However, on the basis of the existing evidence, it seems unlikely that it plays a major part in explaining excess mortality in Scotland and Glasgow.

^{III} The authors reviewed, and compared, both prospective cohort studies and randomised trials. The majority of the former showed strong associations between low vitamin D and a range of health outcomes including mortality; the latter did not. The authors concluded that "the discrepancy between observational and intervention studies suggests that low 25(OH)D is a marker of ill-health. Inflammatory processes involved in disease occurrence and clinical course would reduce 25(OH)D which would explain why low vitamin D status is reported in a wide range of disorders".

^{liii} Two RCTs are underway: one in the USA (see <u>http://www.vitalstudy.org/</u>), due to report in October 2017, and another in Finland (see <u>https://clinicaltrials.gov/ct2/show/NCT01463813</u>), due to report by the end of 2018.

Assessment of evidence of causality

Bradford Hill's criteria for causality	Assessment ^{liv}	Comments
Strength of	U	
association		
Temporality	U	
Consistency	U	
Specificity	U	There is uncertainty about whether vitamin D deficiency is a cause or effect of disease. Randomised
Biological gradient	U	trials to clarify this relationship are currently underway.
Plausibility	Y	
Coherence	Y	
Experiment	U	
Analogy	Y	

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	Y	Vitamin D deficiency is higher in Scotland. The trends in this are
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	unknown.

^{liv} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	Y	The meteorological data suggest that Glasgow has lower exposure to sunlight
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	than Liverpool and Manchester (but not Belfast), but no vitamin D data are available.

A7 Climate: winter deaths

Description of hypothesis

Disproportionately higher numbers of deaths in Scotland and Glasgow in winter contribute to the high levels of excess mortality.

Rationale

Winters are colder in Scotland than in England and are thus likely to result in higher mortality rates at that time of year than in England. The 2011 synthesis report described the recognised impact of winter deaths on overall levels of mortality in Scotland, including the fact that Glasgow, and deprived areas of Scotland more generally, were disproportionately affected.

Links to other hypotheses

Other climatic hypotheses: vitamin D deficiency, rainfall.

Evidence overview

Met Office data confirm that since the early 1980s Scotland has had, on average, colder winters than in the rest of the UK. The same is also true of Glasgow in comparison with Liverpool and Belfast, although not necessarily when compared with Manchester (the average maximum winter temperature was slightly lower in Glasgow between 1981 and 2001, but the average minimum temperature was slightly higher).

However, for similar age and cause of death related reasons to those presented in relation to the vitamin D hypothesis (see Appendix A6), higher winter-related mortality appears unlikely to be a major contributory factor to the high levels of excess Scottish mortality. While the overall excess is driven by higher mortality among those of working age (relating in large part to higher numbers of deaths from alcohol, drugs and suicide), analyses of additional winter deaths^{IV} in both Scotland and England show that the majority relate to people aged over 75 years, with the bulk of such deaths associated with respiratory and circulatory diseases^{337,338}. Furthermore, additional winter mortality figures in Glasgow over the period 2003/04 to 2006/07 were similar to those which took place in Liverpool (although higher than in Manchester)^{IVI}. The figures for Scotland and England are also similar^{IVII}.

^{1v} This is defined as the "difference between the number of deaths in the four 'winter' months (December to March) and the average of the numbers of deaths in the two four-month periods which precede winter (August to November) and follow winter (April to July)"³³⁷.

^{ivi} Published data for Scottish³³⁷ and English³³⁸ local authority areas for these years show that the average annual number of higher winter deaths in Glasgow, Liverpool and Manchester in 2011/12 were approximately 323, 250 and 168 respectively. Expressed as crude rates per 100,000 population the figures are 55.9, 57.2 and 37.8.

^{Ivii} On average between 1991/92 and 2010/11 there were 28,854 excess winter deaths in England compared with 2,899 in Scotland. Expressed as a crude rate per 100,000 population, the figures are 57.6 and 56.6. Clearly, however, a more sophisticated analytical approach (standardising for differences in the age structure of the Scottish and English populations) would be required to

Conclusion

The evidence suggests that it is highly unlikely that differences in the scale of winter deaths impacts on excess mortality in Scotland and Glasgow.

Assessment of evidence of causality

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

Bradford Hill's	Assessment ^{Iviii}	Comments
criteria for causality		
Strength of association	N/A	
Temporality	N/A	
Consistency	N/A	
Specificity	N/A	As the hypothesis relates to a particular cause of
Biological gradient	N/A	applied in general terms.
Plausibility	N/A	
Coherence	N/A	
Experiment	N/A	
Analogy	N/A	

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	Ν	Recent data show (crudely) similar numbers of winter deaths.
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	However, historical data are not readily available.

investigate this fully: however, from these crude calculations, it does not appear that excess winter deaths are higher in Scotland than they are in England.

^{Iviii} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	Ν	Recent data show (crudely) similar numbers
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	of winter deaths to Liverpool (although they were higher than Manchester). Historical data are not readily available.

A8 Culture of dependency

Description of hypothesis

It has been suggested that there is a greater 'culture of dependency' in Glasgow (and, by association, Scotland) compared with other parts of the UK and that this has a negative impact on health among sections of the city's/country's population.

Rationale

Such a 'dependency' on the welfare state, it is argued, diminishes personal responsibility and, via particular causal pathways related to health behaviours and choices, has a detrimental effect on health outcomes. It is closely linked to the notion of intergenerational worklessness, and increased levels of family breakdown and lone parenthood, as well as encouraging a lack of aspiration, thereby impeding social mobility³³⁹⁻³⁴¹.

Links to other hypotheses

As outlined above, this suggestion links to a number of other hypotheses such as individual values, social mobility, 'anomie' (i.e. in relation to 'underclass' and 'culture of poverty' theories), employment/labour market, health behaviours and more.

Evidence overview

There is very little evidence to support the notion of a 'dependency culture' in the UK generally³⁴². A number of studies have shown 'intergenerational worklessness' to be more myth than reality, and the links between welfare and family breakdown are similarly unproven^{342,343}. Furthermore, no evidence exists to suggest that, in any case, any such culture might be any more prevalent in Scotland. Comparisons of Glasgow, Liverpool and Manchester have shown that the percentages of the adult population in receipt of low income related social security benefits to be very similar^{29,30}, and the profiles of the cities in terms of social indicators such as lone parent households and teenage pregnancies are also very alike. As discussed elsewhere in this report, there is no evidence of lower levels of social mobility or a lack of aspiration among people in Glasgow compared with those in Liverpool and Manchester.

Brown *et al.* did show rates of Incapacity Benefit (IB) uptake to be higher in Glasgow than in Liverpool and Manchester between 2000 and 2008³⁴⁴: however, that arguably just reflects the higher levels of poor health in the city which are already well documented. Furthermore, the analyses of IB showed rates in Glasgow to be falling faster than in any other UK city (including Liverpool and Manchester): this was driven by relatively higher 'off-flow' and relatively lower 'on-flow' rates in Glasgow over the period^{lix}.

More generally, the timing of the emergence of the excess mortality coincides with a period of reduced welfare generosity and increased conditionality, which, if anything, would suggest that the policy direction over the last 35 years has been counterproductive³⁴⁵⁻³⁴⁷.

^{lix} The authors defined 'on-flow' as "those starting to claim IB" and 'off-flow' as "those whose claim has terminated".

Conclusion

The weight of evidence strongly suggests this is a very unlikely hypothesis.

Assessment of evidence of causality

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

Bradford Hill's criteria for	Assessment ^{ix}	Comments
causality		
Strength of association	N/A	
Temporality	N/A	
Consistency	N/A	There is little evidence for the existence of such a culture: this makes assessment using the Bradford Hill
Specificity	N/A	criteria impossible.
Biological gradient	N/A	The evidence base around this hypothesis is politicised and contested, but there is little evidence to support the
Plausibility	N/A	existence of such cultures or evidence to support such a culture as a cause of poverty or ill-health.
Coherence	N/A	
Experiment	N/A	
Analogy	N/A	

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	U	We have not examined data to compare measures at the Scotland
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	level. Given the lack of evidence for the theory, it is an unlikely contributor.

^{1x} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	N	For those markers of dependency that the
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	is little difference between the cities. However, these markers did increase at the time of the emergence of the excess.

A9 Culture of substance misuse

Description of hypothesis

Over and above any differences in the prevalence of substance misuse in the population, or the amount of substances used by individuals, it is suggested that differences in the *way* in which substances are used and understood (i.e. the cultural representations, meanings and use) in the population may result in the outcomes being worse in the Scottish populations. This is particularly in relation to alcohol, tobacco and illicit drugs such as heroin.

Rationale

The causal relationships between substances such as tobacco, alcohol and illegal drugs and a variety of health and social outcomes have been described extensively in the literature. There is also evidence, however, that it is not simply the prevalence of substance misuse or quantity of a substance that is (mis)used that determines outcomes, but also the way in which they are used and the surrounding context.

Links to other hypotheses

This hypothesis links to those on health behaviours (particularly alcohol and drugs) and to the other cultural hypotheses (individual values, social capital, anomie).

Evidence overview

The evidence base relating to differences in the cultures of substance misuse in Scottish populations compared with elsewhere is sparse.

Hepatitis C virus (HCV) infection is higher in Scotland (at 0.7% compared with 0.4% in England)^{348 349} and this may markedly increase the risk of mortality for injecting drug users - and itself may be a marker of different drug behaviours including sharing of needles and greater risk taking. Alcohol misuse (among drug misusers) carries a further additional risk³⁵⁰. Given that alcohol-related deaths³⁵¹ and HCV infection rates are higher in Scotland, this may exacerbate the risk of mortality in drug misusers in Scotland. More importantly, it may indicate that there are cultural factors in Scottish populations which may exacerbate the already negative impacts of drug misuse; however, there remains substantial uncertainty about the nature, extent and impact of any differences.

The alcohol culture in the UK compared with the rest of Europe is recognised to be different, but converging (towards the UK cultural attributes)³⁵². However, little difference in cultures have been detected between Scotland and England, and where this has been specifically studied, the similarities have been notable, rather than differences³⁵³. Negative alcohol use cultures, particularly in relation to alcohol use by predominantly young adults in the city centres at night, are not only associated with Glasgow³⁵⁴. However, there are some clues to differences: alcohol sales per capita are higher in Scotland and the volume of spirits (particularly vodka) used is on average higher. There is also a suggestion that alcohol is generally drunk in a more concentrated manner in Scotland (i.e. binging), and more often at home, than in England & Wales³⁵⁵.

Conclusion

This is a plausible hypothesis, but an assessment of its validity is hindered by a lack of comparable data and previous relevant research. There are some suggestions of differences in alcohol misuse culture (in relation to more concentrated drinking patterns and more home drinking).

Assessment of evidence of causality

Bradford Hill's	Assessment ^{lxi}	Comments
criteria for		
causality		
Strength of	U	
association		
Temporality	U	
Consistency	U	The evidence about the importance of substance misuse
Specificity	U	cultures in determining the mortality profile of a population is mixed and disputed. There are historical
Biological	U	examples (e.g. at the time of the industrial revolution and
gradient		in the early 1990s in the former USSR) where alcohol
Plausibility	Y	determining the mortality profile of the population.
Coherence	Y	
Experiment	U	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

^{ki} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	U	There are some, limited, differences in alcohol cultures between
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	Scotland and England & Wales, although these are not thought to be overly influential in explaining difference over time. We have not identified comparative evidence for other substance misuse cultures.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	U	We have not identified any comparative evidence looking at differences in
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	the cultures of substance misuse for Glasgow relative to other populations.

A10 Early years: family, gender relations and parenting differences

Description of hypothesis

It has been proposed that family breakdown, acrimony between partners and/or dysfunctional parenting are more prevalent in Scotland (and Glasgow), and ultimately have a negative influence on population health.

Rationale

There is considerable evidence linking adverse early years experiences to negative adult health and wellbeing related outcomes^{215, 356, 357}. On this basis it has been proposed as a potential explanation for Scottish excess mortality, linked to greater rates of family breakdown, acrimony between partners and poor parenting^{70,73,340,358}.

Links to other hypotheses

Inadequate measurement of deprivation; anomie; culture of dependency.

Evidence overview

The previous synthesis by McCartney *et al.* in 2011 found very little evidence to support the suggestion that such characteristics of parenting, upbringing or relationships were likely to be different in Scotland compared with other parts of the UK (highlighting, for example, similarities in rates of teenage pregnancies and lone parent households in Glasgow, Liverpool and Manchester (and between Scotland and England more generally)). Since then, a report published in 2013 considered in detail the issue of early years environment, based on analyses of a number of well-known UK longitudinal cohort studies. The study found virtually no relevant evidence of differences in early years' experiences between children born between 1946 and 2000. This was true of comparative analyses of cohort members in Scotland and England, and more specifically (where sample sizes allowed), in so-called 'city regions' of Glasgow and the Clyde Valley, Merseyside and Greater Manchester. The same negative finding emerged from subsequent more formal statistical testing of the hypothesis, which examined the impact of area of childhood residence on a range of childhood educational, behavioural and health outcomes (while controlling for a range of socioeconomic (e.g. father's social class) and other explanatory variables).

In addition, some limited questions on early years experiences (rating of happiness of childhood, and rating of childhood relationship with parents) were included within the three-city survey of Glasgow, Liverpool and Manchester (discussed elsewhere in this report): these also provided no evidence of more negative early years experiences on the part of the Scottish sample. An acknowledged, and potentially important, weakness of both those approaches, however, is that populations most at risk of experiencing such circumstances may not be represented in such population surveys. However, even within surveys that are subject to responder bias (such as the Scottish Health Survey (SHeS) and Health Survey for England (HSE)), an excess mortality in Scotland is still observed: this suggests that any residual impact of early years' experiences which might not have been captured within the samples because of responder bias is unlikely to be sufficient to explain the excess.

Further discussion of the potential link between childhood adversity, parent-child attachment style and Scotland's excess mortality is ongoing, based particularly on the knowledge that certain adverse adult health outcomes (e.g. substance misuse, suicide) are known to adversely affect attachment processes, and thus also act as risk factors for the future health of children – this, it is argued, is highly relevant because such negative outcomes are known to be more prevalent in Scotland/Glasgow compared with other parts of the UK³⁵⁹. That said, however, to date no empirical evidence has been unearthed to support the overall hypothesis that negative early years experiences (in relation to parenting, relationships and indeed attachment styles) are any more common in Scotland than other relevant parts of the UK.

Conclusion

All identified evidence to date does not support the notion that early years plays a major part in the Scottish excess mortality phenomenon. It is acknowledged, however, that considerable limitations are associated with those data. We did not identify any data to test the other aspects of this hypothesis, particularly in relation to gender relations.

Assessment of evidence of causality

Bradford Hill's criteria	Assessment ^{Ixii}	Comments
for causality		
Strength of	Y	
association		
Temporality	Y	
Consistency	Y	
Specificity	Y	Early years' experiences are well evidenced to be
Biological gradient	Y	causally related to subsequent health outcomes.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

^{bdi} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments	
Is the hypothesised causal factor worse in Scotland?	Ν	From the limited data available, there are few differences evident.	
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	although these are limited in their scope.	

Comparison	Assessment	Comments
Is the hypothesised causal factor	Ν	From the limited data
worse in Glasgow?		available, there are few
		differences evident.
Assuming a difference in exposure,	U	although these are limited
would this have occurred prior to		in their scene
the outcome?		in their scope.

A11 Educational attainment

Description of hypothesis

It has been proposed that levels of educational attainment are lower in Scotland (compared with England & Wales) and Glasgow (compared with similar cities such as Liverpool and Manchester) and that this may explain some of the excess levels of mortality observed in Scotland.

Rationale

The link between educational attainment and population health is well established. As a key 'social' determinant of health, education can impact on health status by means of interactions with other important determinants such as employment and income, and is thus often used in analyses of socioeconomic inequalities of health^{108,215-220}.

Links to other hypotheses

Inadequate measurement of deprivation.

Evidence overview

Education systems in Scotland and England

There have been very little comparative analyses of the education systems of Scotland and England (and elsewhere in the UK). Responding to this 'paucity of research', Raffe and colleagues assessed and summarised the similarities and differences between the systems in place in the four UK countries up to 1998 (the paper was published in 1999). Their commentary emphasised that Scotland's education system has been different to that of England for several centuries ("Scottish education had... begun to develop as a national system before the union with England in 1707, and it has remained more distinct ever since"), with its distinctiveness shaped by a range of historical and contemporary factors. These include: the incorporation of education within the separate control of the Scottish Office as early as the late 19th century; the development of different sets of educational qualifications; the selection system for secondary education (primarily comprehensive in Scotland, and more diverse in England), and different attitudes to education in Scotland, including (at the time of writing in the 1990s) a greater commitment to comprehensive education, more respect for teachers and head teachers, and greater confidence in local authorities to deliver education services. Looking forward from 1998, the authors suggested that devolution would further these differences – and this has arguably been shown to be the case given, for example, the recent development of 'academy' schools in England (but not Scotland).

Despite the above, however, Raffe *et al.* argued that the similarities of the systems in place in the UK may be more important than the differences. Furthermore the similarity of the 'social context' in Scotland and England (and in Wales and Northern Ireland) may also outweigh differences in the manner of delivering school education: this includes the similarities of the British labour market and the fact that the different education systems "interact with an economy which is integrated and organised at a UK level". This, however, should not be overstated, given that differences in the social context obviously exist across the UK and that: "education systems interact with their contexts; differences in systems reflect and sustain differences in social relations".

These latter points are highly relevant to the conclusions of an independent review of the Scottish school education system undertaken and published by the OECD in 2007: this also emphasised the importance of the social context (especially the socioeconomic context) in assessing performance in Scottish schools. The review highlighted a number of positive aspects of the Scottish system, stating that: "Scotland performs at a consistently very high standard in the Programme for International Student Assessment (PISA). Few countries can be said with confidence to outperform it in mathematics, reading and science. Scotland also has one of the most equitable school systems in the OECD". However, the review also outlined important "challenges", principally in relation to the stark inequalities in education-based outcomes, the "achievement gap that opens up about Primary 5 and continues to widen throughout the junior secondary years (S1 to S4)". Crucially, however, the report emphasised that this was primarily due to the socioeconomic context, rather than to the school system itself:

"Little of the variation in student achievement in Scotland is associated with the ways in which schools differ. Most of it is connected with how children differ. Who you are in Scotland is far more important than what school you attend... Socioeconomic status is the most important difference between individuals. Family cultural capital, lifestyle, and aspirations influence student outcomes through the nature of the cognitive and cultural demands of the curriculum, teacher values, the programme emphasis in schools, and peer effects".

This is further supported by results of the few other comparative studies of the UK nations: these found that attainment levels are largely due to differences in the social background of children rather than the school systems in place (and found that levels of attainment were higher in Scotland but with greater inequalities in attainment)³⁶⁰⁻³⁶².

International research in a number of countries has demonstrated that social context (especially socioeconomic context) is more influential than school characteristics in predicting educational outcomes^{133,221,222}. Indeed, as the sociologist Bernstein stated in the 1970s: "education cannot compensate for society", and sociologists generally have explained variation in educational attainment between social classes in terms of three forms of so-called 'capital' (all overlapping): economic capital, cultural capital (related to particular cultural practices such as reading, and associated with levels of parental education) and social capital. The latter is discussed in detail elsewhere in this report, but with regard to education, it relates to links between families, schools and communities. This is obviously not to say, however, that education systems and schools are unimportant. One recent review suggested that around 20% of variation in educational progress was explained by schools and other reviews have highlighted, for example, the importance of teacher quality in explaining some outcomes^{221,222}. However, the point here – with relevance to this excess mortality synthesis – is that most commentators agree that social and economic contexts

matter much more – and potentially unmeasured differences in the latter are potentially important for population health.

Routine indicators of educational attainment in Scotland and England

Scotland, and relevant parts of Scotland such as the West Central Scotland post-industrial conurbation and Glasgow City itself, tends to be characterised by a complex, polarised, educational profile: a relatively high percentage of its population has tertiary level educational qualifications, but a relatively high percentage also have no educational qualifications compared with elsewhere in the UK and Europe.

At the national level, the most recent (2011) census data show a higher percentage of the adult (age 16+ years) population in Scotland have no educational qualifications (27%) compared with England (22%). However, the percentages with tertiary level^{1xiii} qualifications were similar in both countries (approximately 27%)^{213,214}.

Regionally, comparisons of West Central Scotland (WCS) with other post-industrial regions across Europe confirmed the 'polarised' profile: a relatively high proportion of the adult population in WCS had low/no qualifications, and a relatively high proportion was educated to tertiary level.

At the city level 2011 Census data showed a higher percentage of Glasgow's adult population to have degree level qualifications compared with Liverpool (26% compared with 22%), but not Manchester (29%). However, Glasgow had the highest percentage of its adult population recorded as having no educational qualifications: 32% compared with 29% (Liverpool) and 23% (Manchester).

Data from the previous census (2001) showed a similar overall pattern to that seen in 2011 i.e. a higher percentage of Glasgow's population without any qualifications compared with Liverpool and Manchester^{lxiv}, and a higher percentage with degree level qualifications compared with Liverpool but not Manchester^{lxv}. In terms of those without any qualifications, more in-depth analyses of this indicator show notable variations in its distribution across the three cities, with a more unequal distribution in Glasgow. Grouping each city's population into ten equally-sized groups, Glasgow's highest rates of lack of qualifications (deciles 6-10) are much higher than the equivalent 'worst' groups in the English cities (Figure A11.1).

^{kill} Classed in the census data as Level 4 and above. For England this included: Degree (for example BA, BSc), Higher Degree (for example MA, PhD, PGCE), NVQ Level 4-5, HNC, HND, RSA Higher Diploma, BTEC Higher level, Foundation degree (NI), Professional qualifications (for example teaching, nursing, accountancy). For Scotland the categories were: Degree, Postgraduate qualifications, Masters, PhD, SVQ level 5 or equivalent; Professional qualifications (for example, teaching, nursing, accountancy); Other Higher Education qualifications not already mentioned (including foreign qualifications).

 ^{kiv}41% of 16-74 year-olds in Glasgow compared with 38% and 34% in Liverpool and Manchester respectively. Note that the analyses of 2001 Census data are based on 16-74 year-olds, whereas the 2001 Census data discussed earlier was based on the whole adult (16+ years) population.
^{kv} Glasgow: 18%; Liverpool: 15%; Manchester: 20%.

Figure A11.1^{lxvi}



Analysing these rates by the same income deprivation deciles used in the 2010 analyses of deprivation and mortality for Glasgow, Liverpool and Manchester (which showed very similar levels and distributions of deprivation in all three cities) again highlights differences between Glasgow and the two English cities, with considerably higher rates of 'no qualifications' associated with the five most deprived deciles in Glasgow. This is shown in Figure A11.2.

^{lxvi} Deciles in Figure A11.1 are derived from data calculated for comparably sized geographical units across the three cities: 'Lower Super Output Areas' (LSOAs) in Liverpool and Manchester, and merged 'datazones' in Glasgow²⁹. Data taken from 2001 Census.





Although these differences in educational attainment may be important (given the links between education and population health), it should be remembered that educational attainment has been controlled for in a number of national analyses of mortality in which a high level of excess has been demonstrated among Scottish populations. Thus its impact on excess mortality is likely to be small. For example, analyses of pooled Scottish Health Survey (SHeS) and Health Survey for England (HSE) data over the period 1994-2008 adjusted for differences in educational attainment (as well as a range of other socioeconomic, behavioural and biological risk factors) but still showed 29% higher mortality among Scottish respondents. However, the definition of educational attainment was limited, as it was only based on school leaving age. More recent analyses have shown that all-cause mortality in Scotland compared with England & Wales was 10.0% higher after adjustment for age, gender and Carstairs deprivation, but this was reduced to 8.7% higher when further adjusted for differences in no educational qualifications^{Ixvii,227}.

Note finally that a limitation of this assessment is that we have been unable to obtain long-term trend data in educational attainment for Scotland, England and the three cities^{lxviii}.

^{kvii} Excess after adjustment for age, gender, Carstairs area deprivation: 10,0% (95% Cls 9.45-10.63); after further adjustment for area-based measure of educational attainment (defined as no educational qualifications among those of working age, grouped into deciles): 8.7% (95% Cls (7.90-9.07).

^{kviii} Census forms suggest comparable data for 1971 were collected. However, these do not appear to be available (or only for sections of the population)³⁶³.

Conclusion

There are differences in aspects of educational attainment – specifically in relation to a lack of educational qualifications among sections of the population – between Scotland and England, and between Glasgow, Liverpool and Manchester. It is possible that this is relevant to the issue of excess mortality, especially that observed among the more deprived population – although statistical modelling analyses suggest any impact is small.

Assessment of evidence of causality

1)	General	assessment	of likelihood o	of causality in	terms of	F Bradford H	lill criteria
-	General	assessment	of likelihood (n causanty m	i termis ui	Diautoru	ini criteria.

Bradford Hill's criteria	Assessment ^{lxix}	Comments
for causality		
Strength of association	Y	
Temporality	Y	
Consistency	Y	
Specificity	Y	
Biological gradient	Y	Educational attainment is causally linked to subsequent health outcomes.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

^{kix} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments	
Is the hypothesised causal factor worse in Scotland? Assuming a difference in	Y	Scotland is worse than England & Wales in terms of the percentage of the	
exposure, would this have occurred prior to the outcome?		population with no educational qualifications however this accounts for only a small proportion of the excess mortality.	

ssment Comments
Glasgow is worse than Liverpool and Manchester, especially among the most deprived, in terms of the percentage of the population with no educational qualifications; however, it is likely that this accounts for only a small proportion of the excess mortality.
5

A12 Employment/labour market

Description of hypothesis

Differences in the nature of employment and the labour market in Scotland (compared with the rest of the UK) and Glasgow (compared with cities such as Liverpool and Manchester) may, through particular causal pathways related to, for example, income, safety, control, and 'precariousness' of employment, have a detrimental impact on the health status of sections of the Scottish population. These impacts would be over and above that already explained by higher unemployment rates experienced in Scotland in recent decades.

Rationale

Employment is one the key social determinants of health³⁶⁴. Rates of mortality and morbidity have been shown to increase among those experiencing unemployment, and to reduce among those in 'good' employment^{121,365-367}. Four pathways have been proposed through which employment is thought to impact on population health³⁶⁸: participation in/exclusion from the labour market (impacting on income levels, psychological distress, social participation/capital and more); wages/salaries (the major component of income); exposure to work environment hazards; and exposure to employment related 'psychosocial' risk factors e.g. from employment instability and insecurity, low control/high demand work, low self-esteem.

Links to other hypotheses

Inadequate measurement of deprivation; social mobility; deindustrialisation.

Evidence overview

Differences in overall unemployment rates are not relevant to this hypothesis, as these have been controlled for in analyses of Scottish excess mortality^{21,25}. Whether other aspects of employment/labour market differ between Scotland and England, and between Glasgow, Liverpool and Manchester, is unknown. To partly address this, a review of the existing evidence was undertaken in 2014/15 by Robertson et al.. The authors further elucidated and expanded existing theories linking employment to mortality, identifying seven characteristics of employment that were potentially relevant to the concept of excess mortality. These were: overall employment levels; job types; precarious employment; pay; physical work environment; psychosocial work environment; and employee representation. Literature searches relating to these characteristics were undertaken, focusing on Glasgow, Liverpool and Manchester. Little evidence was identified that suggested Glasgow had fared worse that the two English comparator cities in relation to these characteristics – although the amount of comparable evidence available was extremely small (with particular gaps in relation to precariousness, the physical and psychosocial work environments and employee representation). The review concluded that "there is currently insufficient evidence to link employment as a major explanatory factor for the excess in mortality in Glasgow and Scotland", but that "there are gaps in the research that could be explored further".

Previous analyses have confirmed the similarities of the cities in terms of their industrial employment history, and their current (principally service-sector) based economies.

Conclusion

The available evidence suggests that differences in employment and the labour market do not contribute to the high level of Scottish excess mortality. However, it is acknowledged that the available evidence is limited.

Assessment of evidence of causality

Bradford Hill's criteria	Assessment ^{Ixx}	Comments
for causality		
Strength of association	Y	
Temporality	Y	
Consistency	Y	
Specificity	Y	
Biological gradient	Y	Employment, particularly high quality jobs, are known to be protective for health.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments	
Is the hypothesised causal factor worse in Scotland?	U	There was insufficient data identified to be able	
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	England & Wales across a range of employment indicators.	

^{bx} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	U	There was insufficient data identified to be able to compare Glasgow with
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	Liverpool and Manchester across a range of employment indicators, although where data were available, few differences were identified.

A13 Ethnicity

Description of hypothesis

Lower ethnic diversity in Scotland and Glasgow, compared with the rest of the UK and particular UK cities, has contributed to the mortality phenomena.

Rationale

Ethnicity is a contested concept which encompasses a range of factors including genetic differences, race (itself contested), culture (which is often itself associated with religious differences) and context-specific factors such as discrimination and stigma against minority populations. As such, ethnicity can mean different things to different audiences and has different implications. In relation to the excess mortality, it is also possible that differential immigration rates of healthy migrants (principally highly educated individuals moving to the UK to work) have improved population health more in those areas with more migrants (and consequently more ethnic diversity). These factors therefore have the potential to explain part of the excess mortality phenomenon.

Links to other hypotheses

Ethnicity links to migration, social capital (including religion), the various cultural hypotheses and, more tangentially, sectarianism.

Evidence overview

In a Scottish context, ethnicity is recorded via the proxy of geography of identity (i.e. where individuals were born, or where their ancestors were born) alongside a proxy of skin colour. This leads to ethnicities being described as 'White Scottish', 'White Irish', 'Afro-Caribbean', 'South Asian', 'Chinese', etc³⁶⁹. These categories have been used to investigate the associations with mortality and morbidity in the Scottish context, but also internationally³⁷⁰.

In many contexts (e.g. the USA) being in one of the minority ethnic categories is associated with greater poverty and worse health, although this is dependent on the historical (and more recent) experience of ethnic minority 'community', including the reasons for their presence in a particular country and the welcome or discrimination they have encountered (e.g. there is a marked difference in the health of 'Latino' immigrants to the USA compared with that of 'Afro-Caribbeans', likely to be explained in part by the history of slavery in the latter and economic migration in the former). This is important since the mortality experience of ethnic minorities in Scotland is substantially better than 'White Scots'³⁷¹, and only some ethnic minorities disproportionately live in the more deprived areas (e.g. Black Africans) while others are less likely to do so (e.g. South Asians).³⁷² Compared with Scotland, a much greater proportion of the ethnic minority rates). As a result, ethnicity explains an additional proportion of the variation (inequality) in mortality than in Scotland over and above that due to deprivation (probably through the protective effect of being in an ethnic minority).

Scotland and Glasgow are less ethnically diverse than England overall, and than Manchester in particular. However, in the last ten years the diversity within Scotland and Glasgow has increased rapidly with increasing in-migration of both European and non-European populations.

In relation to analyses of excess mortality, ethnicity has not been included in national analyses which have examined the relationship between deprivation and mortality in Scotland and England & Wales. However, similarly high levels of excess mortality (including particularly high excess *premature* mortality) have been shown for Glasgow in comparison with cities with a similarly sized ethnic minority population (Liverpool^{bxxi}), with a larger ethnic minority population (Manchester^{bxxii}) and with a smaller ethnic minority population (Belfast^{bxxii})^{29,31}.

Conclusion

It is possible that the lack of ethnic diversity in Scotland and Glasgow, given the lower mortality of minorities in these populations and the time period over which it was lower, may have played some role in the excess mortality phenomena (particularly in relation to Manchester). However, the very similar ethnic diversity within Glasgow and Liverpool suggests that ethnicity may not be a key component in explaining the relevant outcomes.

^{bxi} The percentage of the (16+ years) population classed as member of ethnic minority group in 2011 Census was 10.2% in Glasgow 10.2% and 10.0% in Liverpool.

^{bxii} The percentage of the adult (16+ years) population classed as member of ethnic minority group in 2011 Census was 29.4% in Manchester.

^{Ixxiii} The percentage of the total population classed as member of ethnic minority group in the 2011 Census was Belfast was 3.5%.

Assessment of evidence of causality

Bradford Hill's	Assessment ^{lxxiv}	Comments
criteria for		
causality		
Strength of association	Y	
Temporality	Y	
Consistency	Y	
Specificity	Y	Ethnic diversity, especially when associated with
Biological gradient	Y	economic in-migration and the healthy migrant effect, is known to confer low mortality risks.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	Y	Scotland has lower ethnic diversity and this difference emerged prior
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	to the emergence of the excess.

^{bxiv} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

Comparison	Assessment	Comments	
Is the hypothesised causal factor worse in Glasgow?	Y	Glasgow's ethnic diversity is less than Manchester's,	
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y		

A14 Genetics

Description of hypothesis

It has been suggested that genetic differences in the Scottish populations might confer either a predisposition to negative health behaviours or a greater vulnerability to their impacts. More frequently, epigenetic changes (defined below) have been postulated as being important in explaining the Scottish mortality phenomena³⁷³⁻³⁷⁶.

Rationale

It is known that some genetic traits, particularly in relatively closed populations where 'genetic drift' can take place (i.e. where substantial genotype or phenotype differences emerge), are associated with differential incidence and mortality from particular conditions and with different tolerances of particular behaviours (e.g. alcohol use is rare among those who struggle to metabolise it as a result of genetic trait)³⁷⁷. If there was a higher prevalence of a relevant specific genetic trait, or a range of traits, in the Scottish populations then this may be part of the explanation for the excess.

Epigenetic changes refer to the heritable changes in gene expression caused by mechanisms other than changes in the underlying DNA sequence, caused by environmental exposures (defined broadly to include aspects such as poverty) and which can confer an increased risk of disease and death. It has been suggested that this exposure may have occurred either during an individual's life-course³⁷⁸, or may have occurred in the previous generation with the genetic changes being transferred to the subsequent generation³⁷⁹. Epigenetics therefore represents a *mechanism by which environmental exposures become embodied*, not an exposure in and of its own. The implication of this for excess mortality is that exposures during the life-course of individuals alive now, or among their parents, may have generated a subsequent increased risk (which may have been delayed by some time, or even inter-generationally).

Links to other hypotheses

The hypothesis links to that on migration, given the potential for migration differences to introduce genetic diversity (and for migration studies to examine the impact of genotype). The epigenetic component also links to hypotheses around early years, the lagged effects of deprivation, housing, urban change and the inadequate measurement of poverty and deprivation.

Evidence overview

Genetic differences

There are a small number of studies which have attempted to document genotypes in Scotland or Glasgow in comparison with other populations:

• A small sample of Caucasian individuals whose four grandparents were all born within 80km of their current residence within rural parts of the UK (a sampling method designed to maximise the identified differences in genotype, and which

appears to have substantially oversampled some areas such as Orkney) identified genotype clusters which displayed within-group similarities, and diversity between clusters³⁸⁰. The sampled individuals within West Central Scotland (WCS) were genotypically similar to those in Northern Ireland and Northern England. There was more divergence in the samples for rural Aberdeenshire and the Orkney Islands. The sample was not, however, representative of the population overall (excluding the non-White population, the urban population, any family with immigration within three generations) and was very small. It therefore described the maximal difference in genotype between the populations, and although there were differences between WCS and Southern England and the English Midlands, there were similarities to the Northern Ireland and Northern England populations (which have been shown to have lower mortality than WCS).

 A Europe-wide genotyping study sampled 700 people from Aberdeen and found that there were some subtle differences compared with the English samples, but that these were less profound than the differences between the English and Irish samples. 'Haplotype diversity' was lowest in the Irish and Scottish samples (in other words the Scottish sample was among the most homogeneous of the genotypes sampled)³⁸¹.

There is, therefore, an absence of evidence comparing the genotype of the Scottish and Glasgow populations with others (given the limited sampling approaches detailed above). However, even in these studies in which sampling focused on identifying divergent genotypes, the differences were small and tended to be shared with other populations (especially Northern Ireland and Northern England) which do not share the mortality profile of Scotland. It would therefore be expected that the differences in genotype between Glasgow and (especially) Liverpool (given the similar levels of ethnic diversity) are small.

Although not definitive, there are also other aspects of the excess mortality phenomena which make it less likely that genotype is a critical causal factor.

First, genotype-determined disease tends to lead to specific causes of illness and death³⁸². In Scotland, a wide range of causes of death for which there is an excess in Scotland have been identified, and many of these are clearly based on social rather than genetic processes (e.g. alcohol- and drug-related deaths, suicide and violence).

Second, the changing epidemiology of the causes of death implicated in the excess (in terms of the changing specific causes of death responsible and age groups affected (discussed in Part 4 of the report)) is over a timescale that would not have been expected if genetics were primarily responsible. Migration studies have shown that Scottish and Glaswegian migrants to other parts of the UK retain a higher risk of mortality after they leave, while mortality among people born in England but resident in Scotland is lower than the native population^{23,383,384}, which supports the idea that either genetics, early environmental exposures, or retained cultures play a role in the excess. However, the higher mortality of migrants from Scotland to elsewhere is less than that in the population which stayed, suggesting that genotype is not deterministic for the higher mortality.

Epigenetic differences

There are no data comparing epigenetic differences (for example, the extent of changes in gene expression, as approximated by DNA methylation^{bxxv}) in Scotland or Glasgow compared with elsewhere. However, the environmental exposures postulated to be important in causing this damage have been examined in the other hypotheses (and in this way epigenetic changes can be seen as a potential mechanism linking the social determinants of health to subsequent health outcomes).

It is important to recognise that were epigenetic differences identified, these would represent differences in a pathway linking earlier exposures (including exposures in the early years, socioeconomic circumstances, etc³⁸⁵) to health outcomes, not an independent exposure or cause³⁸⁶. Furthermore, the evidence for intergenerational transmission of genetic damage or vulnerability is very limited and has recently summarised as being 'unlikely'³⁸⁷. As a result, the suggestions that epigenetics are responsible for the mortality phenomena in Scotland are not evidenced.

Conclusion

The potential for there to be substantive genotype differences in the Scottish and Glasgow populations which explain the excess is supported by some migration studies but undermined by the similarities in genotype (in the limited studies available which have focused on finding diversity rather than similarities) to populations in Northern Ireland and Northern England; the variety of outcomes for which there are differences; and the changes in the important causes of the excess over time.

There are no comparative measures of epigenetic damage available, but any differences that were present would represent differences in exposures to factors covered by other hypotheses (as epigenetic differences represent a mechanism linking exposure to outcome rather than an independent exposure).

^{bxv} Methylation is the modification of a strand of DNA which impacts on the ability of a gene to be expressed (i.e. generally to be switched off, hence stopping the 'gene product' from being produced.

Assessment of evidence of causality

• 1			C 1.11 1.11 1.	c 1			
1)	General	assessment	of likelihood c	of causality in	terms of	Bradford	Hill criteria.

Bradford Hill's	Assessment ^{lxxvi}	Comments
criteria for		
causality		
Strength of	Y	
association		
Temporality	Y	
Consistency	Y	
Specificity	Y	There is some evidence, for some populations, of increased risk of disease, or increased susceptibility to
Biological gradient	Y	particular exposures (e.g. in relation to breast cancer ³⁸⁸).
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

^{bxvi} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments	
Is the hypothesised causal factor worse in Scotland? Assuming a difference in exposure, would this have occurred prior to the outcome?	U Y	It is unlikely that the wide range of causes that are responsible for the excess could all be due to genetic factors. The change in outcomes within a single generation make changes in the genetic make-up of the population unlikely, although pre-existing weaknesses may have become apparent as exposure changes. Out- migrants from the Scottish population retain a higher mortality risk which may reflect genetics, early years' experiences or retained cultures.	
Is the hypothesised causal factor worse in Glasgow? Assuming a difference in exposure, would this have occurred prior to the outcome? V V V V V V V V V V V V V	Comparison	Assessment	Comments
--	---	------------	--
cultures.	Is the hypothesised causal factor worse in Glasgow? Assuming a difference in exposure, would this have occurred prior to the outcome?	U Y	It is unlikely that the wide range of causes that are responsible for the excess could all be due to genetic factors. The change in outcomes within a single generation make changes in the genetic make-up of the population unlikely, although pre-existing weaknesses may have become apparent as exposure changes. Out- migrants from the Scottish population retain a higher mortality risk which may reflect genetics, early years' experiences or retained cultures.

A15 Health and social services

Description of hypothesis

It has been hypothesised that differences in the quantity, quality, distribution or use of health and social services may contribute to Scottish excess mortality.

Rationale

If differential advantages are gained from health and social services between the Scottish population and the comparators, this could explain aspects of the excess mortality through opportunities for preventative therapies and interventions being missed, or the treatment for acute or chronic conditions being suboptimal.

Links to other hypotheses

There are no clear links to any of the other hypotheses considered in the report.

Evidence overview

No evidence comparing the quality or availability of social services in Scotland or Glasgow with the comparator populations was identified.

There is evidence that the variation in health service use between Scotland and the rest of the UK is minimal³⁸⁹.

Scotland has higher spending per capita on health services than England & Wales (E&W) overall, and the greatest number of doctors and dentists per capita of all the constituent parts of the UK, probably reflecting political priorities and greater health needs³⁹⁰. Using a crude set of data for comparison, there was greater healthcare spending in Greater Glasgow than in Liverpool or Manchester³⁹¹. There has been some criticism that the productivity of the Scottish NHS is lower than in England, although this is disputed on the basis of the accuracy of the data and the higher needs and rurality in Scotland³⁹². Even if the Scottish NHS were less productive, this would not immediately be construed as a cause of the mortality phenomenon. Another possibility is that the way in which NHS resources are deployed in Scotland is different from elsewhere (e.g. that a greater proportion might be spent in hospital care rather than on primary care, rehabilitation or prevention). No evidence was identified which looks at this question, although the improvements in mortality amenable to healthcare have been faster in Scotland (at least since 1990) than elsewhere. The quality of healthcare services is also as high (or higher) in Scotland as in other areas of the UK, as measured by the Quality and Outcomes Framework (QOF) in primary care, suggesting that there is no evidence to suggest that the quality of care in Scotland is any worse^{390, 393}. There are a number of difficulties in comparing other aspects of the quality of NHS care in Scotland with elsewhere, and in assessing whether the higher spending in Scotland per capita simply reflects greater needs, making it difficult to draw further conclusions.

No evidence was identified to facilitate comparison of broader social service provision (either in terms of quantity or quality).

In general, the effective health policies which have been introduced differentially between Scotland and E&W have tended to be introduced first in Scotland (e.g. the ban on smoking in public places³⁹⁴ and the ban on quantity discounting of alcohol³⁹⁵). However, there are few examples of differing health policy prior to devolution in 1999.

The hypothesis that health and social services in Scotland are either poorer quality, less accessible or less frequently demanded than elsewhere and that this is an important causal factor in generating the excess mortality phenomenon is not consistent with how we understand the social causes of alcohol-related deaths, drug-related deaths, suicide and violent deaths – all major contributors to the higher mortality.

Conclusion

Assessment of this particular hypothesis is hindered by an absence of evidence (in particular in relation to any potential differences in relevant social services (e.g. addictions)). The weight of available evidence, however, does not support the hypothesis.

Assessment of evidence of causality

Bradford Hill's criteria	Assessment ^{lxxvii}	Comments
for causality		
Strength of association	Y	
Temporality	Y	
Consistency	Y	
Specificity	Y	
Biological gradient	Y	Various aspects of healthcare, and healthcare systems, are evidenced to improve health.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

^{bovii} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	U	From the limited available evidence, which looks at a very limited range of
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	measures, there are no differences evident. There is a particular gap in relation to social services.

Comparison	Assessment	Comments
Is the hypothesised causal factor	U	No data were identified to
worse in Glasgow?		be able to compare
		Glasgow with elsewhere.
Assuming a difference in exposure,	U	0
would this have occurred prior to		
the outcome?		

A16 Health behaviours – alcohol

Description of hypothesis

The hypothesis is that the people of Glasgow, and Scotland, consume more alcohol than comparable populations (and more after adjusting for deprivation), or that alcohol is consumed differently (in terms of the patterning of drinking within and between individuals, or the type of alcohol consumed).

It is important to note that, as with other hypotheses, a similar consumption of alcohol in Scotland and Glasgow may still be a causal factor in the mortality phenomena if it interacts with other factors to generate disproportionately worse impacts.

Rationale

Alcohol consumption is a recognised cause of increased mortality rates. Unlike many other hypothesised causal factors, the proportion of deaths for which alcohol is implicated is well described (with 100% of alcohol-related deaths having alcohol consumption implicated and varying proportions of alcohol-attributable deaths (e.g. heart disease) having alcohol consumption implicated). Given that alcohol-related mortality is a key cause of deaths responsible for the Scottish excess mortality phenomena, the causes of the higher alcohol consumption are therefore important.

Links to other hypotheses

The alcohol hypothesis links very closely to: different culture of substance misuse; individual values (e.g. hedonism); anomie; and to other health behaviours (smoking, diet, illicit drugs, physical activity) because of the clustering of negative health behaviours³⁹⁶.

Evidence overview

Alcohol as a proximal cause

Alcohol-related harms (i.e. those which are directly due to exposure to alcohol), including alcohol-related mortality and morbidity, are substantially higher in Glasgow and Scotland than in comparable populations and are responsible for a substantial proportion of the excess mortality^{29,25}, inequalities in mortality, and the higher mortality compared with the rest of Europe. Alcohol-related mortality was low in Scotland relative to the rest of Europe from 1950 until the 1980s, but this increased rapidly during the 1990s such that it became the highest in Europe after marked increases in absolute terms. The definition of alcohol-related health harms are known to underestimate the total contribution of alcohol because of the indirect, and partially attributable, causes of mortality and morbidity which are not counted in this definition³⁹⁷. The impact of alcohol on social harms, which may also contribute to health outcomes through a myriad of pathways not counted even through estimated alcohol-attributable health harms, may mean that the prominence of alcohol in the explanation of the mortality phenomena may be underestimated.

Alcohol-related mortality was 2.3 times higher in Glasgow than in Liverpool and Manchester in 2003-7, and was 72% higher in Scotland than England & Wales before adjustment for

Carstairs deprivation and 54% higher after adjustment (representing the excess) in 2011. Alcohol is particularly important in explaining all of the mortality phenomena as they developed during the 1990s, and particularly for younger adults where alcohol-related causes constitute a much greater proportion of all deaths than in later life. Alcohol-related mortality started to decline in Glasgow and Scotland since around 2003 (particularly for men), as did inequalities in alcohol-related mortality, bringing the Scottish rates closer to the European median. As noted above, alcohol is also likely to play an important role in the broader group of alcohol-attributable deaths which includes heart disease, cancer and stroke (i.e. many of the causes of death which explain the excess mortality phenomena among older adults and a larger proportion of all deaths).

In both absolute and relative terms, Scotland consumes more spirits per adult than in England & Wales and compared with Northern England. It is not clear whether the consumption of a unit of spirits is more harmful than the consumption of a unit of another alcohol type, although it is clear that high strength alcohol types (particularly spirits and white ciders) are the preferred drink types of those who are the most harmful drinkers³⁹⁸. The higher consumption of spirits in Scotland therefore may simply represent the higher proportion of harmful drinkers in the population.

It is also possible that alcohol is drunk in a different way, or as part of a culture which has a compounding impact, in Scottish populations. This is discussed in more detail in relation to the *culture of substance misuse* hypothesis.

Determinants of alcohol harms

The causes of the rise and decline in alcohol-related harms (including mortality) in Scotland have been discussed in detail elsewhere^{355,399}.

Alcohol-related harms in Scotland followed the rapid and substantial rise in unemployment and income inequality during the 1980s and 1990s following the introduction of the neoliberal economic and social policies of the governments from 1979 onwards^{119, 346,} ³⁴⁷. These trends in the 'upstream' factors occurred at the same time as alcohol affordability and availability increased (due to increased income, reduced alcohol taxation and liberalisation of alcohol sales regulations), and this has been associated with increased alcohol misuse⁴⁰⁰. From 2003 onwards, real incomes in the least affluent groups declined leading to a divergence in the affordability of alcohol across income groups. For those at greatest risk (i.e. those living in the most deprived areas, exposed to unemployment or poverty) this was associated with a decline in alcohol harms (mortality, morbidity and crime) and because of the marked inequalities in alcohol harms, this trend was large enough to reduce the mean alcohol-related mortality rate in Scotland by approximately a third by 2013^{351,355,399}. Some of the differences between Scotland and E&W in the rise and fall in alcohol-related mortality from 1991 to 2011 can be explained by differences in income trends (probably through the consequent changes in the affordability of alcohol) interacting with the different patterning of alcohol-related mortality by deprivation and the higher prevalence of deprivation in Scotland.

However, the population of Liverpool and Manchester have very similar socioeconomic profiles to Glasgow and it seems less likely that this explains the differences between the cities (although income trends are not available at this level to test this).

It is plausible that some of the steeper decline in harms in Scotland since 2007 may be due to implementation of the new alcohol strategy by the Scottish Government (which includes restrictions on alcohol availability, investment in treatment services and funding for alcohol brief interventions).

There is also some evidence^{58,401}, although it is contested⁴⁰², that there may be a cohort effect (relating to exposure to negative socioeconomic conditions during the 1980s) operating in Scotland that may partially explain both the rise during the 1990s and the decline during the late 2000s.

It is likely that the trends in alcohol harms in Scotland are due to changes in both the upstream and alcohol-specific determinants of health, and that it is the interaction of general and specific factors which is associated with the catastrophic rise in harm. The greater vulnerability to the neoliberal politics of the 1980s, and the interaction with income trends and alcohol-specific changes (i.e. affordability and regulation which occurred across the UK), are the most likely explanations for the differing trends in alcohol-related mortality in Scotland and Glasgow compared with elsewhere^{355,399}.

Conclusion

The consumption of alcohol and related harms are higher in Scotland and Glasgow than in comparable populations. Alcohol is an important proximal explanation for all of the mortality phenomena but has to be contextualised by the determinants of alcohol-related harm. Increased unemployment, deindustrialisation and rising inequality and poverty which resulted from the shift to neoliberal politics during the 1980s led to increased alcohol-related harm, particularly in the context of increased alcohol affordability and reduced regulation of alcohol sales.

Assessment of evidence of causality

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

Bradford Hill's criteria for causality	Assessment ^{lxxviii}	Comments
Strength of association	Y	
Temporality	Y	
Consistency	Y	
Specificity	Y	
Biological gradient	Y	determinant of health.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	Y	Alcohol-related deaths are higher in Scotland and increased at the time of
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	the excess.

^{baxviii} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	Y	Alcohol-related deaths are higher in Glasgow compared with elsewhere
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	and increased at the time of the excess.

A17 Health behaviours: diet

Description of hypothesis

It is proposed that the excess levels of Scottish mortality are explained in large part by a worse dietary profile among the Scottish population.

Rationale

It is well known that good nutrition throughout the life-course impacts positively on health status. Poor diet and nutrition is linked with a greater risk of a considerable number of conditions including coronary heart disease, stroke, various cancers, obesity, type 2 diabetes, high blood pressure and more^{403,404}.

Links to other hypotheses

As a proximal cause of disease, diet is linked directly to more 'downstream' hypotheses, in particular to other health behaviours, and the 'individual values' hypothesis. It is also influenced by the cultural hypotheses.

Evidence overview

A review of the evidence relating to differences in diet between Scotland and England, and between Glasgow, Liverpool and Manchester, was undertaken in 2014-15 by Wrieden *et al.*. This included a 'rapid review' of the existing published literature, alongside new analyses of diet in Scotland and England from the Expenditure and Food Survey/Living Costs & Food Survey (EFS/LCFS) for the period 2001-12.

<u>At the national level</u> (Scotland versus England), comparisons of diet in the published literature were very limited, with results often inconsistent. Some evidence did emerge, both among children and adults, of lower intake of fruit and vegetables, vitamins, and fibre in Scotland compared with England. However, interpretation of these results was often constrained by small sample sizes for Scotland and limited adjustment for confounding factors.

Analyses of EFS/LCFS data (based on food purchases, and controlling for food waste) suggested that compared with England as a whole, the Scottish population had a lower intake of fruit and vegetables, oily fish, fibre, folate and vitamins A, C and D, and also had a higher intake of red meat, processed meat, whole milk, butter, savoury snacks, confectionary, soft drinks, saturated fat, non-modifiable extrinsic sugars (NMES; added sugars and sugar in fruit juice), sodium and alcohol. Stratifying the analyses by equivalised income suggested that lower and higher intakes respectively of fruit & vegetables and saturated fat were true of all income groups, while a lower intake of fibre and higher consumption of processed meats were more prevalent in comparison of lower, rather than higher, income groups.

However, <u>at the regional level</u> (Scotland versus North West England) less clear, more inconsistent, differences were observed. <u>At the city level</u>, no relevant comparisons of diet were found in the published literature – although it has been noted previously that in the

early to mid-2000s survey data indicated that there were no meaningful differences in levels of consumption of fruit and vegetables between Glasgow, Liverpool and Manchester. No other data were available to analyse in the study by Wrieden *et al.*.

It should be noted that analyses of the Scottish Health Survey (SHeS) and the Health Survey for England (HSE) (SHeS data from 1995, 1998 and 2003, HSE data from 1994-2008) suggested that higher mortality among the Scottish population persisted after controlling for differences in diet: all-cause mortality was approximately 40% higher after adjustment for age and gender only (Hazard Ratio (HR) 1.40 (95% CI 1.34 – 1.47), which was attenuated to 31% higher (HR 1.31 (95% CI 1.22, 1.44)) after adjustment for diet. Mortality for cardiovascular disease and cancer was approximately 51% and 39% higher respectively among Scottish respondents after adjustment for diet. It should also be noted, however, that in these analyses 'diet' was measured only in terms of consumption of fruit and vegetables, which is clearly an extremely limited definition.

Conclusion

It is likely that differences in diet play some part in the higher mortality experienced in Scotland compared with England. However, the determinants of the differences in diet in Scotland compared with England, and how those fit into a broader causal chain, are not well understood.

It is also less clear if any such dietary differences apply to Glasgow in comparison with cities such as Liverpool and Manchester.

Assessment of evidence of causality

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

Bradford Hill's criteria for	Assessment ^{Ixxix}	Comments
causality		
Strength of association	Y	
Temporality	Y	
Consistency	Y	
Specificity	Y	
Biological gradient	Y	Diet is a known determinant of health.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	Y	The limited data available suggest that some aspects of the Scottish diet are
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	worse than that in England & Wales. Few trend data were available.

^{lxxix} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	Ν	The very limited data available suggest no differences in diet, but
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	this was for a very limited range of measures.

A18 Health behaviours: drug misuse

Description of hypothesis

The prevalence of drug misuse in Scotland and Glasgow is higher than elsewhere in the UK (including in comparable cities like Liverpool and Manchester), and this contributes to the high levels of excess Scottish mortality.

Rationale

As is the case with other health behaviour related hypotheses, the rationale for this hypothesis is obvious: there are well documented links between drug misuse and mortality⁴⁰⁵. However, given that most health behaviours are socially patterned and the excess mortality is defined as higher mortality over and above that explained by socioeconomic factors, the assumption is that drug misuse contributes to the excess independently of deprivation and socioeconomic conditions.

Similar to the alcohol misuse hypothesis, mortality due to exposure to illicit drugs can be clearly defined for those causes which are completely attributable i.e. drug-related deaths. However, the potential contribution of illicit drugs use to a wider group of causes of deaths means that the use of drug-related deaths data alone is likely to underestimate the total contribution of this exposure⁴⁰⁶.

Links to other hypotheses

There are similarities to the other health behaviour hypotheses, and links to a 'culture of substance misuse', 'anomie', and individual values.

Evidence overview

National analyses of deprivation and mortality in Scotland compared with England & Wales showed that after adjustment for differences in area-based deprivation, drug-related poisonings^{bxxx}, were almost 250% higher in 2011 (% excess: 248.4 (95% CIs: 239.6-257.4)). This has increased markedly in the last 30 years: in 1981 no was excess recorded, in 1991 the figure was 11%, and in 2001 it was 84%. It is estimated that for the period 2010-12 this equated to almost 300 extra premature (age <65 years) deaths per year in Scotland (or 16% of the total number of excess premature deaths)^{bxxxi}.

Similar analyses for Glasgow relative to Liverpool and Manchester produced virtually identical results. Over the period 2003-2007, drug-related poisonings were also 248% higher after adjustment for differences in neighbourhood levels of income deprivation. Analysis suggested that drugs deaths made up 17% of the total number of excess premature deaths experienced over the period in Glasgow relative to Liverpool and Manchester.

^{box} This is the drugs-related grouping of ICD (international Classification of Disease) codes which, for analytical purposes, is deemed to be most comparable between Scotland and England⁴⁰⁷.

^{bxxi} Between 2010-2012, there were an estimated 299 excess deaths at all ages from drug-related poisoning, accounting for 6% of the total number of excess deaths. Of these, 290 were for people aged <65 years (16% of all excess deaths in that age group).

In a separate study Bloor and colleagues suggested that "the higher prevalence of problem drug use in Scotland than in England accounts for a third of Scotland's excess mortality over England"⁴⁰⁸. The accuracy of this claim has been questioned⁴⁰⁹, particularly as it is based on the analysis of a relatively small cohort of drug users (n=1,033). What is clear, however, is that drug misuse contributes considerably and directly to the overall levels of excess mortality (and especially premature mortality) in Scotland and Glasgow. As with other health behaviours, however, there is a need to understand why that is the case i.e. the 'causes of the causes'^{3,245-247}.

Although numbers of drug-related deaths in Scotland have increased steadily in the last 20 years, it has been suggested that there may be a 'cohort' effect, and that numbers may start to decrease in the near future. This is supported by the fact that the median age of a drug-related death has also increased steadily, from 28 in 1996 to 40 in 2014. More positive news is also provided by recent Scottish Schools Adolescent Lifestyle and Substance Use Surveys (SALSUS) which have shown that reported drug use among children (13 and 15-year-olds) has decreased notably since 1998^{lxxxii,410}.

Conclusion

Drug misuse clearly makes a substantial contribution to excess mortality in both Glasgow and Scotland. However, as discussed elsewhere in this report, we have to place this in the context of what is known regarding causal pathways, and in this sense drug misuse can be seen as more of an outcome than a determinant. The underlying causes of high rates of drug misuse in Scotland are what are important to understand.

^{boxii} For example, the percentage of 15 year olds reporting using illicit drugs in the previous month (before the survey) fell from 24% in 1998 to 9% in 2013. The equivalent figures for 13 year olds were 8% in 1998 to 2% in 2013.

Assessment of evidence of causality

Bradford Hill's	Assessment ^{Ixxxiii}	Comments
criteria for causality		
Strength of association	Y	
Temporality	Y	
Consistency	Y	
Specificity	Y	Within the context of current drugs policy, illicit
Biological gradient	Y	drugs use is associated with negative health outcomes.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland? Assuming a difference in exposure, would this have occurred prior to the outcome?	Y Y	Drug-related deaths in Scotland are higher, and there is some evidence to suggest that drug misuse in Scotland currently accounts for a greater number of deaths than this narrow definition accounts for.

^{boxiii} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	Y	Drug-related deaths in Glasgow are higher, and there is some evidence to
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	suggest that drug misuse in Scotland currently accounts for a greater number of deaths than this narrow definition accounts for.

A19 Health behaviours: physical activity

Description of hypothesis

The hypothesis is that the people of Glasgow, and Scotland, do less physical exercise than comparable populations and that this leads to relatively worse health outcomes.

Rationale

Physical activity is recognised as an important causal factor in determining a wide range of health outcomes⁴¹¹⁻⁴¹³. If physical activity is less prevalent in Scotland and Glasgow than in other populations then it would be an important negative influence on both physical and mental health outcomes.

Links to other hypotheses

Physical activity is determined by a range of other factors including the characteristics of the prevalent employment (e.g. are the available jobs desk-based or reliant on physical effort), urban planning and transport systems (particularly in respect to the prevalence of active travel), the weather, the availability and accessibility of leisure services, and the prevalent culture (e.g. in respect to sport, travel and leisure activities)⁴¹⁴⁻⁴¹⁶.

Evidence overview

Assessing and comparing physical activity levels in the relevant populations is complicated by measurement bias (all population data rely on retrospective self-reported estimates) as well as the more common sampling biases associated with the national surveys. A further issue is the narrow range of survey questions which are directly comparable between the populations of interest, meaning that a less nuanced measure relating to the proportion meeting the national guidelines is the only available option for comparison.

Data from the 2008 Scottish Health Survey and the Health Survey for England suggest that physical activity levels are identical in Scotland and England (measured as the proportion of those who had participated in \geq 30 minutes of at least moderate activity on \geq 5 days a week, counting bursts of activity of at least 10 minutes) at 45% for men and 33% for women⁴¹⁷. Data from earlier surveys have shown slight differences whereby the proportion meeting the guidelines was higher in Scotland in 2003, the same in 1998, and lower in 1995. Comparable data are not reported for Glasgow, Liverpool and Manchester.

Gray used similar health survey data from the early 2000s to compare physical activity rates between Greater Glasgow and a number of UK regions⁴¹⁸. After adjustment for socioeconomic status, there were no meaningful differences between levels of physical activity in Greater Glasgow, Greater Manchester and Cheshire & Merseyside (which includes Liverpool) among men; among women, physical activity levels were higher in Greater Glasgow compared with Greater Manchester (but not compared with Cheshire & Merseyside).

Given all the above, it is no surprise that national analyses of the same health survey data (in this case Scottish Health Survey (SHeS) data from 1995, 1998 and 2003, and the Health

Survey for England (HSE) data from 1994-2008) showed that higher mortality among the Scottish population persisted (actually increasing slightly) after adjusting for differences in physical activity.

Conclusion

There are severe limitations in the data available to make meaningful and comprehensive comparisons between Scotland and England, and between Glasgow, Liverpool and Manchester, in terms of levels of physical activity undertaken by the populations. The weight of the available evidence, however, suggests there are few, if any differences, and thus that it is unlikely that physical activity plays a major role in explaining aspects of Scottish excess mortality.

Assessment of evidence of causality

Bradford Hill's criteria	Assessment ^{lxxxiv}	Comments
for causality		
Strength of association	Y	
Temporality	Y	
Consistency	Y	
Specificity	Y	<u>-</u> ,
Biological gradient	Y	physical activity is causally protective to health.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

^{boxiv} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	N	Physical activity levels, from the limited data available, seem to be
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	similar in Scotland and the comparator areas.

Comparison	Assessment	Comments
Is the hypothesised causal factor	N	Physical activity levels
worse in Glasgow?		from the limited data
		available, seem to be
Assuming a difference in exposure,	U	similar in Glasgow and the
would this have occurred prior to		comparator areas.
the outcome?		

A20 Health behaviours: smoking

Description of hypothesis

Smoking prevalence in Scotland/Glasgow is higher than elsewhere in the UK (including in comparable cities like Liverpool and Manchester), and that this contributes to the high levels of excess Scottish mortality.

Rationale

The rationale is obvious, given the well documented links between smoking and mortality^{419,420}. However, given that most health behaviours are socially patterned and the excess mortality is defined as higher mortality over and above that explained by socioeconomic factors, the assumption is that smoking contributes to the excess independently of deprivation and socioeconomic conditions.

Links to other hypotheses

See other health behaviours. In addition this (alongside alcohol and drug misuse) links to the 'culture of substance misuse' hypothesis.

Evidence overview

a) National analyses

At the national level, survey data suggest adult smoking prevalence rates have been consistently higher in Scotland compared with England over the past four decades. For example in the early to mid-1970s, the figures were approximately 48% in Scotland and 46% in England; in 2010-12, rates averaged around 23% and 19% respectively. Since the early 1970s the difference between the two countries has been, on average, approximately 4 percentage points.

Analysis of recent (2011/13) data by social class (here, the National Statistics Socio-Economic Classification (NS-SEC)) showed, for males, little difference between Scotland and England for most socioeconomic groups with the exception of those employed in 'semi-routine occupations': in that group smoking rates were higher in Scotland (37%) compared with England (32%) (Figure A20.1). Rates were also higher among female Scots in this group (33% versus 26% in England), but was lower in Scotland (21% compared with 29% in England) among those of "lower supervisory and technical occupations" (Figure A20.2). However, these data are limited by the fact they do not include the "never worked & unemployed" category.

Figure A20.1



Figure A20.2



Comparison of pooled national health survey data for Scotland and England (from the Scottish Health Surveys of 1995, 1998 and 2003, and the annual Health Survey for England

from 1994-2008) showed a greater difference in prevalence rates between Scotland (33%) and England (25%) over the period. This is likely to relate in part to differences in the age definition of adults in two of the three Scottish surveys^{bxxxv}, and also because the English health survey data were shown to be rather less representative of the total English population than was the case with the Scottish data in relation to all Scotland. However, despite the large difference in smoking rates, a large excess level of mortality (29%) was shown for Scottish respondents *after* adjustment for smoking status (as well as after adjustment for socioeconomic status and a range of other health behavioural, and biological, risk factors). Adjustment for smoking status in the analysis did attenuate the excess (the age/sex adjusted hazard ratio (HR) decreased from 1.40 to 1.31 (the HR in the fully-adjusted model was 1.29)), but a large excess remained. Furthermore, despite adjusting for smoking status, an even larger excess was observed for deaths from smoking-related cancers: 62% higher mortality in the fully-adjusted model.

b) City-level analyses

Estimates of smoking prevalence from national surveys show little difference in rates between Glasgow, Liverpool and Manchester. Smoking data from 2003 were reported alongside the results of the comparative analyses of deprivation and mortality in Glasgow, Liverpool and Manchester: adult smoking rates were around 34-35% in all three cities. A question on smoking was included within the 2011 three-city survey, and the results compared with contemporary data from other sources: those data are combined within Figure A20.3, but again suggest little difference in prevalence rates between the cities (albeit that national sources show a slightly higher rate in Glasgow compared with Liverpool at that time: however, those figures are based on different age groups, making precise estimation of the difference difficult). The three-city survey data showed few differences when analysed by age; stratification by social class, however, suggested relatively lower prevalence rates in Glasgow among those of high SES, and relatively higher rates among those of low SES, although the small sample sizes which result from such stratification again make it difficult to draw firm conclusions from those data (Figure A20.4).

Despite these similarities, lung cancer mortality in 2003-07 was 27% higher in Glasgow compared with Liverpool and Manchester after adjustment for area-based deprivation.

Of course, as with all health behaviours, it is important to consider smoking alongside, and not in isolation from, other contextual factors – what Marmot and others have referred to as 'the causes of the causes'^{3,245-247}, i.e. the upstream, rather than downstream, drivers of poor health and poor health behaviours.

^{boxv} Adults were defined as 16+ years in all the English surveys. However, the 1995 Scottish Health Survey (SHeS) included adults aged 16-64 years only, while the age group included in the 1998 survey was 16-74 years. All adults aged 16 years and over were included in the 2003 SHeS.





Figure A20.4 (Source: Walsh et al. 2013).



Conclusion

Higher rates of smoking in Scotland as a whole (and in particular among those of lower SES) compared with England & Wales contributes, but only in part, to Scottish excess mortality. In national analyses, the inclusion of smoking within statistical modelling analyses lowers the level of excess mortality – but a high excess still remains. Furthermore, as with all health behaviours, although smoking is the proximal cause of morbidity and mortality, there is the need to understand underlying factors (the 'causes of the causes'). Finally, it seems unlikely that smoking contributes in any meaningful way to the high excess level of mortality recorded in Glasgow compared with the English comparator cities.

Assessment of evidence of causality

Bradford Hill's criteria	Assessment ^{Ixxxvi}	Comments
for causality		
Strength of association	Y	
Temporality	Y	
Consistency	Y	
Specificity	Y	
Biological gradient	Y	There is high quality evidence which shows the negative health impacts of smoking.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

^{boxvi} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland? Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	Smoking prevalence is higher in Scotland, and has been for at least 40 years. However, the modelled impact of this higher prevalence explains only a small part of the excess.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	N	Smoking prevalence in Glasgow is very similar to that in Liverpool and
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	Manchester.

A21 Housing quality and provision

Description of hypothesis

It has been proposed that Scotland's and Glasgow's high levels of excess mortality may have been influenced by differences (particularly historical differences) in aspects of housing between Scotland and the rest of the UK.

Rationale

There are two, overlapping, components of this hypothesis: first, that housing in Scotland compared with England & Wales (and more specifically in Glasgow compared with Liverpool and Manchester) has been of poorer quality since the latter half of the 20th century (the period which includes the emergence of Scottish excess mortality); and second, that social housing allocation policies in Scotland (and particularly Glasgow) have disadvantaged sections of the population in a manner not experienced by similar populations in England.

Both these components suggest that sections of the Scottish population have been exposed to a more negative housing environment: this is relevant to the issue of excess mortality given the well-known links between housing and health^{421,422}. Furthermore, the availability of housing may have had an influence on the social capital of communities through the impact this may have on where people live in relation to their sources of social support.

Links to other hypotheses

Inadequate measurement of poverty and deprivation; lagged effects of deprivation; social capital; nature and scale of urban change; quality of physical environment; political influences and vulnerability.

Evidence overview

A review of the evidence of differences in housing quality and provision for Scotland, England and the three cities of Glasgow, Liverpool and Manchester was published in 2016⁴²³. This showed (as have other analyses) that over the period 1939-2011, Scotland (and Glasgow) was markedly more disadvantaged compared with England (and Liverpool/Manchester) in levels of overcrowding. Related to this was a similar disadvantage in terms of the smaller size of Scottish houses compared with those in England. Other differences in housing stock (e.g. a relatively greater numbers of flats (rather than houses)) were identified, although it was also acknowledged that there is no evidence of negative impacts on health for such differences.

As other analyses have also highlighted, the report also stressed the higher rates of local authority (and latterly housing association) owned accommodation in Scotland and Glasgow, and, importantly, the deterioration in the quality of those homes over the approximately 70 year period covered (from being "of relatively good to relatively poor quality compared to other homes").

Although the review did not identify directly comparable measures of dampness, there was some evidence from the literature that damp housing may have been more prevalent in

Scotland/Glasgow than in England/Liverpool & Manchester in the latter half of the 20th century.

There was either no evidence, or mixed evidence, in relation to any potential relative disadvantage in Scotland for housing indicators such as amenities and the age of homes.

The review also highlighted that there has been substantial improvements in housing conditions in Scotland relative to the rest of the UK in recent decades, with the gap between Scotland and England having narrowed (indeed by 2011, overcrowding was lower in Scotland than in England).

Finally, no evidence was uncovered in the review of Scotland/Glasgow having been disadvantaged in terms of housing allocation policies. However, other, relevant policy decisions described elsewhere in this report (e.g. the more socially selective nature of the post-war New Towns programme in Scotland compared with England, the development of larger-scale within-city peripheral estates alongside lower investment in local authority housing repairs and maintenance in Glasgow) are likely to be highly relevant and, together with the issue of housing quality, to have contributed to a greater vulnerability of the population (as also discussed elsewhere in this report). Furthermore, the differing rehousing policies in Glasgow may have done more damage to the social networks and sources of support in the city compared with the situation in the comparison cities.

Conclusion

The clearest evidence for differences (especially historical differences) in housing quality between Scotland and England relates to overcrowding. In that sense, this hypothesis links directly to several others – in particular the inadequate measurement of deprivation, the lagged effects of poverty & deprivation, the nature and scale of urban change, lower 'social capital', political influences and vulnerability – all of which are likely to have contributed to the development of excess mortality in Scotland and Glasgow.

Assessment of evidence of causality

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

Bradford Hill's criteria	Assessment ^{Ixxxvii}	Comments
Strength of association	Y	
Temporality	Y	
Consistency	Y	
Specificity	Y	
Biological gradient	Y	Housing availability and quality is a known and well-evidenced determinant of health.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland? Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	Until very recently, levels of overcrowding, and possibly dampness, were worse in Scotland. We have been unable to identify other data sources that would provide reliable comparisons between Scotland and England & Wales

^{boxvii} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow? Assuming a difference in exposure, would this have occurred prior to the outcome?	Y Y	Overcrowding was worse in Glasgow compared with Liverpool and Manchester over the period of (and decades before) the emergence of excess mortality. We have been unable to identify other data sources with which to meaningfully and
		aspects of housing quality.

A22 Impacts of the world wars

Description of hypothesis

It has been suggested that a greater number of deaths (or a disproportionate number of deaths among younger people) or levels of disability resulting from the world wars has impacted disproportionately on the Scottish populations.

Rationale

If Scottish populations have experienced relatively high war-related mortality or morbidity, or at younger ages than the comparison populations, it is possible that subsequent health may be diminished through a number of mechanisms:

- Disruption to families and social networks could have caused lagged impacts. There is evidence that the existence of strong social networks and family ties are important to maintaining health (See Appendix A36) (although much of that is drawn through cross-sectional studies and different forms of social change which might not be applicable to this scenario).
- Greater relative loss of economically active populations would have a more detrimental impact on household incomes, and the economic performance of the Scottish areas more generally, leading to greater poverty and/or vulnerability.

Links to other hypotheses

This hypothesis could link to social capital and (the inadequate measurement of) deprivation (in terms of the impacts on economic activity), as well as underlying vulnerability, and levels of deindustrialisation (the latter in relation to economic impact).

Evidence overview

The crude number, and proportion of mobilised armed forces, who died in the first world war I (WWI) from Scotland compared with elsewhere has been disputed because of difficulties in counting the number mobilised (e.g. do you include those from the Scottish diaspora in the numerator or denominator, or those who joined the armed forces before hostilities started; and deaths may not have been registered in the home nation)^{164, 424}.

The routine administrative datasets for Scotland (and England & Wales) do not include deaths abroad among the armed forces (apart from those injured abroad but dying at home)^{425, Ixxxviii}. The routine datasets therefore show the trends for (predominantly) civilian deaths.

^{boxviii} Note that the decrease in life expectancy shown in Figure A22.1 around the second world war (WWII) has therefore been attributed to: "a combination of factors including deaths amongst returning wounded, deaths associated with air raids and military activities in Scotland, and a significant increase in road accident deaths (particularly those involving pedestrians) caused by the imposition of blackout regulations"⁴²⁵. As stated below, a large part of the decrease around WWI was attributable to the Spanish Influenza epidemic of the time.

Figure A22.1 shows the life expectancy at birth for civilian men and women in Scotland and England & Wales from the mid-19th century, which covers the relevant period (and includes the influenza pandemic which occurred in the immediate aftermath of WWI). This dataset shows that the civilian life expectancy dropped much further in England & Wales than in Scotland^{Ixxxix}.

Age-specific civilian mortality rates were higher in Scotland than in England & Wales during the first part of the second world war (WWII), but this rapidly equalised during the course of the war among both men and women.

There is therefore uncertainty about the extent to which armed forces deaths may have impacted on subsequent mortality in Scotland compared with England & Wales. Differences in civilian deaths following WWI seem to have disadvantaged England & Wales rather than Scotland, and the mortality rates were not substantially dissimilar for a prolonged period during WWI.

Figure A22.1 Life expectancy at birth for men and women in Scotland and England & Wales (source: Human Mortality Database).



^{boxix} For male life expectancy, the difference between pre-WWI (1909-13) and WWI period (1914-18) in England was -10.8 years (-21%). The equivalent figures for Scottish males were -0.6 years (-1%). For WWII, comparison of 1936-1938 with 1939-1945 shows a decrease of -2.2 years (-3.6%) for England and -0.9 years (-1.6%) for Scotland.

Conclusion

There is uncertainty about whether or not the Scotland had a higher rate of armed forces deaths during the WWI and WWII. Civilian mortality rates were substantially lower in Scotland around WWI (and the associated influenza outbreak) and slightly higher at the start of WWII. There is currently insufficient evidence to support this hypothesis being an important part of the explanation for excess mortality, but further clarity on the relative armed forces mortality rates would be enlightening.

Assessment of evidence of causality

Bradford Hill's	Assessment ^{xc}	Comments
criteria for causality		
Strength of	N/A	
association		
Temporality	N/A	
Consistency	N/A	
Specificity	N/A	Note: As the hypothesis relates to a particular cause
Biological gradient	N/A	applied in general terms.
Plausibility	N/A	
Coherence	N/A	
Experiment	N/A	
Analogy	N/A	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor	U	There is uncertainty about
worse in Scotland?		whether the relative
		mortality rate during the
Assuming a difference in exposure,	Y	wars was different, and
would this have occurred prior to		therefore the temporality

^{xc} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

Comparison	Assessment	Comments
the outcome?		of an unknown exposure becomes less relevant.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	U	There is uncertainty about whether the relative mortality rate during the wars was different, and therefore the temporality of an unknown exposure becomes less relevant.
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	

A23 Income inequalities

Description of hypothesis

It is hypothesised that Scotland's, and Glasgow's, higher overall levels of mortality (and, related to that, wider socioeconomic inequalities in mortality) are caused by wider inequalities in income across the population compared with elsewhere in the UK.

Rationale

Reflecting the 'neo-materialist' view of the causes of health inequalities, many authors have pointed to widening income inequalities in the UK since the early 1980s as the driving force behind the widening health inequalities that have been observed over the same period^{6,107,109,110,130,132,306,426-430}. Thus, if income inequalities were wider in Scotland (and Glasgow), that might explain the country's (and city's) wider health inequalities compared with elsewhere in the UK.

Separately, it is argued (although disputed by some⁴³¹⁻⁴³⁴) that among wealthy societies, those with wider income inequalities have poorer health and social outcomes across the *whole* population⁴³⁵⁻⁴⁴⁰. It is argued that the psychosocial mechanism behind this (relating to 'status anxiety': that is, that greater income inequalities place people within wide social hierarchies, increasing 'social status competition', which leads to stress and a whole range of adverse social and health outcomes) only operates at the level of whole societies (e.g. entire countries, or US states); however, others have suggested that similar processes may operate at more local levels. This is all potentially relevant to the issue of excess mortality in Scotland and Glasgow.

Links to other hypotheses

Spatial patterning of deprivation; inadequate measurement of deprivation; political influences and vulnerability.

Evidence overview

Income inequalities in the UK are among the widest of all high-income countries, largely explaining the increased, and now very wide, health inequalities across the UK^{119,347}. However, in relation to the issue of Scottish excess mortality, it has been shown that income inequalities are in fact wider in England than in Scotland, and regional estimates of income inequality suggest levels are slightly higher in North West England (including Liverpool and Manchester) compared with West Central Scotland (which includes Glasgow)^{20,441,xci}. Other

^{xci} The national and regional comparisons were for the mid-2000s and based on calculations from data from the Luxembourg Income Study (LIS) (for estimates for Scotland, England and North West England) and the Scottish Household Survey (for West Central Scotland (WCS)). These were based on large, and representative, samples: approximately 4,500 and 20,000 for Scotland and England respectively, 3,000 for NW England, and 11,000 for WCS. It should be pointed out, however, that other authors^{442,443} have argued that income inequality in Scotland is similar to England as a whole, but is wider than in regions such as North England and indeed Merseyside (although identical to Greater Manchester). This was based on analyses of the British Household Panel Survey (BHPS) with much smaller sample sizes (e.g. for 2004: <500 for Scotland, 600 for NW England). However, income

analyses have shown that among those in employment, within-city levels of income inequality have been very similar Glasgow, Liverpool and Manchester (and similar to Britain as a whole) in recent decades, and are not highest in Glasgow. Adding the results of those analyses to the previously cited evidence of very similar distributions of income deprivation across those three cities suggests that it is unlikely that Glasgow is a relatively more unequal city in terms of income.

Conclusion

The widening of income inequalities since the late 1970s has played a fundamental role in the widening of health inequalities across the UK, and is an important component of the context for the emergence of excess mortality in Scotland. However, income inequalities per se are not wider in Scotland/Glasgow than in England & Wales and thus appear an unlikely direct explanation for the excess.

estimates from the BHPS have been criticised on the basis of these relatively small sample sizes, as well as associated worries concerning accuracy²⁰². The same authors above also argue (using the same data sources) that the distribution of 'unearned income' (e.g. from investments, and used as a proxy for 'wealth' as opposed to basic household income) is more unequal in Scotland than in the UK as a whole, although the same caveats regarding sample sizes and accuracy of income estimates apply.
Assessment of evidence of causality

Bradford Hill's	Assessment ^{xcii}	Comments
criteria for		
causality		
Strength of	Y	
association		
Temporality	Y	
Consistency	Y	
Specificity	Y	Although contested, there is a substantial evidence base
Biological	Y	linking income inequalities with mortality. The extent to
gradient		result of income inequality is the main ongoing debate.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	N	Income inequality in Scotland is lower than the
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	from the late 1970s onwards.

^{xcii} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	N	Glasgow's income inequalities are similar to those in NW England
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	

A24 Individual values

Description of hypothesis

It has been proposed that Scotland's, and in particular Glasgow's, population may be characterised by different individual 'values' compared with those in the rest of the UK (especially those in Liverpool and Manchester) and that such differences would influence health behaviours and choices and, therefore, ultimately health outcomes.

Rationale

This particular hypothesis embraces a number of overlapping concepts:

- psychological outlook, i.e. differences in optimism, aspiration/achievement, meaningfulness of life, self-efficacy^{xciii,444,445}
- hedonism
- time and risk 'preferences' that is, are Scots/Glaswegians more 'present-oriented', placing relatively less value on future outcomes, and are more risk-seeking; time and risk preferences are key economic concepts which are related to individuals' 'investments' in their future health⁴⁴⁶⁻⁴⁴⁹.
- individualism
- materialism

There are fairly obvious links between important aspects of health (e.g. health behaviours) and concepts such as time preferences and hedonism. A number of studies have also highlighted the health benefits of 'positive psychological wellbeing'^{450,451}, including, more specifically, an optimistic outlook^{452- 457} and greater 'self-efficacy'^{458- 461}. Individualism and materialism have both been highlighted as features of modern Western culture which have negative impacts on health and wellbeing⁴⁶², and in the case of materialism, a number of studies have pointed to significant associations between materialism and measures of life-dissatisfaction, depression, anxiety, and alienation⁴⁶²⁻⁴⁶⁴.

Links to other hypotheses

Aspects of this broad hypothesis link to, or overlap with, a number of other theories that have been suggested. These include: sense of coherence, anomie, social mobility and social capital.

^{xciii} Self-efficacy has been defined as "the belief that one can perform a novel or difficult task, or cope with adversity – in various domains of human functioning"⁴⁴⁴: it links, therefore, to the notions of optimism (reflecting an 'optimistic self-belief"^{444, 445}), aspirations, as well as another hypothesis discussed in this report, social mobility.

Evidence overview

As with many of the hypotheses discussed in this report, at the time of the 2011 synthesis no comparable data for Scotland (versus England & Wales) or Glasgow (versus other UK cities such as Liverpool or Manchester) were available. This was addressed to a degree by including a number of relevant measures in the three-city survey of Glasgow, Liverpool and Manchester undertaken in 2011. A summary of the results of the analyses of those measures follows below.

A number of different measures were used to assess the hypothesis and its many, often overlapping, sub-components. For the majority there was no strong evidence to support the overall hypothesis (although one or two exceptions were noted).

In terms of 'psychological outlook', the analyses showed the following:

- Levels of **optimism** (measured by use of the Life Orientation Test (Revised) (LOT-R) scale⁴⁶⁵, a scale deemed to be the best available measure of optimism) among the Glasgow survey respondents were not lower than among respondents in the other two cities: the mean LOT-R score among the Glasgow sample was very similar to that of the Liverpool sample, and higher than that of Manchester. This was generally true in comparisons of all social classes.
- As measured by Schwartz's 'human value' of *achievement*⁴⁶⁶⁻⁴⁷¹, **aspirations** were higher, not lower, in the Glasgow sample compared with the samples in Liverpool and Manchester. This was again generally true in analyses of high and low social class.
- Overlapping with the notions of aspiration and optimism, **self-efficacy** (measured in the three-city survey by the Generalised Self-Efficacy (GSE) scale) among respondents from Glasgow was not lower compared with the two other samples: the mean GSE scores were similar in Glasgow and Liverpool, and higher than that of the Manchester sample. A similar pattern was seen across different social classes.
- As discussed in Appendix A35, analysis of the meaningfulness component of the Sense of Coherence scale (assessing respondents' perceptions of the extent to which their lives have meaning and purpose) showed this to be higher, not lower, among Glasgow respondents compared with those in the English cities. This was true of those living in the most, and least, deprived neighbourhoods in the three cities.

Highly related to the concept of psychological outlook, there was no evidence of a greater culture of **hedonism** among the overall Glasgow sample (compared with those in the English cities). However, females in Glasgow were more associated with hedonism compared with females in Liverpool (but not compared with those in Manchester). There were generally no meaningful differences between the samples when analysed by social class or area deprivation.

Similarly, there was no evidence of present-orientated **'time preferences'** (reflecting less 'investment' in future health status) in Glasgow, albeit that the analyses were hindered by

questions about the reliability of the data. However, there was some evidence that the Glasgow sample was more risk-seeking compared with those in Liverpool (but not compared with the Manchester sample).

There *was* evidence of more **individualism** (or at least less 'universalism') among the Glasgow sample, a finding which echoed, and reinforced, results from the analyses of the social capital data that were also collected in the survey (and discussed elsewhere in the report). This was based on analysis of the 'universalism' values of Schwartz's Human Values Scale, a value derived from statements relating to the importance of equal opportunities, tolerance and understanding of others, and care for the environment^{xciv}. In this sense universalism can be considered to be the opposite of individualism, and the relatively lower levels of universalism seen in the Glasgow sample were seen across the social spectrum: however, echoing results of some of the social capital analyses, the greatest differences were seen in comparison of those of *high* social class and those living in the *least* deprived neighbourhoods.

Data limitations made assessment of whether there were differences in levels of **materialism** between the populations difficult^{xcv}. However, there was some suggestion that materialism was more associated with the Glasgow sample in comparison with those in Liverpool, but not in Manchester. This was truer of those living in poorer neighbourhoods in Glasgow compared with similarly deprived parts of the two English cities.

Conclusion

Overall there is very little evidence to suggest that there are differences in individual values between the Scottish and English populations which might be relevant to the issue of Scottish excess mortality. That said, however, the available data are for Glasgow only (rather than Scotland).

^{xciv} The universalism value is derived from three statements, with respondents assessing the extent to which they identify with this type of person. These are (using here the male version of the question wording): 1) He thinks it is important that every person in the world should be treated equally. He believes everyone should have equal opportunities in life; 2) It is important to him to listen to people who are different from him. Even when he disagrees with them, he still wants to understand them; and 3) He strongly believes that people should care for nature. Looking after the environment is important to him.

^{xcv} A previously validated question on materialism was not included in the questionnaire. In its absence, one of the questions which make up Schwartz's 'Human Value' of 'power' was used (as it asks about the importance of being rich and having money/expensive things). An additional materialism-related question was created by the survey team: this also showed greater association with the Glasgow sample than the Liverpool sample (but not compared with the Manchester sample). However, interpretation was difficult because the question had not been previously validated.

Assessment of evidence of causality

Bradford Hill's criteria for	Assessment ^{xcvi}	Comments
causality		
Strength of	Y	
association		
Temporality	U	
Consistency	Y	
Specificity	Y	There are a number of components to this hypothesis,
Biological	Y	efficacy) have been associated with differences in
gradient		health outcomes.
Plausibility	Y	
Coherence	Y	
Experiment	U	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	U	There are no data available to assess differences between
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	Scotland and England & Wales.

^{xcvi} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow? Assuming a difference in exposure, would this have occurred prior to the outcome?	N U	For the vast majority of the components of this hypothesis (optimism, self-efficacy, hedonism, time preferences etc) there is no evidence of the population in Glasgow
		the population in Glasgow being associated with more 'negative' individual values. The exceptions are individualism and materialism: however, the differences in relation to individualism arguably relate more to the 'reciprocity' aspect of social capital (discussed elsewhere in this report),
		while the evidence for differences in materialism is extremely limited. No trend data were available.

A25 Lagged effects of poverty and deprivation

Description of hypothesis

Higher levels of mortality in Scotland/Glasgow that do not appear to be explained by current levels of poverty and deprivation may, however, be influenced by socioeconomic conditions experienced by the Scottish populations in previous years.

Rationale

As stated elsewhere in this report, the link between poverty at all stages of the life-course and subsequent poor health is proven and profound. Levels of current deprivation which are either similar (in comparison of cities) and/or do not explain variations in mortality rates (in comparisons of cities, and of countries) may mask greater differences in poverty historically which could have impacted across the life course in a manner not detected by crosssectional analyses.

Links to other hypotheses

Inadequate measurement of deprivation; spatial patterning of deprivation; housing quality and allocation; the nature and scale of urban change; deindustrialisation; quality of physical environment; early years experiences.

Evidence overview

National comparisons have demonstrated clearly that Scotland was relatively more deprived compared with England & Wales 35 years ago than is the case now (Figure A25.1, showing comparison of the four components of the Carstairs & Morris deprivation index, 1981-2011). However, this is complex and the argument suffers from a lack of consistency in that as Scotland has become less deprived relative to elsewhere in Great Britain since 1981, the excess level of mortality has increased, not decreased.

Figure A25.1. Comparison of the components of the Carstairs & Morris area deprivation index between Scotland and England & Wales, 1981-2011 (Source: Schofield *et al.*, 2016 (from Census data)).



In relation to Glasgow, Liverpool and Manchester, few differences were observed in historical analyses of 'core poverty' and 'breadline poverty' back to the early 1970s, and in analyses of unemployment and male social class back to the 1950s (Figures A25.2-A25.4). Nor did analysis of household amenities over the period 1971-2001 show any consistent, clear differences (Figure A25.5)^{134,xcvii}. However, this is not true of overcrowding which, historically, was much higher in Glasgow compared with any other UK city, including Liverpool and Manchester. Analyses show that not only was overcrowding higher in Glasgow at the level of the whole city, but that the distribution of overcrowding across the city was markedly different to that seen in the English cities (Figure A25.6)^{134,xcvii}.

^{xcvii} Note that the list of household amenities included in census questions has varied at different time points. Figure A25.5 summarises a subset of these data for the period 1971-2001, with the amenities grouped under three headings: lack of access to an indoor flush toilet; lack of access to at least one other amenity other than toilet; not lacking any amenities. The full list of household amenities included are: 1971 - hot water supply, fixed bath or shower, inside WC; 1981 - fixed bath or shower, inside WC; 1991 – fixed bath or shower, inside WC, central heating; 2001 - fixed bath or shower, inside WC, central heating. For amenities such as toilets, hot water, baths etc, the census questions relate to exclusive (rather than shared) access.

^{xcviii} Note that the data presented in Figure A25.6 may, to a degree, overestimate the precise levels of overcrowding in Glasgow (and other Scottish cities) relative to Liverpool and Manchester in 1971. This is because of potential definitional differences between the Scottish and English censuses relating to the calculation of the number of rooms in a household. However, it is difficult to adjust for these potential differences with any accuracy; furthermore, sensitivity analyses have shown that any such









crude adjustment would not change the overall picture of significantly higher levels of overcrowding in Glasgow relative to the other English cities.

Figure A25.4



Figure A25.5



Figure A25.6^{xcix}



Similar analysis of the distribution of deprivation in the 1970s identified a much greater concentration of multiply-deprived neighbourhoods in Glasgow compared with the two English cities⁴⁷². This was particularly influenced by the high levels of overcrowding in Glasgow at the time. The analysis identified the 15% most deprived neighbourhoods across Britain, based on a combination of overcrowding, lacking amenities and unemployment, and showed that almost 600 of these areas were in Glasgow (occupied by 19% of the city's population), compared with 93 in Manchester (housing 7% of the population) and 60 in Liverpool (5% of residents).

This is a complex issue, however, especially since overcrowding (for which the greatest historical differences between Scotland and England, and between Glasgow and Liverpool and Manchester, have been shown) is included within the Carstairs index of deprivation. The latter has been used in many analyses of excess mortality i.e. where a high excess has been observed *after adjustment for* overcrowding. However, the time-specific element is important here e.g. those analyses took account of contemporary, not past, levels of overcrowding. For analysis around the 2011 Census, therefore, overcrowding would not

^{xcix} Source: Walsh, 2014¹³⁴. Note that the data presented in Figure A25.6 may, to a degree, overestimate the precise levels of overcrowding in Glasgow (and other Scottish cities) relative to Liverpool and Manchester in 1971. This is because of potential definitional differences between the Scottish and English censuses relating to the calculation of the number of rooms in a household. However, it is difficult to adjust for these potential differences with any accuracy; furthermore, sensitivity analyses have shown that any such crude adjustment would not change the overall picture of significantly higher levels of overcrowding in Glasgow relative to the other English cities.

have attenuated the excess because it was not higher in Scotland in that period; however it had been higher in at least the previous six decades.

Historically high rates of deprivation (evidenced by overcrowding) are important. As Scotland, and Glasgow, emerged from the post-war period, these stark differences in overcrowding compared with elsewhere in Britain could reasonably be seen to place the population north of the border at a disadvantage in terms of the potential to benefit from opportunities for health improvement that was then emerging in terms of the emergence of the new welfare state.

There is an obvious link between this hypothesis and that relating to early years' experiences. Taulbut *et al.* compared socioeconomic conditions in childhood between Scotland and England, and between regions around the three English cities using longitudinal birth cohort data from the 1940s, 1950s, 1970s and 2000^{52,69}. Few meaningful differences were observed, although the authors acknowledged that issues around sample size, representativeness and attrition rates might impact on the results.

Conclusion

Although few differences have been observed between Glasgow, Liverpool and Manchester in terms of historical comparisons of income and employment based measures (e.g. core poverty, social class, unemployment), the notably higher levels of overcrowding in the Scottish city back to at least the middle of the 20th century are likely to be relevant. This is the case for Scotland as a whole compared with England & Wales – and indeed different indicators show the Scottish population to have been more materially deprived than those in England & Wales for many decades. However, the causal pathways are complex, and the relationship with excess mortality less clear, given that the excess has increased over a period in which Scotland has become relatively less deprived compared with the rest of Britain.

Assessment of evidence of causality

Bradford Hill's	Assessment ^c	Comments
criteria for causality		
Strength of	Y	
association		
Temporality	Y	
Consistency	Y	
Specificity	Y	Poverty and deprivation across the life-course are
Biological gradient	Y	populations.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland? Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	Carstairs deprivation measures from 1981, and overcrowding data prior to this, suggest that the population of Scotland has historically been exposed to greater deprivation than elsewhere in Britain.

^c Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow? Assuming a difference in exposure, would this have occurred prior to the outcome?	Y Y	Glasgow was not notably more deprived in terms of income or employment based measures; however, Glasgow's overcrowding data from 1951 onwards suggest that the residents of the city have been exposed to relatively worse levels of deprivation historically.

A26 Migration

Description of hypothesis

The populations of Scotland and Glasgow experience relatively higher mortality because of changes to their composition brought about by migration.

Rationale

The principal component of this hypothesis is that health in Scotland and Glasgow may have been adversely affected by the loss of healthier people migrating elsewhere, leaving behind a less healthy population more likely to suffer from higher mortality.

As described in the 2011/12 synthesis^{50,51}, some more specific sub-components of this hypothesis have included the effects of outward migration from Scotland to the USA, Canada, Australia, New Zealand etc in the 18th, 19th and early 20th centuries, as well as inward migration to Glasgow from Ireland and the Highlands (again particularly in the 19th and early 20th centuries).

There is a considerable amount of evidence of the effects of such 'selective migration' on health status. This definition of migration is 'selective' in the sense that migrants tend to differ from the general population in a number of ways, and that propensity to migrate is influenced by a number of factors (for example, age, level of education, socioeconomic status (SES)⁴⁷³⁴⁷⁸). Crucially, migration is often selective in terms of health status with, in general, migrants tending to be of above average health compared with non-migrants. With migrants tending to be healthier and better educated, illness and mortality rates can fall in places where population size is increasing, and rise in places experiencing population loss^{384, 479-482}.

It has alternatively (and more speculatively) been hypothesised that Scotland and Glasgow have experienced less population change than elsewhere, resulting in a more static, inwardlooking, homogenous population associated with lower levels of aspiration and social mobility, with resulting negative impacts on health. This clearly links directly to the 'individual values' hypothesis.

Links to other hypotheses

Elements of the hypothesis overlap with: the nature and scale of urban change (including, in particular, the effects of population movement to the New Towns (mentioned briefly below); inadequate measurement of deprivation; social mobility; individual values; genetics.

Evidence overview

a) Selective migration: Scotland and Glasgow

As stated above, there is evidence of the impact of selective migration on population health status. However, the scale at which this operates, and the extent of its impact, is disputed. In terms of *scale*, it has been argued by some that the effects of migration on the health of areas are only felt at a small-area level (e.g. neighbourhood), and not in relation to migration

to and from larger areas⁴⁸³. However, other studies have suggested that its influence can be significant at the level of whole cities⁴⁸⁴ and countries⁴⁸⁵. In terms of impact, one study attributed *all* inequalities in mortality between British districts to migration⁴⁸⁶ – although the accuracy of that finding has been questioned by others⁴⁸⁷. Another study suggested that 50% of the widening socioeconomic gap in mortality that took place in England & Wales in the 1990s was attributable to the effects of selective migration⁴⁸⁸, while further research in England & Wales highlighted the changes in mortality brought about by the flow of healthy migrants between 1971 and 1991 from deprived to less deprived areas (mortality rose in the former, and fell in the latter)⁴⁸⁹.

Crucially, however, the existing research evidence does not support the hypothesis that selective migration has impacted to a large degree on Scotland's and Glasgow's relatively higher mortality rates since the 1980s. For example, it was shown that the widening mortality gap witnessed in Scotland between 1981 and 2001 could not be explained simply in terms of population change⁴⁹⁰, while another study showed deprivation to be more important than population change in explaining changing mortality rates in Scotland over the same 20 year period. Furthermore, analysis of Glasgow's poor health and high mortality compared with other parts of Scotland suggested that in fact migration was not a significant contributory factor⁴⁹¹. Similarly, separate research found that the widening health inequalities *within* Glasgow could not be explained in terms of selective migration⁴⁹². Importantly, Scottish migrants elsewhere in the UK display a mortality pattern very similar to that of the non-emigrating population and retain their higher mortality rates compared with native residents: this has been shown for migrants to England & Wales^{22,23}, and to Northern Ireland.

Despite this, however, it is still very possible that particular aspects of migration and population movement may be pertinent to poorer health in Glasgow compared with Liverpool and Manchester. As described elsewhere in this report, in the post-war period Glasgow lost a higher percentage of its better educated and skilled population to the New Towns than was the case in Liverpool and Manchester, while in the English cities some peripheral estates, housing some of the more disadvantaged population, were built outside the city boundaries, whereas all of Glasgow's were contained within the city limits. Although for the same (or similar) level of deprivation, Glasgow exhibits much higher mortality, the impact of these historical changes on the composition of the city's population may have contributed to masking differences between the populations of the three cities.

The potential effects of historical waves of inward and outward migration are also difficult to quantify. In relation to Glasgow, although there has been some evidence of poorer health among those of Catholic/Irish descent (linked to, but not fully explained by, socioeconomic characteristics⁴⁹³), the two comparator cities (Liverpool and Manchester) also experienced similar waves of historical immigration from Ireland, in particular from Ireland in the mid-19th century following the potato famine^{135,138,139,494 ci}. No evidence is available to assess the

^{ci} For example, in 1848 1,000 Irish emigrants were recorded as arriving in Glasgow each week, and by 1851 almost 20% of the city's population had been born in that country¹³⁵. In Liverpool, an estimated quarter of a million Irish emigrants reached the city in that same late 1840s period, many of whom (especially the poorest who could not afford further travel) remained in the city permanently^{138,494}.

impact of historical outward migration from Scotland in the early 20th century and before. However, this period is less relevant to the emergence of Scottish excess mortality from 1980 onwards.

b) Population characteristics

As described elsewhere in this report, there is no evidence that people in Glasgow are characterised by lower aspirations, or are less motivated to succeed, compared with those in Liverpool and Manchester. Nor is there any evidence of lower rates of social mobility in Scotland and Glasgow relative to other parts of the UK. Even if such differences existed, the extent to which they would be related to differences in population movement is unclear. Trends in rates of population change in Glasgow, Liverpool and Manchester have generally been very similar over the past century, and although Manchester is now a much more ethnically diverse city than Glasgow (the potential impact of which is discussed elsewhere in this report), the ethnic profile of Liverpool is very similar to that of the Scottish city. Thus, it seems unlikely that such factors, linked to differing rates of population change, have impacted on population health status.

Conclusion

The weight of evidence suggests that it is unlikely that selective migration from Scotland from the 1980s onwards has played a major role in the widening levels of excess mortality in Scotland. However, as discussed in the main part of the report (and elsewhere in this Appendix), in the case of Glasgow it appears plausible that the socially selective New Town policy adopted in the two to three decades following the end of the second world war may have contributed in part to a greater vulnerability among the city's population in comparison with those living in Liverpool and Manchester.

Similarly, one third of the population increase in Manchester between 1841 and 1851 was attributable to Irish migration (with 15% of the city's population in 1851 recorded as being Irish)¹³⁹. In the middle of the 19th century almost half of Britain's Irish population were living in Glasgow, Liverpool, Manchester and London¹³⁹.

Assessment of evidence of causality

1)	General assessment	of likelihood of	causality in	terms of Bradfo	rd Hill criteria.
- 1	General assessment		cuusuncy m	Critis of Diddio	

Bradford Hill's criteria for causality	Assessment ^{cii}	Comments
,		
Strength of	Y	
association		
Temporality	Y	
Consistency	Y	
Specificity	Y	Selective migration can be detrimental to the health
Biological gradient	Y	recipient population.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	U	Examination of longitudinal data have
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	snown that, from the 1980s onwards, selective migration has not been responsible for the excess.

^{cii} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor	Υ	Examination of
worse in Glasgow?		longitudinal data has
		shown that, from the
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	1980s onwards, selective
		migration has not been
		responsible for the excess.
		However, selective
		migration to the New
		Towns, prior to the 1980s,
		suggests that migration
		could be important in
		explaining the excess
		through an earlier
		exposure.

A27 Obesity

Description of hypothesis

Prevalence of obesity in Scotland/Glasgow is higher than elsewhere in the UK (including in comparable cities like Liverpool and Manchester), and that this contributes to the high levels of excess Scottish mortality.

Rationale

There are well documented links between obesity and a wide range of health conditions⁴⁹⁵. However, given that obesity is socially patterned and the excess mortality is defined as higher mortality over and above that explained by socioeconomic factors, the assumption is that obesity contributes to the excess independently of deprivation and socioeconomic conditions.

Links to other hypotheses

Health behaviours, in particular diet and physical activity.

Evidence overview

At the national level (Scotland compared with England), survey data suggest that although the upward trends are broadly similar in both countries, levels of adult obesity have tended to be slightly higher in Scotland than in England in recent years, and that this is true for both males and females (Figures A27.1 and A27.2). Figure A27.1^{ciii}







Comparison of 2003 data suggested that, for women only, the difference in obesity levels between Scotland and England was not explained by differences in SES⁴⁹⁶.

^{ciii} Note that in both Figure A27.1 and A27.2 data for Scotland are shown for 1995, 1998, 2003 and then 2008-2012. No data are available for the years 1996-1997, 1999-2002 and 2004-2007 (the time points shown on the chart with a dotted line). English data are not available for 1995.

However, BMI was included in modelling analyses of Scottish excess mortality using the same Scottish and English national health survey data (as described elsewhere in this Appendix, this was based on pooled data over the period 1994-2008): the high level of excess mortality among Scottish adults was <u>not</u> attenuated by inclusion of BMI.

Comparisons of obesity by social class show a clear social gradient among females, but not males (Figures A27.3 and A27.4)^{241,242}. For females, obesity rates are fairly similar in Scotland and England across the different social classes between; however, among males rates tend to be higher in Scotland, particularly among social classes I and II (although the small sample sizes make meaningful comparison difficult).



Figure A27.3

Figure A27.4



At the city level, comparable data for Glasgow, Liverpool and Manchester are very limited. Previously published estimates (for one time point only (c.2003)) showed very similar rates of adult obesity for (Greater) Glasgow (22.8%) and Liverpool (21.9%), with a slightly higher figure for Manchester (25.8%). This similarity was confirmed in analyses of regional data (for Greater Glasgow, Greater Manchester and Cheshire & Merseyside) from the same sources and similar time period with no meaningful differences observed.

Thus, in Scotland (in common with most high income countries), the prevalence of obesity has been rising over the last 20 years, and stark inequalities in obesity have appeared⁴⁹⁷. What happens to these obesity trends in the future, and how they might differentially impact within and between the populations of interest, will clearly be important in determining the future mortality phenomena in Scotland and Glasgow.

Conclusion

The weight of evidence suggests that obesity is unlikely to play a major contributory role in explaining the high levels of Scottish excess mortality. However, given the relatively higher levels of obesity among middle class Scottish residents, it is at least possible that analyses of social class-obesity interactions might shed light on aspects of excess mortality among non-deprived Scottish populations.

Assessment of evidence of causality

1) General assessment of likelihood of causality in terms of Bradford Hill criteria

Bradford Hill's criteria	Assessment ^{civ}	Comments
for causality		
Strength of association	Y	
Temporality	Y	
Consistency	Y	
Specificity	Y	
Biological gradient	Y	causally related to ill-health and mortality.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales

Comparison	Assessment	Comments
Is the hypothesised causal factor Y worse in Scotland? U	Y U	For the short time frame in which data are available, there has been a slightly higher prevalence of obesity in Scotland compared with England & Wales, but this did not explain any of the excess in statistical modelling analyses.
would this have occurred prior to the outcome?		

^{civ} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	N	Obesity levels in Glasgow are similar to those in Liverpool and Manchester. No trend data are available.
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	

A28 Political influences and vulnerability

Description of hypothesis

This incorporates the 'political attack' hypothesis, which is that:

- the UK overall was exposed to a more trenchantly neoliberal policy agenda in the period after 1979 than other countries in Europe, which forced rapid deindustrialisation and used high levels of unemployment as the basis for a wider attack on the institutions and culture of the organised working class (including local government, council housing and trade unions);
- Glasgow and Scotland were both economically (in terms of weak and 'declining' heavy industry and reliance on 'branch plants' in lighter industries) and socially (in terms of higher historical levels of deprivation and overcrowding, and a higher reliance on council housing) more vulnerable to, and cumulatively worse impacted by, the damaging effects of this 'political attack' than other parts of the UK
- there was a distinctive impact in terms of political culture from this wider attack evidenced by election outcomes and the strengthening perception of Scotland's 'democratic deficit'^{50,119}.

It further develops this hypothesis both theoretically and empirically:

- theoretically, it draws on a wider literature across a range of disciplines to develop a theory of vulnerability applicable to the issue of Scottish excess mortality (and for its occurrence across different socioeconomic groups in Glasgow in particular)
- empirically, it looks in detail at the historical policy developments which provide the context in which Glasgow and Scotland can be seen to have acquired the heightened economic and social vulnerability identified above.

Links to other hypotheses

This hypothesis links closely to the nature (and scale) of urban change, housing quality and provision, 'social capital' and the cultural hypotheses; and to an extent it provides the basis for an overall synthesis of these factors.

Rationale

There were marked changes in the mortality epidemiology of Scotland and Glasgow during the 1980s: growing excess mortality; rising health inequalities; increases in alcohol- and drug-related deaths, suicide and violent deaths; and a divergence from the European trends of improving mortality rates overall for young adults. These epidemiological features resemble those seen elsewhere, including Eastern Europe, North America and New Zealand, following the adoption of neoliberal policy agendas, suggesting that these agendas had an important causal role.

The concept of 'vulnerability' has an important role in explaining how the potentially adverse consequences of national level policies manifest to differing degrees in different geographies. Drawing on wider work across a range of disciplines (including disaster

mitigation, development studies, political economy and public health), including that of Galea and colleagues on vulnerability and variability in health outcomes between places¹⁴⁸⁻¹⁵², alongside parallel work to grasp more effectively than has hitherto been the case the processes through which Glasgow and Scotland acquired their particular 'vulnerabilities', might help to explain how and why Scotland and Glasgow may have been more adversely affected by UK government policies after 1979 than comparator populations (Liverpool and Manchester, and England and Wales, respectively).

Evidence overview

UK and Europe – divergence of policy

There is strong evidence that the UK government adopted a different policy agenda to the rest of Europe from 1979, involving rapid deindustrialisation to force up unemployment as the basis for a wider reorganisation of power relations between labour and capital, involving the 'rolling back' of the Keynesian Welfare State^{, 498}. There were both beneficiaries and losers of this agenda, and these were distributed unevenly in terms of both social class and geography⁴⁹⁹. There was a high concentration of people who lost out in the deindustrialising, working class areas of Wales, the Midlands, Northern England, Northern Ireland and Scotland, all areas which suffered marked increases in unemployment and poverty as a result.⁵⁰⁰ This was in contrast to the experience across much of Europe, which took a more managed and mitigated approach to socioeconomic change and deindustrialisation in the same period (including greater protection of manufacturing industry)^{, 501-502, cv}.

West Central Scotland, in comparison with other deindustrialised areas of the UK, has been found to have been cumulatively worst affected by the damaging impacts of this policy agenda in the 1980s.

Impacts of neoliberalism

As the policy programme of the UK government shifted after 1979, West Central Scotland displayed more slowly improving^{19,20}, and greater variation^{cvi} in, life expectancy than the European comparison populations. During the 1980s and 1990s, Great Britain as a whole and Scotland in particular experienced a rapid and sustained rise in health inequalities, to leave Scotland with the widest health inequalities in western and central Europe. It is highly likely

^{cv} It is worth noting that the trend towards neoliberalism has continued since, both in the UK and also more widely in Europe and beyond, with rather more confrontational and 'destructive' phases of 'rolling back' the state (characterising 'Thatcherism' in the UK in the 1980s and arguably the period since 2010) as compared with rather more 'constructive' (although not necessarily less damaging) phases of 'rolling out' of new institutions and arrangements of the kind advocated by neoliberals under John Major and particularly 'New Labour' in the 1990s and 2000s. This 'rolling out' was exemplified by the move away from confrontation with local government, towards actively using local government to lead change via initiatives such as 'City Challenge' and the Single Regeneration Budget in England, and the 'Programme for Partnership' in Scotland, in the process moving from 'government' to 'governance'⁵⁰³. In many ways, Glasgow can be seen to have been at the forefront of this move – several years ahead of Manchester, which has often been portrayed as the prime mover and 'exemplar'.

^{cvi} There was no means of ranking areas available and so the variations in life expectancy noted here are the best available approximation of inequality.

that the change in policy is causally responsible for these impacts at UK level since similar neoliberal exposures have led to similar rises in inequality and lower life expectancy in other countries and at other times^{118,504}, and there is some evidence of there being a dose-response relationship⁵⁰⁵. There is also substantial qualitative evidence of political decisions having direct and indirect impacts on the health, and the determinants of health, of people in Scotland (and in quite explicit terms)^{400,506} perhaps most memorably by Glaswegian community activist Cathy McCormack, who described the treatment of her community as a 'war without bullets'.

Vulnerability as an explanation for differential impacts across the UK

The concept of 'vulnerability' has been developed extensively across a wide range of fields, including disaster mitigation, development studies, environmental change and international political economy, and has been specifically developed within the public health literature to explore why some populations experience different health outcomes despite similar exposures. On this basis, population health can be seen as reflecting the interaction of 'vulnerabilities' (such as poverty), and offsetting 'capacities' (such as supportive social networks) combined with population responses to 'stressors' (such as economic recession) and 'protective events' (such as the provision of salutary community amenities).

Wider applications of the concept of vulnerability prove useful in understanding:

- how susceptibility to harm is a variable feature of places and populations and is a product of social, economic and political processes (rather than being simply, or even primarily, 'naturally given')
- how vulnerabilities can have implications for entire populations, rather than just those sections of the population most obviously displaying susceptibilities
- how vulnerabilities can be generated by forces operating at different levels (macro, meso and micro – with the meso-level having a particular salience in relation to Glasgow and Scotland)
- that the nature of such vulnerabilities can be difficult to perceive until they are seen to be revealed or 'surfaced' by events or processes (such as 'political attack') which impact as 'stressors' to generate adverse outcomes (such as excess mortality)
- that combinations of different features of vulnerability in particular places can interact to produce an overall vulnerability which is greater than the sum of its parts^{142-147,150-153,507 -513}.

The 'vulnerabilisation' of Glasgow, 1945 to 1980

New research, based on extensive archive material (much of it government files made available under the '30 year rule'), sheds light on how the implementation of Scottish Office regional policy adversely affected Glasgow in the post-war decades, in ways which would have contributed significantly to its heightened vulnerability to the damaging effects of the post-1979 central government policy agenda. This research, moreover, provides an important context for situating other research findings about differences in urban change across the three cities in the post-war decades. The changes which transpired in Glasgow were intimately shaped by regional policy and its implementation, driven from the Scottish Office.

The research highlights Scottish Office recognition of the severe challenges faced by Glasgow in the post-war period, in particular in terms of the deep-rooted health, housing and economic problems discussed elsewhere in this report. The Clyde Valley Regional Plan ^{cvii} set out to address these issues through the development of four proximal New Towns at East Kilbride, Cumbernauld, Houston and Bishopton, to which population and industry were to be dispersed. In this way, Glasgow's problems were to be addressed in a somewhat regionalised context - Glasgow's needs were at the centre of the plan. However, only East Kilbride was designated in the early post-war period (1948), against opposition from Glasgow, which feared the consequences of the plan for the city itself in terms of loss of population and industry, and thereafter there was a hiatus of several years due to tight public spending and then the return of a Conservative government sceptical about the policy. It was only in the later 1950s that there was a return to a 'regional policy' approach to the city's problems with the designation of Cumbernauld by the Conservative government, this time with the co-operation of the city itself. However, in this context the wider plan was itself subject to a major reorientation – away from a primary focus on addressing the economic, housing and health needs of Glasgow, and in favour of prioritising an untested and ultimately highly problematic policy for achieving economic growth through the development of newer, lighter industries through inward investment in central Scotland as a whole, *primarily outside of Glasgow*^{cviii}. This was to see the designation of a further three New Towns rather more distant from Glasgow (Livingston, Irvine and Glenrothes) for the purpose of receiving both population and industrial 'overspill' from the city, and the official designation of the city itself as 'declining' - both in terms of its population and its staple, heavy industries. The city's skilled labour was to be 'redeployed' to these New Towns and other overspill reception settlements, which were also to be the priority, not just for economic investment, but also for the wider investments in infrastructure and amenities which were seen to be required to achieve the wider 'modernisation' agenda and attract inward investment. Glasgow was no longer the priority focus of the plan, and indeed the plan required selective removal of population on a mass scale. Particular sections of the city's population (generally younger, skilled workers, in employment, and often with

^{cvii} The Clyde Valley Regional Plan is also known as the Abercrombie Plan, and is perhaps best known in the context of the competing visions for the city between that plan and the Bruce Plan. The latter (properly known as the 1945 Glasgow Development Plan) argued for comprehensive redevelopment of the City of Glasgow and the rehousing of the existing population (c. 1m) at much higher densities within city boundaries. In contrast, the Abercrombie Plan, argued for large-scale dispersal, including the rehousing of 250,000–300,000 Glaswegians outside the city, especially in New Towns. NB This particular context (Bruce vs. Abercrombie) is a different, earlier, issue to that discussed here, which concerns the adoption (and the effects of the adoption) of aspects of the plan from the late 1950s onwards.

^{cviii} The economic plan for Central Scotland had its roots, therefore, in the Clyde Valley Regional Plan. However, the different focus from the late 1950s onwards on prioritising economic growth away from the city was formalised in, first, the 1961 Report of the Committee of Inquiry into the Scottish Economy (often referred to as *The Toothill Report*), produced by the Scottish Council (Development and Industry)¹⁵⁵, and then later in the 1963 White Paper, Central Scotland: A Plan for Development and Growth¹⁵⁶. The latter document included a pull-out map which very clearly demonstrated how development and growth was to be pursued away from the 'declining' city of Glasgow.

families) were 'redeployed' to New Towns and other settlements under formal overspill arrangements, or moved there of their own accord, through 'voluntary overspill'. This policy is referred to in some of the archive material as "skimming the cream of Glasgow".

This was different to what happened in Liverpool with regard to the two New Towns built to deal with that city's overspill, Skelmersdale and Runcorn, and ongoing research points toward this as being indicative of some significant differences in the timing, conception and implementation of regional policy in north-west England as opposed to Central Scotland.

Importantly, the new research demonstrates in some detail how this policy agenda was pursued through the 1960s and 1970s *despite growing concern and awareness around the consequences (socioeconomic and ultimately also health-related) for the city*. For example, the new research quotes Labour MP Hugh Brown in the mid-1960s expressing his concern about 'overspill' in the House of Commons:

"it is true that today we are getting rid of some of our best tenants and are leaving ourselves with this gap, and we are losing the capacity for leadership in the very communities which are creating the social problems [emphasis added]"¹⁵³

By 1971, a review of overspill policy within the Scottish Office (entitled, significantly, 'The Glasgow Crisis') was warning that:

"Glasgow is in a socially... [and] economically dangerous position. The position is becoming worse because, although the rate of population reduction... is acceptable, *the manner of it is destined within a decade or so to produce a seriously unbalanced population with a very high proportion of the old, the very poor and the almost unemployable*... the above factors amount to a very powerful case for drastic action to reverse present trends within the city. [But] there is an immediate question as to how much room exists for manoeuvre [emphasis added]."

Thus it was recognised within the Scottish Office that its policy was having very serious consequences for the remaining residents of Glasgow, but these were pursued nevertheless, for many years to come, with only mildly mitigatory measures adopted for the city itself. In the later 1970s, a specific attempt by Labour Secretary of State for Scotland, Bruce Millan, to adjust spatial investment priorities to respond more proportionately to Glasgow's crisis failed. Glasgow entered the 1980s with its staple industries very seriously weakened by almost two decades of policy-aided decline, and searching for its own path for development and growth.

Glasgow's processes of urban change (Appendix A33) during these decades were more generally shaped by the priority given to the Scottish Office's regional policy objectives of modernisation and growth in *other parts of Central Scotland*. This provided the wider framework within which the national priorities for expenditure were set, and in which the city, notwithstanding its colossal problems, found itself 'deprioritised' not just in terms of economic development, but also in relation to the 'social' investment (in housing and amenities) to which it was inherently linked. There were massive amounts of public expenditure on the 'modernisation' plan for Central Scotland from the late 1950s^{cix}. But clearly Glasgow and its needs were not the priority to benefit from it. The pattern of development in Glasgow, and the problems it created, can be seen, at least in part, to derive from this set of circumstances – large quantities of poor quality housing, often built in remote and unattractive peripheral locations, and with a large emphasis on high rise, still with a tendency to be overcrowded, suffering from low levels of expenditure on repairs and other investment, and inhabited by a population affected by an adverse and policy-induced socio-demographic skew.

Two small, but nonetheless telling, pieces of evidence help to crystallise this perspective. First, a 1959 Glasgow Corporation booklet promoting to city residents the merits of leaving the city through 'overspill'. It posed the question of what such 'leavers' stood to gain, and provided a clear answer: a new home in a healthier environment with good amenities giving "the opportunity of a better life" (and perhaps a longer one). The barely implicit message was that these would be much less likely to be on offer for those who remained – which proved to be the case⁵¹⁵. Second, a mid-1970s discussion among civil servants within the Scottish Office as to the precise location of an event within Glasgow to mark the letting of the millionth new council house in post-war Scotland. The conclusion was that the priority should be to find a house and a development of which government – both local and central – need not feel ashamed. Unfortunately, the view was that this would not be an easy task⁵¹⁶.

Differences in political response to the 1980s

In the Galean model of population health, the impact of stressful events or processes on vulnerable city populations can be offset by population responses to these stressors and by the experience of protective events. Initial conclusions of a synthesis of the literature on local government policies being pursued in Glasgow and Liverpool during the 1970-90s lead to the suggestion that different responses across the three cities to the impact of the UK government's post-1979 policy agenda can be seen as having potentially important implications in terms of how they fit into this model. Specific policies impacting across cities like Liverpool, Glasgow and Manchester in this period included the government's forcing of the pace of deindustrialisation with resulting increases in unemployment, sharp reductions in financial support for council housing, and wider policies which impacted adversely on local government finance, trade union organisation and on those in receipt of social welfare payments. The key point here relates to differences in how local government and other agencies at the time responded to the challenges presented by those policies at city level. These differences were arguably most pronounced in relation to the cities' approaches to urban regeneration and in important aspects of the history and operation of local political and policy processes'^{177,179,180,182,183,517-528}

In the case of Glasgow, there were a number of agencies involved in the policy direction which was taken in this period – in particular the city-specific District Council, as well as the much wider Strathclyde Regional Council, and also the Scottish Development Agency (SDA),

^{cix} As Foster and Woolson note: "Unprecedented sums of money were made available to modernise the central belt of Scotland... the physical face of central Scotland began to undergo unimagined changes"⁵¹⁴.

created in the mid-1970s to seek to deal with the then-growing concerns about the problems of economic development affecting the nation. It should also be noted that the policy direction adopted at city level reflected both the exceptionally difficult circumstances facing the city in light of the maintenance of the main geographical priorities for economic development (i.e. away from Glasgow) which, as indicated earlier in this appendix, had been set by government in the early 1960s and reconfirmed in the later 1970s, and also in light of the election of the Conservative Government to Westminster in 1979. One effect of the latter was to re-orientate the activity of the SDA, which was heavily involved in the development of the city, along lines which reflected the 'neoliberal' approach to economic and social policy favoured by the Thatcher government. However, research – both contemporaneous and more recent – and also expert feedback sought for the purposes of this report, has highlighted the ways in which the city-level District Council came to take the lead in this period on the crucial issue of 'urban regeneration' in particular^{173,176-179,529-532}.

As is described in detail below, the contrast between Glasgow and Liverpool in terms of local responses to UK policy in this period is striking. In Manchester, the approach by local government was somewhat intermediate between those of Liverpool and Glasgow. There was resistance to co-operation with the government until 1987 when, faced with the third consecutive Westminster electoral victory for the Conservative Party, it reversed its previous policy of non-co-operation to work with the Government to promote a neoliberal model of urban renewal and regeneration^{140,171,172}. In this way, Manchester was temporarily protected from the impact of the UK government's policy agenda, and other aspects of the latter were delayed for the greater part of the decade.

In Liverpool, following years of (principally) Liberal control, local politics in the 1980s was characterised by the emergence of a Labour-controlled authority - but in particular characterised by the rise of the so-called 'Militant' group within that ruling Labour Party, and its (and by association, the city's) subsequent highly overt confrontation with the UK Conservative government of the time. Described in great detail by various commentators^{137,170,173,174}, the relevance of this to this synthesis is that the city council's actions, in challenging key aspects of the UK government's 'political attack' and its implications in terms of great hardship for many in the city, and in committing itself to its own large-scale programme of council house building and regeneration focused on the provision of public amenities, conferred protective effects on the city's population. Importantly, it entailed considerable mobilisation and political participation among Liverpool's residents – an opportunity to experience collective action, political voice and, with that, feelings of community power and efficacy. This was a vibrant process, and had further impacts in terms of local government prioritising and addressing at least some of the important issues of the day for the majority of working class Liverpudlians - addressing poverty and providing new, affordable council housing and wider public services/amenities.

This contrasts with the situation in Glasgow at the time. Although Labour was also the ruling party in the city, it had been more or less continuously in power for such a long period of time (from the early 1930s, with only brief periods out of office between 1948-51, 1968-71 and 1977-1980) that it was very much more 'the establishment', politically adapted to working in a conciliatory manner with central government and ill-disposed to challenging it

overtly through politicisation of the city population – and even more so in a context where it was seeking to attract investors to a city with an unwanted historic 'Red Clydeside' reputation. Notwithstanding its Labour majority throughout the 1980s, then, Glasgow's local government rejected the pathway of overt confrontation with central government and took a rather more 'conciliatory' approach. This did not amount to 'compliance', for the Council used creative accounting and was willing to breach spending guidelines, as were other local authorities, to try to protect jobs and services. However, the council's 'defence' of the city did not involve the popular mobilisation and participation seen in Liverpool, and from a 'social determinants of health' perspective, concerned with issues of power, control and alienation, this would seem to be a notable difference.

The preceding should not be taken as a suggestion that Liverpool was a model of what was best in local government in the 1980s. What we are interested in here are differences in the historical experience of the cities which are likely to be relevant, from a social determinants of health perspective and in light of the 'vulnerability' model of population health outlined earlier, to the excess mortality phenomenon under consideration. The "markedly different postures" struck by Glasgow and Liverpool in relation to the central government policy agenda of the 1980s are clearly highly relevant in this context.

A further contrast is that the Council in Glasgow in this period also, in significant respects, experimented and innovated with neoliberal policy measures – drawing on the experience of cities in the northern states of the USA such as Baltimore and Pittsburgh, and taking inspiration from the Mellon Bank and the associated Allegheny Conference^{,180,533}. In this, key figures in the city were guided by the apothegm, which contemporary commentators saw as agreed by key (though not necessarily all) civic and business leaders, that "what's good for business is good for Glasgow". These were seen at the time as quite "astonishing" developments in such a "solidly Labour City"^{173,176}. As indicated above, the developments were initiated in a context in which the Scottish Office had (in the later 1970s) decided against adjusting area-based priorities for development and investment in Scotland to take account of the accumulated evidence of the damage being done to the city by the regional priorities which had prevailed for the previous two decades. However, while in this light the city's anxiety to find its own path towards investment and development is understandable, the consequence of the path the city took was that within a matter of years commentators were identifying Glasgow as a 'dual city' with 'dual urban policy' - high budget, high profile retail and property development in the city centre (Merchant City, Buchanan Street) led by a 'growth coalition' in which the city council and the Scottish Development Agency played a lead role in subsidising private developments, and much lower-resourced and very limited mitigation and management of poverty and intensifying social crisis in the city's poorer areas, including its increasingly troubled peripheral estates ¹⁷⁷⁻¹⁸⁴. By the mid-1980s commentators were warning of the emergence of an "increasingly alienated underclass" benefiting little from Glasgow's city centre regeneration, with the latter tending to produce "islands of prosperity surrounded by a sea of despair", in which "basic urban needs are neglected". The contrast with Liverpool's urban regeneration programme in the same period is marked.

More generally, however, the contrast between the cities in terms of the effects on the local population can be neatly captured in the words of different commentators. In Liverpool:

"Labour's radical rhetoric *struck a chord with despondent voters*. Support for the council reflected *a groundswell of popular opinion against the government* [emphasis added]."

"There is no doubt at all that the politics of the financial crisis *electrified the people* and *alerted them to its problems* in a way that was simply never there before. Everyone knew about it and *everyone had an opinion* [emphasis added]".

On the other hand, Jean McFadden, a councillor and later leader of Glasgow Council wrote in 1982 about how the electoral defeat of Labour in the city in 1977 led to an awareness of "the real sense of alienation, frustration, disaffection and cynicism throughout so many communities in Glasgow". By the later 1980s Glasgow, as Robin Boyle put it, had seen "a radical departure from the substance and style of urban planning policy" from that time, but one quite different from that seen in Manchester and especially Liverpool, in that it had already involved large scale public subsidy for private development. In this process, as he saw it:

"Profit becomes the goal: the original, much wider, objectives covering the economic and social condition of the city begin to fade ... an explicit acceptance of commercial objectives can effectively redirect public policies and programmes away from areas most in need, further reducing the resources available to communities that have no part to play in the investment patterns of private developers".

Mark Boyle and colleagues were later to sum up the experience as follows:

"And so the city [centre] was gradually turned from an area of blight to one of reinvestment by property capital, retailers, hoteliers and leisure capital. Much of this investment came from finance capital, especially pension funds and insurance companies based in London and abroad. ... What scarce public funds were available were being transferred from the provision of welfare relief to promoting speculative high-profile marketing projects which were doing little to arrest social and spatial inequalities in the city".

In this context, as Keating was to put it in 1988: "the peripheral areas of Glasgow are to some extent politically disarmed. Nor is there necessarily a danger of serious danger of social disorder as, geographically isolated, alienated youth would have nothing to attack but their neighbours. Yet there must be dangers that in the long run in a degree of alienation affecting the social cohesion of the country [or at least the city] as a whole".

The new research conducted recently usefully summarises the differences democratisation and politicisation (which may help to explain some of the differences in social capital) as follows:

"In Liverpool the actions of the council in the mid-1980s were, for all the controversy associated with them, genuinely popular and apparently invigorating; even for those who disagreed with them, there was a meaningful discussion about the needs of the city, the damage being done by central government and how best to address all of that. In Glasgow, however, there was little scope for that, and in fact there seems to have been an on-going process of managing and manipulating communities in ways which compounded their problems and led, perhaps, to even more damaging outcomes – breaking down fragile bonds of community and turning frustration into something rather more dangerous".

Finally, the impact of the differing approaches of local government in the 1980s seems to have left a legacy still evident in 2012. For example, evidence from the three-city survey indicated a more 'politicised' Liverpool sample (in terms of, for example, having been more engaged in anti-government demonstrations in the 1980s^{cx}).

Taken altogether, alongside the emergence of a 'democratic deficit' and distinctive national reaction (tending towards despair and despondency) to the perceived imposition of neoliberalism in Scotland by a UK government lacking legitimacy north of the border, there is substantial evidence of direct and indirect pathways through which negative health impacts could reasonably be explained.

Conclusion

There is substantial evidence that the UK pursued a neoliberal policy agenda in the years after 1979 in a way that set it apart from the rest of Europe at that time and that this was a causal factor in several of the mortality phenomena that began to develop in that period and thereafter. Taken as a whole, the evidence of Glasgow being a city more vulnerable to, and overall worse affected by, the damaging impacts of that policy agenda, together with the above detailed evidence as to how this vulnerability was created in the post-war decades, and also the further evidence of different city-level responses to the 'stressor' of 'political attack' and their differing implications in terms of city population access to protective experiences and salutary resources, cumulatively makes for a substantial evidence base, linked to a persuasive 'vulnerability' theoretical model, to facilitate a significant part of the explanation of Glasgow's lagging health outcomes in comparison with Liverpool and Manchester.

^{cx} Respondents were asked whether or not in the 1980s they had attended any public demonstrations about government policies (with demonstrations defined as 'public rallies, meetings, strike actions or other similar events'). In Glasgow and Manchester, only 5% of respondents who had lived through the 1980s reported that they had attended demonstrations of this type. However, the equivalent figure for Liverpool was 14%. Other analyses showed the Liverpool sample to have stronger (more negative) views on the current UK government. For example, 50% agreed or strongly agreed that the UK government was 'undermining' their city: the equivalent figures for Glasgow and Manchester respectively were 30% and 28%.
Assessment of evidence of causality

1) General assessment of likelihood of causality in terms of Bradford Hill criteria

Bradford Hill's	Assessment ^{cxi}	Comments	
cificina for causancy			
Strength of	Y		
association			
Temporality	Y		
Consistency	Y		
Specificity	Y	There are reviews showing that neoliberal politics	
Biological gradient	Y	contexts over time.	
Plausibility	Y		
Coherence	Y		
Experiment	Y		
Analogy	Y		

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales

Comparison	Assessment	Comments	
Is the hypothesised causal factor worse in Scotland?	Y	There is evidence that there was greater	
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	vulnerability to, and greater impacts from, neoliberal approaches.	

^{cxi} Note: '-' indicates no evidence to demonstrate or refute the consideration; 'U' indicates there is uncertainty around whether the evidence supports this criterion; 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester

Comparison	Assessment	Comments	
Is the hypothesised causal factor worse in Glasgow?	Y	There is evidence that there was greater	
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	yulnerability to, and greater impacts from, neoliberal approaches.	

A29 Premature and low birthweight births

Description of hypothesis

There are proportionally more premature and low birthweight babies born in Scotland and Glasgow compared with England & Wales, and this influences the higher rates of Scottish mortality.

Rationale

Premature and babies of low birthweight are known to be associated with greater risk of a range of adverse health outcomes (e.g. diabetes, obesity, heart disease⁵³⁴) in later life.

Links to other hypotheses

There are no clear, direct, links to other hypotheses.

Evidence overview

The weight of evidence suggests that in recent years at least, rates of pre-term and low birthweight babies have <u>not</u> been notably higher in Scotland compared with the rest of the UK. However, there are gaps in some historical trends. Even where some evidence exists, the data have not been adjusted for deprivation – and both rates of pre-term and low birthweight births are highly socially patterned, with much higher rates observed among more deprived populations⁵³⁵.

a) Low birthweight babies

ONS and ISD Scotland data summarised on The Poverty Site⁵³⁶ for the period 2007-09 show that the percentage of babies that were of low birthweight (using the standard definition of <2,500g) in Scotland (approximately 7%) was very similar to many other parts of Britain including North West and North East England, and Wales.

Similar ONS and ISD Scotland data are included within Scottish HfA database^{537,cxii}. These are for a longer period of time but only allow comparison of Scotland with the whole of the UK. These do not suggest that the difference between Scotland and the UK as a whole has varied between the 1980s and mid-2000s (although Scotland's figures improved to a greater degree in the mid to late 2000s) (Figure A29.1)

^{cxii} These are shown within the Scottish and European HfA database as % of all live births which are 2,500g or more. The data shown in Figure A29.1 are derived from those figures (i.e. based on the remaining percentage figure).





Regional analyses for the period 2004-08 compared low birthweight babies (as % of all live births) in West Central Scotland with 11 other post-industrial regions in Europe. As Figure A29.2 shows, the figure for WCS for the period was 7.8% of all live births, which was at the upper end of the spectrum, along with regions of Belgium, Germany and the Netherlands. However, it was also very similar to the figure for Merseyside (7.6%) (although higher than that of Northern Ireland (6%)).





b) Pre-term births

Recent data show the percentage of babies born prematurely (defined as <37 weeks gestation) has not been higher in Scotland than in England. Data from ONS and ISD Scotland show the percentage of all live births classed as premature in the period 2011-2013 to have been 7.1% in Scotland and 7.2% in England. There is some suggestion that the figure may have been slightly higher in earlier years (e.g. in the period 2006-08 it was 7.5% in Scotland and 7.1% in England & Wales); however, the data for England are only available from 2006 onwards. These data are presented in Figure A29.3.

Trend data are similarly limited for sub-national comparisons – indeed they are more limited as Scottish data are routinely published at NHS board, rather than local authority, level. As Figure A29.4 shows, generally they suggest that the percentage of premature babies (here defined very slightly differently as 24-36 weeks gestation) in the period 2006-2012 has been lowest in Manchester and highest in Liverpool. Figures for all three areas have tended to be higher than the respective national figures: this is to be expected given the social patterning associated with these data, and the fact that the three cities are the most deprived in their respective countries.









Conclusion

There is no compelling evidence to suggest that Scotland and the Glasgow conurbation have, or have had historically, higher rates of pre-term or low birthweight births compared with other parts of the UK. Thus this topic is unlikely to be relevant to the issue of Scottish excess mortality.

Assessment of evidence of causality

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

Bradford Hill's	Assessment ^{cxiii}	Comments
criteria for causality		
Strength of association	Y	
Temporality	Y	
Consistency	Y	
Specificity	Y	Prematurity and low birthweight is a known causal
Biological gradient	Y	outcomes.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	N	For the short time periods available, there are no
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	these outcomes for Scotland compared with England & Wales.

^{cxiii} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	N	For the short time periods available, there are no substantial differences in
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	these outcomes for Glasgow compared with Liverpool and Manchester.

A30 Quality of external physical environment: land contamination

Description of hypothesis

It has been proposed that a greater exposure to contaminated land might be responsible for the mortality phenomena in the Scottish populations, particularly in Glasgow.

Rationale

Land can be contaminated by a wide range of toxins, and many of these are known to be harmful to human health (e.g. heavy metals and their compounds). The impact of these toxins on health is, however, dependent on whether humans are exposed to them (e.g. through drinking water, particulates in the air or through ingestion of plants grown in contaminated land) and on their bioavailability (i.e. the compounds and forms in which they are present).

Links to other hypotheses

This hypothesis links closely to the air pollution, quality of the physical environment and deindustrialisation hypotheses.

Evidence overview

The evidence on the impact of contaminated land on health has been reviewed extensively, but remains patchy and contested⁵³⁸. Although there is little evidence for 'widespread' health impacts, there is some contested evidence to suggest that residential proximity to landfill sites is associated with a small excess in congenital abnormalities and low birth weight, but the evidence for an association with cancer is unclear. Several aspects of self-reported health are worse in proximity to such sites and this may be linked to higher exposure to noise and odour. The evidence base for specific exposure to particular contaminants is patchy and often of low quality (e.g. in relation to dioxins, asbestos, hexachlorobutadience, etc). There is clearer evidence of an association between high levels cadmium in soil, consumption in food (through crops) and itai-itai disease (a rare renal and bone disease). There have also been examples of negative health impacts of the physical environments associated with contamination (e.g. unstable spoil tips, mine shafts, etc).

Glasgow is recognised to have a large number of areas of land contaminated with chromium⁵³⁹, although the health risks from the levels of contamination and the routes of exposure in this context (i.e. airborne and through soil rather than groundwater) are thought to be low⁵⁴⁰⁻⁵⁴³. The metal content of the soil around Glasgow is high and seems to be higher in more deprived areas.⁵⁴⁴ There has been investigation of the association between landfill sites in Scotland and foetal abnormalities which have found no elevation in risk^{545 546}, and between soil contamination (particularly nickel) and respiratory ill-health which have found a positive association even after adjustment for deprivation.

It has also been suggested that the population of Glasgow may still be exposed to substantial 'natural' seepages of methane, carbon dioxide and oil (but not hydrogen sulphide) through the extensive coal and iron ore mining that occurred in the area

historically, although the health impacts of these specific exposures have not been investigated ^{547,548}. Radon exposure is low in Glasgow, intermediate in some surrounding areas of Clydeside and high in some (mainly rural parts) of North East Scotland ⁵⁴⁹.

No data were identified to facilitate comparisons of exposure to specific contaminants (except radon), or contaminated land in general, between our comparison populations. The most relevant identified data compared recent trends in brownfield site availability in Scotland and England (but which does not provide data on whether or not this is contaminated nor on the proximity to residential populations). It estimates that some 5% of developed land in England was brownfield, and 7% of developed land within Glasgow, but notes importantly that this varies substantially within areas (e.g. in response to land demand (as in London the percentage of developed land which is brownfield was very low)) and over time (with 'new' brownfield sites accounting for the majority of the 'stock')⁵⁵⁰. Another commentary highlighted the difficulties in making comparisons of the quantity of contaminated land sites between Scotland and England as reported by the respective environment agencies⁵⁵¹.

Conclusion

There are little data available to assess whether Scotland and Glasgow have been more exposed to contaminated land than other areas, although in the case of Glasgow this seems likely given the particular industries which were common in the city in the past. However, extensive study has been undertaken to explore whether there are links between these exposures and health outcomes, and these have provided reassurance that any impacts have been small or limited to odour and noise (in relation to landfill). The health consequences that have been most frequently associated with contaminated land elsewhere (although that evidence base is unclear and disputed) are not those causes which are responsible for the excess mortality phenomena. It is therefore unlikely that contaminated land plays an important role.

Assessment of evidence of causality

Bradford Hill's criteria for causality	Assessment ^{cxiv}	Comments
Strength of association	U	
Temporality	U	
Consistency	U	
Specificity	U	The available evidence around the importance of contaminated land in adverse health outcomes is
Biological gradient	U	contested and often of poor quality. The outcomes most cited in relation to contaminated land are not those most prominent in the excess
Plausibility	Y	most prominent in the excess.
Coherence	Y	
Experiment	U	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	U	There are no comparative data available on
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	land. However, the causes of death hypothesised to be related to this exposure are not an important component of the excess.

^{cxiv} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	U	Although there are no comparative data available on exposure, the
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	available evidence suggests that contaminated land is not responsible for a substantial burden of ill- health in Glasgow.

A31 Quality of external physical environment: vacant & derelict land

Description of hypothesis

Urban areas of Scotland are characterised by a more negative physical environment than urban areas in the rest of the UK – specifically in relation to high levels of vacant and derelict land – and this impacts on health status among the Scottish population. Neighbourhoods may differ in terms of this potentially important health-related environmental aspect even when they share similar socioeconomic characteristics (e.g. levels of wealth, poverty, material deprivation), and these differences have not been detected, and thus taken into account, in the analyses of Scottish excess mortality to date.

Rationale

There is a vast amount of research literature linking aspects of the physical environment to population health. This includes the direct effects on health such as air quality, pollution, traffic, housing, as well as more indirect effects in relation to walkability, green space, access to services and quality of neighbourhood²⁰⁷⁻²¹². Recent analyses in England have shown strong area-level associations between negative land use characteristics (so-called brownfield sites) and premature mortality⁵⁵². Differences in other, related, aspects of land use between Scotland and England would, therefore, be potentially relevant to the issue of excess Scottish mortality.

Links to other hypotheses

Inadequate measurement of deprivation; land contamination; climate; nature and scale of urban change; deindustrialisation.

Evidence overview

Analyses of land use survey data showed that in 2011, 60% of the population of Glasgow lived within 500m of vacant or derelict land, a potentially negative environmental characteristic. Across the city's 'neighbourhoods'^{cxv} this figure ranged from 0% of the population of Carmunnock (a relatively affluent village of approximately 1,500 people which lies within the city's boundary on the south side) to 100% of the population of Ruchill & Possilpark (a relatively deprived area of around 10,000 people) and Parkhead & Dalmarnock (another deprived area in the city with a population of approximately 7,000 people)⁵⁵³. Although there are no directly comparable data for Liverpool and Manchester, similar land use information has been used to estimate that overall 6.8% of Glasgow's land is classed as being vacant or derelict, more than the equivalent figure for Liverpool (5.4%) and over three times the figure for Manchester (2.2%)^{203,204} (Figure A31.1)^{cxvi}. Other analyses suggest similar

^{cxv} There are 56 such 'neighbourhoods' across the city of Glasgow. Previously known as housing forum areas, and created by Glasgow City Council in consultation with housing associations, these have been used extensively in analyses of health and wellbeing in the city, including within the Understanding Glasgow indicators project (<u>www.understandingglasgow.com</u>).

^{cxvi} These data are derived from two different sources, and thus caution should be taken in interpreting differences between the cities. However, the definitions used in both sources are broadly comparable. Numerator data for Glasgow come from the 2013 Scottish Vacant & Derelict Land

differences, particularly between Glasgow and Liverpool^{cxvii}. It is highly likely, therefore, that a higher percentage of Glasgow's population live within proximity of this type of environment compared with the populations of these two English cities.

It is possible that the proportionally greater levels of vacant land in Glasgow relate to the greater scale of urban change (demolition etc) experienced by the city (and highlighted elsewhere in this Appendix, and in the main part of the report). However, further research would be required to verify this.





Survey. Two categories are included: vacant land (defined as 'land which is unused for the purposes for which it is held and is viewed as an appropriate site for development. This land must either have had prior development on it or preparatory work has taken place in anticipation of future development'); and derelict land (and buildings) (defined as 'land which has been so damaged by development, that it is incapable of development for beneficial use without rehabilitation. In addition the land must currently not be used for the purpose for which it is held or a use acceptable in the local plan'). English numerator data come from National Land Use Database of Previously-Developed Land (NLUD-PDL) (supplied by ONS). This source provides 'information on previously developed land that may be available for re-development (also known as "brownfield" land)' and, again, two categories were included in the analysis shown here: previously developed land which is now vacant; and derelict land and buildings. All data were measured in hectares and are shown as a percentage of the total land size of each local authority area.

^{cxvii} For example, analysis of the European Environment Agency's (EEA) Urban Atlas data²⁰⁶ (aerial photography based land-use maps from 2005-08) suggests that a higher percentage of land within Glasgow City is classed as 'land without current use' compared with Liverpool, and this is also true in comparison of the most deprived neighbourhoods in both cities.

Conclusion

It is at least plausible that differences in the physical environment (potentially acting as 'unmeasured' differences in the experience of social deprivation) between Glasgow, Liverpool and Manchester, and between other areas of Scotland compared with England & Wales, contribute, via particular causal pathways linked to aspects of mental and physical health, to levels of excess mortality in Scotland. The size of that contribution, however, is difficult to determine.

Assessment of evidence of causality

Bradford Hill's	Assessment ^{cxviii}	Comments
criteria for		
causality		
Strength of	Y	
association		
Temporality	U	
Consistency	Y	
Specificity	Y	There is evidence that living near derelict land is
Biological	Y	detrimental to nealth, although part of that may be
gradient		slum clearance which generates this dereliction.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

^{cxviii} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	U	Data for all urban areas in England & Wales and Scotland have not been
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	compared.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	Y	There is more derelict and vacant land in Glasgow than in Liverpool and
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	Manchester. Trend data on this are not readily available.

A32 Scale of deindustrialisation

Description of hypothesis

It has frequently been proposed that Scotland, but in particular West Central Scotland and Glasgow, has experienced more severe levels of deindustrialisation which, in turn, has impacted on mortality rates.

Rationale

The link between the effects of deindustrialisation (for example unemployment, poverty, deskilling and role redefinition) and population health is well understood, and many postindustrial areas are characterised by adverse social, economic and health outcomes^{120,121,554-} ⁵⁵⁷. Post-industrial decline has, therefore, been cited as an important underlying cause of high mortality in Scotland as a whole, and in West Central Scotland (WCS), Scotland's most deindustrialised region, in particular¹⁰⁻¹³.

Links to other hypotheses

Inadequate measurement of deprivation; labour market/nature of employment.

Evidence overview

National comparisons

Analyses of historic census data show that between 1931 and 2001 the percentage of total employment that was categorised as industrial employment in each year in both Scotland and England was similar (with a similar decline observed). The figures decreased from 48% (Scotland) and 46% (England) in 1931, to 34% and 37% respectively in 1981, and to 23% and 24% respectively in 2001^{134,558} (Figure A32.1). The corresponding percentage decreases over the period were 51% (Scotland) and 48% (England). However, calculated slightly differently – as the loss of industrial jobs expressed in relation to the size of the industrial employment base in 1931 – Scotland fared worse, with a total industrial employment loss over the period of 47% compared with 30% in England. However, restricting the analysis to the period 1971-2001 shows less difference between Scotland (45%) and England (43%).

Figure A32.1



Regional comparisons

A series of analyses published in 2008, 2011 and 2014 compared West Central Scotland (WCS) with other post-industrial regions of the UK and mainland Europe in terms of their experiences of deindustrialisation, health (mortality), and key health determinants including socioeconomic factors^{18-20,498}. Analyses of the percentage of the total employed population working in industry over an approximate 35-year period (until 2005) showed a greater decline in WCS (58%) compared with the other regions (e.g. approximately 50-52% in Merseyside, The Ruhr (Germany) and Wallonia (Belgium)). Analysing the severity of deindustrialisation in terms of the total loss of industrial jobs over the period (relative to the size of the earlier employment base) showed the greatest impact of deindustrialisation was in Merseyside (63% decrease) and WCS (62%), followed by regions such as Katowice (Poland) (55%), The Ruhr (54%) and the ex-coalfield areas of South Wales (51%). More generally, a range of analyses showed that mortality rates were higher, and improving more slowly, in WCS compared with the other deindustrialised regions, but also suggested that WCS's socioeconomic profile was superior to that of the majority of those other areas. Further in-depth analyses of the political, economic and historical context in a subset of the regions (WCS and four regions in Germany, France, Poland and the Czech Republic) highlighted a number of factors which arguably placed WCS at a relative disadvantage, and made deindustrialisation more damaging for the region. These included disadvantageous economic and industrial policies, less autonomy in terms of regional power, and differences in levels of social protection. However, these disadvantages were shared with other UK regions (e.g. Merseyside).

City comparisons

Identical census-based analyses to those undertaken for Scotland and England (described above) were also carried out for the cities of Glasgow, Liverpool and Manchester for the same period (1931-2001). These showed virtually no difference between the industrial employment base in each period and, therefore, the overall loss experienced over time. All three cities experienced profound industrial employment decline. For example between 1931 and 2001 levels of industrial employment decreased by between approximately 83% (Glasgow and Liverpool) and 86% (Manchester). These data (for the three cities, plus also Scotland and England) are presented in Figures A32.2-A32.4.



Figure A32.2

Figure A32.3



Figure A32.4



Conclusion

There is some evidence that Scotland as a whole suffered higher levels of deindustrialisation compared with England and Wales (although it is likely that this is confounded by higher deprivation), and that in European terms, the region of West Central Scotland was among

the worst affected by post-industrial decline. At the city level, however, the data suggest the experiences of deindustrialisation (at least in terms of total numbers of jobs lost) have been very similar in Glasgow, Liverpool and Manchester. Given that the experience of deindustrialisation coincides with the emergence of the mortality phenomena, and the well evidenced links to health outcomes across time and place, it is likely to be part of the causal pathway in Scotland and Glasgow – but in interaction (as an effect modifier) with other factors in Glasgow's case, given that the degree of exposure is very similar to Liverpool and Manchester.

Assessment of evidence of causality

Bradford Hill's	Assessment ^{cxix}	Comments
criteria for causality		
Strength of	Y	
association		
Temporality	Y	
Consistency	Y	
Specificity	Y	The links between deindustrialisation, employment,
Biological gradient	Y	understood.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

^{cxix} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	Y	Scotland deindustrialised to a greater extent than England & Wales, although
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	the effect of this on mortality is likely to be mediated by greater deprivation and thereby accounted for in the mortality modelling.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow? Assuming a difference in exposure, would this have occurred prior to the outcome?	N Y	Glasgow deindustrialised to a similar extent to Liverpool and Manchester. The analyses of deindustrialising areas across Europe indicate that this alone is unlikely to explain the excess compared with those areas, but it is likely to be part of the explanation in combination with other factors.

A33 Scale and nature of post-war urban change

Description of hypothesis

It has been suggested that the scale of post-war urban change (demolition, building of poor quality housing estates, movement of population, breaking up of communities) may have been greater in Glasgow than in the comparator cities of Liverpool and Manchester, and this may, through particular influences, account for some of the excess levels of mortality observed in the city.

Rationale

In common with other UK cities, Glasgow, Liverpool and Manchester experienced large-scale urban change in the post-war period (c. 1945-1980). This was characterised by slum clearance and the relocation of communities to public housing estates, overspill developments in surrounding areas, high-rise flats and New Towns. It therefore had the potential to influence population health in several ways, especially through the important social determinants of housing, living conditions and social and community networks.

Links to other hypotheses

Inadequate measurement of deprivation; spatial patterning of deprivation; housing quality and allocation; quality of external physical environment; social capital; political influences & vulnerability.

Evidence overview

This hypothesis was investigated recently by means of a literature review and analyses of routine data. The literature review identified both similarities and differences between the cities in terms of the scale of urban change experienced in the period. The similarities included: the immediate, and pressing, housing challenges in 1945 (shortages, overcrowding etc), the types of policies subsequently adopted to address these challenges, and important shortcomings and failures in the conception, design and implementation of many aspects of urban change. Potentially important differences between the cities included: the development of 'outer estates' – council house building outside city boundaries – a policy only pursued in Liverpool and Manchester; a focus on building large (within city boundaries) peripheral estates within Glasgow but to a much lesser extent in Liverpool or Manchester; a much greater emphasis on high-rise development in Glasgow; a larger, more ambitious, and – importantly – 'socially selective' New Towns programme in Scotland; and differences in institutional arrangements between Scotland and England (e.g. the role of the Scottish Office, which had no direct equivalent in England).

Analyses of routine data highlighted some key differences between the cities. These included:

• As discussed elsewhere in this report, the scale of overcrowding in Glasgow (and, importantly, in other Scottish cities as well) in the post-war period was much greater

than in Liverpool and Manchester (Figure A33.1). Although it improved in absolute terms, it remained higher in relative terms throughout the rest of the 20th century.

- Slum clearance (Figure A33.2) and construction of local authority housing (Figure A33.3) (especially high-rise dwellings (Figure A33.4)) was undertaken on a greater scale in Glasgow compared with the English cities. The sizes of newly built peripheral estates were also larger in the Scottish city: four of the six largest estates across all three cities were built on the periphery of Glasgow (Drumchapel, Easterhouse, Pollok and Castlemilk). (The second largest estate (Kirby) was built just beyond Liverpool's city boundaries (Figure A33.3)).
- The difference in the numbers of high rise dwellings is potentially relevant because of the known links between living in such housing and negative impacts on mental health¹⁶⁶⁻¹⁶⁸.
- Crucially, investment in local authority housing repairs and maintenance in Glasgow was lower than in Manchester and (to some extent) than in Liverpool throughout the 1960s and 1970s this despite the Scottish city having an older stock of local authority housing. This is shown in Figure A33.5.
- Population movement: within city boundaries there was more movement of the population from inner areas to large peripheral estates in Glasgow and Manchester; Liverpool did this on a smaller scale, but also expanded its more affluent suburbs. Outside city boundaries, Glasgow saw more of its population move to New Towns or leave the West Central Scotland region entirely; Liverpool and Manchester retained more of their population within their own wider regions (i.e. Merseyside and Greater Manchester).
- There were potentially important differences in the composition of the New Towns which were built to deal with overspill from the cities between the 1940s and 1970s. As discussed elsewhere in this report, analysis of census data shows that by the 1980s East Kilbride and Cumbernauld had a noticeably 'better' (less socioeconomically deprived) profile than Glasgow as a whole, reflecting the more socially selective nature of the process in relation to the Scottish city. Fewer differences were evident when comparing Liverpool with its relevant New Towns, Skelmersdale and Runcorn.





Figure A33.2



Figure A33.3



Figure A33.4



Figure A33.5



Conclusion

Some features of planned urban change (e.g. demolitions, construction of council housing, the size of peripheral estates) occurred on a greater scale in Glasgow than in Liverpool and Manchester, but this was not true of all aspects. However, the testing of this hypothesis led to the view that *differences in the specific way urban change unfolded in Glasgow* (e.g. the more socially selective nature of the New Towns programme in Scotland, lower levels of investment in the local authority housing stock coupled with persistently higher levels of overcrowding and more high-rise dwellings) appear to be just as important as differences in scale. Certainly, Glasgow appears unique particularly in the relative scale of overcrowding that confronted it after the second world war, but also in the specific way in which change came about in the city.

Linking the research on this hypothesis to that conducted on the impact of regional policy on the city (Appendix A28) helps to contextualise these findings further. For it becomes apparent that the process of urban change in Glasgow was very closely connected to the implementation, from the later 1950s, of central government's long-term plan for economic growth and 'modernisation' in Central Scotland, away from Glasgow. This provided the wider framework within which the national priorities for expenditure were set and in which the city, notwithstanding its colossal problems, found itself deprioritised not just for economic development, but also for the 'social' investment (in housing and amenities) to which it was inherently linked. There were massive amounts of public expenditure on the

'modernisation' plan for Central Scotland from the late 1950s^{cxx}. But clearly Glasgow and its needs were not the priority to benefit from it. The pattern of development in Glasgow, and the problems it created, can be seen, at least in part, to derive from this set of circumstances – large quantities of poor quality housing, often built in remote and unattractive peripheral locations, and with a large emphasis on high-rise, still with a tendency to be overcrowded, suffering from low levels of expenditure on repairs and other investment, and inhabited by a population affected by an adverse and policy-induced socio-demographic skew.

It is highly plausible, then, that the precise nature of the processes of urban change in Glasgow in the post-war decades combined to increase the relative vulnerability of Glasgow's population to subsequent stressful impacts (such as further rapid deindustrialisation, unemployment and poverty, after 1979, together with reduced central funding for council housing, the 'right to buy' council housing, and, more recently, changes to local authority boundaries – all of which meant Glasgow had to deal with a greater level of need on the basis of inadequate budgets) and to consequent adverse health outcomes. In this light, the processes of urban change in the post-war decades would be seen to be making a contribution (albeit one that is not easily quantifiable in the strictest epidemiological terms) to the city's excess levels of poor health.

^{cxx} As described elsewhere in this Appendix, Foster and Woolson noted that: "Unprecedented sums of money were made available to modernise the central belt of Scotland... the physical face of central Scotland began to undergo unimagined changes"⁵¹⁴.

Assessment of evidence of causality

Bradford Hill's criteria for causality	Assessment ^{cxxi}	Comments
Strength of association	Y	
Temporality	Y	
Consistency	Y	The literature review described in this appendix
Specificity	Y	highlights evidence that post-war urban change had the
Biological gradient	Y	especially through the important social determinants of housing, living conditions and social and community
Plausibility	Y	networks.
Coherence	Y	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	U	We have not examined the data specifically for Scotland in comparison
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	with England & Wales.

^{cxxi} Note: '-' indicates no evidence to demonstrate or refute the consideration; 'U' indicates there is uncertainty around whether the evidence supports this criterion; 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments	
Is the hypothesised causal factor worse in Glasgow?	Y	Overcrowding was higher, and the urban change resulting from policies to	
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	tackle this was more profound in Glasgow than in Liverpool or Manchester.	

A34 Sectarianism

Description of hypothesis

Scotland, and in particular the Glasgow and the West Central Scotland conurbation, is more affected by religious sectarianism (i.e. between Catholics and Protestants) than other parts of the UK, and that this in turn has negatively impacted on health.

Rationale

It is argued that there is a pervading culture of sectarianism in Scotland and, in particular, Glasgow (stretching as far back as the 1840s)⁵⁵⁹ which might impact on the health of its population in a number of ways including: impeding the social mobility of sections of the population; detrimentally affecting, through psychosocial processes, the health and wellbeing of those discriminated against; through the effects of violence from sectarian attacks; and through the uneasy social relations between population sub-groups.

Links to other hypotheses

This is loosely connected to the employment/labour market and social mobility hypotheses, given the suggestion that historically (although some argue currently), Catholics were excluded from certain employment sectors and/or promotion because of religious discrimination. There are also potential links to social capital, in terms of potentially impacting on components such as trust and reciprocity.

Evidence overview

Although there is evidence to support the existence of religious sectarianism in Scotland and Glasgow, its scale is disputed. More to the point, however, there is little doubt that there is a greater sectarian divide in Belfast, but despite this, mortality is lower in Belfast than in Glasgow (after adjustment for differences in levels of deprivation), and also in Northern Ireland compared with both Scotland as a whole, and the West Central Scotland (WCS) region. Mortality in Northern Ireland has also been improving faster than in Scotland and WCS^{18,19}.

Research published in 2003 suggested that there was a perception in Glasgow that a "culture of prejudice" existed in the city, with sectarianism, and sectarian attacks, seen as prevalent characteristics⁵⁶⁰, an assessment with which others have agreed^{561,562}. Other research by Walls and Williams⁵⁶³ highlighted a lack of social mobility among Glasgow Catholics; the authors discussed whether this was due to 'competence' and 'cultural' factors, or whether it was related instead to 'institutional sectarianism' – and concluded that the latter hypothesis was more likely. Furthermore, others have highlighted the poorer health status and socioeconomic position of Catholics compared with the rest of the West Central Scotland population⁵⁶⁴, with one study highlighting Glasgow Catholics' disadvantage across a range of health measures, with only half of the higher morbidity explained by the socioeconomic characteristics of the sample.

However, the evidence is disputed – described by some as "much exaggerated", and that "in the matter of religious conflict, the history of Scotland is much closer to that of the United States or Australia then it is to that of Northern Ireland"⁵⁶⁵. Indeed, the scale of the religious divide in Northern Ireland is well documented. Numerous studies have shown the extent of the religious divide in Belfast: in particular, the considerably poorer socioeconomic and health profile of its Catholic population relative to the rest of the population^{cxxii}, and the impact of years of conflict on the city's residents^{42,566-569cxxiii}. In light of this, a study was undertaken (published in 2012) comparing deprivation and mortality in Glasgow compared with Belfast, based on a methodology very similar to that used in the comparisons of Glasgow with Liverpool and Manchester. This showed remarkably similar levels of excess mortality in the Scottish city to that shown in comparison with the English cities: 27% higher for premature deaths, 18% higher for deaths at all ages. It was argued, therefore, that if religious sectarianism were indeed impacting significantly on population health in Glasgow, one would expect to see a more striking manifestation of that effect in Belfast, given the latter city's more profound history of such religious division.

There has been less work comparing sectarianism in Glasgow with that in Liverpool or Manchester, but it does not seem likely that the English cities have been entirely immune to sectarianism (and in the case of Manchester, racism, given the greater ethnic diversity in the city). All three cities experienced substantial immigration from Ireland during the 19th and early 20th centuries as people moved from the relatively impoverished circumstances in Ireland at that time to the rapidly industrialising cities where work was more plentiful: by the middle of the 19th century almost half of Britain's large Irish population were living in Glasgow, Liverpool, Manchester and London^{139, cxxiv}. Thus it is likely that all three experienced sectarianism resulting from that inward migration to a greater or lesser degree (indeed, Liverpool elected politicians from the 'Protestant Party' until the early 1970s). However, the relative scale of sectarianism across Glasgow, Liverpool and Manchester, and differences in its development in recent decades, have not been assessed.

The 2011 synthesis report found no robust evidence of whether Scotland is more affected by such religious divisions than elsewhere, and furthermore highlighted not only Northern Ireland's lower, and more rapidly improving, mortality rates, but also the fact that there was no consistent association between peaks in sectarianism and mortality in Scotland.

^{cxxii} It should be noted that, as O'Reilly and Rosato and others have pointed out, the health divide in Belfast is not simply a Catholic-Protestant religious division: Catholics have higher mortality, but this is explained by their poorer socioeconomic profile, and there is considerable variation in mortality rates within the non-Catholic population.

^{cxxiii} As French pointed out, even since the Good Friday Agreement of 1998, "sectarian violence [between the Protestant and Catholic communities] remains a problem for many at home and in the workplace, taking the form of attacks on people and property; less violent forms of aggression such as verbal abuse, harassment, visual displays and graffiti also continue to be significant"⁵⁶⁶.

^{cxxiv} By 1851 almost 20% of Glasgow's population had been born in Ireland¹³⁵; in Liverpool, an estimated quarter of a million Irish emigrants reached the city in that same late 1840s period, many of whom (especially the poorest who could not afford further travel) remained in the city permanently^{138,494}; and, one third of the population increase in Manchester between 1841 and 1851 was attributable to Irish migration (with 15% of the city's population in 1851 recorded as being Irish)¹³⁵.

Conclusion

Overall, the weight of evidence suggests it is unlikely that religious sectarianism contributes to Glasgow's excess mortality compared with Liverpool and Manchester (and Belfast). By association, it seems unlikely that it plays a significant role in the excess observed in Scotland as a whole compared with England & Wales.

Assessment of evidence of causality

1)	General	assessment	of likelihood	of causality	in terms of	f Bradford Hill c	riteria.
-,	General	assessment	01 1110000	or caasancy			in iteriai

Bradford Hill's	Assessment	Comments
criteria for		
causality		
Strength of association	U	
Temporality	U	
Consistency	U	
Specificity	U	There is little high quality evidence linking
Biological gradient	U	theoretically possible and plausible mechanism.
Plausibility	Y	
Coherence	Y	
Experiment	U	
Analogy	Y	

^{cxxv} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	U	There are no data available to facilitate comparison between
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	Scotland and England & Wales.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments	
Is the hypothesised causal factor worse in Glasgow?	U	There are no data available for Glasgow,	
Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	on this measure; however, Glasgow's mortality profile is worse than Belfast's (a city which undoubtedly suffers from greater levels of religious sectarianism).	

A35 Sense of coherence

Description of hypothesis

It has been proposed that the population of Scotland, and in particular Glasgow, may have a lower 'sense of coherence' than those elsewhere in the UK, rendering them less resilient to the negative effects of stress on levels of health and wellbeing, and ultimately at greater risk of early death.

Rationale

The Sense of Coherence (SoC) theory was developed by the American-Israeli sociologist Aaron Antonovsky^{570,571}. Emerging from his work around the concept of salutogenesis (a focus on the mechanisms that promote and support good health, in contrast to pathogenesis, the factors that create disease), and in particular the relationship between health and stress, the theory seeks to capture the extent to which people can manage, or be resilient to, the negative effects of stress on health and wellbeing. It was famously developed from his studies of women who survived Nazi concentration camps in the second world war. It is made up of three components: comprehensibility (the extent to which events in one's life can be readily understood and predicted); manageability (having the necessary skills and resources to manage and control one's life); and meaningfulness (there being a clear meaning and purpose to life), of which the third was viewed by Antonovsky to be the most important. Two versions of the SoC scale were created by Antonovsky, one with 29 questions (SOC-29) and a later one with 13 questions (SOC-13), although a considerable number of modified versions of both have also been used⁵⁷². Overall the measure has been deemed to be a "reliable, valid and cross-culturally applicable instrument", and has been shown to be significantly associated with a wide variety of outcomes, in particular: various measures of quality of life^{573,574} and perceived health status⁵⁷⁵; mental health^{575,576} (e.g. depression, hopelessness^{572,577-580}, anxiety, post-traumatic stress symptoms⁵⁸¹, psychiatric disorders⁵⁸² and suicide⁵⁸³); crime⁵⁸⁴; risk of tobacco use⁵⁸⁵; and alcohol and drug problems^{586,587}. Some reviewers have questioned its association with physical health, citing considerably mixed evidence. However, it has been shown to be significantly associated with, for example, circulatory health problems⁵⁸⁸, diabetes⁵⁸⁹, post-surgery recovery^{590,591}, and a 2008 UK study of almost 20,000 individuals suggested that a strong SoC was associated with a 20% reduction in all-cause mortality⁵⁹². Given the above evidence of links to a variety of health related outcomes, it has been hypothesised, including within official government reports^{77,78}, that SoC may be lower among the Scottish and Glaswegian populations.

Links to other hypotheses

Anomie; individual values.

Evidence overview

Sense of coherence has never been compared between all Scotland and England. However, to partially address this gap in the evidence, it was included in the 2011 three-city survey of Glasgow, Liverpool and Manchester (described elsewhere in this report), based on the SOC-

13 scale mentioned above. Contrary to the suggested hypothesis, SoC was found to be substantially higher, not lower, among the Glasgow sample compared with the samples of the two English cities (Figure A35.1)^{57,68}. Statistical analyses showed that it was higher overall, and in comparison of all strata of gender, age, area deprivation and social class (Figure A35.2). Consistent differences between the cities were also seen in the statistical modelling analyses of the comprehensibility, manageability and meaningfulness scores.



Figure A35.1 (Source: Walsh et al. 2013).


Figure A35.2 (Source: Walsh et al. 2013).

Conclusion

Conclusions are limited by the lack of temporal data, and lack of evidence in relation to country level analyses. However, the city comparisons suggest that (although based on cross-sectional survey data which clearly do not allow any measure of impact, or otherwise, on individuals' subsequent mortality) SoC is an unlikely explanatory factor for the excess mortality recorded in Glasgow compared with Liverpool and Manchester.

Assessment of evidence of causality

Bradford Hill's criteria for	Assessment ^{cxxvi}	Comments
causality		
Strength of	Y	
association		
Temporality	U	
Consistency	Y	
Specificity	Y	There is high quality evidence showing cross-sectional
Biological	Y	health outcomes, but little evidence on changes in
gradient		SoC and its impact.
Plausibility	Y	
Coherence	Υ	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	U	No data are available to compare SoC in Scotland with England & Wales
Assuming a difference in exposure, would this have occurred prior to the outcome?	U	

^{cxxvi} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow? Assuming a difference in exposure, would this have occurred prior to the outcome?	N	The three cities survey showed that SoC is higher in Glasgow compared with Liverpool and Manchester. No trend data are available.

A36 Social capital

Description of hypothesis

Social capital^{cxxvii} (a concept related to the idea of social connectedness and the value of social networks, and shown to be associated with population health outcomes) may be lower in Scotland, compared with the rest of the UK, and in particular in Glasgow compared with similar English cities such as Liverpool and Manchester.

Rationale

The theory of social capital is complex. It entails different dimensions (e.g. structural, cognitive^{191,593-595}) and types (e.g. bonding, bridging), and it has been defined in many different ways and by many different commentators^{190,596-598}, albeit that most definitions overlap to large degrees. Perhaps the most frequently used definition is that of Putnam^{188,598}, who defines it as the "features of social organization such as networks, norms, and social trust that facilitate coordination and co-operation for mutual benefit". Other definitions of social capital tend to be based on four similar, key, notions: "social trust/reciprocity; collective efficacy; participation in voluntary organisations; social integration for mutual benefit"^{,599}. Although by no means exempt from criticism (particularly relating to: how it is measured⁶⁰⁰⁻⁶⁰⁴; whether it is an individual or collective (e.g. of a community) attribute^{190,191,605,606}; and its potential negative effects^{188,190,607}), there is, however, a considerable amount of convincing evidence of the beneficial impact of social capital on health. For example, significant associations between higher social capital and lower mortality have been shown in the USA⁶⁰⁸⁻⁶¹¹, post-communist Eastern Europe^{504,612-614}, Finland⁶¹⁵, Australia⁶¹⁶, and Latin America and the Caribbean⁶¹⁷, and a recent review concluded that "both individual social capital and area/workplace social capital had positive effects on health outcomes, regardless of study design, setting, follow-up period, or type of health outcome"⁶¹⁸.

With regard to the links between social capital and socioeconomic health *inequalities*, although some authors have questioned the causal links⁶¹⁹, a systematic review published in 2013 suggested that there *was* an association, albeit one that required further research to unpick⁶²⁰.

In addition, it has been argued that a 'sub-component' of social capital is *religious* social capital, relating to the benefits of social participation in organised religion. This has been confirmed as a "valid construct"⁶²¹, and there is a considerable amount of evidence (albeit principally from the USA) of the beneficial impact of religious participation on health outcomes: a 'meta-analytic' review of the evidence in 2000 suggested that higher levels of religious attendance were associated with almost 30% lower all-cause mortality compared with those with lower levels of participation⁶²². Other reviews have confirmed the association, and although they point to caveats associated with some of the studies, they

^{cxxvii} Note that, as mentioned in the main part of the report, the term 'social capital' itself can be seen as controversial, given that its origin has been traced back to an 'intellectual ruse' by individuals within the World Bank in the 1990s¹⁸⁹, and could perhaps be less contentiously described as 'social integration' or 'social fabric'.

show that the significantly lower mortality is not explained by potential confounders^{623,624}. Studies have also shown that the association with lower mortality may be stronger in women, while separate research has suggested an important role for religion in impacting specifically on suicide mortality⁶²⁵.

A number of different mechanisms have been suggested as means by which social capital may impact on population health. These include: social and psychological support processes (i.e. greater social support in times of need); more positive health behaviours (influenced both by informal social control (preventing damaging behaviours such as alcohol and drug abuse), and by an increased likelihood of healthy behaviours such as physical activity being adopted); and provision of access to services and amenities (i.e. as more socially cohesive communities can safeguard relevant services under threat through effective local action)⁶²⁶. With regard to social participation in particular, volunteering has been shown to be independently associated with better health outcomes: a recent systematic review suggested better outcomes related to depression, life satisfaction, and wellbeing, with some links to lower all-cause mortality.

Some commentators have also argued that at the city or state level (as opposed to the neighbourhood level), greater social capital impacts on health via political processes: it is argued that social participation (e.g. in voluntary groups, churches) nurtures skills that can lead to political engagement and activity, and greater political activity across the social gradient results in government policies more beneficial for the least advantaged members of society^{608,627-630}: "who participates in politics matters for political outcomes, and in turn the resulting policies have an important influence on the opportunities available to the poor to lead a healthy life". However, the 'beneficial' policies described here relate primarily to better government provision of social support: this is less relevant to comparisons of UK cities, or indeed of Scotland compared with other UK nations, as welfare policies in the UK have been reserved to the Westminster parliament throughout the period in which excess mortality has emerged, and have not varied within UK countries or cities^{cxxviii}.

A number of similar, and overlapping, potential pathways have been proposed to explain the apparent links between religious attendance and better health outcomes (including lower mortality): greater social networks, support and integration; less association with damaging lifestyle factors (alcohol, drugs, violence, risky sexual behaviour and so on) through 'social regulation'; and, more specific to religious social capital than other forms, increased psychological resources and coping mechanisms^{621,624,631-636}. Linking these forms of social capital further is the fact that religious participation has also been shown to encourage volunteering, itself a component of social participation with known links to better health outcomes as stated above.

^{covviii} Kawachi (who is quoted here) cites evidence of greater political engagement correlating with greater care of members of society through more generous social security systems. In contrast, and related to this, "the lower the levels of trust between citizens, the more hostile the social policies geared toward the poor". However, evidence is from national and US state governments which have control over welfare legislation, and not from UK local governments which do not.

Links to other hypotheses

Particular aspects of social capital, i.e. social participation, trust and reciprocity, overlap with the notion of individualism, itself a component of the 'individual values' thesis discussed elsewhere in this Appendix. As stated below, results of analyses of measures of individualism (the 'human values' of *universalism* and *benevolence*) from the survey of the adult populations of Glasgow, Liverpool and Manchester broadly correspond with results of analyses of the above three components of social capital.

As stated earlier, there is also a link between political engagement and social capital, which thus connects to the political influences thesis (Appendix A28).

Evidence overview

At the time of the 2011 synthesis report very little empirical data were available by which potential similarities or differences in social capital could be compared between Scotland and elsewhere in the UK, and between Glasgow and other UK cities.

Nationally, one of the few comparative analyses of social capital between Scotland and England was published in 2005 by Bell and Blanchflower using General Household Survey (GHS) data from 2000-01. The measures analysed were: neighbourliness score; local facilities score; network of friends; family network; not civically engaged. There were no significant differences between Scotland and England in analyses of the first three, while Scotland had marginally higher scores than England for the latter two measures.

At the city level, the first comparative, and comprehensive, analysis of indicators of social capital was undertaken by means of the 2011 three-city survey. A summary of those results suggested that there are differences between Glasgow and Liverpool and Manchester in relation to some, but not all, aspects of social capital. Notably the Glasgow respondents were characterised by lower levels of social participation (in terms of volunteering) and trust compared with both Liverpool and Manchester, and lower levels of a number of measures of reciprocity, principally compared with Liverpool alone. Some of these differences (e.g. volunteering, neighbourhood trust) were greatest among those of higher, rather than lower, SES. Some of these results were supported by results of analyses of Schwartz's 'human values' of 'universalism' and 'benevolence', both of which overlap with the concept of reciprocity. As discussed elsewhere in this Appendix, the former was shown to be lower in Glasgow than in both English cities, and this was the case across the social spectrum: however, echoing results of some of the social capital analyses, the greatest differences were seen in comparison of those of high social class and those living in the least deprived neighbourhoods. Benevolence was lower in Glasgow in comparison with Liverpool (but higher compared with Manchester), a pattern generally seen across different social classes.

Given the overlap between aspects of social capital and political engagement, other evidence from the three-city survey of a more 'politicised' Liverpool sample (in terms of, for

example, having been more engaged in anti-government demonstrations in the 1980s^{cxxix}) is also potentially relevant.

The lower levels of social participation were arguably also supported by results of analyses suggesting lower levels of religious participation in Glasgow – however, this is contentious as the question included in the survey was from the census and asked about religious affiliation, not participation: clearly the one does not necessarily entail the other, as a number of commentators have pointed out⁶³⁷⁻⁶⁴⁰. Nonetheless, relevant to this is the fact that high levels of excess mortality from suicide have been shown in Glasgow compared with Liverpool and Manchester. In a 2003 paper, Dorling and Gunnell modelled the impact of social and economic factors^{CXXX} on suicide rates across Britain. They found that in the vast majority of places (parliamentary constituencies) levels of suicide could be predicted by these ecological variables. However, there were a small number of areas which had significantly lower than expected rates, and areas which had higher than expected rates. The latter included deprived constituencies in Glasgow, while the former included areas in and around Liverpool. The authors speculated - in reference to Durkheim's work discussed elsewhere in this report - that protective factors relating to religion and social integration might be operating in Liverpool, a suggestion that potentially ties in with some of the analyses of social capital and religion in the three-city survey discussed above. Dorling and Gunnell specifically suggested that this may have been influenced by high numbers of "practising or believing" Catholics resident in the Liverpool areas, given the fact that there is international evidence of lower suicide rates among those of Roman Catholic faith compared with Protestants⁶⁴¹ (something of course also shown historically by Durkheim). Their suggestion could not be verified by Dorling and Gunnell because of a lack of data (the English census questions do not differentiate between different Christian religions); however, the three-city survey did allow such differentiation and indeed showed that the percentage of the Liverpool sample describing themselves as Catholic was much higher than in Glasgow (and Manchester): 29% compared with 18% (and 12%). Interestingly, the greatest difference was between those living in the most deprived parts of the cities (i.e. quintile 1) where the figures were 41% compared with 20% (and 8% for Manchester) respectively^{cxxxi}. This is potentially relevant given that suicide rates tend to be highest in areas of high deprivation^{642,643}. That said, however, and as stated above, there is a considerable weakness in the use of a question based on religious affiliation, as opposed to participation, in the

^{cxxix} Respondents were asked whether or not in the 1980s they had attended any public demonstrations about government policies (with demonstrations defined as "public rallies, meetings, strike actions or other similar events"). In Glasgow and Manchester, only 5% of respondents who had lived through the 1980s reported that they had attended demonstrations of this type. However, the equivalent figure for Liverpool was 14%. Other analyses showed the Liverpool sample to have stronger (more negative) views on the current UK government. For example, 50% agreed or strongly agreed that the UK government was "undermining" their city: the equivalent figures for Glasgow and Manchester respectively were 30% and 28%.

^{cxxx} Described as indicators of "social isolation", these were: the percentage of internal migrants, the percentage not in employment and the percentage who were single.

^{cxxxi} There was a very clear social gradient in Liverpool, ranging from 41% of those in living in the most deprived areas (quintile 1) stating they were Roman Catholic down to 21% in the least deprived quintile (quintile 5). There was no such gradient evident in analyses of the data for Glasgow and Manchester.

survey. Furthermore, recent analyses of Scottish and English census data linked to individual mortality records have shown that in comparing all-cause mortality in Scotland with England, and in Glasgow with Liverpool and Manchester, levels of excess Scottish all-cause mortality were not reduced when this census-based religion question was included in the models⁶⁴⁴. Suicide was also examined but sample size restrictions meant that this could not be done in a comparison of the cities, only the countries. In the latter case, addition of the religion variable to the model only marginally lowered the risk of suicide in the Scottish sample: after adjustment for age, sex and SES, the Scottish sample was associated with a 45% higher risk of suicide compared with the English sample, and after further adjustment for religious affiliation, this reduced to 41%.

Conclusion

Some (but not all) aspects of social capital may be different in Glasgow compared with Liverpool and, to a lesser extent, Manchester. For some measures this is particularly true in comparison of those of higher, rather than lower, SES. There is a lack of evidence of lower social capital in Scotland as a whole compared with other UK countries.

It seems at least plausible that some protective factors may be at work in Liverpool in comparison with Glasgow with regard to the higher levels of social participation, trust and reciprocity evidenced for the city, and the previously amassed knowledge of the benefits of these factors for population health. This may extend to the more politicised nature of the Liverpool sample, as well as even to the suggestion that religious social capital may play a part in this.

Assessment of evidence of causality

1)	General	assessment	of likelihood	of causality in	terms of Bradford	Hill criteria.
-,	General	assessment		or caasancy m		

Bradford Hill's criteria for causality	Assessment ^{cxxxii}	Comments
Strength of	Y	
association		
Temporality	U	
Consistency	Y	
Specificity	Y	There is good evidence that high levels of social capital enhance health; however, we did not identify
Biological gradient	Y	evidence of changes in social capital and its health impact.
Plausibility	Y	
Coherence	Y	
Experiment	Υ	
Analogy	Y	

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
to the house the strend or and fraction	NI	
is the hypothesised causal factor	N	The limited available
worse in Scotland?		evidence suggests that
		social capital was not
Assuming a difference in exposure,	U	different in the early
would this have occurred prior to		2000s in Sectland
the outcome?		2000s in Scotland.

^{cxxxii} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow? Assuming a difference in exposure, would this have occurred prior to the outcome?	Y U	The three cities survey suggest that some aspects of social capital are worse in Glasgow compared with Manchester and, especially, Liverpool.

A37 Social mobility

Description of hypothesis

It has been proposed that social mobility may be lower in Scotland/Glasgow than in other parts of the UK, impacting on population health status.

Rationale

Health status in any population is socially patterned: thus, limited movement up the social 'ladder' might result in a population exhibiting poorer health outcomes than where such mobility was more evident. Authors have claimed particular aspects of Scottish culture (low self-confidence, and 'social control') are potential impediments to social mobility among the country's population^{70,645}.

Links to other hypotheses

There are links to the religious sectarianism hypothesis, given the suggestion that religious discrimination impedes (or has impeded in the past) mobility among certain sections of the population. The motivational aspect of social mobility links to the hypothesis around individual values. More speculatively, it links to aspects of migration.

Evidence overview

a) Longitudinal and census-based analyses of social mobility

Longitudinal data represent the best means of assessing social mobility in the population. The research literature confirms the hypothesised health differences between socially mobile and 'stable' individuals. UK analyses have shown consistent evidence that upwardly mobile populations tend to have better health than their social class of origin (although not as good as those in their 'new' social class); similarly, downwardly mobile populations have worse health than those they leave behind (but not as bad health as those in their 'destination' class)⁶⁴⁶⁻⁶⁴⁹. This has been shown in analyses of self-reported health^{646,649,650}, mental health⁶⁵¹ and mortality^{647,648}.

(It is also relevant to note that the above research additionally focuses on two other, related, issues. First, the effect of social mobility on health inequalities: this is disputed, as some have argued that it has little effect', or indeed can constrain or narrow inequality^{646,648}, while others have argued it has the potential to instead widen inequalities^{649,652}. Second, the issue of health selection i.e. the suggestion that health itself determines movement up and down the social ladder. However, the majority of research evidence has demonstrated that this is not the case: it is socioeconomic status across the life-course that influences health status, and not the other way round^{311,483,647,650,653,654}).

That longitudinal analyses have shown that upwardly and downwardly mobile members of the population are, respectively, associated with better and worse health (compared with those whose social class does not change) is potentially important. However, all the existing evidence suggests that there is very little difference in the patterns of social mobility between Scotland and England, and that this has been the case for a long time. For example, Erikson and Goldthorpe highlighted strong similarities between the two countries, based on analyses of data up to the mid-1970s⁶⁵⁵; more recently, Paterson and Ianelli produced similar findings in both patterns and trends of social mobility, based on analyses of longitudinal data up to 1999⁶⁵⁶.

No such comparative longitudinal analyses have been undertaken for Glasgow, Liverpool and Manchester. However, given the similarities in social mobility between Scotland and England, as well as the parallels between the cities in terms of their current economic profiles and their histories of industrialisation and deindustrialisation since the 19th century, it would be surprising if any notable differences existed. Indeed, comparisons of crosssectional census data between 1951 and 2001, although not an ideal method for assessing social mobility, do not particularly support the assertion that there have been differences between the cities in this regard. Figure A37.1 shows the percentages of the adult male population broken down by social class over the 50-year period: in general, the percentages categorised as high (Class 1 & 2), middle (Class 3) and low (Class 4 & 5) social class are fairly similar in each city and each period. More detailed analysis of the five individual social classes do suggest a slightly greater increase over the period in adult males classed as Social Class 1 in Manchester compared with Glasgow and Liverpool (Figures A37.2 and A37.3). However, further research, using longitudinal data, would be required to verify, and properly quantify, this difference^{coxviii}.

Adult male social class 1951-2001 Source: University of Portsmouth/Great Britain Historical GIS Project (www.visionofbritain.org.uk) 100.0 90.0 80.0 70.0 % adult males 60.0 Class 4 & 5 50.0 Class 3 Class 1 & 2 40.0 30.0 42.5 20.0 10.0 16.0 15.3 14.2 13. 14. 12.2 12.1 0.0 Glasgow Glasgow Glasgow Glasgow Glasgow Liverpool Liverpool Liverpool Manchester Liverpool Liverpool Manchester Manchester Manchester Manchester 1951 1971 1981 1991 2001

Figure A37.1

^{cxxxiii} Note also that the dramatic rise in the percentage of adults in both social class I and II between 1981 and 2001 in Glasgow has been described before⁴, and may be influenced by measurement and definitional issues driven by the change in those decades from employment opportunities in an industry-based economy to one dominated by the service sector. This is the subject of ongoing research.

Figure A37.2







b) Obstacles to social mobility: confidence and social control

At the national level, the 2011 synthesis report highlighted some evidence that levels of selfconfidence was lower (although increasing) among Scottish adolescents compared with their peers in other European countries (data from 2002)⁶⁵⁷. However, those analyses did not include comparisons with England: there is no evidence of lower confidence among Scottish/Glaswegian adults compared with other parts of the UK.

At the city level, a number of relevant measures were included within the 2011 three-city survey of Glasgow, Liverpool and Manchester. These were:

- Self-efficacy: measured by the Generalised Self-Efficacy (GSE) scale, this assesses control over adversity and over one's environment, and the self-belief that one can succeed in undertaking tasks (no matter how difficult); it has also been shown to capture aspects of "motivation"⁶⁵⁸, also crucial to the idea of social mobility.
- Achievement: one of the values measured in the Human Values Scale^{466,467}, this also assesses levels of motivation to succeed.
- Self-esteem: measured by a single question on perception of one's own self-esteem.

None of these data supported the idea that people in Glasgow were more impeded in their desire to succeed compared with those in Liverpool and Manchester. There was no evidence of lower self-efficacy, and this was the case across all social classes. Respondents in Glasgow tended to be more (not less) associated with the achievement value compared with those in Manchester and, especially, Liverpool. This was again generally the case across all social classes. Finally, relatively more Glaswegian respondents described themselves as having high self-esteem compared with respondents in Liverpool and Manchester. The greatest difference was in comparison of those of high SES, with the figures either similar, or slightly higher for Glaswegians, in comparisons with other social classes.

All the above findings generally held in comparison of age and gender.

A specific component of the hypothesis of cultural impediments to social mobility relates to Scottish Calvinist values. As the previous (2011) synthesis reported, however, there is no evidence to support this. The association with mortality patterns was described as 'weak', given there is no evidence of worse health outcomes or behaviours among the Scottish Presbyterian population compared with, for example, those of Irish Catholic descent.

Conclusion

Although a plausible hypothesis – given the evidence of differences in health status between socially mobile populations compared with socially 'stable' populations – all existing evidence suggests there are no meaningful differences in levels of social mobility between Scotland and England, and that this has been the case for a long time. Other data strongly suggest that this is also likely to be the case for comparisons of the cities of Glasgow, Liverpool and Manchester.

Assessment of evidence of causality

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

Bradford Hill's criteria for causality	Assessment ^{cxxxiv}	Comments
Strength of association	Y	
Temporality	Y	
Consistency	Y	
Specificity	Y	
Biological gradient	Y	There is evidence that upward social mobility is good for health and vice versa.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	N	There is little or no difference in the changing
Accuming a difference in expective	v	social make-up of
would this have occurred prior to	T	Scotland relative to England & Wales.
the outcome?		

^{cxxxiv} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow? Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	Although there has been no specific study of levels of social mobility in Glasgow compared with Liverpool and Manchester, all available data strongly suggest that – as with Scotland compared with England & Wales – there are few differences in levels of social mobility across the cities.

A38 Spatial patterning of deprivation

Description of hypothesis

It has been proposed – principally in relation to comparisons of Glasgow with Liverpool and Manchester – that there may be important differences between the cities in the spatial patterning of deprivation (i.e. the way in which deprived and affluent areas are distributed across the cities) which may, through particular causal pathways, adversely affect the health of Glasgow's population. This hypothesis can be extended to other parts of urban Scotland, especially in and around the post-industrial region of West Central Scotland.

Rationale

The spatial patterning of affluence and poverty has been shown to have potentially important area effects on health. For example, Sridharan *et al.*⁶⁵⁹ demonstrated within a Scottish context the importance of the patterning and concentration of deprivation on mortality, over and above the impact of deprivation alone: this highlighted the potential influence of levels of deprivation on health in neighbouring localities. Others have demonstrated similar effects in other places⁶⁶⁰.

Two contrasting mechanisms have been suggested as having potential effects: first, that greater concentrations of deprivation (deprived neighbourhoods surrounded by other deprived neighbourhoods) might result in more ingrained poor health in comparison with differently patterned areas (deprived areas mixed with less deprived areas); second, reflecting the income inequalities hypothesis of Wilkinson and others but applied to local areas rather than whole countries or states, that there may be negative psychological effects of living in a deprived area in close proximity to areas of greater affluence.

Links to other hypotheses

Income inequalities; inadequate measurement of deprivation; migration; nature and scale of urban change; quality of external physical environment.

Evidence overview

In general terms, the research evidence is very mixed. Some studies have demonstrated that deprived areas in close proximity to more affluent areas are associated with relatively *better* health outcomes⁶⁶¹⁻⁶⁶³. This has been described in terms of a 'pull up' theory⁶⁶³⁻⁶⁶⁵, whereby deprived areas benefit from, among other aspects, proximity to better services provided to more affluent areas. However, other studies have observed worse outcomes for deprived neighbourhoods that are surrounded by more affluent areas (so-called 'islands of deprivation'⁶⁶⁶) compared with deprived areas situated within larger concentrations of similarly deprived neighbourhoods (so-called "land-locked deprivation").

The hypothesis that the spatial patterning of deprivation was different in Glasgow compared with Liverpool and Manchester, and impacted on mortality rates, was tested in research published in 2013 and 2014^{59,664}. These showed that although there were differences between the cities in this regard – principally that deprived areas tend to be rather more

dispersed across Glasgow, and rather more concentrated within larger areas in the English cities, the scale of these differences was not huge. Effects of surrounding levels of deprivation on neighbourhood mortality were observed in both Glasgow and Liverpool (but not in Manchester), and in both cases the effects were very small compared with the effects of deprivation observed within the neighbourhoods. This suggests that the differences in the patterning of deprivation are unlikely to represent a major contribution to the higher levels of excess mortality observed in the Scottish city. However, this is a complex area of research and further work is being carried to better understand the relevance and impact of the observed differences between the cities.

Other research on the historical development of areas of poverty and affluence in the three cities over a 40-year period has shown that the relatively greater fragmentation of poverty in Glasgow has been present since at least 1971 (the earliest year included in the analysis). The results also show that since 1971, based on a number of different measures of the spatial distribution of deprivation, the three cities have become more alike (contrasting with the mortality profiles which have become less alike)⁶⁶⁷.

Conclusion

The weight of evidence does not support the suggestion that excess mortality in Glasgow compared with Liverpool and Manchester is influenced by differences in the spatial patterning of deprivation between the cities. However, there is an absence of any similar data to assess the relevance of this theory to the high levels of excess mortality observed in Scotland compared with England & Wales.

Assessment of evidence of causality

1 \	Gonoral	accoccmont	of likelihood	of cousolity in	torms of B	radford Hill	critoria
-	General	assessment	of likelihood (Ji causanty n	i terms of D		cincenta.

Bradford Hill's	Assessment	Comments
criteria for		
causality		
Strength of	U	
association		
Temporality	U	
Consistency	U	Although there is evidence of associations between
Specificity	U	differences in the patterning of deprivation and differences in health outcomes, assessment of the
Biological	U	evidence is hampered by a lack of consistency in terms
gradient		of conflicting results (related to opposing theorised
Plausibility	Y	research.
Coherence	Y	
Experiment	U	
Analogy	Y	

^{cxxxv} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	U	It is unknown whether the patterning of deprivation differs between Scotland
Assuming a difference in exposure, would this have	Y	and England & Wales.
occurred prior to the outcome?		Given the timing of large amounts of housing construction, any emerging differences are likely to have been prior to the outcomes manifesting.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow? Assuming a difference in exposure, would this have occurred prior to the outcome?	Y Y	Deprived areas in Glasgow were slightly more dispersed than in Liverpool or Manchester; however, this did not appear to explain the mortality patterns observed.

A39 Terminations of pregnancy

Description of hypothesis

Variations in current levels of mortality between Glasgow and Liverpool and Manchester (and, by implication, between Scotland and England more generally) may have be influenced by differences in rates of termination of pregnancy (ToP) in previous years.

Rationale

This theory was suggested in light of research from the USA linking spatial and temporal variations in ToP rates to differences in social and health outcomes. For example, increases in terminations have been associated with reductions in welfare benefits dependency⁶⁶⁸, child poverty⁶⁶⁹, and child abuse⁶⁷⁰, while restrictions in access to ToP services have been linked to subsequent higher rates of child homicide⁶⁷¹. More generally, economists have attributed decreases in overall crime rates (including homicide) in America in the 1990s to the legalisation of abortion throughout the USA in 1973^{672,673}. The 'causal pathways' inferred by these USA analyses relate to rates of unwanted pregnancy being much higher in socioeconomically deprived areas: thus, higher rates of terminations could reduce the numbers of people being born in disadvantaged areas who might otherwise have been more vulnerable to adverse experiences (including involvement in crime). It was suggested that in a similar way, the gap in socially patterned premature mortality rates between Glasgow and Liverpool and Manchester may have been influenced by earlier differences in similarly patterned rates of ToP.

Links to other hypotheses

There are no clear, direct, links between this theory and others that are considered within this report.

Evidence overview

The research evidence on which this hypothesis is based is primarily from the USA. Clearly the American context with, for example, considerable variation in types of access to ToP facilities across USA states, is quite different from the UK context. Furthermore, childhood mortality in Glasgow is not significantly higher than in Liverpool and Manchester and the causal pathways by which variations in termination rates could impact on adult mortality are potentially quite different. Nonetheless, this seemed a potentially important hypothesis to test, and it was examined in research published in 2013. The research concluded that differences in ToP rates between the cities were unlikely to impact on variations in later mortality rates. This was because although ToP rates in Glasgow were lower than in Liverpool and Manchester over the 30-year period analysed (1969-2009) (and this was also true for Scotland compared with England & Wales), the analyses showed that 90% of the excess deaths that took place in Glasgow compared with Liverpool and Manchester between 2003-07 (i.e. the period in which high levels of excess mortality were shown in the 2010 Glasgow, Liverpool and Manchester research) related to individuals born prior to the 1967 Abortion Act (which legalised, and thereby made accessible, abortion in the UK). These excess deaths in early to mid-2000s, therefore, were not influenced by earlier variations in

ToP rates. Additional analyses of ToP and mortality data suggested it was also highly unlikely that the remaining 10% of the excess deaths were in any way influenced by differential ToP rates^{54, cxxxvi}.

Conclusion

On the basis of the evidence – in particular in relation to temporality – this appears to be an unlikely hypothesis.

Assessment of evidence of causality

Bradford Hill's	Assessment ^{cxxxvii}	Comments
criteria for causality		
Strength of association	Y	
Temporality	Y	
Consistency	Y	
Specificity	Y	There is evidence linking historical rates of
Biological gradient	Y	outcomes.
Plausibility	Y	
Coherence	Y	
Experiment	Y	
Analogy	Y	

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

^{cxxxvi} The paper argued that this was for three reasons. First, for this to have contributed significantly to the excess would have required an extremely high percentage of deaths among the 'cohort': analyses suggested a mortality rate of around seven times the normal rate for the relevant age group would have been required, which seems unlikely. Second, analysis of time trends showed that the gap in ToP rates between Glasgow and the English cities *narrowed* over the period in which the mortality gap instead *widened*. Third, the USA research showed differences in ToP rates impacted on social and health outcomes via poverty and deprivation related mechanisms: by definition, however, the excess relates to higher mortality that is *not* explained by differences in deprivation. Thus an alternative linking mechanism would be required for the hypothesis to be relevant, and this again seems unlikely. ^{cxxxvii} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland?	Different (rather than 'worse')	The timing of the 1967 Abortion Act suggests that changes in the rate of
Assuming a difference in exposure, would this have occurred prior to the outcome?	N	terminations could not have been responsible for the excess.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow?	Different (rather than 'worse')	The timing of the 1967 Abortion Act suggests that
Assuming a difference in exposure, would this have occurred prior to the outcome?	N	terminations could not have been responsible for the excess.

A40 Water hardness

Description of hypothesis

It has been proposed that the high level of excess mortality in Scotland from cardiovascular disease may be attributed in part to differences in the hardness of drinking water between Scotland and England.

Rationale

The hardness of drinking water is determined by the presence of a variety of dissolved metallic ions, in particular calcium and magnesium. Both calcium and magnesium are essential minerals which have a number of different positive effects on human health. Inadequate intake of either can result in adverse outcomes – for example, magnesium deficiency has been shown to be associated with conditions such as hypertension, coronary heart disease and type 2 diabetes. Drinking (tap) water in Scotland is softer (i.e. has lower levels of calcium and magnesium) than in England.

Links to other hypotheses

Diet.

Evidence overview

Cardiovascular disease and excess mortality

The most recent analyses of Scottish excess mortality showed that around 2011, deaths from cerebrovascular disease and ischaemic heart disease were, respectively, approximately 25% and 15% higher in Scotland compared with England & Wales after adjustment for age, sex and socioeconomic deprivation. The levels of excess for both causes were reasonably consistent over the period 1981-2011^{cxxxviii}. In comparisons of Glasgow with Liverpool and Manchester, over a quarter of the total number of excess deaths recorded in the Scottish city between 2003 and 2008 were attributable to diseases of the circulatory system.

Water hardness in the UK

Drinking (tap) water is generally softer in Scotland compared with England (especially compared with eastern and south eastern England), and in Glasgow compared with Liverpool and Manchester²²⁹⁻²³¹. This includes lower levels of magnesium in the Scottish drinking supplies.

Water hardness and cardiovascular disease

Evidence for the association between water hardness and cardiovascular disease (CVD) has been much debated since the first relevant epidemiological studies were published in the late 1950s and 1960s^{674,675}. The evidence has been, and remains, mixed, with a number of

^{cxxxviii} For the census periods of 1981, 1991, 2001 and 2011 the excess for cerebrovascular disease was 28%, 22%, 24% and 24% respectively. The equivalent figures for ischaemic heart disease were: 11.5%, 11%, 10% and 15%.

studies suggesting an inverse association between water hardness and CVD mortality (e.g. a study across 16 municipalities in Sweden in the late 1980s/early 1990s which suggested both magnesium and calcium in drinking water were important protective factors in relation to death from acute myocardial infarction (AMI) among middle-aged women⁶⁷⁶), and a number of other studies failing to do so (e.g. a study of more than 120,000 middle aged men and women in the Netherlands in the mid-1980s to mid-1990s which found no association between tap water calcium, magnesium, overall water hardness and mortality from ischaemic heart disease or stroke⁶⁷⁷). In the UK the British Regional Heart Study suggested at the start of the 1980s that areas of the UK with very soft water had 10-15% higher rates of CVD mortality after adjustment for other risk factors including deprivation. However, analyses of the same cohort published 25 years later (and with 25 years more follow up) found no association⁶⁷⁸ (and later reflections on the earlier study suggested that any levels of increased risk were, in any case, not large enough to warrant any kind of intervention). A study based on Scottish data published in the late 1980s also failed to demonstrate any association between water hardness and coronary heart disease⁶⁷⁹, as did a more recent study of data in England & Wales examining outcomes of CVD mortality⁶⁸⁰.

Generally, it has been suggested that much of the evidence for an association between water hardness and CVD mortality has been derived from ecological studies (with the known weaknesses associated with such analyses) rather than (methodologically preferable) case-control or cohort studies^{228,677,681-683}</sup>. However, that has not always been the case and a number of qualitative and systematic reviews published in recent years⁶⁸¹⁻⁶⁸³ have concluded that although there is no consistent evidence of an association between low levels of calcium in drinking water and cardiovascular mortality, the weight of evidence <u>does</u> support an association between levels of magnesium in water and CVD mortality (albeit that there are still weaknesses associated with a number of the relevant studies)^{cxxxix}. Thus, the most recent guidance from the World Health Organization concludes that "drinking-water may be a contributor of calcium and magnesium in the diet and could be important for those who are marginal for calcium and magnesium intake... Although there is some evidence from epidemiological studies for a protective effect of magnesium or hardness on cardiovascular mortality, the evidence is being debated and does not prove causality".

Conclusion

The weight of evidence suggests that it is plausible (albeit that causal links are still disputed) that there is an association between softer drinking water (specifically relating to lower levels of magnesium) and higher risk of cardiovascular disease in populations. Given that Scotland's and Glasgow's water supplies are softer than in the comparator areas, and that the contribution of cardiovascular disease to Scottish excess mortality has been consistent over a number of decades, it is equally plausible that water hardness plays some role in explaining that part of the excess (i.e. relating to cardiovascular mortality, but not to other

^{cxxxix} A meta-analysis within one systematic review (by Catling *et al.*) calculated (based on results from seven case-control studies) a pooled odds ratio of 0.75 (95%Cl 0.68, 0.82)) for the highest compared with the lowest exposure categories. In other words, those exposed to higher concentrations of magnesium in drinking water were associated with approximately 25% less risk of death from CVD compared with those exposed to lower levels.

forms of mortality). However, any contribution is difficult to quantify – although evidence from one of the UK studies has suggested that any impact on population health generally is likely to be small.

Assessment of evidence of causality

Bradford Hill's criteria for	Assessment ^{cx1}	Comments						
causality								
Strength of	Y							
association								
Temporality	U							
Consistency	Y							
Specificity	Y	The weight of evidence suggests that it is plausible (albeit that causal links are still disputed) that there is an						
Biological	Y	association between softer drinking water (specifically						
gradient		relating to lower levels of magnesium) and higher risk of cardiovascular disease in populations.						
Plausibility	Y							
Coherence	Y							
Experiment	U							
Analogy	Y							

1) General assessment of likelihood of causality in terms of Bradford Hill criteria.

^{cxl} Note: 'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion.

2) Assessment in relation to examination of data for Scotland in comparison with England & Wales.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Scotland? Assuming a difference in exposure, would this have occurred prior to the outcome?	Y	Water is softer, and cardiovascular disease is higher after adjustment for deprivation, over time. We have not identified evidence regarding changes to water hardness in Scotland compared with elsewhere.

3) Assessment in relation to examination of data for Glasgow in comparison with Liverpool & Manchester.

Comparison	Assessment	Comments
Is the hypothesised causal factor worse in Glasgow? Assuming a difference in exposure, would this have occurred prior to the outcome?	Y U	Water is softer, and cardiovascular disease is higher after adjustment for deprivation, over time. We have not identified evidence regarding changes to water hardness in Scotland compared with elsewhere.
		compared with elsewhere.

Appendix B. Assessment of hypotheses – summary table

This appendix summarises the three tables included within each of the 40 assessments presented in Appendix A.

'U' indicates that there is uncertainty around whether the evidence supports the criterion (including an absence of evidence); 'Y' indicates a balance of evidence supports criterion; N indicates that the balance of evidence does not support criterion; * indicates not applicable.

Exposure/ hypothesis		Gen	eral	asses	ssme	ent o	f like	eliho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland	Specific to Glasgow		
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A1 Air pollution	Y	Y	Y	Y	Y	Y	Y	Y	Y	Air pollution is a known negative influence on health and has been shown to be responsible for a substantial burden of disease.	N	U	Exposure to air pollution is better in Scotland than in England & Wales, although little time trend data were available.	N	U	Exposure to air pollution is better in Glasgow than in Liverpool and Manchester, although little time trend data were available.

Exposure/ hypothesis		Gen	eral a	asses	ssme	ent o	f like	eliho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland	Specific to Glasgow			
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments	
A2 (a) Anomie – Durkheim	Y	υ	Y	Y	U	Y	Y	U	Y	Durkheim's original work, and more recent descriptions of events in places like the former USSR, suggest that forms of anomie may be associated with mortality outcomes. The extent to which this has been a consequence of other interventions is much less clear.	U	Y	There are no data available to compare Scotland with England & Wales.	Ν	Y	The limited data available to examine aspects of anomie suggest no difference in Glasgow (or even a lower prevalence) than in Liverpool or Manchester.	

Exposure/ hypothesis		Gene	eral	asse	ssme	ent o	flike	eliho	od of	f causality in terms of Bradford Hill criteria	Specific to Scotland			Specific to Glasgow		
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A2 (b) Anomie – underclass / culture of poverty	*	*	*	*	*	*	*	*	*	There is little evidence for such a culture existing which makes assessment using the Bradford Hill criteria impossible. The evidence base around this hypothesis is politicised and contested, but there is little evidence to support the existence of such cultures or evidence to support such a culture as a cause of poverty or ill- health.	U	Y	We have not examined data to compare measures at the Scotland level. Given the lack of evidence for the theory, it is an unlikely contributor.	N	Y	For those markers of dependency that the advocates argue for, there is little difference between the cities. However, these markers did increase at the time of the emergence of the excess.

Exposure/ hypothesis	General assessment of likelihood of causality in terms of Bradford Hill criteria											Specific to Scotland				Specific to Glasgow			
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments			
A3 Artefact: inadequate measurement of poverty and deprivation	Y	Y	Y	Y	Y	Y	Y	Y	Y	By definition, inadequate measures of poverty and deprivation do not lend themselves to quantification and testing. There is evidence that multiple measures of deprivation are more helpful, and that existing measures have become dated and may not reflect the lived experience and depth of poverty within the population.	Y	Y	Where multiple measures of SES are available, the excess decreases, suggesting that more and better measures would explain a larger proportion of the total excess. Some proxies of lived experience, such as overcrowding, display large differences.	Y	Y	Where multiple measures of SES are available, the excess decreases, suggesting that more and better measures would explain a larger proportion of the total excess. Some proxies of lived experience, such as overcrowding, display large differences.			
A5 Climate: rainfall	N	N	N	N	N	Y	Y	N	Y	The available evidence does not suggest that there is a negative impact of higher levels of rainfall.	Y	Y	Rainfall has been consistently higher in Scotland over the relevant time period.	Y	Y	Rainfall has been consistently higher in Glasgow.			

Exposure/ hypothesis		Gene	eral	asse	ssme	ent o	flike	eliho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland	Specific to Glasgow			
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments	
A6 Climate: vitamin D deficiency	U	υ	υ	U	U	Y	Y	υ	Y	There is uncertainty about whether vitamin D deficiency is a cause or effect of disease. Randomised trials to clarify this relationship are currently underway.	Y	U	Vitamin D deficiency is higher in Scotland. The trends in this are unknown.	Y	U	The meteorological data suggest that Glasgow has lower exposure to sunlight than Liverpool and Manchester (but not Belfast), but no vitamin D data are available.	
A7 Climate: winter deaths	*	*	*	*	*	*	*	*	*	As the hypothesis relates to a particular cause of death, the criteria for causality are less easily applied in general terms.	N	U	Recent data show (crudely) similar numbers of winter deaths. However, historical data are not readily available.	N	U	Recent data show (crudely) similar numbers of winter deaths to Liverpool (although they were higher than Manchester). Historical data are not readily available.	

Exposure/ hypothesis		Gene	eral	asse	ssme	ent o	f like	eliho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland	Specific to Glasgow			
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments	
A8 Culture of dependency	*	*	*	*	*	*	*	*	*	There is little evidence for the existence of such a culture: this makes assessment using the Bradford Hill criteria impossible. The evidence base around this hypothesis is politicised and contested, but there is little evidence to support the existence of such cultures or evidence to support such a culture as a cause of poverty or ill- health.	U	Y	We have not examined data to compare measures at the Scotland level. Given the lack of evidence for the theory, it is an unlikely contributor.	Ν	Y	For those markers of dependency that the advocates argue for, there is little difference between the cities. However, these markers did increase at the time of the emergence of the excess.	

Exposure/ hypothesis		Gene	erala	asse	ssme	ent o	f like	eliho	od of	f causality in terms of Bradford Hill criteria		Specific	c to Scotland	Specific to Glasgow			
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments	
A9 Culture of substance misuse	U	U	U	U	U	Y	Y	U	Y	The evidence about the importance of substance misuse cultures in determining the mortality profile of a population is mixed and disputed. There are historical examples (e.g. at the time of the industrial revolution and in the early 1990s in the former USSR) where alcohol cultures are said to have played an important role in determining the mortality profile of the population.	U	U	There are some, limited, differences in alcohol cultures between Scotland and England & Wales, although these are not thought to be overly influential in explaining difference over time. We have not identified comparative evidence for other substance misuse cultures.	U	U	We have not identified any comparative evidence looking at differences in the cultures of substance misuse for Glasgow relative to other populations.	
A10 Early years: family, gender relations and parenting differences	Y	Y	Y	Y	Y	Y	Y	Y	Y	Early years' experiences are well evidenced to be causally related to subsequent health outcomes.	N	U	From the limited data available, there are few differences evident, although these are limited in their scope.	N	U	From the limited data available, there are few differences evident, although these are limited in their scope.	

Exposure/ hypothesis		Gene	erala	asse	ssm	ent o	f like	eliho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland	Specific to Glasgow			
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments	
A11 Educational attainment	Y	Y	Y	Y	Y	Y	Y	Y	Y	Educational attainment is causally linked to subsequent health outcomes.	Y	U	Scotland is worse than England & Wales in terms of the percentage of the population with no educational qualifications; however this accounts for only a small proportion of the excess mortality.	Y	U	Glasgow is worse than Liverpool and Manchester, especially among the most deprived, in terms of the percentage of the population with no educational qualifications; however, it is likely that this accounts for only a small proportion of the excess mortality.	
A12 Employment/ labour market	Y	Y	Y	Y	Y	Y	Y	Y	Y	Employment, particularly high quality jobs, are known to be protective for health.	U	U	There was insufficient data identified to be able to compare Scotland with England & Wales across a range of employment indicators, although where data were available, few differences were identified.	U	U	There was insufficient data identified to be able to compare Glasgow with Liverpool and Manchester across a range of employment indicators, although where data were available, few differences were identified.	

Exposure/ hypothesis		Gene	eral	asse	ssme	ent o	f like	eliho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland	Specific to Glasgow			
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments	
A13 Ethnicity	Y	Y	Y	Y	Y	Y	Y	Y	Y	Ethnic diversity, especially when associated with economic in-migration and the healthy migrant effect, is known to confer low mortality risks.	Y	Y	Scotland has lower ethnic diversity and this difference emerged prior to the emergence of the excess.	Y	Y	Glasgow's ethnic diversity is less than Manchester's, but not Liverpool's.	
Exposure/ hypothesis		Gen	eral a	asse	ssme	ent o	f like	liho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland		Speci	fic to Glasgow	
-------------------------	-------------------------	-------------	-------------	-------------	----------------------------	--------------	-----------	------------	---------	---	---	--	--	--	--	---	
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments	
A14 Genetics	Y	Y	Y	Y	Y	Y	Y	Y	Y	There is some evidence, for some populations, of increased risk of disease, or increased susceptibility to particular exposures (e.g. in relation to breast cancer)	U	Y	It is unlikely that the wide range of causes that are responsible for the excess could all be due to genetic factors. The change in outcomes within a single generation make changes in the genetic make-up of the population unlikely, although pre-existing weaknesses may have become apparent as exposure changes. Out- migrants from the Scottish population retain a higher mortality risk which may reflect genetics, early years' experiences or retained cultures.	U	Y	It is unlikely that the wide range of causes that are responsible for the excess could all be due to genetic factors. The change in outcomes within a single generation make changes in the genetic make-up of the population unlikely, although pre-existing weaknesses may have become apparent as exposure changes. Out- migrants from the Scottish population retain a higher mortality risk which may reflect genetics, early years' experiences or retained cultures.	

Exposure/ hypothesis		Gen	erala	asse	ssme	ent o	f like	liho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland		Speci	fic to Glasgow
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A15 Health & social services	Y	Y	Y	Y	Y	Y	Y	Y	Y	Various aspects of healthcare, and healthcare systems, are evidenced to improve health.	U	U	From the limited available evidence, which looks at a very limited range of measures, there are no differences evident. There is a particular gap in relation to social services.	U	U	No data were identified to be able to compare Glasgow with elsewhere.
A16 Health behaviours – alcohol	Y	Y	Y	Y	Y	Y	Y	Y	Y	Alcohol is a known determinant of health.	Y	Y	Alcohol-related deaths are higher in Scotland and increased at the time of the excess.	Y	Y	Alcohol-related deaths are higher in Glasgow compared with elsewhere and increased at the time of the excess.
A17 Health behaviours – diet	Y	Y	Y	Y	Y	Y	Y	Y	Y	Diet is a known determinant of health.	Y	U	The limited data available suggest that some aspects of the Scottish diet are worse than that in England & Wales. Few trend data were available.	Ν	U	The very limited data available suggest no differences in diet, but this was for a very limited range of measures.

Exposure/ hypothesis		Gen	eral	asse	ssme	ent o	f like	liho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland		Speci	fic to Glasgow
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A18 Health behaviours – drug misuse	Y	Y	Y	Y	Y	Y	Y	Y	Y	Within the context of current drugs policy, illicit drug use is associated with negative health outcomes.	Y	Y	Drug-related deaths in Scotland are higher, and there is some evidence to suggest that drug misuse in Scotland currently accounts for a greater number of deaths than this narrow definition accounts for.	Y	Y	Drug-related deaths in Scotland are higher, and there is some evidence to suggest that drug misuse in Scotland currently accounts for a greater number of deaths than this narrow definition accounts for.
A19 Health behaviours – physical activity	Y	Y	Y	Y	Y	Y	Y	Y	Y	There is high quality evidence showing that physical activity is causally protective to health.	N	U	Physical activity levels, from the limited data available, seem to be similar in Scotland and the comparator areas.	N	U	Physical activity levels, from the limited data available, seem to be similar in Glasgow and the comparator areas.
A20 Health behaviours – smoking	Y	Y	Y	Y	Y	Y	Y	Y	Y	There is high quality evidence which shows the negative health impacts of smoking.	Y	Y	Smoking prevalence is higher in Scotland, and has been for at least 40 years. However, the modelled impact of this higher prevalence explains only a small part of the excess.	Ν	U	Smoking prevalence in Glasgow is very similar to that in Liverpool and Manchester.

Exposure/ hypothesis		Gene	erala	asse	ssmo	ent o	f like	eliho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland		Speci	fic to Glasgow
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A21 Housing quality and provision	Y	Y	Y	Y	Y	Y	Y	Y	Y	Housing availability and quality is a known and well-evidenced determinant of health.	Y	Y	Until very recently, overcrowding, and possibly dampness, were worse in Scotland. We have been unable to identify other data sources that would provide reliable comparisons between Scotland and England & Wales.	Y	Y	Overcrowding was worse in Glasgow compared with Liverpool and Manchester over the period of (and decades before) the emergence of excess mortality. We have been unable to identify other data sources with which to meaningfully and reliably compare other aspects of housing quality.
A22 Impacts of the world wars	*	*	*	*	*	*	*	*	*	As the hypothesis relates to a particular cause of death, the criteria for causality are less easily applied in general terms.	U	Y	There is uncertainty about whether the relative mortality rate during the wars was different, and therefore the temporality of an unknown exposure becomes less relevant.	U	Y	There is uncertainty about whether the relative mortality rate during the wars was different, and therefore the temporality of an unknown exposure becomes less relevant.

Exposure/ hypothesis		Gen	eral	asse	ssme	ent o	f like	eliho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland		Speci	fic to Glasgow
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A23 Income inequalities	Y	Y	Y	Y	Y	Y	Y	Y	Y	Although contested, there is a substantial evidence base linking income inequalities with mortality. The extent to which this might be conferred by poverty levels as a result of income inequality is the main ongoing debate.	Ν	Y	Income inequality in Scotland is lower than the rest of the UK, but did rise from the late 1970s onwards.	Ν	Y	Glasgow's income inequalities are similar to those in NW England.

Exposure/ hypothesis	G	iene	ral a	sses	ssme	ent o	f like	lihoo	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland		Spec	fic to Glasgow
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A24 Individual values	Y	U	Y	Y	Y	Y	Y	U	Y	There are a number of components to this hypothesis, several of which (e.g. optimism, materialism and self-efficacy) have been associated with differences in health outcomes.	U	U	There are no data available to assess differences between Scotland and England & Wales.	Ν	U	For the vast majority of the components of this hypothesis (optimism, self- efficacy, hedonism, time preferences etc) there is no evidence of the population in Glasgow being associated with more 'negative' individual values. The exceptions are individualism and materialism: however, the differences in relation to individualism arguably relate more to the 'reciprocity' aspect of social capital (discussed elsewhere in this report), while the evidence for differences in materialism is extremely limited. No trend data were available.

Exposure/ hypothesis		Gen	eral	asses	ssme	ent o	f like	liho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland		Spec	ific to Glasgow
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A25 Lagged effects of poverty and deprivation	Y	Y	Y	Y	Y	Y	Y	Y	Y	Poverty and deprivation across the life- course are known to be detrimental to the mortality profile of populations.	Y	Y	Carstairs deprivation measures from 1981, and overcrowding data prior to this, suggest that the population of Scotland has historically been exposed to greater deprivation than elsewhere.	Y	Y	Not true of income or employment based measures; however, Glasgow's overcrowding data from 1951 onwards suggest that the residents of the city have been exposed to relatively worse levels of deprivation historically.

Exposure/ hypothesis		Gene	eral	asse	ssme	ent o	f like	eliho	od of	causality in terms of Bradford Hill criteria		Specific	c to Scotland		Spec	ific to Glasgow
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A26 Migration	Y	Y	Y	Y	Y	Y	Y	Y	Y	Selective migration can be detrimental to the health of the donor population, and beneficial to the recipient population.	U	Y	Examination of longitudinal data have shown that, from the 1980s onwards, selective migration has not been responsible for the excess.	Y	Y	Examination of longitudinal data have shown that, from the 1980s onwards, selective migration has not been responsible for the excess. However, selective migration to the New Towns, prior to the 1980s, suggests that migration could be important in explaining the excess through an earlier exposure.
A27 Obesity	Y	Y	Y	Y	Y	Y	Y	Y	Y	There is good evidence to suggest that obesity is causally related to ill-health and mortality.	Y	U	For the short time frame of data available, there has been a slightly higher prevalence of obesity in Scotland compared with England & Wales, but this did not explain any of the excess in modelling.	N	U	Obesity levels in Glasgow are similar to those in Liverpool and Manchester. No trend data are available.

Exposure/ hypothesis		Gene	erala	asse	ssme	ent o	f like	lihoo	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland		Speci	fic to Glasgow
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A28 Political influences and vulnerability	Y	Y	Y	Y	Y	Y	Y	Y	Y	There are reviews showing that neoliberal politics have been detrimental to health in different contexts over time.	Y	Y	There is evidence that there was greater vulnerability to, and implementation of, neoliberal approaches.	Y	Y	There is evidence that there was greater vulnerability to, and implementation of, neoliberal approaches.
A29 Premature and low birthweight babies	Y	Y	Y	Y	Y	Y	Y	Y	Y	Prematurity and low birthweight is a known causal contributor to subsequent negative health outcomes.	N	U	For the short time periods available, there are no substantial differences in these outcomes for Scotland.	N	U	For the short time periods available, there are no substantial differences in these outcomes for Glasgow.
A30 Quality of external physical environment: land contamination	U	U	U	U	U	Y	Y	U	Y	The available evidence around the importance of contaminated land in adverse health outcomes is contested and often of poor quality. The outcomes most cited in relation to contaminated land are not those most prominent in the excess.	U	U	There are no comparative data available on exposure to contaminated land. However, the causes of death hypothesised to be related to this exposure are not an important component of the excess.	U	U	Although there are no comparative data available on exposure, the available evidence suggests that contaminated land is not responsible for a substantial burden of ill- health in Glasgow.

Exposure/ hypothesis		Gen	eral	asse	ssme	ent o	f like	eliho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland		Speci	fic to Glasgow
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A31 Quality of external physical environment: vacant & derelict land	Y	υ	Y	Y	Y	Y	Y	Y	Y	There is evidence that living near derelict land is detrimental to health, although part of that may be confounded by the processes of deindustrialisation and slum clearance which generates this dereliction.	U	U	Data for all urban areas in England & Wales and Scotland have not been compared.	Y	U	There is more derelict and vacant land in Glasgow than in Liverpool and Manchester. Trend data on this are not readily available.

Exposure/ hypothesis		Gen	eral	asse	ssme	ent o	f like	eliho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland		Speci	fic to Glasgow
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A32 Scale of deindustrialisatio n	Y	Y	Y	Y	Y	Y	Y	Y	Y	The links between deindustrialisation, employment, poverty and other social changes are well understood.	Y	Y	Scotland deindustrialised to a greater extent than England & Wales, although the effect of this on mortality is likely to be mediated by greater deprivation and thereby accounted for in the mortality modelling.	Ν	Y	Glasgow deindustrialised to a similar extent to Liverpool and Manchester. The analyses of deindustrialising areas across Europe indicate that this alone is unlikely to explain the excess compared with those areas, but it is likely to be part of the explanation in combination with other factors.

Exposure/ hypothesis		Gen	eral a	asse	ssme	ent o	f like	liho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland		Speci	fic to Glasgow
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A33 Scale and nature of post- war urban change	Y	Y	Y	Y	Y	Y	Y	Y	Y	The literature review described in this appendix highlights evidence that post- war urban change had the potential to influence population health in several ways, especially through the important social determinants of housing, living conditions and social and community networks.	U	Y	We have not examined the data specifically for Scotland in comparison with England & Wales.	Y	Y	Overcrowding was higher, and the urban change resulting from policies to tackle this was more profound in Glasgow than in Liverpool or Manchester.

Exposure/ hypothesis		Gene	erala	asse	ssme	ent o	f like	eliho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland	Specific to Glasgow			
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments	
A34 Sectarianism	U	U	U	U	U	Y	Y	U	Y	There is little high quality evidence linking sectarianism with poor health; however, it remains a theoretically possible and plausible mechanism.	U	Y	There are no data available to facilitate comparison between Scotland and England & Wales.	U	Y	There are no data available for Glasgow, Liverpool and Manchester on this measure; however, Glasgow's mortality profile is worse than Belfast's (a city which undoubtedly suffers from greater levels of religious sectarianism).	
A35 Sense of coherence	Y	U	Y	Y	Y	Y	Y	Y	Y	There is high quality evidence showing cross-sectional associations between sense of coherence (SoC) and health outcomes, but little evidence on changes in SoC and its impact.	U	U	No data are available to compare SoC in Scotland with England & Wales.	N	U	The three cities survey shows that the SoC is higher in Glasgow compared with Liverpool and Manchester. No trend data are available.	

Exposure/ hypothesis		Gene	erala	asse	ssme	ent o	f like	liho	od of	causality in terms of Bradford Hill criteria		Specific	to Scotland	Specific to Glasgow			
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments	
A36 Social capital	Y	υ	Y	Y	Y	Y	Y	Y	Y	There is good evidence that high levels of social capital enhance health, but we did not identify evidence of changes in social capital and its health impact.	N	U	The limited available evidence suggests that social capital was not different in the early 2000s in Scotland.	Y	U	The three-cities survey suggests that some aspects of social capital in Glasgow are worse than Manchester and, especially, Liverpool.	
A37 Social mobility	Y	Y	Y	Y	Y	Y	Y	Y	Y	There is evidence that upward social mobility is good for health and vice versa.	Ν	Y	There is little or no difference in the changing social make-up of Scotland relative to England & Wales.	Ν	Y	Although there has been no specific study of levels of social mobility in Glasgow compared with Liverpool and Manchester, all available data strongly suggest that – as with Scotland compared with England & Wales – there are few differences in levels of social mobility across the cities.	

Exposure/ hypothesis		Gene	erala	asse	ssmo	ent o	flike	eliho	od of	causality in terms of Bradford Hill criteria	Specific to Scotland Specific to Glasgow					fic to Glasgow
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A38 Spatial patterning of deprivation	υ	U	U	υ	U	Y	Y	U	Y	Although there is evidence of associations between differences in the patterning of deprivation and differences in health outcomes, assessment of the evidence is hampered by a lack of consistency in terms of conflicting results (related to opposing theorised causal pathways) and the cross-sectional nature of the research.	U	Y	It is unknown whether the patterning of deprivation differs between Scotland and England & Wales; however, given the timing of housing construction, any emerging differences would have been prior to the outcomes manifesting.	Y	Y	Deprived areas in Glasgow were slightly more dispersed than in Liverpool or Manchester, however this did not explain the mortality patterns observed.
A39 Terminations of pregnancy	Y	Y	Y	Y	Y	Y	Y	Y	Y	There is evidence linking historical rates of terminations with subsequent health and social outcomes.	Differe nt	N	The timing of the 1967 Abortion Act suggests that changes in the rate of terminations could not have been responsible for the excess.	Differe nt	N	The timing of the 1967 Abortion Act suggests that changes in the rate of terminations could not have been responsible for the excess.

Exposure/ hypothesis		Gene	eral a	asse	ssme	ent o	f like	liho	od of	causality in terms of Bradford Hill criteria	Specific to Scotland Specific to Glasgow					
	Strength of association	Temporality	Consistency	Specificity	Biological gradient	Plausibility	Coherence	Experiment	Analogy	Comments	Hypothesised causal factor worse in Scotland?	Would any difference in exposure have occurred prior to the outcome?	Comments	Hypothesised causal factor worse in Glasgow?	Would any difference in exposure have occurred prior to the outcome?	Comments
A40 Water hardness	Y	U	Y	Y	Y	Y	Y	U	Y	The weight of evidence suggests that it is plausible (albeit that causal links are still disputed) that there is an association between softer drinking water (specifically relating to lower levels of magnesium) and higher risk of cardiovascular disease in populations.	Y	U	Water is softer, and cardiovascular disease is higher after adjustment, over time. We have not identified evidence about changes to water hardness in Scotland compared with elsewhere.	Y	U	Water is softer, and cardiovascular disease is higher after adjustment, over time. We have not identified evidence about changes to water hardness in Scotland compared with elsewhere.

Appendix C. Participants at June 2015 'workshop'.

Harry Burns, University of Strathclyde

Chik Collins, University of the West of Scotland

Peter Craig, MRC/CSO Social & Public Health Sciences Unit (SPHSU), University of Glasgow

Fiona Crawford, NHS Greater Glasgow & Clyde (NHSGGC)/Glasgow Centre for Population Health (GCPH)

Mike Danson, Heriot-Watt University

Sara Dodds, Scottish Government/GCPH

Flora Douglas, University of Aberdeen

Ruth Dundas, MRC/CSO SPHSU, University of Glasgow

John Frank, Scottish Collaboration for Public Health Research & Policy (SCPHRP)

Andrew Fraser, NHS Health Scotland (NHSHS)

Lisa Garnham, GCPH

Donald Henderson, Scottish Government

Russell Jones, GCPH

Lorna Kelly, GCPH

Justin Kenrick, University of Edinburgh

Alastair Leyland, MRC/CSO SPHSU, University of Glasgow

Mark Livingston, University of Glasgow

Mhairi Mackenzie, University of Glasgow

Alison McCallum, NHS Lothian

Gerry McCartney, NHSHS

Tom Moorhouse (retired)

Anita Morrison, Scottish Government

John O'Dowd, NHSGGC

Frank Popham, MRC/CSO SPHSU, University of Glasgow

Tony Robertson, SCPHRP

Pete Seaman, GCPH

Michael Smith, NHSGGC

Carol Tannahill, GCPH

Martin Taulbut, NHSHS

David Taylor-Robinson, University of Liverpool

Katharine Timpson, University of the West of Scotland

Jim Tomlinson, University of Glasgow

Katherine Trebeck, Oxfam

David Walsh, GCPH

Bruce Whyte, GCPH

References

¹ McCartney G., Walsh D., Whyte B., Collins C. Has Scotland always been the 'sick man' of Europe? An observational study from 1855 to 2006. European Journal of Public Health 2012; 22 (6): 756-760

² Whyte B., Ajetunmobi T. Still 'The Sick Man of Europe'? Scottish mortality in a European context 1950-2010: an analysis of comparative mortality trends. Glasgow: GCPH; 2012. <u>http://www.scotpho.org.uk/downloads/scotphoreports/scotpho061107_scotmort_rep.pdf</u> (Accessed November 2015)

³ WHO Commission on Social Determinants of Health. Closing the gap in a generation: health equity through action on the social determinants of health. Geneva: World Health Organization; 2008

⁴ Hanlon P, Walsh D, Whyte B. Let Glasgow Flourish. Glasgow: GCPH; 2006.

⁵ Norman P, Boyle P, Exeter D, Feng Z, Popham F. Rising premature mortality in the UK's persistently deprived areas: only a Scottish phenomenon? *Social Science & Medicine* 2011;73(11):1575-1584.

⁶ Shaw M, Dorling D, Gordon D, Davey Smith G. The Widening Gap: health inequalities and policy in Britain (2nd edition). Bristol: The Policy Press; 1999.

⁷ Eikemo T.A., Mackenbach J.P. (Eds). EURO GBD SE: the potential for reduction of health inequalities in Europe. Final Report. University Medical Center Rotterdam; 2012.

⁸ Popham F., Boyle P. Assessing socio-economic inequalities in mortality and other health outcomes at the Scottish national level. Final report for the Scottish Collaboration for Public Health Research and Policy. University of St. Andrews; 2011. Available from: <u>http://www.scphrp.ac.uk/wp-content/uploads/2014/05/Assessing-socio-economic-</u> inequalities-in-mortality-and-other-health-outcomes.pdf (Accessed January 2016).

⁹ Taulbut M., Walsh D., McCartney G., Parcell S., Hartmann A., Poirier G., Strniskova D., Hanlon P. Spatial inequalities in life expectancy within post-industrial regions of Europe. BMJ Open 2014; 4(6): e004711.

¹⁰ Carstairs V and Morris R. Deprivation: explaining differences in mortality between Scotland and England and Wales. *BMJ* 1989;299(6704):886–889.

¹¹ Scottish Office. *Towards a healthier Scotland – a white paper on health*. The Stationary Office, 1999.

¹² Scottish Executive. *Social justice...a Scotland where everyone matters*. Annual report 2000. Edinburgh: Scottish Executive, 2000.

¹³ Scottish Council Foundation. *The Scottish Effect?* Edinburgh: Scottish Council Foundation, Healthy Public Policy Network, 1998.

¹⁴ Scottish Executive Office of the Chief Statistician. Scottish Index of Multiple Deprivation
 2004. Scottish Executive; 2004. Available at:
 http://www.gov.scot/Publications/2004/10/20089/45173 (accessed January 2016).

¹⁵ Health & Wellbeing Profiles 2015; The Scottish Public Health Observatory (ScotPHO). <u>http://www.scotpho.org.uk/opt/Reports/ScotPHO-Health-Wellbeing-Report-2015-150731-</u> web.pdf (accessed January 2016).

¹⁶ Kelso L, Walsh D, Whyte B. Constituency Health & Well-Being Profiles. NHS Health Scotland; 2004.

¹⁷ Leyland AH, Dundas R, McLoone P, Boddy FA. Inequalities in mortality in Scotland 1981-2001. Glasgow: MRC Social and Public Health Sciences Unit; 2007.

¹⁸ Walsh D, Taulbut M, Hanlon P. The aftershock of deindustrialization – trends in mortality in Scotland and other parts of post-industrial Europe. *European Journal of Public Health* 2010;20(1):58-64.

¹⁹ Walsh D., Taulbut M. and Hanlon P. The aftershock of deindustrialisation – trends in mortality in Scotland and other parts of post-industrial Europe. Glasgow: Glasgow Centre for Population Health; 2008.

²⁰ Taulbut M., Walsh D., Parcell S., Hanlon P., Hartmann A., Poirier G., Strniskova D. Health and its determinants in Scotland and other parts of post-industrial Europe: the 'Aftershock of Deindustrialisation' study – phase two. Glasgow: GCPH; 2011.

²¹ Hanlon P, Lawder RS, Buchanan D, Redpath A, Walsh D, Wood R, Bain M, Brewster DH, Chalmers J. Why is mortality higher in Scotland than in England & Wales? Decreasing influence of socioeconomic deprivation between 1981 and 2001 supports the existence of a 'Scottish Effect'. *Journal of Public Health* 2005;27(2):199-204.

²² Popham F, Boyle PJ. Is there a 'Scottish effect' for mortality? Prospective observational study of census linkage studies. *Journal of Public Health* 2011;33(3):453-458.

²³ Popham F, Boyle PJ, Norman P. The Scottish excess in mortality compared to the English and Welsh. Is it a country of residence or country of birth excess? *Health & Place* 2010;16(4):759-762.

²⁴ McCartney G., Russ T.C., Walsh D., Lewsey J., Smith M., Davey Smith G., Stamatakis E., Batty G.D. Explaining the excess mortality in Scotland compared with England: pooling of 18 cohort studies. Journal of Epidemiology & Community Health, 2014 doi:10.1136/jech-2014-204185

²⁵ Schofield L., Walsh D., Munoz-Arroyo R. *et al.* Dying younger in Scotland: trends in mortality and deprivation relative to England and Wales, 1981-2011'. Submitted to Health & Place, 2015

²⁶ Connolly S., Rosato M., Kinnear H., O'Reilly D. Variation in mortality by country of birth in

Northern Ireland: a record linkage study. Health & Place 2011; 17(3): 801-6.

²⁷ Reid J. Excess mortality in the Glasgow conurbation: exploring the existence of a 'Glasgow effect'. Glasgow: University of Glasgow; 2009.

²⁸ Landy R, Walsh D, Ramsay J. Do socio-economic, behavioural and biological risk factors explain the poor health parole of the UK's sickest city? *Journal of Public Health* 2012;34(4):591-598.

²⁹ Walsh D, Bendel N, Jones R, Hanlon P. It's not 'just deprivation': Why do equally deprived UK cities experience different health outcomes? *Public Health* 2010;124(9):487-495.

³⁰ Walsh D, Bendel N, Jones R, Hanlon P. Investigating a 'Glasgow Effect': Why do equally deprived UK cities experience different health outcomes? Glasgow: GCPH; 2010.

³¹ Graham P, Walsh D, McCartney G. Shipyards and sectarianism: how do mortality and deprivation compare in Glasgow and Belfast? *Public Health* 2012; 126(5): 378–385.

³² Gray L. Comparisons of health-related behaviours and health measures between Glasgow and the rest of Scotland. Glasgow: Glasgow Centre for Population Health; 2007.

³³ Seaman R., Mitchell R., Dundas R., Leyland A.H., Popham F. How much of the difference in life expectancy between Scottish cities does deprivation explain? BMC Public Health 2015; 15:1057

³⁴ Whynes D.K. Deprivation and self-reported health: are there 'Scottish effects' in England and Wales? Journal of Public Health 2008; 31(1): 147–153.

³⁵ Phillimore P.R., Morris D. Discrepant legacies: premature mortality in two industrial towns. Social Science & Medicine 1991; 33(2): 139-152.

³⁶ Barker, D., Osmond, C. Inequalities in health in Britain: specific explanations in three Lancashire towns. BMJ 1987; 294: 749 - 752.

³⁷ Young H., Grundy E., O'Reilly D., Boyle P. Self-rated health and mortality in the UK: results from the first comparative analysis of the England and Wales, Scotland, and Northern Ireland Longitudinal Studies. Population Trends 2010; 139: 11-36.

³⁸ Idler E.L., Benyamini Y. Self-rated health and mortality: a review of twenty-seven community studies. Journal of Health and Social Behavior 1997; 38: 21-37.

³⁹ Fayers, P., Sprangers, M. Understanding self-rated health. Lancet 2002; 359: 187-188.

⁴⁰ OECD. Self-Reported Health and Disability. OECD Social Issues/Migration/Health 2010; 21(2): 34-35.

⁴¹ Jürges H. True health vs response styles: exploring cross-country differences in selfreported health. Health Economics 2007; 16: 163-178.

⁴² O'Reilly D., Rosato M. Dissonances in self-reported health and mortality across

denominational groups in Northern Ireland. Social Science & Medicine 2010; 71: 1011-1017.

⁴³ Salomon J.A., Nordhagen S., Oza S., Murray C.J. Are Americans feeling less healthy? The puzzle of trends in self-rated health. American Journal of Epidemiology 2009; 170: 343–351.

⁴⁴ Bostan C., Oberhauser C., Stucki G., Bickenbach J., Cieza A. Biological health or lived health: which predicts self-reported general health better? BMC Public Health 2014; 14: 189.

⁴⁵ Murray C., Chen L. Understanding morbidity change. Population and Development Review 1992; 18: 481-503.

⁴⁶ Dorling D., Barford A., The inequality hypothesis: Thesis, antithesis, and a synthesis?
 Health & Place 2009;15(4): 1166-9.

⁴⁷ Rees P. Counting people: past, present and future. University of Leeds Review 1993; 36: 247-273.

⁴⁸ Gould M. I., Jones K. Analysing perceived limiting long-term illness using UK Census Microdata. Social Science & Medicine 1996; 42: 857-869.

⁴⁹ Howe G. M. Does it matter where I live? Transactions of the Institute of British Geographers 1986; 11: 387-414.

⁵⁰ McCartney G., Collins C., Walsh D., Batty G.D. Accounting for Scotland's Excess Mortality: Towards a Synthesis. Glasgow Centre for Population Health, April 2011.

⁵¹ McCartney G., Collins C., Walsh D., Batty G.D. Why the Scots die younger: Synthesizing the evidence. Public Health (2012), doi:10.1016/j.puhe.2012.03.007

⁵² Taulbut M, Walsh D. Poverty, parenting and poor health: comparing early years' experiences in Scotland, England and three city regions. Glasgow, Glasgow Centre for Population Health, 2013.

⁵³ Rush L, McCartney G, Walsh D, MacKay D. Vitamin D and subsequent all-age and premature mortality: a systematic review. *BMC Public Health* 2013, 13: 679.

⁵⁴ Walsh D, McCartney G. Trends in terminations of pregnancy in Glasgow, Liverpool and Manchester. *Public Health* 2013; 127: 143-152.

⁵⁵ McCartney G, Shipley M, Hart C, Smith GD, Kivimaki M, Walsh D, Watt GC, Batty GD. Why Do Males in Scotland Die Younger Than Those in England? Evidence From Three Prospective Cohort Studies. *PLoS One* 2012; 7(7): e38860. doi:10.1371/journal.pone.0038860.

⁵⁶ Stanners G., Walsh D., McCartney G. Is 'excess' mortality in Glasgow an artefact of measurement? Submitted to Public Health, 2014

⁵⁷ Walsh D, McCartney G, McCullough S, *et al.* Exploring potential reasons for Glasgow's 'excess' mortality: results of a three city survey of Glasgow, Liverpool and Manchester. Glasgow, Glasgow Centre for Population Health, 2013.

⁵⁸ Shipton D, Whyte B, Walsh D. Alcohol-related mortality in deprived UK cities: worrying trends in young women challenge recent national downward trends. al. *J Epidemiol Community Health* 2013; 0:1–8. doi:10.1136/jech-2013-202574.

⁵⁹ Livingston M, Walsh D, Whyte B, Bailey N. The spatial distribution of deprivation. Glasgow, Glasgow Centre for Population Health, 2013.

⁶⁰ Tunstall H, Mitchell R, Gibbs J, Platt S, Dorling D. Socio-demographic diversity and unexplained variation in death rates among the most deprived parliamentary constituencies in Britain. Journal of Public Health 2011; 34(2): 296–304.

⁶¹ Ji C, Kandala NB, Cappuccio FP. Spatial variation of salt intake in Britain and association with socioeconomic status. *BMJ Open* 2013; 3: e002246.doi:10.1136/bmjopen-2012-002246.

⁶² Desai M, Nolte E, Karanikolos M, Khoshaba B, McKee M. Measuring NHS performance 1990–2009 using amenable mortality: interpret with care. *J R Soc Med* 2011: 104: 370–379.

⁶³ Brown D, O'Reilly D, Gayle V, Macintyre S, Benzeval M, Leyland AH. Socio-demographic and health characteristics of individuals left behind. Health &Place 2012; 18: 440–444.

⁶⁴ van der Pol M., Walsh D., McCartney G. Comparing time and risk preferences across three post industrial UK cities. Social Science & Medicine 2015; 140: 54-61

⁶⁵ Walsh D., McCartney G., McCullough S., van der Pol M., Buchanan D., Jones R. Always looking on the bright side of life? Exploring optimism and health in three UK post-industrial urban settings. Journal of Public Health 2015; doi: 10.1093/pubmed/fdv077

⁶⁶ Walsh D., McCartney G., McCullough S., van der Pol M., Buchanan D., Jones R. Comparing levels of social capital in three northern post-industrial UK cities. Public Health 2015; 129(6): 629-38

⁶⁷ Robinson M., Shipton D., Walsh D., Whyte B., McCartney G. Regional alcohol consumption and alcohol-related mortality in Great Britain: novel insights using retail sales data. BMC Public Health 2015; 15:1

⁶⁸ Walsh D., McCartney G., McCullough S., Buchanan D., Jones R. Comparing Antonovsky's Sense of Coherence scale across three UK post-industrial cities. BMJ Open 2014; 4:e005792 doi:10.1136/bmjopen-2014-005792

⁶⁹ Taulbut M., Walsh D., O'Dowd J. Comparing early years and childhood experiences and outcomes in Scotland England and three city-regions: a plausible explanation for Scottish 'excess' mortality? BMC Pediatrics 2014; 14:259

⁷⁰ Craig C. The Tears That Made The Clyde – Well-Being in Glasgow. Glendaruel: Argyll publishing; 2010.

⁷¹ Mackie P., Sim F. We need to talk about Kelvingrove. Public Health 2010; 124: 485-486.

⁷² George S. It's not just deprivation - or is it? Public Health 2010; 124: 496-497.

⁷³ Donnelly P.D. Explaining the Glasgow effect: could adverse childhood experiences play a role? Public Health 2010; 124: 498-499.

⁷⁴ Gordon D.S. We need to look to broad horizons to understand (and change) health. Public Health 2010; 124: 716-717.

⁷⁵ Wilkinson J.R. What we want to know is... Public Health 2010; 124: 718-719.

⁷⁶ Hussey R., Hennell T. It's not just deprivation. Public Health 2011; 125: 114-115.

⁷⁷ Scottish Government. Health in Scotland 2008 – Annual Report of the Chief Medical Officer. Edinburgh: Scottish Government; 2009.

⁷⁸ Scottish Government. Health in Scotland 2009 Time for change – Annual Report of the Chief Medical Officer. Edinburgh: Scottish Government; 2010.

⁷⁹ Collins C., MacKenzie M., McCartney G. (Eds). Accounting for Scotland's Excess Mortality: Towards a Synthesis - Commentaries and Responses. Glasgow: Glasgow Centre for Population Health; 2014. Available from:

⁸⁰ Bagnall A.M., Raine G., Jones R., Mitchell B., White A.K. A systematic review of proposed explanations for "excess" mortality. Leeds: Institute for Health and Wellbeing, Leeds Beckett University; 2015

⁸¹ Hill A. The environment and disease: association or causation? Proc R Soc Med 1965; 58: 295e300

⁸² Rothman K.J., Greenland S. Modern Epidemiology (2nd edition). Philadelphia: Lippincott-Raven; 1998

⁸³ Identifying the environmental causes of disease: how should we decide what to believe and when to take action? . London: Academy of Medical Sciences; 2007.

⁸⁴ Gordis L. Epidemiology. Philadelphia: Elsevier Saunders; 2013.

⁸⁵ Rutherford L., Sharp C., Bromley C (Eds). The Scottish Health Survey 2011. Edinburgh:
 Scottish Government; 2012. Available from:
 http://www.gov.scot/Resource/0040/00402630.pdf (Accessed January 2016)

⁸⁶ Campbell M., Ballas D., Dorling D., Mitchell R., Mortality inequalities: Scotland versus England and Wales, Health & Place 2013; 23: 179-86.

⁸⁷ McLoone P. Increasing mortality among adults in Scotland 1981-1999. European Journal of Public Health 2003; 13(3): 230.

⁸⁸ Dahlgren G., Whitehead M. Tackling inequalities in health: what can we learn from what has been tried? Working paper prepared for the King's Fund International Seminar on

Tackling Inequalities in Health, September 1993. Ditchley` Park, Oxfordshire: King's Fund; 1993.

⁸⁹ Dahlgren G., Whitehead M. European strategies for tackling social inequities in health: levelling up, Part 2. Copenhagen: WHO Regional Office for Europe; 2007.

⁹⁰ VanLeeuwen J.A., Waltner-Toews D., Abernathy T. *et al*. Evolving models of human health toward an ecosystem context. Ecosystem Health 1999; 5: 239–254.

⁹¹ Blum H.L. Planning for Health: Developmental Application of Social Change Theory. New York, NY: Human Sciences Press; 1974.

⁹² Morris J.N. Uses of Epidemiology. New York, NY: Churchill Livingstone Inc.; 1975.

⁹³ Travis J.W. Wellness for Helping Professionals. 3rd ed. Mill Valley, Calif: Wellness Associates; 1977.

⁹⁴ Hancock T., Perkins F. The mandala of health: a conceptual model and teaching tool.
 Health Education 1985; 24: 8–10.

⁹⁵ Hancock T. Health, human development and the community ecosystem: three ecological models. Health Promotion International 1993; 8: 41–46.

⁹⁶ Hamilton N., Bhatti T. Population Health Promotion: An Integrated Model of Population Health and Health Promotion. Ottawa, Ontario, Canada: Health Promotion Development Division, Health Canada; 1996.

⁹⁷ Starfield B., Shi L. Determinants of health: testing of a conceptual model. In: Adler N.E., Marmot M., McEwen B.S., Stewart J. (Eds.). Socioeconomic Status and Health in Industrial Nations: Social, Psychological, and Biological Pathways. New York, NY: New York Academy of Sciences; 1999.

⁹⁸ Nicholson J.M., Carroll J., Brodie A., Waters E., Vimpani G. Child and youth health inequalities: the status of Australian research 2003, report commissioned by the Australian Government Department of Health and Ageing for the Health Inequalities Research Collaboration, Children, Youth, and Families Network, Centre for Health Research, School of Public Health, Queensland University of Technology, Brisbane; 2004.

⁹⁹ Bronfenbrenner, U. The ecology of human development. Cambridge, MA: Harvard University Press; 1979.

¹⁰⁰ Lynch J.W. Social-epidemiology: some observations on the past, present and future. Australasian Epidemiologist 2000; 7: 7–15.

¹⁰¹ Centers for Disease Control and Prevention (CDC). The Social-Ecological Model: A Framework for Prevention. <u>http://www.cdc.gov/ViolencePrevention/overview/social-</u> <u>ecologicalmodel.html</u> (Accessed December 2015).

¹⁰² Bao W., Ma A., Mao L. *et al*. Diet and lifestyle interventions in postpartum women in

China: study design and rationale of a multicenter randomized controlled trial. BMC Public Health 2010; 10: 103.

¹⁰³ Evans R.G., Stoddart G.L. Producing health, consuming health care. *In:* Evans R.G., Barer M.L., Marmor T.R. (Eds). Why are some people healthy and others not? The determinants of health of populations. Berlin, New York: Walter de Gruyter; 1994.

¹⁰⁴ Evans R.G., Stoddart G.L. Producing health, consuming health care. Social Science & Medicine 1990; 31: 1347–1363

¹⁰⁵ Black D., Morris J., Smith C., Townsend P. Inequalities in health: report of a Research Working Group. London: Department of Health and Social Security; 1980

¹⁰⁶ Bambra C. Health inequalities and welfare state regimes: theoretical insights on a public health 'puzzle'. Journal of Epidemiology & Community Health 2011; 65(9): 740-5.

¹⁰⁷ McCartney G., Collins C., Mackenzie M. What (or who) causes health inequalities: theories, evidence and implications? Health Policy. 2013;113(3): 221-7

¹⁰⁸ Marmot M, Wilkinson RG (Eds). Social determinants of health: the solid facts. 2nd edition. Denmark: World Health Organization; 2003

¹⁰⁹ Link B.G, Phelan J.C. McKeown and the Idea That Social Conditions Are Fundamental Causes of Disease. American Journal of Public Health 2002; 92(5).

¹¹⁰ Scott S., Curnock E., Mitchell R., Robinson M., Taulbut M., Tod E., McCartney G. What would it take to eradicate health inequalities? Testing the fundamental causes theory of health inequalities in Scotland. Glasgow: NHS Health Scotland; 2013.

¹¹¹ Doyal L., Pennell I. The political economy of health. London: Pluto Press; 1979.

¹¹² Navarro V., Borrell C., Benach J. *et al*. The importance of the political and the social in explaining mortality differentials among the countries of the OECD, 1950–1998. International Journal of Health Services 2003; 33: 419–94.

¹¹³ Coburn D. Beyond the income inequality hypothesis: class, neo-liberalism, and health inequalities. Social Science & Medicine 2004; 58: 41–56.

¹¹⁴ Bambra C. Work, worklessness and the political economy of health inequalities. Journal of Epidemiology & Community Health 2011; 65: 746-750.

¹¹⁵ Mooney G. The health of nations: towards a new political economy. London: Zed Books Ltd; 2012.

¹¹⁶ Navarro V., Shi L. The political context of social inequalities and health. Social Science & Medicine 2001; 52(3): 481-91.

¹¹⁷ Navarro V. What we mean by social determinants of health. International Journal of Health Services 2009; 39(3): 423–441.

¹¹⁸ Beckfield J, Krieger N. Epi + demos + cracy: linking political systems and priorities to the magnitude of health inequities - evidence, gaps, and a research agenda. Epidemiologic Reviews 2009; 31: 152-77.

¹¹⁹ Collins C., McCartney G. The impact of neoliberal 'political attack' on health: the case of the 'Scottish effect'. International Journal of Health Services 2011; 41(3): 501-523.

¹²⁰ Bartley M. Unemployment and ill health: understanding the relationship. *Journal of Epidemiology and Community Health* 1994;48(4):333-337.

¹²¹ Dorling D. Unemployment and health. *BMJ* 2009;338:b829.

¹²² Waddell G, Burton AK. Is Work Good for Your Health and Well-being? London: Stationery Office; 2006.

¹²³ Lundberg O, Yngwe MA, Stjärne MK, Elstad JI, Ferrarini T, Kangas O, Norström T, Palme J, Fritzell J; NEWS Nordic Expert Group. The role of welfare state principles and generosity in social policy programmes for public health: An international comparative study. *Lancet* 2008;372(9650):1633-1640.

¹²⁴ Lynch JW, Davey Smith G, Kaplan GA, House JS. Income inequality and mortality: Importance to health of individual income, psychosocial environment, or material conditions. *BMJ* 2000;320(7243):1200-1204.

¹²⁵ Marmot M, Friel S, Bell R, Houweling TA, Taylor S; Commission on Social Determinants of Health. Closing the gap in a generation: Health equity through action on the social determinants of health. *Lancet* 2008;372(9650):1661-1669.

¹²⁶ Wilkinson R, Pickett K. The Spirit Level: Why More Equal Societies Almost Always Do Better. London: Penguin; 2009.

¹²⁷ Krieger N. Epidemiology and the People's Health. Theory and context. Oxford: Oxford University Press; 2011.

¹²⁸ Egan M, Tannahill C, Pettigrew M, Thomas S. Psychosocial risk factors in home and community settings and their associations with population health and health inequalities: a systematic meta-review *BMC Public Health* 2008;8:239.

¹²⁹ Marmot M, Wilkinson RG. Psychosocial and material pathways in the relation between income and health: A response to Lynch *et al. BMJ* 2001;322(7296):1233-1236.

¹³⁰ Dorling D., Rigby J., Wheeler B., Ballas D., Thomas B., Fahmy E., Gordon D. Lupton R.
 Poverty, wealth and place in Britain, 1968 to 2005 - Understanding the Transformation of the Prospects of Places. Bristol: The Policy Press; 2007. Available from:
 <u>http://sasi.group.shef.ac.uk/research/transformation/index.html (Accessed December 2015).</u>

¹³¹ Institute for Fiscal Studies (IFS): Inequality and Poverty Spreadsheet – accompaniment to IFS Commentary No. 124, 'Living Standards, Poverty and Inequality in the UK: 2012'. IFS;

2012. Available from: <u>http://www.ifs.org.uk/publications/6196</u> (Accessed October 2012)

¹³² Thomas B., Dorling D., Davey Smith G. Inequalities in premature mortality in Britain: observational study from 1921 to 2007. British Medical Journal 2010; 341: c3639.

¹³³ Dorling D. Injustice: why social inequality persists. Bristol: The Policy Press; 2009

¹³⁴ Walsh D. An analysis of the extent to which socio-economic deprivation explains higher mortality in Glasgow in comparison with other post-industrial UK cities, and an investigation of other possible explanations. University of Glasgow PhD thesis; 2014. Available from: <u>http://theses.gla.ac.uk/5489</u> (Accessed December 2015)

¹³⁵ Maver I. Glasgow. Edinburgh: Edinburgh University Press Ltd; 2000

¹³⁶ Damer S. Glasgow: Going For a Song. London: Lawrence & Wishart; 1990

¹³⁷ Lane T. Liverpool: Gateway of Empire. London: Lawrence & Wishart; 1987

¹³⁸ Misselwitz P. Liverpool City Profile. In: Shrinking Cities (Schrumpfende Städte),

Complete Works 1 – analysis. Aachen (Germany): ARCH+; 2006. Available from: www.shrinkingcities.com (Accessed May 2013)

¹³⁹ Kidd A. Manchester: a history. Lancaster: Carnegie Publishing; 1993

¹⁴⁰ Misselwitz P. Manchester – City Profile. In: Shrinking Cities (Schrumpfende Städte),
 Complete Works 1 – analysis. Aachen (Germany): ARCH+; 2006. Available from:
 www.shrinkingcities.com (Accessed May 2013).

¹⁴¹ Collins C, McCartney G, Walsh D, Levitt I. The Health of Cities: Variability, Vulnerability and Mortality. Submitted for publication and currently undergoing peer review

¹⁴² Wisner B. Sustainable Suffering? Reflections on Development and Disaster Vulnerability in the Post-Johannesburg World. Regional Development Dialogue. 2003;24:135-48

¹⁴³ Delor F, Hubert M. Revisiting the concept of `vulnerability'. Social Science and Medicine. 2000;50:1557-70

¹⁴⁴ Watts MJ, Bohle HG. Hunger, Famine and the Space of Vulnerability. GeoJournal. 1993;30(2):117-25

¹⁴⁵ Watts MJ, Bohle HG. The space of vulnerability: the causal structure of hunger. Progress in Human Geography. 1993;17(1):43-67

¹⁴⁶ Adger WN. Vulnerability. Global Environmental Change. 2006;16(3):268-81

¹⁴⁷ Turner BL, Kasperson RE, Matson PA, McCarthy JJ, Corell RW, Christensen L, *et al*. A framework for vulnerability analysis in sustainability science. Proceedings of the National Academy of Sciences. 2003;100(14):8074-9.

¹⁴⁸ Galea S, Freudenberg, N, Vlahov D. Cities and Population Health. *Social Science &Medicine* 2005; 60(5): 1017-1033.

¹⁴⁹ Galea S, Vlahov D. Urban Health: Evidence, Challenges, and Directions. *Annual Review of Public Health* 2005; 26: 341-365.

¹⁵⁰ Galea S, Ahern J, Nandi A, Tracy M, Beard J, Vlahov D. Urban Neighborhood Poverty and the Incidence of Depression in a Population-Based Cohort Study. *Annals of Epidemiology* 2007; 17(3): 171-179.

¹⁵¹ Gallopín, G. C. Linkages between vulnerability, resilience, and adaptive capacity. *Global Environmental Change* 2006; 16(3): 293-303.

¹⁵² Galea, S., Ahern, J., and Karpati, A. A model of underlying socioeconomic vulnerability in human populations: evidence from variability in population health and implications for public health. *Social Science and Medicine* 2005; 60(11): 2417-30.

¹⁵³ Collins C. and Levitt I. The 'modernisation' of Scotland and its impact on Glasgow, 1955-1979: 'unwanted side effects' and vulnerabilities. Scottish Affairs 2016; 25(3).

¹⁵⁴ Abercrombie P., Matthew R.H. Clyde Valley Regional Plan 1946. Edinburgh: HMSO; 1949

¹⁵⁵ Toothill J.N. Report on the Scottish Economy 1960-1961: Report of a Committee Appointed by the Scottish Council (Development and Industry) under the Chairmanship of J.N. Toothill Esq., CBE. Edinburgh: Scottish Council (Development and Industry); 1961

¹⁵⁶ Scottish Development Department. Central Scotland: A Programme for Development and Growth (Cmnd. 2188). Edinburgh: HMSO; 1963

¹⁵⁷ 'Understanding the Lagging Health Outcomes in West Central Scotland – An Applied Social Science Contribution'. PhD Studentship, School of Media, Culture and Society, University of the West of Scotland (part-funded by Public Health Observatory, NHS Health Scotland).

¹⁵⁸ James Arnott, Glasgow City Council. Personal communication. 2015

¹⁵⁹ Parr JB. Growth-pole Strategies in Regional Economic Planning: A Retrospective View.Part 2. Implementation and Outcome. Urban Studies. 1999;36(8):1247-68.

¹⁶⁰ Parr JB. Growth-pole Strategies in Regional Economic Planning: A Retrospective View: Part 1. Origins and Advocacy. Urban Studies 1999;36(7):1195-215

¹⁶¹ Firn JR. Industry. In: Smith R, Wannop U, editors. Strategic Planning in Action: The Impact of the Clyde Valley Regional Plan, 1946-1982. Aldershot: Gower; 1986. p. 100-38.

¹⁶² Foster J. The Economic Restructuring of the West of Scotland 1945-2000: Some Lessons from a Historical Perspective. In: Blazyca G, editor. Restructuring Regional and Local Economies: Towards a Comparative Study of Scotland and Upper Silesia. Aldershot: Ashgate; 2003.

¹⁶³ Foster J. The Twentieth Century, 1914-1979. In: Houston RA, Knox WWJ, editors. The New Penguin History of Scotland. London: Penguin Press/Museums of Scotland; 2001.

¹⁶⁴ Devine T. The Scottish Nation: 1700-2000. London: Penguin; 1999.

¹⁶⁵ Taulbut M., Walsh D., McCartney G. Collins C.. Excess mortality and urban change: Investigating similarities and differences in the extent of urban change in Glasgow, Liverpool and Manchester and their surrounding regions from 1945, and the extent to which this might be part of the excess mortality explanation. Edinburgh: NHS Health Scotland; 2016.

¹⁶⁶ Evans G, Wells NM, Moch A. Housing and mental health: a review of the evidence and a methodological and conceptual critique. Journal of Social Issues 2003; 59(3): 475-500.

¹⁶⁷ Moore NC. The personality and mental health of flat dwellers. British Journal of Psychiatry 1975; 128: 256-261.

¹⁶⁸ Hartig T, Mang M, Evans GW. Restorative effects of natural environment experiences. Environmental Behaviour 1991; 23(1): 3-26.

¹⁶⁹ McCormack C. The Wee Yellow Butterfly. Glendaruel: Argyll publishing; 2009.

¹⁷⁰ Collins C. Comparison of Liverpool and Glasgow in the 1970s and 1980s: Issues arising with potential relevance to causation of health differences. Report to NHS Health Scotland (as part of ongoing commissioned research), 2015.

¹⁷¹ Kidd A. Timeline: Manchester 1750-2002. In: Shrinking Cities (Schrumpfende Städte),
 Complete Works 1 – analysis. Aachen (Germany): ARCH+; 2006. Available from:
 www.shrinkingcities.com (Accessed May 2013).

¹⁷² Peck J., Ward K. (Eds). City of Revolution: Restructuring Manchester. Manchester: Manchester University Press; 2002.

¹⁷³ Carmichael P. Central-Local Government Relations in the 1980s: Glasgow and Liverpool Compared. Aldershot: Avebury; 1995.

¹⁷⁴ Parkinson M. Liverpool on the Brink: One City's Struggle Against Government Cuts. Hermitage, Berkshire: Policy Journals; 1985;

¹⁷⁵ Parkinson, M. Creative accounting and financial ingenuity in local government: the case of Liverpool. Public Money 1986; 5(4): 27-32

¹⁷⁶ Keating, M.J., Boyle, R. Remaking Urban Scotland: Strategies for Local Economic Development. Edinburgh: Edinburgh University Press; 1986.

¹⁷⁷ Boyle M., McWilliams C., Rice G. The spatialities of actually existing neoliberalism in
Glasgow, 1977 to present. Geografiska Annaler; series B, Human Geography 2008; 90: 31325.

¹⁷⁸ Boyle R. Changing Partners: The Experience of Urban Economic Policy in West Central

Scotland, 1980-90. Urban Studies 1993;30:309-24.

¹⁷⁹ Keating M. The City that Refused to Die. Glasgow: The Politics of Urban Regeneration. Aberdeen: Aberdeen University Press; 1988

¹⁸⁰ Boyle R. Partnership in Practice: An Assessment of Public-Private Collaboration in
 Regeneration - A Case Study of Glasgow Action. Local Government Studies. 1989;15(2):17 28.

¹⁸¹ Robson B. Those Inner Cities: Reconciling the Economic and Social Aims of Urban Policy. Oxford: Oxford University Press; 1989.

¹⁸² Keating M, Mitchell J. Easterhouse: An urban crisis. Strathclyde Papers on Government and Politics. 1986;47.

¹⁸³ Keating M, Mitchell J. Glasgow's Neglected Periphery: The Easterhouse and Drumchapel Initiatives. In: McCrone D, editor. Scottish Government Yearbook 1987. Edinburgh: Unit for the Study of Government in Scotland, University of Edinburgh; 1987.

¹⁸⁴ Keating M, Mitchell J. Inner City Decay: Glasgow sees need to look beyond the fringe. Municipal Journal. 1986:1312-3.

¹⁸⁵ Kemp A. The Hollow Drum: Scotland Since The War. Edinburgh: Mainstream Publishing; 1993

¹⁸⁶ Harvey C. Scotland after 1978 - from referendum to millennium. *In*: Houston R.A., Knox W.W.J. (Eds.) The New Penguin History of Scotland. London: Penguin Press; 2002.

¹⁸⁷ Harvie C. No Gods and Precious Few Heroes: Twentieth Century Scotland. Edinburgh:Edinburgh University Press; 1998

¹⁸⁸ Putnam R.D. Bowling alone: the collapse and revival of American community. New York, NY, USA: Simon and Schuster; 2000.

¹⁸⁹ Feeney M., Collins C. Tea in the Pot: Building 'social capital' or a 'great good place' in Govan? University of the West of Scotland-Oxfam Partnership, 2015. Available from: <u>http://uwsoxfampartnership.org.uk/wp-content/uploads/2014/10/Tea-in-the-Pot-Report-No.3-WEB1.pdf</u> (Accessed January 2016).

¹⁹⁰ Portes A. Social capital: its origins and applications in modern sociology. Annual Review of Sociology 1998; 24: 1-24

¹⁹¹ Islam M.K., Merlo J., Kawachi I., Lindström M., Gerdtham U.G. Social capital and health: Does egalitarianism matter? A literature review. International Journal for Equity in Health 2006; 5: 3

¹⁹² Jenkinson C.E., Dickens A.P., Jones K. *et al.* Is volunteering a public health intervention? A systematic review and meta-analysis of the health and survival of volunteers. BMC Public Health 2013; 13: 773.

¹⁹³ Low N, Butt S, Paine A, Smith J: Helping out: a national survey of volunteering and charitable giving. London, UK: National Centre for Social Research and the Institute for Volunteering Research; 2007.

¹⁹⁴ Dorling D., Gunnell D. Suicide: the spatial and social components of despair in Britain 1980–2000. Transactions of the Institute of British Geographers 2003; 28(4): 442–460

¹⁹⁵ Seaman P., Fiona E. Exploring socio-cultural explanations of Glasgow's 'excess' mortality. Glasgow: Glasgow Centre for Population Health; 2015. Available from: <u>http://www.gcph.co.uk/publications/548_exploring_socio-</u> <u>cultural_explanations_of_glasgow_s_excess_mortality</u> (Accessed January 2016).

¹⁹⁶ Townsend P. Deprivation. Journal of Social Policy 1987; 16: 124-46

¹⁹⁷ Townsend P., Phillimore P., Beattie A. Health and Deprivation: Inequality and the North. London: Croom Helm; 1988.

¹⁹⁸ Bailey N., Flint J., Goodlad R., Shucksmith M., Fitzpatrick S., Pryce G. Measuring Deprivation in Scotland: Developing a Long-Term Strategy - Final Report. Scottish Executive Central Statistics Unit; 2003. Available at: http://www.scotland.gov.uk/Publications/2003/09/18197/26547 (Accessed January 2016).

¹⁹⁹ Davey Smith G, Hart C, Hole D. Education and occupational social class: which is the more important indicator of mortality risk? J Epidemiol Community Health 1998;52:153–160.

²⁰⁰ Gordon, D., Adelman, A., Ashworth, K., Bradshaw, J.R., Levitas, R., Middleton, S., Pantazis,
 C., Patsios, D., Payne, S., Townsend, P. and Williams, J. Poverty and social exclusion in
 Britain. York: Joseph Rowntree Foundation; 2000

²⁰¹ MacInnes T., Tinson A, Hughes C., Born T.B., Aldridge H.. Monitoring poverty and social exclusion 2015. York: Joseph Rowntree Foundation 2015; 2015

²⁰² Palmer G. The Poverty Site: <u>www.poverty.org.uk</u> (Accessed: January 2016).

 ²⁰³ Scottish Government. Statistical Bulletin: Scottish Vacant & Derelict Land Survey 2013.
 Edinburgh: Scottish Government; 2014. Available from: http://www.gov.scot/<u>Resource/0044/00444542.pdf</u> (Accessed November 2015)

²⁰⁴ Office for National Statistics (ONS) Neighbourhood Statistics website (data extracted from the National Land Use Database of Previously-Developed Land (NLUD-PDL), 2010). Available from: <u>https://www.neighbourhood.statistics.gov.uk</u> (Accessed November 2015)

²⁰⁵ Understanding Glasgow – The Glasgow Indicators Website (from data from Scottish Vacant & Derelict Land Survey):

<u>http://www.understandingglasgow.com/indicators/environment/proximity_to_derelict_site</u> s/scottish_cities (Accessed January 2016).

²⁰⁶ European Environment Agency (EAA). Urban Atlas data. Available from: <u>http://www.eea.europa.eu/data-and-maps/data/urban-atlas</u>. (Accessed August 2015) ²⁰⁷ Croucher K., Myers L., Jones R., Ellaway A., Beck S. Health and the Physical Characteristics of Urban Neighbourhoods: a Critical Literature Review. Glasgow: Glasgow Centre for Population Health; 2007.

²⁰⁸ Cummins S., Stafford M., Macintyre S. *et al*. Neighbourhood environment and its association with self-rated health: evidence from Scotland and England. Journal of Epidemiology & Community Health 2005; 59: 207-231.

²⁰⁹ Duncan M., Spence J., Mummery W. Perceived environment and physical activity: a metaanalysis of selected environmental characteristics. International Journal of Behavioural Nutrition and Physical Activity 2005; 2(11).

²¹⁰ Mitchell R., Popham F. Greenspace, urbanity and health: Relationships in England. Journal of Epidemiology & Community Health 2007; 61(8): 681-683.

²¹¹ Mitchell R., Popham F. Effect of exposure to natural environment on health inequalities: an observational population study. Lancet 2008; 372: 1655-1660.

²¹² Lee A.C.K., Maheswaran R. The health benefits of urban green spaces: a review of the evidence. Journal of Public Health 2011; 33(2): 212–222.

²¹³ Office for National Statistics. 2011 census data for England & Wales. Available from: <u>http://www.ons.gov.uk/ons/guide-method/census/2011/census-data/index.html</u> (Accessed January 2016).

²¹⁴ Scotland's census 2011 website: <u>http://www.scotlandscensus.gov.uk/census-results</u> (Accessed January 2016).

²¹⁵ Graham H and Power C. *Childhood disadvantage and adult health: a lifecourse framework*. Health Development Agency, 2004.

²¹⁶ Lawlor DA, Batty GD, Morton SMB, Clark H, Macintyre S and Leon DA. Childhood socioeconomic position, educational attainment, and adult cardiovascular risk factors: the Aberdeen children of the 1950s cohort study. *American Journal of Public Health* 2005;95:1245-1251.

²¹⁷ Morris JN, Blane DB, and Whyte IR. Levels of mortality, education, and social conditions in 107 local education authority areas in England. *Journal of Epidemiology and Community Health* 1996;50:15-17.

²¹⁸ Whity G, Aggleton P, Gamarnikow and Tyler P. Education and Health Inequalities. Education Policy 1998;13(5):41-52.

²¹⁹ Strand BH, Grøholt EK, Steingrímsdóttir OA, Blakely T, Sidsel Graff-Iversen S, Næss Ø. Educational inequalities in mortality over four decades in Norway: prospective study of middle aged men and women followed for cause specific mortality, 1960-2000. *BMJ* 2010;340.

²²⁰ Bromley C, Cunningham SC. Growing Up In Scotland: Health Inequalities in the Early Years. Edinburgh: The Scottish Government, 2010.

²²¹ Connelly R., Sullivan A., Jerrim J. Primary and secondary education and poverty review.
 London: Centre for Longitudinal Studies, University of London; 2014. Available from:
 http://www.education.gg/CHttpHandler.ashx?id=97486&p=0 (Accessed October 2015)

²²² Joseph Rowntree Foundation (JRF). Reducing poverty in the UK: a collection of evidence reviews. York: JRF; 2014. Available from: <u>https://www.jrf.org.uk/report/reducing-poverty-uk-collection-evidence-reviews</u> (Accessed October 2015).

²²³ Bernstein B. Education cannot compensate for society. New Society 1970; 26: 344-347

²²⁴ Rasbash J., Leckie G., Pillinger R., Jenkins J. Children's educational progress: partitioning family, school and area effects. Journal of the Royal Statistical Society Series A: Statistics in Society 2010; 173(3): 657-682

²²⁵ Raffe D., Brannen K., Croxford L., Martin C. Comparing England, Scotland, Wales and Northern Ireland: the case for 'home internationals' in comparative research. Comparative Education 1999; 35(1): 9-25.

²²⁶ OECD. Reviews of national policies for education: quality and equity of schooling in Scotland. OECD 2007.

²²⁷ Schofield L., ISD Scotland. Personal communication, 2015.

²²⁸ World Health Organisation (WHO). Hardness in drinking water. Geneva: WHO; 2011

²²⁹ Pocock SJ, Shaper AG, Cook DG *et al*. British Regional Heart Study: geographic variations in cardiovascular mortality, and the role of water quality. BMJ 1980;280:1243–49

²³⁰ Scottish Water website: <u>http://www.scottishwater.co.uk/you-and-your-home/water-</u> <u>quality/waterqualitysearch</u> (Accessed June 2015).

²³¹ Environment Agency (England). Freedom of Information request. Received June 2015

²³² Walker M., Whincup P.H., Shaper A.G. The British Regional Heart Study 1975-2004. Int J Epidemiol. 2004; 33(6):1185-92

²³³ Mays N., Chinn S. Relation between all cause standardised mortality ratios and two indices of deprivation at regional and district level in England. Journal of Epidemiology & Community Health 1989; 43: 191–9.

²³⁴ Focas C. The four world cities transport study. London: The Stationery Office; 1998.

²³⁵ Church A., Frost, M., & Sullivan, K. Transport and social exclusion in London. Transport Policy 2000; 7: 195–205.

²³⁶ Currie G., & Senbergs, Z. Exploring forced car ownership in metropolitan Melbourne. 30th Australasian transport research forum. Melbourne: ATRF; 2007.

²³⁷ Johnson V., Currie G., Stanley J. Measures of Disadvantage: is Car Ownership a Good Indicator? Social Indicators Research 2010; 97:439–450.

²³⁸ Wannop U., Cherry G.E. The development of regional planning in the United Kingdom. Planning Perspectives 1994; 9(1): 29-60

²³⁹ Turok I. Urban Policy in Scotland: New Conventional Wisdom, Old Problems? *In*: Keating
 M. (Ed.) Scottish Social Democracy: Progressive Ideas for Public Policy. Brussels: PIE Peter
 Lang; 2007

²⁴⁰ Bell D., Blanchflower D. The Scots May Be Brave But They Are Neither Healthy Nor Happy.Scottish Journal of Political Economy 2007; 54(2): 151-307.

²⁴¹ Adams D., Public Health Team, Scottish Government. Personal communication (incorporating analysis of 2008 Scottish Health Survey), February 2015.

²⁴² National Obesity Observatory (NOO) (England). NOO fact sheet: Adult Obesity and
 Socioeconomic Status (incorporating analysis of Health Survey for England 2005-09), 2012.
 Available from:

http://www.noo.org.uk/uploads/doc/vid_16966_AdultSocioeconSep2012.pdf (Accessed December 2014)

²⁴³ Wrieden W., Chambers S., Barton K., Albani V., Anderson A. Diet and excess mortality in Glasgow and Scotland: Exploring differences in diet and nutrition. Glasgow: NHS Health Scotland, 2016

²⁴⁴ ScotPHO website (from data from General Household Survey, General Lifestyle Survey and Integrated Household Survey): <u>http://www.scotpho.org.uk/behaviour/tobacco-</u> <u>use/data/adult-smoking-gb-and-international-comparison</u> (Accessed June 2015)

²⁴⁵ McDowell I. From risk factors to explanation in public health. Journal of Public Health 2008; 30: 219-23.

²⁴⁶ McPherson K. Wider 'causal thinking in the health sciences'. Journal of Epidemiology & Community Health 1998; 52: 612-8.

²⁴⁷ Coggon D., Martyn C. Time and chance: the stochastic nature of disease causation. Lancet 2005; 365: 1434-7.

²⁴⁸ Rothman K. J. Causes. American Journal of Epidemiology 1976; 104: 587-592

²⁴⁹ Scotland Act 2016. See: <u>http://services.parliament.uk/bills/2015-16/scotland.html</u> (Accessed APril 2016)

²⁵⁰ NHS Health Scotland. Health inequalities briefing: income, wealth, poverty and health inequalities. Glasgow: NHS Health Scotland (forthcoming)

²⁵¹ Joseph Rowntree Foundation. Anti-poverty strategy, 2016. (Forthcoming)

 ²⁵² Ostry J.D., Berg A., Tsangarides C.G. Redistribution, Inequality, and Growth. International Monetary Fund Research Department, 2014. Available from: https://www.imf.org/external/pubs/ft/sdn/2014.

²⁵³ OECD. In it together: why less inequality benefits us all. Paris: OECD; 2015.

²⁵⁴ Scottish Government. Scotland's economic strategy. Edinburgh: Scottish Government,2015

²⁵⁵ Eisenstadt N. Shifting the curve: a report to the First Minister. Edinburgh: Scottish Government; 2015

²⁵⁶ Fair Work Convention – see: <u>http://www.fairworkconvention.scot/index.html</u> (Accessed April 2016)

²⁵⁷ Scottish Government. Working together for a fairer Scotland – Scottish Government
 Response to the Working Together Review. Edinburgh: Scottish Government; 2015

²⁵⁸ McCartney G, Taulbut M, Scott E, Macdonald W, Burnett D, Fraser A. Response to the Scottish Government's Expert Working Group on Welfare (EWGW) call for evidence. Glasgow: NHS Health Scotland; 2013.

²⁵⁹ Trebeck K., Stuart F. Our Economy: Towards a new prosperity. Oxford: Oxfam GB; 2013. Available from: <u>http://policy-practice.oxfam.org.uk/publications/our-economy-towards-a-new-prosperity-294239</u> (Accessed January 2016)

²⁶⁰ The Poverty Alliance. A Response to the Scottish Government Discussion Paper on Tackling Poverty Inequality and Deprivation in Scotland. 2008. Available from: <u>http://www.gov.scot/Resource/Doc/241165/0067088.pdf</u> (Accessed January 2016)

²⁶¹ World Health Organisation. Health in all policies training manual. Geneva: WHO; 2015. Available from: <u>http://who.int/social_determinants/publications/health-policies-manual/en/</u> (accessed November 2015).

²⁶² Scottish Housing Regulator. Scottish Housing Quality Standard. Progress update 2012/13.Glasgow: Scottish Housing Regulator; 2014

²⁶³ Place Standard: <u>http://www.placestandard.scot</u> (Accessed January 2016)

²⁶⁴ McConnell A. Scottish Local Government. Edinburgh: Edinburgh University Press; 2004

²⁶⁵ Fraser D. "The council map of Scotland is a glaring indictment of naked political gerrymandering". The Herald. 2006 December 1st.

²⁶⁶ The British Academy. "If you could do one thing..." - nine local actions to reduce health inequalities. London: British Academy; 2014.

²⁶⁷ UK Government. Glasgow and Clyde Valley City Deal. UK Government 2014. Available from:
https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/346278/G lasgow_Clyde_Valley_City_Deal.pdf (Accessed January 2016)

²⁶⁸ Robertson T., Estradé M., Jepson R., Muir G. The Nature of Employment and Excess Mortality in Glasgow and Scotland. Glasgow: NHS Health Scotland; 2016

²⁶⁹ Review of evidence on health aspects of air pollution – REVIHAAP project technical report. Copenhagen, WHO Europe, 2013.

²⁷⁰ Hoek G, Krishnan RM, Beelen R, *et al*. Long term air pollution exposure and cardiorespiratory mortality: a review. *Environmental Health* 2013; 12: 43.

²⁷¹ Brunekreef B, Holgate ST. Air pollution and health. *Lancet* 2002; 360: 1233-42.

²⁷² Effects of air pollution. London, Department for the Environment and Rural Affairs [accessed at <u>http://uk-air.defra.gov.uk/air-pollution/effects on 14th December 2015</u>].

²⁷³ Gerber A, Hofen-Hohloch AV, Schulze J, Groneberg DA. Tobacco smoke particles and indoor air quality (ToPIQ-II) – a modified study protocol and first results. J Occup Med Toxicol. 2015; 10: 5.

²⁷⁴ Rajagopalan, S and Brook, RD. The indoor-outdoor air-pollution continuum and the burden of cardiovascular disease: an opportunity for improving global health. Glob Heart.
2012; 7: 207–213.

²⁷⁵ Menon R, Porteous C. Healthy Low Energy Home Laundering. MEARU (Mackintosh Environmental Architecture Research Unit), Glasgow, The Glasgow School of Art, 2012.

²⁷⁶ Ambient (outdoor) air quality and health. Fact sheet number 313. Geneva, World Health Organization, 2014 [available at <u>http://www.who.int/mediacentre/factsheets/fs313/en/]</u>.

²⁷⁷ Lee D, Rushworth A, Sahu SK. A Bayesian localised conditional autoregressive model for estimating the health effects of air pollution. *Biometrics* 2014; 70: 419-429.

²⁷⁸ Searl A, Hurley F, Holland M, King K, Stedman J, Vincent K. Quantifying the Health Impacts of Pollutants Emitted in Central Scotland. Edinburgh, Institute of Occupational Medicine & AEA Technology, 2003.

²⁷⁹ Gowers AM, Miller BG, Stedman JR. Estimating local mortality burdens associated with particulate air pollution. Chilton, Public Health England, 2014.

²⁸⁰ Air pollution in the UK: 2013. London, DEFRA, 2014.

²⁸¹ Bower J, Willis P, Broughton G, Kent A, Lampert J. Air pollution in the UK: 2003. London, Natcen, 2004.

²⁸² Elliot P, Shaddick G, Wakefield JC, *et al*. Long-term associations of outdoor air pollution with mortality in Great Britain. *Thorax* 2007; 62: 1088-1094.

²⁸³ De Vos Irvine H, Lamont DW, Hole DJ, Gillis CR. Asbestos and lung cancer in Glasgow and

the west of Scotland. BMJ 2003; 306: 1503-6.

²⁸⁴ Durkheim E. The Division of Labour in Society. New York, NY, USA: The Free Press; 1984.

²⁸⁵ Durkheim E. Suicide. New York, NY, USA: The Free Press; 1951.

²⁸⁶ McKee M, Leon DA. Social transition and substance abuse. *Addiction* 2005;100(9):1205-1209.

²⁸⁷ Crawford F, Beck S, Hanlon P. Will Glasgow Flourish? Learning from the past, analysing the present and planning for the future. Glasgow: GCPH; 2007.

²⁸⁸ Buckingham A. Is there an underclass in Britain? *British Journal of Sociology* 1999;50:49-75.

²⁸⁹ Dean M. Christmas, the poor, and the development of a UK underclass. *Lancet* 1989;334:1536-1537.

²⁹⁰ Murray C. The Underclass Revisited. Washington, DC, USA: AEI Press; 1999.

²⁹¹ Kelso WA. Poverty and the Underclass: Changing Perceptions of the Poor in America. New York, NY, USA: New York University Press; 1994.

²⁹² Murray C. The emerging British underclass. London: Institute of Economic Affairs; 1990.

²⁹³ Srole L. Social integration and certain corollaries: an exploratory study. American Sociological Review, Vol 21, 1956, 709-716.

²⁹⁴ Dwight G, Dean DG. Alienation: Its Meaning and Measurement. *American Sociological Review* 1961;26(5):753-758.

²⁹⁵ Nettler G. A Measure of Alienation. *American Sociological Review* 1957;22:670-677.

²⁹⁶ Middleton R. Alienation, race, and education. *American Sociological Review* 1963;28:973-977.

²⁹⁷ Streuning EL, Richardson AH. A factor analytic exploration of the alienation, anomia and authoritarianism domain. *American Sociological Review* 1965;30:768-776.

²⁹⁸ Srole L. Social integration and certain corollaries: an exploratory study. American Sociological Review, Vol 21, 1956, 709-716.

²⁹⁹ Dwight G, Dean DG. Alienation: Its Meaning and Measurement. *American Sociological Review* 1961;26(5):753-758.

³⁰⁰ Nettler G. A Measure of Alienation. *American Sociological Review* 1957;22:670-677.

³⁰¹ Middleton R. Alienation, race, and education. *American Sociological Review* 1963;28:973-977.

³⁰² Streuning EL, Richardson AH. A factor analytic exploration of the alienation, anomia and

authoritarianism domain. American Sociological Review 1965;30:768-776.

³⁰³ Hills J, Brewer M, Jenkins S, *et al*. Anatomy of economic inequality in the UK: report of the National Equality Panel. London; 2010

³⁰⁴ MacDonald R, Marsh J. Employment, unemployment and social polarization. In:
 Crompton R, Devine F, Savage M, Scott J, eds. Renewing class analysis. Oxford: Blackwell;
 2000

³⁰⁵ Marshall G, Roberts S, Burgoyne C. Social class and underclass in Britain and the USA. The British Journal of Sociology 1996;47:22-44

³⁰⁶ Beeston C, McCartney G, Ford J, Wimbush E, Beck S, MacDonald W, and Fraser A. Health Inequalities Policy review for the Scottish Ministerial Task Force on Health Inequalities. NHS Health Scotland, Glasgow, 2013.

³⁰⁷ Macintyre S. The Black Report and beyond: what are the issues>? Soc Sci Med. 1997; 44(6):723-45.

³⁰⁸ Whitehead, M. 'The Health Divide" in Townsend, P., Whitehead, M., and Davidson, N. "Inequalities in Health: New Edition", London, Penguin, 1992.

³⁰⁹ Inequalities in Health: Report of a research working group, London, DHSS, 1980.

³¹⁰ Closing the Gap in a Generation: Health Equity through Action on the Social Determinants of Health. Geneva, WHO, 2008.

³¹¹ Power C, Matthews S. Origins of health inequalities in a national population sample. Lancet 1997; 350 (9091): 1584–9

³¹² Stringhini S, Dugravot A, Shipley M, Goldberg M, Zins M, KivimaM, *et al.* Health behaviours, socioeconomic status, and mortal-ity: further analyses of the British Whitehall II and the FrenchGAZEL prospective cohorts. PLoS Medicine 2011;8(2):e1000419,http://dx.doi.org/10.1371/journal.pmed.1000419.

³¹³ Shale J., Balchin K., Rahman J., Reeve R., Roli M. Households Below Average Income: an analysis of the income distribution 1994/95 – 2013/14. London: DWP; 2015. Available from: <u>https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/437246/h</u> <u>ouseholds-below-average-income-1994-95-to-2013-14.pdf</u> (Accessed January 2016)

³¹⁴ Richard Mitchell, Danny Dorling, Mary Shaw. Inequalities In Life and Death - What If Britain Were More Equal - The Technical Report. Bristol: The Policy Press; 2000. Available from: <u>http://www.dannydorling.org/wp-content/files/dannydorling_publication_id3005.pdf</u> (Accessed January 2016).

³¹⁵ Met Office website: <u>http://www.metoffice.gov.uk</u> (Accessed February 2015)

³¹⁶ Rosenthal N.E., Sack D.A., Gillin J.C., Lewy A.J., Goodwin F.K., Davenport Y., Mueller P.S., Newsome D.A., Wehr T.A. Seasonal affective disorder. A description of the syndrome and preliminary findings with light therapy. Arch Gen Psychiatry 1984; 41(1): 72-80.

³¹⁷ Ajdacic-Gross V., Lauber C., Sansossio R., Bopp M., Eich D., Gostynski M., Gutzwiller F., Rossler W.. Seasonal associations between weather conditions and suicide - Evidence against a classic hypothesis. Am. J. Epidemiol. 2007; 165(5): 561-569

³¹⁸ Hiltunen L., Ruuhela R., Ostamo A., Lönnqvist J., Suominen K., Partonen T. Atmospheric pressure and suicide attempts in Helsinki, Finland. Int J Biometeorol 2012; 56(6): 1045-53

³¹⁹ Chiu L.P.W. Do weather, day of the week, and address affect the rate of attempted suicide in Hong Kong? Soc. Psychiatry Psychiatr. Epidemiol. 1988; 23(4): 229-235

³²⁰ Geltzer A.J., Geltzer A.M., Dunford R.G., Hampson N.B. Effects of weather on incidence of attempted suicide by carbon monoxide poisoning. Undersea Hyperb Med. 2000; 27(1): 9-14

³²¹ Huibers M.J.H., de Graaf L.E., Peeters F.P.M.L., Arntz A.. Does the weather make us sad? Meteorological determinants of mood and depression in the general population. Psychiatry Res. 2010; 180(2-3): 143-146

³²² de Craen A.J.M., Gussekloo J., van der Mast R.C., le Cessie S., Lemkes J.W., Westendorp R.G.J. Seasonal mood variation in the elderly: The Leiden 85-plus study. Int. J. Geriatr.
 Psychiatry. 2005; 20(3): 269-273

³²³ Molin J., Mellerup E., Bolwig T., Scheike T., Dam H. The influence of climate on development of winter depression. J. Affect. Disord. 1996; 37(2-3): 151-155.

³²⁴ Denissen J.J.A., Butalid L., Penke L., van Aken M.A.G. The Effects of Weather on Daily Mood: A Multilevel Approach. Emotion 2008; 8(5): 662-667

³²⁵ Salib E., Sharp N. Relative humidity and affective disorders. Int. J. Psychiatry Clin. Pract.
2002; 6(3): 147-153

³²⁶ McWilliams S., Kinsella A., O'Callaghan E. The effects of daily weather variables on psychosis admissions to psychiatric hospitals. International Journal of Biometeorology2013; 57(4): 497-508

³²⁷ Bulbena A., Pailhez G., Acena R., Cunillera J., Rius A., Garcia-Ribera C., Gutierrez J., Rojo C.. Panic anxiety, under the weather?. Int. J. Biometeorol 2005 49(4):238-243

³²⁸ Watson, D. Mood and temperament. New York: The Guilford Press; 2000

³²⁹ Enquselassie F., Dobson A.J., Alexander T.M., Steele P.L.. Seasons, temperature and coronary disease. Int. J. Epidemiol. 1993; 22(4): 632-636

³³⁰ Feinglass J., Lee J., Semanik P., Song J., Dunlop D., Chang R. The effects of daily weather on accelerometer-measured physical activity. Journal of Physical Activity and Health 2011; 8(7): 934-943

³³¹ Gillie O. Scotland's health deficit: an explanation and a plan. Health Research Forum

occasional reports: No 3. Health Research Forum; 2008.

³³² Met Office: <u>http://www.metoffice.gov.uk</u> (Accessed February 2015).

³³³ Hypponen E., Power C. Hypovitaminosis D in British adults at age 45 years: nationwide cohort study of dietary and lifestyle predictors. American Journal of Clinical Nutrition 2007; 85(3): 860–868.

³³⁴ Autier P., Boniol M., Pizot C., Mullie P. Vitamin D status and ill health: A systematic review. The Lancet Diabetes and Endocrinology 2014; 2(1): 76-89.

³³⁵ Paul Welsh, University of Glasgow. Personal communication, 2015.

³³⁶ Afzal S, Brøndum-Jacobsen P, Bojesen SE, Nordestgaard BG. Genetically low vitamin D concentrations and increased mortality: mendelian randomisation analysis in three large cohorts. BMJ 2014;349:g6330.

³³⁷ National Records of Scotland. Winter mortality 2011/12. <u>http://www.gro-scotland.gov.uk/statistics/theme/vital-events/deaths/winter-mortality</u> (Accessed September 2013).

³³⁸ ONS. Excess Winter Mortality in England and Wales, 2011/12 (Provisional) and 2010/11 (Final). ONS 2013. Available from: <u>http://www.ons.gov.uk/ons/rel/subnational-</u> <u>health2/excess-winter-mortality-in-england-and-wales/2012-13--provisional--and-2011-12--</u> <u>final-/index.html</u> (Accessed January 2014).

³³⁹ Breakthrough Britain: Economic Dependency and Worklessness. London, Centre for Social Justice, 2007.

³⁴⁰ Breakthrough Glasgow: ending the costs of social breakdown. London, Centre for Social Justice, 2008.

³⁴¹ The Centre for Social Justice. 21st Century Welfare: Response of the Centre for Social Justice. London: Centre for Social Justice; 2010. Available from: http://www.centreforsocialjustice.org.uk/UserStorage/pdf/Pdf%20reports/21stCenturyWelf are.pdf (Accessed January 2016)

³⁴² Harkness S., Gregg P., MacMillan L. Poverty: the role of Institutions, behaviours and culture. Bath: University of Bath/Joseph Rowntree Foundation; 2012. Available from: <u>https://www.jrf.org.uk/sites/default/files/jrf/migrated/files/poverty-culture-behaviour-full.pdf</u> (Accessed January 2016)

³⁴³ Macmillan L. Measuring the intergenerational correlation of worklessness. Bristol: University of Bristol; 2011

³⁴⁴ Brown J., Smith J., Webster D., Arnott J., Turok I., Macdonald E., Mitchell R. Changes in incapacity benefit receipt in UK Cities, 2000 – 2008. Glasgow: Scottish Observatory for Work and Health/University of Glasgow; 2010 ³⁴⁵ Taylor-Gooby P, Stoker G. The Coalition Programme: A New Vision for Britain or Politics as Usual? The Political Quarterly 2011; 82(1): 4–15,

³⁴⁶ McCartney G, Myers F, Taulbut M, MacDonald W, Robinson M, Scott S, Mitchell R, Millard D, Tod E, Curnock E, Katikireddi SV, Craig N. Making a bad situation worse? The impact of welfare reform and the economic recession on health and health inequalities in Scotland (baseline report). Edinburgh: NHS Health Scotland; 2013.

³⁴⁷ Scott-Samuel A, Bambra C, Collins C, Hunter DJ, McCartney G, Smith K. The impact of Thatcherism on health and wellbeing in Britain. International Journal of Health Services 2014; 44(1): 53–71.

³⁴⁸ Hutchinson SJ, Roy KM, Wadd S, *et al*. Hepatitis C virus infection in Scotland:
epidemiological review and public health challenges. *Scottish Medical Journal* 2006; 51(2): 8-15.

³⁴⁹ Harris H, Costella A, Goldberg D, *et al*. Hepatitis C in the UK: 2014 report. London, Public Health England, 2014.

³⁵⁰ Merrall ELC, Bird SM, Hutchinson SJ. Mortality of those who attended drugs services in Scotland 1996-2006: record linkage study. *International J Drug Policy* 2012; 23: 24-32.

³⁵¹ Beeston C, Reid G, Robinson M, Craig N, McCartney G, Graham L and Grant I (on behalf of the MESAS project team). Monitoring and Evaluating Scotland's Alcohol Strategy. Third Annual Report. Edinburgh: NHS Health Scotland; 2013.

³⁵² Gordon R, Heim D, MacAskill S. Rethinking drinking cultures: A review of drinking cultures and a reconstructed dimensional approach. *Public* Health 2012; 26; 3-11.

³⁵³ Gordon R, Heim D, MacAskill S, *et al.* Snapshots of drinking: a rapid review of drinking cultures and influencing factors: Australia, Canada, France, Germany, Spain, Sweden and the United Kingdom and Scotland. Edinburgh, NHS Health Scotland, 2008.

³⁵⁴ Whiteford M, Byrne P. Back to the future: understanding and responding to alcohol use in Liverpool. *People, Place & Policy Online* 2012: 6/3: 108-121.

³⁵⁵ McCartney G, Boutell J, Craig N, *et al*. Explaining trends in alcohol-related harms in Scotland 1991-2011 (II): policy, social norms, the alcohol market, clinical changes and a synthesis. Public Health 2016 (in press).

³⁵⁶ Felitti, V.J. Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults. American Journal of Preventative Medicine 1998; 14(4): 245-258.

³⁵⁷ Brown D.W., Anda R.F., Tiemeier H., Felitti V.J., Edwards V.J., Croft J.B., Giles W.H. Adverse childhood experiences and the risk of premature mortality. American Journal of Preventative Medicine 2009;37(5):389-396.

³⁵⁸ Craig C. Women and children last. Scottish Review of Books 2010; 6(1): 4-6.

³⁵⁹ Smith, M., Williamson, AE., Walsh, D., McCartney, G. Is there a link between childhood adversity, attachment style and Scotland's excess mortality? Evidence, challenges and potential research. Submitted to BMC Public Health, November 2015

³⁶⁰ Paterson L, Iannelli C. Social class and educational attainment: a comparative study of England, Wales and Scotland. Edinburgh, University of Edinburgh and ESRC, 2006 [accessed at . <u>http://www.ces.ed.ac.uk/research/Recent%20projects/SocMobility/papers/WP2.pdf on 18th November 2015</u>].

³⁶¹ Machin S, McNally S, Wyness G. Education in a Devolved Scotland: A Quantitative Analysis. ESRC, 2013. Available from:

http://cep.lse.ac.uk/pubs/download/special/cepsp30.pdf on 1th November 2015 (Accessed November 2015)

³⁶² Croxford L. Inequality in Attainment at Age 16: A 'Home International' Comparison. Edinburgh, University of Edinburgh, 2000 [accessed at http://www.ces.ed.ac.uk/PDF%20Files/Brief019.pdf on 18th November 2015].

³⁶³ UK Data Service. Personal communication. August 2015

³⁶⁴ Bartley M, Ferrie J, Montgomery SM. Living in a high-unemployment economy: understanding the health consequences. In: Marmot M, Wilkinson RG. (eds.). Social Determinants of Health. Oxford: Oxford University Press; 1999

³⁶⁵ Roelfs DJ, Shor E, Davidson KW, Schwartz JE. Losing life and livelihood: A systematic review and meta-analysis of unemployment and all-cause mortality. Social Science & Medicine2011; 72(6): 840–854..

³⁶⁶ Bambra C, Eikemo TA. Welfare state regimes, unemployment and health: a comparative study of the relationship between unemployment and self-reported health in 23 European countries. Journal of Epidemiology and Community Health 2009; 63: 92-98.

³⁶⁷ Brown J, Demou E, Tristram MA, Gilmour H, Sanati KA, Macdonald EB. Employment status and health: understanding the health of the economically inactive population in Scotland. BMC Public Health 2012; 12: 327

³⁶⁸ Siegrist J, Benach J, McKnight A, *et al*. Employment arrangements, work conditions and health inequalities: Report on new evidence on health inequality reduction, produced by task group 2 for the strategic review of health inequalities post 2010. London, UK: Marmot Review; 2010.

³⁶⁹ Gordon D, Graham L, Robinson M, Taulbut M. Dimensions of diversity: Population differences and health improvement opportunities. Edinburgh, NHS Health Scotland and ScotPHO, 2010.

³⁷⁰ Bhopal RS. Migration, Ethnicity, Race, and Health in Multicultural Societies. Oxford, OUP, 2013.

³⁷¹ Fischbacher CM, Steiner M, Bhopal R, Chalmers J, Jamieson J, Knowles D, *et al*. Variations in all cause and cardiovascular mortality by country of birth in Scotland, 1997-2003.erratum appears in Scott Med J. 2008 May;53(2):66]. Scott Med J 2007 11;52(4):5-10.

³⁷² Overview of Equality results from the 2011 Census: release 2. Edinburgh, Scottish Government, 2014.

³⁷³ Hawkes N. Solving the mystery of health inequality. *BMJ* 2015; 350: h3389.

³⁷⁴ McGuinness D, McGlyne LM, Jonson PCD, *et al*. Socio-economic status is associated with epigenetic differences in the pSoBid cohort. *International Journal Epidemiology* 2012: 1-10, doi: 10.1093/ije/dyr215.

³⁷⁵ Burns H. 2012. Annual Report of the Chief Medical Officer. Health in Scotland 2011. Transforming Scotland's Health. Edinburgh, Scottish Government.

³⁷⁶ McLaughlin M. Babies born into poverty are damaged forever before birth. Edinburgh, Scotsman, 24th January 2014.

³⁷⁷ Chen L, Davey Smith G, Harbord R, Lewis S. Alcohol intake and blood pressure: a systematic review implementing a Mendelian randomization approach. *PLoS Med* 2008; 5: e52.

³⁷⁸ McGowan PO, Szyf M. 2010. The epigenetics of social adversity in early life: Implications for mental health outcomes. Neurobiology of Disease; 39: 66–72.

³⁷⁹ Kaati G, Bygren LO, Edvinsson S. 2002. Cardiovascular and diabetes mortality determined by nutrition during parents' and grandparents' slow growth period. European Journal of Human Genetics; 10: 682 – 688.

³⁸⁰ Leslie S, Winney B, Hellenthal G, *et al*. The fine-scale genetic structure of the British population. Nature 2015, 519; 309-314.

³⁸¹ O'Dushlaine CT, Morris D, Moskvina V. European Journal of Human Genetics 2010; 18: 1248–1254.

³⁸² Coggon DIW, Martyn CN. Time and chance: the stochastic nature of disease causation. Lancet 2005; 365: 1434–37.

³⁸³ Popham F, Boyle P, O'Reilly D, Leyland A. Selective internal migration. Does it explain Glasgow's worsening mortality record? Edinburgh, GRO Scotland, 2009.

³⁸⁴ Exeter D, Feng Z, Flowerdew R, Boyle P. Shrinking areas and mortality: an artefact of deprivation effects in the West of Scotland? *Health & Place* 2005; 59: 924-6.

³⁸⁵ Richmond RC, Simpkin AJ, Woodward G, *et al*. Prenatal exposure to maternal smoking and offspring DNA methylation across the life-course: findings from the Avon Longitudinal Study of Parents and Children (ALSPAC). Human Molecular Genetics 2015; 24(8): 2201–2217. ³⁸⁶ Davey Smith G. Epigenetics for the masses: more than Audrey Hepburn and yellow mice? International Journal of Epidemiology 2012;41:303–308.

³⁸⁷ Heard E, Martienssen RA. Transgenerational Epigenetic Inheritance: Myths and Mechanisms. Cell 2014; 157: 95-109.

³⁸⁸ Brandt-Rauf SI, Raveis VH, Drummond NF, Conte JA, Rothman SM. Ashkenazi Jews and Breast Cancer: The Consequences of Linking Ethnic Identity to Genetic Disease. Am J Public Health 2006; 96(11): 1979–1988.

³⁸⁹ Healthcare across the UK: a comparison of the NHS in England, Scotland, Wales and Northern Ireland. London, National Audit Office, 2012.

³⁹⁰ Sutherland K, Coyle N. Quality in healthcare in England, Wales, Scotland, Northern Ireland: an intra-UK chartbook. London, Health Foundation, 2009.

³⁹¹ McLaughlin R. A comparison of the health expenditure of the cities of Glasgow, Dundee, Edinburgh, Liverpool and Manchester. Glasgow, Glasgow Centre for Population Health [unpublished analysis].

³⁹² McLaren J, Armstrong J, Harris R. Spending on health. Glasgow, Centre for Public Policy for Regions in association with KPMG, 2010.

³⁹³ Bevan G, Karanikolos M, Exley J, *et al*. The four health systems of the United Kingdom: how do they compare? London, Health Foundation and Nuffield Trust, 2014.

³⁹⁴ Pell JP, Haw S, Cobbe S, *et al*. Smoke-free Legislation and Hospitalizations for Acute Coronary Syndrome. New England Journal of Medicine 2008; 359(5):482-491.

³⁹⁵ Robinson M, Geue C, Lewsey J, *et al*. Evaluating the impact of the alcohol act on off-trade alcohol sales: a natural experiment in Scotland. Addiction 2014; 109(12): 2035–2043.

³⁹⁶ Lawder R, Harding O, Stockton D, Fischbacher C, Brewster DH, Chalmers J, Finlayson A, Conway DI. Is the Scottish population living dangerously? Prevalence of multiple risk factors: the Scottish Health Survey 2003. BMC Public Health 2010, 10: 330.

³⁹⁷ Grant I, Springbett A, Graham L. Alcohol attributable mortality and morbidity: alcohol population attributable fractions for Scotland. Edinburgh, NHS National Services Scotland, 2009.

³⁹⁸ Gill J, Tsang C, Black H, Chick J. Can Part of the Health Damage Linked to Alcohol Misuse in Scotland be Attributable to the Type of Drink and its Low Price (by Permitting a Rapid Rate of Consumption)? A Point of View. *Alcohol & Alcoholism* 2010; 45(4): 398–400.

³⁹⁹ McCartney G, Bouttell J, Craig N, *et al*. Explaining trends in alcohol-related harms in Scotland, 1991-2011 (I): the role of incomes, effects of socio-economic and political adversity and demographic change. Public Health 2016 (in press).

⁴⁰⁰ Mackenzie M, Collins C, Connolly J, Doyle M, McCartney G. "I don't smoke and I don't

drink. The only thing that's wrong with me is my health": Working class discourses about health, its macro-level determinants and intervening mechanisms. Policy and Politics 2016 (in press).

⁴⁰¹ Shipton D. Alcohol-related harm in Glasgow: a national, city and neighbourhood perspective. Glasgow: Glasgow Centre for Population Health; 2014.

⁴⁰² Giles L. Developing a model to project alcohol-related mortality in Scotland. Glasgow: Glasgow University; 2013.

⁴⁰³ Lim SS, Vos T, Flaxman AD, *et al*. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 2012; 380(9859): 2224–2260.

⁴⁰⁴ Wang X, Ouyang Y, Liu J, Zhu M, Zhao G, Bao W, Hu FB. Fruit and vegetable consumption and mortality from all causes, cardiovascular disease, and cancer: systematic review and dose-response meta-analysis of prospective cohort studies. BMJ 2014; 349: g4490.

⁴⁰⁵ National Records of Scotland (NRS). Drug-related deaths in Scotland in 2014. Edinburgh: NRS; 2015. Available from: <u>http://www.gro-scotland.gov.uk/files//statistics/drug-related-deaths/drd14/drugs-related-deaths-2014.pdf</u> (Accessed January 2016)

⁴⁰⁶ Nutt DJ, King LA, Phillips LD, *et al*. Drug harms in the UK: a multicriteria decision analysis. Lancet 2010; 376(9752): 1558–1565.

⁴⁰⁷ Corkery J. University of Hertfordshire, and previously of National Programme on Substance Abuse Deaths National Programme on Substance Abuse Deaths, St George's Hospital Medical School, London. Personal communications; 2009 and 2015.

⁴⁰⁸ Bloor M., Gannon M., Hay G., Jackson G., Leyland A.H., McKeganey N. Contribution of problem drug users' deaths to excess mortality in Scotland: secondary analysis of cohort study. British Medical Journal 2008; 337: a478.

⁴⁰⁹ BMJ rapid responses to Bloor *et al*.

http://www.bmj.com/content/337/bmj.a478.full/reply#bmj_el_200773 (Accessed August 2011).

⁴¹⁰ Dodds B., Grant I., Bainbridge R., Wood L. Robb S. Scottish Schools Adolescent Lifestyle and Substance Use Survey (SALSUS): drug use among 13 and 15 year olds in Scotland 2013. Edinburgh: NHS National Services Scotland; 2014. Available from:

http://www.isdscotland.scot.nhs.uk/Health-Topics/Public-Health/Publications/2014-11-25/SALSUS_2013_Drugs_Report.pdf (Accessed January 2016).

⁴¹¹ Samitz G, Egger M, Zwahlen M: Domains of physical activity and all-cause mortality:
systematic review and dose response meta-analysis of cohort studies. Int J Epidemiol 2011,
40: 1382-1400.

⁴¹² Nocon M, Hiemann T, Müller-Riemenschneider F, Thalau F, Roll S, Willich SN. Association of physical activity with all-cause and cardiovascular mortality: a systematic review and meta-analysis. Eur J Cardiovasc Prev Rehabil. 2008 Jun; 15(3): 239-46.

⁴¹³ Clays E, Lidegaard M, De Bacquer D, Van Herck K, De Backer G, Kittel F, de Smet P, Holtermann A. The combined relationship of occupational and leisure-Time physical activity with all-cause mortality among men, accounting for physical fitness. Am J Epidemiol. 2014; 179(5): 559–566.

⁴¹⁴ Sallis J, Floyd M, Rodríguez D, Saelens B. Role of built environments in physical activity, obesity, and cardiovascular disease. Circulation 2012;125(5):729-737.

⁴¹⁵ Handy S. Does the Built Environment Influence Physical Activity? Examining the Evidence. Critical Assessment of the Literature of the Relationships among Transportation, Land Use and Physical Activity. Transportation Research Board Special Report 282; 2005.

⁴¹⁶ Brownson RC, Baker EA, Housemann RA, Brennan LK, Bacak SJ. Environmental and Policy Determinants of Physical Activity in the United States. Am J Public Health 2001; 91(12): 1995–2003.

⁴¹⁷ Bromley C, Shelton N. The Scottish Health Survey: Topic Report UK Comparisons. Edinburgh, Scottish Government, 2010.

⁴¹⁸ Gray L. Comparisons of health-related behaviours and health measures in Greater Glasgow with other regional areas in Europe. Glasgow: Glasgow Centre for Population Health; 2008

⁴¹⁹ Peto R, Darby S, Deo H, Silcocks P, Whitley E, Doll R. Smoking, smoking cessation, and lung cancer in the UK since 1950. British Medical Journal 2000;321:323-9.

⁴²⁰ World Health Organisation. Fact sheet no. 339: Tobacco. Available from: <u>http://www.who.int/mediacentre/factsheets/fs339/en/</u> (Accessed January 2015)

⁴²¹ Kjellstrom T, Mercado S, Sattherthwaite D, McGranahan G, Friel S, Havemann K. Our cities, our health, our future: acting on social determinants for health equity in urban settings. Japan: World Health Organization; 2007

⁴²² US Task Force on Community Preventive Services. Recommendations to promote healthy social environments. Am J Prev Med. 2003;24(suppl 3):21–24

⁴²³ Tunstall R., Wright K. Housing provision and excess mortality in Glasgow and Scotland.Glasgow: NHS Health Scotland; 2016

⁴²⁴ Celebrating Scotland's disproportionate WW1 deaths. The Scottish Military Research Group, 2014 [accessed at <u>http://scottishmilitary.blogspot.co.uk/2014/07/celebrating-</u> <u>scotlands-disproportionate.html on 30th October 2015</u>].

⁴²⁵ General Register Office for Scotland. Scotland's Population 2005: The Registrar General's Annual Review of Demographic Trends, 151st Edition. Edinburgh: General Register Office for Scotland; 2005. Available from: <u>http://www.nrscotland.gov.uk/files/statistics/old/ar05.pdf</u> (Accessed December 2015)

⁴²⁶ Davey Smith G., Dorling, D., Mitchell, R., and Shaw M. Health inequalities in Britain: continuing increases up to the end of the 20th century. Journal of Epidemiology & Community Health 2002; 56: 434-435.

⁴²⁷ Dorling D., Thomas B. People and Places – a 2001 census atlas of the UK. Bristol: The Policy Press; 2004.

⁴²⁸ Dorling D. Death in Britain. How local mortality rates have changed: 1950s - 1990s. York: Joseph Rowntree Foundation; 1997.

⁴²⁹ Mitchell R., Dorling D., Shaw M. Inequalities in life and death – what if Britain were more equal? Bristol: The Policy Press; 2000.

⁴³⁰ Macintyre S. Inequalities in health in Scotland: what are they and what can we do about them? Glasgow: MRC Social & Public Health Sciences Unit. Occasional Paper No. 17; 2007.

⁴³¹ Goldthorpe J. Analysing Social Inequality: a Critique of Two Recent Contributions from Economics and Epidemiology. European Sociological Review 2009; 26(6): 731–44.

⁴³² Saunders P. Beware False Prophets: Equality, the Good Society and The Spirit Level.London: Policy Exchange; 2010.

⁴³³ Lynch J, Davey Smith G, Harper S, Hillemeier M, Ross N, Kaplan GA, Wolfson M. Is Income Inequality a Determinant of Population Health? Part 1. A Systematic Review. Milbank Quarterly 2004; 82(1): 5–99.

⁴³⁴ Lynch J, Davey Smith G, Harper S, Hillemeier M. Is Income Inequality a Determinant of Population Health? Part 2. U.S. National and Regional Trends in Income Inequality and Ageand Cause-Specific Mortality. Milbank Quarterly 2004; 82(2): 355–400.

⁴³⁵ Wilkinson R., Pickett K. The Spirit Level - Why More Equal Societies Almost Always Do Better. London: Allen Lane; 2009.

⁴³⁶ Preston S. The Changing Relation between Mortality and Level of Economic Development.Population Studies 1975; 29: 231–48.

⁴³⁷ Rodgers G.B. Income and inequality as determinants of mortality: an international cross section analysis. International Journal of Epidemiology 1979; 31: 182–91.

⁴³⁸ Wilkinson R. Income distribution and life expectancy. British Medical Journal 1992; 304: 165–8.

⁴³⁹ Wilkinson R. Unhealthy societies: the afflictions of inequality. London: Routledge; 1996.

⁴⁴⁰ Wilkinson R. The impact of inequality. London: Routledge; 2005.

⁴⁴¹ Luxembourg Income Study (LIS) Database: http://www.lisproject.org (Accessed March

2014).

⁴⁴² Mooney, G., Morelli, C. Seaman, P. The question of economic growth and inequality in contemporary Scotland. Scottish Affairs 2009; 67 (Spring): 92–109

⁴⁴³ Mooney G., Morelli C.J., Seaman P.T. 'Tackling Inequality and Disadvantage in the Devolved Scotland'. In: Leith M., McPhee I., Laxton T. (Eds). Scottish Devolution and Social Policy: Evidence from the First Decade. Newcastle upon Tyne: Cambridge Scholars Publishing; 2012

⁴⁴⁴ Schwarzer R, Jerusalem M. Generalized Self-Efficacy scale. In: Weinman J, Wright S, Johnston M, Measures in health psychology: A user's portfolio. Causal and control beliefs (pp. 35-37). Windsor, UK: NFER-NELSON; 1995

⁴⁴⁵ AHP Research/NHS Health Scotland. Review of scales of positive mental health validated for use with adults in the UK: Technical report. Edinburgh: NHS Health Scotland; 2007.
 Available from: <u>http://www.healthscotland.com/documents/2655.aspx</u> (Accessed January 2015)

⁴⁴⁶ Cairns J, van der Pol M. The estimation of marginal time preference in a UK-wide sample (TEMPUS) project. *Health Technology Assessment* 2000;4(1).

⁴⁴⁷ Cairns J, van der Pol M. Repeated follow-up as a method for reducing non-trading behaviour in discrete choice experiments. *Social Science and Medicine* 2004;58:2211-2218.

⁴⁴⁸ van der Pol M, Cairns J. Negative and zero time preference for health. *Health Economics* 2000;9(2):171-175.

⁴⁴⁹ Robinson A, Dolan P, Williams A. Valuing health status using VAS and TTO: what lies behind the numbers? *Social Science & Medicine* 1997;45(8):1289-1297.

⁴⁵⁰ Pressman SD, Cohen S. Does positive affect influence health? *Psychological Bulletin* 2005;131(6):925-971.

⁴⁵¹ Diener E, Chan MY. Happy people live longer: Subjective well-being contributes to health and longevity. *Applied Psychology: Health and Well-Being* 2011;3(1):1-43.

⁴⁵² Lyons A, Chamberlain K. The effects of minor events, optimism and self-esteem on health. *British Journal of Health Psychology* 1994;33(Pt 4):559-570.

⁴⁵³ Fournier MA, de Ridder D, Bensing J. How optimism contributes to the adaptation of chronic illness. A prospective study into the enduring effects of optimism on adaptation moderated by the controllability of chronic illness. *Personality and Individual Differences* 2002;33(7):1163-1183.

⁴⁵⁴ de Ridder D, Fournier MA, Bensing J. Does optimism affect symptom report in chronic disease? What are its consequences for self-care behaviour and physical functioning? *Journal of Psychosomatic Research* 2004;56(3):341-350.

⁴⁵⁵ Mahler HIM, Kulik JA. Optimism, pessimism and recovery from coronary bypass surgery:
 Prediction of affect, pain and functional status. *Psychology, Health & Medicine* 2000;5(4):347-358.

⁴⁵⁶ Boehm JK, Kubzansky LD. The heart's content: The association between positive psychological well-being and cardiovascular health. *Psychological Bulletin* 2012;138(4):655-691.

⁴⁵⁷ Rasmussen HN, Scheier MF, Greenhouse JB. Optimism and physical health: a metaanalytic review. *Annals of Behavioral Medicine* 2009;37(3):239-256.

⁴⁵⁸ Conner P, Norman P (eds.) Predicting health behaviour (2nd edition). Buckingham,England: Open University Press; 2005.

⁴⁵⁹ Marks R, Allegrante JP, Lorig K. A Review and Synthesis of Research Evidence for Self-Efficacy-Enhancing Interventions for Reducing Chronic Disability: Implications for Health Education Practice (Part II). *Health Promotion Practice* 2005;6(2):148-156.

⁴⁶⁰ Jones F, Riazi A. Self-efficacy and self-management after stroke: a systematic review. *Disability and Rehabilitation* 2011;33(10):797-810.

⁴⁶¹ Korpershoek C, van der Bijl J, Hafsteinsdottir TB. Self-efficacy and its influence on recovery of patients with stroke: a systematic review. *Journal of Advanced Nursing* 2011;67(9):1876-1894.

⁴⁶² Eckersley R. Is modern Western culture a health hazard? *International Journal of Epidemiology* 2006;35(2):252-258.

⁴⁶³ Kasser T. The High Price of Materialism. Cambridge, MA, USA: MIT Press; 2002.

⁴⁶⁴ Eckersley R. Well and Good: Morality, Meaning and Happiness (2nd edition). Melbourne, Australia: Text Publishing; 2005.

⁴⁶⁵ Scheier MF, Carver CS, Bridges MW. Distinguishing optimism from neuroticism (and trait anxiety, self-mastery, and self-esteem): A re-evaluation of the Life Orientation Test. *Journal of Personality and Social Psychology* 1994;67:1063-1078.

⁴⁶⁶ Schwartz SH. Basic Human Values. Cross-National Comparison Seminar on the Quality and Comparability of Measures for Constructs in Comparative Research: Methods and Applications, Bolzano (Bozen), Italy, June 10-13, 2009. Available from: <u>http://www.ccsr.ac.uk/qmss/seminars/2009-06-10/documents/Shalom_Schwartz_1.pdf</u> (accessed April 2013)

⁴⁶⁷ Schwartz SH. Les valeurs de base de la personne: Théorie, mesures et applications [Basic human values: Theory, measurement, and applications]. *Revue Française de Sociologie* 2006;42:249-288.

⁴⁶⁸ Bilsky W. Janik M, Schwartz SH. The structural organization of human values – evidence from three rounds of the European Social Survey (ESS). *Journal of Cross-Cultural Psychology*

2011;42:759.

⁴⁶⁹ Schwartz SH. Universals in the content and structure of values: Theory and empirical tests in 20 countries. In Zanna M (ed.) Advances in experimental social psychology (volume 25). New York, NY, USA: Academic Press; 1992. pp1-65.

⁴⁷⁰ Schwartz SH. A Proposal for Measuring Value Orientations across Nations. Chapter 7 in the Questionnaire Development Package of the European Social Survey; 2003. Available at: <u>www.Europeansocialsurvey.org</u>

⁴⁷¹ Schwartz SH. Basic human values: Their content and structure across countries. In Tamayo A, Porto JB (eds.) Valores e comportamento nas organizações [Values and behaviour in organisations] Petrópolis, Brazil: Vozes; 2005. pp21-55.

⁴⁷² Holtermann S. Areas of urban deprivation in Britain: an analysis of 1971 census data.
 Social Trends 1975; No. 6

⁴⁷³ Champion, T., Fotheringham, S., Rees, P., Boyle, P., Stillwell, J. The Determinants of Migration Flows in England: a Review of Existing Data and Evidence. Newcastle: Department of the Environment, Transport and the Regions; 1998. Available from: <u>http://www.geog.leeds.ac.uk/publications/DeterminantsOfMigration/report.pdf</u> (Accessed May 2014).

⁴⁷⁴ Fielding T. Migration and poverty: A longitudinal study of the relationship between migration and social mobility in England and Wales. Institute of Development Studies Bulletin 1997; 28: 48–57.

⁴⁷⁵ Leon D., Strachan P. Socioeconomic characteristics of interregional migrants in England and Wales, 1939–71. Environment and Planning A 1993; 25(10): 1441–1451.

⁴⁷⁶ Meen G., Gibb K., Goody J., McGrath T., MacKinnon J. Migration and location. *In*:
 Economic segregation in England: Causes, consequences and policy. England: The Policy
 Press; 2005.

⁴⁷⁷ O'Reilly D., Stevenson M. Selective migration from deprived areas in Northern Ireland and the spatial distribution of inequalities: Implications for monitoring health and inequalities in health. Social Science & Medicine 2003; 57(8): 1455–1462.

⁴⁷⁸ Richey P. Explanations of migration. Annual Review of Sociology 1976; 2: 383.

⁴⁷⁹ Davey Smith G., Shaw M., Dorling D. Shrinking areas and mortality. Lancet 1998; 352: 1439–1440.

⁴⁸⁰ Boyle P., Gatrell A., Duke-Williams, O. Do area-level population change, deprivation and variations in deprivation affect individual level self-reported limiting long-term illness? Social Science & Medicine 2001; 53: 795–799.

⁴⁸¹ Regidor E., Calle M.E., Dominguez V., Navarro P. Inequalities in mortality in shrinking and growing areas. Journal of Epidemiology & Community Health 2002; 56: 919–921.

⁴⁸² Brown D., Leyland A.H. Population mobility, deprivation and self-reported limiting longterm illness in small areas across Scotland. Health & Place 2009; 15(1): 37–44.

⁴⁸³ Brimblecombe N., Dorling D., Shaw M. Migration and geographical inequalities in health in Britain. Social Science & Medicine 2000; 50: 861-878.

⁴⁸⁴ O'Reilly D., Browne S., Johnson Z., Kelly A. Are cities becoming more unhealthy? An analysis of mortality rates in Belfast and Dublin between 1981 and 1991 to illustrate a methodological difficulty with ecological studies. Journal of Epidemiology & Community Health 2001; 55: 354-355.

⁴⁸⁵ Krieger N., Rehkopf D.H., Chen J.T. *et al*. al. The fall and rise of US inequities in premature mortality: 1960–2002. PLoS Med 2008; 5(2): e46.

⁴⁸⁶ Brimblecombe N., Dorling D. Shaw, M. Mortality and migration in Britain, first results from the British Household Panel Survey. Social Science & Medicine 1999; 49: 981-988.

⁴⁸⁷ Boyle P. Population geography: Migration and inequalities in mortality and morbidity.Progress in Human Geography 2004; 28(6): 767–776.

⁴⁸⁸ Connolly S., O'Reilly D., Rosato M. Has the increasing inequality in health in England and Wales been caused by the selective movement of people between areas? A 12-year mortality study using the Longitudinal Study. Social Science & Medicine 2007; 64: 2008-2015.

⁴⁸⁹ Norman P., Boyle P., Rees P. Selective migration, health and deprivation: A longitudinal analysis. Social Science & Medicine 2005; 60(12): 2755–2771

⁴⁹⁰ Boyle, P., Exeter, D., & Flowerdew, R. The role of population change in widening the mortality gap in Scotland. Area 2004; 36(2): 164–173.

⁴⁹¹ Popham F., Boyle P., O'Reilly D., H Leyland A.H. Selective internal migration. Does it explain Glasgow's worsening mortality record? Scottish Longitudinal Study (SLS) Research Working Paper Series, Research working paper 5. Longitudinal Studies Centre Scotland; 2009.

⁴⁹² Popham F., Boyle P., O'Reilly D., H Leyland A.H. Exploring the impact of selective migration on the deprivation-mortality gap within Greater Glasgow. Glasgow: Glasgow Centre for population Health; 2010. Available at:

http://www.gcph.co.uk/publications/177 exploring the impact of selective migration (Accessed May 2014).

⁴⁹³ Abbotts J., Williams R., Ford G. Morbidity and Irish Catholic descent in Britain Relating health disadvantage to socio-economic position. Social Science and Medicine. 52 (7) (pp 999-1005), 2001.

⁴⁹⁴ Murden J. Timeline: Liverpool 1699-2004. In: Shrinking Cities (Schrumpfende Städte),
 Complete Works 1 – analysis. Aachen (Germany): ARCH+; 2006. Available from:

www.shrinkingcities.com (Accessed January 2016).

⁴⁹⁵ World Health Organization (WHO). (2000). Obesity: Preventing and Managing the Global Epidemic. WHO:Geneva

⁴⁹⁶ Shelton N. Regional risk factors for health inequalities in Scotland and England and the "Scottish Effect". Social Science and Medicine 2009;69:761-7.

⁴⁹⁷ Zhu J, Coombs N, Stamatakis E. Temporal trends in socioeconomic inequalities in obesity prevalence among economically-active working age adults in Scotland between 1995 and 2011: a population-based repeated cross-sectional study. BMJ Open 2015;5:e006739. doi:10.1136/bmjopen-2014-006739.

⁴⁹⁸ Daniels GA. Underlying influences on health and mortality trends in post-industrial regions in Europe. Glasgow, Glasgow University, 2013.

⁴⁹⁹ Jessop B., Bonnett K., Bromley S., Ling T. Thatcherism: a tale of two nations. Cambridge:Polity; 1988

⁵⁰⁰ Smith R. "Please never let it happen again": lessons on unemployment from the 1930s. *BMJ* 1985; 291: 1191-5.

⁵⁰¹ Birch K, MacKinnon D, Andrew C. Old industrial regions in Europe: a comparative assessment of economic performance. *Regional Studies* 2010; 44(1): 35–53.

⁵⁰² Birch K Mykhnenko V. Varieties of neoliberalism? Restructuring in large industrially dependent regions across Western and Eastern Europe. *J Econ Geogr* 2009; 9(3): 355-380.

⁵⁰³ Peck J, Tickell A. Neoliberalizing Space. Antipode 2002; 34(3): 380-404

⁵⁰⁴ Stuckler D, King L, McKee M. Mass privatisation and the post-communist mortality crisis: a cross-national analysis. *Lancet* 2009; 373: 399-407.

⁵⁰⁵ McCartney G, Walsh D, Whyte B, Collins C. Has Scotland always been the 'sick man' of Europe? European Journal of Public Health 2012; 22(6): 756–760.

⁵⁰⁶ Perchard A. "Broken Men" and "Thatcher's Children": Memory and Legacy in Scotland's Coalfields. International Labor and Working-Class History 2013; 84: 78-98.

⁵⁰⁷ Ahern J, Galea S, Hubbard A, Karpati A. Population vulnerabilities and capacities related to health: a test of a model. Social Science and Medicine. 2008;66(3):691-703

⁵⁰⁸ Hadley C, Rudenstine S, Galea S. How Vulnerabilities and Capacities Shape Population Health after Disasters. In: Freudenberg N, Klitzman S, Saegert S, editors. Urban Health and Society: Interdisciplinary Approaches to Research and Practice. San Francisco, CA: Wiley; 2009

⁵⁰⁹ Karapati A, Galea S, Awerbuch T, Levins R. Variability and Vulnerability at the Ecological Level: Implications for Understanding the Social Determinants of Health. American Journal of Public Health. 2002;92(11):1768-72.

⁵¹⁰ Kirby P. Theorising globalisation's social impact: proposing the concept of vulnerability.Review of International Political Economy. 2006;13(4):632-55

⁵¹¹ Naudé W, McGillivray M, Rossouw S. Measuring the vulnerability of subnational regions in South Africa. Oxford Development Studies. 2009;37(3):249-76

⁵¹² Guillaumont P. An economic vulnerability index : its design and use for international development policy. Oxford Development Studies. 2009;37(3):193-228

⁵¹³ Briguglio L, Cordina G, Farrugia N, Vella S. Economic vulnerability and resilience : concepts and measurements. Oxford development studies. 2009;37(3):229-47

⁵¹⁴ Foster J., Woolfson C. The Politics of the UCS Work-In. London: Lawrence and Wishart; 1986 (p.31)

⁵¹⁵ Corporation of Glasgow. Industry on the Move Glasgow: Corporation of Glasgow; 1959

⁵¹⁶ These exchanges are to be found in the Scottish Office archives, part of the National Records of Scotland located in General Register House in Edinburgh. See file DD28/41 (Urban Renewal 1977 Glasgow District: housing and related topics. UAF/2/2 Pt 4), esp. memo of 17 Nov 1977.

⁵¹⁷ Campbell I. Glasgow's peripheral housing estates: an examination of how their problems surfaced on the policy agenda [unpublished MSc in Public Policy]. Glasgow, University of Strathclyde, 1984.

⁵¹⁸ Cochrane A, Peck J, Tickell A. Olympic dreams: visions of partnership. In: Peck J, Ward K (eds). City of revolution: restructuring Manchester. Manchester, Manchester University Press, 2002.

⁵¹⁹ Jones M, Ward KG. Grabbing grants? The role of coalitions in urban economic development. Local Economy 1998; 13(1): 29-38.

⁵²⁰ Matheson M. Discussion papers in planning, no. 8: housing associations in Glasgow. Glasgow, Department of Town and Regional Planning, University of Glasgow, 1976.

⁵²¹ Pacione M. Quality of life in Glasgow: an applied geographical analysis. *Environment and Planning A* 1986; 18: 1499-1520.

⁵²² Pacione M. The urban crisis: poverty and deprivation in the Scottish city. *Scottish Geographical Magazine* 1989; 105(2): 101-115.

⁵²³ Pacione M. Glasgow: the socio-spatial development of the city. Chichester, Wiley, 1995.

⁵²⁴ Robertson DS. Pulling in opposite directions: the failure of post-war planning to regenerate Glasgow. *Planning Perspectives* 1998; 13(1): 53-67.

⁵²⁵ Sim D. Planners and the peripheral estates. *The Planner* 1990; 76(6): 17-9.

⁵²⁶ Sim D. Beginning to tackle the outer city. *The Planner* 1985; 76(6): 17-9.

⁵²⁷ Wannop U. The evolution and roles of the Scottish Development Agency. *Town Planning Review* 1984; 55(3): 313-321.

⁵²⁸ Sim D. Urban regeneration: not just the inner area. *Area* 1984; 16(\$): 299-306.

⁵²⁹ Boyle M., Acting Director of Social Sciences Institute and Professor of Geography, The National University of Ireland, Maynooth. Personal communication, 2016.

⁵³⁰ Keating M. Chair & Professor of Scottish Politics. Aberdeen University. Personal communication, 2016.

⁵³¹ Carmichael P. Dean of the Faculty of Social Sciences and Professor of Public Policy/Government, University of Ulster. Personal communication, 2016.

⁵³² Boyle R. Chair and Professor of Urban Planning, Wayne State University, Detroit, Michigan. Personal communication, 2016.

⁵³³ McCrone G. Urban Renewal: The Scottish Experience. Urban Studies. 1991;28(6):919-38

⁵³⁴ Johnson R.C., Schoeni R.F. Early-Life Origins of Adult Disease: National Longitudinal
 Population-Based Study of the United States. American Journal of Public Health 2011;
 101(12): 2317–2324

⁵³⁵ ISD Scotland. Births in Scottish Hospitals, Year ending 31 March 2015. Edinburgh: ISD Scotland; 2015. Available from: <u>https://isdscotland.scot.nhs.uk/Health-Topics/Maternity-and-Births/Publications/2015-11-24/2015-11-24-Births-Report.pdf</u> (Accessed January 2016)

⁵³⁶ <u>http://www.poverty.org.uk/s20/index.shtml</u>

⁵³⁷ <u>http://www.scotpho.org.uk/comparative-health/scotland-and-european-hfa-database</u>

⁵³⁸ Potential health effects of contaminants in Soil - SP1002. London, DEFRA, 2009 [available at

http://randd.defra.gov.uk/Default.aspx?Menu=Menu&Module=More&Location=None&Proj ectID=16185&FromSearch=Y&Publisher=1&SearchText=soil&SortString=ProjectCode&SortO rder=Asc&Paging=10#Description].

⁵³⁹ Farmer JG, Jarvis R. Strategies for improving human health in contaminated situations: a review of past, present and possible future approaches. *Environ Geochem Health* 2009; 31: 227–238.

⁵⁴⁰ Broadway A, Cave MR, Wragg J. Determination of the bioaccessibility of chromium in Glasgow soil and the implications for human health risk assessment. *Science of the Total Environment* 2010; 409: 267–277.

⁵⁴¹ Eizaguirre-Garcia D, Rodriguez-Andrew C, Watt GC, Hole D. A study of leukaemia in Glasgow in connection with chromium-contaminated land. *J Public Health Medicine* 1999;

21: 435-438.

⁵⁴² Eizaguirre-Garcia D, Rodriguez-Andrew C, Watt GC. Congenital anomalies in Glasgow between 1982 and 1989 and chromium waste. *J Public Health Medicine* 2000; 22: 54-58.

⁵⁴³ Irvine H. Personal communication, 2012.

⁵⁴⁴ Morrison S, Scott M, Fordyce F. Assessment of Geo-Environmental Inequalities in the Glasgow Conurbation [research poster]. Glasgow, University of Glasgow, undated.

⁵⁴⁵ Morris SE, Thomson AOW, Jarup L, *et al*. No excess risk of adverse birth outcomes in populations living near special waste landfill sites in Scotland. *Scottish Medical Journal* 2003; 48(4): 105-107.

⁵⁴⁶ Irvine HJ. Are Scottish landfill sites really safer than English ones? *Scottish Medical Journal* 2003; 48(4): 102-4.

⁵⁴⁷ Appleton JD, Hooker PJ, Smith NJP. 1995. Methane, carbon dioxide and oil seeps from natural sources and mining areas: characteristics, extent and relevance to planning and development in Great Britain: British Geological Survey, Analytical Geochemistry Series, Technical Report WP/95/1.

⁵⁴⁸ Barron H. Heat from minewaters: energy in the right place [available at <u>https://www.youtube.com/watch?v=xVlie63TbUs]</u>.

⁵⁴⁹ Miles JCH, Appleton JD, Rees DM, *et al*. Radon: indicative atlas in Scotland. Chilton, Health Protection Agency & British Geological Survey, 2011.

⁵⁵⁰ Adams D, De Sousa C. Brownfield Development: A Comparison of North American and British Approaches.

Paper presented at the European Urban Research Association Conference 'The Vital City' held at the University of Glasgow, 12 – 14 September, 2007 [available at http://www.gla.ac.uk/media/media_47883_en.pdf].

⁵⁵¹ Orr G. Lifting the stones. J Law Society Scotland, 17th August 2009 [available at <u>http://www.journalonline.co.uk/Magazine/54-8/1006879.aspx</u>].

⁵⁵² Bambra C., Robertson S., Kasim A., Smith J., Cairns-Nagi J.M., Copeland A, Finlay N, Johnson K. Healthy land? An examination of the area-level association between brownfield land and morbidity and mortality in England. Environment and Planning A 2014; 46: 433-454.

⁵⁵³ Understanding Glasgow Neighbourhood Profiles: http://www.understandingglasgow.com/profiles (Accessed January 2016)

⁵⁵⁴ Riva M., Terashima M., Curtis S., Shucksmith J., Carlebach S. Coalfield health effects:
Variation in health across former coal field areas in England. Health & Place 17(2011) 588–
597.

⁵⁵⁵ Champion, A., Townsend, A., 1990. Contemporary Britain: A geographical Perspective. Edward Arnold, London.

⁵⁵⁶ Beynon H., Hollywood E., Hudson R. Health Issues in the Coal Districts. The Coalfields Research Programme, Discussion Paper No. 7, 1999.

⁵⁵⁷ Mitchell R., Gleave S., Bartley M., Wiggins D., Joshi. H. Do attitude and area influence health? A multilevel approach to health inequalities. Health & Place 6 (2000) 67-79.

⁵⁵⁸ University of Portsmouth and the Great Britain Historical GIS Project: <u>http://www.visionofbritain.org.uk</u> (Accessed May 2013).

⁵⁵⁹ Foster J, Houston M, Madigan C. Irish immigrants in Scotland's shipyards and coalfields: employment relations, sectarianism and class formation. Historical Research 2010 DOI: 10.1111/j.1468-2281.2010.00554.x

⁵⁶⁰ NFO Social Research. Sectarianism in Glasgow - Final report. Prepared for: Glasgow City Council. Glasgow: NFO Social Research; 2003.

⁵⁶¹ T. M. Devine, 'Then and now: Catholicism in Scottish society, 1950–2000', in Scotland's Shame? Bigotry and Sectarianism in Modern Scotland, ed. T. M. Devine (Edinburgh, 2000), pp. 261–5.

⁵⁶² Kelly E. Challenging sectarianism in Scotland: the prism of racism. Scottish Affairs, No. 42, Winter 2003

⁵⁶³ Walls P., Williams R. Accounting for Irish Catholic ill health in Scotland: a qualitative exploration of some links between 'religion', class and health. Sociology of health & illness.
26 (5) (pp 527-556), 2004

⁵⁶⁴ Abbotts J., Williams R., Ford G., Hunt K., West P. Morbidity and Irish Catholic descent in Britain: An ethnic and religious minority 150 years on. Social Science and Medicine. 45 (1) (pp 3-14), 1997

⁵⁶⁵ Bruce S., Glendinning T., Paterson I., Rosie M. Sectarianism in Scotland. Edinburgh University Press, 2004.

⁵⁶⁶ French D. Residential segregation and health in Northern Ireland. Health and Place. 15 (3) (pp 873-881), 2009

⁵⁶⁷ O'Reilly D., Rosato M. Religious affiliation and mortality in Northern Ireland: Beyond Catholic and Protestant. Social Science and Medicine. 66 (7) (pp 1637-1645), 2008

⁵⁶⁸ O'Reilly D., Stevenson M . The two communities in Northern Ireland: Deprivation and ill health. Journal of Public Health Medicine. 20 (2) (pp 161-168), 1998

⁵⁶⁹ Jamison J., O'Reilly D., Carr-Hill R., Stevenson M. Equity of use of inpatient services by the two communities in Northern Ireland. Journal of Health Services Research and Policy. 9 (1) (pp 34-38), 2004

⁵⁷⁰ Antonovsky A. Health, stress and coping. San Francisco: Jossey-Bass; 1979.

⁵⁷¹ Antonovsky A. Unravelling the mystery of health. How people manage stress and stay well. San Francisco: Jossey-Bass; 1987.

⁵⁷² Eriksson M, Lindström B. Validity of Antonovsky's sense of coherence scale: a systematic review. Journal of Epidemiology and Community Health 2005;59(6):460-466.

⁵⁷³ Eriksson M., Lindström B. Antonovsky's sense of coherence scale and its relation with quality of life: a systematic review. Journal of Epidemiology and Community Health 2007;61:938-944.

⁵⁷⁴ Van Leeuwen CMC, Kraaijeveld S, Lindeman E, Post MWM. Associations between psychological factors and quality of life ratings in persons with spinal cord injury: a systematic review. Spinal Cord 2012;50(3):174-187.

⁵⁷⁵ Eriksson M, Lindström B. Antonovsky's sense of coherence scale and the relation with health: a systematic review. Journal of Epidemiology and Community Health 2006;60:376-381.

⁵⁷⁶ Flensborg-Madsen T, Ventegodt S, Merrick J. Sense of Coherence and Physical Health. A Review of Previous Findings. The Scientific World Journal 2005;5:665-673.

⁵⁷⁷ Coward D. Self-transcendence and correlates in a healthy population. Nursing Research 1996;45(2):116-121.

⁵⁷⁸ Edwards D, Besseling E. Relationship between depression, anxiety, sense of coherence, social support and religious involvement in a small rural community affected by industrial relations conflict. South African Journal of Psychology 2001;31(4):62-72.

⁵⁷⁹ Gibson LM. Inter-relationships among sense of coherence, hope, and spiritual perspective (inner resources) of African-American and European-American breast cancer survivors. Applied Nursing Research 2003;16(4):236-44.

⁵⁸⁰ Matsuura E, Ohta A, Kanegae F. Haruda Y, Ushiyama O, Koarada S, Togashi R, Tada Y, Suzuki N, Nagasawa K. Frequency and analysis of factors closely associated with the development of depressive symptoms in patients with scleroderma. Journal of Rheumatology 2003;30(8):1782-1787.

⁵⁸¹ Eriksson NG, Lundin T. Early traumatic stress reactions among Swedish survivors of the m/s Estonia disaster. British Journal of Psychiatry 1996;169:713-716.

⁵⁸² Kouvonen AM, Vaananen A, Vahtera J, Heponiemi T, Koskinen A, Cox SJ, Kivimaki M. Sense of coherence and psychiatric morbidity: a 19-year register-based prospective study. Journal of Epidemiology and Community Health 2010;64(3):255-261.

⁵⁸³ Sjostrom N, Hetta J, Waern M. Sense of coherence and suicidality in suicide attempters: a prospective study. Journal of Psychiatric and Mental Health Nursing 2012;19(1):62-69.

⁵⁸⁴ Ristkari T, Sourander A, Ronning JA, Elonheimo H, Helenius H. Sense of coherence and criminal offences among young males. Findings from the Finnish 1981 Nationwide Birth Cohort Follow-Up Study. European Child and Adolescent Psychiatry Conference: 14th International Congress of ESCAP European Society for Child and Adolescent Psychiatry: Helsinki Finland; 2011

⁵⁸⁵ Glanz K, Maskarinec G. Ethnicity, Sense of Coherence, and Tobacco Use among Adolescents. Society of behavioural medicine 2005;29:192-199.

⁵⁸⁶ Nilsson KW, Starrin B, Simonsson B, Leppert J. Alcohol-related problems among adolescents and the role of a sense of coherence. International Journal of Social Welfare 2007:16(2):159-167.

⁵⁸⁷ Arevalo S, Prado G, Amaro H. Spirituality, sense of coherence, and coping responses in women receiving treatment for alcohol and drug addiction. Evaluation and Program Planning 2008;31:113-123.

⁵⁸⁸ Lundberg O, Peck MN. Sense of coherence, social structure and health. Evidence from a population survey in Sweden. European Journal of Public Health 1994;4:252-257.

⁵⁸⁹ Agardh EE, Ahlbom A, Andersson T, Efendic S, Grill V, Hallqvist J, Norman A, Stenson CG. Work Stress and Low Sense of Coherence Is associated with type 2 Diabetes in Middle-aged Swedish Women. Diabetes Care 2002;26:719-724.

⁵⁹⁰ Ristner G, Andersson R, Johansson LM, Johansson SE, Ponzer S. Sense of coherence and lack of control in relation to outcome after orthopaedic injuries. Injury 2000;31(10):751-756.

⁵⁹¹ Ray EC, Nickels MW, Sayeed S, Sax HC. Predicting success after gastric bypass: the role of psychosocial and behavioral factors. Surgery 2003;134(4):555-564.

⁵⁹² Wainwright NWJ, Surtees PG, Welch AA, Luben RN, Khaw KT, Bingham SA. Sense of coherence, lifestyle choices and mortality. Journal of Epidemiology and Community Health 2008;62(9):829-831.

⁵⁹³ Krishna A, Uphoff N. Mapping and Measuring Social Capital: A Conceptual and Empirical Study of Collective Action for Conserving and Developing Watersheds in Rajastan, India. *In*: Social Capital Initiative Working Paper no. 13 Washington, DC, USA: World Bank; 1999.

⁵⁹⁴ McKenzie K, Whitley R, Weich S. Social Capital and mental health. *British Journal of Psychiatry* 2002;181:280-283.

⁵⁹⁵ Harpham T, Grant E, Thomas E. Measuring social capital within health surveys: key issues. *Health Policy and Planning* 2002;17(1):106-111.

⁵⁹⁶ Coleman J.S. Foundations of Social Theory. Cambridge, MA, USA: Harvard University Press; 1990.

⁵⁹⁷ Cote S., Healy T. The Well-being of Nations. The role of human and social capital. Paris: Organisation for Economic Co-operation and Development; 2001. ⁵⁹⁸ Putnam RD. Bowling alone: America's declining social capital. Journal of Democracy 1995; 6(1): 65-78.

⁵⁹⁹ Lochner K., Kawachi I., Kennedy B.P. Social capital: a guide to its measurement. Health & Place 1999; 5(4): 259-270.

⁶⁰⁰ Glaeser E.L., Laibson D., Sacerdote B. An Economic Approach to Social Capital. The Economic Journal 2002; 112: F437-F458.

⁶⁰¹ Durlauf S., Fafchamps M. Social Capital. National Bureau of Economic Research (NBER)
 Working Paper 10485 National Bureau of Economic Research; 2004. Available at:
 <u>http://www.nber.org/papers/w10485</u> (accessed April 2013)

⁶⁰² Baum F. Social capital: is it good for your health? Issues for a public health agenda. Journal of Epidemiology & Community Health 1999; 53(4): 195-196.

⁶⁰³ Durlauf S. Bowling Alone: A Review Essay. Journal of Economic Behavior and Organization 2000; 47(3): 259-273.

⁶⁰⁴ Manaski C.F. Economic analysis of social interactions. Journal of Economic Perspectives 2000; 14(3): 115-136.

⁶⁰⁵ Kawachi I., Kim D., Coutts A., Subramanian S.V. Reconciling the three accounts of social capital. International Journal of Epidemiology 2004; 33(4): 682-690.

⁶⁰⁶ Macinko J., Starfield B. The utility of social capital in research on health determinants. The Milbank Quarterly 2001; 79(3): 387-427.

⁶⁰⁷ Lundborg P. Having the wrong friends? Peer effects in adolescent binge drinking, smoking and illicit-drug use. *In*: Risky Health Behavior Among Adolescents. Lund Economic Studies 10, Department of Economics, Lund University, Sweden; 2003.

⁶⁰⁸ Kawachi I. Social Capital and Community Effects on Population and Individual Health. Annals of the New York Academy of Sciences 1999; 896(1): 120-130.

⁶⁰⁹ Kawachi I., Kennedy B., Lochner K., Prothrow-Stith D. Social Capital, Income Inequality, and Mortality. American Journal of Public Health 1997; 87(9): 1491-1498.

⁶¹⁰ Wilkinson R.G., Kawachi I., Kennedy B.P. Mortality, the social environment, crime and violence. Sociology of Health and Illness 1998; 20(5): 578-597.

⁶¹¹ Lochner K., Kawachi I., Brennan R.T., Buka S.L. Social capital and neighborhood mortality rates in Chicago. Social Science & Medicine 2003; 56(8): 1797-1805.

⁶¹² Kennedy B., Kawachi I., Brainerd E. The role of social capital in the Russian mortality crisis. World Development 1998; 26(11): 2029-2043.

⁶¹³ Skrabski A., Kopp M., Kawachi I. Social capital in a changing society: cross sectional associations with middle aged female and male mortality rates. Journal of Epidemiology &

Community Health 2003; 57(2): 114-119.

⁶¹⁴ Skrabski A., Kopp M., Kawachi I. Social capital and collective efficacy in Hungary: cross sectional associations with middle aged female and male mortality rates. Journal of Epidemiology & Community Health 2004; 58(4): 340-345.

⁶¹⁵ Hyppä M.T., Mäki J. Why do Swedish-speaking Finns have longer active life? An area of social capital research. Health Promotion International 2001; 16(1): 55-64.

⁶¹⁶ Siahpush M., Singh G.K. Social integration and mortality in Australia. Australian and New Zealand Journal of Public Health 1999; 23(6): 571-577.

⁶¹⁷ Kripper C.E., Sapag J.C. Social capital and health in Latin America and the Caribbean: a systematic review. Pan American Journal of Public Health 2009; 25(2): 162-170.

⁶¹⁸ Murayama H., Fujiwara Y., Kawachi I. Social capital and health: a review of prospective multilevel studies. Journal of Epidemiology 2012; 22(3): 179-187.

⁶¹⁹ Pearce N., Davey Smith G. Is Social Capital the Key to Inequalities in Health? American Journal of Public Health. 2003; 93: 122–129

⁶²⁰ Uphoff E.P., Pickett K.E., Cabieses B. Small N., Wright J. A systematic review of the relationships between social capital and socioeconomic inequalities in health: a contribution to understanding the psychosocial pathway of health inequalities. International Journal for Equity in Health 2013, 12:54

⁶²¹ Maselko J., Hughes C., Cheney R. Religious social capital: Its measurement and utility in the study of the social determinants of health. Social Science & Medicine 2011; 73(5): 759-767.

⁶²² McCullough M.E., Hoyt W.T., Larson D.B., Koenig H.G., Thorensen C.E. Religious involvement and mortality: a meta-analytic review. Health Psychology 2000; 19(3): 211-222.

⁶²³ Coruh B., Ayele H., Pugh M., Mulligan T. Does Religious Activity Improve Health
Outcomes? A Critical Review of the Recent Literature. Journal of Science and Healing 2005;
1(3): 186-191.

⁶²⁴ Hummer R.A., Ellison C.G., Rogers R.G., Moulton B.E., Romero R.R. Religious involvement and adult mortality in the United States: Review and perspective. Southern Medical Journal 2004; 97(12): 1223-1230.

⁶²⁵ Gearing R.E., Lizardi D. Religion and suicide. Journal of Religion and Health 2009; 48(3):332-341

⁶²⁶ Kawachi I, Berkman LF. Social cohesion, social capital, and health. *In*: Berkman LF,Kawachi I, editors. Social epidemiology. Oxford: Oxford University Press; 2000. pp 174-190.

⁶²⁷ Verba S., Schlozman K.L., Brady H.E. Voice and Equality. Civic Voluntarism in American Politics. Cambridge, MA, USA: Harvard University Press; 1995.

⁶²⁸ Kawachi I., Kennedy B.P., Wilkinson R.G. Income Inequality and Health: A Reader. New York, NY, USA: The New Press; 1999.

⁶²⁹ Kawachi I., Kennedy B.P. Health and social cohesion: why care about income inequality? British Medical Journal 1997; 314(7086): 1037-1040.

⁶³⁰ Hill K.Q., Leighley J.E. The policy consequences of class bias in state electorates. American Journal of Political Science 1992; 36: 351-363.

⁶³¹ Koenig H.G., McCullough M.E., Larson D.B. Handbook of Religion and Health. Oxford: Oxford University Press; 2001.

⁶³² Ellison C.G., Levin J.S. The religion-health connection: Evidence, theory, and future directions. Health Education & Behavior 1998; 25(6): 700-720.

⁶³³ Pargament K.L. The psychology of religion and coping. Theory, research, practice. New York, NY, USA: The Guilford Press; 1997.

⁶³⁴ Jonker L., Greeff A.P. Resilience factors in families living with people with mental illnesses. Journal of Community Psychology 2009; 37(7): 859-873.

⁶³⁵ Gall T.L., Charbonneau .C, Clarke N.H. Grant K., Joseph A., Shouldice L. Understanding the nature and role of spirituality in relation to coping and health: a conceptual framework. Canadian Psychology-Psychologie Canadienne 2005; 46(2): 88-104.

⁶³⁶ Borgonovi F. Doing well by doing good. The relationship between formal volunteering and self-reported health and happiness. Social Science & Medicine 2008; 66(11): 2321-2334.

⁶³⁷ Nicolet S., Tresch A. Changing religiosity, changing politics? The influence of 'belonging' and 'believing' on political attitudes in Switzerland. Politics and Religion 2009; 2(1): 76-99.

⁶³⁸ Pollack D., Pickel G. Religious individualism or secularism? Testing hypotheses of religious change – the case of Eastern and Western Germany. British Journal of Sociology 2007; 58(4):
603-632.

⁶³⁹ Davie G. Religion in Britain since 1945: Believing without belonging. Oxford, UK: Blackwell; 1994.

⁶⁴⁰ Davie G. Religion in Modern Europe. Oxford, UK: Oxford University Press; 2000.

⁶⁴¹ Spoerri A., Zwahlen M., Bopp M. *et al*. Religion and assisted and non-assisted suicide in
Switzerland: National Cohort Study. International Journal of Epidemiology 2010;39(6):148694.

⁶⁴² Boyle P., Exeter D., Feng Z. Flowerdew R. Suicide gap among young adults in Scotland: population study. British Medical Journal 2005; 330: 175.

⁶⁴³ Platt S., Boyle P., Crombie I., Feng Z., Exeter D. The epidemiology of suicide in Scotland 1989-2004: an examination of temporal trends and risk factors at national and local levels. Edinburgh: Scottish Executive Social Research; 2007.

⁶⁴⁴ Ralston K., Walsh D., Feng Z., Dibben C., McCartney G., O'Reilly D. Religious affiliation and excess mortality in the UK. Forthcoming.

⁶⁴⁵ Craig C. The Scots' Crisis of Confidence. Glasgow: Big Thinking; 2005.

⁶⁴⁶ Bartley M., Plewis I. Does health-selective mobility account for socioeconomic differences in health? Evidence from England and Wales, 1971 to 1991. Journal of Health & Social Behavior 1997; 38(4): 376-386.

⁶⁴⁷ Hart C. L., Smith G. D., Blane D. Social mobility and 21 year mortality in a cohort of Scottish men. Social Science & Medicine 1998; 47(8): 1121-1130

⁶⁴⁸ Blane D., Harding S., Rosato M. Title Does social mobility affect the size of the socioeconomic mortality differential?: evidence from the Office for National Statistics Longitudinal Study. Journal of Royal Statistical Society, Series A 1999; 162 (1): 59-70.

⁶⁴⁹ Boyle P.J., Norman P., Popham, F. Social mobility: Evidence that it can widen health inequalities. Social Science and Medicine 2009; 68(10): 1835-1842.

⁶⁵⁰ Power C. Inequalities in self rated health in the 1958 birth cohort: lifetime social circumstances or social mobility? BMJ 1996; 313: 449.

⁶⁵¹ Das-Munshi J., Leavey G., Stansfeld S. A., Prince M. J. Migration, social mobility and common mental disorders: critical review of the literature and meta-analysis. Ethnicity & Health 2012; 17 (1-2): 17-53.

⁶⁵² Simons A.M.W., Groffen D. A. I., Bosma H. Socio-economic inequalities in all-cause mortality in

Europe: an exploration of the role of heightened social mobility. European Journal of Public Health, Vol. 23, No. 6, 1010–1012

⁶⁵³ Fox A. J., Goldblatt P. O., Jones D. R. Social class mortality differentials: artefact, selection or life circumstances? Journal of Epidemiology & Community Health 1985; 39(1): 1-8.

⁶⁵⁴ Smith GD, Hart C, Watt DG, Hole D, Hawthorne V. Individual social class, area-based deprivation, cardiovascular disease risk factors, and mortality: the Renfrew and Paisley study. Journal of Epidemiology & Community Health 1998;52:399-402.

⁶⁵⁵ Erikson R., Goldthorpe J. The Constant Flux: a study of class mobility in industrial societies. Oxford: Clarendon; 1993

⁶⁵⁶ Paterson L., Iannelli, C. Patterns of absolute and relative social mobility: a comparative study of England, Wales and Scotland. Sociological Research Online 2007; 12: 6. Available from: <u>http://www.socresonline.org.uk/12/6/15.html</u> (Last accessed July 2015)

⁶⁵⁷ Currie C, Todd J. Mental well-being among schoolchildren in Scotland: age and gender

patterns, trends and cross-national comparisons. Edinburgh: Child & Adolescent Health Research Unit, The University of Edinburgh; 2003.

⁶⁵⁸ Schwarzer R, Urte Scholz U. Cross-cultural assessment of coping resources: the general perceived self-efficacy scale. Paper presented at the Asian Congress of Health Psychology 2000: Health Psychology and Culture, Tokyo, Japan, August 28-29, 2000.

⁶⁵⁹ Sridharan, S., Tunstall, H., Lawder, R., & Mitchell, R. An exploratory spatial data analysis approach to understanding the relationship between deprivation and mortality in Scotland. Social Science & Medicine 2007; 65: 1942–1952.

⁶⁶⁰ Allender S., Scarborough P., Keegan T., Rayner M. Relative deprivation between neighbouring wards is predictive of coronary heart disease mortality after adjustment for absolute deprivation of wards. Journal of Epidemiology & Community Health 2012; 66(9): 803-808.

⁶⁶¹ Maheswaran, R., Craigs, C., Read, S., Bath, P. & Willett, P. A graph-theory method for pattern identification in geographical epidemiology - a preliminary application to deprivation and mortality. International Journal of Health Geographics 2009; 8: 28

⁶⁶² Astell-Burt, T., Feng, X. Investigating 'place effects' on mental health: implications for population-based studies in psychiatry. Epidemiology and Psychiatric Sciences 2015; 24: 27-37.

⁶⁶³ Cox, M., Boyle, P. J., Davey, P. G., Feng, Z. & Morris, A. D. 2007. Locality deprivation and Type 2 diabetes incidence: a local test of relative inequalities. Social Science & Medicine 2007; 65: 1953-64

⁶⁶⁴ Livingston M, Lee D. "The Glasgow effect?"- the result of the geographical patterning of deprived areas? Health & Place. 2014 Sep;29:1-9.

⁶⁶⁵ Zhang, X., Cook, P., Jarman, I. & Lisboa, P. Area effects on health inequalities: The impact of neighbouring deprivation on mortality. Health & Place 2011; 17: 1266-1273.

⁶⁶⁶ Dunn, J. R. & Cummins, S. 2007. Placing health in context. Social Science & Medicine, 65, 1821-1824

⁶⁶⁷ Stewart J. *et al*. Using population surfaces and spatial metrics to track the development of spatial patterns of poverty over time. Forthcoming.

⁶⁶⁸ Ananat, E. O., Gruber, J., Levine, P., & Staiger, D. (2009). Abortion and selection. Review of Economics and Statistics, 91(1), 124-136.

⁶⁶⁹ Gruber, J., Levine, P., & Staiger, D. (1999). Abortion legalization and child living circumstances: who is the "marginal child"? Quarterly Journal of Economics, 114, 263-291.

⁶⁷⁰ Bitler, M. P., & Zavodny, M. (2002). Child abuse and abortion availability. AEA Papers and Proceedings, 92(2), 363-367

⁶⁷¹ Sen B., Wingate M.S., Kirby R. The relationship between state abortion-restrictions and homicide deaths among children under 5 years of age: a longitudinal study. Social Science & Medicine 75 (2012) 156-164.

⁶⁷² Donohue, John J. and Levitt, Steven D., The Impact of Legalized Abortion on Crime (2000). Quarterly Journal of Economics. Available at: <u>http://dx.doi.org/10.2139/ssrn.174508</u> (last accessed: May 2012)

⁶⁷³ Levitt S., Dubner S.J. Freakonomics: a rogue economist explores the hidden side of everything. William Morrow: 2005.

⁶⁷⁴ Kobayashi J. On geographical relationship between the chemical nature of river water and death rate from apoplexy. Ber des Ohara Institutes fur Landurtschaftuche Biologie 1957;11:12–21.

⁶⁷⁵ Schroeder H. Relationship between mortality from cardiovascular disease and treated water supplies. Variations in states and 163 largest municipalities of the United States. J Am Med Assoc 1960;172:1902–8.

⁶⁷⁶ Rubenowitz E, Axelsson G, Rylander R. Magnesium and calcium in drinking water and death from acute myocardial infarction in women. Epidemiology 1999; 10:31–36

⁶⁷⁷ Leurs L.J., Schouten L.J., Mons M.N., Goldbohm R.A., Van Den Brandt P.A. Relationship between tap water hardness, magnesium, and calcium concentration and mortality due to ischemic heart disease or stroke in the Netherlands. Environmental Health Perspectives 2010; 118 (3): 414-420.

⁶⁷⁸ Morris R.W., Walker M., Lennon L.T., Shaper A.G., Whincup P.H. Hard drinking water does not protect against cardiovascular disease: New evidence from the British Regional Heart Study. European Journal of Cardiovascular Prevention and Rehabilitation 2008; 15 (2): 185-189.

⁶⁷⁹ Smith WC, Crombie IK. Coronary heart disease and water hardness in Scotland. Is there a relationship? J Epidemiol Community Health 1987; 41:227–228

⁶⁸⁰ Lake I.R., Swift L., Catling L.A., Abubakar I., Sabel C.E., Hunter P.R. Effect of water hardness on cardiovascular mortality: An ecological time series approach. Journal of Public Health. 32 (4) (pp 479-487), 2010

⁶⁸¹ Monarca S., Donato F., Zerbini I., Calderon R.L., Craun G.F. Review of epidemiological studies on drinking water hardness and cardiovascular diseases. European Journal of Cardiovascular Prevention & Rehabilitation 2006; 13(4):495-506.

⁶⁸² Catling L.A., Abubakar I., Lake I.R., Swift L., Hunter P.R. A systematic review of analytical observational studies investigating the association between cardiovascular disease and drinking water hardness. Journal of water and health 2008; 6 (4): 433-442.

⁶⁸³ Rylander, R. Magnesium in drinking water - A case for prevention? Journal of Water and

Health 2014; 12 (1): 34-40.







