

ACCOUNTING FOR SCOTLAND'S EXCESS MORTALITY: TOWARDS A SYNTHESIS

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SUMMARY

Introduction

Scotland, because of the higher mortality of its population, has been dubbed 'the sick man of Europe'. This higher mortality is most apparent in the west of Scotland and in the city of Glasgow in particular.

Yet Scottish mortality was not always higher: it was only after 1950 that the rates improved more slowly than elsewhere in Europe. As late as 1981, most of the excess in Scotland as compared to England & Wales could be explained by deprivation. Over the next 20 years this excess increased, and the scope to account for it by reference to deprivation declined, raising the question of how to account for the balance.

The immediate causes are known: high rates of alcohol and drug-related deaths, suicide, violence, cardiovascular disease, stroke and cancer. But what are the underlying causes? There are multiple candidate hypotheses regarding both:

- the divergence of the Scottish mortality pattern from the rest of Europe from around 1950;
- the rise in excess mortality unexplained by deprivation from 1980 in Scotland (and Glasgow) as against the rest of the UK.

This report uses the criteria developed by Bradford-Hill for causation in observational epidemiology to evaluate each of these candidate hypotheses. As none of the hypotheses seems likely to provide for a 'total' explanation, a synthesis is attempted. Finally recommendations are made for future research.

Methods

A structured literature review was carried out to identify candidate hypotheses. This was informed and supplemented by notes taken from discussions arising in the dissemination events for previous research into the Scottish mortality phenomenon. Each hypothesis was then described and critically evaluated in relation to the Bradford-Hill criteria. The fit of each hypothesis with the criteria was tabulated. A synthesis of the hypotheses which appeared robust against the criteria was then attempted, using a broadly 'dialectical' approach. That is, an attempt was made to develop a 'higher order' hypothesis, retaining and integrating the partial truths and insights achieved in the pre-existing hypotheses, but in a new context which aspires towards a broader and deeper grasp of the totality of the problem.

Results

Seventeen hypotheses were identified including: artefactual explanations (deprivation and migration); 'downstream explanations' (health behaviours and individual values); 'midstream' explanations (culture of substance misuse; boundlessness and alienation; family, gender relations and parenting differences; lower 'social capital'; sectarianism; culture of limited social mobility; health service supply or demand, and deprivation concentration); and 'upstream' explanations (inequalities; deindustrialisation; 'political attack', and climatic differences). Finally, a genetic explanation has been proposed.

The higher mortality in Scotland between 1950 and 1980 was largely due to a slower reduction in deaths from cardiovascular disease, stroke, respiratory disease and cancer. There is little evidence available to determine why this happened. On balance, it is likely that a greater exposure to negative health behaviours goes some way to explaining it. Cultural factors may have been operant, but it is less likely that they are the cause of the divergent pattern. It is unclear whether Scotland suffered from greater poverty and inequality than other European nations around 1950, but it is possible, and in terms of historical accounts plausible, that particular industrial employment patterns, together with particular kinds of housing and urban environments, all linked to specific community and family dynamics, and in turn to negative health behaviour cultures, cumulatively led to a slower improvement than in other populations. This would fit with the mortality pattern in Glasgow being similar to those in Liverpool and Manchester during this time. However, significant new work would be required to investigate this. It has been suggested that there may be a role for lower levels of vitamin D in driving this higher mortality, but the evidence for this at present remains unclear.

The higher mortality in Scotland and Glasgow from 1980 was largely due to alcohol-related deaths, drug-related deaths, suicides, violent deaths and road-traffic accidents in young adults (in addition to the continuing high rates of mortality for cardiovascular disease, cancer and stroke). Furthermore, the available measures of deprivation accounted for proportionately less of the growing excess mortality after this time. It is likely that a proportion of the excess mortality not explained by these measures is in fact due to deprivation, and that the measures have become dated. However, comparisons of Glasgow, Liverpool and Manchester provide strong evidence that there is an excess in the Scottish city beyond that attributable to deprivation. The cause which fits best with the Bradford-Hill criteria concerns the prevalence of various health behaviours. Although these exposures are causal, the synthesis requires an explanation for their higher prevalence or more profound effects in the Scottish context. More culturally orientated hypotheses are plausible and have some evidence, but require further comparative research and are challenged in terms of explaining why the Scottish and Glaswegian populations were worse affected than other areas from the early 1980s (rather than at an earlier time). Structural explanations fit well with the Bradford-Hill criteria and are likely to have played an important causal role, but on the basis of the available evidence and the application of the Bradford-Hill criteria, the political attack hypothesis seems best placed to bring together the most likely behavioural, cultural and structural determinants of health into a coherent narrative which can explain the post-1980 mortality phenomenon. This hypothesis

suggests that the neoliberal government policies implemented from 1979 onwards disproportionately affected the Scottish, and in particular the west of Scotland and Glaswegian populations, and that this in turn was causally implicated in changing behavioural patterns leading to negative health outcomes. However, other posited explanations, for which there is currently little or no research evidence, may prove relevant. It is important, therefore, that further research relevant to these hypotheses is undertaken.

Conclusions

The reasons for the high Scottish mortality between 1950 and 1980 are unclear, but poverty and deprivation linked to particular industrial employment patterns, poor housing and unhealthy cultural and behavioural patterns seem the more likely explanations. From 1980 onwards the mortality pattern changed and this seems most likely to be attributable to the changed political context, produced by neoliberal political attack, and the consequent hopelessness and community disruption experienced in Scotland and Glasgow. This perspective may have relevance to faltering health improvement in other countries, such as the USA. Further research, linked to integration and synthesis of the most likely causal explanations, is merited, as is further work to design policies and interventions to create a healthier future for Scottish communities.

Keywords

Scotland; Glasgow; mortality; Scottish Effect; Glasgow Effect.

PURPOSE

This purpose of this report is to:

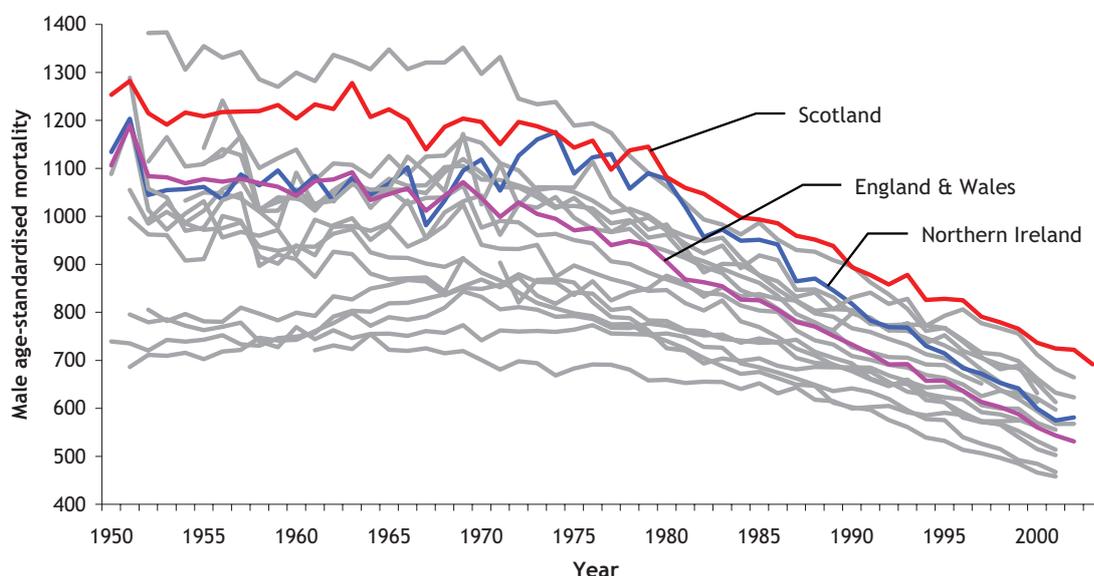
1. Identify the various hypotheses that have been proposed to explain the higher mortality rate in Scotland, the West of Scotland and Glasgow, as compared to the rest of Europe; and the additional mortality relative to the rest of the UK unexplained by markers of deprivation.
2. Evaluate how well each hypothesis explains the trends by reviewing the current evidence base in light of the Bradford-Hill criteria for causation in observational epidemiology.
3. Begin the process of synthesis required to create a higher level of understanding of the mortality phenomenon in Scotland.
4. Suggest future research approaches that might help to clarify which factor(s) are most important in explaining the trends.

INTRODUCTION

Divergence of Scottish mortality from the rest of Europe

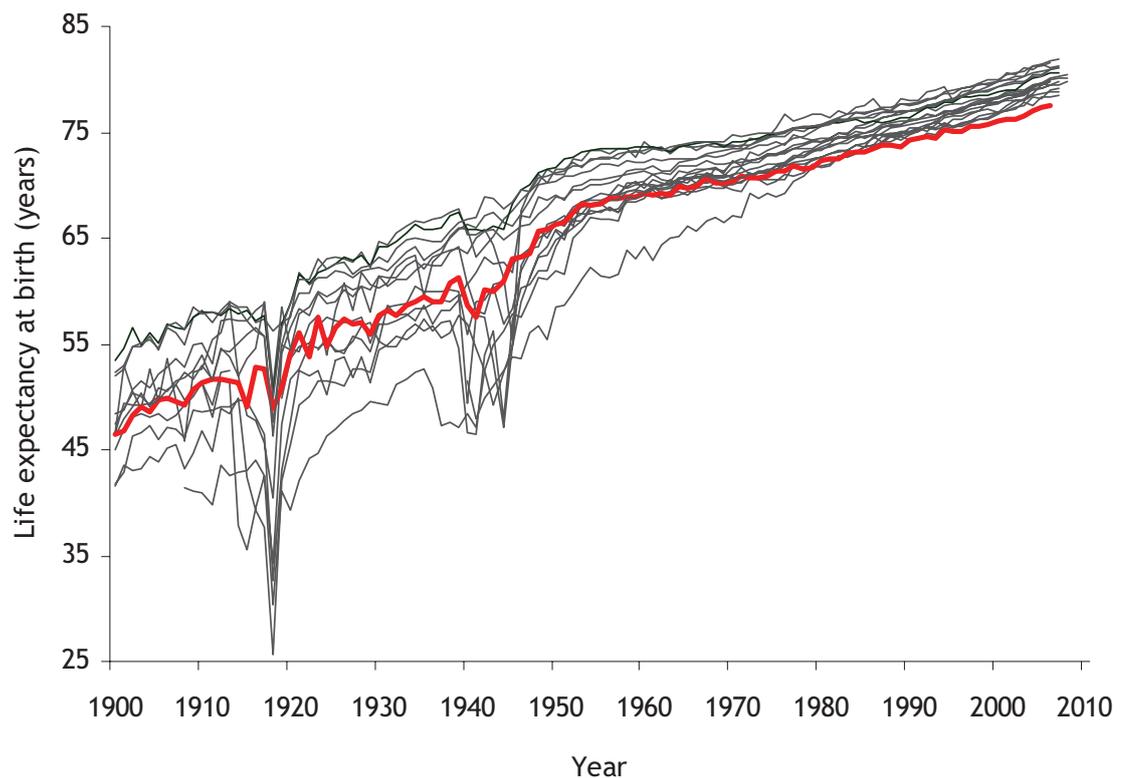
The mortality rate in Scotland, and even more so in the West of Scotland and Glasgow, is an outlier compared to the rest of Western Europe and the rest of the UK (Figure 1). Yet mortality has not always been worse in Scotland: it was only from around 1950 onwards that Scottish life expectancy diverged from that of other European countries (Figure 2). For the first 50 years of the 20th Century, Scottish mortality rates were broadly comparable with those of other Western European countries and there was no distinctive Scottish mortality problem.¹ Indeed at the end of the 19th Century infant mortality rates in Scotland were lower than in England & Wales and Ireland.² A similar divergence is seen with smaller geographical areas, where mortality in the West of Scotland improved at a slower rate than other similar UK and European areas in the latter half of the 20th Century, even where the other areas started from having higher mortality, or experienced greater poverty or unemployment.^{3,4} The higher mortality from 1950 in Scotland was largely attributable to cardiovascular disease, stroke and cancer (alcohol- and drug-related deaths, 'external' causes and suicide were all lower in Scotland from 1950 to 1980 compared to other Western European countries).¹

Figure 1 - Trends in Scottish male mortality in comparison to other Western European countries and other parts of the UK (data from Whyte 2007; the pattern for females is similar)⁵



Data from: Austria, Belgium, Denmark, Finland, France, Germany, Greece, Ireland, Italy, Northern Ireland, Netherlands, Norway, Portugal, Spain, Sweden, Switzerland, England & Wales, and Scotland.

Figure 2 - Trends in life expectancy of the Scottish population (in red) in comparison to other Western European countries* during the 20th Century (data from the Human Mortality Database)⁶



*Western European nations for which data was available were: Austria, Belgium, Denmark, England & Wales, Finland, France, Ireland, Iceland, Italy, Luxembourg, Netherlands, Northern Ireland, Norway, Portugal, Scotland, Spain, Sweden, Switzerland and West Germany.

The emergence of the Scottish (and Glasgow) Effect

The higher adult mortality in Scotland (and the West of Scotland and Glasgow in particular) from the 1980s onwards can be attributed to: alcohol-related and drug-related deaths; 'external causes' (principally violent deaths and road-traffic accidents), and suicide - as well as continuing trends of higher cardiovascular disease,⁷ cancer and stroke.^{3-5, 8} The higher mortality in young males in Scotland was almost entirely accounted for by the most deprived areas of the West of Scotland whereas the higher mortality for women was more evenly spread across Scotland.⁸ More recently, the age-standardised mortality trends for Glasgow were compared to those of Liverpool and Manchester (English cities with deprivation profiles near identical to that of Glasgow). The experience in Glasgow was worse than in the English cities across all deprivation deciles (i.e. was ubiquitously worse for rich and poor within the cities (Figure 3)). Again, most of the divergence emerged after 1980 (Figure 4).⁹

Figure 3 - Standardized all-cause mortality ratios 2003–2007 for Glasgow relative to Liverpool and Manchester (combined), broken down by deprivation decile, for (a) all deaths and (b) deaths under 65 years of age (from Walsh 2010).⁹

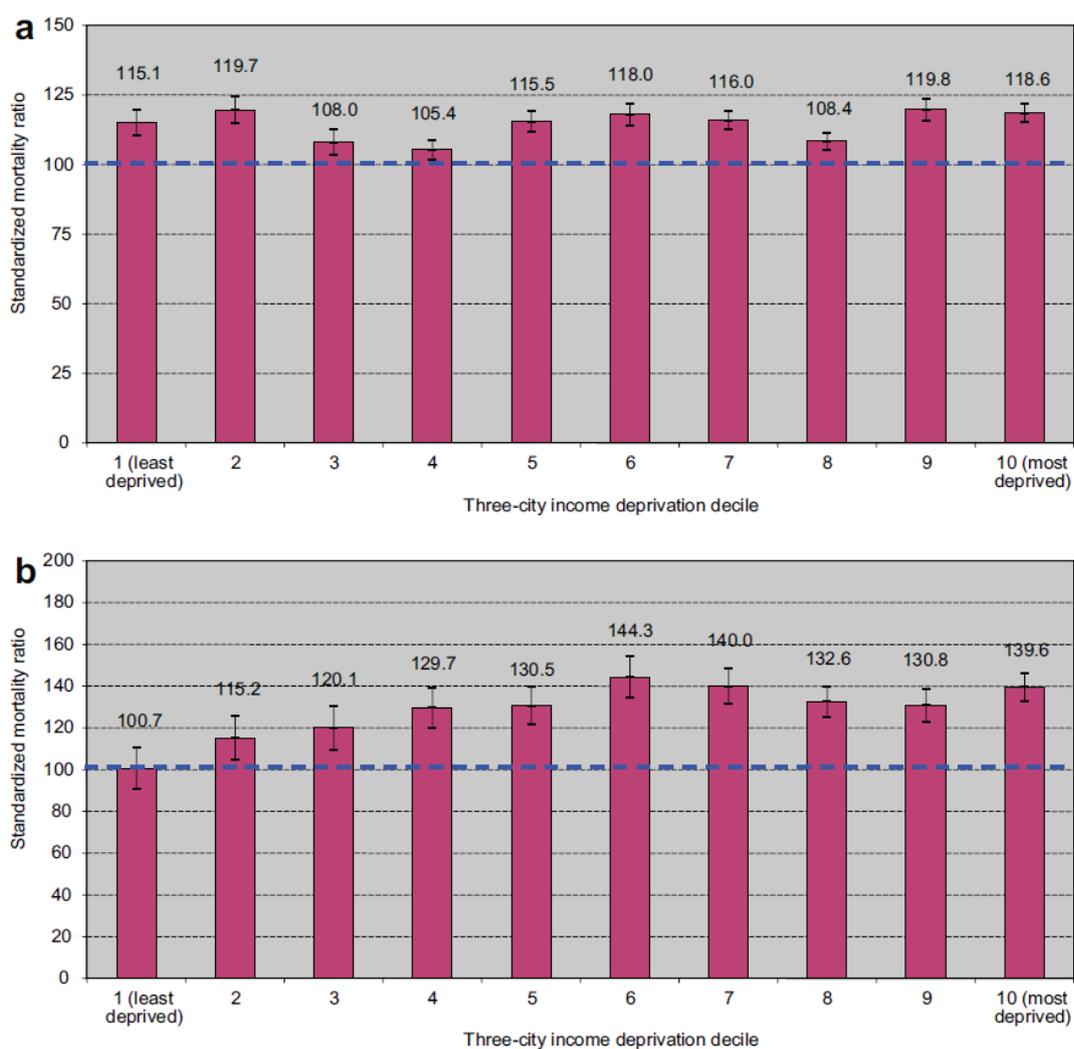
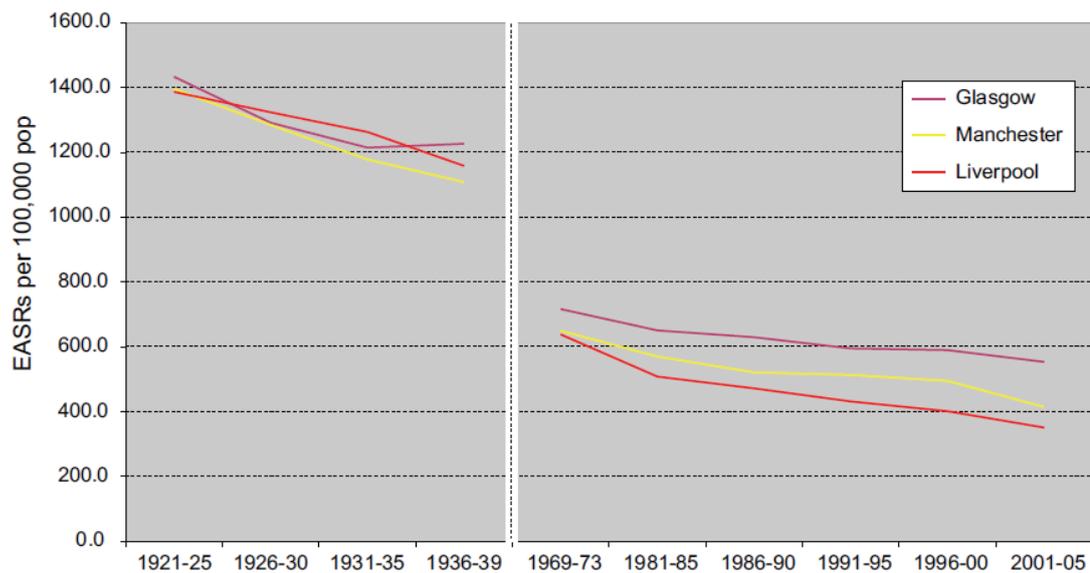


Figure 4 - Male premature (<65 years) mortality: European age-standardized mortality rates (EASRs) per 100,000 population, 1921/25–2001/05, Glasgow, Liverpool and Manchester (from Walsh 2010).⁹



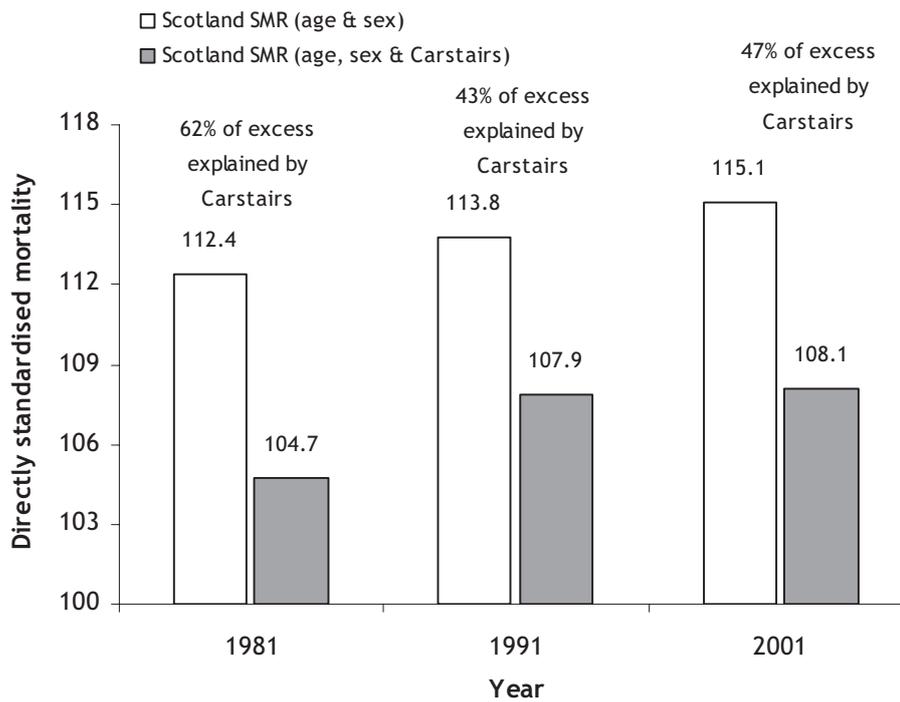
A large proportion of the higher rate of mortality in Scotland, the West of Scotland and Glasgow relative to the rest of the UK can be explained by higher deprivation (i.e. when an adjustment is made for markers of wealth and poverty, such as car ownership and overcrowding from census data, the differences in mortality between areas reduces). The links between deprivation and mortality are well researched and uncontroversial, although there remains uncertainty, as well as theoretical disagreement, about which mechanisms are most important in linking deprivation, poverty, low social class and mortality (i.e. how deprivation gets ‘under the skin’).¹⁰⁻¹²

However, while the excess mortality has increased since 1981, the proportion explicable by the use of the Carstairs index (a measure of area deprivation based on car ownership, overcrowding, male unemployment and social class derived from census data) has declined from around two thirds in 1981 to under half in 1991 and 2001 (Figure 5).¹³ ^a The reason(s) is (are) not clear, and this is indicated by the name which has been adopted for the phenomenon - the ‘Scottish Effect’.^b However it is recognised that the phenomenon is more profound in the West of Scotland in general and in the City of Glasgow in particular (the ‘Glasgow Effect’, Figure 6).^{14, 15} There is a much smaller ‘Scottish Effect’ for self-reported poor health. In this case the Scottish excess over England & Wales *can* largely be accounted for by deprivation.¹⁶

^a It is not possible to examine the extent to which the Carstairs index accounts for variation in mortality prior to 1981 as the census did not capture the same data prior to this date, nor were postcode sectors available in 1971.

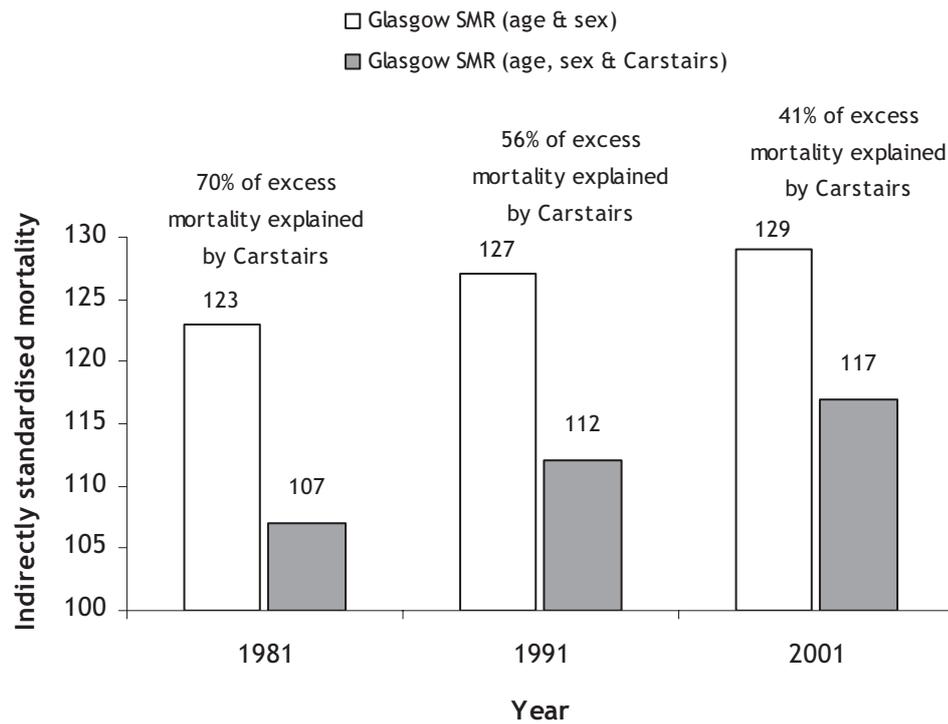
^b There is a slight difference between the excess mortality rates quoted in Hanlon 2005 and Carstairs 1989 due to a subsequent adjustment in the measurement of overcrowding in the census.

Figure 5 – Trend in the ability of the Carstairs index to explain variation in all age mortality between Scotland and England & Wales (Britain^c = 100; data from Hanlon 2005)¹⁵



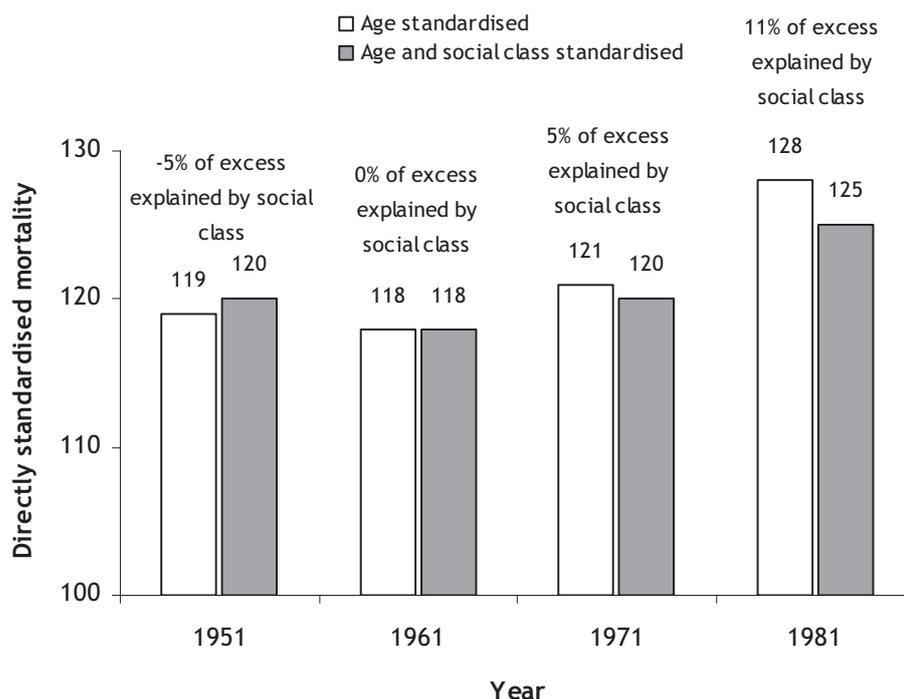
^c Britain comprises England, Wales and Scotland; the UK includes Britain and Northern Ireland.

Figure 6 – Trend in the ability of the Carstairs index to explain the variation in mortality between the City of Glasgow and UK (UK = 100; data from Reid 2008)¹⁷



Social class (a component of the area-based Carstairs index) has not always been able to explain the excess mortality in Scotland as compared with England & Wales. Figure 7 shows that Scottish mortality was higher than that in England & Wales from at least 1951, and that social class could only explain a very marginal proportion (11%) of the higher mortality in 1981. It actually demonstrated an inverse relationship at the beginning of the time series.

Figure 7 – Trends in excess Scottish male mortality compared with England & Wales (E&W = 100) standardised by age and by age and social class (data from Marang-van de Mheen 1998)¹⁸



Comparing Glasgow with the rest of Scotland

The divergence of mortality from 1980 onwards is also seen when Glasgow is compared to Scotland as a whole for all-cause mortality (Figure 8), deaths due to ischaemic heart disease (Figure 9), mortality due to cerebrovascular disease (stroke, Figure 10) and suicide (Figure 11). Furthermore, as the life expectancy of the most affluent fifth of Glaswegians improved at a similar rate to the rest of Scotland, it is clear that the most deprived populations in Glasgow drove this change (Figure 12).¹⁹ Similar patterns are also seen in females.

Figure 8 – Trend in male directly age-standardised death rates per 100,000 population for Scotland and Greater Glasgow (15-74 years, from Hanlon 2006)¹⁹

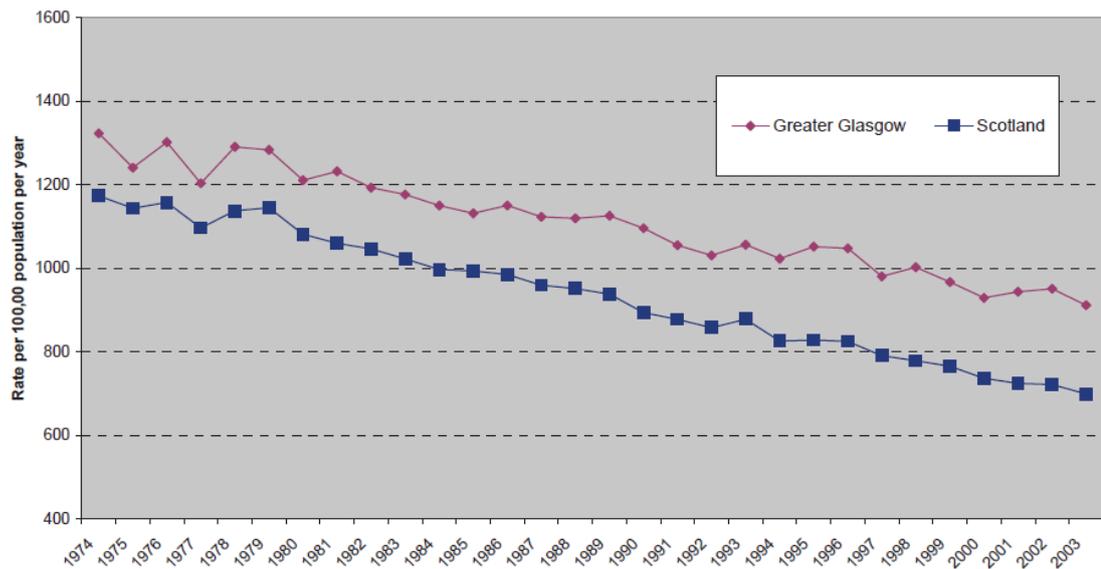


Figure 9 – Trend in male ischaemic heart disease death rates per 100,000 population for Scotland and Greater Glasgow (15-74 years, from Hanlon 2006)¹⁹

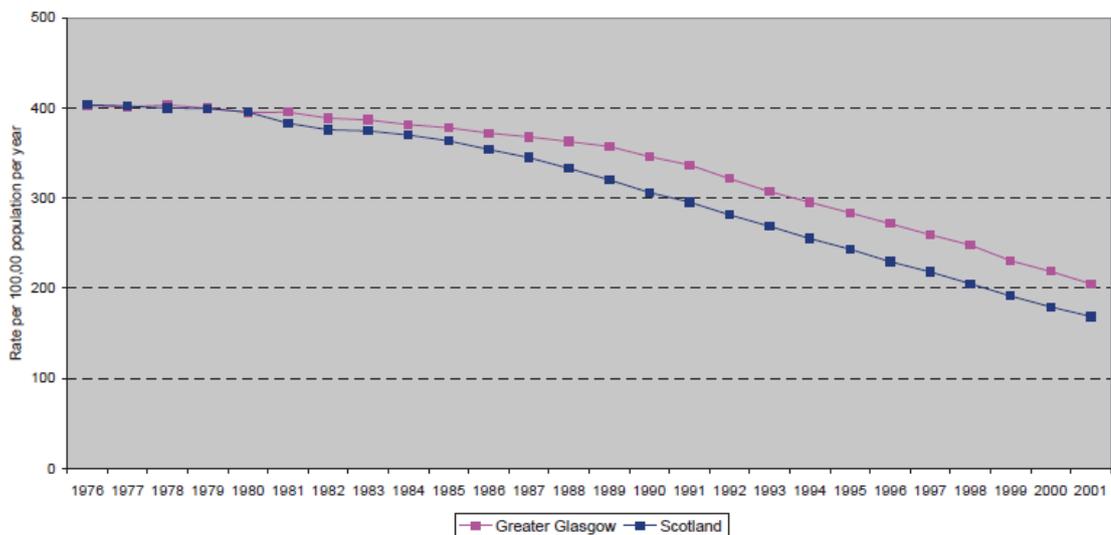


Figure 10 - Trend in male cerebrovascular disease death rates per 100,000 population for Scotland and Greater Glasgow (15-74 years, from Hanlon 2006)¹⁹

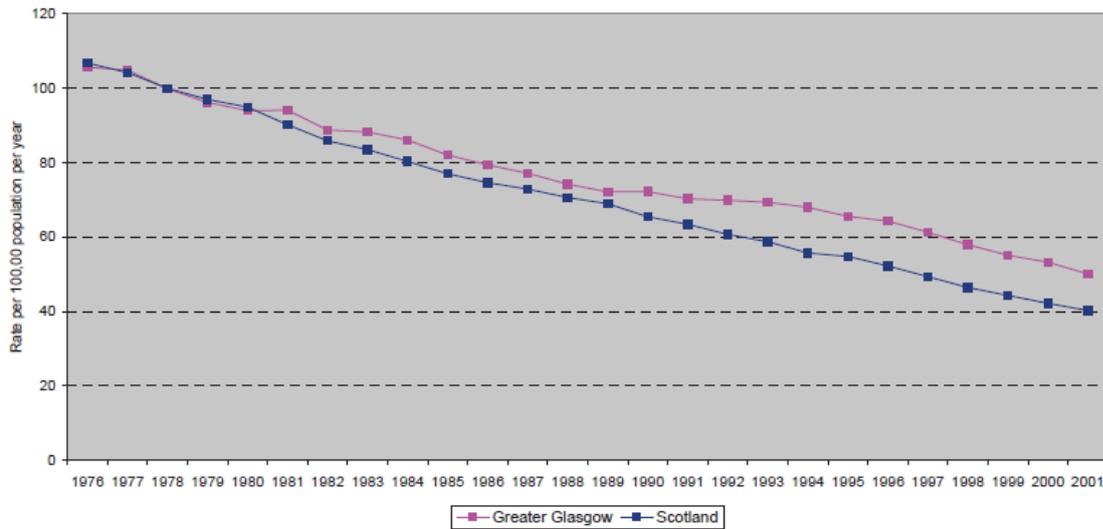


Figure 11 - Trend in male suicide death rate per 100,000 population for Scotland and Greater Glasgow (15-74 years, from Hanlon 2006)¹⁹

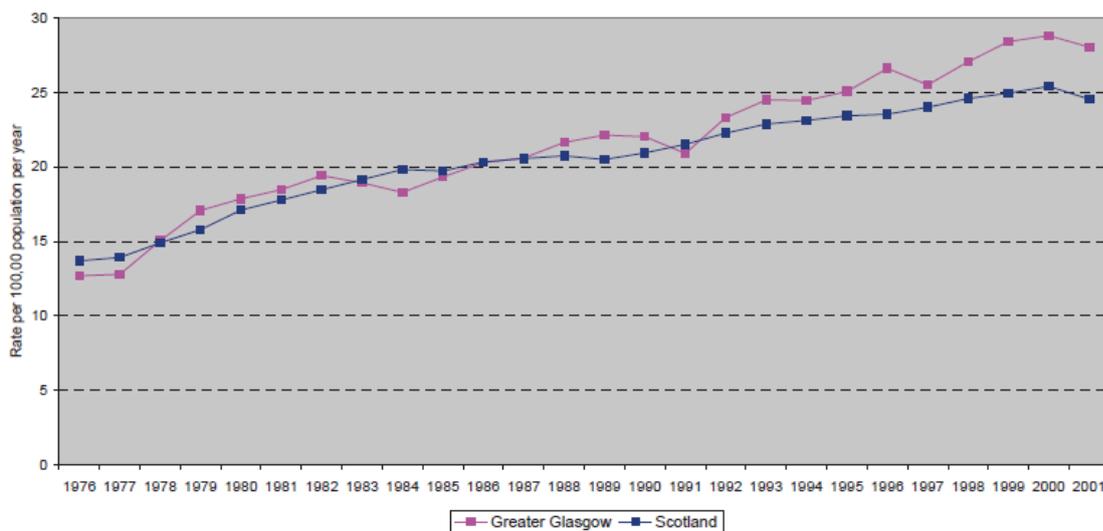
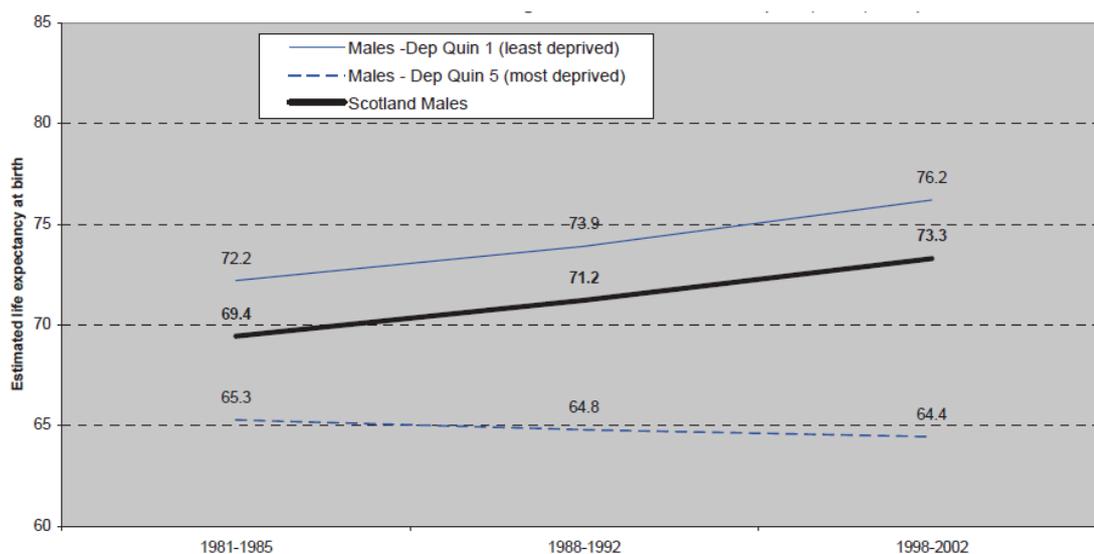


Figure 12 - Trend in male life expectancy in Scotland and for the most deprived population fifth and least deprived fifth of the population of Greater Glasgow (from Hanlon 2006)¹⁹



Summary

Scottish mortality was, until around 1950, comparable to that of its European neighbours. In the second half of the 20th Century Scottish (and Glaswegian) mortality improved more slowly than in other nations, leaving Scotland as an outlier with the highest mortality.

When compared to England & Wales, relative mortality increased between 1981 and 2001 (from 12% to 15% higher) while the proportion of this higher mortality which could be explained by higher deprivation declined. The higher mortality can be attributed to alcohol, drugs, suicide, violence, road-traffic accidents, as well as higher rates of cardiovascular disease, cancer and stroke. What is unclear is why these health outcomes, and the behavioural, cultural and social problems that underlie them, constellate in Scotland (and the West of Scotland and Glasgow in particular), and why they constellate to a greater extent than would be explicable by deprivation alone.

The purpose of this report is to: identify the various hypotheses that have been proposed to explain the higher mortality in Scotland, the West of Scotland and Glasgow, including the additional mortality unexplained by deprivation; evaluate how well each hypothesis explains the trends by reviewing the current evidence base in light of the Bradford-Hill criteria of causality; and suggest future research approaches that might help to clarify causes.

METHODS

Identification of hypotheses

Review of literature

A five-pronged approach was used in our search for evidence: (1) Embase (1980-April 2010) and Medline (1950-April 2010) electronic databases were searched using the search strategy outlined in the appendix (Table 2); (2) a search of key authors' websites for other publications was carried out; (3) the reference sections of key papers were checked; (4) the citations of these key papers were also checked; and (5) an internet (Google) search was undertaken based on the search terms 'Scottish Effect', 'Glasgow Effect', 'excess mortality Scotland', and 'excess mortality Glasgow', with the first 50 hits being scrutinised. Additional literature searches for those hypotheses where no evidence was uncovered in the formal literature review were also performed. These were targeted towards the specific hypothesis rather than the generality of the Scottish Effect or higher Scottish mortality, but were not systematic.

Discussion groups

The original authors of the work on the 'Scottish Effect' have been involved in numerous presentations of their work at conferences and seminars. These have been a fruitful source of hypotheses on the cause(s) of the Effect and the hypotheses noted during these presentations were included.

Induction and deduction in the creation of hypotheses

Research into the Scottish mortality phenomenon has been based on a mix of inductive and deductive approaches. Observation of the higher mortality and its patterning has clearly sparked a number of hypotheses and theories, providing impetus (and funding) for this area of work (in the inductive mode). Yet the phenomenon has also been approached from a deductive perspective, particularly where health outcomes are seen in their wider connection to changing social and economic circumstances. Both approaches are valid and can be evaluated using the Bradford-Hill criteria. However, it is possible that some hypotheses generated in the deductive mode may have been missed since they may not always refer to outcomes in the health terms deployed in the literature search.

Evaluation compared to Bradford-Hill's criteria

Two factors (e.g. X and Y) can appear to be associated with one another if: X causes Y; Y causes X; X and Y are caused by a third factor Z (confounding); X and Y are associated only by an artefact (erroneous association); or if the association is by chance. Bradford-Hill outlined in 1965 a series of 'criteria' which can be used to evaluate whether a particular association between two factors is of a causal nature.²⁰

These criteria are:

1. Strength of association

The stronger the association between an outcome and an exposure, the more likely it is that the relationship is causal. Thus, any exposure which was very common in an area of high mortality, but much less common in areas of lower mortality, would tend to be a clear candidate for consideration in attributing causality.

2. Consistency

If an outcome is consistently observed in association with the exposure by different research groups, in different populations, contexts and time periods, the more likely the exposure is to be the cause of the outcome. This criterion is more useful in confirming rather than rejecting a causal relationship, particularly with rare outcomes. It would mean that the putative cause of the higher mortality being discussed here would be associated with the same impact in other populations.

3. Specificity

If a specific (or narrowly defined) outcome and exposure are repeatedly observed to be associated, this again supports causality. In Scotland, this would mean that exposures that are observed in different contexts to cause the specific range of higher death rates (e.g. alcohol- and drug-related, suicide etc.) would be more likely to be a causal factor. Specificity allows a causal conclusion to be drawn quickly, but its absence is not as useful at disproving a putative hypothesis.

4. Temporality

Where the exposure precedes the outcome this makes causality more likely, and where the outcome precedes exposure it can help to disprove a causal hypothesis. It should be remembered that life course epidemiology has taught us that this exposure may have occurred several decades prior to the outcome being witnessed,²¹ and that a single outcome may have a range of 'causal' factors which may act with different lag times and through different mechanisms (some of which may be necessary but not sufficient).²²

5. Biological gradient

Where intensity of exposure is associated with a greater occurrence of the outcome, this supports causality. Thus, a putative causal factor for the Scottish mortality pattern would be supported if there was evidence of a similar pattern where the exposure was intense, and less of the mortality pattern where the exposure was minimal.

6. Plausibility

If the relationship between the exposure and outcome is biologically plausible, this helps to support causality, but there are numerous examples in history where it was only later that the biological knowledge caught up with the epidemiology to make the pathway plausible (e.g. as was the case with H.pylori infection and peptic ulceration).²³

7. Coherence

If a hypothetical causal exposure fits with the existing knowledge base and does not require a fundamental rethink of science, this again makes it easier to conclude a causal relationship. However, a non-coherent explanation does not preclude causation (for example, as was seen in the development of physics during the time of the Enlightenment) but would require much more work before causality could be established.

8. Experiment

If experimental, or quasi-experimental evidence can be gathered which evaluates whether a change in exposure is associated with a change in the outcome, this provides very strong evidence of causality. In the case of the Scottish mortality phenomena laboratory-style experimentation is impossible but there may be natural experiments which allow this observation to be made.

9. Analogy

Where a similar exposure has caused a similar outcome, this increases the acceptability of potential hypotheses. To use the example above, putative infective causes of gastrointestinal diseases (such as peptic ulceration) are much more likely to be accepted now that a causal link between H.pylori and peptic ulceration has been established.

None of these 'criteria' individually 'proves' or 'disproves' causation, but each can have a role in supporting or casting doubt on a particular hypothesis. These criteria are different from statistical tests of significance, which seek to expose the likelihood of any association being due to chance, and do not negate the need for caution against bias and confounding in any observed association.²⁰

More recently, a report by the Academy of Medical Sciences identified three of these criteria as being most useful when considering the 'upstream' causes of public health problems: strength of association; a temporal relation; and plausibility.²⁴ Others have supported specificity as being the most important criterion.²⁵

Ecological and atomistic fallacies

The well-known ecological fallacy is when an inference is drawn at the individual level based on group level data. For example, the richer a country the higher the prevalence of obesity, yet within countries obesity is usually associated with the poorest individuals. The related, but less well known, fallacy relevant to this study is the atomistic fallacy.²⁶ This is when group level conclusions are drawn on the basis of individual data. The Scottish mortality pattern and the Scottish Effect are group level phenomena which require studies based on populations rather than individuals. This is particularly important to remember with the cohort study data arising from Scotland which, by definition, will be unable to detect group level exposures which a high proportion of the Scottish population might be exposed to, and yet which might explain a difference in mortality compared to other countries. Despite this limitation, studies using individual data remain useful in ascertaining the plausibility of the linking mechanisms between exposure and outcome. The potential for these fallacies to interfere with the evaluation of the hypotheses is recognised and made explicit where relevant.

Synthesis

Placement on a theory of change

Hypothetical causes of the higher mortality rates in Scotland (and the West of Scotland and Glasgow), and the additional mortality unexplained by deprivation, can be classified into 'upstream' or 'fundamental' causes and 'downstream' causes.²⁷⁻²⁹ This classification fits with the theoretical work by Lynch,³⁰ Whitehead,³¹ and Evans & Stoddart³² which links the 'upstream' and 'downstream' causes of health and illness. If a pathway was to be described for the development of typhus epidemics in London in the 1800s, it would include a range of 'upstream' and 'downstream' causes all of which contribute towards an overall explanation of the mechanisms leading to death and help to highlight possible opportunities for intervention (Figure 13).³³ However, where there is an exposure causing a range of negative outcomes, hypotheses at the more 'upstream' level are more likely to be required in providing an explanation for the entire phenomenon (Figure 14).

Figure 13 – A simplified representation of some of the 'upstream' and 'downstream' causes of mortality from typhus in 19th Century London

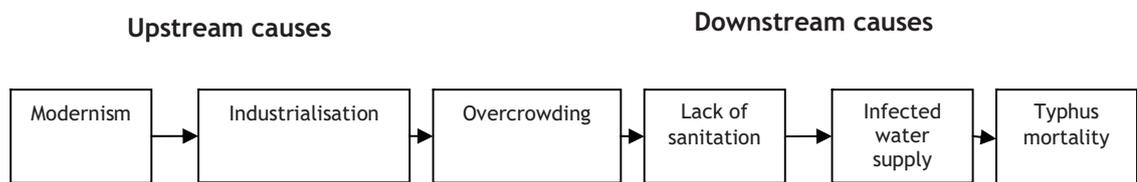
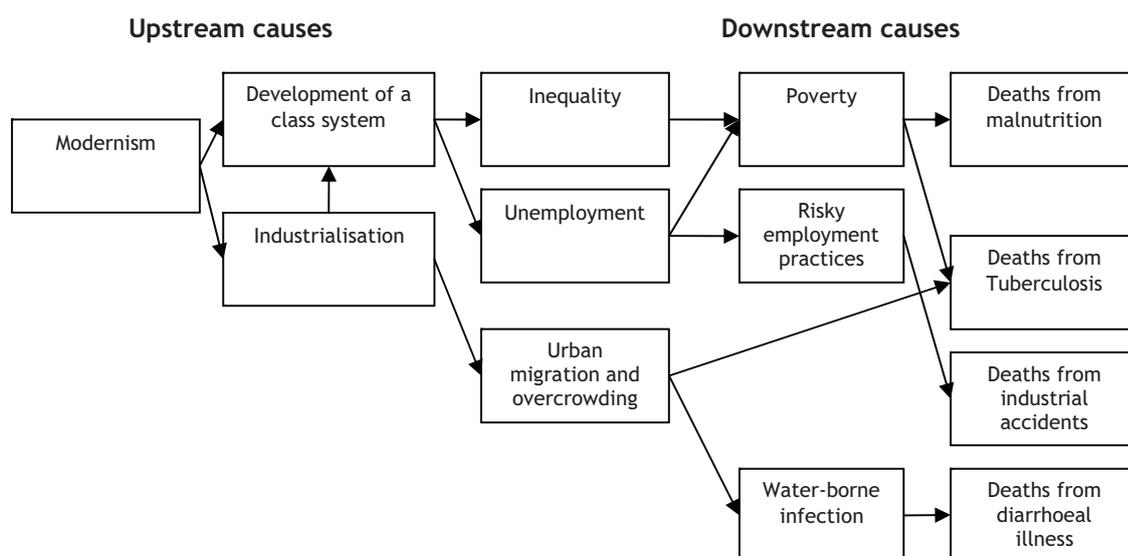


Figure 14 – A simplified representation of how ‘upstream’ causes are more likely to generate a range of diverse mortality outcomes than a ‘downstream’ cause (using 19th Century London as a context)



Figures 13 and 14 represent theoretical links between ‘upstream’ and ‘downstream’ causes of mortality as if the mechanisms are linear. In reality, the links are likely to be more complex and subject to the defining features of complex adaptive systems (emergent properties, path dependency etc.).³⁴ Furthermore, some explanations for a mortality pattern might be necessary but not sufficient, or might catalyse other important pathways.

A further complication is that many of the hypotheses may in fact reflect differing emphases of the same phenomenon, or represent different parts of a single causal pathway. Others may represent only part of a causal chain (e.g. ‘Scots have a different relationship with alcohol than other populations’, without any suggestion as to why this difference has occurred).

In light of these kinds of considerations, this report will seek to identify where hypotheses might fit together and where progress towards a comprehensive hypothetical chain from the ‘causes of the causes’ to the mortality outcomes might be possible.²⁷ Along these lines it might be possible to elaborate a theory linking the most ‘upstream’ determinants of health at the social, political and environmental level through a range of relevant mechanisms to explain both the lagging health outcomes of Scotland compared to the rest of Europe, and the unexplained mortality after deprivation has been accounted for in relation to England and Wales. Such a theory would be most useful in providing a comprehensive understanding that could lead to policy solutions.

Dialectical synthesis

A useful way of conveying what is here being attempted might be to invoke the idea of a dialectical progression from more partial understanding of a problem towards a more developed and holistic understanding of its totality. Such an approach would draw in some way on the dialectical philosophy of G.W.F. Hegel, for whom understanding proceeds through a critical process of comparison, evaluation and progression.^{35, 36} In this process ‘lower’ forms of knowledge are first negated by rivals of comparable level, and then all are transcended with the emergence of a higher form which retains the limited ‘truths’ of its antecedents – but in a new context which aspires towards a broader and deeper grasp upon the totality of the problem. The attraction of such a perspective and approach in addressing the multiple hypotheses for the Scottish mortality phenomenon is that it focuses us not just on the competition between rival contenders, but on synthesising the most valid insights of the competing hypotheses in aspiring towards a higher level of understanding.^{35, 36}

RESULTS

Results of searches and discussion group

The electronic database search identified 309 potentially relevant papers. From these 76 were selected for detailed examination on the basis of the relevance of the title and abstract.

The notes from the various discussions around dissemination of previous research identified a further set of hypotheses. There was considerable overlap between the hypotheses extracted from the literature and from the notes, although there were ideas generated from the latter that were not reflected in the academic literature.

Identified hypotheses

Seventeen hypotheses were identified which addressed either: the higher mortality in Scotland as compared to Western Europe; the higher mortality in Glasgow (or the West of Scotland) compared to the rest of Scotland; the excess mortality in Scotland after accounting for deprivation compared to England (Scottish Effect); or the excess mortality in Glasgow (or the West of Scotland) compared to the rest of Scotland (or to comparable English cities – Liverpool and Manchester) after accounting for deprivation (Glasgow Effect). Two of these hypotheses (deprivation and migration) suggest that artefact may explain some of the phenomena, whilst the other 15 suggest potentially causal mechanisms that might contribute to the pattern, or account for the pattern entirely. As indicated previously, some of the hypotheses are clearly closely related, and others can also be linked.

The hypotheses are dealt with below in the following order: artefactual explanations, 'downstream' explanations, 'midstream' explanations, and 'upstream' explanations. The evidence identified through the literature review, which includes evidence for each of the mortality phenomena and comparisons listed above, is then summarised and appraised using the Bradford-Hill criteria. For some of the hypotheses there has been little or no research. This does not preclude the possibility that they will prove in future to be relevant, and a programme of research which will consider a number of them is due to begin soon. Table 1 then summarises how, in light of the currently available evidence, each of the hypotheses fits the criteria for each of the time periods under consideration. This is used as a first stage in working towards the synthesis presented in the later part of the report.

1. Deprivation

Description of hypothesis

For the purpose of this discussion, deprivation is defined as absolute or relative poverty (the broader cultural aspects of inequalities are covered under hypothesis 14 – ‘greater inequalities’).

The deprivation hypothesis suggests that the ‘Scottish Effect’ would actually be explained by deprivation, but that the measures have, since 1981, become increasingly unsuitable in capturing the changing nature of the deprivation experienced by the population. Four aspects of the measurement of deprivation which could explain the apparent (but artefactual) Scottish Effect have been highlighted:

1. The Carstairs index has become outdated.
2. The proxy measures of deprivation available do not adequately capture how deprivation is experienced.
3. Issues around the size, and consistency of size, of the small areas at which historical measures of deprivation (Carstairs) were calculated.
4. The population denominator for Scotland is underestimated.

The application of this hypothesis to the divergent mortality pattern of Scotland (compared to its European neighbours from 1950 onwards) is that greater deprivation, compared to the rest of Europe, can explain the phenomenon.

Evidence generally relevant to the hypothesis

The geographical areas with the greatest deprivation in 1981 are the areas with the greatest unexplained excess mortality in 2001.^{8,19} This is consistent with deprivation being the most important explanation for the higher mortality.

Comparing Glasgow with the rest of Scotland, Gray was able to explain the higher female mortality using deprivation. However, the same author was unable to explain either all causes of male mortality (including cancer, liver disease and drug-related deaths) or some self-reported measures of health (particularly self-reported mental health in women) in the same way.³⁷ This suggests that the Glasgow Effect is partially explained by Carstairs deprivation. Using more up-to-date and detailed deprivation markers used in the Scottish Health Survey, almost all of the excess for various measures of self-reported poor health (in 2008/9) in Glasgow relative to the rest of Scotland could be explained.³⁸

Evidence relating to Scottish Effect – 1. Carstairs Index has become outdated

The identification of the ‘Scottish Effect’ was based on the decreasing ability of the Carstairs index to account for variations in mortality between areas after 1981.¹⁵ The Carstairs index is a pragmatic measure which utilises data available from the Census: car ownership, social class, employment and overcrowding.

There are two distinct reasons why this is a plausible hypothesis. First, overcrowding and car ownership have become less important markers of relative poverty as housing conditions have improved and as the cost of driving has declined (i.e. the distribution of areas for these measures has become much narrower over time making it less likely that the measure is able to distinguish well between areas – Figures 15 & 16).

Figure 15 – The changing ability of the overcrowding element of the Carstairs index to discriminate between areas over time (from Reid 2008)¹⁷

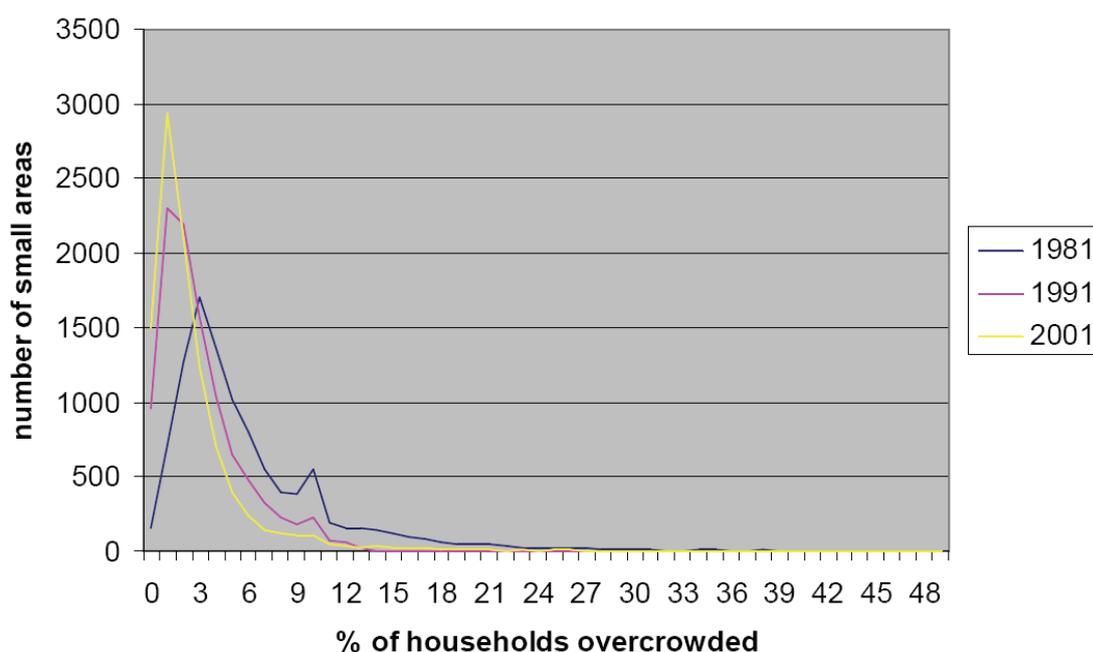
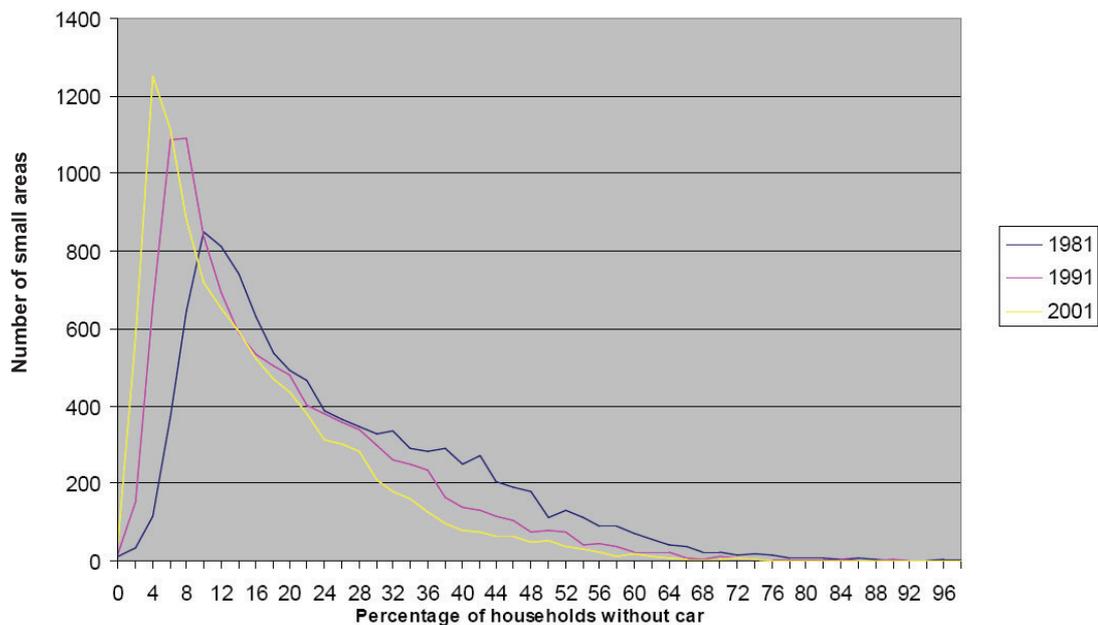


Figure 16 - The changing ability of the car ownership element of the Carstairs index to discriminate between areas over time (from Reid 2008)¹⁷



Thus, the Carstairs index may no longer be able to capture poverty in the same way as it did in the past. By way of analogy, if a deprivation measure included whether or not a household had an outside toilet, this would have been a good discriminator of poverty in the early 20th Century. But so few houses were without an inside toilet by the end of the 20th Century that this measure would have become outdated. It is therefore plausible that deprivation measures require to be updated periodically. Indeed it should be expected that this would be required.

Reid's work demonstrates how the very negatively skewed deprivation profile for Scotland in 1981 (Figure 17) seemed almost entirely resolved by 2001 when using the Carstairs index (Figure 18). Either deprivation in Scotland improved markedly in comparison to England during this 20 year period, or the measure itself became less good as an indicator of what was happening.¹⁷

Figure 17 - The distribution of the Scottish and English populations by deprivation decile using the Carstairs index (1981; from Reid 2008)¹⁷

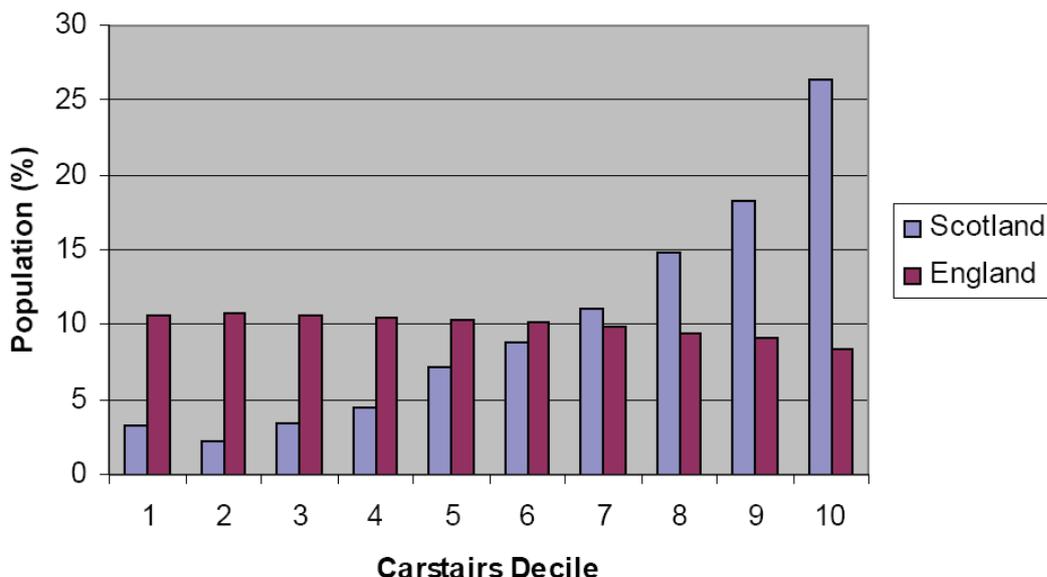
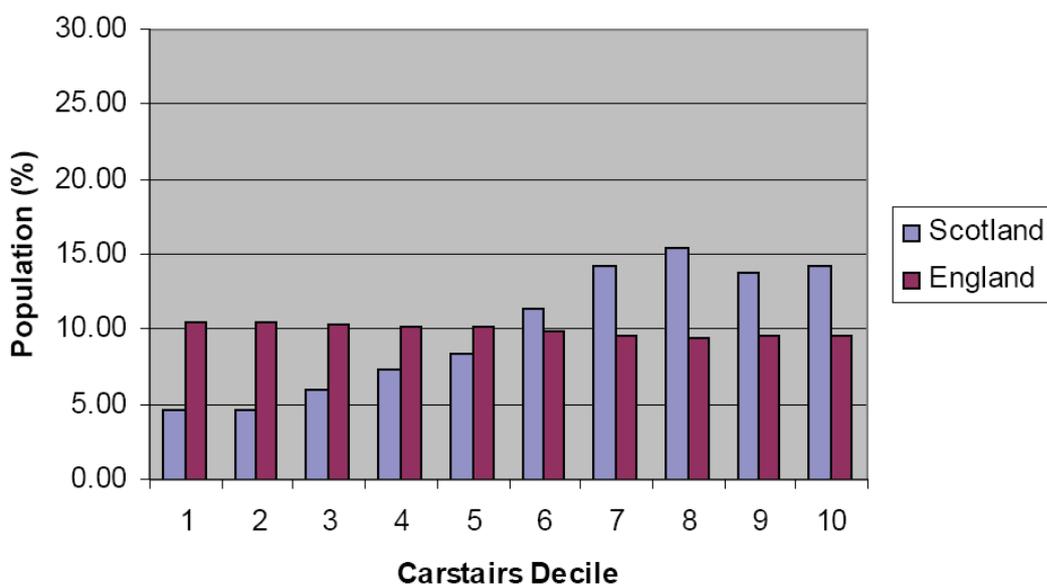


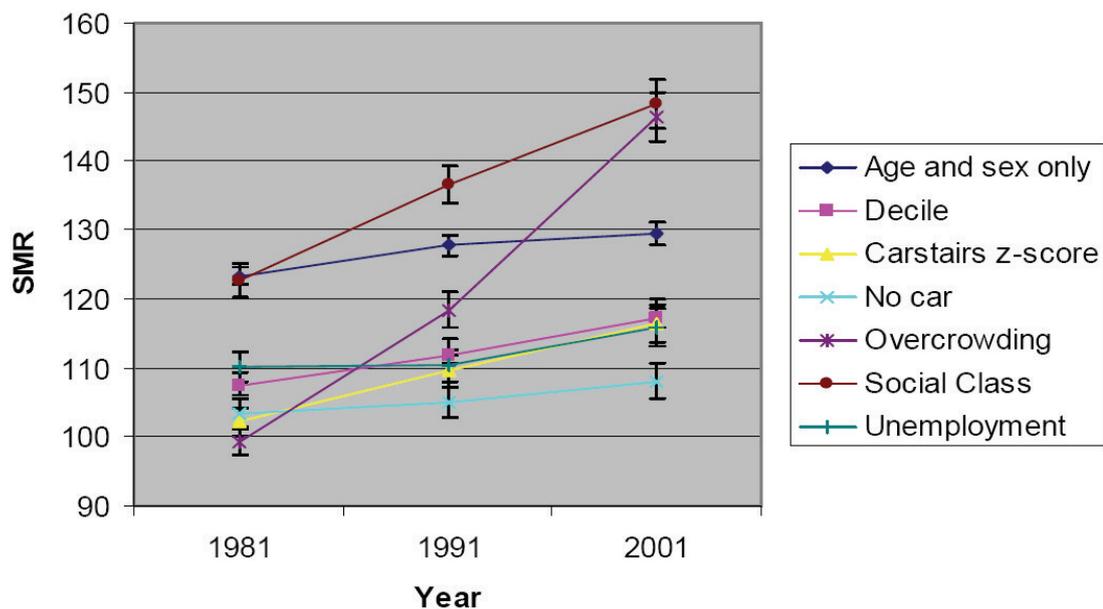
Figure 18 - The distribution of the Scottish and English populations by deprivation decile using the Carstairs index (2001; from Reid 2008)¹⁷



Reid also performed an experiment to explore whether a different measure of deprivation might explain a greater proportion of Glasgow's excess mortality than was possible using the Carstairs index. This experiment was based on the observation that while overcrowding no longer discriminates well between areas (Figure 15), there remains some greater spread in terms of car ownership (Figure 16). This work showed that the use of Carstairs z-scores and car

ownership explained a much greater proportion of the excess mortality (72% of the variation in 2001 was explained using this method, calculated using Reid's data of an age-adjusted SMR of 129 and an age- and 'alternative' deprivation index- adjusted SMR of 108; Figure 19).¹⁷ In contrast, by using social class and overcrowding it was only possible to explain a much smaller proportion of the excess mortality (particularly in 1991 and 2001). However, Reid's work is vulnerable to the use of different sized areas in its comparison of Glasgow with English cities.

Figure 19 - The changing ability of different sub-components of the Carstairs index to explain the excess mortality in Glasgow compared to other UK cities over time (from Reid 2008)¹⁷



Evidence relating to Scottish Effect – 2. Deprivation is not adequately captured

Another potential explanation is that, separately from whether Carstairs has become outdated, deprivation is not adequately captured by the index.³⁹ All measures of deprivation are proxy measures, and as such all are limited in their ability to capture the experience of deprivation through time. Various measures have been designed using available data from the Census and other surveys, many of which are aggregated to the area level.⁴⁰ It is therefore plausible and coherent that deprivation will never be completely captured by any measure and so there will always be at least an element of this which remains outstanding.

Evidence relating to Scottish Effect – 3. Size and consistency of areas

It is also possible that the difference in Carstairs-adjusted mortality between Scotland and England & Wales reflects the different sizes of geographical area used. In Scotland, postcode sectors were used which had a mean population of around 5,500 in 2001. England & Wales had their Carstairs indices calculated on the basis of local authority electoral wards. Although the mean population sizes of the areas was very similar between Scotland and England & Wales, there was marked differences in the population size of the different geographies in the cities (e.g. the areas in Manchester and Liverpool in 2001 had mean populations of 11,900 and 13,300 respectively).⁴¹ It is therefore possible that the Carstairs measure in Scotland is more sensitive

to identifying small areas of concentrated deprivation and that the distribution of deprivation using the Carstairs measure, even in equally deprived populations, might be different (with a flattened distribution expected in England & Wales). It is therefore plausible that this might be partially responsible for the apparent Scottish Effect.

To investigate whether this is the case for Glasgow, Walsh compiled areas of similar size for Glasgow, Manchester and Liverpool using 'Lower Super Output Areas' in Liverpool and Manchester, and by combining 'datazones' in Glasgow to create geographies of equal size. Then, an income deprivation measure (which was very highly correlated with the official Scottish and English indices of multiple deprivation) was created to facilitate comparison of the three cities in terms of income deprivation and mortality. The deprivation profiles of the three cities were found to be virtually identical but Glasgow had higher mortality for all deprivation deciles of its population.⁹ This therefore supports the existence of an excess mortality in addition to that explained by deprivation, even after matching the population size for the small areas and using a different measure of deprivation (income deprivation). This provides experimental evidence against this artefactual explanation and against deprivation explaining the Glasgow Effect.

Evidence relating to Scottish Effect – 4. Population denominator

Another potential source of artefact in measuring the Scottish Effect is that there is significant uncertainty about the population denominator in Scotland and in Glasgow from 1991 onwards. The 'poll tax' in 1990 was blamed for the large drop in the male population in the 1991 Census in Glasgow.³⁶ On this basis, the population in the 1991 census was adjusted upwards to account for the suspected 'hidden population'; but this population remained absent at the time of the 2001 Census. It is therefore possible that some of the 'excess' mortality is actually due to inaccuracy in the population denominator, in particular that a larger proportion of the population in Scotland (and the West of Scotland and Glasgow in particular) is not captured by the Census. No further research was identified relating to this sub-hypothesis.

Evidence relating to the divergence of mortality from the rest of Europe

Scotland and the West of Scotland are less materially deprived than comparator nations and regions in Europe, yet, as we have seen, have both higher mortality and divergent mortality trends in relation to their comparators. However, it is unclear whether material deprivation at the time of divergence (particularly 1940-1980) was different. There is therefore uncertainty about whether there is an association, temporal relationship or biological gradient to explain the higher mortality in Scotland.^{3, 4, 42}

Furthermore, there is evidence amongst rich nations such as the UK that absolute income is not associated with mortality, which would make the hypothesis inconsistent and incoherent with existing knowledge.^{43, 44} This suggests that absolute poverty or deprivation is not responsible for the overall trend. Relative poverty and inequality are covered in other hypotheses later. However, the hypothesis is plausible (since material resources are known to be important in determining health within countries), and has many analogies and natural experiments in history where rapidly developing countries whose mean wealth increased improved their mortality whilst those without development did not. There is evidence to suggest that there is a specific

deprivation pattern in Scotland and other European areas for a large range of outcomes such as cardiovascular disease, violence, suicide etc.; but a more useful comparison might be that of incident breast cancer since it is more specific (using its inverse association with deprivation).

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Is the Scottish Effect due to the difference in size of the comparison populations between Scotland and England & Wales?	Use a method similar to that used by Walsh in relation to Glasgow, but for Scotland as a whole. ⁴¹
Is there a better measure of deprivation than Carstairs or income deprivation which can explain a greater proportion of the variation in mortality between Scotland (and the West of Scotland and Glasgow) and other populations (such as car ownership)?	Use a method similar to that of Reid, except using similar population sizes. ¹⁷
Does variation in the deprivation profile of cities explain the variation in mortality outcomes between Glasgow and other cities?	Additional quantitative analysis of Glasgow, Liverpool and Manchester could be undertaken using census data and the geographical areas developed by Walsh. ⁴¹ Comparisons of nations is more complicated as there may not be common measures.
Does the census population accurately represent the actual population in Scotland and Glasgow, and are there systematic differences with other populations?	It might be possible to compare the census population with other measures, such as the community health index (CHI), to evaluate whether there is a divergence in the denominator in Scotland as distinct from other areas.
Can deprivation explain the variation in Scottish mortality compared to other nations?	This would entail an extension to the Aftershock report to include nations rather than deindustrialised regions. ³
Is there a North-West England Effect, Manchester Effect or Liverpool Effect?	This would replicate the methodology in Hanlon 2005 ¹⁵ , but would focus on finding other areas with unexplained excess mortality?
Does deprivation produce a specific mortality pattern which is witnessed in Scotland and Glasgow?	It would be possible to identify a series of cause-specific mortalities which are either positively or negatively associated with deprivation, and then observe whether this patterning is seen in Scotland and Glasgow.

2. Migration

Description of hypothesis

The most frequently proposed version of this hypothesis is that Scotland and Glasgow suffered from a greater degree of emigration of healthy individuals than other areas, leaving behind a more unhealthy population which would be more likely to suffer from higher mortality. This is in effect the well-known 'healthy migrant effect', except that the population of interest is that which is left behind.⁴⁵

There are two alternative versions of the migration hypothesis: that there are historical differences in migration patterns in Scotland (either in terms of immigration from countries such as Ireland or in terms of emigration during the 18th, 19th and early 20th Centuries to the United States, Canada, Australia, New Zealand etc.);⁴⁶ or that the Scottish and Glasgow population is more static and homogenous than is found in other areas, and that this has had a detrimental effect on the culture and aspiration of the City and had consequent negative health impacts.

Evidence

Some evidence suggests that the construction of the New Towns (principally East Kilbride and Cumbernauld, but also Glenrothes, Irvine and Livingston) and 'overspill' housing developments (such as in Greenock, Paisley, Dumbarton and Hamilton) around Glasgow during the 1960s created a movement of a large proportion of the Glasgow population outside the City boundary, and that those most likely to move were the better off (or at least less badly off) groups.⁴⁷ It has been suggested that the opposite may have been the case in some other cities such as Liverpool, where gentrification took place and there was a movement of the poorest populations to outside the City boundary. It is also known that there was a large movement of populations in and out of Glasgow and Scotland over several centuries (both to and from other countries and within Scotland).⁴⁶

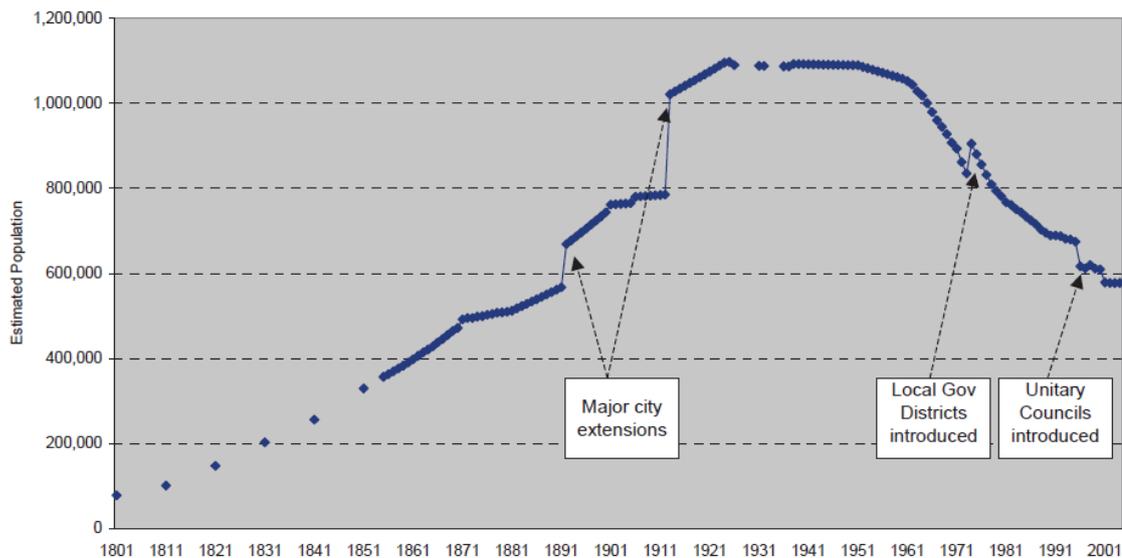
Popham found that English & Welsh born immigrants to Scotland live longer than native Scots, and that those born in Scotland who move to England & Wales do not live as long as those who were born and continue to live in England & Wales, despite emigrants reflecting the Carstairs deprivation distribution of the recipient nation.⁴⁸ This was confirmed by a similar study which examined the death rates of different immigrant groups in England & Wales which showed Scottish and Irish immigrants had higher death rates (32% higher for Scottish men and 36% higher for Scottish women).⁴⁹ This casts doubt on a healthy migrant effect being responsible for the higher mortality in Scotland.

Furthermore, people born in Scotland who die in England and Wales have higher rates of alcohol related mortality compared to native English and Welsh populations (SMR = 187), suggesting that one of the rising causes of premature mortality in Scotland is not unique to current Scottish residents.⁵⁰ A similar pattern was evident for cardiovascular mortality for migrant Scots.⁵¹

Popham et al found that migration within Scotland was not responsible for the increasing gap in mortality between Glasgow and the rest of the nation.⁵² Exeter showed that the higher mortality associated with Scottish health board areas with a declining population (principally those in the West of Scotland) was completely explained by markers of deprivation.^{53, 54} Thus the evidence regarding migration and Glasgow is consistent with the evidence for Scotland.

Glasgow's population did decline markedly from the 1960s onwards which does suggest an appropriate temporal relationship with a rise in excess mortality in the following decades (Figure 20).¹⁹

Figure 20 – Trends in the population of Glasgow (1801-2004 data sourced from the reports of the Medical Officer of Health for Glasgow and Registrar General; from Hanlon 2006)¹⁹



Evidence from across Britain suggests that selective migration might be more important in explaining local concentrations of higher mortality rather than regional or national differences, casting doubt on the potential for a migration hypothesis to explain the higher mortality in Scotland or the West of Scotland as compared to England & Wales or other regions.^{55, 56}

In summary, the healthy migrant hypothesis is plausible, and coherent with much of the existing knowledge base around healthy migrants with many analogies, but there is no association between migrating Scots and better health and the experimental models do not suggest the effect is an artefact due to migration (in contrast to that found in other migrant populations who adopt the mortality patterns of their adopted countries relatively quickly).⁵⁷ There is a lack of evidence regarding specificity (for example that a migration effect has resulted in a similar pattern of health outcomes in other areas) or a biological gradient.

No evidence was gathered relating to the prospect of the mortality phenomenon being explained by migration prior to the 1950s, and so little can be said about whether it was likely to have had an effect on the mortality divergence in Scotland from the 1950s. However, it seems implausible that it could be responsible for the mortality picture from the 1980s onwards.

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Was the patterning of migration in Glasgow similar to other cities and does this explain differences in mortality (especially with Liverpool or Manchester)?	This would require work describing the migration patterns for each of the cities (in terms of timing, which groups moved) and the surrounding context (regeneration patterns, push-pull factors etc.).
Does selective migration explain the differences in mortality between Scotland and England & Wales?	Could the method adopted by Popham for migration within Scotland using the ONS longitudinal study be adapted to include migrants from outwith the country, or at least to examine whether less deprived individuals were more likely to emigrate?
Did the migration patterns in Glasgow and Scotland from 1750-1950 influence the mortality patterns in Glasgow and Scotland after 1950 in a way which might be relevant to the problem?	Historical records of migration to and from Scotland and Glasgow would be compared with those of other areas, and the potential relevance of the results explored.

3. Genetic differences

Description of hypothesis

The genetic hypothesis suggests that the Scottish (and particularly the West of Scotland and Glasgow) population is either predisposed to negative health behaviours, or is particularly vulnerable to their effects, as a result of differences in the genetic mix of the population.

Evidence

There are analogies for this hypothesis: such as the different tolerance of alcohol in different racial groups.⁵⁸

Adjusting for IQ was found to account for a proportion of the variation in health outcomes (including mortality) within the West of Scotland after adjusting for deprivation.⁵⁹ Exposures in early life (including education and family environment), residual confounding (perhaps relating to deprivation) or genetic difference could account for such findings, making a genetic predisposition possible, but plausibility would require an ability to account for the changing nature of the Scottish mortality relative to other nations. Furthermore there is an absence of evidence comparing the IQ or genotype of Scots (and Glaswegians) with other populations, and consequently no evidence to assess whether there is strength of association.

The migration studies detailed in the previous section which suggest that Glaswegian migrants to other parts of Scotland and Scottish migrants to other parts of the UK retain a higher risk of mortality, do provide some (natural) experimental evidence that either genetics, early environmental exposures or retained cultures play a consistent role.^{48, 52, 53}

The wide range of cause-specific mortality categories which are in excess in Scotland and Glasgow (including alcohol-related, drug-related, cardiovascular, stroke, suicide etc.) makes a genetic cause unlikely. Genetic causes are more likely to explain specific causes of death rather than a wide range of health outcomes (the cause(s) of which are more likely to be 'upstream' or social) and this hypothesis is therefore incoherent with current knowledge.

There is no evidence available regarding the strength of association or biological gradient of the outcomes.

The ill-fitting nature of the trends in Glasgow and Scotland compared to the much slower intergenerational effects of genetic change suggest that the temporal relationship between a genetic 'exposure' and the mortality pattern 'outcome' could not explain most of the variation in mortality over this time, and at best could only explain a small proportion of the longstanding excess mortality. However, it is possible that genetics confer a vulnerability to a change in the environment which reveals itself only in retrospect. For this to be important, Scotland would have to have a distinctive common genotype, something which seems unlikely given the history of migration to the country.

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Is the higher mortality in Scotland and Glasgow explained by differences in genotype between populations?	A research programme to investigate whether genetics plays an important role in the higher mortality of Scots and Glaswegians would require longitudinal work from birth to eliminate the role of environmental exposures on outcomes.

4. Health behaviours

Description of hypothesis

The health behaviours hypothesis asserts that a large proportion of the higher mortality in Scotland and Glasgow can be attributed to alcohol-, smoking- and drug-related deaths because of a higher prevalence of these negative health behaviours. The hypothesis can also extend to worse dietary habits (in terms of the intake of fat, salt and sugar, and lower intakes of fruit and vegetables) and lower physical activity.

Evidence – illicit drugs

Bloor et al found that illicit drug-related deaths (including deaths due to blood-borne viruses, violent deaths in drug-users, suicides in drug-users etc.), using a modelled cohort based on the DORIS study, account for a third of the excess mortality in Scotland after deprivation is taken into account. This modelled difference is not due to a differing mortality experience of drug users between countries (the Scottish mortality rate was applied to the English population) but to the higher prevalence of problem drug use (2.7% (95% CI 2.1-4.2%) in Scotland versus 1.3% in England, amounting to an association between drug use and the higher mortality in Scotland). Furthermore, Bloor et al note that,

“a rapid increase in problem drug use (and particularly heroin use) occurred in the 1980s in Scotland, at the very point at which deprivation measures (Carstairs deprivation scores) began to account for less than half of the cross-national variance in rates” (Figure 21).⁶⁰

If true, this provides some (natural) experimental and appropriately temporal evidence in favour of higher drug misuse being a causal factor. More contemporary evidence suggests that illicit drug use in Scotland is higher than in other parts of the UK and than is found in most other Western European nations.⁶¹

Figure 21 – Crude number of drug-related deaths in Greater Glasgow (1995-2004, from Hanlon 2006)¹⁹



Evidence – alcohol

Gray found that alcohol consumption in Glasgow was higher than in the rest of Scotland even after adjustment for deprivation³⁷ (although the self-reported alcohol consumption may underestimate real consumption levels).⁶² There is also a clear temporal relationship between the appearance of the Scottish effect (Figure 22) and alcohol-related deaths, and between the divergent Scottish mortality trend and alcohol-related deaths (Figure 23). There is also evidence of a biological gradient in that Glasgow's liver cirrhosis rates are worse than Scotland's (Figure 23).¹⁹ However, comparison of reported binge-drinking in Greater Glasgow compared with Liverpool and Manchester shows a lower rate and therefore suggests that exposure to self-reported binge drinking is unlikely to explain the higher mortality (although self-reported alcohol consumption is likely to be underestimated and this underestimation may itself be socially patterned).⁴¹

Figure 22 – Crude number of alcohol-related deaths in Greater Glasgow (where alcohol is a primary or secondary cause, from Hanlon 2006)¹⁹

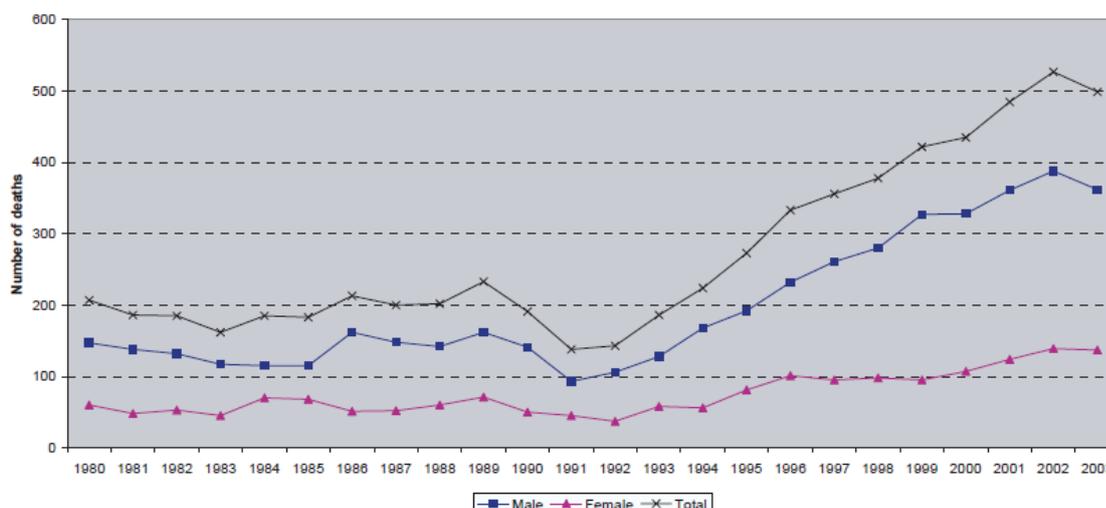
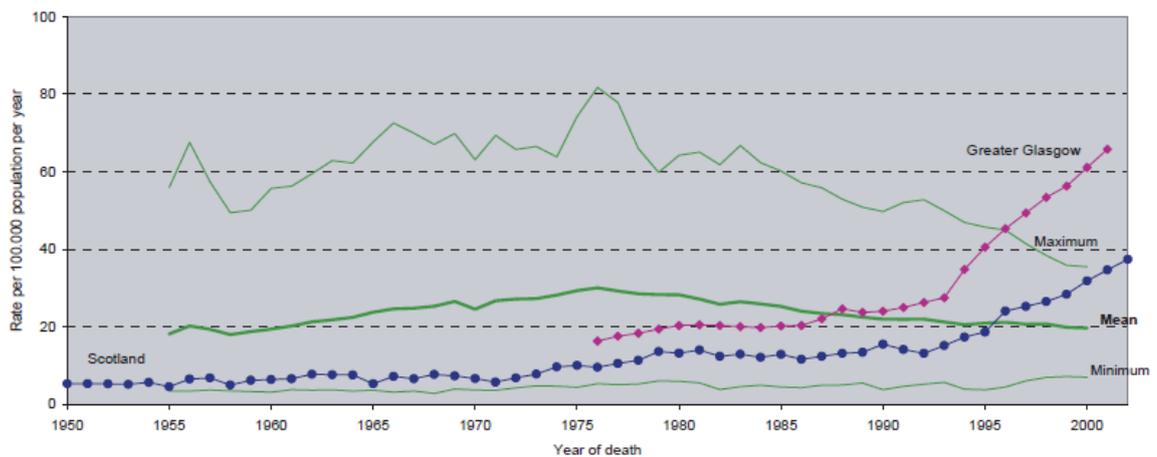


Figure 23 – Male age-standardised liver cirrhosis deaths in Greater Glasgow and Scotland in the context of the maximum, minimum and mean cirrhosis death rates for 16 Western European countries (from Hanlon 2006)¹⁹



Evidence – smoking

Gray showed that smoking prevalence in Greater Glasgow was 30% higher for men and 43% higher for women than in the rest of Scotland, but that this was entirely explained by greater deprivation. This study therefore supports the hypothesis that smoking can explain the higher mortality in Glasgow compared with the rest of Scotland, but questions whether there is an effect unexplained by deprivation (although there was no comparison with England & Wales to evaluate the Scottish Effect).⁶³ This is supported by individual data from the Renfrew/Paisley study.⁶⁴ The report on this cohort after 28 years shows that much of the all-cause mortality variation within the cohort can be explained by smoking, but that there is an additional negative impact of lower social class.⁶⁵ A case-control study and cohort study in the West of Scotland also suggests that smoking behaviour alone does not explain the higher mortality from lung cancer in this population.^{66, 67} In short, smoking is strongly associated with deprivation and can explain much of the higher mortality, but cannot explain the excess mortality once deprivation is accounted for. One possible explanation for this is that self-reported smoking either underestimates the number of cigarettes, or the variation in toxins inhaled.⁶⁸

Mitchell found that the higher self-reported ischaemic heart disease (IHD) in Scotland could not be entirely explained by known risk factors such as smoking and deprivation, suggesting that health behaviours alone do not explain the Scottish Effect in IHD.⁶⁹ This observation was supported by self-reported smoking in Glasgow, Liverpool and Manchester which shows that the overall prevalence is very similar in the three cities.⁴¹

Evidence – diet

Shelton, using the Scottish Health Survey and the English Health Survey did not find that regional variations in: fruit & vegetable consumption; smoking; obesity; or diabetes were consistently able to explain variations in mortality (with the exception of obesity and mortality in women).⁷⁰ Gray analysed the Scottish Health Survey and found that much of the unhealthy diet evident in Glasgow as compared to the rest of Scotland could be accounted for by markers of deprivation (including area-based measures and individual measures) although some aspects of diet related to vegetable intake in men and butter and salt in women were not.⁷¹ A direct comparison of healthy eating (measured as self-reported eating of five or more portions of fruit/vegetables per day) in Greater Glasgow, Liverpool and Manchester shows very similar rates and therefore casts doubt on diet being an important explanatory factor for differences in mortality. A similar pattern is witnessed for adult obesity prevalence.⁴¹

Evidence – physical activity

No evidence was identified which compared physical activity rates between relevant populations.

Evidence – general

The hypothesis that more prevalent negative health behaviours might be associated with higher mortality is consistent with numerous cohort studies in individuals and is coherent with existing knowledge. Furthermore, there is evidence that trends in smoking and alcohol exposure lead to changes in mortality patterns, providing natural experimental evidence in favour of the hypothesis. Many of the problem causes of death in Scotland (including alcohol-related, drug-related, cardiovascular and cerebrovascular disease) are strongly associated with health behaviours and this provides evidence of specificity. The hypothesis is plausible and has numerous analogies from other time periods and populations.

It has been suggested that countries transit through phases of an 'epidemic' in relation to tobacco use, where males and richer social groups take up (and subsequently quit) smoking first, followed later by females and poorer social groups.⁷² This is in many ways similar to the obesity epidemic which affected men and richer groups first before spreading to the rest of the population. It may therefore be the case that Scotland (and Glasgow) is simply passing through a negative health behaviour epidemic slightly later than other areas. Even if this is the case, it does not explain why Scotland might be such a laggard.

Summary

There is sufficient evidence to suggest that alcohol use and illicit drug use are more prevalent in Scotland and Glasgow, and that this difference is likely to be in excess of what might be explained by deprivation. Although smoking and poor diet are more prevalent in Scotland and Glasgow, they are well explained by the patterning of deprivation. There is no evidence to evaluate the variation in physical activity rates.

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Are there differences in physical activity rates between relevant populations?	Comparative survey work between populations could address this knowledge gap.
Are there differences in smoking, alcohol, drugs, diet and physical activity rates which are more extreme in Glasgow or Scotland which might not be captured in routine surveys but might explain the variation in mortality?	This would require sensitive sampling methods to ascertain whether there are differences in health behaviours between populations, in combination with a suitably large sample size.

5. Individual values

Description of hypothesis

If Scots (and the population of the West of Scotland and Glasgow in particular) were to have a different psychological outlook to others, in terms of their aspirations or time preferences (favouring immediate rewards to delayed gratification - hedonism) it is said that this may have a consequent negative impact on mortality (most likely through a variety of negative health behaviours relating to alcohol, drug and tobacco use). This hypothesis suggests that the individuals with the highest mortality are more hedonistic than others or have lower aspirations, and that this in turn leads to a higher prevalence of adverse health behaviours and higher mortality.

Later hypotheses address whether there is a cultural difference in values (i.e. a group-level effect relating to this hypothesis).

Evidence

No evidence was identified which reported a comparison of the values held by Scots with other groups (to assess strength of association), nor any evidence relating to specificity, temporality, gradient or to any experiments.

A study by Gilhooly et al in the West of Scotland investigated whether those healthy and unhealthy individuals aged 70-90 years differed in their personality, beliefs and spirituality. It found that:

“the healthy participants were less neurotic, more likely to endorse an internal locus of control belief, and to report a greater sense of coherence ... the unhealthy group scored higher on the religiosity/spirituality measure”⁷³

This study was based on individuals rather than populations and could therefore be subject to the ecological fallacy; but it does suggest that the values of populations may play a role in determining the population mortality pattern – making the hypothesis plausible and consistent with self-reported health. The study could however be confounded by a selection effect, for example if those terminally ill are more likely to embrace religiosity/spirituality.

There is a growing research base which suggests that individualistic and consumerist values might be detrimental to health, and this has provided numerous analogies and would make the hypothesis coherent with existing knowledge.⁷⁴⁻⁷⁸

Research to test hypothesis

Research question	Possible methodological approach
Is there a difference in the values held by Scots or Glaswegians compared to other populations, and if there are differences, when did they emerge?	Comparative survey work addressing questions regarding the time preferences and aspirations of individuals could identify any current differences.

6. Different culture of substance misuse

Description of hypothesis

This hypothesis suggests that the way in which substances (illicit drugs, tobacco and alcohol) are used in Scotland differs from that elsewhere, and/or that there is a unique culture surrounding their use which exacerbates their effect. The hypothesis does not require the per capita use of substances, or the distribution of their use amongst the population, to be different.

Evidence

A review comparing the alcohol cultures internationally reported that Scotland was not different from England in terms of its alcohol culture, and not distinctive in relation to other comparison nations.⁷⁹

However, there is evidence that some groups inhale more toxins per cigarette than others, and also that simple reports of tobacco use might mask the true exposure to risk.⁶⁸ There is, moreover, some suggestion from the media of the emergence of a different drug misuse identity in Scotland (classically associated with the cult film ‘Trainspotting’), but no evidence was uncovered to examine whether there is a real difference to other populations.⁸⁰

No evidence was identified relevant to the consistency, specificity, temporality, biological gradient, coherence or experimental evidence for this hypothesis. It is plausible and there are analogies with particular cultures of substance misuse elsewhere leading to distinctive patterns of harm (as with alcohol culture in the former USSR in the early 1990s).

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Is there a different culture of substance misuse in Scotland or Glasgow compared to other populations?	This would require sensitive sampling methods to ascertain whether there are differences in health behaviours between populations reflecting a distinctive culture of misuse, in combination with a suitably large sample size.

7. Culture of boundlessness and alienation

Description of hypothesis

It has been suggested that Scotland and Glasgow may have a culture different to other areas in terms of boundlessness, hopelessness and alienation. This has some parallels to cultural patterns observed during the 19th Century in some industrial cities.⁸¹⁻⁸³ It is proposed that where emergent cultural norms undermine pre-existing behavioural restraints, greater risk taking and self-destructive behaviour (including alcohol/drug misuse and violence) may ensue.

Evidence

There is conflicting evidence around whether a distinctive subculture exists in parts of the UK, and this debate revolves around whether the most disadvantaged group simply lack material resources to participate in societal norms and cultural activities, or whether there is a different set of values and behavioural norms compared to the rest of society.⁸⁴ There is no doubt that social polarisation increased radically during the 1980s,⁸⁵ but there is no evidence to suggest that this has generated a distinctive culture⁸⁶ – instead the evidence suggests that behaviours have been shared across society, but are socially patterned.^{87, 88} The evidence is therefore largely against there being an association between exposure and outcomes and it is incoherent with existing knowledge around the existence of subcultures in the UK. However, no evidence was identified which sought to address the question of whether there is a distinctive culture in Scotland, the West of Scotland or Glasgow in particular (and therefore a biological gradient, consistency, temporality, experimental evidence and specificity cannot be assessed).

It is not clear if there are contemporary examples of this type of culture in other populations which can be used to evaluate whether similar mortality outcomes are seen (although it may be that the former USSR from 1990 exhibited many of these features). It is plausible that if a distinct culture arose, without social norms which reduced excessive risk-taking, this could lead to higher mortality from the specific causes of death witnessed in Scotland (alcohol-related, drug-related, violence and suicide). Analogies to negative health outcomes arising from cultures without limiting social norms were found in the 19th Century by Durkheim within newly industrialising cities.

Research to test hypothesis

Research question	Possible methodological approach
Is Scotland, especially the West of Scotland and Glasgow, suffering from a cultural anomie distinct from other areas?	Measures of this could be developed and added to any comparative survey work. This would involve defining anomie in this context and its key elements.

8. Family, gender relations and parenting differences

Description of hypothesis

If family breakdown, acrimony between partners or dysfunctional parenting were more prevalent in Scotland (and the West of Scotland and Glasgow in particular),⁸⁹ it is hypothesised that this would have a negative influence on health. This hypothesis has been forwarded by Iain Duncan Smith's Centre for Social Justice and has featured in some of the work by Carol Craig.^{47, 90, 91}

Evidence – gender relations

No evidence was identified that compared inter-gender relationships in Scotland with other countries (or Glasgow or the West of Scotland with other areas), other than the rates of reported domestic violence being higher in Glasgow than Scotland as a whole.¹⁹ Hughes suggests that the rebirth of family life seen in the aftermath of World War II, which was built around new freedoms and consumerism across Britain, clashed with Glasgow's "macho culture", but that Glasgow did not adopt a more progressive perspective on gender relations because of the dominant heavy industry, council housing patterns and unemployment which restricted the transition. Alcohol misuse and gambling have been seen to have maintained a quite negative male identity when it was challenged by these new cultural trends.^{92, 93} Craig's interesting account of Glasgow's divergence in the first part of the 20th Century relies quite heavily on fictional and biographical accounts, perhaps to a greater extent than more conventional historical work, and it is therefore difficult to assess whether there is a true association between exposure and outcomes.⁴⁷

The hypothesis suggests that unhealthy gender relations were evident in Scotland from at least the early decades of the 20th Century. If true, this would mean that the deleterious mortality impacts associated were delayed by some 30 or 40 years in terms of the Scottish comparison to the rest of Europe, and some 60 or 70 years in terms of creating a mortality impact different from cities with similar deprivation such as Liverpool and Manchester. The temporality in the relation to the 'confidence' aspects of the hypothesis is similarly problematic, given that the Calvinist culture, if prevalent and generating an effect, might be expected to have been most profound in the 19th and early 20th Centuries, rather than from 1950 or 1980 onwards.

Evidence – family disruption and parenting

Family disruption is closely associated with a range of negative social and health outcomes.⁹⁴ Scotland, the West of Scotland and Glasgow have high rates of lone parent households and teenage pregnancies, both markers of the kinds of changes in family structure emphasized by those suggesting that this hypothesis is causal.¹⁹ There is also a biological gradient between exposure to lone parenthood and family breakdown and mortality.⁹⁵ However, there is little difference in the proportion of households with children who are headed by a lone parent in Glasgow as compared to Liverpool and Manchester (and the proportion in Scotland as a whole

is less than 3% above that in England); and the teenage pregnancy rates in Glasgow are lower than in Manchester and only slightly higher than Liverpool (and are almost identical between Scotland and England).⁴¹

There has been a marked rise in lone parenthood between 1971 and 2001 (from 4.3% to 25.6% of families with dependent children) in Britain. Rowthorn accounts for 38-59% of the variation in this rise across Britain by the rise in male unemployment and deindustrialisation.⁹⁶ This rise was particularly acute during the 1980s which demonstrates an appropriate temporal relationship with the overall mortality phenomenon and Scottish Effect.⁹⁷

The hypothesis is coherent with a large body of evidence relating to the importance of childhood attachment to parents and childhood development,⁹⁸ although much more research is required to be clear how parenting can be improved or to understand the precise mechanisms involved.⁹⁹

No evidence was identified on the consistency, specificity or presence of analogies for this hypothesis, nor were any experiments identified. The hypothesis is however plausible.

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Are there differences in family breakdown, gender relations and/or parenting styles between Scotland, Glasgow and other populations?	This could be established using comparative survey work between populations.

9. Lower ‘social capital’

Description of hypothesis

It is suggested that ‘social capital’ in Scotland (and the West of Scotland and Glasgow in particular) is lower than elsewhere and that this has had a detrimental impact on health.

Evidence

No evidence was identified which compared the extent or patterning of social capital in Scotland (or the West of Scotland or Glasgow), in the original sense described by Granovetter, with other areas.¹⁰⁰ However, there have been attempts to measure and compare proxies for social capital including the proportion of the population volunteering and electoral attitudes, registration and turnout.¹⁹ These suggest that while lower social capital may be more associated with Glasgow than with Scotland as a whole, voter turnout was similar in comparisons between Glasgow, Liverpool and Manchester.⁴¹ There are quite distinctly lower levels of religious affiliation in Glasgow as compared to Liverpool and Manchester (a difference that is also seen between Scotland and England) which might suggest that social capital of this type could be protective.⁴¹ Some measures in the household surveys for the West of Scotland indicate that some limited social capital measures relating to the fear of crime may be lower than elsewhere in Scotland, but there was no difference between Scotland and England in the measures of ‘bridging capital’, and ‘bonding capital’ may be higher in Scotland.¹⁰¹

The hypothesis is consistent with the findings of Stuckler in relation to the protective effect of social capital in Eastern Europe in the face of neoliberalism.¹⁰² No evidence was identified to address whether the association is specific, temporal, whether there is a biological gradient or which demonstrated experimental evidence. It is a plausible hypothesis that is coherent with what we know about disrupted communities^{43, 44} and there are analogies in relation to social isolation and health.

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Is there a difference in social capital between Glasgow/Scotland and other populations?	Measures of social capital could be gathered in comparative survey work.
Have there been changes in social capital and have they preceded or followed changes in mortality?	It may be possible to use historical data from the household survey to evaluate changes in social capital over time.

10. Sectarianism

Description of hypothesis

Another hypothesis that has been advanced to explain both the higher mortality in Scotland and the distinct Scottish Effect, is that Scotland is more affected by sectarianism than other areas, and that this in turn has negatively impacted on health.

Evidence

Scotland (and Glasgow and the West of Scotland in particular) is recognised as having problems with sectarianism revolving around a divide between Catholic and Protestant communities, and this provides some evidence for a strength of association between exposure and outcome. However, no evidence was identified that examined whether Scotland is more affected by sectarian or similar divisions than other areas, and consequently no evidence to allow assessment of a biological gradient.^{47, 103}

The effect is not consistent: Northern Ireland, a part of the UK which undoubtedly has a greater sectarian problem than Scotland, has reduced its mortality faster than Scotland since the middle of the 20th Century (Figure 1). Furthermore, there is evidence that at times when sectarianism was at its worst in both Northern Ireland (during the height of the civil conflict in the 1970s) and in Glasgow (during the peak times of Irish immigration in the 19th and 20th Centuries), the mortality patterning was not at its worst (going against a temporal relationship and consistency).⁴⁶ There was no evidence that could address whether sectarianism generates specific effects or to provide experimental evidence. It is plausible and coherent that sectarianism would generate such impacts and there are analogies with other types of discrimination.¹⁰⁴

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Is there a difference in sectarianism between Glasgow/Scotland and other areas?	Measures of this could be developed and added to any comparative survey work.

11. Culture of limited social mobility

Description of hypothesis

It has been suggested that there is a culture of limited aspiration in Scotland which has led to limited social mobility. This has been linked to both a cultural lack of confidence fostered through the influence of Calvinism, and also to a distinctive culture of social control where people have been discouraged from being seen to be doing better than their peers.^{47, 105} The hypothesis focuses on the emergence of these distinctive cultural patterns in the first decades of the 20th Century.

Evidence - confidence

No evidence was identified to suggest that Scottish or Glaswegian adults are more or less confident than other populations, or have a different culture in terms of their relationships and ability to support each another. There is some evidence amongst adolescents that Scots are lower (but rising) in some measures of confidence than in other European countries in recent years.¹⁰⁶

Evidence – Calvinist values

Calvinism is most commonly thought to be synonymous with certain religious affiliations, particularly Presbyterianism. However, some have suggested that ‘cultural Calvinism’ may be more socially pervasive. No evidence was identified to allow an assessment of whether this has been the case in Scotland.

If Calvinist culture was to be a driving force in creating the Scottish mortality phenomenon it would be expected that populations most immersed in it would be worst affected. Second (and subsequent) generation young Irish Catholics in the West of Scotland, despite suffering higher levels of deprivation than non-Catholics, have similar health outcomes. This suggests that there might be some protective effect from being part of an Irish Catholic culture in Glasgow.¹⁰⁷ There is however a pattern of greater exposure to an unhealthy diet in West of Scotland Catholics,¹⁰⁸ and working men with Irish surnames in the West of Scotland have higher mortality after adjusting for smoking and deprivation (particularly from cardiovascular disease).¹⁰⁹

The geographical areas of Scotland worst affected by higher mortality (such as Glasgow) and the populations worst affected (such as those of Irish Catholic origin)¹¹⁰ are those with a less distinctive Calvinist influence in contrast to areas of the Highlands, Islands and Grampian (as measured by Presbyterianism) which also have lower mortality. Thus the biological gradient runs counter to Calvinism being associated with higher mortality or higher mortality unexplained by deprivation in Glasgow. The temporal relation is also ill-fitting, since Scottish adult mortality rates were similar to England and other European nations in the first half of the 20th Century (although infant mortality rates were briefly higher).^{1, 47}

Craig relates the rise in consumerism in Glasgow to the Calvinist culture, yet it is much more coherent and consistent to see such consumerism as bound up with the rise of secularism and materialism from the 1960s, and from the 1980s as bound up with the ascendancy of neoliberalism. ^{74, 75} No experimental evidence was identified, nor any which addresses the specificity of the hypothesis, but the hypothesis is plausible and there are analogies for cultural trends impacting significantly on health outcomes.

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Do Scots, and in particular those in the West of Scotland and Glasgow, show a difference in their confidence as compared to other populations?	Measures could be developed and added to any comparative survey work.
Does the dominant culture in Scotland, particularly the West of Scotland and Glasgow, limit social mobility?	Measures could be developed and added to any comparative survey work.

12. Health service supply and demand

Description of hypothesis

If the quality, accessibility, or demand for health services was lower in Scotland (and the West of Scotland and Glasgow in particular) compared to other areas, this might increase the differential mortality – since populations with equal need for services might benefit inequitably from them, thereby generating differential outcomes.

Evidence

There is evidence that the variation in health service use between Scotland and the rest of the UK is minimal compared to the variation in the provision of services within regions and cities with respect to need (the ‘inverse care law’).^{111, 112} However, there was no evidence identified to ascertain whether this pattern of variation was different from that in England or elsewhere.¹¹³⁻¹¹⁵

Health and social services were also very similar across the UK until 1999 (after which time devolution allowed for some divergence in the models of service provision in England, Wales, Northern Ireland and Scotland), and this does not fit with a divergence in mortality from around 1980 (therefore the temporal relationship is problematic). The same is true for the general divergence in mortality patterns in Scotland from the rest of Europe, as there was a marked increase in accessibility to health and social services in Scotland (and the rest of the UK) from the late 1940s following the Beveridge reforms and the introduction of the NHS. This again does not fit with health and social service provision being an important determining factor. Scotland has the highest spending per capita on health services and the greatest number of doctors and dentists per capita of all the constituent parts of the UK. This suggests that there is no association with service provision and the mortality phenomenon, nor a biological gradient.¹¹⁶

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 Scotland.¹¹⁷ 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.

The quality of health care 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, vaccination rates, the care of patients with acute heart attacks and waiting times. Again this suggests that there is no clear association between the quality of care and the mortality phenomenon, nor a biological gradient.¹¹⁶ 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.¹¹⁸

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 mortality phenomenon is not coherent with how we understand the social causes of alcohol-related deaths, drugs-related deaths, suicide and violent deaths – all major contributors to the higher mortality. Furthermore, no analogy was identified for health and social services generating such a phenomenon, though it is plausible that, in extremis, communities without access to services could be unsupported in dealing with negative health behaviours such as drug misuse, and that this would have consequent mortality impacts.

It is not clear if there are natural experiments, consistent effects seen elsewhere with exposure to poorer or less accessible health services, or specific outcomes that are related.

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Is NHS or social care spending patterned differently in Scotland or Glasgow compared to elsewhere?	Routine data could potentially be utilised to expose any difference in how resources are allocated between areas.
Is there a greater problem with the inverse care law in Scotland (and the West of Scotland and Glasgow) than in England (and the rest of Europe)?	This would require comparative work between Scotland, Glasgow and other populations. Some initial work in Glasgow has been performed looking at this. ¹¹⁹ Another approach might be to compare disease incidence with mortality (on the basis that incidence is ultimately less liable to be affected by healthcare).

13. Deprivation concentration ('area effects')

Description of hypothesis

This hypothesis proposes that deprived areas in Scotland (or Glasgow) are more 'concentrated' than in England & Wales (or in other Scottish cities), in that they are more extensive and form unique, large, deprived, monocultural communities. This patterning of deprivation is hypothesised to have an impact additional to that of deprivation (an 'area effect') and could therefore help to explain the 'Scottish Effect' and 'Glasgow Effect'.

Evidence

Sridharan demonstrated that in 2001 the variation in mortality in Scotland was better explained by including a measure of the deprivation of surrounding geographical areas in addition to the deprivation of the specific area in question, rather than using the latter alone.¹²⁰ This suggested that the hypothesis about the spatial arrangement of deprivation is plausible and coherent with what evidence is currently available, and furthermore that there is an association between proximity to broader deprivation in addition to the deprivation of the immediate area of residence which might explain some of the Scottish Effect.¹²⁰ There is no research to evaluate whether there is a consistent effect elsewhere, a biological gradient, or specific effects. Nor is there any on changes in deprivation patterning to provide either experimental evidence or a temporal relationship. An analogy for this hypothesis would be the work of Wilkinson on the effects of national inequality on health.^{43, 44}

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Is there a difference in the geographical patterning of deprivation between Glasgow, Scotland and other populations - either now, or in the past, and is this associated with mortality patterns?	Work is currently planned by the GCPH to address this hypothesis by examining the effects of concentrated deprivation in Glasgow.

14. Greater inequalities

Description of hypothesis

There is a large body of evidence which suggests that richer nations with greater income inequality do worse on a range of health and social outcomes than more equitable comparison nations.^{43, 44} An implication is that if Scotland (and the West of Scotland or Glasgow) suffers from greater income inequality,^d this would have an additional impact to that of deprivation per se and thereby help to explain the 'Scottish Effect'.

Evidence in favour

Weich found that higher regional income inequality, as measured by the Gini coefficient,^e was associated with worse self-rated health in Britain, consistent with the wider body of international work which associates greater inequality in rich nations with worse health and social outcomes (along with demonstrable biological gradients and specific outcomes).^{43, 44, 121} This makes this hypothesis both plausible and coherent with existing knowledge and provides analogies.

However, it seems that income inequality is lower in Scotland than in England, meaning there is a lack of association.^{122, 123} At the same time, the UK has a higher income inequality than other areas of Europe and the existence of an association may depend on what size of geographical area is most important in assessing the exposure (i.e. is inequality best measured on a UK basis, Scotland-wide, regional or city basis to capture the damaging effects that inequality engenders?).

There is an appropriate temporal relationship between the rise in income inequality witnessed during the 1980s and the divergence of Scottish mortality from England.¹²⁴ It is not clear if countries which have radically changed their inequality profiles (in 'natural' experiments) have had consequent changes in the mortality profile.

d Income inequality is the disparity between high income and low income households.

e The Gini coefficient is a measure of the dispersion of income amongst a population such that zero represents equality and one represents total inequality (i.e. all the wealth is held by one individual). It is mathematically defined by the ratio of the area between a line of equality and the Lorenze curve (depicting the proportion of the wealth held by each proportion of the population) over the total area under the line of equality.

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Does variation of the geographical area in which income inequality is calculated help explain variations in mortality (i.e. calculation of income inequality at a city, regional, national level)?	Data is available at the UK, Scottish and regional level for income inequality. Calculation at a more local level would require careful consideration of appropriate measures to ensure comparability, and appropriate sampling and sample size.
Are there populations in countries, similar in their inequality to the UK, which suffer higher mortality, which is also not entirely accounted for by higher deprivation?	It may be possible to use routine or survey data on other countries to examine whether other areas exhibit phenomena similar to those seen in Glasgow.
Do countries which have had radical changes in income inequalities, similar to the UK, also have areas with mortality patterns similar to those in the West of Scotland and Glasgow?	Identify developed countries which have had a similar rise in inequalities and then examine the patterning of mortality within the identified countries.

15. Deindustrialisation

Description of hypothesis

A popular hypothesis has been that Scotland (and Glasgow and the West of Scotland in particular) suffered from more profound deindustrialisation than other areas. For this hypothesis to be true, deindustrialisation would need to compound the impact of deprivation to exacerbate mortality.

Evidence

Scotland (and Glasgow and the West of Scotland) has higher mortality than comparable deindustrialised areas across northern Europe despite lower poverty levels and lower unemployment. The West of Scotland lost the greatest number of industrial jobs as a proportion of total employment when compared to 10 such deindustrialised areas across the UK and mainland Europe (losing 58% compared to 52% in the Ruhr, 51% in Merseyside and 24% in Northern Ireland). Merseyside and Glasgow lost a similar number of industrial jobs (greater than other deindustrialised areas which provides some strength of association).^{3, 4, 125} Furthermore, if the larger Glasgow conurbation (the West of Scotland or Clydeside) is considered, the scale of deindustrialisation is greater than elsewhere in the UK (although Merseyside is a close second).¹²⁶

The presence or otherwise of a biological gradient between Scotland or Glasgow and other populations has not been tested, but the mortality pattern was not specific or consistent since mortality is improving faster in other European deindustrialised areas than in the West of Scotland.^{3, 4} Deindustrialisation did occur in the West of Scotland during the period of the emergence of the Scottish Effect, and there were periods of deindustrialisation earlier in the 20th Century which could account for the divergence of Scotland's mortality from other European countries. There is therefore an appropriate temporal relationship. The relation between deindustrialisation and higher mortality is plausible, has individual experimental evidence and has analogies using studies on individual unemployment and health.¹²⁷ It is also coherent with existing knowledge about unemployment and health outcomes.

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Were there important differences in how deindustrialisation occurred and in the associated policy contexts between areas?	GCPH-funded work to examine this hypothesis, using a range of routine data and a review of the sociological evidence, is currently underway.
Were there important differences between nations, pertaining to the societal determinants of health, which either mitigated or exacerbated the mortality impact of deindustrialisation (e.g. pace of deindustrialisation, availability of alternative employment, welfare policy)?	There is currently work underway funded by the GCPH to examine this hypothesis using a range of routine data and a review of the sociological evidence.
Is there a biological gradient linking the degree of deindustrialisation and the cause-specific mortality outcomes that have become more prevalent in the West of Scotland?	It may be possible to examine this using routine data.

16. Political attack

Description of hypothesis

There are three aspects to this hypothesis. First, the UK as a whole was exposed to neoliberal ideology and policies from 1979 onwards in a way in which other European countries were not. This neoliberalism amounted to a political attack on the institutions and culture of the organised working class. Second, Scotland, the West of Scotland and Glasgow were more vulnerable to the damaging effects of this neoliberal 'political attack' than other areas of the UK. Third, there was in Scotland a distinctive cultural reaction to all of this because of the perceived problems of democratic illegitimacy (formulated in some circles as a 'democratic deficit').¹²⁸ This hypothesis has significant currency amongst social commentators, social historians, political scientists and some epidemiologists,¹²⁹ and was perhaps most succinctly termed a 'war without bullets' by Cathy McCormack.^{128, 130}

Evidence

Deprivation, poverty and class explain most of the variation in mortality in Britain until after the early 1980s. It is only during the term in Government of Margaret Thatcher that the mortality record in Glasgow begins to diverge from that of equally deprived Liverpool and Manchester, and that the excess mortality in Scotland as a whole becomes less explicable in relation to deprivation (although there is some missing data which makes the trend in mortality between cities difficult to interpret, and it is possible that the divergence emerges earlier).⁴¹ The mortality trends for Glasgow compared to the rest of Scotland for outcomes such as cardiovascular disease, stroke and suicide all diverge in the years around 1980.¹⁹ This demonstrates an ideal temporal relationship for all the relevant mortality outcomes for the Scottish Effect. However, the divergence of Scotland from the rest of Europe appears earlier than the 'political attack' (around 1950).

A mortality pattern similar, if more profound, to Scotland appeared in the former USSR after 1989 as it undertook a rapid transition from state communism to free market capitalism. This included rapid rises in alcohol-related deaths and suicide (and therefore a specific set of outcomes from a similar exposure).^{131, 132} The rise in mortality has been explicitly linked to neoliberal policies such as privatisation in Eastern Europe, providing a natural experiment, coherence and consistency (as well as plausibility and analogy).¹³³

There is evidence that neoliberalism was not adopted in all the deindustrialising nations in Europe, and to the extent that it was adopted in some of them it was also more mitigated and tempered in its effect than was the case in the UK.¹³⁴ Furthermore, Glasgow, amongst UK cities, and its Labour politics notwithstanding, is noted as having had a particular emphasis on accommodating and promoting neoliberalism during the 1980s, a feature which may help explain the greater vulnerability of the City during the Thatcher years (i.e. an association between exposure and outcome with a biological gradient).^{135, 136}

The West of Scotland had a higher proportion of council housing and industrial employment than other UK regions during the late 1970s. Both were specific and early targets for the neoliberal ‘political attack’, and this would have made the region particularly vulnerable to the damaging effects of such an attack. In addition, the specific opposition from the region to the neoliberal politics of an earlier Conservative administration (that of Edward Heath) may have made the region a particular target for the attack.¹³⁷ Furthermore, the electoral trends and historical accounts of the period after 1979 show a particular reaction, in terms of sociopolitical culture, against neoliberalism – indicative of a strongly intensifying sense of disempowerment.¹²⁸

It was not the case that the political attack exposure was present from the mid-20th Century and this hypothesis does not therefore explain the higher mortality in Scotland prior to the 1980s. However, the hypothesis may well contribute towards an explanation of the continued higher mortality in Scotland (explained or unexplained by deprivation) after this date.

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Was neoliberalism implemented to a greater degree in Glasgow/Scotland and was its impact felt to a greater degree than elsewhere in Europe and in the UK?	There is currently work underway at Glasgow University seeking to evaluate the differences in how neoliberalism was implemented in some sample countries in Western and Eastern Europe compared to Scotland.
What were the consequences on health of the implementation of neoliberalism in other countries, and are there other populations which have experienced similar consequences to Scotland, the West of Scotland and Glasgow?	Identify other countries where there was a similar neoliberal approach (? USA or New Zealand) and areas within these countries that might be vulnerable to its damaging effects, then consider whether any differences in health outcomes can be explained by deprivation (e.g. is there a ‘Pittsburg Effect’, ‘Detroit Effect’ etc.).
What factors increase the vulnerability of populations to neoliberalism?	Stuckler has suggested that social capital protected some populations in Eastern Europe from the worst impacts of neoliberalism. ¹⁰² A similar methodology could be adopted to evaluate whether there are factors within the UK which made some areas more vulnerable.

17. Climatic differences

Description of hypothesis

There are two separate hypotheses regarding the role of the Scottish climate in explaining the mortality phenomenon: that Scots suffer from a lack of sunlight and a consequent deficit in vitamin D; and that harsher winters increase mortality through the effects of the cold.

Evidence – winter deaths

Excess winter deaths are a well recognised phenomenon in Scotland. Howieson described how there were 51,600 excess winter deaths (i.e. the number of deaths over the four winter months as compared to the four summer months) between 1989 and 2001, and that almost all of these occurred in those aged over 65 years. The most deprived areas were affected disproportionately (and counter to temperature records across Scotland which affected the richer and rural areas hardest). Glasgow, being most deprived, was worst affected.^{138, 139}

Gemmell's work suggests that a one degree drop in temperature approximates to a one percent rise in mortality one week later, although the amount of excess winter mortality is decreasing over time.¹⁴⁰ A socioeconomic gradient in excess winter mortality was not found in a British cohort study of those aged over 75 years.¹⁴¹

Despite these observations, it is clear that the higher premature mortality in Scotland from 1980 onwards is being driven by alcohol- and drugs-related deaths, violence, suicide, road-traffic accidents as well as cardiovascular disease, cancer and stroke; and that these excess deaths are occurring in young and middle-aged adults as well as amongst the elderly.⁸ Excess winter mortality is therefore neither a plausible nor coherent explanation for the entirety of the mortality phenomenon, despite there being an association. Excess winter mortality may play a small role in higher mortality in the older groups, but the reason for this remains unclear (although poorer housing conditions, poverty and fuel poverty have been suggested).¹³⁸ It is possible that it has contributed to the divergence of Scottish mortality from the rest of Europe in the second half of the 20th Century.

Evidence – vitamin D

Gillie is the main proponent of this thesis and has helpfully published the main arguments in a short book.¹⁴² The crux of the argument is that Scotland, and the West of Scotland in particular, receives less sunlight (which is essential for the natural production of vitamin D by the skin) than other areas, and that there is therefore a strength of association between lack of exposure to sunlight and mortality. There is evidence of a gradient in vitamin D levels in population blood samples between Scotland and the rest of the UK.¹⁴³ However, although a lack of vitamin D has been linked to a number of conditions, to be coherent and plausible it would require that a pathway be established from low vitamin D through mental health issues to drug-related, alcohol-related, violent deaths. It is also unclear how vitamin D can be an important cause unless there has been a temporal change in exposure. It is possible that changes in housing or

diet may play a role.¹⁴³ For the main drivers of premature mortality (drugs, alcohol, etc.) in recent decades, there is no consistency, specificity nor biological gradient that could explain the phenomenon, as more Northern areas of Scotland are less affected than Glasgow, and as there has not been the emergence of this pattern in other areas similarly affected by low sunlight exposure. However, these factors could be important for the emergence of the mortality divergence earlier in the 20th Century and further work to synthesise this growing literature base and to consider how it might explain the earlier epidemiological trends is merited. There are hormonal analogies but no experimental evidence for low vitamin D as a cause was identified.

Research to test hypothesis

<i>Research question</i>	<i>Possible methodological approach</i>
Can vitamin D deficiency explain the specific causes of death seen in Scotland?	More comprehensive reviews of the available literature are merited to examine whether the cause-specific mortality pattern in Scotland can be related to vitamin D.
Is there a difference between the West and East of Scotland levels of sunlight exposure (taking account of potential dressing differences due to temperature and wind etc.) and serum vitamin D levels?	It might be possible to construct a multivariate model including differences in sunlight exposure, weather and diet to try to explain variation in mortality in Scotland.
Is there a difference in either sunlight exposure or serum vitamin D levels between affluent and deprived populations?	Specific literature searches to examine the social patterning of serum vitamin D are merited.
Are there variations in the historical patterning of the Scottish diet which might account for variations in the mortality patterning over time?	A specific literature search to try to identify dietary patterns in vitamin D intake in Scotland is merited.

Table 1 - Summary of hypotheses and their fit with the Bradford-Hill criteria

Hypothesis	Upstream or downstream hypothesis	Mortality phenomenon	Number of criteria supporting	Number of criteria not supporting	Bradford-Hill's criteria for causality*									
					Strength of association	Consistency	Specificity	Temporality	Biological gradient	Plausibility	Coherence	Experiment	Analogy	
1. Deprivation	NA	Higher mortality	2	2	-	X	-	-	-	-	-	X	✓	✓
		Scottish/Glasgow effect	5	0	-	✓	-	-	✓	-	✓	✓	U	✓
2. Migration	NA	Higher mortality	4	2	X	-	-	-	✓	-	✓	✓	X	✓
		Scottish/Glasgow effect	4	2	X	-	-	-	✓	-	✓	✓	X	✓
3. Genetic differences	Downstream	Higher mortality	3	4	-	✓	X	X	X	-	X	X	✓	✓
		Scottish/Glasgow effect	3	4	-	✓	X	X	X	-	X	X	✓	✓
4. Health behaviours	Downstream	Higher mortality	9	0	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
		Scottish/Glasgow effect	8	0	U	✓	✓	✓	✓	✓	✓	✓	✓	✓
5. Individual values	Midstream	Higher mortality	4	0	-	✓	-	-	-	-	✓	✓	-	✓
		Scottish/Glasgow effect	4	0	-	✓	-	-	-	-	✓	✓	-	✓
6. Different culture of substance misuse	Midstream	Higher mortality	2	0	-	-	-	-	-	-	-	-	-	✓
		Scottish/Glasgow effect	2	0	-	-	-	-	-	-	✓	-	-	✓
7. Culture of boundlessness & alienation	Midstream	Higher mortality	2	2	X	-	-	-	-	-	✓	X	-	✓
		Scottish/Glasgow effect	2	2	X	-	-	-	-	-	✓	X	-	✓
8. Family, gender relations & parenting	Midstream	Higher mortality	4	0	✓	-	-	-	✓	-	-	✓	-	-
		Scottish/Glasgow effect	4	0	✓	-	-	-	✓	-	-	✓	-	-

Hypothesis	Upstream or downstream hypothesis	Mortality phenomenon	Number of criteria supporting	Number of criteria not supporting	Bradford-Hill's criteria for causality*									
					Strength of association	Consistency	Specificity	Temporality	Biological gradient	Plausibility	Coherence	Experiment	Analogy	
9. Lower social capital	Midstream	Higher mortality	4	0	-	✓	-	✓	-	-	✓	✓	-	✓
		Scottish/Glasgow effect	5	0	✓	✓	-	✓	-	✓	✓	✓	-	✓
10. Sectarianism	Midstream	Higher mortality	4	2	✓	X	-	✓	X	-	✓	✓	-	✓
		Scottish/Glasgow effect	4	2	✓	X	-	✓	X	-	✓	✓	-	✓
11. Culture of limited social mobility	Upstream	Higher mortality	2	4	-	X	-	X	X	-	✓	X	-	✓
		Scottish/Glasgow effect	2	4	-	X	-	X	X	-	✓	X	-	✓
12. Health service supply and demand	Midstream	Higher mortality	1	4	X	-	-	X	X	-	✓	X	-	-
		Scottish/Glasgow effect	1	4	X	-	-	X	X	-	✓	X	-	-
13. Deprivation concentration	Midstream	Higher mortality	4	0	✓	-	-	-	-	-	✓	✓	-	✓
		Scottish/Glasgow effect	4	0	✓	-	-	-	-	-	✓	✓	-	✓
14. Greater inequalities	Upstream	Higher mortality	7	0	-	✓	✓	✓	✓	✓	✓	✓	-	✓
		Scottish/Glasgow effect	7	0	-	✓	✓	✓	✓	✓	✓	✓	-	✓
15. Deindustrialisation	Upstream	Higher mortality	6	2	✓	X	X	✓	✓	✓	✓	✓	✓	✓
		Scottish/Glasgow effect	5	2	-	X	X	✓	✓	✓	✓	✓	✓	✓
16. Political attack	Upstream	Higher mortality	4	1	-	-	-	X	-	-	✓	✓	✓	✓
		Scottish/Glasgow effect	9	0	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
17. Climatic differences	Upstream	Higher mortality	2	0	✓	-	-	-	-	-	-	-	-	✓
		Scottish/Glasgow effect	2	3	✓	X	X	-	X	-	X	-	-	-

* - = no evidence to demonstrate or refute the consideration; U = there is uncertainty around whether the evidence supports this criterion;

✓ = balance of evidence supports criterion; X = balance of evidence does not support criterion.

Evaluating the hypotheses

All of the hypotheses would seem to have at least some validity in helping to explain the mortality pattern in Scotland. Having identified the candidate hypotheses, it is clear that a more thorough reviewing of the literature is required to ensure that important elements of the evidence base have not been missed, misinterpreted or incorrectly appraised. Some of the hypotheses also clearly require further research to clarify their validity in the face of the Bradford-Hill criteria. A current lack of research relevant to these hypotheses does not mean that they will have no relevance in terms of developing – or altering – our understanding.

Figure 24 details, in light of the currently available evidence, which hypotheses met most of the Bradford-Hill criteria regarding the divergent mortality pattern for Scotland from the middle of the 20th Century. The current weight of evidence is clearly not very strong for some hypotheses being causal – most notably: health service supply and demand; the culture of limited social mobility; and genetic differences. In contrast some hypotheses are supported as causative explanations, most notably: health behaviours; greater inequalities; deindustrialisation; deprivation concentration; individual values; and lower 'social capital'.

Figure 24 – The number of Bradford-Hill criteria met by each hypothesis for the divergence of the Scottish mortality pattern from the rest of Europe in the middle of the 20th Century

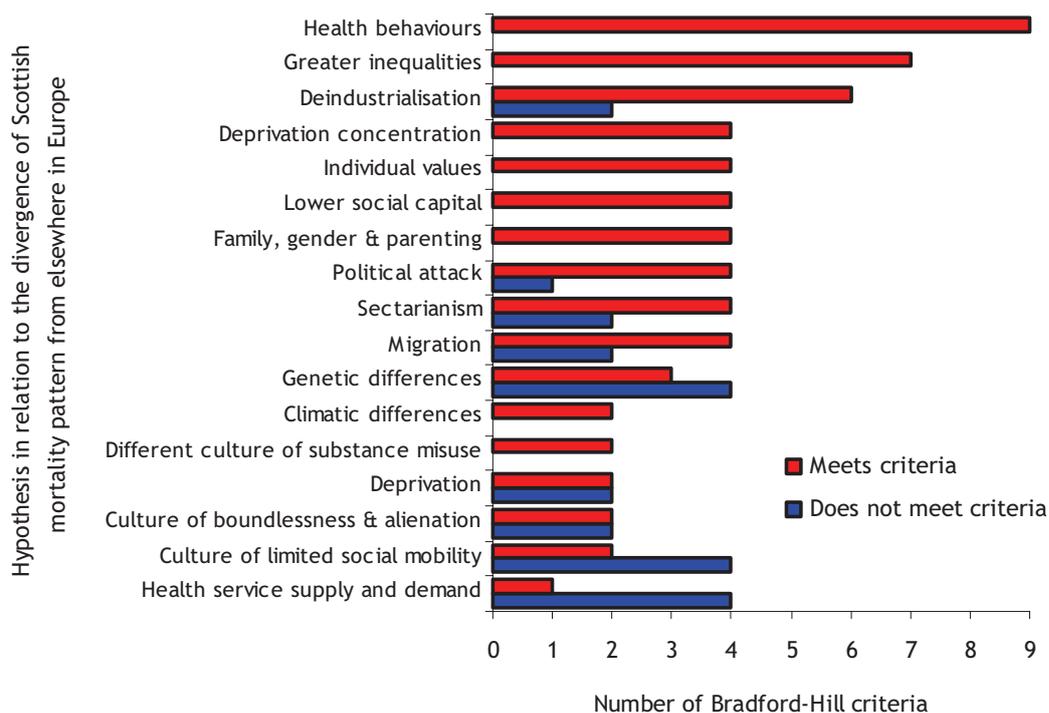
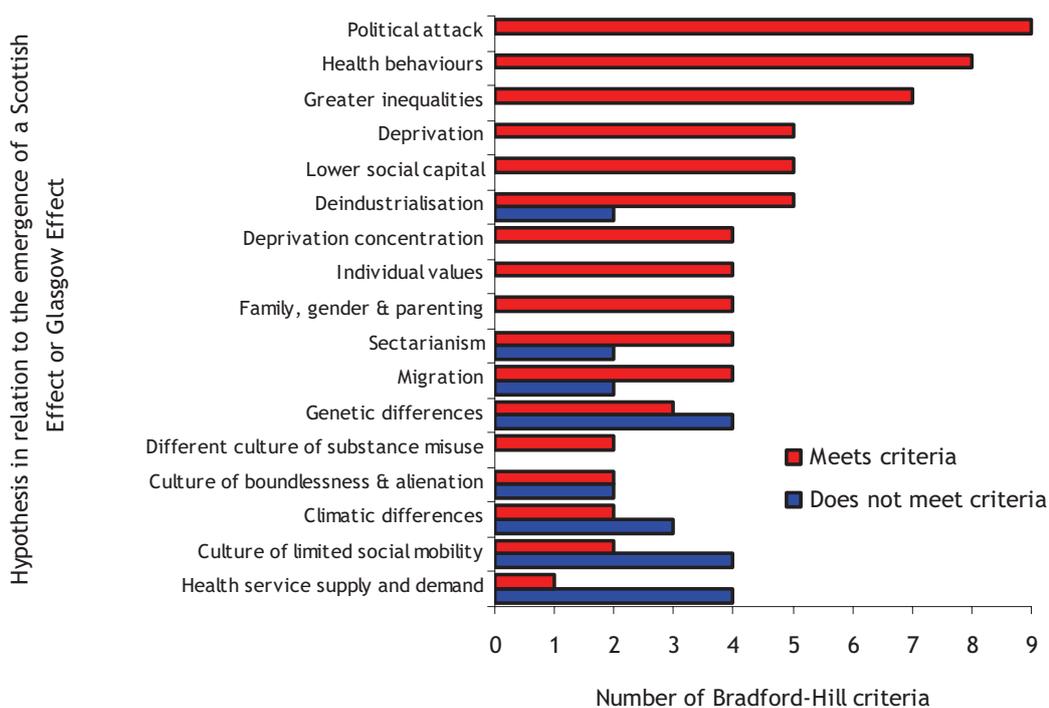


Figure 25 details the candidate hypotheses for the Scottish Effect and Glasgow Effect. Some hypotheses are well supported as being causal: political attack; health behaviours; greater inequalities; deprivation (artefact); deindustrialisation; deprivation concentration, and individual values are all supported as causal explanations.

The next section seeks to explore the potential to build a greater and more integrated understanding through synthesizing some of the stronger hypotheses in a larger hypothetical framework which can encompass more of the totality of the problem.

Figure 25 – The number of Bradford-Hill criteria met by each hypothesis for the emergence of the Scottish Effect or Glasgow Effect



Synthesis of the mortality divergence from 1950 onwards

The divergence of Scottish mortality from that of its European neighbours from 1950 onwards was largely due to a slower reduction in mortality from cardiovascular disease, stroke, respiratory disease and cancer. It was not until the 1980s that alcohol-related deaths, drug-related deaths, suicide and road-traffic accidents became drivers of higher mortality rates. There has been less research into the divergence of Scottish mortality from 1950, and the precise nature of the causal pathways in this earlier period is, therefore, far from certain.

Downstream explanations

The balance of evidence suggests that a greater exposure to negative health behaviours such as smoking, alcohol, poor diet and low levels of physical activity should play a role in explaining the divergence, but there is little evidence available which compares Scotland with other populations to assess the degree to which this is the case. However, the main causes of death (cardiovascular disease, respiratory disease, stroke and cancer) are known to be strongly associated with tobacco use, alcohol, diet and physical activity. A transition through a tobacco epidemic has been witnessed internationally, and it may be that Scotland passed through its tobacco epidemic later than other European countries.

Midstream explanations

A variety of midstream explanations, often with a distinctive cultural bent, have been proposed for the higher mortality pattern including: family, gender relations and parenting differences; a culture of limited social mobility, and a different culture of substance misuse. These factors may well have been present in Scottish society, yet it is less likely that they are the cause of the divergent pattern. For example, the kinds of cultural norms described are likely to predate the mortality divergence by perhaps 50 years – or even more. Furthermore, Scots had a consistently lower rate of alcohol-related mortality than other European countries until around 1990. However, some of the cultural factors highlighted may well have resulted from the underlying causes of the divergence and have fed into or reinforced negative and damaging behaviours; and others may have interacted with the underlying causes to a similar end.

Upstream explanations

It is unclear whether Scotland suffered from greater poverty than other European nations around 1950. No evidence was identified that specifically looked at this for the relevant time period. It is possible that unemployment (higher than in England), more precarious industrial employment, poverty, inequality, overcrowded housing conditions and the large scale reconstruction which resulted in the peripheral council housing estates around Glasgow, Edinburgh and other large Scottish conurbations could be important in providing a causal explanation (Figure 26). More work is required to determine whether climatic influences (including the role of vitamin D) are important in explaining the higher mortality in Scotland during this time.

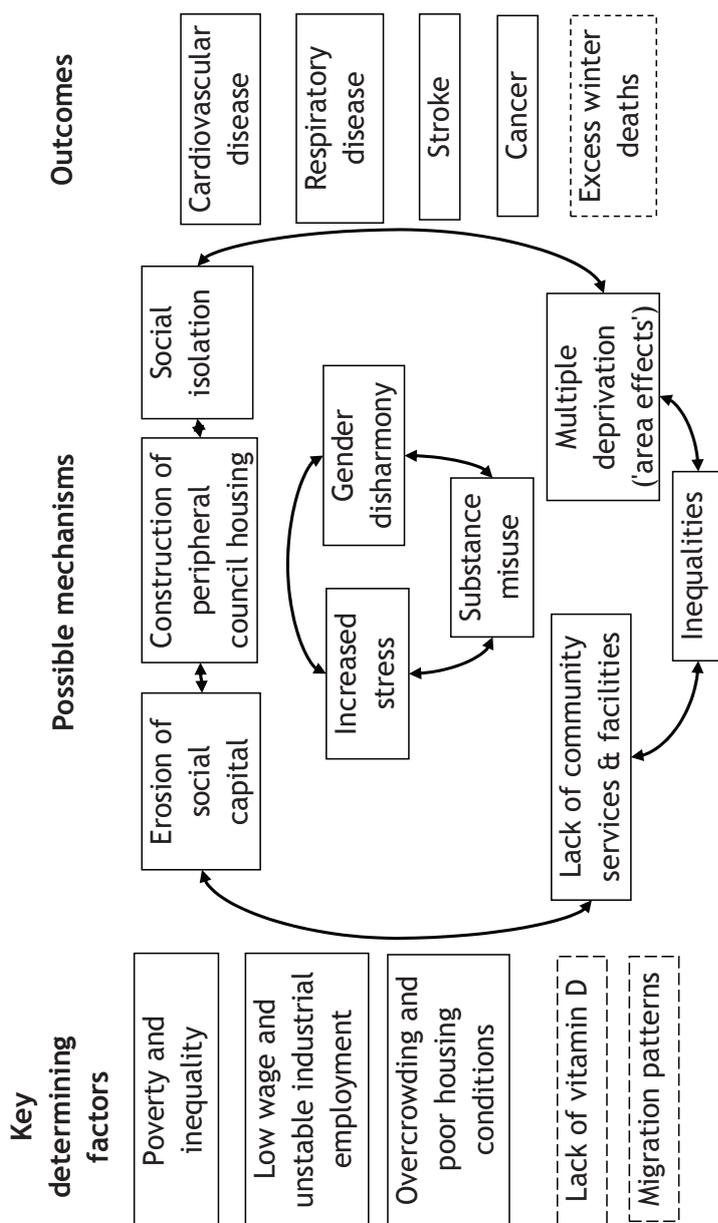
Unlikely explanations

It does not seem likely that genetic factors or health service quality, supply or demand are important explanations for the mortality divergence, although there is currently little evidence to examine them.

Summary

The cause of the divergence of Scottish mortality from its neighbours from 1950 onwards is uncertain and requires further thought and research. The task of describing and explaining this historical phenomenon has been perhaps somewhat neglected in light of the salience of the more recent divergence from around 1980. The most likely explanation is that Scotland suffered a combination of more precarious employment, overcrowding, poverty and ill-conceived reconstruction than other countries during this period, and this fits with what is known about the housing conditions and industrial history. A representation of this perspective is shown in Figure 26. However, a better understanding will require more comparative historical work to determine the key differences between Scotland and other nations during this time period.

Figure 26 – A simplified representation of the synthesis of the cause of the divergence of Scottish mortality from the rest of Europe from 1950 onwards



Synthesis of the causes of the Scottish Effect and Glasgow Effect

Artefactual explanations

At the beginning of the 1980s, deprivation (as measured by the Carstairs index) was able to account for most of the excess mortality in Scotland (and Glasgow) as compared to England & Wales. Over the next two decades the excess increased while the proportion of it which could be explained by this measure of deprivation fell – from almost two thirds to less than a half. Some have suggested that this decline is simply an artefact, reflecting the ‘dating’ of the Carstairs measure, and there is some evidence to suggest that this may be at least partially true. However, even where equally deprived cities are compared (Glasgow, Liverpool and Manchester), Glasgow’s mortality is seen to improve more slowly from the late 1970s onwards. It seems clear therefore that poverty and deprivation continue to play an important role in explaining Scotland’s (and Glasgow’s) higher mortality (quite possibly somewhat more important than is suggested by use of the Carstairs measure), but that there are additional causal factors at play. It is also possible that there was a migration of the healthier members of the Scottish and Glaswegian population around this time, which might have played a small role in increasing the relative mortality. Nonetheless, such artefactual explanations seem unlikely to be able to explain the entirety of the Scottish Effect or Glasgow Effect.

Downstream explanations

The cause of the Scottish Effect and Glasgow Effect which fits best with the Bradford-Hill criteria concerns the prevalence of various health behaviours (particularly alcohol, and drug misuse). Although exposure to alcohol and illicit drugs are clearly causal for many mortality outcomes, the synthesis requires an explanation about why the prevalence of these negative health behaviours is higher, or why their effects are more profound, in Scotland and Glasgow.

Midstream explanations

There is a series of related hypotheses, again often of a culture orientation, some of which are clearly plausible and have some evidence, but require comparative work to ascertain whether there is a different patterning in Scotland and Glasgow compared to other areas. These include: individual values (particularly with respect to whether Scots and Glaswegians are more hedonistic or have lower aspirations); lower ‘social capital’; family, gender relations and parenting differences; different culture of substance misuse; culture of boundlessness and alienation, and culture of limited social mobility. All of these are likely to have some relevance (to a greater or lesser extent) in explaining the emergence of the Scottish Effect and Glasgow Effect, but are challenged in terms of explaining why the Scottish and Glaswegian populations were worse affected than other areas from the early 1980s, rather than at an earlier point in time. These hypotheses, like the hypothesis relating to negative health behaviours, therefore appear necessary and important explanations in describing the causes of the Scottish Effect and Glasgow Effect, but are not sufficient.

Figure 27 attempts to depict the cultural and health behavioural hypotheses as a series of inter-related mechanisms which are, to a degree, self-reinforcing and -perpetuating. The role of each mechanism may be great or small, but taken together, they are likely to be representative of the ways in which other factors (which are discussed below) get 'under the skin' and generate mortality from drugs, alcohol, suicide, road-traffic accidents, cardiovascular disease, respiratory disease, stroke and cancer.

Culture can influence structures in society (such as public services and the economy) and how individuals perceive themselves (such as religious beliefs and self-esteem), but culture is itself profoundly influenced by the socio-economic and political environment in which it exists. To explain why the kinds of cultural patterns described in the midstream hypotheses emerged, or made themselves felt, in Scotland and Glasgow, to a greater degree than in other areas from the late 1970s, requires an assessment of how culture changes. Four related hypotheses have been proposed which attempt to explain why the culture of Scotland and Glasgow might have changed at this time and why negative health behaviours became more prevalent. One of these has been classified here as a midstream hypothesis – deprivation concentration. However this clearly connects closely to the main contenders in the upstream category, which are: greater inequalities; deindustrialisation, and political attack. Accordingly, these four hypotheses are discussed below.

Upstream explanations

Inequalities and the patterning of deprivation as candidate explanations for the post-1980 divergence within Scotland and Glasgow fit well with the Bradford-Hill criteria. The UK as a whole rapidly became more unequal during the 1980s and early 1990s, with Scotland and in particular Glasgow being the poorest parts. It is less clear whether inequality within Scotland or within the West of Scotland or Glasgow has been higher than other areas, but the UK is the most unequal country in Europe and the West of Scotland is the poorest area of the UK. It is therefore quite plausible that the West of Scotland has suffered disproportionately as a consequence of the increasing inequality within the UK. It has also been suggested that deprivation in Scotland (and Glasgow in particular) has been more concentrated than elsewhere, particularly in the large peripheral housing estates such as, in Glasgow, Easterhouse, Castlemilk, Pollok and Drumchapel, as well as in places like Ferguslie Park in Paisley, and Wester Hailes and Craigmillar in Edinburgh. It seems highly plausible therefore that housing policies, in their interaction with other social changes, have contributed to some degree in amplifying the effects of poverty and deprivation in these communities.

The West of Scotland may have suffered a more rapid and profound process of deindustrialisation than other areas of the UK and Europe, although the evidence for Glasgow City in comparison to Manchester and Liverpool is less certain. Sharp deindustrialisation was a particular feature of Scotland from the late 1970s onwards and the loss of employment that was associated with it contributed to the inequalities in the UK between the South-East of England and elsewhere. Such a rapid loss of employment and community structure is likely to have had an important influence on the culture of Scotland and Glasgow, and on the health-related behaviours.

There is increasing evidence to suggest that the pace and manner of deindustrialisation was forced in the UK, in comparison to other European countries, and that this was part of a broader 'political attack'. The 'political attack' hypothesis is consistent with the other upstream mechanisms (deindustrialisation, deprivation concentration and inequalities) and the midstream/cultural explanations relating to disempowerment, hopelessness, family breakdown etc. It suggests that in the years after Edward Heath's abandonment of his neo-liberal 'Selsdon agenda' in 1972, and particularly after the loss of the 1974 General Elections, elements within the Conservative Party planned a political attack against the organised working class – which had proved such an obstacle for Heath. After 1979 this plan was implemented with a high degree of resolve, and with very significant adverse impacts. Similar health outcomes relating to a yet more profound implementation of a neoliberal policy agenda were seen in the former USSR. This makes the political attack hypothesis a potentially important one in tying the down-, mid- and upstream determinants of health together in a coherent narrative which can explain the problematic mortality phenomenon. What seems less clear is why other deindustrialised areas of the UK did not suffer in quite the same way as Scotland and Glasgow during the 1980s. On this question, there is evidence to suggest that Scotland was in some ways particularly targeted in the broader UK attack, and in key respects more vulnerable to its damaging effect than other areas – in light of its pre-existing poverty, high dependence on industrial employment, and very high reliance on council housing. There is also some evidential basis to indicate that Scotland had a distinctive cultural response to the political attack – in terms of a national feeling of disempowerment and loss of control.

Unlikely explanations

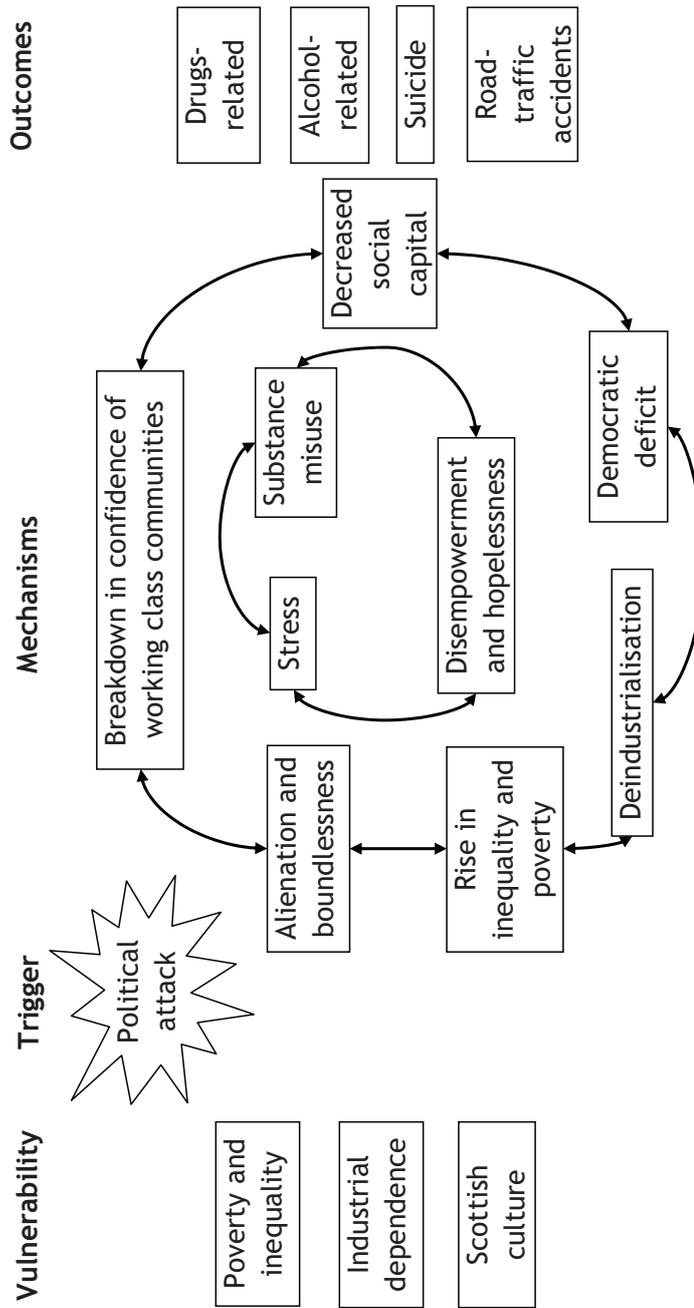
Other hypotheses may also play a role. The studies on migration thus far suggest that it does not explain the higher mortality pattern, but it seems plausible that migration should have had some minor impact. It is also consistent with the overall synthesis in that such a breakdown of communities would be likely to create an incentive for some to move to other areas for work or to avoid the social problems that became more prevalent.

It does not seem likely that genetic differences or health service supply and demand are important explanations for the Scottish Effect or Glasgow Effect. A genetic explanation cannot explain the cause-specific mortality patterns or the relatively rapid change in outcomes. The evidence, such as is available, suggests that health services in Scotland are at least as high in quality as elsewhere, and the cause-specific mortality patterns for this period are more likely to be related to the social determinants of health rather than health service quality. There is little available evidence to support a role for vitamin D and the Scottish climate in being an important causal factor at present, but further work to synthesise the growing literature base in this area and relate this to epidemiological trends is merited.

Summary

There is no single explanation for the cause of the Scottish or Glasgow Effects, but it is likely that a collection of negative health behaviours related to a cluster of cultural phenomena has been responsible (Figure 27). It is likely that the higher mortality occurred at this point in time as a result of increased inequality, deindustrialisation and a breakdown of community structures as a consequence of a neoliberal political attack during the 1980s which affected Scotland and Glasgow more profoundly than other parts of the UK. There is more evidence for the downstream elements of this synthesis and it is not clear which of the mechanisms are most important in generating the outcomes, but it is unlikely that any of the mechanisms described (such as gender, family relations and parenting differences) provide sufficient explanation alone. Therefore, most of the proposed hypotheses are likely to contribute something towards explaining the Scottish Effect and Glasgow Effect, with the exception of migration, genetic differences, climatic differences, and health service supply and demand.

Figure 27 - A simplified representation of the synthesis of the cause of the Scottish Effect (and Glasgow Effect)



DISCUSSION

Strengths and limitations

It is unlikely that there are important hypotheses for the mortality phenomenon in Scotland (and Glasgow) that were missed by the search strategy. The appraisal process using the Bradford-Hill criteria was robust and is likely to have minimised bias in the synthesis process. It is however possible that some deductive hypotheses arising from outside the health research field may have been missed. Furthermore, it may be that some evidence relating to a particular hypothesis has been overlooked since the search strategy for each hypothesis, once identified, was not systematic. This could have significantly underestimated the quantity and quality of the evidence available for a particular explanation and could have biased the results of the synthesis. To address this fully, a comprehensive and systematic search of the literature for each hypothesis would be required. There is a difficulty in relating exposures to outcomes given that a time lag might be expected for some outcomes (and the length of any timelag will vary dependent on the nature of the exposure and the outcome of interest). Further work is required to clarify how this might relate to the temporality of putative causal factors in this synthesis. Finally, for some of the hypotheses there has been, as indicated previously, little or no research. Such hypotheses might yet prove to be relevant and the application of the Bradford-Hill criteria in this report should not be seen in any way to preclude that possibility. A further programme of research which will investigate a number of them is due to begin soon.

Implications

The causes of the higher mortality in Scotland and Glasgow are complex and relate to the cumulative exposures of its population over many decades. Further research is required to fully understand why mortality is higher and the implications for future research are discussed in the next section. It is clear that there is a difference between how the Scottish mortality problem is commonly viewed and the findings of this preliminary synthesis. Usually, the higher mortality is attributed to higher rates of smoking, greater alcohol consumption and poorer diet, and these are of course true. However, these explanations are not sufficient to understand why the mortality picture in Scotland is so very different to that of other, seemingly comparable, areas. Leyland was touching on this kind of problem when he stated:

“Without denying the importance of specific public health interventions – such as those directed at smoking and alcohol consumption – it is difficult to escape the conclusion that the inequalities we describe have more fundamental origins in lifestyles determined by poverty ... [the] growing number of young deaths from essentially negative lifestyles, is evidence of a need for social policies which have a more direct influence on poverty and its correlates.”⁸

This is a common theme in public health, and exemplifies the need to consider the ‘upstream causes of the causes’.¹⁴⁴⁻¹⁴⁶ This report has attempted to use the Bradford-Hill criteria to examine which hypotheses seem best placed to provide causal explanations for the phenomenon, and has sought to begin the process of elaborating a synthesis which can encapsulate the partial insights and understandings achieved in existing research in a ‘higher order’ hypothesis.

Research implications

There are clearly a large number of outstanding deductive hypotheses which could be investigated for their potential causal role in generating the mortality pattern in Scotland (and Glasgow in particular). There remains room, however, for further inductive work into the divergence of Scottish mortality from the rest of Europe around 1950.

CONCLUSION

The study of public health outliers is an important means to understanding the causes and patterning of health and disease. Scotland suffers from higher mortality than its UK and continental European neighbours, and has for the last 30 years suffered from a new, and troubling, mortality pattern which seems in some respects closer to that witnessed in Eastern Europe.

It is unlikely that any single cause is responsible for this phenomenon, and there is uncertainty around why Scotland started to diverge from elsewhere in Europe around 1950. It is clearer that the health and social patterns that emerged during the 1980s and 1990s are more closely linked to negative health behaviours (e.g. alcohol consumption), but these behaviours are in turn heavily influenced and shaped by the social, cultural and economic disruption which occurred as the political and economic policies of the UK abruptly changed from the later 1970s onwards.

Any understanding of the Scottish mortality patterning requires, as well as a clear focus on behaviours, an understanding of the most 'upstream' determinants of health, including economic, social and political history. Any analysis which only refers to tobacco use or alcohol, or even to early years — massively significant as these factors undoubtedly are — will inevitably fail to identify the overall causality of this profoundly troubling phenomenon, and will be liable to generate at best partial policy interventions, which are in turn most liable to prove disappointing in their outcomes.²⁷

APPENDIX

Table 2 – Search strategy for electronic database literature search

Search	Search term	Number of studies identified
1	Glasgow effect (keyword)	7
2	Scottish effect (keyword)	12
3	Glasgow effect (title)	5
4	Scottish effect (title)	7
5	1 or 2 or 3 or 4	19
6	Remove duplicates from 5	13
7	Mortality (keyword)	727,034
8	Glasgow (keyword)	22,808
9	Scotland (keyword)	28,081
10	Mortality (title)	97,479
11	Glasgow (title)	2,171
12	Scotland (title)	5,990
13	8 or 9 or 11 or 12	49,090
14	7 or 10	727,034
15	13 and 14	7,373
16	Deprivation (keyword)	83,166
17	Deprivation (title)	18,277
18	Carstairs (keyword)	267
19	Carstairs (title)	15
20	Poverty (keyword)	39,875
21	Poverty (title)	3,359
22	Class (keyword)	441,983
23	Class (title)	68,657
24	Inequal* (keyword)	18,693
25	Inequal* (title)	5,609
26	16 or 17 or 18 or 19 or 20 or 21 or 22 or 23 or 24 or 25	572,719
27	26 and 15	447
28	Remove duplicates from 27	296
29	6 and 28	309
	Final search result	309

AUTHORSHIP

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Gerry McCartney and Chik Collins devised the methodology for the report. Gerry McCartney undertook the literature review and framed the initial synthesis. All authors contributed to the synthesis and critically revised the manuscript.

The views expressed in this report do not necessarily reflect the views of the employing organisations or funders of the contributing authors. The employers and funders played no part in the design or conduct of this work, although the programme of the Glasgow Centre for Population Health (GCPH) relating to the mortality phenomenon is guided by the GCPH Management Board and Advisory Committee which have representatives of the funders thereon.

REFERENCES

1. Leon D, Morton S, Cannegieter S, McKee M. Understanding the health of Scotland's population in an International context: A review of current approaches, knowledge and recommendations for new research directions. London: London School of Hygiene and Tropical Medicine & Public Health Institute for Scotland; 2003 (Accessed at http://www.scotpho.org.uk/home/Comparativehealth/InternationalComparisons/int_mortality_comparisons.asp on 20th April 2010).
2. Aykroyd W, Kevany J. Mortality in infancy and early childhood in Ireland, Scotland and England and Wales 1871-1970. *Ecology of Food & Nutrition* 1973;2:9-11.
3. Walsh D, Taulbut M, 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.
4. Walsh D, Taulbut M, Hanlon P. The aftershock of deindustrialization - trends in mortality in Scotland and other parts of post-industrial Europe. *The European Journal of Public Health* 2010;20:58-64 (doi:10.1093/eurpub/ckp063).
5. Whyte B. Scottish mortality in a European context 1950-2000: an analysis of comparative mortality trends. Edinburgh Scottish Public Health Observatory; 2007 [downloaded from <http://www.scotpho.org.uk/nmsruntime/saveasdialog.asp?IID=3591&sID=3079> on 20th April 2010].
6. Human Mortality Database: University of California, Berkeley (USA) & Max Planck Institute for Demographic Research (Germany) [Accessed at www.mortality.org on 4th June 2010].
7. Davies C, Dundas R, Leyland A. Increasing socioeconomic inequalities in first acute myocardial infarction in Scotland, 1990-92 and 2000-02. *BMC Public Health* 2009;9.
8. Leyland A, Dundas R, McLoone P, Boddy F. Cause-specific inequalities in mortality in Scotland: two decades of change. A population-based study. *BMC Public Health* 2007;7.
9. 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;124:487-495 (doi:10.1016/j.puhe.2010.02.006).
10. Marmot M, Bosma H, Hemmingway H, Brunner E, Stansfeld S. Contribution of job control and other risk factors to social variations in coronary heart disease incidence. *Lancet* 1997;350:235-9.
11. Smith G, Harding S. Is control at work the key to socioeconomic gradients in mortality? *Lancet* 1997;350:1369-70.
12. Adler N. When one's main effect is another's error: material vs. psychosocial explanations of health disparities. A commentary on Macleod et al., "is subjective social status a more important determinant of health than objective social status? Evidence from a prospective observational study of Scottish men" (2005;61(9):1916-1929). *Social Science and Medicine* 2005;63:846-50.

13. Carstairs V, Morris R. Deprivation: explaining differences in mortality between Scotland and England and Wales. *BMJ* 1989;299:886-9.
14. The Scottish Effect? Edinburgh: Scottish Council Foundation Healthy Public Policy Network; 1998.
15. Hanlon P, Lawder R, Buchanan D, Redpath A, Walsh D, Wood R. 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;27:199-204.
16. Popham F. Is there a "Scottish effect" for self reports of health? Individual level analysis of the 2001 UK census. *BMC Public Health* 2006;6.
17. Reid J. Excess mortality in the Glasgow conurbation: exploring the existence of a Glasgow effect. Glasgow: University of Glasgow; 2008.
18. Mheen PM-vD, Smith G, Hart C. Socioeconomic differentials in mortality among men within Great Britain: time trends and contributory causes. *Journal of Epidemiology and Community Health* 1998;52:214-8.
19. Hanlon P, Walsh D, Whyte B. Let Glasgow Flourish. Glasgow: Glasgow Centre for Population Health; 2006.
20. Hill A. The environment and disease: association or causation? *Proceedings of the Royal Society of Medicine* 1965;58:295-300.
21. Smith GD, Ben-Shlomo Y, Lynch J. Life course approaches to inequalities in coronary heart disease risk. In: Stanfeld S, Marmot M, eds. *Stress and the Heart: Psychosocial Pathways to Coronary Heart Disease*. London: BMJ Books; 2002:20-49.
22. Kuh D, Hardy R, Langenberg C, Richards M, Wadsworth M. Mortality in adults aged 26-54 years related to socioeconomic conditions in childhood and adulthood: post war birth cohort study. *BMJ* 2002;325:1076-80.
23. Oshowo O, Hobsley M. Helicobacter pylori and duodenal ulcers. *Gut* 1997;40:155-6 (doi:10.1136/gut.40.1.155-b).
24. Identifying the environmental causes of disease: how should we decide what to believe and when to take action? London: Academy of Medical Sciences; 2007.
25. Weiss NS. Can the "specificity" of an association be rehabilitated as a basis for supporting a causal hypothesis? *Epidemiology* 2001;13:6-8.
26. Roux AD, Schwartz S, Susser E. Ecological variables, ecological studies, and multilevel studies in public health research. In: Detels R, McEwan J, Beaglehole R, Tanaka H, eds. *Oxford textbook of public health (4th edition)*. Oxford: Oxford University Press; 2004.
27. Link B, Phelan J. McKeown and the idea that social conditions are fundamental causes of disease. *American Journal of Public Health* 2002;92:730-2.

28. Rose G. Sick individuals and sick populations. *International Journal of Epidemiology* 1985;14:32-8.
29. McLaren L, McIntyre L, Kirkpatrick S. Rose's population strategy of prevention need not increase social inequalities in health. *International Journal of Epidemiology* 2010;39:372-7.
30. Lynch J. Socio-epidemiology: some observations the past, present and future. *Australasian Epidemiologist* 2000;7:7-15.
31. Dahlgren G, Whitehead M. Policies and strategies to promote social equity in health. Stockholm: Institute of Futures Studies; 1991.
32. Evans R, Stoddart G. Producing health, consuming health care. *Social Science and Medicine* 1990;31:1347-63.
33. Snow J. On the mode of communication of cholera (2nd edition). New York: Commonwealth Fund; 1936 (originally published 1855).
34. Hunter DJ. Leading for Health and Wellbeing: the need for a new paradigm. *Journal of Public Health* 2009;31:202-4.
35. Williams H. Hegel, Heraclitus, and Marx's Dialectic: Harvester Wheatsheaf; 1989.
36. Hegel G. Hegel's Science of Logic. London: Allen & Unwin; 1812.
37. Gray L. Comparisons of health-related behaviours and health measures between Glasgow and the rest of Scotland. Glasgow: Glasgow Centre for Population Health; 2007.
38. Landy R, Walsh D, Ramsay J. The Scottish Health Survey: the Glasgow effect (topic report). Edinburgh: Scottish Government; 2010.
39. George S. It's not just deprivation - or is it? *Public Health* 2010;124:196-7.
40. Deprivation key data sources. ScotPHO, 2010. (Accessed at http://www.scotpho.org.uk/home/Lifecircumstances/Deprivation/deprivation_keydatasources.asp on 23rd May 2010).
41. Walsh D, Bendel N, Jones R, Hanlon P. Investigating a 'Glasgow Effect': why do equally deprived UK cities experience different health outcomes. Glasgow: Glasgow Centre for Population Health; 2010.
42. 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.
43. Wilkinson R. The impact of inequality: how to make sick societies healthier. Abingdon: Routledge; 2005.
44. Wilkinson R, Pickett K. The Spirit Level: why more equal societies almost always do better. London: Penguin; 2009.
45. Boffeta P, Brennan P, Saracci R. Neoplasms. In: Detels R, McEwan J, Beaglehole R, Tanaka H, eds. *Oxford Textbook of Public Health*. Oxford: Oxford University Press; 2004:1156.

46. Devine TM. *The Scottish nation*. London: Penguin; 1999.
47. Craig C. *The tears that made the Clyde – well-being in Glasgow*. Glendaruel: Argyll publishing; 2010.
48. 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;16:759 (doi:10.1016/j.healthplace.2010.03.007).
49. Wild S, McKeigue P. Cross sectional analysis of mortality by country of birth in England and Wales, 1970-92. *BMJ* 1997;314:705-10.
50. Bhala N, Bhopal R, Brock A, Griffiths C, Wild S. Alcohol-related and hepatocellular cancer deaths by country of birth in England and Wales: analysis of mortality and census data. *Journal of Public Health* 2009;31:250-7.
51. Wannamethee S, Shaper A, Whincup P, Walker M. Migration within Great Britain and cardiovascular disease: early life and adult environmental factors. *International Journal of Epidemiology* 2002;31:1054-60.
52. Popham F, Boyle P, O'Reilly D, Leyland A. *Selective internal migration. Does it explain Glasgow's worsening mortality record?* Edinburgh: GRO Scotland; 2009.
53. Exeter D, Boyle P, Feng Z, Boyle M. Shrinking areas and mortality: an artefact of deprivation effects in the West of Scotland? *Health & Place* 2009;15:399-401.
54. Exeter D, Feng Z, Flowerdew R, Boyle P. Shrinking areas and mortality: an artefact of deprivation effects? *Journal of Epidemiology and Community Health* 2005;59:924-6.
55. Brimblecombe N, Dorling D, Shaw M. Migration and geographical inequalities in health in Britain. *Social Science and Medicine* 2000;50:861-78.
56. Brimblecombe N, Dorling D, Shaw M. Mortality and migration in Britain, first results from the British Household Panel Survey. *Social Science and Medicine* 1999;49:981-8.
57. Thomas D, Karagas M. Migrant studies. In: Schottenfeld D, JF D FJJ, eds. *Cancer Epidemiology and Prevention*. 2nd ed. New York: Oxford University Press; 1996:236-54.
58. Chen L, Smith GD, Harbord R, Lewis S. Alcohol intake and blood pressure: A systematic review implementing a Mendelian randomization approach. *PLoS Med* 2008;5:e52.
59. Batty G, Der G, Macintyre S, Deary I. Does IQ explain socioeconomic inequalities in health? Evidence from a population based cohort study in the West of Scotland. *BMJ* 2006;332:580-3.
60. Bloor M, Gannon M, Hay G, Jackson G, Leyland A. Contribution of problem drug users' deaths to excess mortality in Scotland: Secondary analysis of cohort study. *BMJ* 2008;337:a478.
61. *World Drug report 2010*. New York: United Nations Office on Drugs and Crime; 2010.

62. Catto S. How much are people in Scotland really drinking? A review of data from Scotland's routine national surveys. Glasgow: NHS Health Scotland; 2008.
63. Gray L. Is the "Glasgow Effect" of cigarette smoking explained by socio-economic status?: A multilevel analysis. *BMC Public Health* 2009;9:245(doi:10.1186/471-2458-9-245).
64. Hart C, Hole D, Gillis C, Smith G, Watt G, Hawthorne V. Social class differences in lung cancer mortality: risk factor explanations using two Scottish cohort studies. *International Journal of Epidemiology* 2001;30:268-74.
65. Gruer L, Hart CL, Gordon DS, Watt GC. Effect of tobacco smoking on survival of men and women by social position: a 28 year cohort study. *BMJ* 2009;338:b480.
66. Gillis C, Hole D, Boyle P. Cigarette smoking and male lung cancer in an area of very high incidence. I Report of a case-control study in the West of Scotland. *Journal of Epidemiology and Community Health* 1988;42:38-43.
67. Gillis C, Hole D, Hawthorne V. Cigarette smoking and male lung cancer in an area of very high incidence. II Report of a general population cohort study in the West of Scotland. *Journal of Epidemiology and Community Health* 1988;42:44-8.
68. West R, Zatonski W, Przewozniak K, Jarvis M. Can we trust national smoking prevalence figures? Discrepancies between biochemically assessed and self-reported smoking rates in three countries. *Cancer Epidemiology, Biomarkers and Prevention* 2007;16:820-2.
69. Mitchell R, Fowkes G, Blane D, Bartley M. High rates of ischaemic heart disease in Scotland are not explained by conventional risk factors. *Journal of Epidemiology & Community Health* 2005;59:565-7.
70. Shelton N. Regional risk factors for health inequalities in Scotland and England and the "Scottish Effect". *Social Science and Medicine* 2009;69:761-7.
71. Gray L, Leyland A. A multilevel analysis of diet and socio-economic status in Scotland: investigating the 'Glasgow effect'. *Public Health Nutrition* 2008;12:1351-8.
72. Lopez A, Collishaw N, Piha T. A descriptive model of the cigarette epidemic in developed countries. *Tobacco Control* 1994;3:242-7.
73. Gilhooly M, Hanlon P, Cullen B, Macdonald S, Whyte B. Successful ageing in an area of deprivation: Part 2 - a quantitative exploration of the role of personality and beliefs in good health in old age. *Public Health* 2007;121:814-21.
74. James O. *Affluenza*. London: Vermillion; 2007.
75. Schwartz B. *Paradox of choice: why more is less*. London: Harper Collins; 2005.
76. Hanlon P, Carlisle S. Is 'modern culture' bad for our health and well-being? *Global Health Promotion* 2009;16:27-34.
77. Carlisle S, Hanlon P. 'Wellbeing': A collateral casualty of modernity? *Social Science and Medicine* 2009;69:1556-60.

78. Carlisle S, Hanlon P. Well-being and consumer culture: A different kind of public health problem? *Health Promotion International* 2007;22:261-8.
79. 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.
80. Yule E. Scots Miserablism. In: GCPH Seminar (Series 6, lecture 3); 2010 16th February; Glasgow; 2010.
81. Durkheim E. *The division of labour in society*. New York: The Free Press; 1984.
82. Durkheim E. *Suicide*. New York: The Free Press; 1951.
83. Marx K. *Estranged Labour*. In: *Economic and Philosophic manuscripts of 1844*. New York: Dover publications; 2007.
84. Buckingham A. Is there an underclass in Britain? *The British Journal of Sociology* 1999;50:49-75.
85. Hills J, Brewer M, Jenkins S, et al. *Anatomy of economic inequality in the UK: report of the National Equality Panel*. London; 2010.
86. 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.
87. Marshall G, Roberts S, Burgoyne C. Social class and underclass in Britain and the USA. *The British Journal of Sociology* 1996;47:22-44.
88. Dean M. Christmas, the poor, and the development of a UK underclass. *Lancet* 1989;334:1536-7.
89. Donnelly P. Explaining the Glasgow effect: could adverse childhood experiences play a role? *Public Health* 2010;124:498-9.
90. *Breakthrough Glasgow: ending the costs of social breakdown*. London: The Centre for Social Justice; 2008.
91. Craig C. Women and children last. *Scottish Review of Books* 2010;6:4-6.
92. Hughes A. Working class culture, family life and domestic violence on Clydeside, c1918-1939: a view from below. *Scottish Traditions* 2002.
93. Hughes A. Representation and counter-representations of domestic violence on Clydeside between the two World Wars. *Labour History Review* 2004;69:169-84.
94. O'Neill R. *Experiments in living: the fatherless family*. London: Civitas; 2002.
95. *Breakdown Britain. Interim report on the state of the nation*. London: Social Justice Policy Group; 2006.

96. Rowthorn R, Webster D. Male worklessness and the rise of lone parenthood in Great Britain. *Cambridge Journal of Regions, Economy and Society* 2008;1:69-88.
97. *Social Trends 38*. London: ONS; 2008.
98. Hoghughi M. The importance of parenting in child health. Doctors as well as the government should do more to support parents. *BMJ* 1998;316:1545.
99. Barlow J, McMillan AS, Kirkpatrick S, Ghate D, Smith M, Barnes J. *Health-led Parenting Interventions in Pregnancy and Early Years*. London: Department for Children, Schools and Families and the University of Warwick; 2008.
100. Granovetter MS. The strength of weak ties. *American Journal of Sociology* 1973;78:1360-80.
101. Bell D, Blanchflower DG. *The Scots may be brave but they are neither healthy nor happy*. Bonn: Institute for the Study of Labor; 2005.
102. Stuckler D, King L, McKee M. Mass privatisation and the post-communist mortality crisis: A cross-national analysis. *Lancet* 2009;373:399-407.
103. *Sectarianism in Glasgow – final report*. Edinburgh: NFO Social Research; 2003.
104. Beckfield J, Krieger N. Epi + demos + cracy: Linking Political Systems and Priorities to the Magnitude of Health Inequities—Evidence, Gaps, and a Research Agenda. *Epidemiol Rev* 2009:mxp002.
105. Craig C. *The Scots' Crisis of Confidence*. Glasgow: Big Thinking; 2005.
106. 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.
107. Abbots J, Williams R, Sweeting H, West P. Poor but healthy? The youngest generation of Irish catholics in west Scotland. *Health Bulletin* 2001;59:373-80.
108. Mullen K, Williams R, Hunt K. Irish descent, religion and food consumption in the West of Scotland. *Appetite* 2000;34:47-54.
109. Abbots J, Williams R, Smith G. Association of medical, physiological, behavioural and socio-economic factors with elevated mortality in men of Irish heritage in West Scotland. *Journal of Public Health* 1999;21:46-54.
110. Williams R. Britain's regional mortality: a legacy from disaster in the Celtic periphery? *Social Science & Medicine* 1994;39:189-99.
111. Tudor-Hart J. The inverse care law. *Lancet* 1971;1:405-12.
112. Haynes R. Inequalities in health and health service use: evidence from the general household survey. *Social Science and Medicine* 1991;33:361-8.
113. Mercer S, Watt G. The inverse care law: Clinical primary care encounters in deprived and affluent areas of Scotland. *Annals of Family Medicine* 2007;5:503-10.

114. McLean G, Sutton M, Guthrie B. Deprivation and quality of primary care services: evidence for persistence of the inverse care law from the UK Quality and Outcomes Framework. *Journal of Epidemiology & Community Health* 2006;60:917-22.
115. Smith C. Equity in the distribution of health and welfare services: Can we rely on the state to reverse the 'inverse care law'? *Social Science and Medicine* 1986;23:1067-78.
116. Sutherland K, Coyle N. Quality in healthcare in England, Wales, Scotland, Northern Ireland: an intra-UK chartbook. London: Health Foundation; 2009.
117. Connolly S, Bevan G, Mays N. Funding and performance of healthcare systems in the four countries of the UK before and after devolution. London: Nuffield Trust; 2010.
118. McLaren J, Armstrong J, Harris R. Spending on health. Glasgow: Centre for Public Policy for Regions in association with KPMG; 2010.
119. MacKay D, Sutton M, Watt G. Deprivation and volunteering by general practices: Cross sectional analysis of a national primary care system. *BMJ* 2005;331:1449-51.
120. 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 and Medicine* 2007;65:1942-52.
121. Weich S, Lewis G, Jenkins S. Income inequality and self rated health in Britain. *Journal of Epidemiology and Community Health* 2002;56:436-41.
122. Households Below Average Income (HBAI) 1994/95-2007/08. London: Department for Work and Pensions; 2009.
123. Grainger S, Stewart S. Income distribution in Scotland. In: *Scottish Economic Statistics*. Edinburgh: Scottish Executive; 2007.
124. McLoone P, Boddy F. Deprivation and mortality in Scotland, 1981 and 1991. *BMJ* 1994;309:1465-70.
125. Turok I, Edge N. *The Jobs Gap in Britain's Cities: Employment Loss and Labour Market Consequences*. Bristol: The Policy Press; 1999.
126. Owen D, Gillespie A, Coombes M. 'Job shortfalls' in British local labour market areas: A classification of labour supply and demand trends, 1971-1981. *Regional Studies* 1984;18:469-88.
127. Waddell G, Burton A. *Is work good for your health and well-being?* London: TSO; 2006.
128. Collins C, McCartney G. Is a 'political attack' an explanation for the 'Scottish Effect' in health outcomes? Forthcoming in *International Journal of Health Services*.
129. Watt G. All together now: why social deprivation matters to everyone. *BMJ* 1996;312:1026-9.
130. McCormack C. *The Wee Yellow Butterfly*. Glasgow: Argyll Publishing; 2009.

131. Leon D, Chenet L, Shkolnikov V, et al. Huge variation in Russian mortality rates 1984-1994: Artefact, alcohol, or what? *Lancet* 1997;350:383-8.
132. Shkolnikov V, McKee M, Leon D. Changes in life expectancy in Russia in the mid-1990s. *Lancet* 2001;357:917-21.
133. Stuckler D, King L, McKee M. Mass privatisation and the post-communist mortality crisis: a cross-national analysis. *Lancet* 2009;373:399-407.
134. Birch K, Mykhnenko V. Varieties of neoliberalism? Restructuring in large industrially dependent regions across Western and Eastern Europe. *Journal of Economic Geography* 2009;9:355-80.
135. Gray N. The tyranny of rent. *Variant* 2010;37:37-43.
136. Boyle M, McWilliams C, Rice G. The spatialities of actually existing neoliberalism in Glasgow, 1977 to present. *Geografiska Annaler; series B, Human Geography* 2008;90:313-25.
137. Phillips J. *The industrial politics of devolution: Scotland in the 1960s and 1970s*. Manchester: Manchester University Press; 2008.
138. Howieson S, Hogan M. Multiple deprivation and excess winter deaths in Scotland. *The Journal of the Royal Society for the Promotion of Health* 2005;125:18-22.
139. Bowie N, Jackson G. *The raised incidence of winter deaths*. Occasional paper no.7. Edinburgh: General Registrar's Office for Scotland; 2002.
140. Gemmell I, McLoone P, Boddy F, Dickinson G. Seasonal variation in mortality in Scotland. *International Journal of Epidemiology* 2000;29:274-9.
141. Wilkinson P, Pattenden S, Armstrong B, et al. Vulnerability to winter mortality in elderly people in Britain: Population based study. *BMJ* 2004;329:647(doi:10.1136/bmj.38167.589907.55).
142. Gillie O. *Scotland's health deficit: An explanation and a plan*. London: Health Research Forum Publishing; 2008.
143. Hypponen E, Power C. Hypovitaminosis D in British adults at age 45y: Nationwide cohort study of dietary and lifestyle predictors. *American Journal of Clinical Nutrition* 2007;85:860-8.
144. McDowell I. From risk factors to explanation in public health. *Journal of Public Health* 2008;30:219-23.
145. Coggon D, Martyn C. Time and chance: The stochastic nature of disease causation. *Lancet* 2005;365:1434-7.
146. McPherson K. Wider "causal thinking in the health sciences". *Journal of Epidemiology and Community Health* 1998;52:612-8.

