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Political economy and population health: from theory to an empirical assessment of the impact of austerity on mortality trends

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Submitted in fulfilment of the requirements of the Degree of PhD

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November 2022

Dedication

This thesis is dedicated to all people whose lives have been cut short as a result of government policy that was designed to increase the wealth and power of the privileged, when it should instead have been designed to achieve ecological sustainability, equity, health and social outcomes.

Author's declaration

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

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Signature:

Abstract

The life expectancy of most high income countries had increased consistently over the second half of the 20th Century. However, after around 2012 these improving trends stalled in many countries. This thesis seeks to explain these trends through a political economy lens.

There are three central arguments made in this thesis. First, that health is best defined as a structural, functional and emotional state that is compatible with effective life as an individual and as a member of society; and that health inequalities are best defined as the systematic, avoidable and unfair differences in health outcomes that can be observed between populations, between social groups within the same population, or as a gradient across a population ranked by social position.

Second, that political economy is crucial to understanding the health of populations. The existing evidence linking political economy to population health was systematically reviewed and synthesised. Although there were risks of bias, social democratic welfare states, higher public spending, fair trade policies, extensions to compulsory education provision, microfinance initiatives in low-income countries, health and safety policy, improved access to health care, and high-quality affordable housing were found to have positive impacts on population health. 'Neoliberal' restructuring was associated with increased health inequalities. Higher income inequality was associated with lower self-rated health and higher mortality.

Third, countries with more austere policy in recent decades experienced slower improvements in mortality, especially when measured as changes in Government Expenditure and Public Social Spending, and when implemented during economic downturns, albeit with imprecise effect estimates. The stalled mortality trends observed across many high income countries is therefore likely to be at least partly due to austerity policies.

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Publications from thesis

McCartney G, Popham F, McMaster R, Cumbers A. Defining health and health inequalities. *Public Health* 2019; 172: 22-30, <https://doi.org/10.1016/j.puhe.2019.03.023>.

McCartney G, Hearty W, Arnot J, Popham F, Cumbers A, McMaster R. Impact of Political Economy on Population Health: A Systematic Review of Reviews. *American Journal of Public Health* 2019; 109: e1_e12, <https://doi.org/10.2105/AJPH.2019.305001>.

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Acknowledgements

I am grateful to NHS Health Scotland who agreed to fund my PhD and give me time to work on it, to Public Health Scotland for honouring this commitment, and to the University of Glasgow who had faith in me to complete my studies whilst an employee.

I have benefited from the expertise, wisdom and counsel of four supervisors over my period of study: Robert McMaster, Ruth Dundas, Frank Popham and Andy Cumbers. I am particularly grateful to Frank for his patience in correcting my R code and providing solutions to the endless problems I presented him with. Prospective PhD students would do well to find supervisors as helpful as they have been.

I have had many co-authors and colleagues who I have worked with on the specific outputs of this thesis, and on the wider endeavour to understand the reasons for the stalled mortality trends, and, crucially, to catalyse action to improve these trends once again. I would therefore like to acknowledge the contributions of my co-authors for work contributing to this thesis: Frank Popham, Robert McMaster, Andrew Cumbers, Wendy Hearty, Julie Arnot, Lynda Fenton, Jon Minton, Colin Fischbacher, Martin Taulbut, Kirsty Little and Ciaran Humphreys. Other colleagues have been important collaborators on other work in relation to the stalled mortality trends that has been important in developing my thinking and research in this area: David Walsh, Phil Broadbent, Paige Somerville, Justine Fitzpatrick, Allan Baker, Jim McMenanim, Josie Murray and Rebecca Campbell.

There are many people around the world who contribute their labour without pay to the greater good by writing, sharing, peer-reviewing and offering advice in overcoming the bugs in R code and R packages. I will never know them, but I couldn't have done this work without them.

Similarly, I used data from surveys and routine mortality records that were compiled diligently by people working for statistical agencies around the world. Without those workers, and the members of the public who responded to those surveys, none of this research would be possible. I also want to acknowledge Pat Thomson's blog (www.patthomson.net) which has been an invaluable guide throughout the work of the thesis, and one I recommend wholeheartedly to PhD candidates across disciplines.

I would like to thank the following people/organisations for granting permission to reproduce Figures in this thesis: Elsevier for Figures 3.1 and 3.6; Göran Dahlgren for Figure 3.2 ; Finn Diderichsen for Figure 3.3; Orielle Solar for Figure 3.4; and Nancy Krieger for Figure 3.6. Special thanks is also due to Veronica Toffolutti who checked and corrected earlier published analyses of AAFI and mortality that she led on, and then shared code to allow for cross-checking between my results and hers.

My work has been subject to anonymous and open peer review which has improved the quality of my work and provoked me to think more deeply and to improve. I am deeply grateful to all those who undertake peer review and who are rarely thanked or recognised.

I also wish to thank my three wonderful children, Eilidh, Daniel and Sarah for their patience and irreverence, and Wendy for her love, support and encouragement through the more difficult times.

List of abbreviations

AAFI	Alesina-Ardagna Fiscal Index
AMSTAR	Assessing the Methodological Quality of Systematic Reviews
ASMR	Age-Standardised Mortality Rate
CAPB	Cyclically-Adjusted Primary Balance
ESP	European Standard Population
GDP	Gross Domestic Product
GDPpc	Gross Domestic Product per capita
HMD	Human Mortality Database
ILO	International Labour Organisation
IMF	International Monetary Fund
OECD	Organisation of Economic Co-operation and Development
ONS	Office for National Statistics
RII	Relative Index of Inequality
SRH	Self-Rated Health
SII	Slope Index of Inequality
UK	United Kingdom (of Great Britain and Northern Ireland)
UN	United Nations
USA	United States of America
UAE	United Arab Emirates
WHO	World Health Organisation

1. Introduction

1.1 The problem of stalled mortality rates

Mortality rates in high income countries, with a few specific exceptions, have improved since the start of the agricultural revolution (Floud et al., 2014). The exceptions to this are important. Pandemics, war, and the rapid transition of the Eastern Bloc countries to a particular form of capitalism all created periods of increasing mortality (McCartney et al., 2011; Shkolnikov et al, 2001).

However, in the decade prior to the COVID-19 pandemic, the rate of mortality improvement has dramatically slowed across many high income countries. In fact, for some countries such as the USA and (on some measures) the constituent nations of the UK, mortality has increased in some years in the last decade (Fenton et al., 2019a; Ho & Hendi, 2018; Raleigh 2019). The cause(s) of this change in trend is disputed and politicised, with a marked absence of clear and unambiguous recommendations from public health bodies as to what should be done. A key debate, and limiting factor in achieving clear policy recommendations to sufficiently address the stalled mortality trends, has been the extent to which it is agreed that austerity policies are the principal cause (PHE, 2018; Hiam et al., 2018; Hiam et al., 2017; Baker et al., 2018; Newton et al., 2017).

The lay definition of austerity, particularly in the UK, has been derived from the policies of the Conservative-Liberal Democrat coalition government that came to power in 2010. The coalition argued that a period of austerity was required to reduce government debt to boost economic growth and make the public finances “sustainable” (HM Treasury, 2010). This meant, for most of the public, the imposition of a series of spending cuts, most notably on social security benefits for working-age adults and for local government

(Reed & Portes, 2018). However, for economists the definition of austerity is more specific. Austerity can be defined as reductions in government spending, and/or increases in taxation, after accounting for changes in spending or taxation that are related to changes in economic growth rates (Blyth, 2013; Konzelmann, 2016). However, even within this definition there are many possible variants. First, there may be different impacts depending on whether austerity is implemented mostly by way of government spending reduction or through tax rises. Second, the impact of these changes may differ depending on which population groups are affected (e.g. whether tax rises are implemented on richer or poorer groups, or whether spending cuts are implemented on areas or services that have unequal impacts). Finally, the impact may be different depending on when in the economic cycle it is implemented, in particular whether it is implemented during a period of economic growth (often to pay down government debt) or during an economic downturn (Alesina et al., 2019a; Guarjardo et al., 2011; Blyth, 2013; Perotti, 2013).

Austerity has come to be associated with some countries of the European Union which were perceived to be overspending following the deep recession from 2008. Greece in particular, but also Italy, Ireland, Portugal and Spain were identified as having had austerity policies imposed upon them by the European Commission (EC), the European Central Bank (ECB) and the International Monetary Fund (IMF), resulting in a combination of government spending cuts, privatisations, asset sales and tax rises (Varoufakis, 2017). As such, a series of studies was undertaken shortly after the recession to examine the impact of austerity, focusing in particular on these countries (Stuckler & Basu 2013; McKee et al., 2012; Stuckler et al., 2011; Stuckler et al., 2010; Stuckler et al., 2009a). The results of these studies were then subsequently used in comparison to the experience of the UK and USA in discussions of the role of austerity on mortality trends. Yet, very few of these studies included empirical measures of austerity or data from the period after 2010 when the stalled trends became evident. In

short, this left a gap in the evidence base which left sufficient uncertainty that clear public health messages did not arise.

1.2 Warrant for the thesis

As the lead for the Scottish Public Health Observatory between 2010 and 2021, it has been my task to describe and explain the trends in health and health inequalities to policymakers, practitioners and the public. As noted above, the stalled trends in mortality rates have arguably been the greatest public health challenge faced in Scotland, and across most high income countries, for many decades. The lack of clear guidance from public health for policymakers to act has been a failure which is likely to have led to unnecessary mortality and morbidity. One crucial factor in this failure has been the debate over the role of austerity policies in causing these trends, stemming from a lack of robust empirical studies and a politicised context for public health employees to work in.

This thesis builds towards an empirical analysis, for the first time, of the extent to which a range of measures of austerity can explain the recent trends in mortality rates in high income countries, directly addressing this knowledge gap.

1.3 Structure of the thesis

This thesis begins by defining health, health inequalities and population health (Chapter 2). Using a structured review of the literature and critical appraisal of different existing definitions, it exposes the wide range of underlying assumptions and values that percolate the literature on health. The tensions between different definitions are exposed and a justification for new, adapted, definitions is made.

Having established definitions of the outcomes of interest, Chapter 3 summarises the existing theories that explain the health of populations. There is a long historical and academic record of such theories which have guided empirical studies over time. Common to most modern theories of

population health is the role of political economy (although not always termed as such). Following Adam Smith (1982), a political economy approach takes a broad and contextualised view of the economy, in which the economy is understood as a socially embedded array of provisioning institutions in which people attempt to address their needs and wants. As such, the study of political economy acknowledges evolutionary change, emergent properties, power relations, geographical and historical contingencies, and that individuals and institutions are mutually constitutive (Dow et al., 2018; Hodgson 2001). This builds upon the public health traditions and literature concerning the social and commercial determinants of health, but takes this further by explicitly incorporating political economy, including power and social class relationships, politics and institutions. By taking a political economy perspective, and through adaptation of existing population health theories, a new framework for understanding the political economy of population health is proposed.

Chapter 4 then takes the theoretical framework for the influence of political economy on population health and systematically reviews the literature (through a systematic review of reviews) to provide a robust overview of the empirical evidence for these relationships. This review details the areas in which there is already a robust evidence base, and where there is an absence of synthesised, reviewed evidence. Note that a systematic review on the narrower question of the relationship between austerity and population health (from existing studies) has also been undertaken, but as it has been led by a colleague it is outside the scope of this thesis.

Having established the importance of political economy to population health, theoretically and from the empirical literature, the problem of the stalled mortality trends is then described in detail in Chapter 5. Here the extent of the stalling across time and space, its epidemiological features, and the evidence for different hypothesised causes of the changed trends, are all summarised.

Chapter 6 provides the methods for the subsequent empirical analyses. It details a pre-analytical protocol which was developed to reduce the risks of bias in the approaches taken, but also the changes in methods that were adopted as the structure of the available data became known. This chapter also provides detail on the derivation of the four different measures of austerity used in this thesis, and the rationale for each.

Chapter 7 gives a detailed descriptive analysis of the trends in the austerity exposures and of each of the mortality-derived outcome measures. This chapter also describes the timing of the change in trends across countries.

The final substantive chapter (Chapter 8) then provides the results of the empirical analyses of the impact of austerity on mortality-derived outcomes. A series of sensitivity analyses are also undertaken, some of which were pre-specified, others were deemed necessary as the structure of the data became apparent.

This is then followed by a concluding chapter (Chapter 9) which summarises the key findings of the thesis, the strengths and weaknesses of the approaches taken, and the implications for future research, policy and practice.

2. Defining health and health inequalities

2.1 Chapter synopsis

This chapter will discuss the variety of ways in which health and health inequalities have been defined in the literature, and the key features of these definitions. A critical appraisal of these features on the basis of the utility, strengths, weaknesses and parsimony is then undertaken to inform a synthesis of existing definitions. This process is undertaken initially to create a definition of health, and then to create a definition of health inequalities.

Note that the work in this chapter was published in the peer reviewed literature as part of this programme of postgraduate study:

McCartney G, Popham F, McMaster R, Cumbers A. Defining health and health inequalities. *Public Health* 2019; 172: 22-30, <https://doi.org/10.1016/j.puhe.2019.03.023>.

2.2 Background

'Health' is a term that is widely used but which is subject to differing interpretations and meanings. For many economists health is functional and reduced to a form of 'capital' ('health capital') which allows people to participate in the labour force as an input to production (Schneider-Kamp, 2021; Grossman, 1972). Within the capabilities tradition health is seen as a 'resource for living' that facilitates the participation and functioning of people within society (Sen, 1999). This remains a conceptualisation of health as a form of capital, but one which is needed by people to live well. Others, including the World Health Organisation (WHO), define health in terms of an outcome - a desirable state that people can experience (WHO, 2006). There is a further complexity in relation to how the distribution of health within and between populations is conceptualised and defined. Terms such as 'health inequalities', 'health inequities', 'health disparities' and 'health variations' have often been used to describe similar phenomena, but with important differences (Braveman et al., 2017).

Public health research and policy action should be built upon a shared definition and understanding of health and health inequalities, because of the influence this has on how these concepts are measured, the analyses undertaken, and the interpretations drawn as a result (Krieger, 2011). Furthermore, there is a risk that the underlying assumptions, emphasis and values for different uses of the terms are obscured by a lack of a common definition. In turn, this could lead to researchers in different disciplines and traditions talking at cross-purposes, and misunderstandings between researchers and policymakers.

2.3 Methods

The Embase and Medline databases were searched without time limits, but limiting to studies published in English on human subjects. The following terms were searched for in the article titles: ‘definition\$’ OR ‘glossary’; AND, ‘health’ OR ‘inequ\$’. A similar search was performed in Google to identify relevant grey literature. A total of 671 citations after de-duplication were identified in the research databases of which 30 were screened as potentially relevant. Sixteen additional citations were identified from my own collection and the grey literature. All these papers were then obtained in full text and read for relevance to the research question, in particular whether they proposed a relevant definition. The key features of each of the definitions were extracted and tabulated iteratively such that any new features from subsequent definitions were added to the list, and any similar features integrated. Each of these key features were then critically appraised, using the logic and argumentation presented for each of the definitions by the original authors. In this way the case and for and against particular features of definitions were drawn out. Using explicit reasoning, new definitions synthesising the most useful aspects of existing definitions, were then created (McCartney et al., 2019a).

2.4 Results

WHO definitions of health and their critiques

The most widely used definition of health is that originally published by the WHO in 1948 (and republished in 2006) which described health as,

“...a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity” (WHO, 2006, p.1).

The 1986 Ottawa Charter, in which the WHO articulated its health promotion agenda, expanded upon this definition as follows:

“Health promotion is the process of enabling people to increase control over, and to improve, their health. To reach a state of complete physical, mental and social well-being, an individual or group must be able to identify and to realize aspirations, to satisfy needs, and to change or cope with the environment. Health is, therefore, seen as a resource for everyday life, not the objective of living. Health is a positive concept emphasizing social and personal resources, as well as physical capacities. Therefore, health promotion is not just the responsibility of the health sector, but goes beyond healthy life-styles to well-being” (WHO, 1986, p.1).

These WHO definitions have three important dimensions. First, they emphasise health as a positive phenomenon rather than as the absence of illness or disability. Second, they describe health as being multidimensional. Finally, they articulate a high aspiration for health by describing the experience of health as being one when the achievement across dimensions is ‘complete’.

These definitions have, however, been the subject of a range of critiques. Some have argued that the WHO definition conflates health with happiness and well-being. Instead, it is argued that these are actually separate dimensions that can vary independently of health (Huber et al., 2011; Saracci, 1997). For example, it is argued that positive and negative dimensions of mental health can co-exist, and that it is possible for people to experience mental illness (e.g. schizophrenia) concurrently with positive health (e.g. well-being or happiness) (Tennant et al., 2007).

Another important debate in relation to the WHO definition is its aspirational nature. Although some support this approach, “...using the term ‘euxia’ to describe an ‘optimal’ health-fitness standard characterised by physical vigour, long lifespan and freedom from chronic disease” (Elrick, 1980), the counter case is that it makes the attainment of health near impossible (Huber et al., 2011; McGrail et al., 2016).

In response, some alternative health definitions have attempted to reduce or remove this aspirational (or absolute) aspect. For example, Starfield proposed that:

“[health is] the extent to which an individual or group is able, on the one hand, to realise aspirations and satisfy needs and, on the other hand, to cope with the interpersonal, social, biological, and physical environments. Health is therefore a resource for everyday life, not the objective of living; it is a positive concept embracing social and personal resources as well as physical and psychological capacities” (Starfield, 2001, p.453).

An even less aspirational definition was proposed by Leonardi, who argued that health is simply:

“...the capability to cope with and to manage one’s own malaise and well-being conditions” (Leonardi, 2018, p.742).

Another approach, which is still compatible with an aspirational approach, emphasises instead that health is a continuum in which people sit at different points on a scale:

“Health is the experience of physical and psychological well-being. Good health and poor health do not occur as a dichotomy, but as a continuum. The absence of disease or disability is neither sufficient nor necessary to produce a state of good health” (Card, 2017, no page number).

These proposed amendments avoid the binary and absolutist difficulties of the WHO definitions and instead introduce an analogous concept (‘the extent to which’) based on the realisation of aspirations, the ability to satisfy needs and to cope with a range of environments. The clear advantage of this alternative approach is that health becomes contextually defined and thus can evolve over time (e.g. with increasing life expectancy the expectation and definition of a healthy lifespan might increase).

However, it can mean that poor health within any particular population might be underestimated or overlooked because it is only defined in relation to local or socially bounded norms (Popham, 2016; Popham, 2014). Krieger (2012) has discussed this in depth in relation to who counts (and is counted) within a population, and what that means for how comparisons are made and what aggregate health statistics show.

Population and individual health

Rose (1985) drew a clear distinction within epidemiology between the causes of cases, and the causes of prevalence in a population. By highlighting the importance of understanding the unit and level of analysis (linking to the risks of the ecological and atomistic fallacies wherein findings at one unit of analysis are inappropriately applied to a different level of analysis), the importance of clarifying the level at which health is defined (i.e. is it a population or individual phenomenon) is emphasised.

Indeed, some researchers have made the case that health is a collective condition with the property of a public good, i.e. whereby the enjoyment of it by one person does not diminish its use by others:

“Health is a condition in which people achieve control over their lives due to the equitable distribution of power and resources. Health is thus a collective value; my health cannot be at the expense of others, nor through the excessive use of natural resources” (Scott-Samuel, 2011).

However, this latter definition, through its focus on achieving control and its description of health as a collective value, may preclude an adequate lens through which to understand different individual experiences of health within a population. It may be better to have a definition which allows discussion of both the health and determinants of health for both populations and for individuals (Rose, 1985). For example, it would be possible to have a high degree of control over one’s life yet die prematurely, because control may be a cause of incidence but not of cases

within a population. It also limits the definition of health to that which is obtained through the equitable distribution of power and resources, which are not necessarily the only routes through which health can be achieved. Similar limitations apply to the suggested definition by the International Union for Health Promotion and Education (IUHPE) which defines health in terms of its determinants (power and control over life, and where needs and rights are supported):

“Health is created when individuals, families, and communities are afforded the income, education, and power to control their lives; and their needs and rights are supported by systems, environments, and policies that are enabling and conducive to better health” (Shilton et al., 2011).

Last’s dictionary of public health offers two alternative definitions of health that have merit (Last, 2001, p.81):

“A sustainable state of equilibrium or harmony between humans and their physical, biological and social environments that enables them to coexist indefinitely”;

and,

“A structural, functional, and emotional state that is compatible with effective life as an individual and as a member of family and community groups”.

The former of these definitions derives from an ecological perspective whereby health is dependent on its sustainability and its inter-relation with the surrounding environment (similar to Charlier et al. (2017)). The attraction of this definition is that a longer-term perspective is adopted, and it avoids a purely anthropocentric approach. However, it fails to provide a conceptualisation of health that describes the experience of health: it is possible to be in equilibrium at a level of health that is low (or characterised by illness and disease). It is also interesting that it defines it

in such a way as to suggest that it may not be akin to a 'public good' in that the achievement of health may be at the expense of others (both human and other species).

The latter definition offered by Last contains the multi-dimensional components of the earlier WHO definition, including an experiential element that is missing from many of the proposed definitions, but avoids an absolutist position of health having to be a 'complete' state.

Furthermore, this definition relates health to the ability function and to participate socially (which is also common in many definitions of poverty).

Table 2.1 provides a summary of the common features and themes of the definitions described above. This approach is similar to that of Leonardi who identified nine features by which health should be defined (Leonardi, 2018). Defining health by the achievement of an absolute standard rather than a context specific one is contested. However, the disadvantages of a purely contextual definition is that causes of better or worse health within populations can only be uncovered through comparison, and this would not be possible if health was not defined to a common standard. For this reason, avoiding a definition that follows a purely context-specific approach is preferable. However, this does not necessarily mean that health needs to be defined aspirationally such that people cannot be defined as healthy if they do not meet an 'ideal' standard, but there is a tension with adopting a common standard for comparison.

Another difference between definitions is whether health should be defined as something people experience and an end in itself, or whether health should instead be defined in terms of the capacity it gives people to function and participate in society (Bircher, 2005). Some of the proponents of the former are at risk of ignoring the importance of being healthy in order to be a social being and to participate; whilst some proponents of the latter are at risk of reducing health merely to a factor of production in the economy. A more balanced perspective might recognise the value of both.

Clearly health is a state of being that is experienced - to be in pain,^a or to enjoy positive mental health is real and important. However, the capacity that health provides to participate and function is also essential and provides a contextualisation of how health is a relative phenomenon.

As noted above, some have proposed that health should either defined by its determinants, by the control people have over their lives, or by the extent to which it is sustainable (both in terms of the sustainability of health and how this is interdependent on environmental sustainability). Although each of these are important issues, it is not useful to define health by its causes as this can confuse cause and effect and create a circular logic. It is however useful to have a definition which incorporates the different dimensions of health, including physical and mental health, and which is applicable to both individuals and populations.

^a Davis & McMaster (2017, p.63) note that: "...the International Association for the Study of Pain focuses their definition on "unpleasant sensory and emotional experience" associated with actual or potential damage to tissue; thereby somewhat de-emphasizing Saunders' emphasis on spiritual, social, and existential sources. This contrasts with the American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders, which explicitly recognizes a relationship between sensory and existential factors".

Table 2.1 - Features of different health definitions

Feature	Sources	Commentary
Health is achievement of a common standard.	(WHO, 2006)	Some define health as the achievement of a defined (aspirational) standard, whilst others describe a more analogue scale whereby health can be achieved to a greater or lesser extent (and possibly with lower expectations given contextual and personal circumstances). For epidemiological study, a common definition that is not context specific can help identify exposures which create limits on the experience of positive health which might otherwise be ignored.
Health is achievement of an 'ideal' outcome.	(WHO 1986; WHO 2006)	The definitions of health which categorise people into healthy or not on the basis of whether they have achieved a 'complete' state of health or wellbeing are good for recognising aspiration and potential. However, they may not recognise that people can see themselves as healthy whilst living with some forms of disability or conditions, and they may not recognise the process of 'healthy ageing' whereby some loss of functionality may not represent a loss of health.
Health is experiential.	(Card, 2017)	The experience of positive or negative health as an experience in and of itself (i.e. separate from the capacity this may provide to function or participate in the economy or society) is not a ubiquitous feature of definitions. Some argue that it is not the experience of health that matters (or indeed that can be defined), but instead the capacities it provides which are important. Clearly, the two are linked and it is difficult to envisage a scenario whereby negative health is experienced without capacity being reduced. However, this may reduce the human experience to an overly functional or mechanistic phenomenon (or even to reduce health to the ability to be productive in society) and therefore undermine the experience and value of health for its own sake.
Health is the ability to function and participate.	(WHO, 1986; Starfield, 2001)	Some define health solely on the (in)ability to participate in society (otherwise framed as a resource for living or the ability to 'function'), whilst others include this as an essential component alongside the physical and mental aspects. Defining health narrowly on the basis of participation in society means that experiential elements (pain, low mood, etc.)

	(Leonardi, 2018; Last, 2001)	are only relevant to the extent that they impact on the ability to participate. The advantage of including this aspect is that health is recognised as a contextualised phenomenon in which the extent to which a society enables and includes (for example) people with particular disabilities influences the experience of health.
Health is defined by its determinants.	(Shilton et al., 2011)	Without a definition of the outcome or experience of health, defining health by its determinants alone is imprecise and unsatisfactory. For example, if health is determined by adequate income, all outcomes that are due to adequate income would constitute 'health'. This would be too broad a definition to be useful. In this way such definitions of health are better covered within a theoretical framework of health causation than in a definition of health.
Health is an individual <i>and</i> population phenomenon.	(Starfield, 2001)	Some definitions focus only on health as a population phenomenon but this restricts its applications.
Health is a multi-dimensional phenomenon.	(WHO, 2006; WHO 1986; Card 2017)	This recognises the holistic nature of the experience of health. Most recent definitions of health recognise the physical and mental components of health and so this is uncontentious.
Health is defined by the control people have over their lives.	(WHO, 1986; Scott-Samuel, 2011)	Health is clearly a resource which determines the control people have over their lives, their ability to realise expectations and to satisfy needs; but it is not the only determining factor (for example, the political and socio-economic context are also very important).
Health has to be sustainable.	(Scott-Samuel, 2011; Last, 2001)	Some definitions of health focus largely, or entirely, on its sustainability. However, this confuses the outcome of interest (health) with the processes through which health is determined.

Taking all these factors into account, it is argued here that the best available definition is that used by Last. However, to make the definition more parsimonious, it is proposed that it should be amended slightly such that health is defined as:

A structural, functional and emotional state that is compatible with effective life as an individual and as a member of society.

Definitions of health inequalities

Health experiences can vary widely between different individuals and groups. Much of the difference in health outcomes between individuals is due to chance (Coggon & Martyn, 2005; Smith, 2011). Nonetheless, the systematically different outcomes for groups that share common characteristics, and the changes over time in the health of populations, is both the substrate for public health research (by facilitating the research into why some people experience different health outcomes than others), and the purpose of public health action (to improve health and health inequality outcomes) (Krieger, 2012). Like health, health inequalities have been defined in many different ways.

At the outset it is important to recognise a particular continental difference in the lexicon. In the Americas, it is common to use health inequalities to refer to variations or differences between groups that are not necessarily unfair, such as might be the case if elderly people are more likely to die than young adults (Arcaya et al., 2015).^b Health inequity is the term used, and linguistically most correctly to define unfair differences where there is an issue of social (in)justice (Krieger, 2001a). However, in Europe, the term health inequity is not used routinely and the term ‘health inequalities’ is used instead (Krieger, 2011). Further confusion can arise with the use of the term ‘health disparities’ which has been defined either as simple

^b Note that this does not preclude the possibility of intergenerational unfairness and inequalities.

differences between groups or differences after accounting for a variety of other explanations (Fink, 2009).

It is also worth noting that the mean health of a population is often dependent on the extent to which there is inequality in health outcomes within that population. This is demonstrated by showing that populations with the greatest lifespan variation also have the highest mean mortality rates (Popham et al., 2013). If the differences between ranked groups are considered in terms of the simple difference (i.e. subtraction of one from another) between or across groups, this is termed the absolute inequality (even though it is a difference of one or more groups relative to another). Alternatively, the difference can be considered as a ratio (i.e. one divided by the other), and this is termed the relative inequality. This is important because, on a declining mean trend it is frequently the case that the absolute inequality decreases at the same time as the relative inequality increases (Blakely et al., 2017). This is not only an arithmetical phenomenon, but the importance put on relative and absolute measures also raises a question of values. It is further complicated that, with the same data, a trend can be increasing or decreasing depending on whether it is presented as a positive or negative measure (i.e. life expectancy or mortality) (Kjellsson et al., 2015). With these issues in mind, the definitions identified are now reviewed.

A definition used in a prominent WHO report from 1990 stated that health inequalities can be defined as follows:

“Social inequities in health are systematic differences in health status between different socioeconomic groups. These inequities are socially produced (and therefore modifiable) and unfair” (Whitehead, 1991, p.2).

The key components of this definition are that the differences of interest are in health outcomes; that the differences occurring between social groups are systematic rather than random; and that they have to be

understood at a population rather than individual level. Finally, it is explicit that these differences are avoidable.

A similar, if more perfunctory, definition has been offered by Graham (2009), but omits reference to their avoidability:

“Health inequalities ... are the systematic differences between more and less advantaged groups” (Graham, 2009, p.3).

In a more extensive definition, Krieger defines *social inequalities in health* as:

“...health disparities, within and between countries, that are judged to be unfair, unjust, avoidable, and unnecessary (meaning: are neither inevitable nor unremediable) and that systematically burden populations rendered vulnerable by underlying social structures and political, economic, and legal institutions” (Krieger, 2001a, p.698).

This adds three additional components to the definition. First that the systematic differences between populations are unfair or unjust, and in the surrounding text to the definition given here, the necessity of taking action to redress the injustice is made clear. Second, that the inequalities are a result of underlying social structures and institutions. Third, that the differences are avoidable and can be changed (in common with other authors) (Arcaya et al., 2015). The extent to which a health outcome is understood as avoidable or remediable also changes over time. Disease processes that in the past were either misunderstood, not appreciated and for which no effective preventative or treatment measures were available, have often subsequently become avoidable, preventable or treatable. As such, what is defined as an inequality can also change. Furthermore, even when a disease process is poorly understood, if other populations have a lower burden of that disease, it suggests that it is avoidable and treatable and therefore represents an inequality.

A quite different approach to defining health inequalities has been taken by other authors. For example, Kawachi and colleagues define health inequalities as:

“a term used to designate differences, variations, and disparities in the health achievements of individuals and groups” (Kawachi et al., 2002, p.647).

The only common feature between this definition and the others is the interest in differences in health outcomes, and the other definitional aspects are all either implicitly or explicitly contested (Krieger, 2001a).

A further alternative approach has been proposed by Fleurbaey and Schokkaert (2009), which defines (health) inequalities as only being unfair when:

“...they follow from causes which do not belong to the sphere of individual responsibility”.

This is operationalised by proposing a method for adjusting health outcome differences for a range of ‘lifestyle’ factors to isolate the inequalities that are argued to be unfair (on the basis that ‘lifestyle’ factors are based on individual choices rather than differences in opportunity). The immediate and obvious problem with this is the assumption that ‘lifestyle’ factors and ‘choices’ are independent of social background (and can thereby be adjusted away to reveal ‘equality of opportunity’).

This thesis has been discussed extensively, and was a prominent component of the Black and Whitehead reports in 1980 and 1987 respectively (Black et al., 1988). The evidence has since been updated and revised, with the same conclusions reached (Marmot et al., 2008). In short, there is good evidence that ‘lifestyle factors’ and individual agency are very closely shaped by social background, including parental background, early years experiences, education, etc. (Jones et al., 2011; McCartney et al., 2013; Power & Matthews, 1997; Phelan et al., 2010; Scott et al., 2013). As such, the ethical

assertion that individual ‘lifestyles’ can be separated from social background seems to be at best unsafe, and weak basis for justifying this distinction.

Related to the definition of health inequalities, but framed positively, Braveman and colleagues have provided a range of definitions of ‘health equity’ with varying brevity and with versions for general and technical audiences (Braveman et al., 2017). The most detailed definition for a general audience they offer is:

“Health equity means that everyone has a fair and just opportunity to be as healthy as possible. This requires removing obstacles to health such as poverty, discrimination and their consequences, including powerlessness and lack of access to good jobs with fair pay, quality education and housing, safe environments, and health care” (Braveman et al., 2017, p.2).

Two further versions for a general audience are also offered, depending on whether health equity is defined as an outcome or process:

“Health equity means that everyone has a fair and just opportunity to be as healthy as possible” (Braveman et al., 2017, p.2).

“Health equity means removing economic and social obstacles to health such as poverty and discrimination” (Braveman et al., 2017, p.2).

And the definition for a technical audience is:

“For the purposes of measurement, health equity means reducing and ultimately eliminating disparities in health and its determinants that adversely affect excluded or marginalised groups” (Braveman et al., 2017, p.2).

The criteria that Braveman et al. argue that the definition should:

“Reflect a commitment to fair and just practices across all sectors of society; be sufficiently unambiguous that it can guide policy priorities; be actionable; be conceptually and technically sound, and consistent with current scientific knowledge; be possible to operationalise for the purpose of measurement, which is essential for accountability; be respectful of the groups of particular concern, not only defining the challenges they face but also affirming their strengths; resonate with widely held values, in order to garner and sustain broad support; and, be clear, intuitive, and compelling without sacrificing the other criteria, in order to create and sustain political will” (Braveman et al., 2017, p.3).

Missing from all the Braveman et al. definitions is an explicit recognition that for ranked social groups, such as occupational social class or income, the inequalities in health can be seen to occur stepwise as a gradient across the entire population. This gradient cannot be described where the social groupings are not rankable (e.g. gender or ethnicity), but it is (arguably) an important feature of health inequalities to capture in the definition because all social groups with the exception of the most advantaged within a society are negatively affected (Wilkinson & Pickett, 2009), and a failure to recognise this can make the phenomenon less relevant for the majority of the population and/or tend to feed a narrative of ‘othering’. Moreover, if the most advantaged within any particular society were to compare themselves within similarly advantaged groups in other societies, they may also find that they do less well. Wilkinson and Pickett (2009) have suggested that this is the case within the most unequal societies.

Norheim and Asada (2009) make the point that definitions of health inequality should recognise that equality should not necessarily be prioritised over the overall level of health in the population or other social goods such as education. Although this may be the case, it could be argued that that is a question of priorities and values rather than of definition.

Table 2.2 summarises the key features proposed in the different definitions of health inequalities. Although all definitions start from the point of describing a difference in health between groups, only some are explicit that the differences of interest are systematic and non-random. More contested is whether the definition should state that the differences between groups are avoidable and unnecessary, or whether they are unfair. Given that health inequalities have varied over time and between populations, and that their causes are due to class and political economy (McCartney et al., 2013), it seems important to state their systematic, avoidable and unfair nature, and that they arise between social groups who occupy different positions of power in society. As social groups may or may not be rankable, as with social class and gender, a definition needs to be able to describe both forms of inequality. Finally, some definitions seek to define health inequalities by their causes. This confuses cause and effect and therefore this approach has been avoided.

Table 2.2 - Features of different health inequality definitions

Feature	Sources	Commentary
Differences in health are the outcomes of interest.	All	This is the only aspect that is common across all of the definitions.
Differences in health are systematic and not random.	(Whitehead & Dahlgren, 1991; Graham, 2009; Krieger, 2001a)	That the differences in health are systematic is important because it indicates that the health outcomes are due to some causal forces which cannot be explained by random variation.
The differences are avoidable and unnecessary.	(Whitehead & Dahlgren, 1991; Krieger, 2001a)	This is a more contentious part of the definition and makes clear that the observed differences require political attention. It is also helpful, however, in focussing on aspects of health which are genuinely due to injustice. For example, differences in the prevalence of dementia between age groups would not necessarily be deemed an injustice (although differences in medical research funding for dementia as opposed to heart disease might be). This definition does not entirely protect against claims that some observed differences are unavoidable (as has been wrongly claimed in the past in relation to racial differences in health), but it does emphasise the need for such claims to be justified.
The differences are unfair and unjust.	(Whitehead & Dahlgren, 1991; Krieger, 2001a)	This aspect naturally flows from defining health inequalities as being systematic and avoidable and in some ways should not be necessary in the definition. However, stating that the differences in health outcomes are unfair and unjust makes clear that they are important and require political action.

<p>The differences are observed between different social groups.</p>	<p>(Whitehead & Dahlgren, 1991; Graham, 2009; Kawachi et al., 2002; Braveman et al., 2017)</p>	<p>There are two implications of this aspect. First, that health inequalities are a population or group phenomenon (and between groups with common sociological features) rather than an individual phenomenon. The second is that variations within a population, if they are not ranked or categorised as being differences between social groups, would not constitute a measure of inequality.</p>
<p>The differences can be observed between categorical social groups or as a gradient across the whole population of ranked social groups.</p>	<p>(Whitehead & Dahlgren, 1991)</p>	<p>Categorical social groups can include ethnicity, sex or nationality. It is proposed that health inequalities can be observed between such groups because such differences are unjust and avoidable and the definition must therefore be able to incorporate this. However, ranked social groups (such as social class, educational attainment, income, deprivation of the area of residence), which often cover all or most of the population, can provide another view of health inequalities which constitutes a stepwise gradient in the health outcomes. The definition therefore requires to be able to incorporate both views of inequality and, ideally, the concept of the gradient. Note that gradients may be linear or non-linear, and this may in turn depend on the sensitivity of the ranking measure and the relative size of different social groups.</p>
<p>The differences are due to the vulnerabilities created by social structures and institutions.</p>	<p>(Krieger, 2001a)</p>	<p>This aspect of the definition seeks to include information about the causal processes but may thereby exclude other relevant exposures.</p>

In order to effectively encapsulate the best aspects discussed above, a new definition is therefore proposed:

Health inequalities are the systematic, avoidable and unfair differences in health outcomes that can be observed between populations, between social groups within the same population, or as a gradient across a population ranked by social position.

2.5 Discussion

To be comprehensive, any definition of health must contain experiential and functional elements, physical, mental and social dimensions, and be applicable to both individuals and populations. Defining the outcome by the causes, or the sustainability of the outcome, is arguably better covered within a causal theory framework. It is therefore argued that an adaptation of Last's (2001) definition is best for public health policy, practice and research:

‘A structural, functional, and emotional state that is compatible with effective life as an individual and as a member of society’.

For health inequalities, there is a strong reason to include all the features in Table 2.2 with the exception of the inclusion of the causal factors. As none of the existing identified definitions does this, an amalgam is proposed:

‘Health inequalities are the systematic, avoidable and unfair differences in health outcomes that can be observed between populations, between social groups within the same population, or as a gradient across a population ranked by social position’.

It is worth noting in passing that the term ‘population health’ is a much looser term that has been used to describe both the mean (or median) health and the distribution of health within a population (Manwell et al., 2015; Thirunavurakasu et al., 2013; Vaillant, 2012; Galderisi et al., 2015).

This seems like a reasonable working definition for this term, and one that will be used as relevant throughout this thesis.

The strengths of the approach taken here include the transparent and structured literature search, the explicit process of identifying the key features of each definition, and the critical appraisal of these features. However, alternative approaches to generating definitions have started with qualitative research which has then been thematically analysed to identify the key relevant components (Song & Kong, 2015). This type of approach could be further used to develop the experiential aspects of a health definition. It is likely that other definitions have been proposed that have not been included in this paper, and these may include other valuable themes. For example, the concept articulated by Grossman (1972) of health capital, and how people might trade off their health against other considerations, is not fully discussed here. Further work to systematically review the available definitions and to expand on the themes they propose, the values that underlie them, the assumptions they use and their utility for different purposes would be worthwhile. Finally, this definitional process has not involved consensus building nor negotiation with key stakeholders. This therefore runs the risk of creating just another definition that could therefore cause confusion rather than clarity in future research, practice and policymaking.

2.6 Conclusion

This chapter proposes definitions for health and health inequalities after reviewing commonly used definitions for their common and divergent features, examining the assumptions and value underlying these features, and then combining those with greatest utility into a short and accessible definition for use within public health research, policy and practice. In doing so, it makes the rationale for the use of these definitions explicit, and would also facilitate the development and use of alternative definitions for other purposes.

Definitions of health and health inequalities are important if a shared understanding between researchers, policymakers and practitioners is to be achieved. The wide range of definitions that are available reflects the inclusion or exclusion of different components and emphases, use varying assumptions and have differing underlying values. Definitions are proposed here that combine the greatest utility for those working in public health and policy, with brevity and accessibility.

The next chapter (Chapter 3) uses these definitions to shape the scope of a review and critical appraisal of theories of how the health of populations is shaped. Beyond that, Chapter 4 again uses these definitions to structure a review of the evidence linking political economy to health. However, in the later empirical chapters narrower measures of health and health inequalities are used than are commensurate with these definitions, as the scope of the work had to be narrowed to fit within time and data constraints, and the length requirements for the thesis.

3. Theories of population health

3.1 Introduction

How we theorise and understand the causes of population health, incorporating the mean, distribution and inequalities in health within a society (Kindig & Stoddart, 2003; McCartney et al., 2019a), influences the priority it is accorded, what research questions we ask, what recommendations we make and ultimately who we hold responsible for it (McCartney et al., 2013). This chapter reviews theories of population health, particularly focusing on commonly used heuristic diagrammatic models or frameworks, to identify their common and divergent features, and to interrogate the underlying assumptions, values and implications of each. Following this an augmented theory of population health is proposed which synthesises the best features of the existing theories and which addresses the gaps in current theorisation.

3.2 Early population health theories

Health has long been a concern for populations and governments, and the theories to explain it have changed substantially over time. Ancient and medieval understandings of individual and population health revolved around spiritual interpretation. Following the reformation, the emphasis shifted from the spiritual and mythical to more scientific understandings (Rosen, 2015). This led to the hygienist movement that went further than that seen in the Roman Empire, leading to the creation of separate sewerage systems, the provision of clean(er) water, isolation of the sick, safer burial of the dead, and exclusion of livestock from houses (Chadwick, 1842). With greater acceptance and use of scientific methods, the germ theory of disease gained prominence through the work of Louis Pasteur and

Robert Koch (Ullmann, 2007). Other causes of disease were increasingly being understood with the use of the new methods (e.g. that scurvy was caused by a lack of vitamin C (Baron, 2009)). John Snow took action (albeit belatedly) to disable a suspect water pump in Soho after epidemiological study to determine that cholera was water borne rather than miasma-related (Snow, 1936 (originally published 1855)), and Rudolf Virchow described how the health of his local population was a product of their political and economic situation in ((Virchow, 2006) originally published 1848). These developments demonstrated that health was beginning to be understood as a population phenomenon and as a product of the social context in which people lived.

It was around the time of World War II that a new phase of public health action, and by implication population health theory, developed (Hanlon et al., 2011). In Britain, influential reports on the living conditions and poverty experienced within cities had been published by Booth and Rowntree, albeit with a conflation of moral and material judgements (Booth, 1889, Rowntree, 1901). The economic depression in the 1930s and the devastating impacts of two wars, especially in Europe, was the context for social reforms across the continent and led to the construction of welfare states. These changes in social policy were also reflected in public health theory where addressing the social determinants of health (employment, housing, income, etc.) was increasingly identified as being important in achieving health improvement (Hanlon et al., 2011).

The substantial increases in life expectancy, particularly across Europe from the time of the industrial revolution, led to substantial theorisation and debate (in hindsight) of the relative contribution of different factors. McKeown proposed that most of the improvement was due to increased availability of nutritious food as a result of economic development, with smaller contributions from public health measures such as cleaner water and sanitation, or medical developments such as vaccination (McKeown et al., 1972; McKeown, 1976). These themes were expanded and challenged by

others who highlighted the importance of improved housing and reduced overcrowding, social institutions, safer workplaces, protective legislation, and economic factors in explaining the increase in life expectancy over this time (Floud et al., 2014; Fogel, 2004; Szreter, 1997; Szreter, 1988).

In the second half of the 20th Century, increased use of case-control and cohort studies led to the identification of individual ‘risk factors’ for disease and death. The first major example of this approach to theory was the Doll-Hill study, which identified the role of smoking in causing lung cancer (Doll, Hill, 1950). A plethora of such studies followed with a huge range of individual (and individualistic) risk factors subsequently identified, leading to a new form of behaviourist theory whereby the causes of diseases were framed as being the product of individual decisions in an often decontextualized manner. These sequential, but overlapping, developments of population health theory and action has been summarised as ‘four waves’ of public health (Hanlon et al., 2011; Lyon, 2003).

More recently, the importance of ecological, commercial and historical determinants of health have been described, alongside a more detailed understanding of the role of structural discrimination, economic and power relationships (Krieger, 2011; Krieger, 2001b; McCartney et al., 2020a; Sayer & McCartney, 2021; Phelan & Link, 2015; Phelan et al., 2010).

There is therefore a rich historical background of epidemiological and theoretical work to help understand the long-run changes in population health and its causes. The following sections go on to discuss the commonly used heuristic models of population health theory that seek to describe the causal factors leading to population health outcomes and the mechanisms involved. It then proposes a new model of the relationship between political economy and population health, drawing upon the best aspects of the existing models and addressing their weaknesses.

3.3 Methodological approach

As noted in Chapter 2, population health is defined as a term that encompasses the average, distribution and inequalities in health within a society; health is the structural, functional and emotional state that is compatible with effective life as an individual and as a member of society; and health inequalities is the systematic, avoidable and unfair differences in health outcomes that can be observed between populations, between social groups within the same population, or as a gradient across a population ranked by social status. As stated in the introduction, political economy is defined here as the historical contingencies, contemporary economics, production and consumption activities, power relations, governance, policies, politics (or institutions), legal rules, culture, values and ecology of societies (Dow et al., 2018; Hodgson, 2001).

These theories were identified in a non-systematic way, covering the models commonly used in teaching and practice in public health in the UK (sourced from the core textbooks listed for the Glasgow Master in Public Health course, the core textbook for the Membership of the Faculty of Public Health part A examination, and from snowball sampling from included models). Each model was examined to uncover the insights that it offers in terms of theorised causal processes, relationships between different exposures, the range of exposures and its utility as a means for furthering research and understanding of population health. These aspects were then tabulated to identify common themes and tensions between the models. A new, adapted, heuristic model of the political economy determinants of population health was then derived by synthesising the most useful aspects of the existing models.

It is worth noting that some authors have used the 'Social Determinants of Health' to describe and investigate the relationship between political economy and population health, and when used in its widest sense, this will include all of the exposures and relationships included within the definition of political economy above. However, it is arguably more commonly used in

a narrower sense, whereby social, economic and environmental exposures are listed as exposures relevant to population health, but without articulating how these exposures have come into being, how they are maintained, how they are distributed, and the social and economic relationships that underlie them (Wilkinson & Marmot, 2003). Krieger has neatly summarised this as a 'web of causation without the spider' (Krieger, 1994).

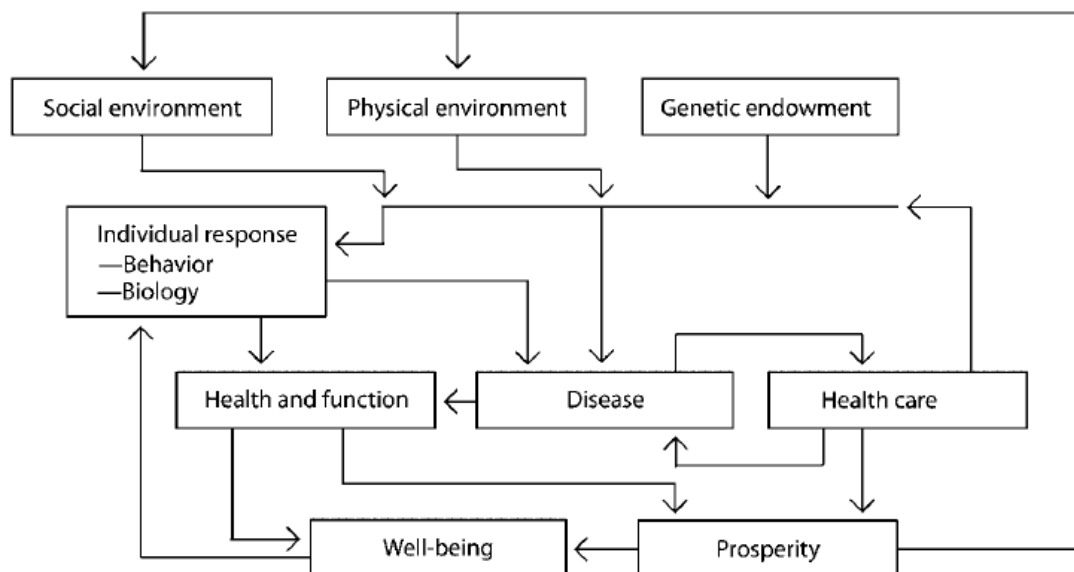
3.4 Population health theories and heuristic models

Black report, Evans and Stoddart, Dahlgren and Whitehead

The Black report identified four theories of health inequalities (artefact, selection, behavioural/cultural, structural) and critically appraised the evidence for each. The report was clear that the available evidence supported the structural theory best and, flowing from this conclusion, a range of policies to equalise the socio-economic circumstances in society were proposed (Black et al., 1988). Note that the Black Report did not discuss average outcomes for populations and why the average for any particular country might be different from another. Black's conclusions were consistent with the work of others in placing political economy at the heart of explaining population health (Doyal & Pennell, 1979; Hart et al., 1982), Doyal cast doubt on the ability of capitalism, through the mechanism of economic growth, to achieve improved population health because the social processes inherent to that system were said to be a key part of the causal pathway leading to population health problems (Doyal & Pennell, 1979). However, the relationship between capitalism and population health was argued to be complicated by the fact that capitalism had delivered rapid improvements in average living standards, which had been a vast improvement on the pre-existing economic systems. The questions for Doyal and Pennell were therefore whether, how and for whom capitalism remained a positive force for population health (Doyal & Pennell, 1979).

Although the Black Report was not associated with a heuristic illustrative model, several were produced in the following years that built upon the insights that it offered. For example, the Evans and Stoddart model described health outcomes in three forms (health, function, and well-being) and suggested a complicated (although logical) set of causal relations which included individual factors (individual behavioural, biological responses and genetic endowment) and societal factors (social and physical environment) (Evans & Stoddart, 1990) (Figure 3.1). However, some interconnections between exposures were unclear: (e.g. genetic endowment and physical environment seem to influence a pathway from health care to individual response). The role of social processes such as class relations, the contribution of different aspects of political economy, as well as health inequalities as an outcome, were not included.

Figure 3.1 - The Evans and Stoddart model of population health (1990, p. 1356)



Dahlgren and Whitehead also developed a population model for the WHO closely aligned to the Black Report (Figure 3.2) (Whitehead & Dahlgren, 1991; Dahlgren & Whitehead, 1991). This described 'layers' of exposure from socioeconomic, cultural and environmental conditions on the outside,

through living and working conditions, social and community networks, individual lifestyle factors, to individual age, sex and constitutional factors in the centre. This was generally understood as nesting individual determinants of health (such as age or alcohol consumption) within the context of the broader socio-economic context in which people lived. For many people this model therefore represented a helpful framing of population health as a social phenomenon where there was a dynamic between population-wide determinants of health and individual factors. It also helpfully articulated a wide range of social determinants of health in sufficient detail to focus policy action without the over-definition that might have excluded relevant exposures. However, the model lacked a clear set of causal relations or understanding of social processes and tended towards an approach of simply placing all the relevant factors in a diagram without connecting them or explaining their relationships. As Krieger has suggested, it represented a ‘web of causation’ without a ‘spider’ (Krieger, 1994, p.887). It is easy, however, to be critical of a model on criteria that it was never designed to meet. It was never designed to model the determinants of health inequalities; nor was it designed to describe the causal pathways between the determinants and health outcomes (Dahlgren & Whitehead, 2021). Indeed, Dahlgren and Whitehead (2021) point to the Diderichsen model as a better approach to understanding causal relationships (Figure 3.3) (Diderichsen et al., 2001). The main drawback of this framework is the under-articulation of political economy, including the economic and social determinants of health.

Figure 3.2 - The Dahlgren and Whitehead rainbow model (Dahlgren & Whitehead, 1991, p.20)

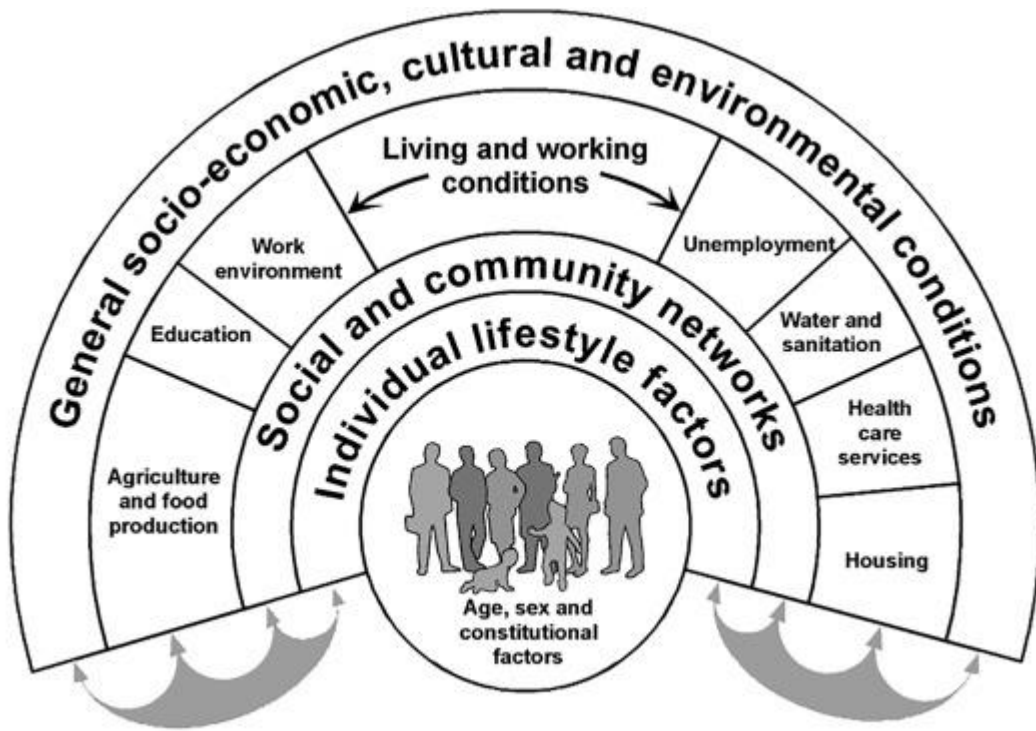
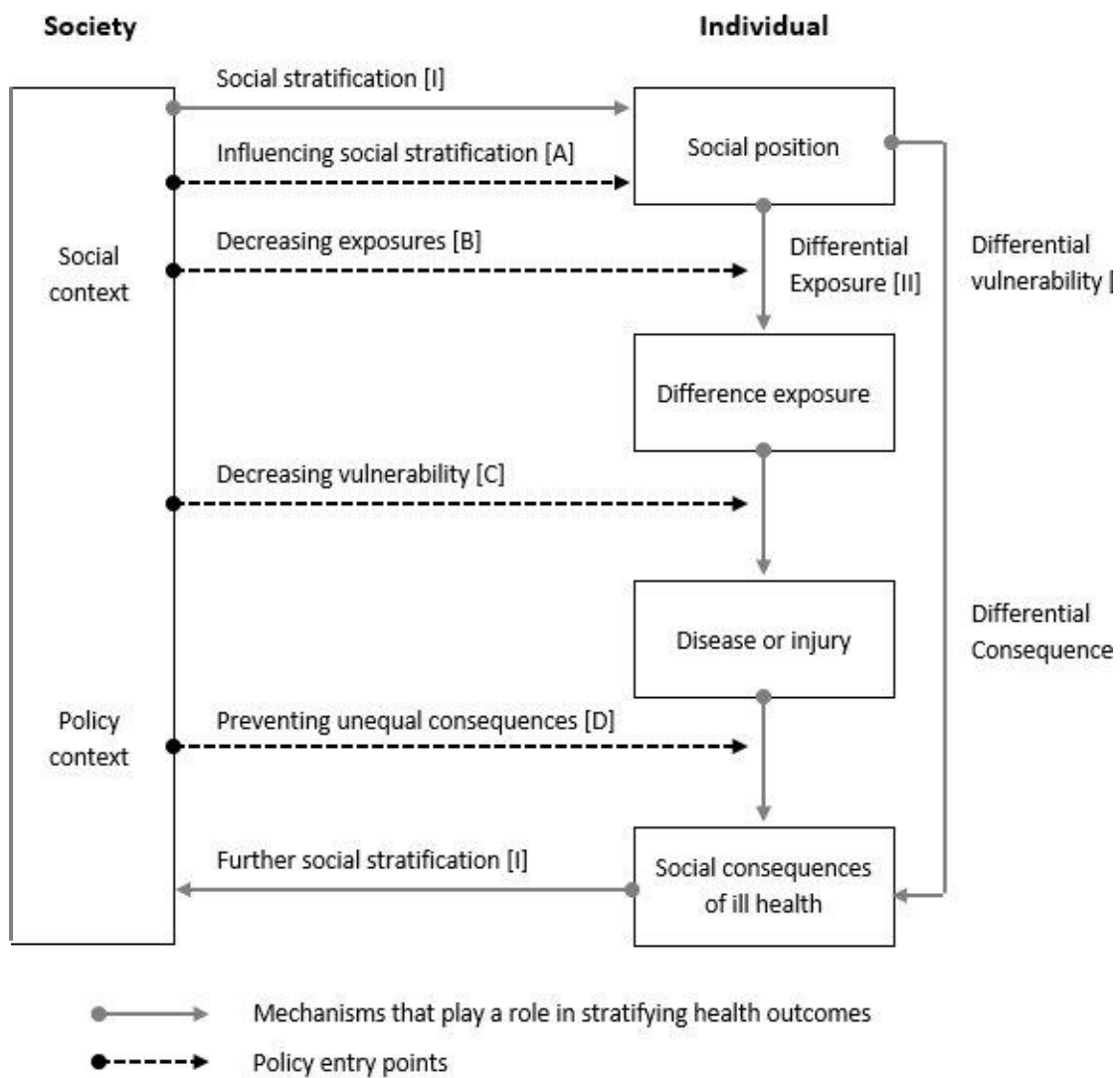


Figure 3.3 - The Diderichsen model (Diderichsen et al., 2001, p.15)



Selection theories

Macintyre (1997) described 'hard' and 'soft' interpretations of Black's selection hypothesis, based on West's (1991) reframing. West argued that people are more likely to experience a social slide as a consequence of illness in some societies than others, depending on the available social protection policies and prevalent discrimination. In doing so, he attempts to distance his argument from Social Darwinism, and instead reconceptualise social selection as a social process that operates to a greater or lesser

degree depending on the context. For example, the degree to which becoming unemployed leads to a drop in income varies between countries with some (such as Germany) tending to provide individuals with a high proportion of their salary for prolonged periods of ill-health whilst others provide only basic income no matter what the level of original salary. In this way, selection theory (resulting in a steeper social slide) would operate more fully in countries with only a residual income provision. Another example is the extent to which people with disabilities are viewed, stigmatised and supported within different societies. Where people with disabilities are nurtured and supported, they are more likely to retain their social position (including, for example, having statutory provision made to facilitate ongoing employment through workplace adaptations), whereas a society that does not value those with disability can be substantially disadvantaged and discriminated against, leading to a marked social slide.

Linked to this specific articulation of the operation of selection theory is the more general use of the idea of meritocracy in explaining how social ordering might occur within societies and the implications this may have for the measurement of health inequalities (Campos-Matos & Kawachi, 2015; Simons et al., 2013). The argument made is that individuals will experience greater change in their social position (up or down) in more 'meritocratic' societies because the policy and culture within such societies values virtuous individual characteristics (such as ability and hard work) rather than social class background. If there is greater social sorting within a society then it has been suggested that (if the markers of social status are measured contemporaneously rather than at birth) each social stratum will be more homogenous leading to larger distinctions in health outcomes between groups (Bambra, 2011a). Using lifespan variation (i.e. mortality data unranked by social status) a very different picture of which nations have the largest health inequalities has been found (Popham et al., 2013a). The relative importance of selection theory in explaining health inequalities is therefore contested (McCartney et al., 2013; Benzeval et al., 2014).

Lifecourse, Intelligence and Genetics

A particular form of selection theory has been articulated in relation to ‘intelligence’ or ‘genetics’ in which it is argued that health inequalities arise as the inevitable result of the individual and innate talents from which individuals in particular social groups benefit (that arise from genetic endowment, but which are best measured or articulated as intelligence) (Batty et al., 2009; Gottfredson, 2004). However, this social Darwinist view tends not to account for the influence of socio-economic exposures in the early years (McCartney et al., 2013; Lawlor et al., 2006), the numerous measurement difficulties surrounding intelligence (and consequent introduction of systematic bias) (McCartney et al., 2013).

A more important aspect of health inequalities theory, and related to this critique of the role of intelligence and genetics, is the need to measure socio-economic exposures across the entire lifecourse given the known lagged effects of early experiences on health and the systematically different exposures between social groups (Power & Matthews, 1997; McCartney et al., 2016, Walsh et al., 2016). The importance of differential historical experiences across social groups in terms of direct exposure, and through the social processes described in more detail below at the level of economic and social development, are therefore crucial to a comprehensive theory of population health (Bambra, 2011b).

Cultural/behavioural theories

Cultural and behavioural theories were considered in the Black Report and have been commonly described and explained since. In essence, these theories to a greater or lesser extent foreground the role of health behaviours (such as smoking, alcohol consumption, diet, physical activity) and common behaviours and understandings within population sub-groups (frequently ‘the poor’, but also at times to include particular ethnic groups or cultural groups) as the most important causes of health inequality outcomes. In doing so, they either explicitly or implicitly diminish the

importance of structural factors and the political economy in influencing or determining cultures and behaviours, and put the onus and causal influence on individuals and those suffering most from health inequalities rather than those in positions of power (Gruer et al., 2009).

The extent to which this is true is less clear. For example, smoking and excess alcohol intake are well evidenced to be particularly harmful for a wide range of health outcomes, and this is not seriously contested. However, this ignores the extent to which cultures and behaviours are shaped, constrained and determined by structural factors. Furthermore, there is evidence that changes in the inequalities in behaviours does not change the inequalities in all-cause mortality outcomes (Stringhini et al., 2011) (as predicted by fundamental cause theory - discussed in more detail later in this section). Overall, although cultural and behavioural factors are important mechanisms linking exposures to structural factors, they do not provide much explanation of health inequalities outside of this context (McCartney et al., 2013).

Unequal societies

Wilkinson (1992, 2005), and more recently Wilkinson and Pickett (2006, 2009), have described through a series of correlational analyses that more unequal societies encounter a range of inferior health and social outcomes when compared to more equal societies. Crucially, this approach restricts itself to the mean health and social outcomes for a society (e.g. average life expectancy) and does not seek to describe health inequality outcomes (Wilkinson & Pickett, 2008). The evidence advanced for this thesis is that nations, and other population units (such as the US states), with greater income inequality tend to have lower life expectancy (and as such even the more affluent within an unequal society tend to do worse than the less affluent within more equal societies) (Wilkinson & Pickett, 2006). The theorised mechanism here is that steep social hierarchies within societies (as approximated by wide income inequalities) create substantive levels of

stress across the population as people worry about losing their social position or suffer by being in a stigmatised and relatively lowly social position (Wilkinson & Pickett, 2008). This builds upon the work by Marmot and colleagues on the Whitehall cohort studies, which articulated the importance of 'job control' (the extent to which people have autonomy over their own work or whether this is determined by others higher in the occupational social hierarchy) (Marmot et al., 1997). A substantial body of work exists which elaborates these psycho-social mechanisms which link steep social hierarchies with negative health outcomes (Wilkinson, 1992).

There have been two substantive critiques of Wilkinson and Pickett. First, that the data used to show an association between unequal societies and worse average health is not consistent across datasets and population unit sizes (i.e. by examining smaller areas the association weakens or disappears) (Lynch et al., 2004b). Second, the apparent relationship between income inequality and average health is better explained by more unequal societies having a larger population in relative poverty, and thereby exposed to relative deprivation. This relatively deprived population thus suffers from poorer health and this reduces the population average. Importantly, the implication of this critique is that it is materialist or structural mechanisms that are important in driving the more modest health outcomes rather than psychosocial mechanisms (Lynch et al., 2004b).

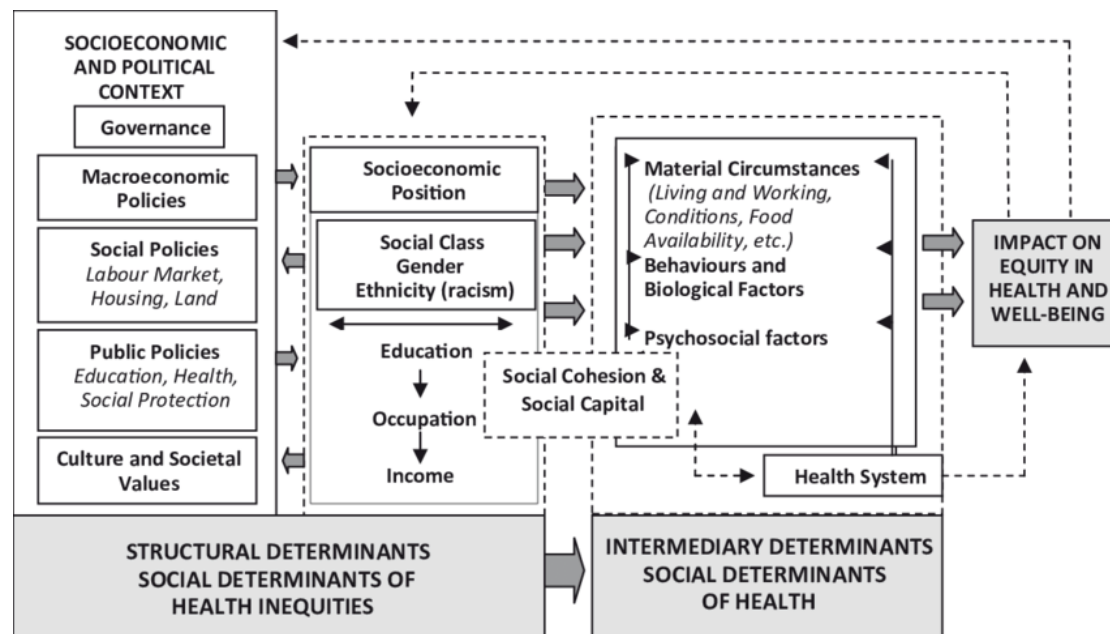
Commission for the Social Determinants of Health (CSDH)

The more recent work by Marmot and colleagues for the WHO under the auspices of the Commission for the Social Determinants of Health (CSDH) has continued to develop and reaffirm the conclusions of the Black Report and the work of Whitehead and Dahlgren (Marmot et al., 2008). Background work for the Commission provided a much expanded explanation of the social and political processes underlying population health and accompanying models, creating a sophisticated theoretical model for understanding inequalities in health (Solar & Irwin, 2007).

In particular, this work aimed to highlight the difference between levels of causation and distinguish between the mechanisms by which social hierarchies are created (and the resulting conditions of daily life). It also sought to incorporate an explicitly collective interpretation of the human right to health. It also used the concept of power to link social position to health outcomes (although this does not feature explicitly in the model). Finally, it includes within its scope the theorisation of processes that lead to the distribution of the social determinants of health (and in doing so renders these processes amenable to change rather affixed characteristics of a society).

The framework developed demonstrates that social, economic and political mechanisms generate differential social positions for population groups and that these social positions determine the exposure to specific determinants of health and health outcomes (Figure 3.4) (Solar & Irwin, 2007).

Figure 3.4 - the Solar and Irwin theoretical framework developed to support the Commission for the Social Determinants of Health (Solar & Irwin, 2007, p.6)



The Solar and Irwin framework, therefore, has a number of strengths. It includes theorisation of the social, economic and political processes through which differential exposure to the determinants of health arises - a feature missing in most other models (McCartney et al., 2013; Krieger, 1994; Solar & Irwin, 2007; Krieger, 2011; Krieger, 2008a; Krieger, 2008b). Second, there is a clear causal flow from the social context. There are, however, some weaknesses in the model. The arrows linking the boxes are not described, missing an opportunity to uncover the social processes at work (such as differential power). The arrows in the socioeconomic position box suggest that education determines occupation, which in turn determines income. Although this is clearly a set of causal relations that is likely to be important in most contexts, it is not the only influence on occupation and income (e.g. the role of wealth inheritance is important here (Krieger, 2018)). Furthermore, income is an important determinant of the quality of education that can be afforded and the likely early years' experiences that influence the capacity that individuals have to benefit from education

(Walsh et al., 2019). The health system (which refers the provision for health care services) is not linked explicitly to the box on public policy, although clearly policy will largely determine the funding and model of health care provision. The text describing the model discusses the risks of the use of the term ‘social capital’ in the model; in particular, the potential for social capital to be a depoliticised concept and one which is not amenable to political remedy. However, the model is unlikely to be used consistently in conjunction with the explanatory text and so the risk is that social cohesion and social capital are interpreted in such a depoliticised manner (reflecting again the missed opportunity to explain the linking mechanisms or arrows in the model).^c

Navarro’s Marxist approach

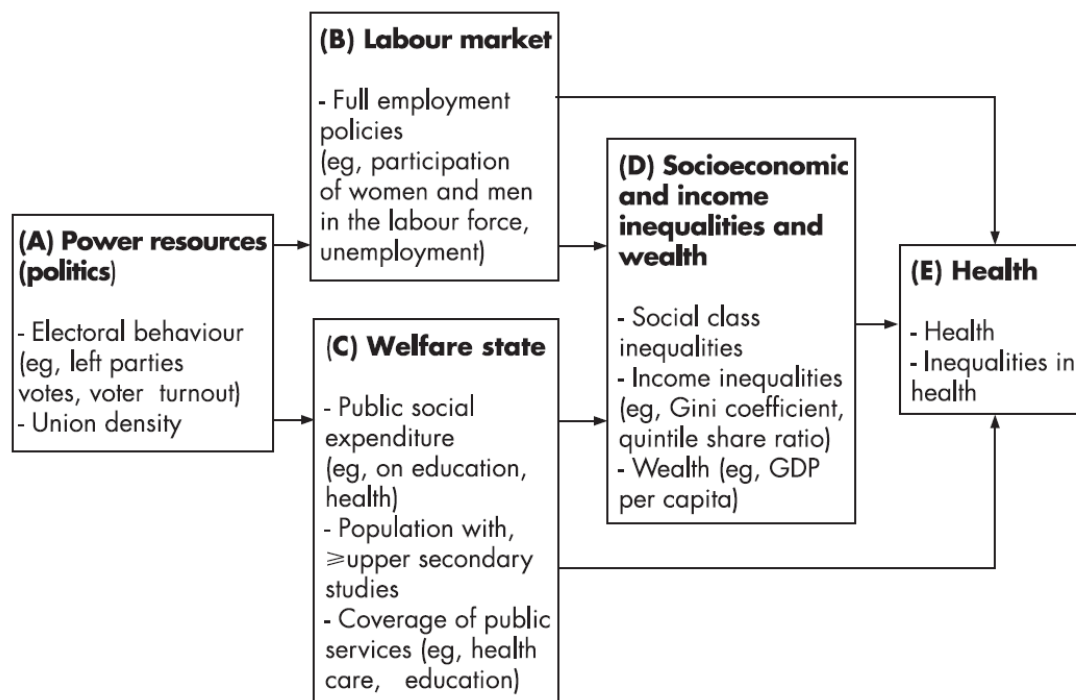
In common with those arguing for an articulation of the social processes leading to socio-economic and health inequalities, Navarro has argued for a more explicitly class-based analysis which draws upon the Marxist tradition of understanding how societies have developed, the power relations this generates within societies and the consequent distribution of resources and exposures across populations (Navarro, 1976). To test this thesis, attempts have been made by Navarro with colleagues over the subsequent years to look at the influence of labour power and the nature of the state in determining health outcomes across countries (Navarro et al., 2003; Muntaner et al., 2002; Navarro, Shi, 2001; Navarro, 1992; Navarro, 2009; Navarro et al., 2006; Borrell et al., 2007).

The strengths of this model include the explicit incorporation of the historical development of the economic system, class and power relations, and clear causal relations are proposed with a distinction made between deterministic and facilitative relationships. That said, the model does not include other forms of discrimination than those relating to class, and

^c It is worth noting that the Solar and Irwin paper also includes a separate framework for guiding policy and practice actions to reduce health inequalities.

crucially it does not seek to cover the relationship between the economic and class system and health (only the health service). As such, the model has limited utility as a heuristic for explaining population health, but the associated literature is a helpful exposition of the social processes involved in generating the distribution of the social determinants of health in societies. However, subsequent work did theorise the links between politics and power to health and health inequalities (Borrell et al., 2007). In contrast to the earlier Navarro model, this heuristic is linear, simplified and extends to include health and health inequality outcomes. The pathways linking to health are, unfortunately, not described; nor are the social processes underlying the causal relations implied across the model explained (Figure 3.5).

Figure 3.5 - Theory linking power resources, labour market, welfare state, socioeconomic inequalities, health and health inequalities (Borrell et al., 2007, p.658)



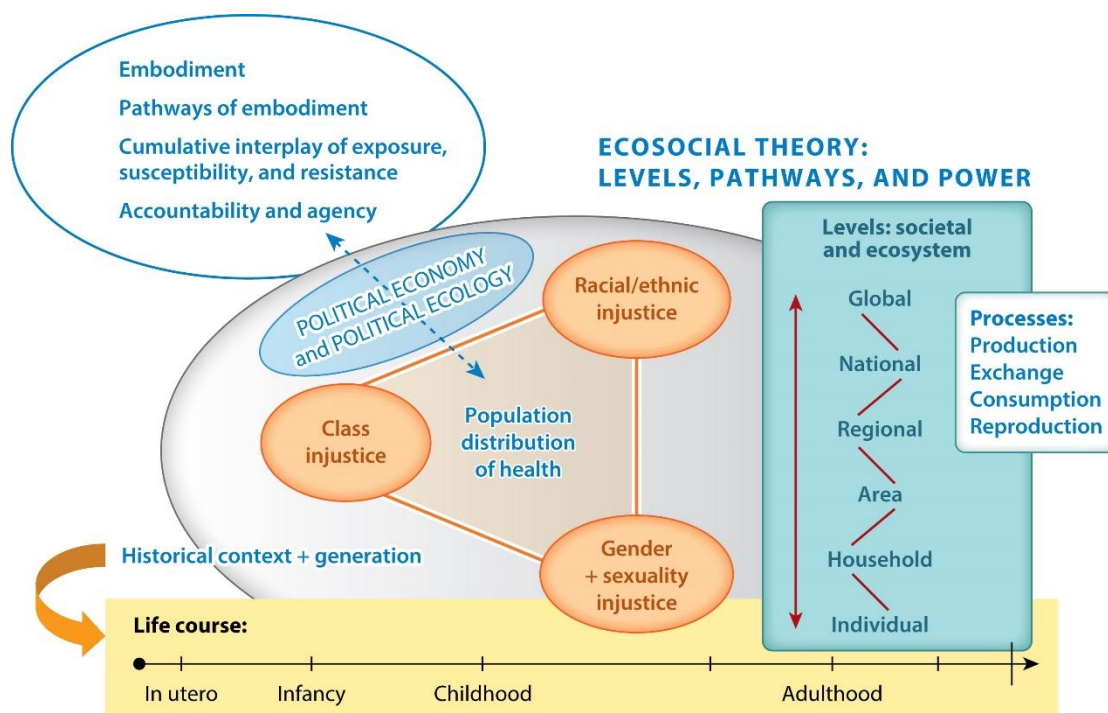
Another attempt to theorise the links between the social structure of society to health inequalities was undertaken by Hofrichter (Hofrichter,

2010). The resultant model explicitly includes three social processes (class exploitation, institutional racism and sexism) as being responsible for power and wealth imbalances, which, in turn, are described as leading to six other processes that determine the distribution of the social determinants of health. The model also proposes that these determinants are linked to health inequalities through psychosocial stress and behaviours. Nonetheless, the model is not comprehensive in its description of the pathways through which health inequalities occur and although it does cover some of the social processes leading to the distribution of the social determinants of health, these are unhelpfully described as being determined by the something termed 'social structure' which is probably a product rather than a determinant of the social processes the model describes.

Eco-social theory

Eco-social theory was developed by Krieger (2001b, 2008a, 2008b, 2011) to overcome the limitations she identified in pre-existing models. Figure 3.6 provides the visual representation of eco-social theory. The core components of the theory are: embodiment (the literal incorporation of exposures into bodily systems); pathways of embodiment (the mechanisms through which exposures become incorporated); the cumulative interplay of exposure, susceptibility and resistance across the lifecourse; and accountability and agency in society (Krieger, 2001b; Krieger, 2008a). However, the model is difficult to use as there is no 'starting point' to reading it and, moreover, there is no logical flow. The individual components within it are important, but are not linked by hypothesized causal relations.

Figure 3.6 - Krieger's ecosocial theory heuristic model (Krieger, 2020, p. 46)



Krieger N. 2020. *Annu. Rev. Public Health*. 41:37–62

Fundamental cause theory

Link and Phelan (1995) argued that health inequalities are explained by the distribution of the fundamental causes (such as income, power and resources) within a society, and therefore not determined by the mechanisms linking the fundamental causes to health outcomes (such as smoking or alcohol intake) (Phelan et al., 2010). The case for fundamental cause theory is built upon the observation that there are no inequalities in mortality for the specific causes of death for which there has been no known avoidable cause or effective treatment (e.g. brain cancers) and which, therefore, those with greater resources at their disposal cannot avoid or gain a treatment advantage. Nevertheless, for those causes of death for which the causes become clearer, or for which treatments become effective, inequalities arise and then persist as it becomes possible for those

with greater access to income, power and resources to gain an advantage. Examples of this include cardiovascular disease during the second half of the Twentieth Century and HIV/AIDS in the 1990s. Empirical work to test this theory has shown that the mechanisms (including the cause-specific mortalities) responsible for health inequalities have changed radically over time, yet the overall all-cause mortality inequalities have persisted in line with the inequalities in the fundamental causes (Link & Phelan, 2002; Phelan et al., 2004).

The implications of fundamental cause theory are important. First, policies and interventions that address income, wealth, power and resource inequalities are likely to be the most effective in changing health inequalities. Second, policies and interventions that address intermediate mechanisms may impact on the mean health outcomes of population, but are unlikely to impact on health inequalities. This includes action on alcohol, tobacco, food and physical activity because other mechanisms linking the fundamental causes to the health inequality outcomes will become more important in determining the outcomes (i.e. competing causes of health outcomes will be revealed) (Phelan et al., 2010; Scott et al., 2013).

Although there is substantial evidence to show that preventable and treatable causes of death have larger inequalities, and that the causes of death responsible for health inequalities have changed substantially over time, there is an absence of evidence about whether changes in the intermediate mechanisms (in the absence of change in the distribution of fundamental causes) have an impact on health inequalities. There is also an absence of evidence in relation to non-mortality health inequality outcomes (Scott et al., 2013; Mackenbach et al., 2015).

Fundamental cause theory has not been expressed in pictorial form, but it has the potential to be a highly important in helping explain how and why

particular exposures are shaped and determined by the political economy and the dominance of that in explaining health inequality outcomes.

Components of a robust population health theory

An effective population health theory has to be able to provide an explanation of trends and differences in health outcomes within and between populations. In doing so it has to be accessible and relatively easy to use and understand (and to achieve this a good theory also benefits hugely from an accompanying pictorial representation). The theory also has to be consistent with the accumulated evidence base, and testable in the face of emergent evidence.

In the work to develop eco-social theory, Krieger articulates the aspects it covers and contrasts these with other theories and models (Krieger, 2011, p.166). Eco-social theory is argued to cover the following:

- Political, economic and social production and reproduction of society
- Politics, polities^d and social movements
- Social determinants (as factors)
- Psychosocial exposures
- Biological pathways of embodiment
- Exposure across the lifecourse
- Levels in the hierarchy of systems (from global to biological)
- Spatiotemporal scale
- History and historical contingency
- Ecosystem
- Reflexivity of theory and theorists

As with the approach to appraising the utility of definitions of health and health inequality in Chapter 2, there is merit in tabulating the different

^d Interpreted as the collectives and organisations within a particular society, including the state.

components of health inequalities theory and the heuristic models used to explain them, and the relative value of the different aspects. In doing so, it is acknowledged that eco-social theory is one of the most advanced and comprehensive theories of population health currently available.

Nonetheless, eco-social theory (or more specifically, the heuristic model depicting the theory) also has a number of limitations.

First, as noted, the model lacks a logical direction or causal pathway. The nearest approximation to this is the lifecourse line at the bottom, but the relationship between this and the other factors in the model is unclear. In a sense, it appears to err on the side of including all the relevant factors, but without clearly and logically linking them together. This makes the model difficult to use and explain, and difficult to test. Second, although the population distribution of health is included, there is no articulation of the overall (mean) level of health in the population. Third, although there are certain forms of social inequality incorporated (class, gender and racial/ethnic), other forms of social inequality (for example, homophobia) are excluded because this specificity is only provided for those three social categorisations.

Building on, and adapting, the criteria offered by Krieger (2011), Table 3.1 suggests a range of factors to judge the utility of different population health theories. These criteria are challenging to represent adequately within a pictorial model, yet there is substantial value in doing so if the theorised causes of health inequalities are to be clearly and accessibly understood.

Table 3.1 - Suggested features of an enhanced population health theory and heuristic model

Feature	Comment
Able to explain average health and health inequality outcomes within and between populations.	This is not explicit in many models of population health (although it is more frequently included in the background text). Including both sets of outcomes explicitly may help in ensuring a broader understanding of the differences and links between them. Population must be defined such that it can incorporate shared characteristics (e.g. sexual orientation) or exposures (e.g. location).
Includes exposures at all levels from ecological to individualistic.	This is a more common feature of the available models, although only some are explicit in the exposures found at different levels (which is helpful in exemplifying what the exposure actually are, but can be prone to over-specification and therefore missing key exposures in some contexts or time periods).
Incorporates social and political processes, institutions and polities which generate differential exposures and outcomes.	This aspect of theory is least well developed in the available models and where it is included tends to simply name some social inequalities likely to be extant rather than expose the processes underlying them or how they link to history, exposures, mechanisms of embodiment, or outcomes.
Incorporates historical exposures (both within individuals as part of their lifecourse and in terms of the history of societies).	This feature is important for theories of population health because of the time lags between exposures and outcomes experienced by people over their lifecourse.
Articulates the embodiment of exposures (i.e. the mechanisms linking exposure to health outcomes).	Understanding how particular exposures result in health consequences is vitally important if attempts are to be made to interrupt or promote particular pathways. Furthermore, being able to demonstrate the mechanisms of action provides credibility to claims of the importance of particular exposures.
Incorporates a clear causal direction and pathway from exposures to outcomes.	This flows from the above feature. In demonstrating the mechanisms linking exposures and outcomes, causal pathways are described to provide a clear means of ‘reading’ the model and following the logic of the theory. The limitation of this feature is that the evidence base for particular pathways and causal directions may be unclear or contested.
Is simple enough to facilitate widespread use and understanding.	The risk of incorporating all of the features above is that the model becomes too complicated and thereby does not help support greater understanding. The complexity of the model therefore has to be balanced against its utility for a range of audiences.

Using these criteria, and the best elements of the available population health models and theories described above, a new working model is proposed below (Figure 3.7). The model is designed to incorporate all of the elements detailed in Table 3.1, but with a causal flow through the diagram from left to right and top to bottom. It starts on the left with ‘historical contingency’, emphasising the importance of historical context in shaping societies. For example, the extent to which societies have experienced war, imperial domination, slavery, industrialisation, environmental degradation, etc., has profound influence on the political economy, culture and ecology within which particular populations live.

Moreover, this approach has some theoretical appeal and alignment to other disciplines. In particular, it incorporates the concept of stratification economics.^e This argues that inequalities, stigmatisation, discrimination, and power asymmetries are group based (Davis, 2015; Darity, 2009). Thus, inequalities in society are a consequence of inter-group rivalries. The approach goes beyond social class processes to acknowledge structural racism and a range of other forms of structural discrimination and their intersections. The deeper the stratification in a society, the greater its social conflict and the more uneven are distributions in wealth, income, and resources. This impacts on health outcomes.

The central importance of the fundamental causes and political economy in determining population health outcomes is represented by their centrality in the model. The two-way relationship between the fundamental causes, ecology and culture (given that both ecology and culture are both determined by, and determinants of, the fundamental causes and political economy) is represented by the dual-headed arrows linking them up.

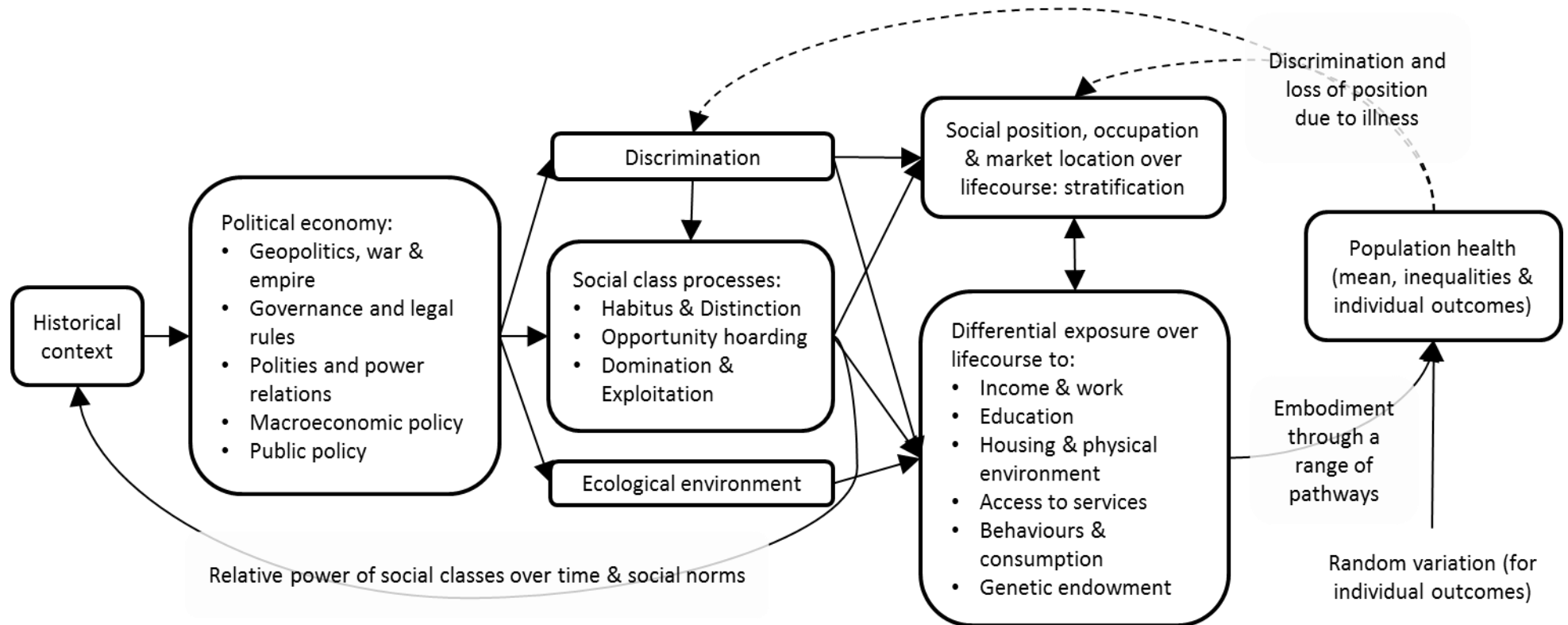
The fundamental causes are in turn linked causally to all other exposures, and also to how these exposures are differentially experienced. For

^e Stratification economics has been defined as, “...an important new approach devoted to explaining economic inequality in terms of how social groups are separated or stratified according to relative group status” (Davis, 2015).

example, the means by which those with similar genotypes can experience markedly different outcomes depending on social position and contextual exposures as a result of gene-environment interactions, is consistent with the model. Separating out the intersectional forms of social position from the exposures highlights that it is not social position *per se* that causes health outcomes, but the interaction between social position and exposures (in determining the degree of exposure and impact). Listing the different forms of social position also provides the means to consider how these can be compounded or mitigated by groups holding different social positions simultaneously, and across the lifecourse, all of which have immediate or lagged impacts. The model then describes how these exposures are then embodied by populations (both biologically and psychologically) to produce health outcomes (positive and negative, and differentially and unequally across social groups), and also incorporates the role of random variation to highlight that for individuals the model is not deterministic.

Finally, there is a dotted line to represent the potential for the health outcomes, through one form of health selection (whereby those who become ill or disabled are discriminated against directly or who experience social slide because of loss of income and status in societies where the welfare state does not maintain social position in such circumstances) to influence social position and subsequent exposures. Overall, this putative model meets the criteria set out for a population health theory. In Chapter 6, Figure 3.7 will be adapted to focus more specifically on theoretical links between fiscal austerity policies and health outcomes. Figure 6.2 details these relationships, including factors that lie on the causal pathway and those which are confounders.

Figure 3.7 - An enhanced theory of political economy and population health



3.5 Conclusion

This chapter has described the early understandings of the causes of population health, how these have developed over time, and the key debates that pertain today. The most commonly used heuristic models of population health are detailed, with their key features, strengths and limitations appraised.

None of the current models are sufficient for the purposes for this thesis, not least because of the difficulties in accurately incorporating the wide range of social processes which determine population health. In an attempt to improve and clarify the theory, the key tenets of a model were set out. This builds upon the best elements and structures of previous models but avoids the limitations identified with them. However, it details a theory which, although is rooted within the epidemiology of population health, remains to be tested. It also has to balance simplicity and utility with complexity, as it would be possible to have a more expansive and complex model which might be more accurate and comprehensive, but ultimately be unusable.

The following chapter will use this theory to inform a systematic review of the literature which considers the relationship between political economy and population health.

4. Impact of Political Economy on Population Health: a systematic review of reviews

4.1 Chapter synopsis

As noted in Chapter 3, although there is a substantive theoretical literature examining the relationship between a wide range of political economy exposures and health outcomes, the extent to which there is empirical evidence for the influence of different aspects of political economy on health is only partially understood. This chapter describes a systematic review of reviews of the literature describing the impact of political economy on population health.

A review of reviews approach was taken for several reasons. First, the scope of the primary literature linking all political economy exposures and health is too broad to be synthesised systematically. Second, this approach can provide an overview of the more focused questions and topics that have been systematically reviews, critically appraised and synthesised, and the extent to which this has been undertaken robustly. As such, clarity can be generated on the areas where there is quality evidence that has been synthesised robustly, where there are known gaps in the primary literature, and where there is a need for quality syntheses of the primary literature.

The literature for the review was identified through systematic searches of Medline, Embase, International Bibliography of the Social Sciences (IBSS), Proquest Public Health, Sociological Abstracts, Applied Social Sciences Index and Abstracts (ASSIA), EconLit, SocIndex, Web of Science and the grey literature via 'Google Scholar'. To make the review manageable, the scope

was limited to studies that were themselves a review of the literature. Relevant exposures were differences or changes in: policy, law or rules; economic conditions; institutions or social structures; politics, power or conflict. Relevant outcomes were any overall measure of population health such as self-assessed health, mortality, life expectancy, survival, morbidity, well-being, illness, ill-health and lifespan. All citations were reviewed independently by two reviewers. Critical appraisal of all included reviews was undertaken using modified 'Assessing the Methodological Quality of Systematic Reviews' (AMSTAR) criteria and then synthesised narratively giving greater weight to the higher quality reviews.

From 4,912 citations, 58 reviews were included. Both the quality of the reviews and the underlying studies within the reviews was variable. Social democratic welfare states, higher public spending, fair trade policies, extensions to compulsory education provision, microfinance initiatives in low income countries, health and safety policy, improved access to healthcare, and high quality affordable housing have positive impacts on population health. 'Neoliberal' restructuring (including deregulation, privatisation, greater recourse to pricing at the point of consumption, erosion of subsidies) seems to be associated with increased health inequalities and higher income inequality with lower self-rated health and higher mortality.

Unsurprisingly, the review shows that politics, economics and public policy are important determinants of population health. Countries with social democratic regimes, higher public spending and lower income inequalities have populations with better health. There are substantial gaps in the synthesised evidence on the relationship between political economy and health and there is a need for higher quality reviews and empirical studies in this area. However, there is sufficient evidence in this review, if applied through policy and practice, to have marked beneficial health impacts.

Note that the work in this chapter was published in the peer reviewed literature as part of this programme of postgraduate study:

McCartney G, Hearty W, Arnot J, Popham F, Cumbers A, McMaster R. Impact of Political Economy on Population Health: A Systematic Review of Reviews. *American Journal of Public Health* 2019; 109: e1_e12, <https://doi.org/10.2105/AJPH.2019.305001>.

4.2 Introduction

The determinants of population health may be different to the determinants of the health of individuals (Rose, 1985). Although we know that people who lose their jobs (Roelfs et al., 2011) and have lower incomes (Marmot et al., 2008) have higher mortality, this does not necessarily mean that populations with higher unemployment or with lower mean incomes have higher mortality (Wilkinson & Pickett, 2009; Mackenbach, 2012). It is therefore important to understand the health of populations at societal level by considering the overall context in which populations live.

Societies are complex and dynamic systems shaped by their historical contingencies as well as their contemporary economics, production and consumption activities, power relations, governance, policies, politics (or political institutions), legal rules, culture, values and ecology. The term 'political economy' is used here to describe these aspects of societies, their inter-relationships and power dynamics. As discussed in Chapter 3, there are several theories linking political economy and population health (Krieger, 2011; Whitehead & Dahlgren, 1991; Solar & Irwin, 2007; Beckfield et al., 2015), as well as some attempts to systematically evaluate the relationship between political economy and population health (Beckfield & Krieger, 2009). A political economy understanding of societies makes clearer why and how specific policies are implemented in different places and times.

Many aspects of political economy and health have been extensively studied. One of the longest running themes has been the study of the extent to which economic growth and economic development has been responsible for the reduction of mortality rates (McKeown et al., 1972; McKeown, 1976; Floud et al., 2014; Szreter, 1997; Szreter, 1988). In general terms, economic development measured in a variety of ways has been associated with improved health across time and place, but the causal mechanisms have been disputed. Some have argued that medical developments have been particularly important in explaining the health trends, whilst others have

stated that greater consumption and production (as approximated by Gross Domestic Product (GDP)), the building of social institutions such as the welfare state and social services, or public health measures have been more important (Floud et al., 2014; Fogel, 2004; Cutler et al., 2006; Deaton, 2013). It has also been noted that many measures of health, including happiness, well-being and life expectancy, have not consistently improved in tandem with economic growth (Lane, 2001; Easterlin, 1974; Tapia Granados, 2017); and that mortality rates continue to improve in some countries, such as Japan and Cuba, which experienced prolonged periods without growth (Borowy, 2011).

In addition to this work on economic development and health, there are many studies considering the health impact of short-run recessions, and the interaction with different economic policy responses. It seems that some health outcomes such as road-traffic fatalities and alcohol-related mortality tend to decline in the short-run following recessions, but others such as suicide worsen (Tapia Granados, 2017; Toffolutti & Suhrcke 2019). Given that there is very strong evidence that people who lose their jobs experience substantial increases in subsequent mortality (Roelfs et al., 2011), understanding the differential impacts and the contextual interactions with economic and social policy is important. This has led to the finding that a combination of austerity policies in response to recession, and recessions in countries with minimal welfare state provision, exacerbates the negative health impacts of recession (Stuckler & Basu, 2013; McKee et al., 2012; King et al., 2009; Stuckler et al., 2009c). Indeed, countries which have pursued more 'neoliberal' approaches to economic policy have been found to have worse health inequalities and higher mortality rates, amongst high income countries (Walsh et al., 2016; McCartney et al., 2011; Stuckler et al., 2009c; Beckfield & Krieger, 2009). Neoliberalism is used to refer to that suite of theories and policies that advocate individualism, marketization and privatisation of industry, goods

and services, and the financialisation of large sections of the economy (Mirowski, 2013; Collins & McCartney, 2011; Scott-Samuel et al., 2014).

Income inequality has been proposed as an important cause of health and social problems across high income countries (Wilkinson & Pickett, 2009). The association is much stronger at country level and for states in the USA than it is for analyses at smaller scales (Wilkinson & Pickett, 2006; Backlund et al., 2007). There remains substantial debate about the extent to which the association is due to worse health (and social) outcomes for those living in poverty; due to a variety of mechanisms between wider inequalities and health outcomes (e.g. psychosocial stress); or whether both income inequalities and the health/social outcomes are due to other political economy factors (Lynch et al., 2004b; Starfield & Birn, 2007).

Another focus of study has built upon Esping-Andersen's classification of European nations into different welfare state types (Esping-Andersen, 1990). By extending the classification and identifying the common features of how different countries provide public services and the extent to which government uses taxes and benefits to (re)distribute incomes, several authors have found that Nordic welfare states tend to have lower overall mortality rates than other European welfare types (Bambra, 2007), but greater health inequalities (Mackenbach, 2012; Bambra, 2011a). However, this is not the case if inequalities are measured using lifespan variation (Popham et al., 2013). The impact of welfare state regimes on health is now a well theorised and studied phenomenon globally (Chuang et al., 2011; Raphael & Bryant, 2015; Muntaner et al., 2011a; Muntaner et al., 2011b).

The experience of democracy has been extensively considered as potentially important factor in explaining differences in population health (Muntaner, 2013; Lin et al., 2012; Pushkar, 2011). In Europe from the 1970s the democratisation of Greece, Spain and Portugal from fascist dictatorships was associated with faster improvements in life expectancy, but the

relationship with democracy was less clear across Eastern Europe after 1990 as that period of democratisation was also associated with substantial economic restructuring and rapidly increasing income and wealth inequalities (Beckfield & Krieger, 2009; Mackenbach et al., 2013). Although using only cross-sectional data, an association has been identified between greater democratisation and higher population self-rated health (Krueger et al., 2015).

Despite this wide range of research, the extent to which the different aspects of political economy influence health, and through which mechanisms and in what contexts is only partially understood. Furthermore, there is no overall picture of the field whereby policymakers and academics can reliably know the areas which have seen extensive study or where there are gaps. There is also no review where studies of sufficient quality have been synthesised to clarify which relationships are causal and generalizable across populations or where there remains substantial uncertainty or debate. This review of reviews seeks to understand the extent to which political economy, and important aspects of it, explain differences in health outcomes within and between populations over time.

4.3 Methods

Protocol and reporting

The study design was a systematic review of review studies. The protocol for the review was published on the International prospective register of systematic reviews (PROSPERO) website:

https://www.crd.york.ac.uk/prospero/display_record.php?RecordID=65352.

The results are reported in line with the PRISMA guidelines for the reporting of systematic reviews, including the additions in relation to review focusing on the impacts of equity (Welch et al., 2012).

Inclusion and exclusion criteria

Studies were included that: were a review of the literature; included any overall measure of health as an outcome such as self-assessed health, mortality, life expectancy, survival, morbidity, well-being, illness, ill-health and lifespan; and included at least one aspect of political economy as the exposure. Following the theoretical understanding of the nature of political economy and population health detailed in Figure 3.8, political economy exposures were defined in terms of a difference, or change in: policy, law or rules; economic conditions; institutions or social structures; or politics, power or conflict. The following were excluded: book reviews; reviews that looked only at specific conditions, specific diseases or specific causes of death; specific interventions within services rather than overall policy, practice, institutions, legal rules, or political-economy exposures, or where the exposures were not linked to political economy processes; reviews that simply use a measure of social position to describe a gradient in an outcome rather than use an aspect of political economy as an exposure; protocols for reviews; and where the health outcomes were limited to a subset of the population (except age/sex strata - i.e. reviews pertaining to, for example women, children, or adults aged 35-70 years were included). There were no restrictions on the countries of interest or on the publication date.

Search strategy

The following research databases were searched in May 2017 for relevant citations: Medline, Embase, International Bibliography of the Social Sciences (IBSS), Proquest Public Health, Sociological Abstracts, Applied Social Sciences Index and Abstracts (ASSIA), EconLit, SocIndex and Web of Science. The grey literature was searched using similar terms in 'Google Scholar'.

The full database search strategy, including the search terms and combinations, is available at

https://www.crd.york.ac.uk/prospero/display_record.php?RecordID=65352.

No language restrictions to the search were put in place, but in practice the search terms used would have been unlikely to identify relevant papers not

published in English and some potentially relevant studies had to be excluded because no translation facilities were available. These are noted in the results section.

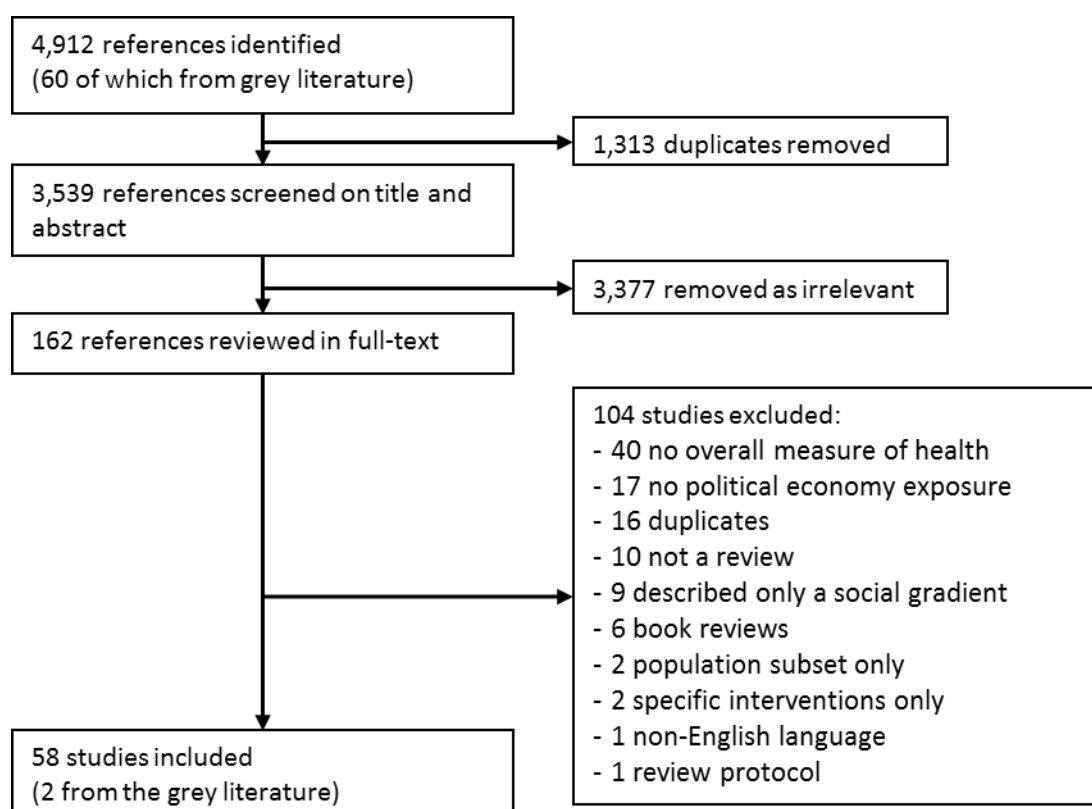
Screening, critical appraisal, data extraction and synthesis

Studies identified via the grey literature were screened by copying the relevant citation information into a document which was then screened independently by two reviewers. Any differences in interpretation were resolved by discussion until a consensus emerged. Critical appraisal of all studies deemed to meet the inclusion/exclusion criteria was undertaken using modified AMSTAR criteria (Shea et al., 2017). Specifically: was an 'a priori' design for the review provided; was a comprehensive search undertaken (including relevant search terms and at least two databases); were the studies selected for inclusion by at least two independent researchers; were there clear inclusion and exclusion criteria; was the status of publication (e.g. grey literature) ignored in the inclusion/exclusion criteria; were the data extracted independently by at least two researchers; was the scientific quality of the included studies assessed and documented; was the scientific quality of the included studies used appropriately in formulating conclusions; were the methods used to combine the findings of studies appropriate; was the likelihood of publication bias assessed; and were there important conflicts of interest that may have impacted on the conclusions? Data from the reviews were tabulated back to the original studies to prevent duplication across reviews. Given the mix of qualitative and quantitative data these were synthesised narratively giving greater weight to the data from higher quality reviews and higher quality original studies. Following Kim (2017), where a review provided insights across multiple themes, these were disaggregated for synthesis. The results are presented by theme in order to synthesise across political economy types rather than synthesising by country or time period which would have made the findings much more context-specific.

4.4 Results

A total of 4,912 references were screened to identify the 58 review studies that were included in this systematic review (Figure 4.1). There was substantial overlap in the underlying primary studies that were used by reviews in the same thematic area, thus care was taken to avoid ‘double-counting’ the insights that arose from the same primary sources. There were only three relevant reviews that were classified as ‘very high’ quality, and only a further seven as ‘high’ quality. Although each review was assessed to ascertain whether or not they had looked for and identified publication bias in the studies they included, this was found not to be a relevant criterion for any of the included studies and it did not help to discriminate between higher and lower quality reviews.

Figure 4.1 - PRISMA flowchart showing the points at which studies were included/excluded



Economic recession

Eight reviews considered the health impacts of economic recession (Table 4.1). There was little high quality evidence in this area for the reviews to draw upon - reflecting an absence of evidence in some areas (relating to resilience to economic crises (Glonti et al., 2015) and the impacts on child mortality (Palma et al., 2009)) and a combination of low quality systematic reviews or low quality studies in others. The reviews also tended to focus on specific aspects of recession (e.g. on one specific recession) or a specific outcome (e.g. mental health). The available evidence did however suggest that mental health (Frasquilho et al., 2016; Parmar et al., 2016; Rajmil et al., 2014; Downing, 2016), self-rated health and mortality (Rajmil et al., 2014; Simou & Koutsogeorgou, 2014; Falagas et al., 2009) worsened in many populations in association with recession. The lack of a high quality review of recession and adult mortality, particularly in light of the contradictory findings of some high quality primary studies (e.g. Tapia Granados, 2017; Toffolutti & Suhrcke 2019) mean that this result needs to be treated very cautiously.

Table 4.1 - Reviews considering the health impacts of economic recession

Author	Critical appraisal ^a	Review quality summary ^b	Reported quality of included studies	Context	Summary of insights
(Glonti et al., 2015)	2 3 4 6* 7 8 9 10 11*	High	Mixed	Mostly drawn from Europe, USA and south-east Asia	There is an absence of evidence of political economy factors that increase resilience to economic crises
(Parmar et al., 2016)	2 3 4 5 7 8 9 10 11	High	Low	Mostly southern Europe and UK	The post-2008 recession in southern Europe was associated with a worsening of mental health outcomes and mixed impacts on other health outcomes
(Frasquilho et al., 2016)	2 3 4 7* 8* 9 10 11	Moderate	Low	Most studies were from high income countries, especially Europe and North America	Periods of economic recession in high income countries may be associated with worsening mental health
(Palma et al., 2009)	2 8* 9 10 11*	Low	High	Not clearly reported	There was an absence of evidence of the impact of economic factors on the child mortality
(Rajmil et al., 2014)	2 3* 4 5 7 9 10 11	Low	Mixed	Wide range of countries including sub-Saharan Africa and Bangladesh, but most studies from high income nations	Infant mortality increased or an excess was observed (in Greece and Sub-Saharan Africa) in association with recession, but not in Spain. SRH worsened in adolescents in the USA and inequalities in SRH worsened in Catalunya.
(Simou & Koutsogeorgou, 2014)	2 3 4* 6* 9 10 11	Low	Not reported	Greece	SRH worsened in 2007-9 in Greece in association with the recession and austerity
(Falagas et al., 2009)	2* 4 5 9 10 11*	Low	Not reported	South Korea, Peru, Madagascar, Mexico, Bulgaria, Russia	Periods of economic recession were associated with an increase in all-cause mortality in a selection of countries.
(Downing, 2016)	2* 4 7* 9 10 11*	Low	Not reported	Not clearly reported, but many from the USA	Housing foreclosure in the USA is associated with mental health problems and violence with an absence of evidence for other health outcomes.

^aCritical appraisal criteria: 1 = an 'a priori' design for the review was provided; 2 = a comprehensive search was undertaken (including relevant search terms and at least two databases); 3 = studies were selected for inclusion by at least two independent researchers; 4 = there were clear inclusion and exclusion criteria; 5 = the status of publication (e.g. grey literature) was ignored in the inclusion/exclusion criteria; 6 = the data were extracted independently by at least two researchers; 7 = the scientific quality of the included studies was assessed and documented; 8 = the scientific quality of the included studies was used appropriately in formulating conclusions; 9 = the methods used to combine the findings of studies was appropriate; 10 = the likelihood of publication bias was assessed (if possible); 11 = there were no important conflicts of interest that may have impacted on the conclusions. *denotes a partially fulfilled criterion.

^b Quality assessment: all (very high); at least 2, 4, 7, 8, 9 (high); at least partially 2, 4, 7, 8, 9 (moderate); all others (low).

Healthcare policy

Changes to healthcare policy form another substantial area for systematic reviews although there was only one of high quality (Table 4.2). The impact of changes to healthcare insurance coverage (Hadley, 2003; Yuan et al., 2014; Acharya et al., 2013; Liang et al., 2012; Kesselheim et al., 2015) and of conditional cash transfer schemes (Yuan et al., 2014; Gopalan et al., 2014; Murray et al., 2014) were the most common subjects for reviews. The only high quality review found an absence of evidence of the health impacts of changes to health insurance coverage for prescriptions drugs (Kesselheim et al., 2015). The other reviews in this area were of lower quality and the studies on which they relied was either low or unclear (Hadley, 2003; Yuan et al., 2014; Acharya et al., 2013; Liang et al., 2012). One reported that becoming healthcare insured was associated with increased self-rated health and reduced mortality (Hadley, 2003), whilst others either reported mixed (Liang et al., 2012), uncertain (Acharya et al., 2013; Liang et al., 2012) or an absence (Yuan et al., 2014) of evidence.

A conditional cash transfer scheme in India was associated with mixed impacts on perinatal, neonatal and maternal mortality in some (Yuan et al., 2014; Gopalan et al., 2014). Two low quality reviews report that the impacts of changes to healthcare governance were contextually dependent, but notable in some countries (Ciccone et al., 2014; Sumahab et al., 2016). There was also low quality review evidence that increased primary care provision (Kruk et al., 2010) and increased public health spending could have positive health impacts (Singh, 2014).

Table 4.2 - Reviews considering the health impacts of healthcare policy

Author	Critical appraisal ^a	Review quality ^b	Included study quality	Context	Summary of insights
(Kesselheim et al., 2015)	2 3 4 6* 7 8 9 10 11*	High	Low	USA	There was an absence of evidence on the impact of changes to insurance coverage for prescription drugs on health in the USA.
(Gopalan et al., 2014)	1* 2 3* 4 5 7* 8 9 10	Moderate	High	India	The Janani Suraksha Yojana conditional cash transfer for skilled birth attendance in India was associated with a reduction of 14.2 (95% CI -2.7 to -31) perinatal deaths per 1,000 pregnancies, and a reduction of 6.2 (95% CI -8.1 to 20.4) neonatal deaths per 1,000 live births.
(Sumahab, et al., 2016)	2 3 4 6* 7 8* 9 10 11	Moderate	High	Spain and Canada	There is evidence that inequalities in self-rated health are smaller in Spain and Canada in association with decentralisation of healthcare governance.
(Liang et al., 2012)	2 3 4 5 6 7* 8 9 10 11	Moderate	Mixed	Rural China	The health impact of the Chinese NCMS healthcare insurance scheme varied widely across the available studies and so the overall impacts were unclear.
(Yuan et al., 2014)	2 3 4 5* 6* 7* 8* 9 10 11	Moderate	Not reported	India and Philippines	There was an absence of evidence in relation to the impact of conditional cash transfers and health insurance policies on maternal mortality.
(Acharya et al., 2013)	2 4 5 9 10 11*	Low	Not reported	Wide coverage of low and middle income countries	The impact of healthcare insurance schemes in the informal sector have uncertain impacts on health.
(Hadley, 2003)	2* 4 5 7* 8* 9	Low	Unclear	Not clearly reported, but many from the USA	A change to becoming healthcare insured was associated with a reduction in mortality ranging between 4% and 25% across studies and improved SRH.

Author	Critical appraisal ^a	Review quality ^b	Included study quality	Context	Summary of insights
(Murray et al., 2014)	1 2 3 4 5 7* 9 10 11*	Low	High	India, Nepal, Mexico & Tanzania	Different studies of conditional cash transfers have been associated with: an 11% reduction in maternal mortality; no change in neonatal mortality; and 17% and 2% reductions in infant mortality. Some studies have associated payments to offset the costs of health service access with declines in neonatal mortality whilst others have shown no change. Maternity service vouchers were associated with a 1 percentage point decline in stillbirths but no effect on neonatal deaths compared to comparison areas.
(Ciccone et al., 2014)	2 3* 9 10 11*	Low	Not reported	Mostly Africa, with some from Asia and two in South America	Different aspects of governance in low and middle-income countries were associated with health outcomes, but the exposure and outcomes measures, and contexts, were highly variable as was the degree of association and extent to which the effect was mediated through other factors.
(Kruk et al., 2010)	2 3 4 5 9 10 11*	Low	Not reported	Wide range - Caribbean, Latin America, Central America, Sub-Saharan Africa and Asia	There is some evidence that primary care programmes in middle and low income countries have reduced child mortality and in some cases wealth-based mortality inequalities.
(Singh, 2014)	2* 3* 4 5 8* 9 10 11*	Low	Not reported	USA	There is consistent evidence that public health spending in the USA is associated with better population health outcomes

^aCritical appraisal criteria: 1 = an 'a priori' design for the review was provided; 2 = a comprehensive search was undertaken (including relevant search terms and at least two databases); 3 = studies were selected for inclusion by at least two independent researchers; 4 = there were clear inclusion and exclusion criteria; 5 = the status of publication (e.g. grey literature) was ignored in the inclusion/exclusion criteria; 6 = the data were extracted independently by at least two researchers; 7 = the scientific quality of the included studies was assessed and documented; 8 = the scientific quality of the included studies was used appropriately in formulating conclusions; 9 = the methods used to combine the findings of studies was appropriate; 10 = the likelihood of publication bias was assessed (if possible); 11 = there were no important conflicts of interest that may have impacted on the conclusions. *denotes a partially fulfilled criterion.

^b Quality assessment: all (very high); at least 2, 4, 7, 8, 9 (high); at least partially 2, 4, 7, 8, 9 (moderate); all others (low).

Income inequality

Table 4.3 details all the reviews considering the impact of income inequality on health. Despite there being some 13 reviews in this area, none were high quality and only two, both of which were based on the same review process, that were moderate quality (having been downgraded for insufficient detailing of the quality of the included studies, a lack of incorporation of study quality into the synthesis and having not explicitly included grey literature) (Kondo et al., 2012, Kondo et al., 2009). These showed that income inequality was detrimental for health, especially where the Gini coefficient (an indicator of income inequality where 1 represents all income going to one person and zero complete equality) is >0.3 , when analysed with larger population units, including data after 1990, and where adjustment is made for time lags. The low quality reviews in this area did not report a sufficiently robust approach to searching and selecting relevant studies and frequently did not make any attempt at critical appraisal of the individual studies they relied upon (Kim, 2017; Adjaye-Gbewonyo & Kawachi, 2012; Furnée & Pfann, 2010; Judge et al., 1998; Lynch et al., 2004a; Macinko et al., 2003; Pickett & Wilkinson, 2015; Spencer, 2004; Wilkinson & Pickett, 2006; Rowlingson, 2011; Lago et al., 2018). The strengths of some of these reviews lie instead in the theoretical elaboration they offer rather than in the quality of the reviews and synthesis (Lynch et al., 2004a; Wilkinson & Pickett, 2006).

Table 4.3 - Reviews considering the health impacts of income inequality

Author	Critical appraisal ^a	Review quality summary ^b	Included study quality	Context	Summary of insights
(Kondo et al., 2009)	2 3 4 6 7* 8* 9 10 11	Moderate	Unclear	Mostly high income, but some Latin American and Eastern European and China	There is an adverse effect of income inequality on health, especially when the Gini is >30, after 1990, and when time lags are accounted for.
(Kondo et al., 2012)	2 3 4 6 7* 8* 9 10 11	Moderate	Unclear	Mostly high income, but some Latin American and Eastern European and China	The health-income inequality relationship is stronger with larger population units (for SRH); and that lag, threshold and period effects all remain important explanatory factors after adjusting for area size.
(Adjaye-Gbewonyo & Kawachi, 2012)	2* 4 5 9 10 11*	Low	Not reported	USA, GB, China, Japan, Australia and South Africa	Greater relative deprivation was associated with higher mental ill-health, higher mortality and lower SRH.
(Furnée & Pfann, 2010)	2* 5 9*	Low	Not reported	Europe (mostly northern Europe) and USA	SRH at lower income levels is worse in more unequal countries.
(Judge et al., 1998)	4* 9* 10 11*	Low	Not reported	Large range of countries included in panel data, including low and middle income nations	Greater income inequality in high income countries is associated with higher mortality and lower life expectancy but the sample and measures are limited.
(Lynch et al., 2004a)	9* 10 11*	Low	Not reported	Wide range of countries, but most were high income	There is little evidence that income inequality is a major generalisable determinant of population health difference within or between rich countries but there is stronger evidence that greater poverty is.
(Macinko et al., 2003)	2 4* 9* 10 11*	Low	Not reported	Not clear	The relationship between income inequality and health is unclear.

Author	Critical appraisal ^a	Review quality summary ^b	Included study quality	Context	Summary of insights
(Pickett & Wilkinson, 2015)	10	Low	Not reported	High income countries	There is sufficient evidence to conclude that income inequality causes lower life expectancy in high income countries.
(Spencer, 2004)	4 7* 8* 9 10 11*	Low	Unclear	Mostly Europe and North America with some studies of Australia and Japan	Greater income inequality and less redistribution was associated with higher infant mortality rates in high income countries.
(Wilkinson & Pickett, 2006)	7* 8* 9* 10 11*	Low	Not reported	Wide range of countries, but mostly high income	There are more studies than not showing a strong association between income inequality and mortality when large population units are compared and where potential mediators are not adjusted for.
(Rowlingson, 2011)	10 11	Low	Not reported	Not clear	There is evidence of a link between income inequality and worse health.
(Kim, 2017)	2 4 9 10 11	Low	Not reported	Higher income countries, especially Nordic countries	In high income countries, income inequality is associated with worse infant and child mortality, but not at other ages. The Scandinavian welfare regime is associated with better infant and child mortality but not at other ages.
(Lago et al., 2018)	2* 9 10 11	Low	Not reported	Not clear	Income inequality is associated with greater health inequality across the population and worse population health.

^aCritical appraisal criteria: 1 = an 'a priori' design for the review was provided; 2 = a comprehensive search was undertaken (including relevant search terms and at least two databases); 3 = studies were selected for inclusion by at least two independent researchers; 4 = there were clear inclusion and exclusion criteria; 5 = the status of publication (e.g. grey literature) was ignored in the inclusion/exclusion criteria; 6 = the data were extracted independently by at least two researchers; 7 = the scientific quality of the included studies was assessed and documented; 8 = the scientific quality of the included studies was used appropriately in formulating conclusions; 9 = the methods used to combine the findings of studies was appropriate; 10 = the likelihood of publication bias was assessed (if possible); 11 = there were no important conflicts of interest that may have impacted on the conclusions. *denotes a partially fulfilled criterion.

^b Quality assessment: all (very high); at least 2, 4, 7, 8, 9 (high); at least partially 2, 4, 7, 8, 9 (moderate); all others (low).

Housing and physical environment

There is strong evidence from a very high quality review that warmth and energy efficiency measures have positive health impacts although the impact of general housing condition improvement is unclear. There was an absence of evidence on the health impacts differences in, or changes in, housing tenure (Thomson et al., 2013; Thomson et al., 2006), although housing rent assistance was found to be beneficial for health (Table 4.4) (Bambra et al., 2010). There was an absence of evidence of the impacts on health of interventions to improve 'slums' in low- and middle-income countries (Turley et al., 2013). Within high income countries there was evidence that moving people to areas with lower poverty improves SRH, but that the impacts of regeneration programmes in poor areas are mixed, with evidence of no greater improvement in mortality in regenerated areas (Gibson et al., 2011; McCartney et al., 2017; Smith et al., 2009).

Table 4.4 - Reviews considering the health impacts of housing and regeneration policy

Author	Critical appraisal ^a	Review quality summary ^b	Included study quality	Context	Summary of insights
(Thomson et al., 2013)	1 2 3 4 5 6 7 8 9 10 11	Very high	Mixed	Wide range of countries	Improvements to housing can lead to health improvements, especially when they focus on people in ill-health and those living in low quality housing (especially cold homes).
(Turley et al., 2013)	1 2 3 4 5 6 7 8 9 10 11	Very High	Low	Low and middle-income countries	There was an absence of evidence of ‘slum’ improvements in low and middle-income countries on health outcomes.
(McCartney et al., 2017)	2 4 5 7 8 9 10 11	High	Mixed	High income countries	There is evidence that refurbishment of housing can have positive health impacts. The impacts of regeneration programmes on health is more mixed.
(Gibson et al., 2011)	2 3 4 7 8 9 10 11	High	Mixed	UK	Moving people to areas with lower poverty improves SRH. The evidence on efforts to improve high poverty areas is mixed with some positive and negative impacts reported. There is strong evidence that warmth and energy efficiency measures have positive health impacts although the impact of general housing condition improvement is unclear. There is a lack of evidence on the health impacts of housing tenure.
(Smith et al., 2009)	2 4 5* 6* 7 8* 9 10 11*	Moderate	Mixed	England	Health in Health Action Zones and New Deal for Communities areas did not increase faster than in comparison areas.
(Thomson et al., 2006)	2 3* 4 5 7* 8* 9 10 11	Moderate	Not reported	UK	There is some low quality evidence of mixed impacts of regeneration programmes on health in the UK.
(Bambra et al., 2010)	2* 3 4 6 8* 9 10 11	Low	Unclear	Not clear	Housing rent assistance associated with improved SRH, lower mental health problems, lower substance misuse, increased safety, reduced disorder and violence. Privatisation was associated with worse mental health and some physical health outcomes. Legal restrictions on working at height were associated with reduced fall injuries. Reduced legal drinking ages were associated with increased road injuries. Reduced blood alcohol driving limits were associated with decreased vehicle crashes. Welfare to work interventions may increase employment but may be confounded by labour market conditions.

^aCritical appraisal criteria: 1 = an ‘a priori’ design for the review was provided; 2 = a comprehensive search was undertaken (including relevant search terms and at least two databases; 3 = studies were selected for inclusion by at least two independent researchers; 4 = there were clear inclusion and exclusion criteria; 5 = the status of publication (e.g. grey literature) was ignored in the inclusion/exclusion criteria; 6 = the data were extracted independently by at least two researchers; 7 = the scientific quality of the included studies was assessed and documented; 8 = the scientific quality of the included studies was used appropriately in formulating conclusions; 9 = the methods used to combine the findings of studies was appropriate; 10 = the likelihood of publication bias was assessed (if possible); 11 = there were no important conflicts of interest that may have impacted on the conclusions. *denotes a partially fulfilled criterion.

^b Quality assessment: all (very high); at least 2, 4, 7, 8, 9 (high); at least partially 2, 4, 7, 8, 9 (moderate); all others (low).

Welfare state

Table 4.5 summarises the six reviews which consider the health impacts of differences and changes in welfare states. None of these were high quality and thus the conclusions that can be drawn in this area are more tentative. There is a substantial body of evidence arising from the rapid economic restructuring in Eastern Europe which suggests that its rapid transition to capitalism increased health inequalities (Beckfield & Krieger, 2009). How welfare states are classified and the consequent associations with health outcomes are inconsistent across papers, and this is not helped by three of the four reviews being low quality. Mortality rates are lower in social democratic welfare states, as defined by Esping-Andersen, but the relationship with health inequalities is rather inconsistent (Beckfield & Krieger, 2009; Bergqvist et al., 2013). Social democratic states also seem to be able to mitigate against the negative health impacts of precarious employment (Kim et al., 2012). There is some evidence from a lower quality review that higher social security payments to unemployed workers can reduce the negative impacts of unemployment (O'Campo et al., 2015). Political incorporation of subordinated racial/ethnic, indigenous and gender groups reduces health inequities, and dual family earner models and greater public spending are associated with lower mortality (Bergqvist et al., 2013; Brennenstuhl et al., 2012; Borrell et al., 2014).

Table 4.5 - Reviews considering the health impacts of welfare state type

Author	Critical appraisal ^a	Review quality summary ^b	Included study quality	Context	Summary of insights
(Beckfield & Krieger, 2009)	2 3* 4 7* 8* 9 10 11	Moderate	Mixed	Mostly Eastern Europe, USA and New Zealand, with spread across other countries	The transition to a capitalist economy and 'neoliberal' restructuring probably increases health inequalities. Welfare state regimes are inconsistently related to health inequalities. Political incorporation of subordinated racial/ethnic, indigenous and gender groups reduces health inequities.
(Bergqvist et al., 2013)	2* 4* 9* 10 11	Low	Not reported	High income countries	There is substantial diversity in how welfare states are classified with contradictory classifications across different schemes and authors. The institutional approach shows the most consistent results where more generous policies and benefits are associated with better mean population health. Greater expenditure on health and social services is associated with better health and lower inequalities.
(Brennenstuhl et al., 2012)	2 4* 6* 9 10 11	Low	Not reported	Not clear	Mortality is lower in social democratic regimes and where spending is greater on some specific policies (healthcare, public health, dual family earner policies, benefit generosity), but there is little support for the thesis that socio-economic inequalities in health are smaller in social-democratic regimes.
(Borrell et al., 2014)	2 3 4 9 10 11	Low	Not reported	Mostly Europe and USA	Nordic social democratic welfare regimes and dual earner family models best promote women's health. Enforcement of reproductive health policy across the USA, and longer paid maternity leave, is associated with better mental health in women.
(Kim et al., 2012)	2 4 9 10 11*	Low	Not reported	Mostly high income countries	Precarious workers in Scandinavian states do not suffer from worsening of self-rated health in contrast to those in other welfare state types.
(O'Campo et al., 2015)	2 3 4 5 6* 8* 9 10 11*	Low	Not reported	Not clear	There was weak evidence to suggest that generous unemployment insurance schemes can mitigate the harmful consequences of unemployment.

^aCritical appraisal criteria: 1 = an 'a priori' design for the review was provided; 2 = a comprehensive search was undertaken (including relevant search terms and at least two databases; 3 = studies were selected for inclusion by at least two independent researchers; 4 = there were clear inclusion and exclusion criteria; 5 = the status of publication (e.g. grey literature) was ignored in the inclusion/exclusion criteria; 6 = the data were extracted independently by at least two researchers; 7 = the scientific quality of the included studies was assessed and documented; 8 = the scientific quality of the included studies was used appropriately in formulating conclusions; 9 = the methods used to combine the findings of studies was appropriate; 10 = the likelihood of publication bias was assessed (if possible); 11 = there were no important conflicts of interest that may have impacted on the conclusions. *denotes a partially fulfilled criterion.

^b Quality assessment: all (very high); at least 2, 4, 7, 8, 9 (high); at least partially 2, 4, 7, 8, 9 (moderate); all others (low).

Income, employment and workplaces

There were six reviews considering the impacts of income, employment or workplace policy on health (Table 4.6). Despite a very high quality review being available, there was very little data on the impact of changes in income on health, with only one study reporting health outcomes. This found a 74% decline in child mortality in drought areas in response to an unconditional cash transfer (Pega et al., 2017). Job insecurity and unemployment arising from privatisation was evidenced as being negative for health, particularly mental health (Campos-Serna et al., 2013). There was mixed evidence on the impact of background unemployment rates on the health of those who become unemployed (Roelfs et al., 2015; Jin et al., 1997). Health and safety legislation and legislation to reduce legal blood alcohol levels for driving were all found to be effective means of improving population health (Bambra et al., 2010).

Table 4.6 - Reviews considering the health impacts of income, employment and workplaces

Author	Critical appraisal ^a	Review quality summary ^b	Included study quality	Context	Summary of insights
(Pega et al., 2017)	1 2 3 4 5 6 7 8 9 10 11	Very High	High	Nicaragua and Niger only	Only one study reported relevant data - that unconditional cash transfers at times of drought led to a 74% reduction in child mortality.
(Campos-Serna et al., 2013)	2 3* 4 6* 7* 8* 9 10 11	Moderate	High	Mostly Europe and USA	Drawing largely from studies of high income countries, employed women had worse SRH than men, alongside worse job security, lower control at work, worse contractual working conditions and shorter hours.
(Bambra et al., 2009)	2 3 4 5 6 7 9 10 11	Low	High	High income countries	Higher quality studies suggested that job insecurity and unemployment resulting from privatisation impacted adversely on mental health and on some physical health outcomes.
(Jin et al., 1997)	9 10	Low	Not reported	High income countries	Most studies reported an association between national employment rates and overall mortality. Individuals who became unemployed reported more symptoms and illnesses.
(Roelfs et al., 2015)	2 3 4 5 6* 7* 9 10 11	Low	Not reported	High income countries and Costa Rica	The background unemployment rate within countries did not explain mortality over and above the individual impact.
(Bambra et al., 2010)	2* 3 4 6 8* 9 10 11	Low	Unclear	Not clear	Legal restrictions on working at height were associated with reduced fall injuries. Reduced legal drinking ages were associated with increased road injuries. Reduced blood alcohol driving limits were associated with decreased vehicle crashes.

^aCritical appraisal criteria: 1 = an 'a priori' design for the review was provided; 2 = a comprehensive search was undertaken (including relevant search terms and at least two databases); 3 = studies were selected for inclusion by at least two independent researchers; 4 = there were clear inclusion and exclusion criteria; 5 = the status of publication (e.g. grey literature) was ignored in the inclusion/exclusion criteria; 6 = the data were extracted independently by at least two researchers; 7 = the scientific quality of the included studies was assessed and documented; 8 = the scientific quality of the included studies was used appropriately in formulating conclusions; 9 = the methods used to combine the findings of studies was appropriate; 10 = the likelihood of publication bias was assessed (if possible); 11 = there were no important conflicts of interest that may have impacted on the conclusions. *denotes a partially fulfilled criterion.

^b Quality assessment: all (very high); at least 2, 4, 7, 8, 9 (high); at least partially 2, 4, 7, 8, 9 (moderate); all others (low).

Other aspects of political economy

There are reviews across several different themes presented in Table 4.7. The evidence for the association between trade and trade agreements and health was mixed and varied across study designs and reviews (Burns et al., 2016; Barlow et al., 2017). There was some evidence from a low quality review that fair trade was associated with higher wellbeing and lower child mortality (Terstappen et al., 2013). Within low income countries, microfinance interventions were associated with lower infant and maternal mortality, and especially amongst the poorest groups (Orton et al., 2016). Extensions to compulsory education were associated with consistent reductions in mortality and improved self-rated health (SRH) (Ljungdahl & Bremberg, 2015). One review examined the health impacts of female empowerment in low income countries, but did not find any relevant studies (Pratley, 2016). Finally, one review considered the health impacts of food subsidies and food programmes on health in the USA and the UK. There was some limited evidence of small improvements in SRH in one study but little evidence of consistent impacts across interventions and settings (Black et al., 2012).

Table 4.7 - Reviews considering the health impacts of trade, microfinance, female empowerment, education and food programmes

Author	Critical appraisal ^a	Review quality summary ^b	Included study quality	Context	Summary of insights
(Burns et al., 2016)	2 4 5 7 8 9 10 11*	High	High	Wide range of countries	Countries with higher levels of international trade have better population health but the direction of causality was unclear and the association varied by study design and scale.
(Orton et al., 2016)	2 3 4 5 7 8 9 10 11	High	Mixed	Most studies from Bangladesh, others from India, sub-Saharan Africa and Peru	Membership of microfinance schemes in low income countries was associated with reduced maternal and infant mortality. The declines in infant mortality were substantial and greater amongst poorer households.
(Pratley, 2016)	2 4 9 10 11*	Moderate	Not reported	Mostly sub-Saharan Africa and Asia-Pacific - in particular Ethiopia and India	There is an absence of evidence of the relationship between female empowerment and maternal and child health outcomes in low income countries
(Black et al., 2012)	2 3 4 5 6* 7 8* 9 10 11	Moderate	Mixed	Specific groups in the USA and GB	US food programme (WIC) was not associated with any change in infant mortality rates there were fewer pre-term births. No change in mortality in families in GB in receipt of family food packages compared to controls (although estimates imprecise). Free fruit/veg from community centre had some marginal improvements in SRH in USA.
(Barlow et al., 2017)	2 5 9 10 11	Low	Not reported	Wide range of low and middle income countries	No consistent association between trade agreements and mortality or life expectancy in low or middle-income countries.
(Terstappen et al., 2013)	2 4 9 10 11*	Low	Not reported	Global south, with most studies being in central America	Fair and alternative trade was reported to be associated with lower child mortality and improved household wellbeing in two studies
(Ljungdahl & Bremberg, 2015)	2 4 5 9 10 11*	Low	Not reported	Wide range of European countries	Extended compulsory education was associated with a mortality change in under 40 year olds of -1.3% (95% CI -2.8% to +0.3%) in women and -2.1% (95% CI -3.6% to -0.6%) in men; and in those aged over 40 years by -0.1% (95% CI -1.5% to +1.4%) in women and -0.7% (95% CI -2.2% to +0.8%) in men. Self-rated poor health changed by -4% (95% CI -0.1% to -7.6%) in men and -1% (-6.3% to +4.0%) in women.

^aCritical appraisal criteria: 1 = an 'a priori' design for the review was provided; 2 = a comprehensive search was undertaken (including relevant search terms and at least two databases); 3 = studies were selected for inclusion by at least two independent researchers; 4 = there were clear inclusion and exclusion criteria; 5 = the status of publication (e.g. grey literature) was ignored in the inclusion/exclusion criteria; 6 = the data were extracted independently by at least two researchers; 7 = the scientific quality of the included studies was assessed and documented; 8 = the scientific quality of the included studies was used appropriately in formulating conclusions; 9 = the methods used to combine the findings of studies was appropriate; 10 = the likelihood of publication bias was assessed (if possible); 11 = there were no important conflicts of interest that may have impacted on the conclusions. *denotes a partially fulfilled criterion.

^b Quality assessment: all (very high); at least 2, 4, 7, 8, 9 (high); at least partially 2, 4, 7, 8, 9 (moderate); all others (low).

4.5 Discussion

Main results

A total of 58 reviews that measured the health consequences of changes or differences in political economy within or across populations were identified and included. There was substantial variation in the quality of the reviews, with 10 assessed as high or very high quality, and in the reported quality of the underlying studies. There were clusters of reviews considering the impacts of economic recession, income inequality, welfare state type, some aspects of employment policy, urban regeneration, housing, healthcare policy and trade. Single reviews were identified that investigated the impacts of food subsidies and female empowerment. Comparing the coverage of these reviews with the underlying theoretical framework of political economy and health, there are widespread gaps including: the contingency of population health on historical and ecological context, culture and societal norms; and some areas of social and public policy (e.g. the impacts of housing policy in relation to availability and tenure). The impact of changes and differences in governance, politics, power and macroeconomic policy on health has been partially addressed by the available reviews, particularly in relation to the importance of the welfare state, but there remain substantial gaps, including the impact of monetary policy, fiscal policy, economic growth, trade policy, and the balance of power between capital and labour.

Based on relatively weak evidence, it seems that social democratic welfare states and states with greater public spending have better overall population health, but there is no clear relationship between welfare state type and health inequalities. In societies where there is systematic discrimination against particular groups, political incorporation and inclusion contributes to reduced health inequalities. 'Neoliberal' restructuring of states is associated with increased health inequalities, and privatisation leads to worse mental health for workers. The better quality

reviews shows that income inequality is an independent determinant of SRH and mortality, with greater income inequality being detrimental. There is some low quality evidence that economic recession is damaging for mental health, SRH and mortality but it is not clear how generalizable this is, especially given the contradictory findings of higher quality primary studies that have not been synthesised on the relationship between recession and adult mortality (Tapia Granados, 2017; Toffolutti & Suhrcke 2019). There is some limited evidence that fair trade policies are beneficial to wellbeing and child health. Extensions of healthcare insurance coverage in countries where no comprehensive universal system exists were generally associated with health improvements, in particular for lower income groups. Similarly, increases in primary care provision, public health spending and cash transfers conditional on healthcare engagement in some low income countries all had positive population health impacts. There is compelling evidence that housing rent assistance and improving the physical housing infrastructure, particularly for low income groups and those living in cold homes, improves health. The evidence on the impact of regeneration programmes is mixed with no clear positive health impact in high income countries. Health and safety policies in the workplace and prohibition of driving under the influence of alcohol are found to improve health. Within low income countries, microfinance initiatives are associated with lower infant and maternal mortality, particularly amongst those living in poverty. Finally, extensions to compulsory education have been associated with reductions in subsequent mortality rates.

There are several areas where there are multiple reviews, and in reviews investigating similar research questions, but with marked differences in the conclusions drawn. This is particularly the case in the reviews addressing the mean population health impacts of income inequality, and in the reviews considering the impacts of economic recession. Many of the reviews in that area were low quality because they lacked a comprehensive search, independent dual screening, critical appraisal of included studies and a lack

of clarity in reporting their methods. Many reviews also addressed subtly different questions, or used data from different time periods or populations to draw less generalizable lessons. The lower quality reviews in these areas have greater value in elaborating the potential theoretical mechanisms and limitations, than they do in clarifying the extent to which the relationship holds true.

Note that there was no review found which examines the relationship between austerity and health generally, or austerity and the stalled mortality trends (described in detail in Chapter 5 to follow) specifically. This was therefore identified for a specific systematic review of the primary literature (which is being led by a colleague and is not yet published). The protocol for this review is available at:
www.crd.york.ac.uk/prospero/display_record.php?RecordID=226609.

It is therefore clear that in areas where there are reviews and evidence to draw upon, political economy matters for population health. This spans the welfare state approach, economic policy, public spending, health and education provision, housing provision and policy, access to credit and specific legislation.

Strengths and weaknesses

This systematic review examines the broad scope of political economy and health. In doing so it lays out a framework for understanding the availability and quality of the available evidence. Moreover, it establishes the areas in which policymakers can be more clearly evidence-informed, the research questions for which high quality systematic reviews could usefully be undertaken, and the areas in which further primary study is required. The review also sets out the importance of political economy for population health and thereby supports a fuller engagement of health researchers in sociological, political and economic debates.

Several of the included reviews restricted their searches to English language only. This Anglo-centric approach to the literature clearly carries a risk that the included studies are not truly reflective of the available literature as a consequence of restrictions placed on the search. The approach of undertaking a systematic review of reviews provides some certainty that there are no reviews addressing the areas of political economy identified here as gaps. However, the absence of reviews in a particular area does not indicate a lack of research in that area, but simply that there has not been a review addressing a relevant question. A systematic review addressing this research question, but which did not restrict to including only reviews, would have quickly become unwieldy in size and scope. There is therefore a greater range and depth in the literature examining the population health implications of political economy than is reflected in the review level studies that are synthesised in this paper. There is also a risk of decontextualizing the underlying primary studies in this review of reviews given the process of abstraction and generalisation inherent in the synthesising process.

Finally, caution is required in interpreting the extent to which the relationships described are causal or not at this level of abstraction. Clearly many of the underlying primary studies seek to determine whether or not there is a causal effect, despite them using a wide variety of techniques to remove confounding, risks of reverse causality, etc. However, it is difficult to assess risks to causality at review level.

How it fits with the existing literature

Political economy is well recognised as centrally important in determining the health of populations (Marmot et al., 2008; Beckfield et al., 2015; Beckfield & Krieger, 2009; Ottersen et al., 2014). The general findings of this study resonate with much of the theoretical work, which shows how social democratic states have managed to mitigate against the deleterious impacts of marketised social relations (Navarro et al., 2003; Muntaner et

al., 2002; Navarro & Shi, 2001). This review is also consistent with the policy reviews which describe how the most effective means of reducing health inequalities is to decrease economic and social inequalities, use legislation, regulation and taxation to restrict unhealthy consumption, and addresses the structural and financial barriers to access to services (Marmot et al., 2008; Macintyre, 2007; Beeston et al., 2013).

Implications

Policymakers should be aware that social democratic welfare state types, countries which spend more on public services, and countries with lower income inequalities have better self-rated health and lower mortality. A wide range of social and public policy is important in determining population health. Research funders and researchers should be aware that there remain substantial gaps in the available reviews. One such area concerns the inter-relationship between governance, politics, power, macroeconomic policy, public policy and population health, including how these aspects of political economy generate social class processes and forms of discrimination which differentially impact across social groups. This includes the influence of patterns of ownership (of land and capital), and tax policies. However, there are several relevant individual studies and policy reviews which represent a good starting point (Marmot et al., 2008; Mackenbach et al., 2015; Beckfield et al., 2015; Raphael & Bryant, 2015; Beckfield & Krieger, 2009; Beeston et al., 2013; Muntaner et al., 2015). For some areas, there are many lower quality reviews which leave uncertainties in the relationship between political economy and population health. It may be that a very high quality review could provide the clarity required, however, it is notable that in contested areas such reviews have not provided closure to the debate even where new primary studies have not subsequently become available (Gøtzsche & Nielsen, 2006; Marmot et al., 2013). There are also areas where the available reviews have identified primary research gaps such as the impact of changes to housing policy, availability and tenure (Thomson et al., 2013).

4.6 Conclusion

Politics, economics, and public policy are important determinants of population health. Countries with social democratic regimes, higher public spending and lower income inequalities have populations with better health. There are substantial gaps in the synthesised evidence on the relationship between political economy and health and there is a need for higher quality reviews and empirical studies in this area. However, there is sufficient evidence in this review, if applied through policy and practice, to have marked beneficial health impacts.

The next chapter moves on to discuss the problem of stalled mortality trends that have afflicted many high income countries from 2010 onwards. This is the population health challenge that the rest of the thesis will address, considering a specific aspect of political economy (austerity) as a potential explanation.

5. The problem of the recent mortality trends

5.1 Chapter synopsis

This chapter describes how the rate of mortality trend improvement across many high income countries dramatically slowed over the last decade. The stalling in these trends was unexpected given that they had consistently improved for many decades, and thus represent a stark public health challenge.

Although many high income countries experienced a slowing in the rate of improvement at around the same time, it was not ubiquitous. Within the UK almost all age groups, and both females and males, had a slowdown in the rate of improvement. The stalling also impacted on almost every cause of death. The stalling on average does, however, obscure a marked increase in inequalities, with mortality rates worsening in the most deprived areas and continuing to improve (albeit more slowly) in the least deprived areas.

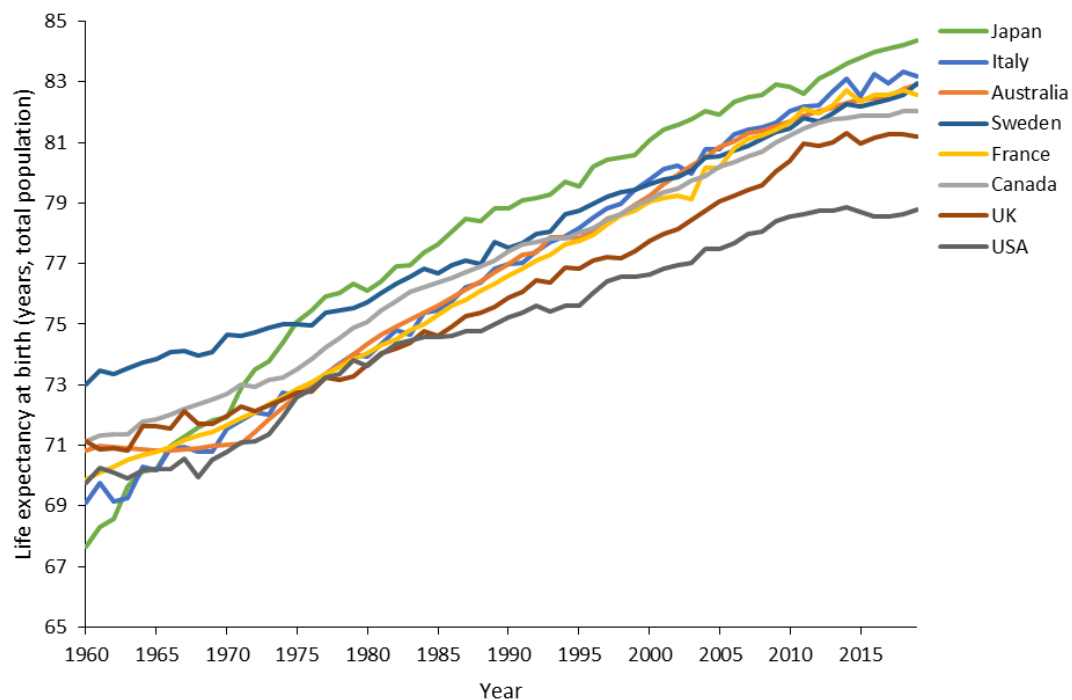
The chapter concludes by considering the hypothesised causes of the stalled trends, and the evidence for each. The role of political economy exposures is highlighted as the most likely explanation, in particular the role of changes in economic policy and the consequences this has had for people's incomes.

5.2 Introduction

The previous chapters have defined health, outlined the theories for what influences the health of populations, and synthesised the evidence on how political economy shapes population health. This chapter introduces the population health challenge of stalled mortality trends during the 2010s across many high income countries. Assessing whether measures of austerity explain this change in trend will be the subject of the subsequent empirical chapters.

Except for periods of war and pandemic, average national life expectancy and mortality rates have demonstrated a long-term trend towards improvement since the 1850s (McCartney et al., 2011; Roser et al., 2013). There have been some exceptions, such as the nations of the former USSR which demonstrated a sudden and dramatic fall in life expectancy in the early 1990s (Shkolnikov et al., 2004; Shkolnikov et al., 2006; Shkolnikov et al, 2001). However, for western and central Europe, and for North America, life expectancy consistently improved between 1960 and 2010 (Figure 5.1).

Figure 5.1 - Long-term trends in life expectancy for selected high income countries (1960-2019, Source: Redrawn from World Bank data)



The improving trend in life expectancy changed for many high income countries around 2011 (Raleigh, 2019; Ho & Hendi, 2018; ONS, 2018; Marshall et al., 2019; Fenton et al., 2019a). Figures 5.2 and 5.3 show the rates of improvement in life expectancy between 1992 and 2016 (inclusive) in five year periods for selected high income countries for females and males respectively. They show that between 1992 and 2011 for most countries there was around 10 weeks of life expectancy improvement for females, and around 13 weeks of life expectancy improvement for males, although this varied somewhat between countries and over time. However, the rate of improvement for the period 2012-2016 was markedly slower for several of the included countries for men and women compared to the earlier time periods. The scale of this ‘stalling’ in the rate of improvement varied across countries. For females, the rates for Northern Ireland, USA, England & Wales, the Netherlands, France, Sweden, Germany and Austria all show marked slowdowns for 2012-2016 compared to the earlier periods. For females in Northern Ireland life expectancy actually declined during those

years (Figure 5.2). For males, between 2012 and 2016, life expectancy declined in Iceland and the USA, and demonstrated a markedly slower rate of improvement in England & Wales, Scotland, Germany, the Netherlands and Sweden (Figure 5.3) (Fenton et al., 2019a).

It is notable that the stalling in the rate of improvement seen between 2012 and 2016 for many high income countries is not ubiquitous. Denmark, Croatia, the Czech Republic, Hungary, Japan and Korea demonstrated similar rates of improvement to most previous time periods for females, as was the case for the Czech Republic, Lithuania, Slovakia, Japan, Estonia and Korea for males (Fenton et al., 2019a).

The deficit in life expectancy in 2017 in the UK, compared to what would have been expected had previous trends continued, is now substantial, and due to a steady deviation from previous trends rather than the influence of any specific year (Minton et al., 2020).

Figure 5.2 - Mean annual change in female life expectancy (in weeks) for selected high income countries (ordered by values for 2012-2016 (Redrawn from data from Fenton et al., 2019a))

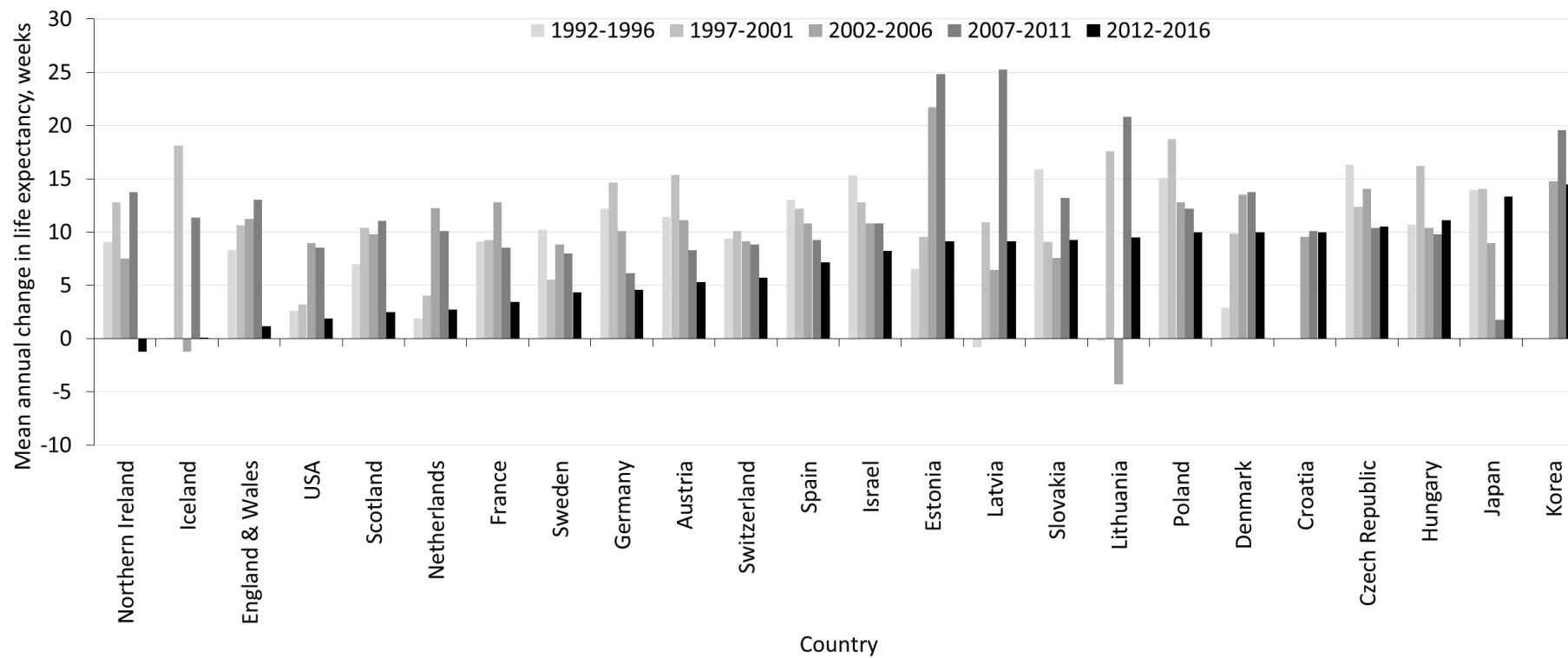
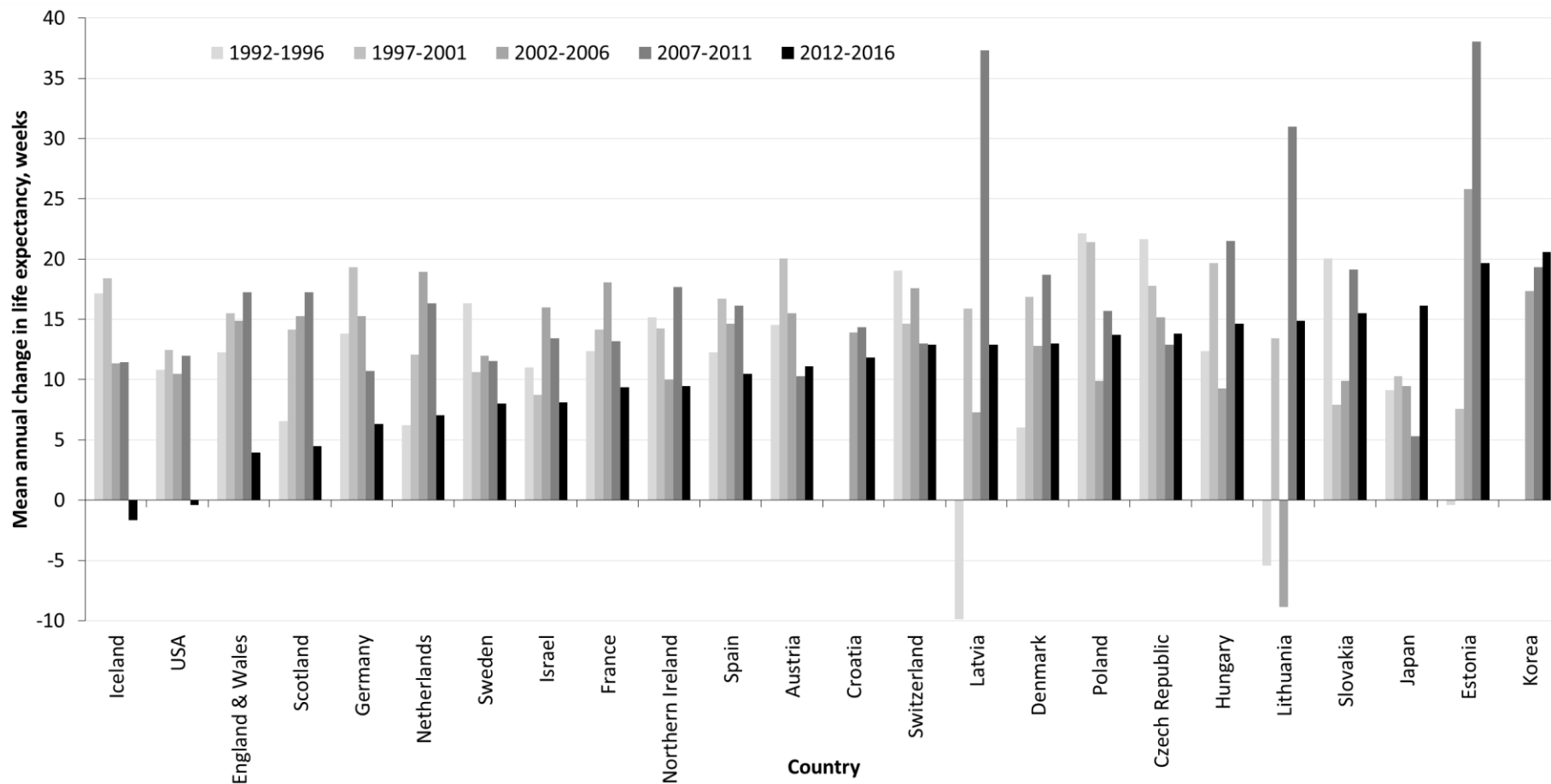


Figure 5.3 - Mean annual change in male life expectancy (in weeks) for selected high income countries (ordered by values for 2012-2016 (Redrawn from data from Fenton et al., 2019a))



5.3 Epidemiology of the stalled trends

Timing

Understanding when the trends changed is important because this informs analyses about the causes of the trends. Clearly, factors that come into being after the change in trends are unlikely to be primary causes.

However, given the year-to-year variation in mortality and life expectancy measures, the precise turning point in a trend can only really be understood in retrospect. Furthermore, in the case of a change in the rate of improvement, rather than a step change, the difference to the pre-existing trend is only slight in the initial years.

In the case of the stalled mortality trends during the 2010s, the change in trend was identified in 2015 in light of higher than expected crude deaths in 2015 compared to 2014, which were initially associated with the influenza epidemic in that year. It was only through more considered analyses of these data, including of age-standardised mortality rates, that the actual change point in the mortality and life expectancy trends were identified.

For Scotland, segmented regression analyses showed that the change in mortality trends amongst men occurred in 2012, and for women in 2013-14 (Fenton et al., 2019a). In England, a turning point of 2011 has been described, but this is based on a qualitative assessment of trends rather than a formal statistical analysis (PHE, 2018). Similarly, analyses of international trends tend to focus on a turning point in the early 2010s, but again based on qualitative assessment of descriptive data (Ho & Hendi, 2018; ONS, 2018). The timing of the changes in trends across high income countries will be empirically analysed and described comprehensively in Chapter 7.

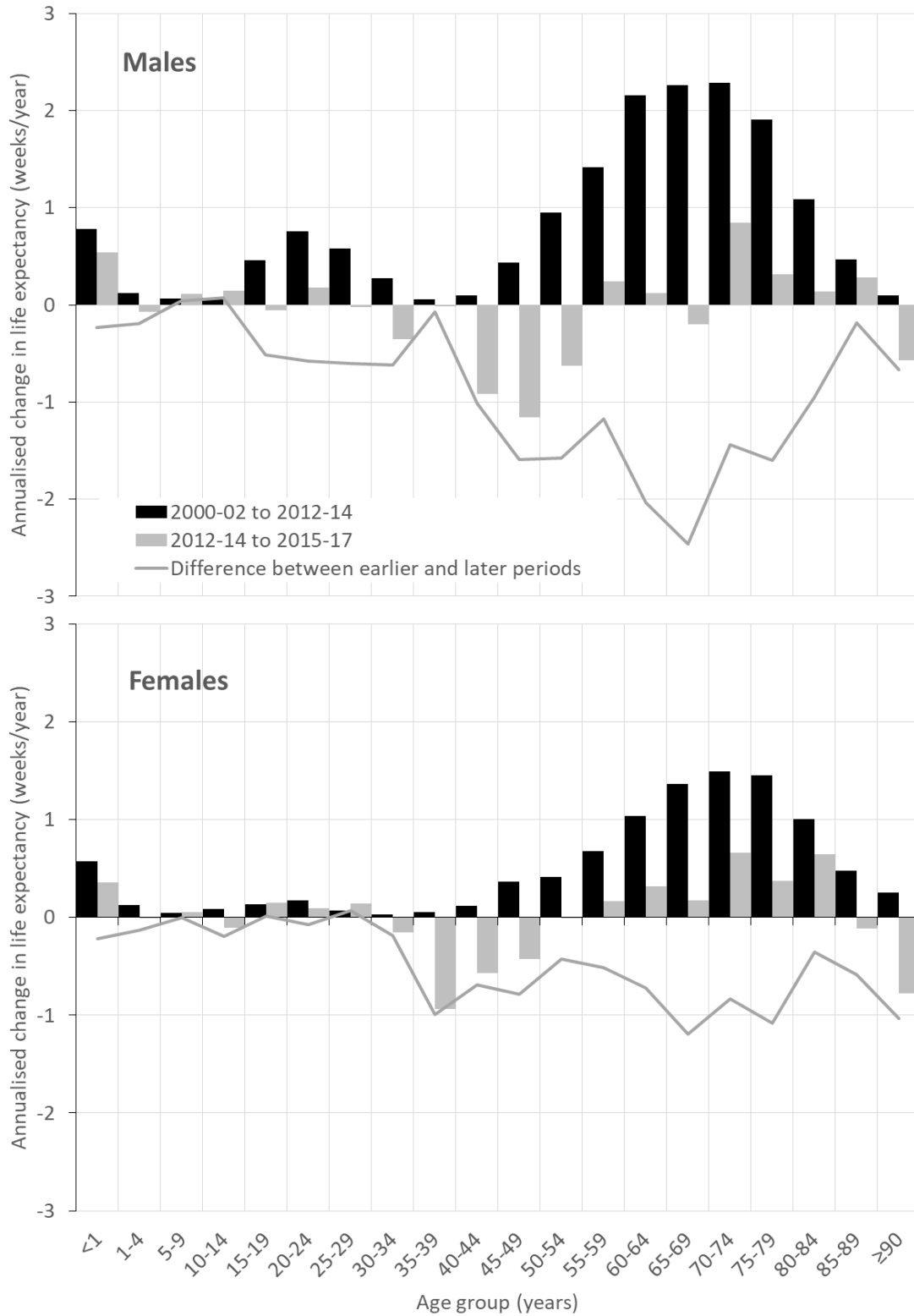
Age and sex

Figure 5.4 shows the contribution of each age strata to the overall changed trends in life expectancy in Scotland (Ramsay et al., 2020). For both females and males, there was declining mortality for all age groups between 2000-02 and 2012-14. Most of the increase in life expectancy was due to rapid improvements in mortality between the ages of 50 and 85 years, partly reflecting the higher number of deaths at those ages which were amenable for reduction. The rate of improvement in this earlier period was greater for males than females.

In the period 2012-14 to 2015-17 there were continuing reductions in mortality for infants and for most of the age strata between 20 and 30 years, and between 55 and 80 years. For women between 30 and 50 years, and for men between 35 and 55 years, mortality rates worsened markedly in this later period. There was also a worsening in mortality rates for females and males aged over 90 years. Overall, for almost every age group, and for females and males, there was a worsening in the rate of improvement (or an actual increase) in mortality in the later period compared to the earlier period.

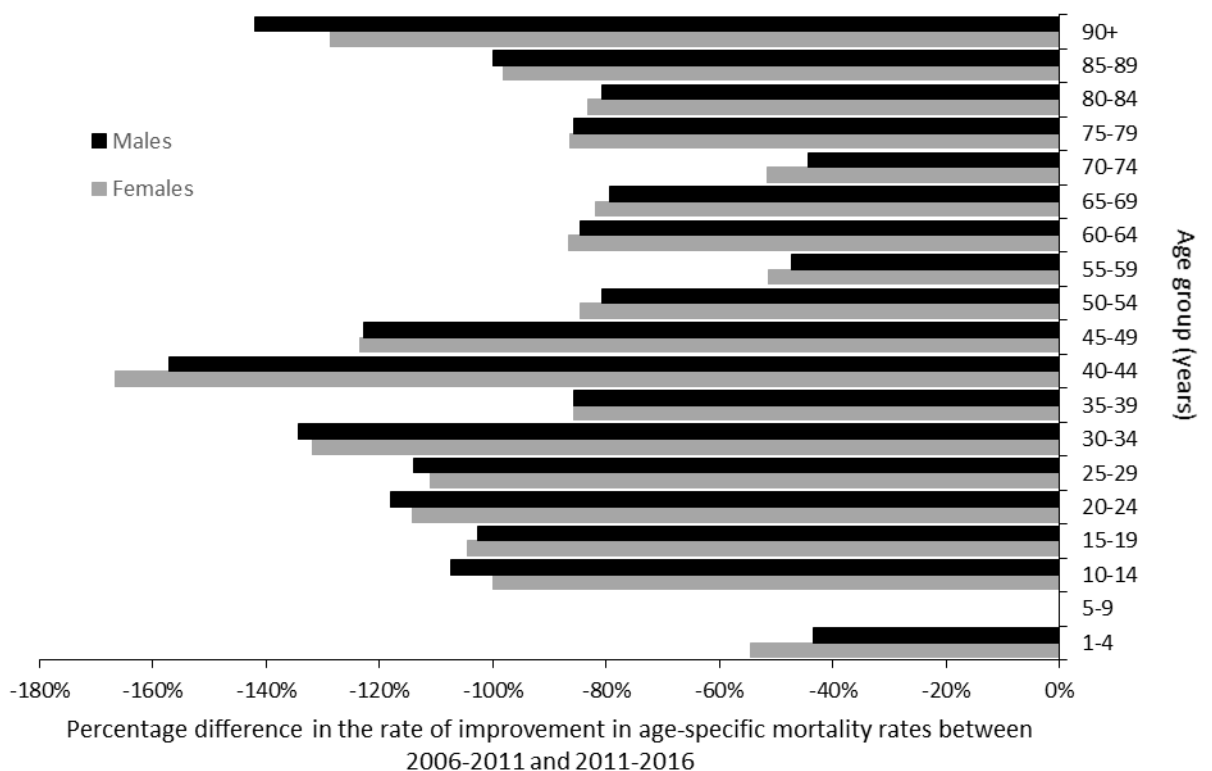
Whatever caused the change in overall life expectancy trends in the last decade in Scotland, impacted on females, males, and almost every age group.

Figure 5.4 - Contribution of changes in age-specific mortality to the change in life expectancy trends, Scotland, males and females (Redrawn from data in Ramsay et al., 2020)



An analyses for England, using slightly different categories for deaths and different time periods than the analyses for Scotland, showed very similar results (PHE, 2018). Figure 5.5 shows that the rate of change in mortality by age and sex in England between 2006-11 and 2011-16 worsened for all age groups (except 5-9 year olds for whom there was no change between the time periods for either sex) and both sexes. The slower rate of improvement was stark for many age groups, and for many of the age groups that contribute most to premature mortality (such as those between 40-49y and 60-69y).

Figure 5.5 - Percentage change in age-specific rates per 100,000 population by age group between 2006-2011 and 2011-2016 in England (redrawn from data in Table 3.1, (PHE, 2018))



In the USA, the decline in life expectancy between 2014 and 2015 was attributed to changes across age groups, but with the greatest contributions

from increased mortality in older women, and increases in mortality amongst middle aged men (Acciai, Firebaugh, 2017). Using longer time trends the substantial contributions from middle aged groups to the overall stalled life expectancy trends in the USA was clearer (Evans, 2018).

Cause-specific trends

Figure 5.6 shows the causes of death contributing most to the change in trends in 2012-14 compared to 2000-02 to 2012-14 in Scotland (Ramsay et al., 2020). In the earlier time period most causes of death were positively contributing to improving life expectancy for females and males, reflecting improving mortality rates. Ischaemic Heart Disease (IHD), other Circulatory causes and Cerebrovascular causes (stroke) were making the most substantial contributions for both females and males in the earlier time period. Some causes, notably drug-related deaths, dementia and Alzheimer's Disease, were however already worsening between 2000-02 and 2012-14 for females and males.

Between 2012-14 and 2015-17 for most causes of death for females, and for almost every cause of death for males, had a slower rate of improvement than was the case in the earlier time period. The causes of death that continued to improve, and to improve more quickly, for females were largely cancers (which generally have a longer lag time between the relevant causes and death than other causes (Thomas, 1988)). Although still improving, there was more than a halving of the rate of improvement in IHD for females and males, and as this contributed so much to the overall life expectancy improvement in the earlier time period, this contributes the largest single contribution to the overall stalling. Some causes of death demonstrated markedly worsening trends in the later time period for both females and males: Drug-related deaths, other Circulatory diseases, dementia and Alzheimer's Disease (Figure 5.5).

In summary, most causes of death contributed to the stalled life expectancy trends. IHD continued to improve, but more slowly, whilst Drug-related

deaths, other Circulatory diseases and dementia and Alzheimer's Disease all worsening in absolute terms in the later time period. The implication is that, in Scotland, the causes of the stalled overall trends impacted not only on females and males, and on almost all age groups, but also on almost every specific cause of death.

Figure 5.6 - Contribution of changes in cause-specific mortality to the change in life expectancy trends, Scotland, males and females (Redrawn with data from Ramsay et al., 2020)

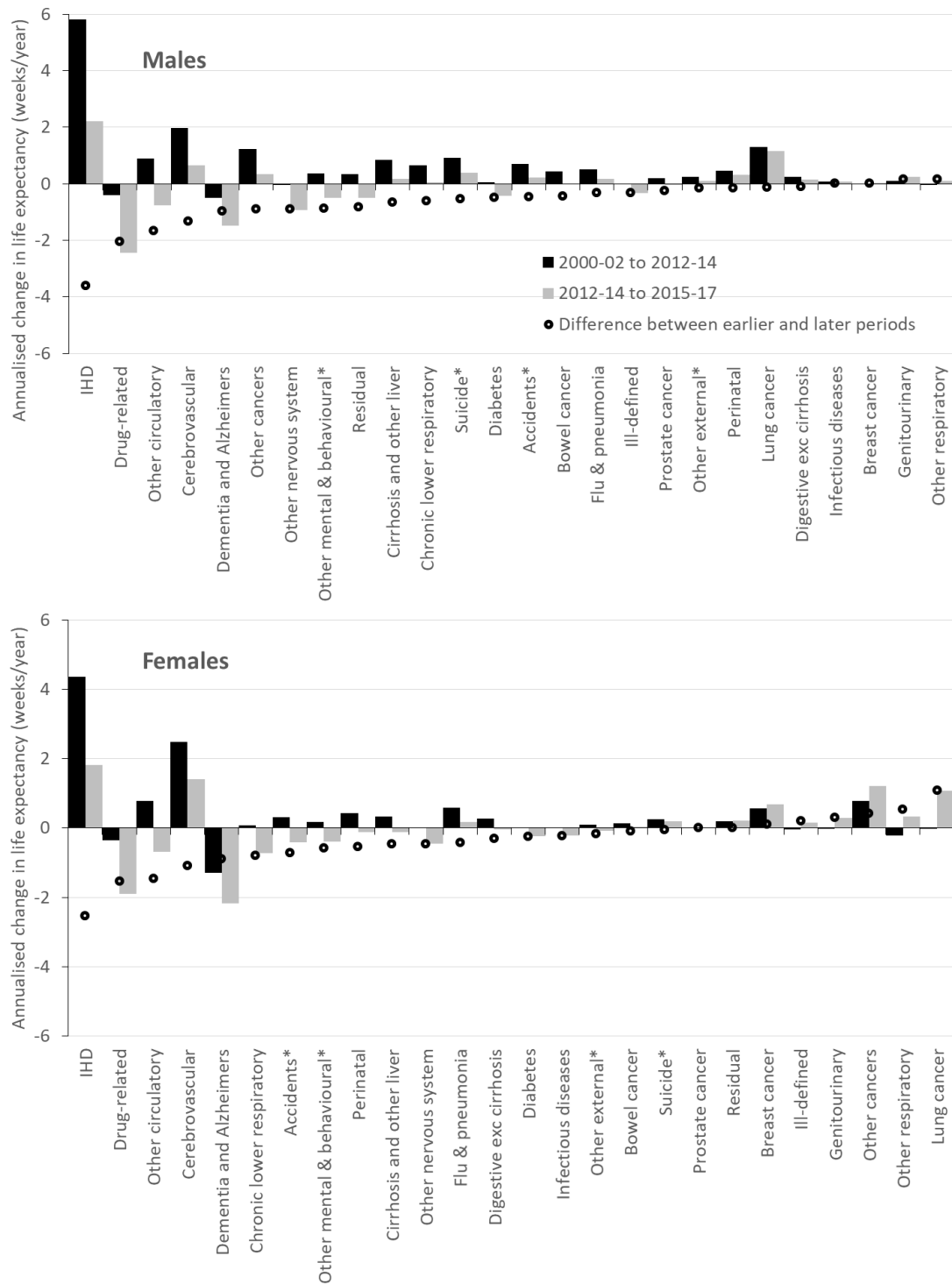
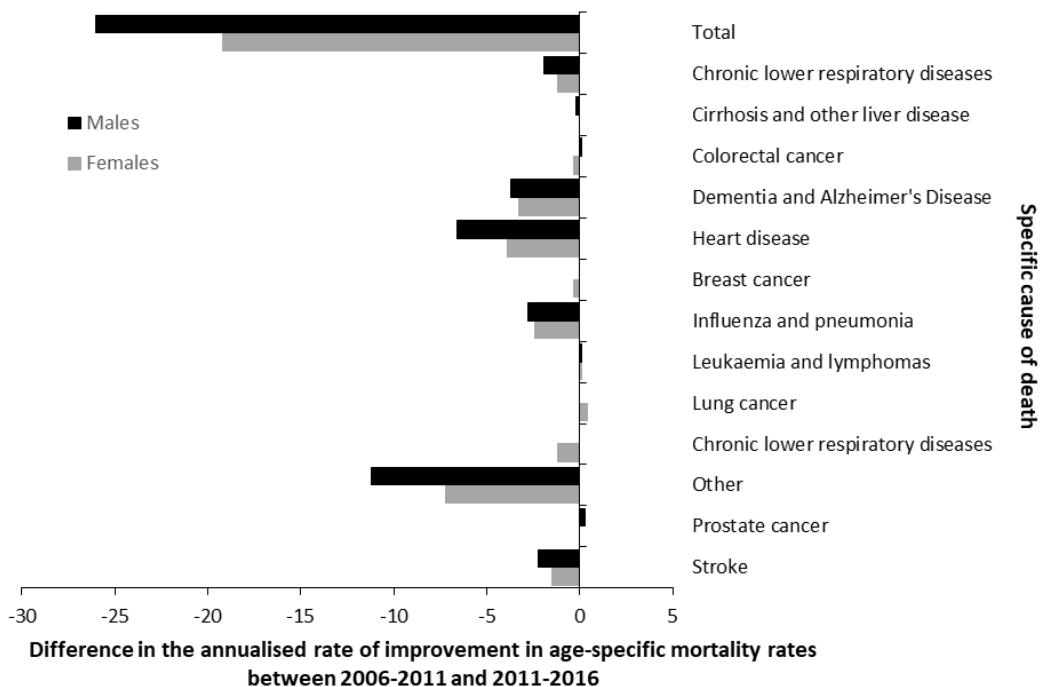


Figure 5.7 shows the annualised change in cause-specific mortality in England between 2006-2011 and 2011-2016. Almost all causes of death showed a slower rate of improvement in the later time period with particularly notable contributions to the overall stalling from ‘Other deaths’ (spread across a large number of causes), Dementia and Alzheimer’s Disease, Heart Disease, Stroke, and Influenza and Pneumonias.

Figure 5.7 - Change in cause-specific and total age-standardised mortality in England 2006-2011 to 2011-2016 (redrawn from data kindly shared from PHE (PHE, 2018))



A similar group of causes was found to be responsible for the stalled trends in life expectancy in Wales with respiratory disease, cancer, circulatory disease and digestive disease the four largest contributors for women; and circulatory disease, cancers, respiratory disease, digestive disease, drug- and alcohol-related conditions, and external causes all making substantial contributions for men (Currie et al., 2021).

Using a different methodological approach, and the data for the year-on-year decline in life expectancy between 2014 and 2015 in the USA, a similarly wide range of specific causes of death were found to be responsible. This included substantial contributions from heart disease, mental illnesses, accidental poisonings, homicide, suicide, and transport accidents (Acciai & Firebaugh, 2017). Other authors considering the USA data over a longer time frame have highlighted the role of adverse trends in drug-related deaths (Barbieri, 2019; Case & Deaton, 2015), suicide and alcohol-related deaths (collectively referred to by the authors as ‘deaths of despair’) (Case & Deaton, 2020). In short, a similarly wide range of causes is contributing to the slowdown and reversal of life expectancy trends in the USA as is the case across the UK nations (Harper et al., 2021). There does not appear to be currently published analyses of the specific causes driving the stalled trends for countries in Europe other than the UK.

Deprivation and ethnicity

This stalling of overall, average, life expectancy trends has masked particularly negative trends for the most disadvantaged groups for several of the countries for which there are data, leading to a rapid widening of health inequalities (Case & Deaton, 2015; Geronimus et al., 2019; Fenton et al., 2019b).

In Scotland, premature mortality rates (i.e. age-standardised mortality for those aged <75 years) are monitored as part of the annual long-term monitoring report for health inequalities (Scottish Government, 2021). This is calculated based on residency within small areas (datazones, with approximately 700 residents in each), ranked by the proportion of working-age adults claiming income and employment benefits. These ranked small area populations are then divided into tenths of the total Scottish population to facilitate surveillance of the extent of health inequalities over time.

The trend in premature mortality for Scotland, for women and men combined, between 1997 and 2019 is provided in Figure 5.8. As expected from the life expectancy trends described above, there is a clear downward, improving trend in premature mortality until around 2014, after which there is little or no change in the rate. The inequality trends in Scotland, shown using tenths of the population ranked by income-employment deprivation, exposes both the three-fold difference between the most and least deprived tenths, but also the divergence in trends after 2014 whereby the most deprived 40% of areas show increasing mortality rates, whilst the least deprived areas continue to improve at a similar rate to that in the earlier period (Figure 5.9). The trends in the Slope Index of Inequality (SII) and Relative Index of Inequality (RII) for these data, which are based on the gradient across these population tenths (and for the RII, divided by the mean in each year) confirm increased premature mortality inequality after around 2014 in Scotland, representing a change in the trend for the SII which had until that point been declining because of the broadly parallel improvement in mortality rates for the earlier period (Figure 5.10).

The contemporaneously available data on mortality inequalities trends is limited to the use of area-based measures of deprivation to rank the population. It is known that most deprived individuals and households do not live in the most deprived areas, and so the gradient across individuals or households ranked by socioeconomic position would arguably be steeper (McLoone, 2001).

Figure 5.8 - Trends in premature age-standardised mortality rates for males and females combined (Scotland, 1997-2019, redrawn from data in (Scottish Government, 2021))

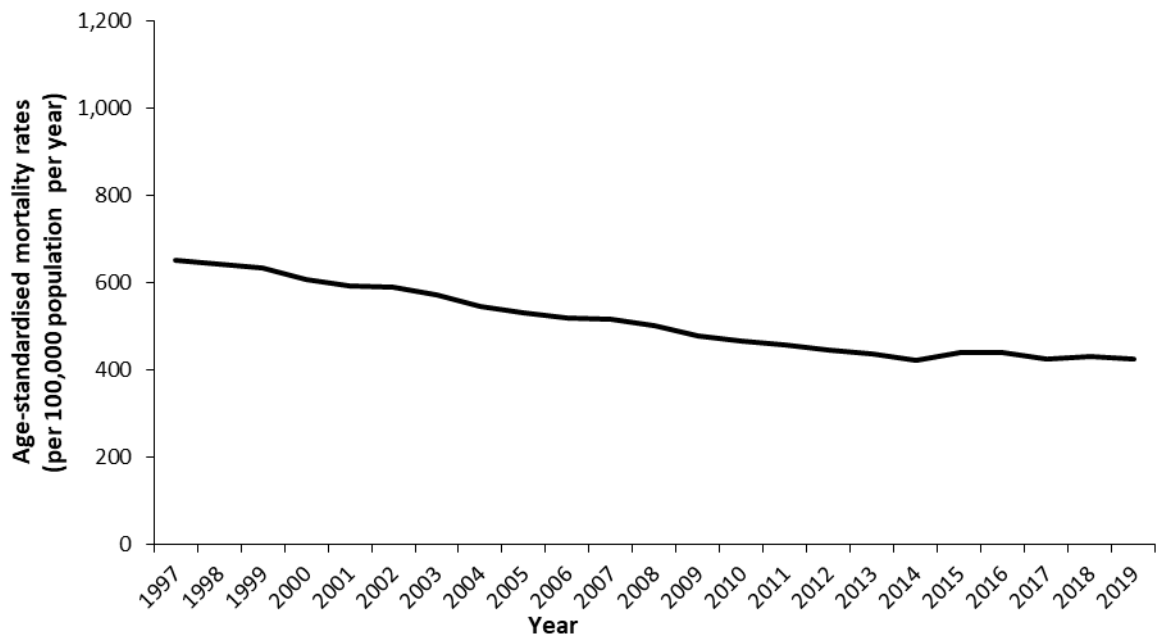


Figure 5.9 - Trends in premature age-standardised mortality rates for males and females combined, by income-employment deprivation (Scotland, 1997-2019, redrawn from data in Scottish Government, 2021)

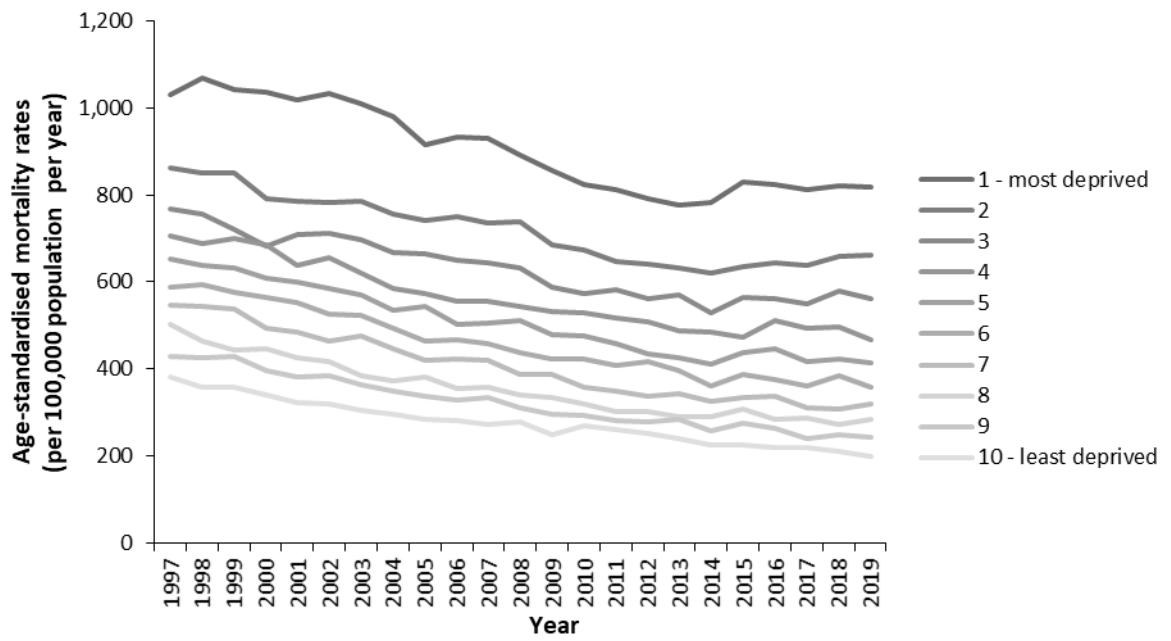
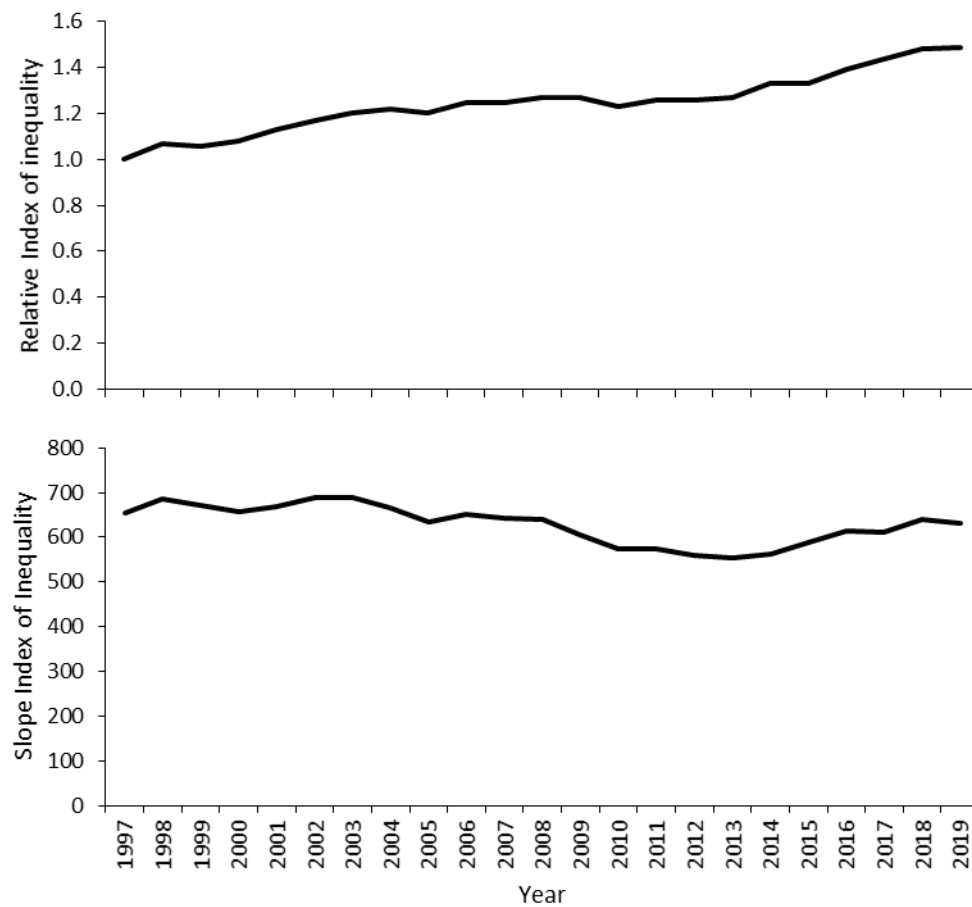


Figure 5.10 - Trends in the Relative Index of Inequality (RII) and Slope Index of Inequality (SII) in premature age-standardised mortality rates for males and females combined (Scotland, 1997-2019, redrawn from data in Scottish Government, 2021)



Case and Deaton have shown that the trends in the USA for the white population are worse than for Blacks and Hispanics (Case & Deaton, 2015; Case & Deaton, 2020), although the mortality rates for Black remains substantially higher than for the white population. More recently, during the COVID-19 pandemic, a disproportionately large negative impact for Blacks in the USA (Siegel et al., 2021; Rentsch et al., 2020), and for Black and Asian ethnicities within the UK, has clearly been shown (Mathur et al., 2021).

To summarise, the stalled trends in life expectancy, although impacting females and males across almost all age groups and causes of death, masks

substantially worse trends for those living in more socioeconomically deprived circumstances.

Morbidity

The focus of this thesis is on explaining the stalled trends in mortality-based measures. However, in keeping the broad definition of health identified in Chapter 2, it would be remiss to make no comment on whether the stalled mortality trends are mirrored by similar trends in broader measures of health (including mental health, morbidity due to any cause, functioning, etc.).

One of the simplest measures of morbidity is the use of survey measures of self-rated health wherein respondents are asked to rate their health on a numerical scale or categorically ordered ladder. These measures are routinely included in most health surveys, and have been validated and made comparable across countries and cultures. Despite this, many self-rated health measures show somewhat surprising results. An example of this is in Scotland where, despite markedly higher mortality and hospital admission rates, self-rated health is higher than in England (Young et al., 2010).

Routine, comparable data across countries on (some of) these broader measures are being developed by teams around the world under the auspices of Burden of Disease studies ("GBD 2019 Diseases and Injuries Collaborators", 2020). These combine a count of the mean Years of Life Lost (YLL, by comparing the age at death to an aspirational standard) with an estimate of the years of perfect health lost to illness (Years Lived with Disability, YLD), to create a combined metric called the Disability-Adjusted Life Year (DALY) which allows the counting of the health deficit for populations in particular years to either premature mortality or ill-health. There was no evidence using this combined DALY measure of any slowing in the rate of improvement globally. However, several countries did display a

change in the rate of improvement on this measure, most notably the USA ("GBD 2019 Diseases and Injuries Collaborators", 2020).

However, these Burden of Disease studies are heavily dependent on proxy data to estimate the actual prevalence trends for populations. For example, the prevalence of many non-fatal conditions is estimated through the trends in the number of people attending healthcare services or using treatments such as pharmaceutical therapies. Clearly these sources of data are subject to changes in availability and acceptability of treatments over time (e.g. in the use of antidepressant medications), as well as any changes in underlying prevalence. For many of the mental health conditions contained within the studies there is a reliance on survey data which in turn is subject to various forms of bias in terms of non-response (almost half of those asked to participate in the Scottish Health Survey, a key source of data for such studies do not do so) and measurement bias (as the tools and instruments are not necessarily sensitive or specific enough to clearly identify underlying mental health conditions). The Gates Foundation funded Global Burden of Disease (GBD) study further relies on synthetic modelling of prevalence for its estimates, using the socioeconomic conditions of the population as a means of predicting the disease burden.

A more nuanced (although currently unpublished) analysis of the survey data on self-rated health measures in Great Britain, and a combined measure with life expectancy ('healthy life expectancy'), shows a quite different trends to those of the GBD study, with falling self-rated health over the last decade and declining healthy life expectancy (Walsh et al, unpublished).

Taken together, it is clear that surveillance of morbidity is a much more challenging endeavour than is the case for mortality. This makes it much less certain what the recent trends in morbidity are, and the extent to which they have changed in line with mortality measures.

The next section discusses the theories that have been offered to explain the stalled trends described above, and summarises the evidence that has been published to support or refute each of these proposed causes.

5.4 Evidence of the causes of the changed trends

Across the UK nations the overall life expectancy trend change was due to a decrease in the rate of improvement (or a decline) for men and women, across all age groups, and almost all specific causes of death, with particularly large contributions due to slower improvements in cardiovascular mortality and increased mortality from drug-related deaths and dementias (Ramsay et al., 2020; PHE, 2018). These changes have impacted most on those living in the most deprived areas, widening mortality inequalities.

Several grey literature reviews and many academic commentaries have now been published offering theories for the cause of the stalled mortality trends (Marshall et al., 2019; PHE, 2018; Hiam et al., 2018; Dorling, 2019; Raleigh, 2018; Murphy et al., 2019). Some of these favoured or foregrounded a single explanation (particularly influenza in the case of the early work from Public Health England; and austerity by Dorling, Hiam and their co-authors) (PHE, 2018; Hiam et al., 2018; Dorling, 2019; Hiam et al., 2017). Other reviews have tried to assess the contributions from different causes as part of a multifactorial explanation (Murphy et al., 2019) or have been more focused on the specific causes of death and the search for medicalised causes (Raleigh, 2019; Raleigh, 2018).

Ageing population and limits to life expectancy

The first data that indicated that there may be a problem with mortality trends in the UK was the publication of crude mortality counts. In 2015 there was a notable rise in the weekly death counts across the UK. These are routinely monitored by the national statistical agencies and by the

teams of public health workers with a particular responsibility for communicable disease control. The standard comparison used by the health protection teams for monitoring was to compare the weekly death counts in any particular week and year with the average number of deaths in the same population for that week averaged across the previous five years (e.g. for week four of 2015, the crude death count would be compared with the average number of deaths in week four of the previous five years).

This approach is helpful for communicable disease control where it is important to understand quickly whether the trends in mortality are deviating from previous years in order to ensure that health and social care services can be adapted to cope with changing demands (e.g. by reducing planned operations to free up intensive care unit capacity for expected demand from influenza-related cases). In 2015 in the UK there was evidence of a substantial and sustained increase in crude weekly mortality which, when interpreted along with other evidence (discussed in more detail below), was attributed to a severe influenza epidemic.

Age-standardised mortality data, and cause-specific mortality data are not available in the UK (or indeed in most countries) so rapidly. This meant that some of the early analysis of the changed mortality trends, analysis that had prompted by the 'spike' in crude mortality in 2015, was reliant on comparisons of crude mortality trends. In turn, this meant that changes in the underlying age structure of the population (i.e. an ageing population) could have been responsible for a rise in crude mortality rates (i.e. deaths per unit population) without a change in the mortality rates in each age group. This led to some hypotheication that some or all of the observed rise in mortality could have been due to an ageing population.

Subsequent publication of age-standardised mortality rates, and of life expectancy calculations, was sufficient to address this concern however, and as a hypothesis for explaining the stalled mortality trends it was quickly able to be excluded (notwithstanding some critique of poorly-standardised

mortality rates in some quarters that did overestimate the stalled trends by failing to consider ageing within broad age strata) (Minton et al., 2017).

A more persistent, but related, hypothesis for explaining the stalled trends has been that there is a 'natural' limit to human life expectancy such that as populations (on average) age, the rate of improvement can be expected to decline. If true, this could explain a stalled mortality trend in high income countries where life expectancy is already high. This hypothesis was rarely explicit in scientific discourse (Dong et al., 2016; Vaupel et al., 2021), but it did regularly appear in private discussions amongst public health researchers and policymakers.

There is some evidence to support this understanding. Amongst high income countries there are analyses showing that as countries improve their mean life expectancies over time there is a decline in the rate of improvement over the second half of the 20th Century (Parry et al., 2018). Part of this relates to the difference in life expectancy gains made by improvements in infant mortality compared to improvements in mortality at older ages. In short, reductions in mortality rates at young ages add more substantially to life expectancy because of the greater capacity to gain years of life cumulatively over the lifespan. Thus, high income countries that have already successfully reduced infant mortality rates to low levels have little room for further improvement as those rates approach zero. Therefore, for life expectancy at birth, maintenance of the historical rates of improvement require faster rates of mortality improvement at older ages to compensate for the lack of room for improvement at younger ages.

This observation, of a long-term tendency to a slower rate of improvement in life expectancy as countries improve their mortality rates at younger ages, is robust (White, 2002). However, the gradual decline in the rate of improvement is very slow (noticeable only over several decades) and occurs in the presence of continuing linear improvements in mortality rates at all

age groups. This is thus distinct from the stalled mortality trends described earlier that have occurred across several high income countries after 2010.

There are several key differences that demonstrate that the recent stalled trends are not due to the longer-term phenomenon of a lack of room for continued improvements in mortality rates at younger ages. First, the change in mortality and life expectancy trends in the last decade is sudden and rapid rather than gradual and long-term. Second, the change in mortality trends is not due in any substantive way to a reduction in the rate of improvement at younger ages. Instead, much of the change in life expectancy trend is due to slower rates of improvement in those aged 50-85 years and worsening trends in mortality for those aged 35-50 and 90+ years. Third, the worsening trends in mortality are seen particularly amongst those living in the most deprived areas who already have the lowest life expectancy. If the attainment of a 'natural' ceiling to life expectancy was important this would be expected to be witnessed first amongst the social groups who already enjoy the highest life expectancy. Instead, the opposite is true. Finally, those high income countries who already have the highest life expectancy globally (including Japan and Korea) have demonstrated continued improvements over the last decade, whilst those high income countries with lower life expectancy (such as the USA) have been amongst those with the most severe stalls (or declines).

Taking this evidence together, the possibility that the attainment of a 'natural limit to life expectancy' is a contributing explanation to the stalled trends in the last decade can be dismissed.

Influenza

As noted above, the first publications warning of the problem of stalled mortality trends were published in the context of high weekly crude mortality counts (compared to the same weeks in previous years) and an influenza epidemic (Hiam et al., 2017; Hawkes, 2016; Newton et al., 2017; Dorling, 2016; Pebody et al., 2018). As a result, many of these publications

were led by communicable disease specialists who were managing the influenza epidemic, and influenza thus dominated these early explanations. A further issue was that 2015 was initially understood as an anomalous year, a 'spike', with the comparator methods emphasising the seasonal nature of the higher mortality.

The importance of influenza was supported by there being a rise in mortality amongst older age groups and the evidence gathered from virological, serological and primary care surveillance systems which all provided some evidence of high influenza activity at the same time as the rise in deaths was observed (Newton et al., 2017; Pebody et al., 2018).

However, there were many weaknesses in the approaches taken to surveillance that subsequently became apparent. First, many comparisons focused on changes between years, or excess deaths in comparison to the previous three or five years. Given that the trends changed around 2012 in the UK this meant that the inflection point was missed, and indeed obscured, by the higher mortality in 2015. Second, the use of crude mortality counts rather than age-specific mortality rates meant that the change in trends at younger ages was missed. Third, the lack of analysis of cause-specific mortality meant that the wide range of specific causes of death that were demonstrating changes in trends was missed (Raleigh, 2018; Hiam et al., 2017; Dorling, 2018).

Influenza, as a specific cause of death with a clear virological explanation (and particularly prior to the COVID-19 pandemic when the importance of social and political factors in influencing the impact of a viral epidemic was not as well understood) was also a much more politically acceptable explanation for the stalled trends than many alternative hypotheses. Influenza could be framed as an unfortunate, but 'natural', cause of higher mortality that was largely outside government control or influence, and as such was not due to political decision-making or mistakes by any particular agency or individual. This is clearly evidenced in the hostility expressed

towards academics questioning the role of influenza in explaining the trends, and also in the difference in the quality of evidence required for different hypotheses for them to be included within official explanations (Pebody et al., 2018; Baker et al., 2018; Hiam et al., 2017; Hiam et al., 2018).

For example, in a Public Health England report summarising the causes of the stalled mortality trends, there is a large section summarising the evidence for influenza as a putative cause, and despite the shortcomings of this evidence base it remained an important explanation in the overall conclusions drawn. In contrast, austerity was not examined as a possible cause, despite widespread discussion and research, with the possibility of it being a cause only being briefly referred to in the discussion. The difference between the research and conclusions being drawn by the wider academic community, and the publications of governments and public health agencies, was stark (PHE, 2018).

For influenza to be a plausible explanation for the changed mortality trends over the last decade several things would have to be true. First, the age groups and causes of death responsible for the changed trends would have to have influenza as a contributing causal force. Second, influenza outbreaks and epidemics would have to have become more frequent or more severe in their impact in the last decade compared to previous time periods. Third, this changed impact of influenza would have to have consistently occurred in some high income countries (such as the UK, USA and the Netherlands) but not at all in others.

There is a further possibility relating to the interaction between influenza and broader social and political changes. A simple example could be that countries pursuing greater austerity, or which have a greater involvement of the private sector in public service delivery, have populations that are more vulnerable to mortality from a similar influenza exposure because of poorer quality services or less health and social care access. Another example

would be that countries that have divergent trends in poverty would have populations that experience different scales of impact with similar exposure, through a range of material, social and psychological mechanisms relating to the embodiment of poverty.

Thus, there are three possibilities in relation to how influenza has influenced the stalled mortality trends. First, influenza has been an important causal force and as such the changing epidemiology of how influenza spreads and impacts has independently contributed. Second, influenza has been an important cause, but only as a mechanism linking broader political economy exposures to mortality outcomes (as has been widely discussed in relation to how COVID-19 has created different scales of mortality across countries (COVID-19 Excess Mortality Collaborators, 2022; Cassana & Van Steenvoort, 2021)). Third, the mortality impact of influenza (whether or not influenced by broader political economy factors) has not changed in scale over the last decade.

The available evidence points towards a combination of the second and third of these possibilities: that there has been a small change in the degree of mortality linked to influenza, but any change in impact cannot be disassociated from political economy. As discussed earlier, the cause-specific mortality trends have changed for almost all causes of death and almost all age groups. The trends for influenza and pneumonia (taken together to avoid coding issues at death which might underestimate the contribution of influenza) have changed, but very much in line with the mean change in trend for all other causes. Influenza and pneumonia do not make a disproportionately large contribution to the changes (as is the case for drug-related deaths). This is strong evidence that influenza is not an important cause of the change in trend. Indeed, the fact that almost all causes of death demonstrate a change in trend at the same time, and across almost all age groups (which is important as influenza is known to differentially impact by age with some strains disproportionately impacting on younger or older age groups), indicates that the likely causes of the

stalled trends lie in political economy rather than in specific disease mechanisms.

There are some caveats to this conclusion, however. Although influenza's most widely understood mortality mechanism is respiratory infection and pneumonia, there is emerging evidence that influenza also contributes to other specific causes of death such as Ischaemic Heart Disease (IHD) (Siriwardena, 2012) and what is often coded as Dementias. Both IHD and Dementias are large contributors to the overall stalled trends, and if influenza was an important driver of those specific causes, it could become a more plausible overall explanation. The evidence around the role of IHD adverse events being triggered by influenza seems robust, but the attributable fraction (i.e. the proportion of IHD mortality that includes a role for influenza) is unknown, as is whether there has been a *change* in the attributable fraction (which would be required for influenza to be a plausible explanation). There is a related issue with the role of influenza in causing deaths coded as Dementias. When an elderly person dies who has been living with a dementia, there is often an understandable reluctance to subject the individual to extensive medical tests during a period of illness (which might include virological or serological tests) or to autopsy after death. As a consequence, there may be insufficient evidence after an individual with dementia dies from influenza for a doctor to put that onto the death certificate. Taken together, these leaves some uncertainty about the true scale of influenza in contributing to changes in the trends for several important, specific, causes of death.

A study by Public Health England (which is as yet unpublished) used the widespread testing for influenza virus of individuals at the point of admission to hospital in England to estimate the trends in subsequent mortality for people infected with influenza. This approach reduces the underestimation of the mortality contribution from influenza for people who die following admission to hospital. It shows that improvement in life expectancy attributable to influenza (defined in this broader way) reduced

to approximately zero having made a contribution to improving life expectancy in previous decades. The estimates are broadly in line with the mean changes in rates for all other causes, indicating that influenza is not making a disproportionately large contribution.

Notwithstanding the possibility of influenza contributing to changes in trends in deaths for IHD and Dementias, there are other causes for which a role for influenza seems highly unlikely. Drug-related deaths have risen exponentially across the UK and in the USA, and a substantive role for influenza in driving that change in trend, independent of social, economic and political causes, is not logical.

More specific evidence from influenza surveillance systems also indicates that there is no consistent circulation of influenza virus in every year over the last decade, nor over all weeks of the year. As such, influenza cannot explain the change in trend observed when only summer months are considered, nor in those years where there has been muted influenza activity (PHE, 2018).

In summary, influenza has arguably had disproportionate weight given to it as an explanation for the stalled mortality trends because the initial evidence of a stalled trend emerged in the context of an influenza epidemic and because it was an acceptable explanation for governments and public health agencies to hypothesise, research and use, because it could successfully depoliticise the stalled mortality problem. There are research challenges for accurately estimating the causal contribution of influenza because of inadequacies in the mortality coding systems, but the best available data do not suggest that influenza has contributed more than any other cause. Since the changed trends are observed across almost all causes of death and almost all age groups, including causes for which influenza can only plausibly play a role as an intermediary mechanism, influenza should not be accorded an important role in any explanatory framework.

Tempo effects

Tempo effects are described as distortions of period life expectancy that can overinflate (at a time of improving mortality for life expectancy) or over-deflate (at a time of worsening mortality) the measure at a time of change because of a temporary disconnect between the sizes of the numerator and denominator in the calculation. In light of this discrepancy, an adjustment has been proposed, based on calculation of a 'Total Mortality Rate' (Sardon, 1994) that more accurately describes the actual period life expectancy at any particular point in time (Bongaarts & Feeney, 2002).

Given the description of the changing mortality trends in the last decade, and the characterisation of a decline in life expectancy occurring between one year and the next on some measures, it was proposed by Murphy et al. that some or all of this decline could be explained by these tempo effects (Murphy et al., 2019).

However, the problem described here is a short-term one relating to a shift in the size of denominators between years. If there is a single inflection point in an otherwise stable time series, these tempo effects may be seen briefly in the following year, but not thereafter. They are further diluted by the use of three-year averaged life expectancy data.

The proposed solutions and adjustments to account for tempo effects are also markedly controversial, imposing as they do assumptions of a series of incremental changes rather than a change in the slope. As such, the utility of using estimates of mortality adjusted for tempo effects is at best unclear.

Mortality shifting across time

A similar issue in the interpretation of time trend mortality and life expectancy data arises from the shifting of the timing of mortality for particular groups of people over time. If there is an intermittent or 'once-off' cause of mortality that selectively impacts on a population group who are more vulnerable (e.g. due to pre-existing multi-morbidity) then this will cause a short-run increase in mortality rates, but in the subsequent years there will be a smaller population of vulnerable individuals for any given age

structure in the population, and this might result in lower mortality rates. The converse is also true, in that a prolonged period of relatively low mortality rates can lead to a greater number of people in a given population who are vulnerable to a new mortality threat, and this can therefore lead to a more dramatic increase in mortality than would otherwise be the case.

In short, in the presence of intermittent mortality risks (such as influenza outbreaks), mortality rates may be higher than expected in the face of such an intermittent risk if in there has been a prolonged preceding period of lower risk, and vice versa. This displacement of mortality over time, forward or backward, theoretically causes greater year-to-year variation in mortality because of the selective impacts of intermittent mortality effects.

This was raised as a possible explanation for the reported decline in year-on-year life expectancy trends in England, with some arguing that the decline was less of a worry because it simply reflects a ‘build-up’ of a more vulnerable population over previous years because of prolonged lower mortality (Murphy et al., 2019).

Although this may provide some explanation for year-to-year fluctuations in mortality rates, this does not provide an explanation for the stalled mortality trends over the last decade. The years leading up to the reported decline in life expectancy in England in 2015 were already displaying a stalling in trends and as such do not support the idea of a prolonged period of a more vulnerable population being accumulated. Furthermore, for the period after the reported decline in life expectancy, there was no substantial ‘bounce-back’ which would be expected if this was an important explanation.

Ischaemic heart disease (IHD)

As noted above, rapidly diminishing mortality rates from IHD prior to 2012 made a very substantial contribution to the improving rates in all-cause mortality and life expectancy. Although IHD continued to improve after 2012, the rate of improvement markedly slowed, and because of the

substantial contribution this makes to all-cause mortality (as the most common single cause of death in Scotland), some researchers have focused particularly on the potential for cause-specific mechanisms to be an important explanation to the overall stalled trends (Raleigh, 2017; Raleigh, 2018).

Specifically, two hypotheses have been offered. First, that an increased prevalence of obesity in the preceding period would negatively impact on the rates of improvement in IHD. Second, that there have been fewer major clinical developments to achieve mortality improvements in the last decade compared to those in previous decades (including statins, thrombolysis, and rapid coronary artery stenting).

Across most high income countries there has been a consistent increase in the prevalence of obesity (Fox et al., 2019) (alongside a rightwards shift in the BMI distribution across the whole population (Tod et al., 2017)) from the 1990s until around 2010. However, estimates of the contribution this has made to all-cause mortality suggest that this is relatively minor (Walsh et al., unpublished).

It is also possible that there have been fewer clinical developments that have achieved a step-change in IHD mortality, although there is an absence of evidence which considers change in the number, effectiveness and implementation of new treatments and interventions. However, the potential importance of this mechanism is somewhat undermined by the divergence between IHD mortality trends between the populations living in the most and least deprived areas. If a lack of new effective treatments was a key mechanism it would be expected that the trends would remain parallel over the last decade. Instead, mortality trends have diverged (Scottish Government, 2021), indicating that social rather than clinical factors are likely to be more important.

Clearly, given the substantial contribution IHD makes to overall mortality in high income countries, any factors that impact on that specific cause is

important. However, the trends in IHD have shown a very similar change to those for other specific causes of death, indicating that a common cause across specific causes of death is most likely.

Austerity

‘Austerity’, as an imprecisely defined description of the UK Government’s policy approach implemented from 2010 onwards, was posed as an alternative explanation to influenza for the stalled trends (Hiam et al., 2018; Hiam et al., 2017; Hiam et al., 2021). The UK approach to austerity was characterised by reductions in public service funding and reductions in the real value of social security benefits, and had entered the public lexicon as a term for this policy approach. Many UK studies thus focused on the role of changes in the social security system, and of changes in public service funding, in explaining the trends (Watkins et al., 2017; Wickham et al., 2020; Taylor-Robinson et al., 2019; Loopstra et al., 2016b; Loopstra et al., 2016a; Alexiou et al., 2021).

In parallel, a wider European debate was taking place about austerity following the ‘Great Recession’ (starting around 2008) which caused substantial government debts to accrue, and in some cases challenged government liquidity for those countries unable to raise funds by printing currency or issuing bonds (the archetypical example of which was Greece) (Varoufakis, 2017).

Thus, two different, but complementary, literatures that have emerged. The UK studies have sought to describe the health impacts of the group of policies implemented under the broad, popular understanding of austerity which focuses on the impacts of reduced government spending on social security and local government. These studies have also linked quite explicitly to the stalled mortality trends within the UK and many have sought to explain the degree to which these policies can explain the trends. In contrast, the international studies have generally used a tighter definition of austerity (as a measure of government fiscal surplus) and have not

necessarily focused on the most recent decade as the time frame of interest (Rajmil et al., 2014; Rajmil & Fernández de Sanamed, 2019; Toffoluttia & Suhrcke, 2019; van der Wel et al., 2018), although some have focused on the implementation of fiscal consolidation following the Great Recession in EU countries (Stuckler et al., 2010; McKee et al., 2012; McKee & Stuckler, 2013; Karanikolos et al., 2013; Reeves et al., 2013; Stuckler et al., 2009b). These literatures are briefly discussed in turn below.

Within England, a fixed effects panel regression study examining the relationship between changes in spending on health and social care services and changes in mortality rates, by local authority, found that spending cuts were associated with an additional c.45,000 deaths (Watkins et al., 2017). Although this study identified a large negative mortality impact of reductions in health and social care spending, there was a limited counterfactual as spending reductions were being implemented across England at this time. These findings were similar to a modelling study of the Scottish population which used evidence around the causal impact of changes in income and employment from the broader literature to estimate the likely impact of changes to the real value of social security benefits, wages, taxes and employment on mortality rates (Richardson et al., 2020). The modelled impact of the tax and benefit changes proposed for the period 2010/11 to 2021/22 was a relative decline in life expectancy of 0.38 and 0.44 years for females and males respectively (Richardson et al., 2021).

The health impacts of specific changes to the social security system introduced in the UK as part of the loosely defined austerity measures were examined in a series of studies. Universal Credit was introduced in 2013 and was broadly welcomed across the political spectrum as a means of reducing the complexity of the system. However, on introduction the real terms value of the benefit was continuously reduced and eligibility was tightened. One high quality study comparing those exposed and unexposed to the introduction of the benefit found that an addition c.64,000 people

experienced psychological distress because of the policy (Wickham et al., 2020).

As hinted above, the net result of the changes in tax and social security policy in the UK during the 2010s led to increases in poverty, and especially child poverty. The distribution of this increase in poverty across England was very similar to the distribution of adverse trends in infant mortality (Taylor-Robinson et al., 2019). Using measures of change in local government spending ecological associations were also noted with homelessness (Loopstra et al., 2016b), old-age mortality (Loopstra et al., 2016a), and life expectancy (Alexiou et al., 2021) across England.

As noted earlier, there is also a literature beyond the UK examining the role of austerity policies on health. Some of these papers were published shortly after the 2008 recession and were focused on specific outcomes such as suicide (Stuckler & Basu, 2013; McKee et al., 2012; Reeves et al., 2013). There was also several works examining the impact of, or summarising the literature on, the likely impacts of recession (as opposed to austerity) (Stuckler et al., 2011; Stuckler et al., 2010; Reeves et al., 2013; Stuckler et al., 2009b; Suhrcke & Stuckler, 2012).

Three papers more specifically examined the impact of austerity (as a measure of fiscal balance) on health. In a high quality study examining policy changes between 1991 and 2013 across EU countries, austerity policies were found to increase mortality rates by 0.7% (whilst also noting that recessions generally decrease mortality rates with the exception of suicide deaths) (Toffoluttia & Suhrcke, 2019). Rajmil & Fernández de Sanamed (2019) restricted their analysis Europe in the period 2011-2015 and found that although mortality rates generally declined, they declined most quickly in those countries with the lowest exposure to austerity (although those with intermediate austerity exposure had worse trends than countries with high austerity exposure). Finally, using self-rated health as an outcome

measure, van der Wel et al. (2018) found that countries that pursued austerity had greater increases in health inequalities.

There is thus evidence of negative health (and social) impacts resulting from specific policies in the UK, introduced as part of what is loosely defined (but widely understood) as ‘austerity’ from 2010 onwards. Using more precise definitions of austerity (as a measure of fiscal balance), there is also consistent evidence of negative health and mortality impacts of more austere policies internationally. However, there is no study currently available which considers the role of austerity in explaining the stalling in mortality trends across high income countries over the last decade.

5.5 Chapter summary

This chapter has described the problem of stalled mortality trends across many high income countries that has arisen over the last decade (and prior to the COVID-19 pandemic). These stalled improvements are surprising and important given the long-term improvements in mortality that had been seen across countries since the end of the Second World War. Although mortality measures represent only a part of what is defined as ‘health’, they obviously remain an important marker of the health of populations.

The stalling in the trends has been particularly profound across the UK nations and the USA, but has also been seen to a greater or lesser extent in many other European countries. However, it has not been ubiquitous, with some countries such as Japan and Korea displaying continuing improvements.

Where analyses have been published (principally for the UK nations and the USA), the greatest contributions to the overall stalled trends for populations have been from a slower rate of improvement in mortality for those aged 50-80 years. However, almost all age groups, amongst both women and men, have seen a slower rate of improvement in recent years. Indeed, in some countries there has been a worsening in mortality trends in some

years, and certainly for some age groups (adults aged 40-55 years), with drug-related deaths playing a prominent role. Almost all specific causes of death have demonstrated a slower rate of improvement in recent years, with substantial contributions from the most common specific causes (such as heart disease and stroke) but also from drug-related deaths and suicide in some populations.

The work to understand the underlying causes of the trends has not brought about a rapid consensus amongst researchers or policymakers (Raleigh, 2019; PHE, 2018; Case & Deaton, 2020; Raleigh, 2018; Murphy et al., 2019). Some of this is explicable by the context of the influenza epidemic in 2015 after which many of the initial statistics on the stalled trends came to light. Indeed, the COVID-19 pandemic may further obscure the underlying causes in the very latest years. However, there has also been a difficulty in reconciling research evidence into the contributions from specific causes of death and the contributions from broader socioeconomic policies. As such, there has been a lack of clarity in the messages relayed to policymakers about the appropriate responses.

Understanding more clearly the relationship between political economy and population health is the subject of this thesis. More specifically, the extent to which austerity has had a causal role in the stalled improvements in mortality trends across high income countries over the last decade. Chapter 7 will describe in more detail the trends in mortality outcomes and the changes in these trends over time, as well as the trends in measures of austerity across countries. These data will then be used in Chapter 8 to assess the causal relationship between austerity and mortality trends.

6. Methods for empirical analysis

6.1 Synopsis of the chapter

This chapter details the methodological approaches taken for the empirical analyses that are presented in Chapters 7 and 8. It begins by summarising the research gap that the empirical chapters will address. There is then a restatement of the research questions. Next, the methods followed for the analysis are described in detail alongside any deviations from the published, pre-analytical, protocol.

The protocol was published in order to fulfil best practice in observational epidemiological research as detailed in the ‘Strengthening the Reporting of Observational studies in Epidemiology’ (STROBE) statement (von Elm et al., 2007). The protocol was published as:

McCartney G, Fenton L, Minton J, Fischbacher C, Taulbut M, Little K, Humphreys C, Cumbers A, Popham F, McMaster R. Is austerity responsible for the recent change in mortality trends across high-income nations? A protocol for an observational study. *BMJ Open* 2020; 10: e034832. <https://doi:10.1136/bmjopen-2019-034832>.

6.2 Summary of the research gap the empirical analyses will address

Recession and austerity

The financial crash of 2007-08, the resulting ‘Great Recession’, the implementation of a fiscal stimulus up until around 2010, and subsequently ‘austerity’ in many countries (Wren-Lewis, 2016), preceded the current change in mortality rate trends. Given the clear links between economic policy and mortality (as summarised in Chapter 5), the changes to economic policy in many countries in the wake of recession has been suggested as an important cause of the recent stalling in mortality trends (Hiam et al., 2018; Heggebø et al., 2018; Leão et al., 2018; McKenna et al., 2017; Wolf & Morrissey, 2017).

Austerity is an ambiguous term, which has only been applied in economic and policy discourse since the 1950s (Anderson & Minneman, 2014). However, as Blyth (2013) details, the policy approach itself has a long history, dating back to the arguments put by John Locke and David Hume. Austerity is associated with fiscal consolidation or retrenchment (i.e. cuts in expenditure and/or increases in taxation). That said, some economists, such as Wren-Lewis (2016) argue that austerity is a particular form of fiscal consolidation which leads to a “noticeably larger output gap” that implies increases in involuntary unemployment and counteracts automatic stabilisers. Thus, for Wren-Lewis, fiscal consolidation need not imply austerity, it becomes a question of degree, and indeed timing. For example, fiscal consolidation during a period of sustained economic growth represents (for many) sound Keynesian demand management, in that it is counter-cyclical and not austerity in the sense used by Wren-Lewis. By contrast, austerity is characterised by fiscal consolidation being applied during a downturn or recession. This is likely to further deflate demand (Blyth, 2013), although there are those who dissent from this argument (Alesina & Perotti, 1997). For this thesis, austerity is thus defined as the suite of policies associated with discretionary fiscal consolidation that acts pro-

cyclically. Austerity may be employed for a number of reasons, including a belief that it reduces government deficits, or is a mechanism for correcting past conditions (Anderson & Minneman, 2014).

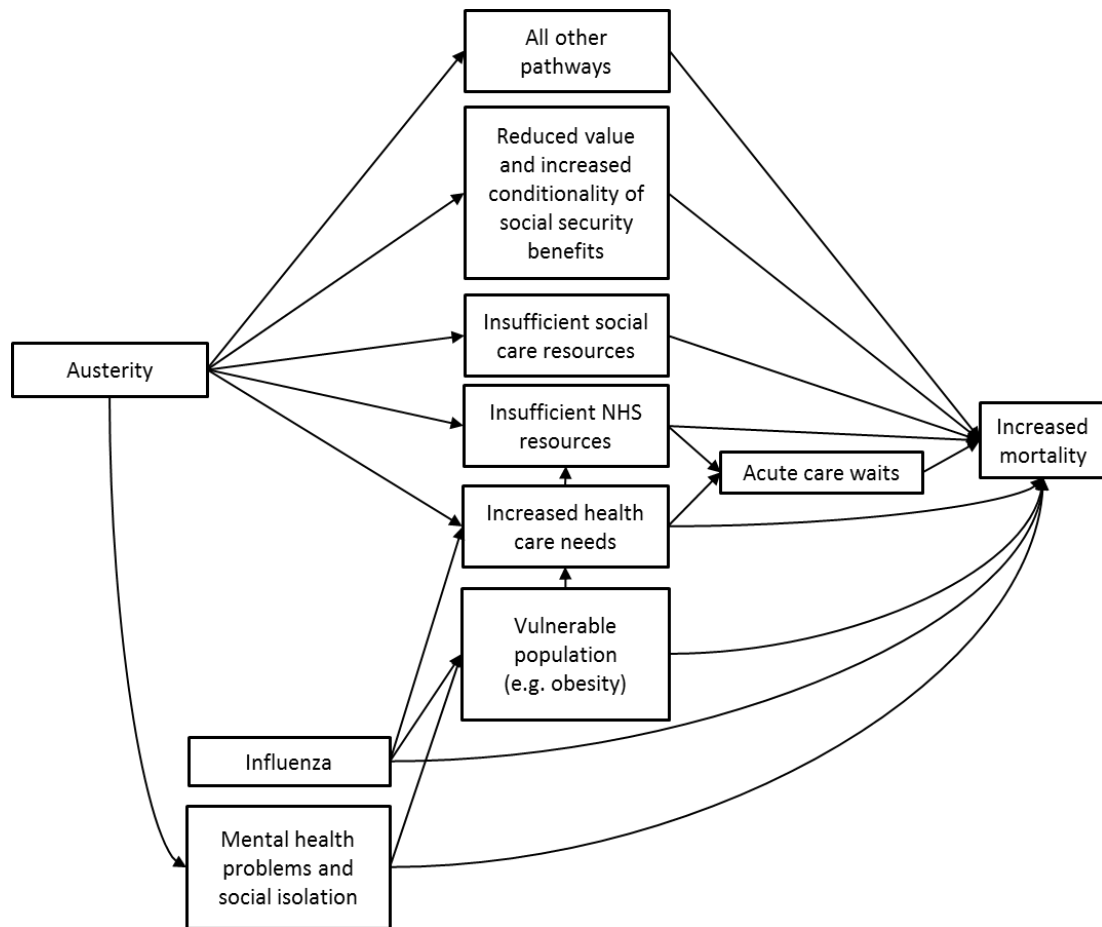
As noted above, some aspects of public spending can increase, even when a government is otherwise committed to an austerity agenda, through the 'automatic stabilisers' within the economy, such as increased spending on unemployment benefits due to an increase in the number of unemployment claimants. Indeed, reducing the spending on such 'automatic stabilisers' in the long-run can be an objective of austerity policies for some politicians. In the recent period, most high income countries pursuing austerity have focused on reducing public spending, rather than increases in taxes (Konzelmann, 2014). As a result, particularly in the UK, these policies have tended to impact most on lower income groups (De Agostini et al., 2016).

The evidence of the impact of economic recession on health and mortality of populations, rather than individuals, is complex and not necessarily negative overall (Floud et al., 2014; Tapia Granados, 2017; McCartney et al., 2019b; Suhrcke & Stuckler, 2012; Toffolutti & Suhrcke 2019). There are several mechanisms through which economic downturns may impact on health. Decreased household and individual incomes can limit the consumption of a range of goods and services that both support health (e.g. paying for heating in the home) and which can damage health (e.g. alcohol) (McCartney et al., 2016; Kawachi et al., 2010). Increased unemployment (as well as under-employment and poor quality work) is well evidenced to be causally related to increased mortality rates in the subsequent 10 years (Roelfs et al., 2011). However, all-cause mortality generally improves more quickly during recessions in comparison to periods of economic growth because reductions in some specific causes of death such as through alcohol-related mortality and road traffic injuries outweigh relative increases in other causes such as suicide (Tapia Granados, 2017; Toffolutti & Suhrcke 2019).

However, the government response to recession is also important for health (McCartney et al., 2019b). In the UK, there have been substantial real-terms reductions in the value of many social security benefits (particularly for those of working-age) and new restrictions on the eligibility and conditionality for receiving those benefits (Loopstra et al., 2016a; Katikireddi et al., 2018; Taulbut et al., 2018). There have also been very substantial reductions in local government funding (Local Government Association, 2018), with greater reductions in England than in Scotland or Wales (Smith et al., 2016). This impacts on a wide range of services, including education, leisure, housing and some support services for those with particular needs (e.g. disabilities or substance misuse issues). A particular impact on health has been proposed through the reduction in the budget available to provide social care services, something that is largely delivered to the elderly either living at home or in residential accommodation (Loopstra et al., 2016b; Loopstra et al., 2016a). It has been suggested that in the UK this meant that fewer people could be adequately cared for outside the NHS, leading to a lower quality social care experience, and increased demand on hospital services. Areas with the largest reductions in spending in England had the greatest mortality rate increases (Hiam et al., 2018; Watkins et al., 2017; Loopstra et al., 2016a).

It is also worth noting that, although austerity has been framed as a distinct hypothesis, it is compatible with many other theories for the stalled trends (Figure 6.1), all of which can be nested within the more general understanding of the links between political economy and health (Figure 3.7). Austerity plausibly sits as part of a long causal chain, or chains, in which other hypotheses fit (Figure 6.1). Indeed, the relationship between austerity and other factors may have complex and non-linear relationships - including the possibility of acting as an effect modifier (Pearce, 2011). Thus, if this study was to find evidence for or against a role for austerity, this does not preclude a role for other factors.

Figure 6.1 - Some potential ways in which the different hypotheses may be related



Limitations of existing research

The difference between exploratory research and causal research, and the risks of conflating the two, have been clearly described in the literature (Academy of Medical Sciences, 2015). A causal approach needs to avoid the risks that can arise from multiple testing within a large dataset in the absence of a clear hypothesis, selective reporting of outcomes or sub-populations, picking particular analytical approaches or baseline time periods without good justification which biases in favour of particular outcomes, or publication bias towards findings that are more interesting or which confirm pre-existing beliefs. There are also risks when different approaches to the data and analysis yield divergent results. For example,

this can occur when the choice of using shorter or longer time periods to compare before and after a change in exposure, or where there are options for which comparison populations to use. There are also risks relating to how data are presented and the extent to which a change in outcomes might be (de-)contextualised from its pre-existing degree of variability.

There is a lack of clear pre-analysis research protocols being published in this area to protect against selective publication or altered analysis approaches after initial work. The risks of this approach are reflected within the current literature examining the causes of the recent slowdown in the improvement of mortality rates. Several studies have suggested that the 'Great Recession' (i.e. the post-2008 economic downturn which occurred across many high-income countries following the financial crash) has been associated with negative health outcomes such as suicide, mental health problems and mortality (Parmar et al., 2016). However, many of these studies have been reliant on very unstable and short baseline periods (Stuckler et al., 2011; Katikireddi et al., 2012; Barr et al., 2012), or have been at risk of analysing only selected outcomes (e.g. only for men) (Reeves et al., 2014). The choice of the baseline period is also very important in determining the magnitude of the recent change in trends, not least because of a period of relatively fast improvement during the late 1990s and early 2000s (Taulbut et al., 2018; Hawkes, 2018).

Where results might change with different decisions about which data to use, over what time series, and with which comparisons and statistical approaches, it is important to be clear on the rationale for those decisions to ensure that they adopt the most robust means of addressing the research question and are at the lowest risk of error, bias and confounding.

Frequently, a lack of good data measuring relevant exposures and outcomes for the populations of interest necessitate pragmatic decisions on the methods adopted, but the extent to which pragmatism has driven research decision-making is not often clear.

To avoid these problems in this area of research, and particularly because of the politicised nature of the implications of findings in this area, it was important to publish a protocol for this programme of work prior to the analysis commencing. This is in line with recent recommendations for the conduct of observational research ("The PLOS Medicine Editors", 2014).

Of the papers published on the impact of austerity and recessions on mortality (Stuckler & Basu, 2013; Tapia Granados, 2017; Parmar et al., 2016; Karanikolos et al., 2013; Reeves et al., 2013; McCartney et al., 2019b; Taulbut et al., 2018; van Gool & Pearson, 2014; Drydakis, 2016; Modrek et al., 2013; Franklin et al., 2017; Margerison-Zilko et al., 2016; Bambra, 2019), recessions are generally found to have negative health impacts for some specific outcomes, but not for overall mortality rates; whilst austerity has negative impacts for both specific and overall outcomes. Although there are studies of the impact of historical periods of austerity, particularly in the UK context (Walsh et al., 2016; Scott-Samuel et al., 2014; Collins & McCartney, 2011), there are only four studies specifically considering austerity (rather than recession) in the post-2010 period (Table 6.1). These also associate greater austerity with relatively high mortality rates, although none use data beyond 2014.

Table 6.1 - Empirical literature relating overall austerity measures and health outcomes

Reference	Exposure	Findings	Quality and interpretation^a
(Rajmil & Fernández de Sanamed, 2019)	Cyclically Adjusted Primary Balance (CAPB) in terciles, Europe (15 countries), 2011-2015	In 2015, compared with countries in the low-austerity group, countries with intermediate austerity had excess mortality of 40.2 per 100,000 per year and those with high austerity had excess mortality of 31.2 per 100,000 per year.	Study at low risk of bias or confounding showing that greater austerity was associated with slower mortality rate improvement in Europe 2011-2015.
(Toffoluttia & Suhrcke, 2019)	Alesina-Ardagna Fiscal Index (AAFI) (also called 'Blanchard Fiscal Index')	Austerity regimes are associated with an increase in mortality of 0.7% after adjusting for recession. Recession is associated with decreased mortality rates.	Study at low risk of bias or confounding showing that greater austerity is associated with worse mortality trends in Europe up to around 2012/3.
(van der Wel et al., 2018)	Spending on social security	Austerity was related to increasing inequalities in self-rated health, with the association growing stronger with time.	At risk of bias due to variable response rates in the European Social Survey across countries. Shows that greater austerity was associated with increasing inequality in self-rated health.
(Franklin et al., 2017)	Mean change in health and social care spending, OECD countries, 2008-2013	Negligible relationship between spending and mortality rates between 2008 and 2013.	Pharmaceutical company funded study with unclear methods showed little relationship between a narrow measure of austerity and mortality up to 2013.

^a No formal quality assessment tool was used but the risk of bias, confounding and conflicts of interest were considered.

In summary, although there is substantial evidence linking austerity policies and the stalled mortality trends within the UK, there is much more limited evidence across countries, and particularly for the most recent period. Studies that examine data up to 2013 find austerity is associated with higher mortality (Toffoluttia & Suhrcke, 2019) and worse self-rated health inequalities (van der Wel et al., 2018). Analyses of austerity and mortality between 2011 and 2015 across Europe also finds a link between austerity and mortality, although the relationship is not linear (Rajmil & Fernández de Sanamed, 2019). There is therefore no available study considering the stalled trends after 2015, nor a wider range of countries beyond Europe.

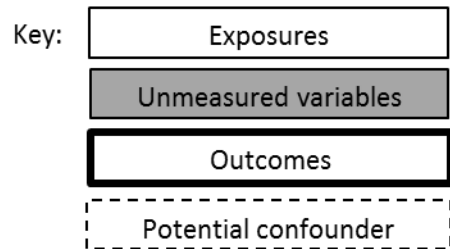
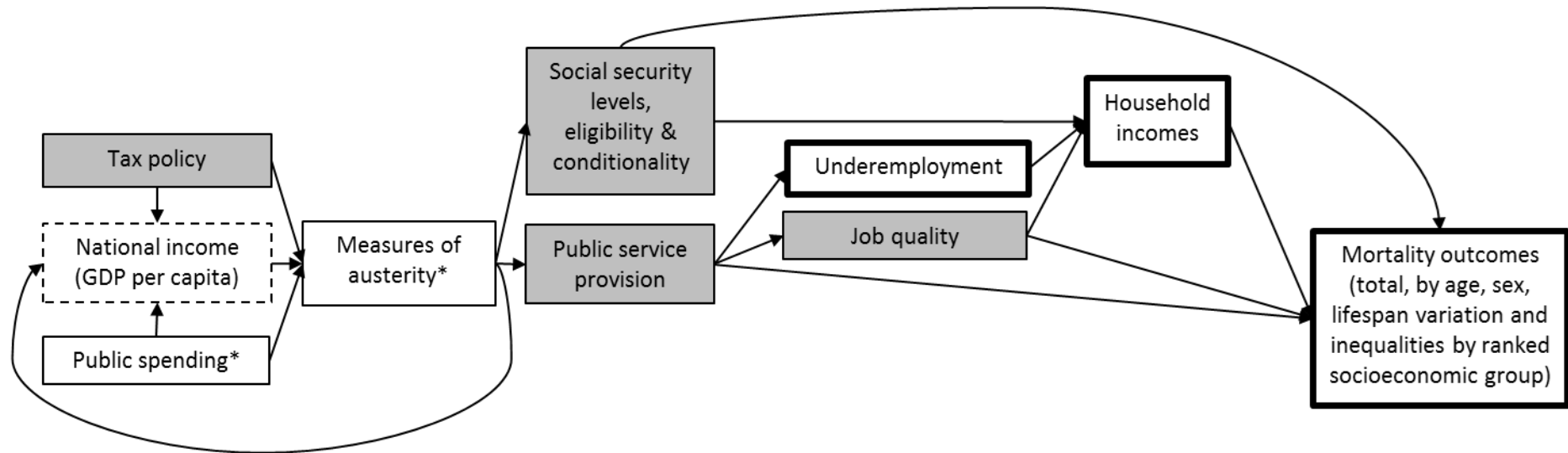
Description of the theory that is to be tested

Taking the general understanding of the relationships between political economy and health laid out in Figure 3.7, and the range of hypotheses proposed to explain the recent stalled mortality trends (Figure 6.1), a more specific and testable theory for the role of austerity needs to be articulated. The analyses to follow in Chapters 7 and 8 study will test the relationships laid out in Figure 6.2. First, it is recognised that there are a range of unmeasured, but time (largely) invariant, confounding factors relating to the welfare state type and broader political economy of countries. Second, changing national incomes, the key indicator of recession, is theorised as both a causal factor in government public spending decisions and a result of government public spending and tax decisions (i.e. austerity or fiscal stimulus). For example, there has been substantial debate about whether the pursuit of austerity causes prolonged economic recessions ((Blyth, 2013; Konzelmann, 2014; Arestis & Sawyer, 2004; Nersisyan & Wray, 2016). However, others have argued that reducing government debt, through austerity, is important to increase economic growth (Reinhart & Rogoff, 2012; Alesina et al., 2019a). Therefore, including Gross Domestic Product (GDP) as a means of adjusting for

recessionary effects on mortality-derived outcomes (and on household incomes and under-employment) risks over-adjustment of the austerity-health relationship because of the potential for austerity to impact on GDP. That said, some of the austerity measures already include some recognition of the impact of changes in GDP either by their calculation as a percentage of GDP, or through inclusion of unemployment adjustment. The approach to how these issues are to be handled in terms of sensitivity analyses is discussed further below.

The other factors in the theory are more clearly linked in a causal direction in the short to medium run. Public spending (overall, on public service provision generally and on specific public services, and spending not on debt repayments), social security policy and personal taxation are all relevant policy decisions that form the overall approach that can be described as more or less orientated towards austerity. Most of these factors have both direct and indirect impacts on mortality outcomes, many through the important mediators of unemployment, wages and household incomes, but also through the changes in the provision of particular public services which could be expected to act differentially on particular population sub-groups (Blyth, 2013; Konzelmann, 2014). The variation in the nature of austerity programmes (e.g. those which might increase taxes on richer or poorer groups, or might cut spending on universal or targeted public services, or those which impact on social security payments differentially by age) might be expected to have different impacts on mortality trends overall, and for specific population groups. However, this more detailed work is beyond the scope of this project, particularly because of limitations in the availability of comparable data. The focus here is on mortality outcomes as an easily measurable outcome, but that is not to downplay the importance of other measures of health (McCartney et al., 2019a).

Figure 6.2 - Theory to be tested linking austerity and mortality outcomes



*These variables are either exposures or unmeasured variables depending on the analysis.

6.3 Research questions

This empirical chapters of this thesis will test the hypothesis that the pursuit of austerity policies (measured in different ways) in high income countries in recent years impacted negatively on a range of mortality outcomes, and on household incomes and underemployment, relative to populations that experienced a different policy approach. The research questions, as detailed in the protocol, are detailed in Table 6.2.

These questions were amended slightly during the course of the analyses. Research question b. could not be fully addressed because of a lack of ranked health data to facilitate calculation of health inequality metrics as defined in Chapter 2. However, to provide some means of considering the differential impacts within each country as a result of austerity, lifespan variation was calculated as a measure of mortality dispersion within population (van Raalte et al., 2018; Seaman et al., 2015). This therefore provides a proxy for health inequality which can be undertaken with the available data.

Table 6.2 - Research questions, null and alternative hypotheses

Research question	Null hypothesis
a. Have higher levels of austerity led to greater negative impacts on life expectancy and mortality rates in high income countries?	Higher levels of austerity have not led to greater negative impacts on life expectancy and mortality rates in high income countries.
b. Have higher levels of austerity led to increases in absolute and relative health inequalities?	Higher levels of austerity have not led to increases in absolute and relative health inequalities.
c. Have higher levels of austerity led to increased underemployment?	Higher levels of austerity have not led to increased underemployment.
d. Have higher levels of austerity led to lower household incomes?	Higher levels of austerity have not led to lower household incomes.
e. Does greater underemployment mediate the relationship between austerity and mortality?	Higher underemployment does not mediate the relationship between austerity and mortality.
f. Does lower household income mediate the relationship between austerity and mortality?	Lower household incomes do not mediate the relationship between austerity and mortality.

6.4 Detailed methods

Design

As the exposure to austerity cannot be manipulated by the researcher, an observational, ecological, ‘natural experiment’ study design was adopted. As the exposure across countries and time in this case is a continuous rather than binary variable, a family of regression models using the country as the unit of analysis was adopted (Craig et al., 2017). More specifically, fixed effects panel models to reduce unmeasured, time invariable, confounding due to pre-existing differences between countries (e.g. welfare state type) was used.

Populations and settings

The sample frame for the study was the total populations of UN-defined high income countries, with sub-group analyses for men, women and specific age groups (<1 year, 1-14 years, 15-29 years, 30-49 years, 50-69 years and 70+ years). The exception to this was that the individual nations of the UK were included separately as mortality-derived outcomes data was available from HMD to facilitate this, but using the UK exposure and confounder data. A wider range of data are shown in Chapter 7 reflecting the available data from the sources, but is done so without prejudice in relation to claims of sovereignty or otherwise.

Exposures

The exposures of interest are listed in Table 6.3 below, detailing the exposure for the primary analysis and the exposures for the sensitivity analyses. It was detailed in the protocol that identification of the timing of the start of the austerity period for each country was to be undertaken by fitting a segmented regression model in R (using the ‘segmented’ package) to identify the first turning point after 2007, using a time series from 1987 (to provide a minimum 20 year baseline period) to the latest data point

available. This year was then to become the point from which the change in exposures and outcomes will be measured.

For each of the austerity measures and the recession measure, the protocol (McCartney et al., 2020b) stated that the cumulative difference from the previous trend was to be calculated and used as the exposure measure. However, as is shown in Chapter 7 to follow, there was no linear trend for three of the four measures of austerity. This necessitated a change in how the measurement of the exposures was undertaken. Therefore, as an amendment to the protocol, the exposures were more simply measured as the value in each year and for each country as part of the panel regression analyses.

It is likely that there is a time lag between changes in the exposure and changes in the outcome. However, the optimal lag time is unknown. Therefore, an initial two-year lag between the exposure and outcome was used, and then varied to zero years (i.e. simultaneous change) and to five years as sensitivity analyses.

Following Toffolutti and Suhrcke (2019), the Alesina-Ardagna Fiscal Index (AAFI) was pre-specified as the primary measure of austerity for the analyses. The basis of this was that this measure was arguably the most sophisticated measure in taking account of the automatic stabilisers in the economy over time. This was derived using the methods specified by Toffolutti and Suhrcke (2019) using data from the IMF whereby:

$$AAFI = (g_{i,t}(U_{i,t-1}) - \tau_{i,t}) - (g_{i,t-1} - \tau_{i,t-1})$$

and:

$g_{i,t}$ = total government expenditure as a percentage of GDP for country i at time t

τ = total government revenues as a percentage of GDP

U = unemployment rate

Three additional measures of austerity were also specified as sensitivity analyses: real per capita Government Expenditure; Public Social Spending as a percentage of GDP; and the Cyclically Adjusted Primary Balance (CAPB). The detailed specification of each measure, and the source data, are provided in Table 6.3.

The pre-analytical protocol specified per capita Government Expenditure data from the World Bank (McCartney et al., 2020b). However, this was an error in that per capita data are not produced routinely for this indicator. Instead, government expenditure from the World Bank, defined as follows, was used:

“General government final consumption expenditure (formerly general government consumption) includes all government current expenditures for purchases of goods and services (including compensation of employees). It also includes most expenditures on national defense and security, but excludes government military expenditures that are part of government capital formation. Data are in constant 2010 U.S. dollars.”

There were two further changes made to the exposure measures described in the protocol (McCartney et al., 2020b). First, Government Expenditures were indexed to 2007 to allow comparison between countries as the levels of Government Expenditures were vastly different because of differences in the size of the population and levels of wealth. Second, year on year change in CAPB, rather than absolute values of CAPB were used, in line with the interpretation of Alesina et al. (2019b).

Table 6.3 - Austerity data definitions and sources

Description	Analytical position	Measure	Definition	Strengths and weakness	Source
Austerity	Exposure (primary analysis measure)	Alesina-Ardagna Fiscal Index (AAFI)	Following Toffolutti and Suhrcke (2019), the AAFI is calculated from the total current government expenditures as a percentage of GDP, unemployment rate, and total government revenues as a share of GDP.	Accounts for fiscal automatic stabilisers and thereby more accurately represents policy decisions, applying data from previous years to generate a counterfactual scenario.	International Monetary Fund (IMF)
Austerity	Exposure (sensitivity analysis 1)	Real per capita Government Expenditure (indexed to 2007)	Real per capita government expenditure (general government final consumption expenditure in constant US \$).	Most intuitive measure of government spending and easily comparable across countries. Does not account for tax changes or automatic stabilisers.	World Bank
Austerity	Exposure (sensitivity analysis 2)	Public Social Spending	Social spending including all financial flows controlled by General Government (including all levels of government and social security funds), as social insurance and social assistance payments, defined as a % of GDP.	Most direct measure of government spending that is likely to impact on health outcomes. May have issues limiting valid comparisons across countries and does not account for tax changes or automatic stabilisers.	Organisation for Economic Co-operation and Development (OECD)
Austerity	Exposure (sensitivity analysis 3)	Annual change in Cyclically adjusted primary balance	Cyclically adjusted balance excluding net interest payments (interest expenditure minus interest revenue).	Accounts for fiscal automatic stabilisers but not changes in asset prices.	IMF

Outcomes

Table 6.4 details each of the outcome measures and the source for the data. Although a wider range of measures of health was desirable, including measures of mental health, wellbeing, functioning and inequalities, only mortality-derived measures were available for a sufficiently large number of countries and recent years.

The age-standardised mortality rates (ASMRs) were calculated from the life tables for each country and year where data was available. The data were standardised to an adapted version of the European Standard Population 2013 (ESP 2013), whereby the age range of the standard population was disaggregated for the older age strata to match the strata provided by HMD (see the Supplementary material for Chapter 6 at the end of the thesis for full details on how this adaptation was made).

It was pre-specified that each of these outcome measures were to be calculated from a start point two years after the year in which a change in exposure occurs (see the analytical approach below for the identification of that year) until the latest available year. It was also specified that all of the outcome data were to be calculated as the mean annual change from the previous trends, to ensure comparability across countries which have differing availability of data after the start of the exposure period and to take into account the potential for differing rates of improvement prior to the recent period. However, because the exposure data did not follow the anticipated linear trends, the data were instead treated as panel data rather than derived single measures for the exposures and outcomes. Thus, instead of the pre-specified approach, the annual data for each country was used as part of panel regression analyses.

For each of the outcome measures described above, the data were analysed using routines in R, and graphed using the `ggplot2` package, to allow the visual identification of any unusual or non-linear trends. Using the 'segmented' package within R, breakpoints in the trends for any of the

outcome measures were formally identified. This was run on the time period between 2000 and 2019 in order to avoid the identified worsening in trends during the 1990s for several Eastern European countries. For the life expectancy analyses Luxembourg had to be excluded because of an unresolvable issue with the underlying data.

Table 6.4 - Data definitions and sources for the outcomes data

Description	Analytical position	Measure	Definition	Strengths and weakness	Source
Life expectancy	Outcome (primary outcome measure)	Period life expectancy at birth	Period life expectancy calculated using the Chiang II method (Chiang, 1979) derived from Human Mortality Database (HMD) data.	Summary measure of mortality rates across the population.	Human Mortality Database (HMD)
Mortality	Outcome (secondary outcome measure 1)	Age-sex-standardised mortality rate	Mortality data standardised to the 2013 European Standard population.	Summary measure of mortality in the population which accounts for age and sex differences in the population structure over time and between countries.	HMD
Mortality	Outcome (secondary outcome measure 2)	Age-standardised mortality rate for men and women and for specific age groups (<1, 1-14, 15-29, 30-49, 50-69 and 70+ years)	Mortality data standardised within sex and age strata to the 2013 European Standard population.	Allows for identification of age-specific effects in the population.	HMD
Lifespan variation	Outcome (secondary outcome measure 3)	Lifespan variation	Lifespan variation calculated as eT for mortality at all ages (Seaman et al., 2015, Shkolnikov, Andreev, 2010).	Allows for a comparison across countries of a proxy measure of inequality.	HMD

Confounders

Time invariant confounders were accounted for by the use of fixed effects panel regression models. As per the theoretical relationships between the variables detailed in Figure 6.2, economic growth/recession, as measured by Gross Domestic Product per capita (GDPpc), was identified as a potential time-varying confounder. However, as noted above, changes in GDPpc is also likely to be a consequence of austerity policies. To avoid over-adjustment, GDPpc was therefore only adjusted for in the analyses as a sensitivity analyses. As a deviation from the protocol and because of the panel regression analysis approach being adopted, GDPpc was operationalised as an annual value for each country rather than the percentage change between 2007 and a subsequent trough as originally anticipated in the pre-analytical protocol (McCartney et al., 2020b) (Table 6.5).

Two other variables were identified in Figure 6.2 as potential mediators between austerity and mortality (and also as outcomes of interest in their own right): under-employment and household incomes. These were therefore adjusted in the models as a crude approximation of the degree to which the relationship between austerity and mortality was mediated by these; but also included as outcomes in their own right (Table 6.5). Under-employment was measured as the percentage share of employed persons who are willing and available to increase their working time and worked fewer hours than a specified time threshold) was obtained from the International Labour Organisation (ILO). Household incomes was approximated using data on household spending (Households and non-profit institutions serving households' final consumption expenditure, purchasing power parity in constant 2011 international \$, per capita) obtained from the World Bank for each country and year. In an amendment to the protocol (McCartney et al., 2020b), household income was operationalised *per capita* to account for changes in the population size of countries over time.

Table 6.5 - Data definitions and sources for confounders, mediators and secondary outcomes

Description	Analytical position	Measure	Definition	Strengths and weakness	Source
Recession	Confounder (only in sensitivity analysis as likely to be a mediator)	GDP per capita	GDP per capita (measured as Purchasing Power Parity (PPP) in constant US \$).	Measure accounts for changes in the population size over time and helps disentangle the impacts of austerity from recession (but likely to be a mediator in the relationship between austerity and mortality-derived measures).	World Bank
Under-employment	Outcome and mediator (secondary outcome measure 4)	Time-related under-employment rate	Measured as the share of employed persons who are willing and available to increase their working time and worked fewer hours than a specified time threshold.	Measure of labour demand which does not depend on individuals claiming benefits. It is limited by being a survey measure with associated response biases.	International Labour Organization (ILO)
Household incomes	Outcome and mediator (secondary outcome measure 5)	Approximated using household spending	Household spending (Households and Non-profit institutions serving households (NPISHs) Final consumption expenditure, PPP (constant 2011 international \$)), per capita.	Comparable measure of spending power which adjusts for currency differences. Spending only approximates for incomes however, as debt and saving behaviour are unmeasured.	World Bank

Analytical approach

A family of fixed-effects regression models were used to estimate the relationship between the exposures and outcomes. The first step of the analysis was to derive the variables that were not available from the data providers (AAFI and lifespan variation).

Second, a simple descriptive characterisation of the trends over time was undertaken by graphing the time trends in each variable over time for each country (shown in Chapter 7). Checks were made for linearity before segmented regression models were fitted for the outcome measures to identify when the trends in exposures that displayed linear trends changed in each country. The rate of change in the mortality-derived measures before and after the identified breakpoints were compared in scatterplots.

Scatterplots of the exposure and outcome measures were produced to check for spurious (e.g. due to two clusters) and non-linear correlations.

Scatterplots were also produced to check the correlations between austerity measures for each country and year to examine the extent to which each of the measures were consistent. These descriptive results are provided in Chapter 7.

The full list of fixed effects panel regression models run, including the sensitivity analyses, are presented in Table 6.7. The protocol stated that an additional sensitivity analysis would be undertaken which limited the time period of interest to two years after the austerity measure returned to baseline. As three of the four austerity measures did not display linear trends this did not make sense. However, two additional sets of sensitivity analyses were undertaken. First, because the countries that have economies dominated by hydro-carbon extraction displayed exaggerated revenue flows that impacted on government budget balances, and therefore measures of austerity, an analysis excluding those countries was undertaken. This was operationalised as excluding countries where 'oil rents' were >9.16% of GDP (i.e. excluding the two largest categories of 'oil rents as a percentage of

GDP' for countries in the latest available year, using data from the World Bank).

Second, an analysis was undertaken which was restricted to periods of economic downturn to try to disentangle periods of fiscal austerity/stimulus that were implemented in such circumstances. The rationale for this was that, given the panel regression approach instead of the originally envisaged analysis which identified and restricted to the post-recession austerity period, the panel regression would include periods of fiscal consolidation both at times of economic growth and economic downturn. This was operationalised by creating a variable for each country and each year that indicated whether GDPpc was lower than any previous year. Thus, a country was defined as being in an economic downturn for all years where GDPpc was declining, and those years where it was increasing but still lower than the peak year.

Ethics

All data used for this study were publicly available, aggregated datasets with no individuals identifiable. There is therefore no requirement for ethical committee approval for the study. The study was lodged within the NHS Health Scotland research governance system (which, over the course of the study was amalgamated into the Public Health Scotland research governance system as part of an organisational change).

Table 6.7 - Fixed effects panel regression models fitted

Model	Exposure	Outcome*	Adjustment	Interpretation
1	Alesina-Ardagna Fiscal Index (AAFI)	Life expectancy	Nil	Primary evaluation of austerity hypothesis.
2	Real per capita Government Expenditure	Life expectancy	Nil	Sensitivity analysis 1 using alternative austerity measure.
3	Public Social Spending	Life expectancy	Nil	Sensitivity analysis 2 using alternative austerity measure.
4	Cyclically adjusted primary balance (CAPB)	Life expectancy	Nil	Sensitivity analysis 3 using alternative austerity measure.
5-8	As per models 1-4	Mortality rates	Nil	Evaluation of austerity hypotheses across primary and alternative measures using mortality rate outcome.
9-12	As per models 1-4	Under-employment	Nil	Impact of austerity on under-employment.
13-16	As per models 1-4	Mean household income	Nil	Impact of austerity on mean household income.
17-20	As per models 1-4	Life expectancy	GDP per capita	Impact of austerity after accounting for recession, but noting the potential for austerity to cause recession.
21-24	As per models 1-4	Life expectancy	Under-employment	Estimate of the mediating role of under-employment.
25-28	As per models 1-4	Life expectancy	Mean household income	Estimate of the mediating role of household incomes.
29-32	As per models 1-4	Life expectancy	Nil	Sensitivity analyses changing lag time to 0 years.
33-36	As per models 1-4	Life expectancy	Nil	Sensitivity analyses changing lag time to 5 years.
37-40	As per models 1-4	Mortality rates	Nil	Sensitivity analyses changing lag time to 0 years.
41-44	As per models 1-4	Mortality rates	Nil	Sensitivity analyses changing lag time to 5 years.
45-48	As per models 1-4	Life expectancy	Nil	Restricted to countries with <9.6% of GDP as oil rent (i.e. restricted to countries not dominated by oil extraction).
49-52	As per models 1-4	Life expectancy	Nil	Restricted to years where GDPpc was lower than the previous peak year (i.e. restricted to periods of economic downturn).

*Life expectancy is calculated for the total population and separately for men and women. The mortality rates are calculated as age-standardised rates for the total population, separately for men and women, and for separate age strata.

7. Where and since when have stalled mortality trends and periods of austerity been evident?

7.1 Chapter synopsis

This chapter describes the public health ‘problem’ of stalled mortality and life expectancy trends and provides data on the periodisation of austerity across countries. As noted in Chapter 2, measures of mortality and life expectancy are not the only measures of health. However, they are readily available, comparable between countries, and provide important information on the health of populations. In the absence of a measure of health by ranked social position available across countries, alternative measures were sought. Lifespan variation is a measure of mortality dispersal within a population which does not require ranking by social position, but which can be readily derived from routinely available mortality data. It is argued that it provides an alternative view of inequality which cannot be dissociated from socially ranked inequality measures (Seaman et al., 2015). Therefore, it provides a useful perspective on the variation in mortality experienced within populations that is likely to be relevant to better (but less readily available) measures of health inequalities.

As discussed in Chapters 3 and 4, political economy is an important influence on the health of populations, and there is some evidence that the recent stalling in the rate of improvement in life expectancy and mortality trends across many high income countries may be, at least in part, due to changes in economic policy (Chapter 5). As described in more detail in the methods (Chapter 6), a first step to testing the extent to which austerity is causally related to these stalled trends is to examine the trends in relevant outcome measures. The outcome measures are: period life expectancy at birth, age-standardised mortality rates (ASMRs) for the whole population of

each country, the ASMRs for each specific age group, lifespan variation, underemployment and mean household income.

The chapter begins with a brief summary of the methods employed. Following this, time trends and breakpoints are identified for each of the outcome measures and exposures. The chapter concludes with an examination of the correspondence of the four austerity measures in each country over time.

7.2 Methods summary

A fuller version of the methods is provided in Chapter 6. Briefly, data were obtained from the Human Mortality Database (HMD) for countries within the sample frame (high income and with data available between 1987 and 2012 as a minimum) for life expectancy at birth. Life tables were also obtained to allow calculation of age-standardised mortality rates (ASMRs) for each year for the total, female and male populations of each country. The data were standardised to an adapted version of the European Standard Population 2013 (ESP 2013). In addition, ASMRs were calculated for specific age groups (<1, 1-14, 15-29, 30-49, 50-69 and 70+ years) to examine whether there were different trends by age. Lifespan variation ($e\ddagger$) for the total population, females and males, for each country and year were also calculated from the life tables. Data on under-employment over time was obtained from the ILO; and data on household incomes (approximated from final consumption expenditure) was obtained from the World Bank. Of the austerity measures only AAFI required to be derived, the others were available from international agencies (Table 6.3). The trends in the exposure and outcomes data were line graphed with segmented regression analyses and scatterplots implemented as appropriate using R packages.

7.3 Results

Note that Tables and Figures enumerated with a prefix of ‘S’ (e.g. ‘Table S7.1’) appear in the supplementary material at the end of the thesis.

Data availability

Table S7.1 details the availability of data on each of the exposures and outcome variables. There were critical outcome data gaps for many of the countries with small populations within the sample frame. The following countries and territories had no data available between 1987 and 2012 for ASMR, age specific mortality, lifespan variation and life expectancy, and were thus excluded from further the explanatory analyses in Chapter 8: Andorra, Antigua and Barbuda, Aruba, the Bahamas, Bahrain, Barbados, Bermuda, British Virgin Islands, Brunei Darussalam, Cayman Islands, Channel Islands, Curaçao, Cyprus, Faeroe Islands, French Polynesia, Gibraltar, Greenland, Guam, Isle of Man, Liechtenstein, Macao SAR (China), Malta, Monaco, New Caledonia, Northern Mariana Islands, Oman, Palau, Puerto Rico, Qatar, San Marino, Saudi Arabia, Panama, Seychelles, Singapore, Sint Maarten (Dutch part), St. Kitts and Nevis, St. Martin (French part), Trinidad and Tobago, Turks and Caicos Islands, United Arab Emirates, Uruguay, and the Virgin Islands (U.S.). Several other countries had missing data for some of the years or for one or more of the outcome measures, but were included in the panel for the years for which data was available. For example: Kuwait has life expectancy data but not the data required for the calculation of ASMR, age-specific mortality or lifespan variation; Germany re-unified in 1990 and thus only has data available from this point; and Croatia only has data from 2002 following the end of the Balkans war.

Time trends: life expectancy

The trends in life expectancy between 1988 and 2019 for the available countries and years are shown in Figures 7.1, S7.1 and S7.2 for the total population, females and males respectively. There are several notable

features of the trends. First, as expected, life expectancy is higher amongst women than men across countries and time. Second, the general trend is for life expectancy to increase over time. However, three East European countries (Estonia, Latvia and Lithuania) had marked declines in life expectancy during the 1990s, and to a lesser extent the 2000s, before returning to improving trends. Several countries also had slower improvements at the start of the time period - notably Australia, Austria, Denmark, Hungary and Slovenia. Finally, several countries seem to have a slower rate of improvement in life expectancy in the most recent years (especially England and Wales, Iceland, Luxembourg, Northern Ireland, Scotland, Slovenia, Spain, the UK and the USA). This latter phenomenon is the stall in life expectancy trends that was described in Chapter 5 and which will be the subject of further analysis in Chapter 8.

To identify those countries where there has been a stalling in the life expectancy trends since the 'Great Recession' more formally, segmented linear regression lines were fitted to the data between 2000 and 2019. With a longer time frame of data (1987-2019) breakpoints were identified for a wider range of years across countries reflecting the trends described in the previous paragraph. Note that p values are not presented for these breakpoints for several reasons. First, there would be a high risk of spurious precision because a large number of models are being fitted and it would be expected that five percent would be deemed significant simply by chance. Second, this would risk confusing the certainty of their being a breakpoint with the scale of the change in slope, p values only providing information on the former. Third, substantial qualitative interpretation of the data are required because the time series for some countries before or after the statistically identified breakpoint is very short (but highly significant). To avoid these issues a qualitative assessment of the breakpoints is made throughout this section to ensure reasonable interpretation.

Figures 7.2, S7.3 and S7.4 show the results of the segmented regression models for the total population, females and males respectively, with the breakpoint for each country indicated with a vertical line. Table S7.2 provides the breakpoint, and regression coefficients for the pre- and post-breakpoint regression lines for each country. This is best summarised in Figure 7.3 which compares the rate of life expectancy improvement for the time periods before and after the breakpoint. This shows that most countries had improving life expectancy during both time periods (i.e. are in the top right quadrant of the scatterplot). However, Lithuania had a declining life expectancy in the earlier time period and the USA, Scotland and Iceland had declining life expectancy in the later period. Crucially, the majority of countries (30/38)^f sit below the line of equality (whereby they had a slower rate of improvement in the later period compared to the earlier period). Furthermore, Northern Ireland, England & Wales and the UK overall had almost no improvement in the later time period.

This pattern is broadly similar for females (Figure S7.5) and males (Figure S7.6) as for the total population, with the majority of countries displaying slower rates of improvement in life expectancy in the later time period, but with some important differences. For females, only Scotland stands out as having a life expectancy trend substantially falling in the later period, although Iceland, the USA, England & Wales and the UK overall have rates that are broadly static. The decline in life expectancy for Lithuania was smaller for women than for men, and there was a notable and substantial drop for women within New Zealand in the earlier period (Figure S7.5). The earlier trends for males in Estonia are very different from those for women and seem to indicate a substantial declining trend. However, this should not be over-interpreted as this reflects only a single data point prior to the break point identified by the model in 2001. The earlier trends amongst

^f Note that the UK is included here despite its constituent countries (England & Wales, Northern Ireland and Scotland) also being included. Removing this double counting would mean 29/37 countries being below the line of equality.

Lithuanian males was worse, and the trend for New Zealand markedly better, than for females in the same countries. In the later period the trends for males in Iceland looks particularly bad, but this is again dependent on a single data point identified by the model and should not be over-interpreted. The trends for males in Scotland, USA, the Czech Republic, and Iceland (albeit for a single year), all declined in the later period (Figure S7.6). The precise break points and slope coefficients for each country for the total population, males and females, are provided in Table S7.2.

Figure 7.1 - Trend in life expectancy at birth for the total population (1987-2020, note shortened y-axis)

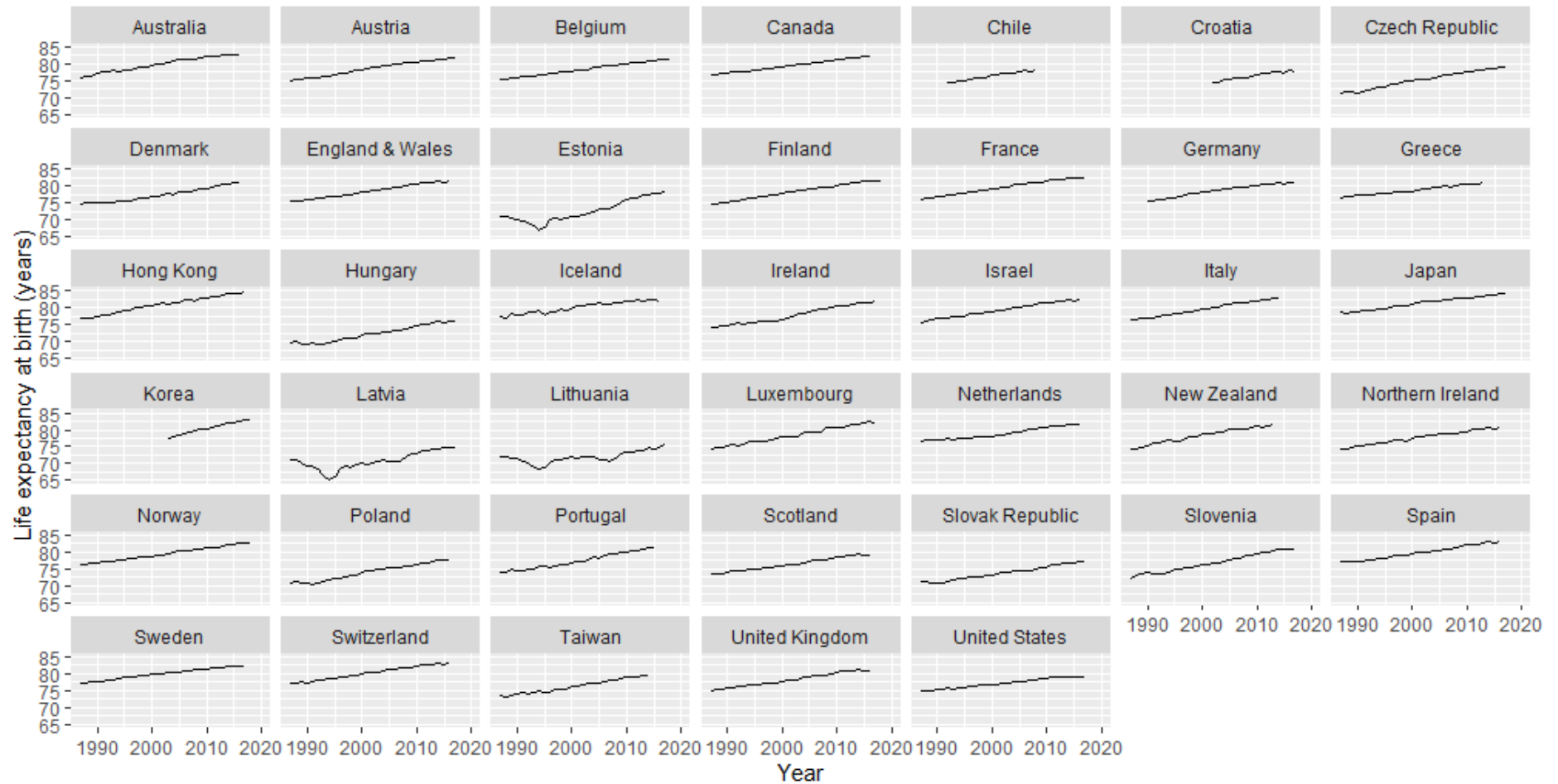


Figure 7.2 - Segmented regression fitted to the life expectancy trends for the total population (2000-2019, note shortened y-axis, vertical lines indicate breakpoint)

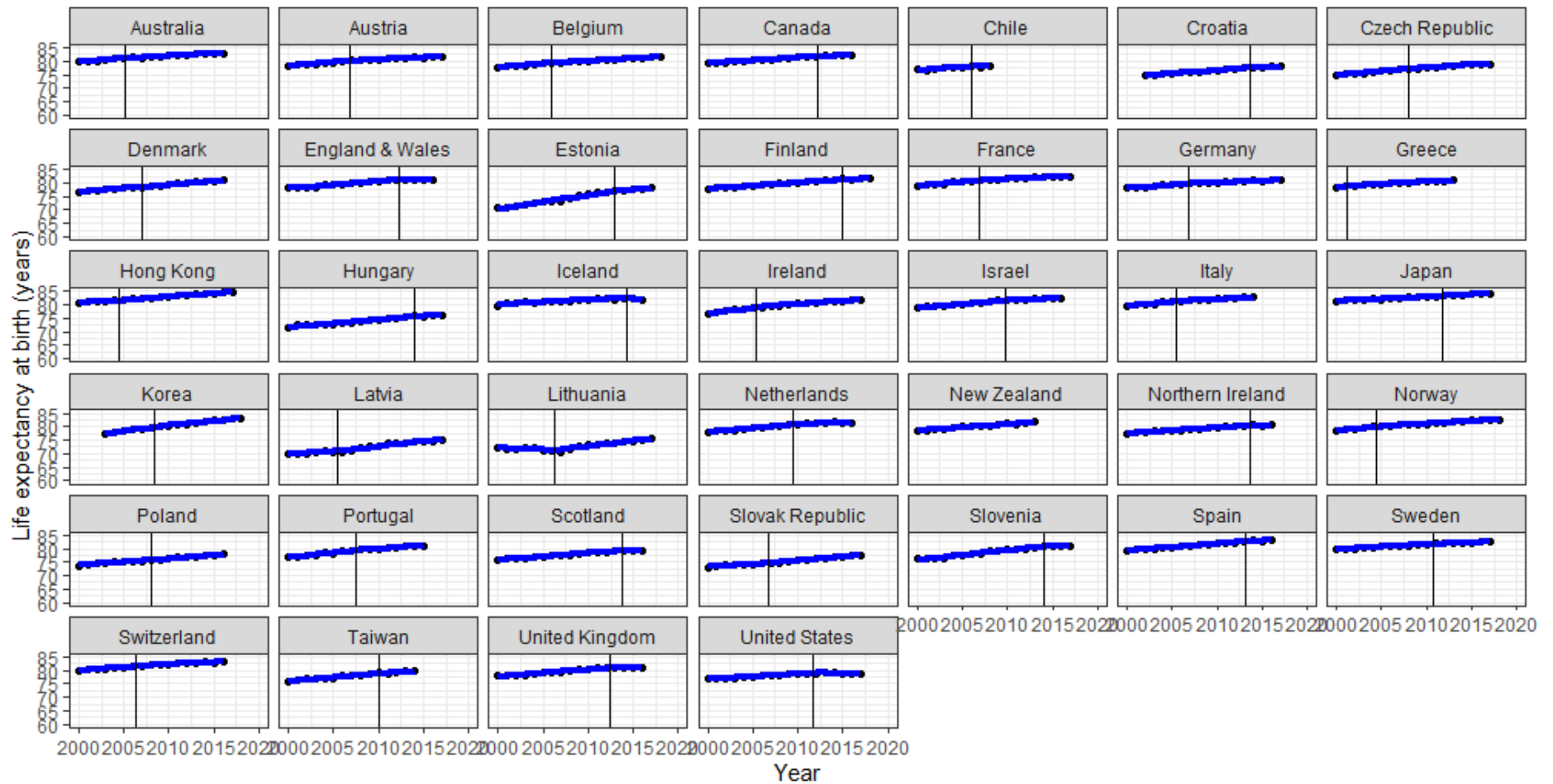
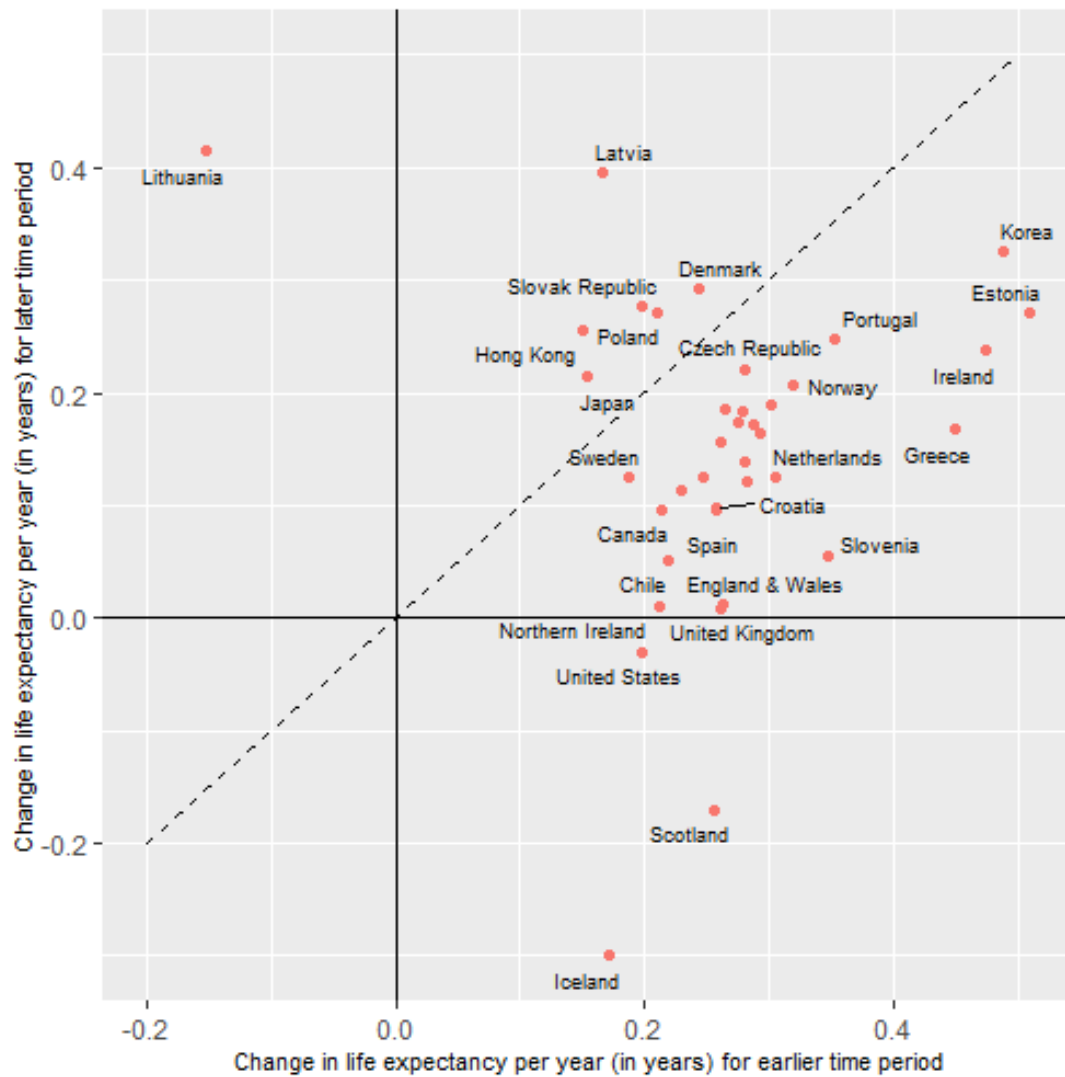


Figure 7.3 - Scatterplot of the rate of change in life expectancy for the total population before and after the breakpoint (2000-2019)



Time trends: Age Standardised Mortality Rates (ASMRs)

All ages

The trends in ASMRs for all age groups combined within each country were very similar to those for life expectancy, but in mirror image (Figures 7.4, S7.7 and S7.8). ASMRs were higher for males than for females, generally declined over time, but with some anomalous trends in Estonia, Latvia and Lithuania (which had increases in mortality during the 1990s). Again, similar to the life expectancy data, there were some periods of slower improvements for several countries towards the end of the time series (e.g. England & Wales, Scotland, the UK overall and the USA).

Fitting segmented regression lines to the data from 2000 onwards confirms breakpoints in the trends in the same way as for the life expectancy data (Figure 7.5 and Table S7.3). Breakpoints for the total population life expectancy data could not be identified for Chile, Hungary or New Zealand (although these were identified for males in Hungary and New Zealand). There was a wide range of breakpoint years identified across countries, with the UK and USA at 2011 and 2010 respectively, Finland and France earlier (at 2006 and 2007), and Greece, Luxembourg and Spain later (at 2013, 2016 and 2013).

Figure 7.6 summarises the scale of change in the ASMR trends for the total population of each country before and after these breakpoints. For ASMRs, data points in the bottom left quadrant indicate that the trends were improving before and after the break points, with points to the upper left of the dashed line indicating that trends improved more slowly (or got worse in absolute terms) in the later period. Only four countries (Hong Kong, Slovak Republic, Latvia and Lithuania) improved more quickly in the later period, indicating a widespread slowing in the rate of improvement. Greece, Luxembourg and Poland were identified as having worsening trends in the

later period, but Luxembourg (and to a lesser extent Poland) are based on very short time periods after the identified break point.

The segmented regression trends for ASMRs for females and males are shown in Figures S7.9 and S7.10, and the scatterplots of the rate of change in ASMRs before and after the breakpoints in Figures S7.11 and S7.12. The identified breakpoints were very similar for women and men (and to the breakpoints for the total populations), but slightly fewer countries had a deteriorating trends for men than was the case for women.

Age-specific ASMRs

Figures S7.13-S7.15 provide the trends in Infant Mortality Rates (IMRs), the mortality rates for those aged <1 year, for all countries for the total population, females and males respectively; and Figure S7.16 the segmented regression analysis for the total population aged <1 year. As for the ASMRs at all ages, the IMRs tended to decline over time, although Latvia and Lithuania again displayed worsening trends for a time during the 1990s. The trends were very similar between infant females and infant males. Figure S7.17 indicates that the later trends tended to be worse across the majority of countries, with some demonstrating worsening trends in both time periods. However, for some countries (e.g. Latvia) this was based on a very short post-break point trend. It is also notable that, for all countries, the IMRs are approaching zero and it might be expected to some degree that trends would not be linear.

The ASMRs for 1-14 year olds fluctuated substantially from year to year, especially in the countries with a smaller population such as Iceland and Luxembourg, but the trend was generally an improving one for all countries for the total population, and for males and females separately, throughout the time series (Figures S7.18-S7.20). Similar to the IMRs noted earlier, the ASMRs for this age group were tending towards zero in many countries and so it was not a surprise that the breakpoint analysis (Figure S7.21) revealed

that most countries had a slower rate of improvement in the later time period than in the earlier one (Figure S7.22). Again, the outlying values for the changes in trends require to be interpreted with caution because of the short time series for either the period before or after the breakpoint (e.g. Iceland).

The ASMRs for 15-29 year olds were more stable over time and displayed the same peaks in mortality for Estonia, Latvia and Lithuania seen in the life expectancy data during the 1990s, for women and men, but with higher peaks in men (Figures S7.23-S7.25). Several countries seemed to display a more rapid period of improvement during the 1990s than before or after (e.g. Czech Republic, Hungary, Italy, Switzerland); and there was some evidence of a stalling in the latest time period across a small number of countries (e.g. Canada, Spain, United States). This was confirmed by the identification of breakpoints after 2012 for many countries for this age group (Figure S7.26), and the evidence of increasing mortality for 12 countries in the later period (Figure S7.27).

For those aged 30-49 years, there was again evidence of a 1990s peak for Estonia, Latvia and Lithuania, but also for Hungary and to a lesser degree Poland (Figure S7.28-S6.30). These peaks were all more marked for males than females. The segmented regression analyses again identified changes in trends across countries with most countries experiencing slower improvements in the later period (Figure S7.31), and some, such as Scotland and the USA, experiencing markedly deteriorating trends (albeit with fairly short time series) (Figure S7.32).

The ASMRs for the populations aged 50-69 years demonstrate the now familiar 1990s peak for Estonia, Hungary, Latvia and Lithuania (and to a lesser extent Poland and the Slovak Republic), with generally declining trends for all other countries and times (Figures S7.33-S7.35). For many countries there is evidence of a stalling in improvements after 2010 (Figure S7.36) and increasing rates for some (e.g. Scotland and USA, also

Luxembourg but for a very short time period). Again, the majority of countries experienced slower improvement (or worsening) in the later time period, with only two (Latvia and Lithuania, both of which had early breakpoints) displaying faster improvement (Figure S7.37).

For the oldest age group (70+ years), there was a much steadier decline in ASMR over time, with much more muted increases or stalls in the 1990s (for Estonia, Latvia, Lithuania, and also Denmark) compared to the trends for younger age groups (Figures S7.38-S7.40). There are several countries which have trends suggestive of slowed improvements in the most recent years, and this is confirmed by the segmented regression analyses (Figure S7.41). Although most countries had improving trends before and after the breakpoint, the majority again had a slower rate of improvement in the later time period (Figure S7.42). The outliers (Croatia and Poland) are due to there being a very short time period either before or after the breakpoint, making the comparison less stable.

Figure 7.4 - Trends in ASMR for the total population (1987-2020)

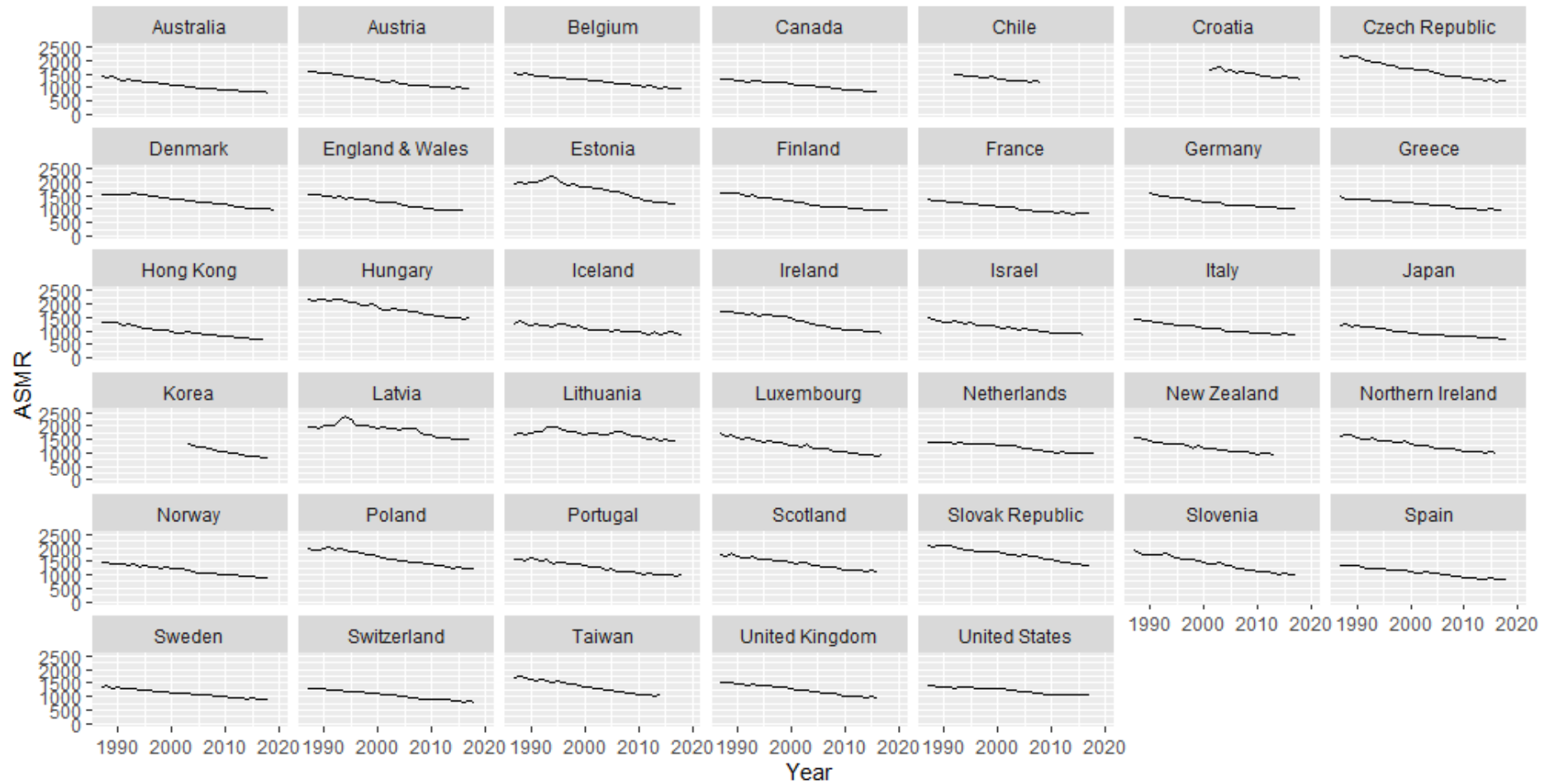


Figure 7.5 - Segmented regression fitted to the ASMR trends for the total population (2000-2019, vertical lines indicate breakpoint)

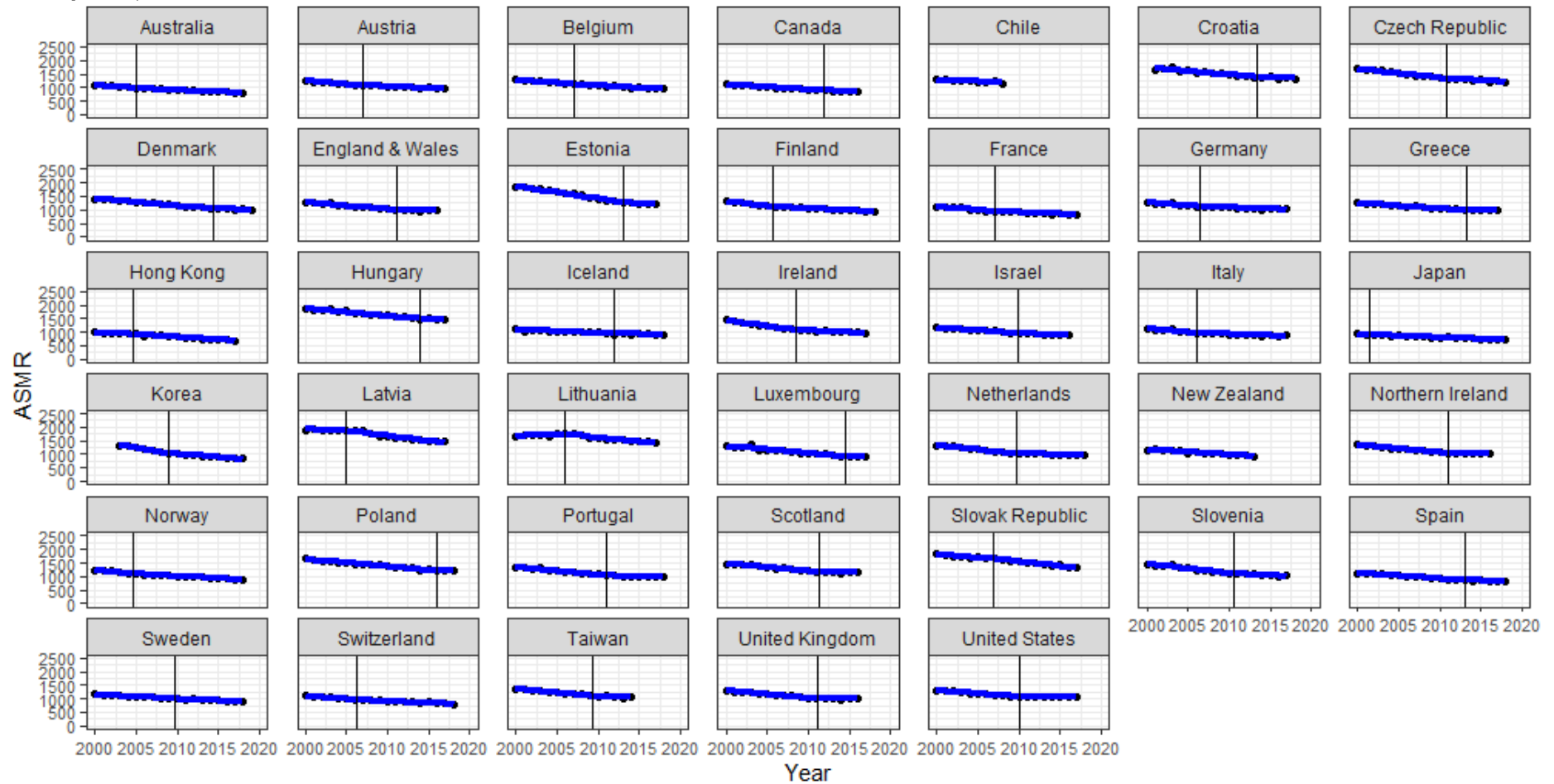
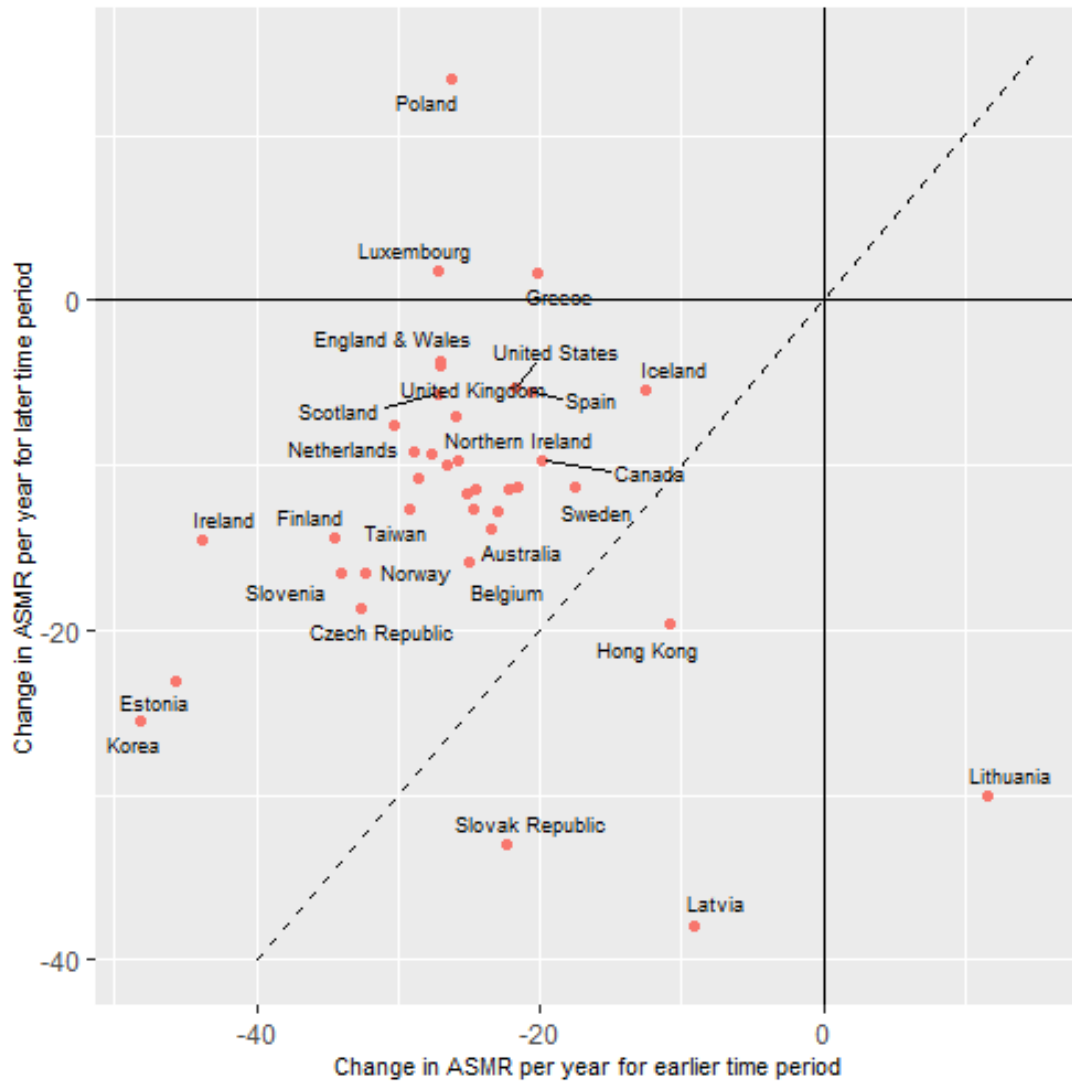


Figure 7.6 - Scatterplot of the rate of change in ASMR for the total population before and after the breakpoint (2000-2019)



Time trends: Lifespan variation

Lifespan variation (e_{\dagger}) is a measure of the variability of age at death among individuals within a defined population. The trends in lifespan variation for the total population are very similar to the ASMR trends for all ages, but the mirror image of life expectancy (Figure 7.7). The general trend is improvement over time across countries, with slightly higher lifespan variation amongst males than females (reflecting the higher mortality rates

at early ages, Figures S7.43 and S7.44). Again, some countries saw worsening trends during the 1990s (Estonia, Latvia and Lithuania) reflecting the rise in mortality rates at younger ages at this time.

The segmented regression analyses of the lifespan variation trends show a wide range in breakpoints, with some countries displaying a breakpoint shortly after 2000 and subsequent faster improving trends (e.g. Denmark, Hungary, Ireland and the Slovak Republic), whilst many others showed a later breakpoint and a subsequent rate of improvement that was slower (Figure 7.8). The analysis of the rate of change in lifespan variation before and after the breakpoint across country was more balanced than for life expectancy, ASMR across all ages, or for the ASMRs for specific age strata, in that 12 countries had better trends after the breakpoint than for before. However, the scatterplot is spread out by Iceland and Hong Kong which have apparently large changes in the later time period, but these are based on very short time series (Figure 7.9).

Figure 7.7 - Trends in lifespan variation for the total population (1987-2020)

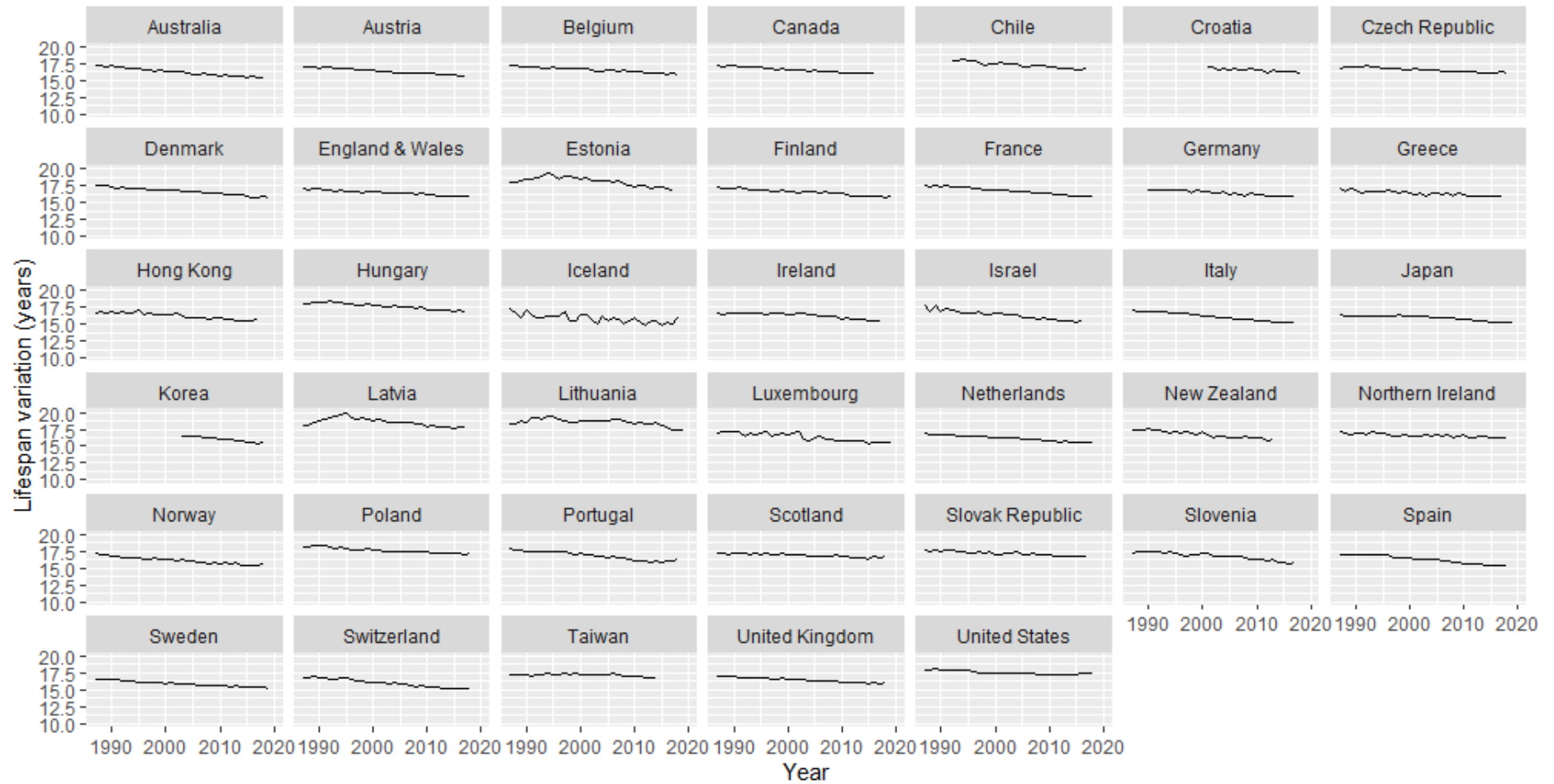


Figure 7.8 - Segmented regression fitted to the lifespan variation trends for the total population (2000-2019, vertical lines indicate breakpoint)

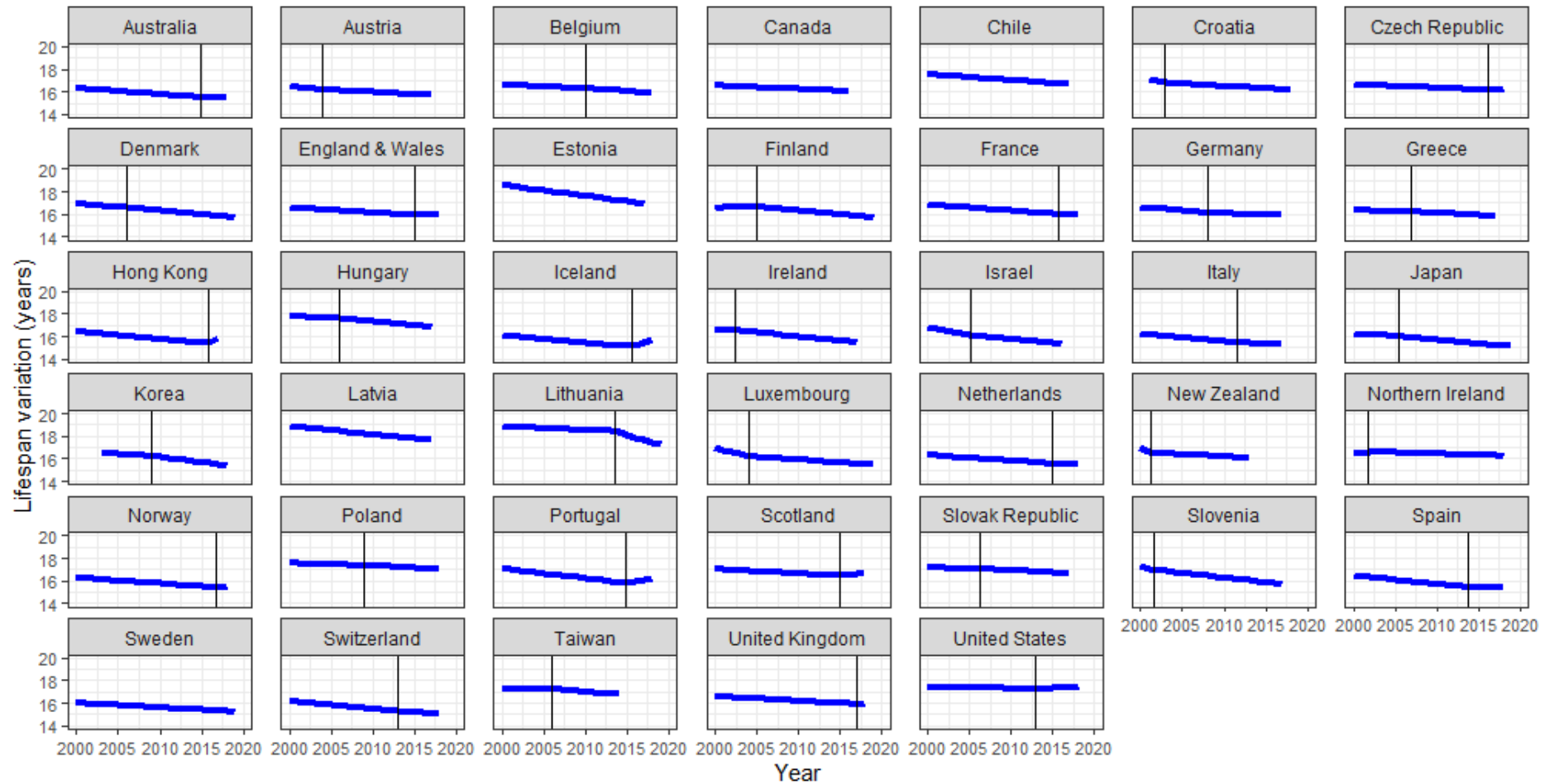
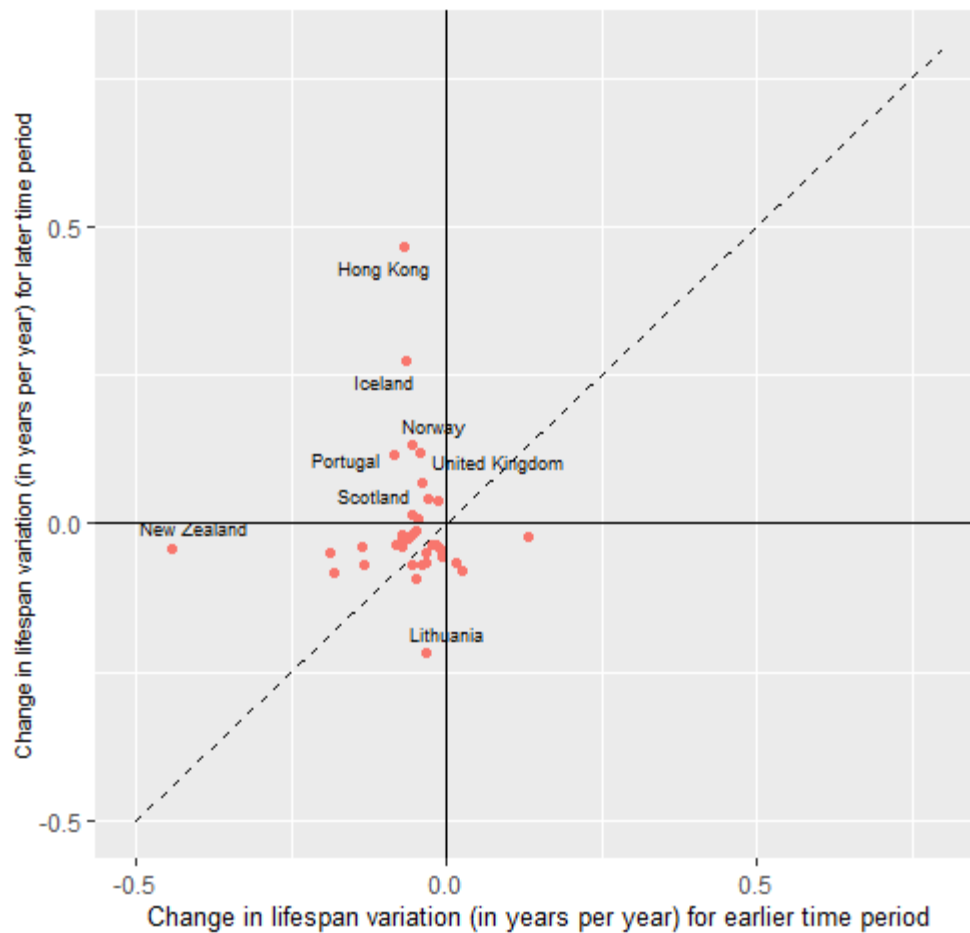


Figure 7.9 - Scatterplot of the rate of change in lifespan variation for the total population before and after the breakpoint (2000-2019)



Time trends: underemployment

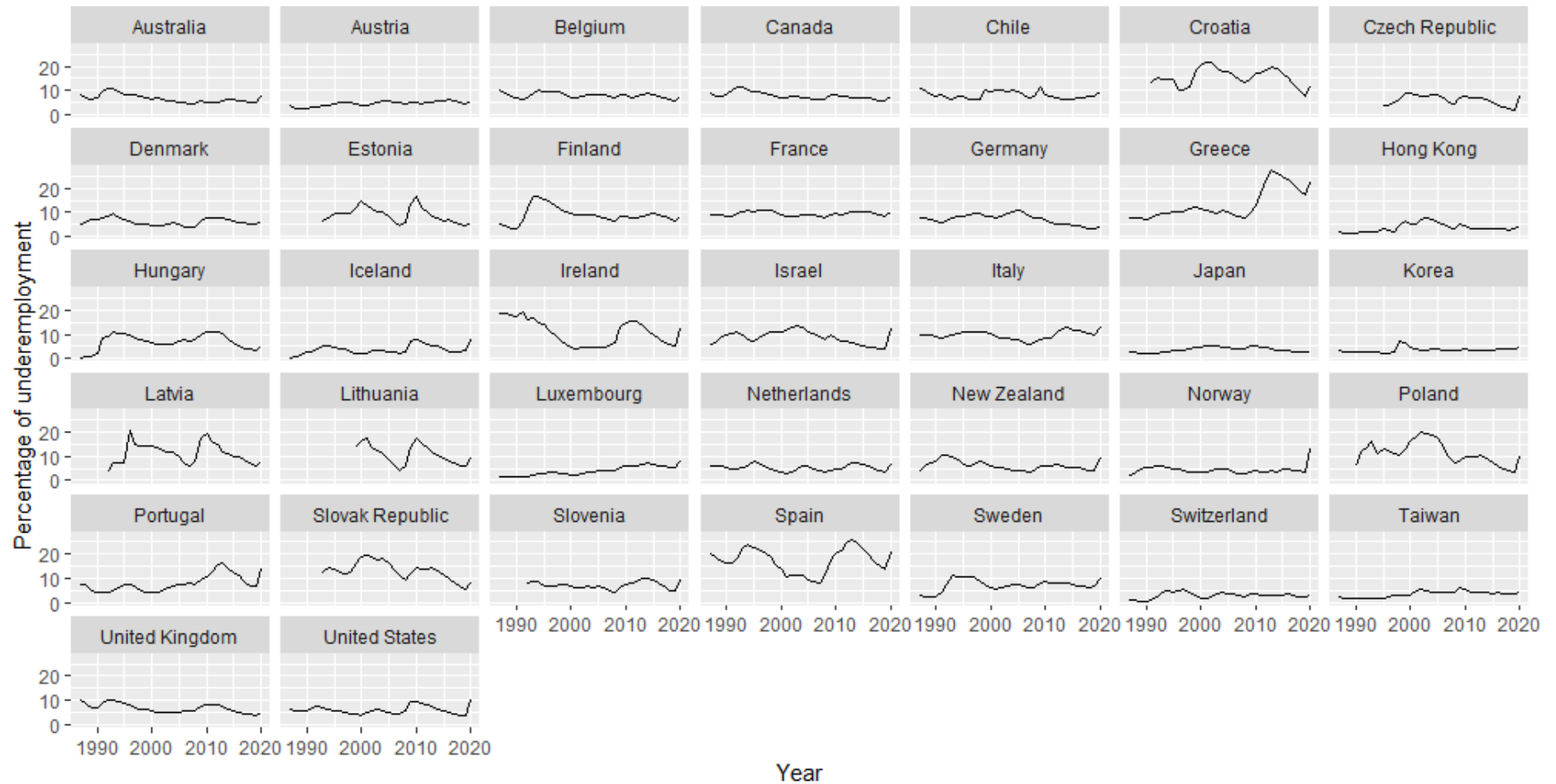
The trends in underemployment (a measure constructed by the International Labour Organisation (ILO)) relating to the percentage of people in paid work who worked less than a threshold (equivalent to working 'full-time') indicate that there were many countries with relatively stable trends over time, and others with very marked swings (Figure 7.10). The difference in the scale of variability between countries makes comparison on the same scale difficult as a doubling of the percentage from (for example) 2% to 4%

within one country (e.g. the fluctuations in Switzerland) might not seem important when another country (e.g. Spain or Greece) experiences changes from under 10% to over 25%. It is also worth remembering that this measure is survey-based, and although a substantial amount of work has been undertaken by the ILO to harmonise these across countries, there are likely to be differences in how these relate to the experiences across countries.

There are several countries which experienced marked increases in reported underemployment following the Great Recession from 2008. Croatia, Estonia, Greece, Hungary, Iceland, Ireland, Italy, Latvia, Lithuania, the Netherlands, Poland, Portugal, the Slovak Republic, Slovenia, Spain, Taiwan, the UK and the USA all display peaks around 2010. However, the magnitude of these peaks, and the stability of the trends before and after are markedly contrasting.

The lack of linear trends meant that the application of linear regression methods, and segmented regression methods, to these trends was not appropriate.

Figure 7.10 - Trends in underemployment (1987-2020)

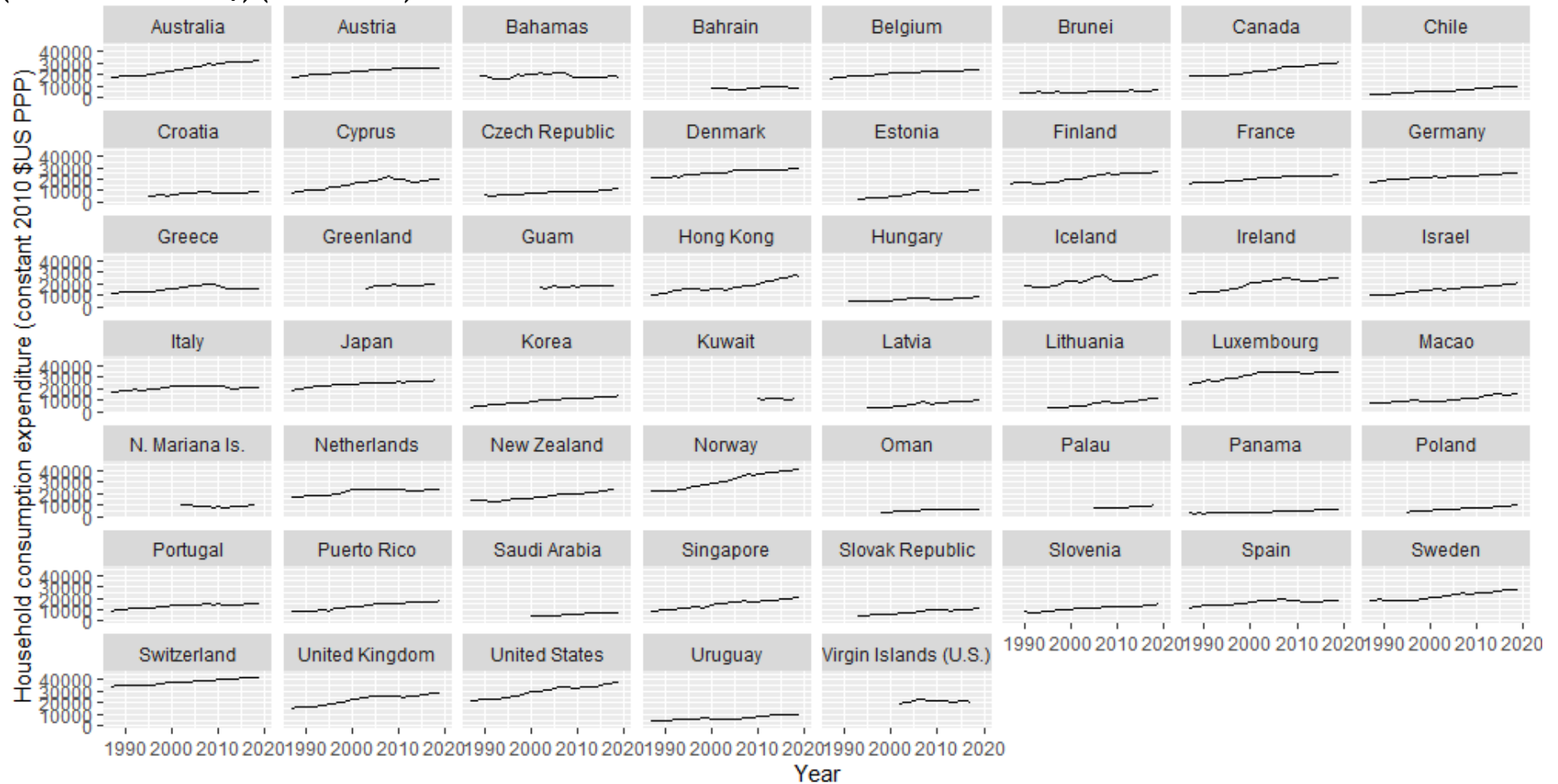


Time trends: Household incomes

Mean household incomes tended to increase over time for most countries for which trend data were available (Figure 7.11). There were some countries where there was less evidence of improvement (The Bahamas, Bahrain, Brunei Darussalam, Croatia, Greenland, Guam, Hungary, Kuwait, Northern Mariana Islands, Oman, Uruguay and the US Virgin Islands), and evidence across many countries of a dip in household incomes around 2008-2010 (e.g. Cyprus, Iceland, Ireland, Italy, Latvia, Lithuania, Luxembourg, Netherlands, Portugal, Spain, UK and USA).

Note that there was only a single data point for Antigua, Aruba, Barbados, Bermuda, Faroe Islands, Malta, Qatar and the Seychelles, and so these have not been graphed).

Figure 7.11 - Trends in Household incomes (Households and NPISHs Final consumption expenditure per capita), PPP (constant 2010 US\$) (1987-2020)



Time trends: Alesina-Ardagna Fiscal Index (AAFI)

As discussed in more detail in Chapter 6, part of the calculation of the AAFI requires a regression of government expenditure as a percentage of GDP on time, adjusted for the unemployment rate (Figure S7.45) in the previous year. Figure S7.46 shows that fitting a linear regression equation to the trends in government expenditure as a percentage of GDP, is appropriate for most countries for which data are available but with some exceptions. The trend in Kuwait approximates a V-shape which does not lend itself to a linear regression for the whole time period, and so the results should be interpreted with caution. Several countries also display substantial, but short-lived, increases in government expenditure around 2010 corresponding to the global financial crisis (especially Ireland, but to a lesser extent Latvia, Lithuania and Slovenia).

The trends in government revenues as a percentage of GDP, also used in the calculation of AAFI, are shown in Figure S7.47. Using these data the predicted and actual government expenditures as a percentage of GDP can be calculated as a means of estimating the automatic stabilisers in the economy (Figure S7.48).

From these data, the trends in the AAFI can then be calculated (Figure S7.49, Table S7.5). Figure 7.12 below shows these trends colour coded such that the blue dots indicate years when there is a degree of fiscal stimulus and grey dots when there is a degree of austerity. Note that the scale of the y-axis Figure 7.10 below ranges from -20 to +20, much larger than would be necessary for most values of fiscal stimulus or austerity. This is however necessary to accommodate the data from the oil-producing states of Kuwait and Saudi Arabia whose economies and government revenues are very dependent on the prevalent oil price, somewhat distorting the overall pattern. However, for some other countries (notably Iceland and Ireland) there are values >10 and <-10 in some years, particularly around the 'Great

Recession' around 2007-2010. The different in scale between countries creates a risk of overlooking the trends in the periods of fiscal stimulus and austerity within other countries. Restricting the y-axis and instead colouring the data points red if the AAFI is less than -1.5, a threshold suggested by Toffolutti and Suhrcke (2019) (Figure 7.13), reveals that many countries have experienced prolonged periods of austerity⁹ over the time period. All countries demonstrate some periods of austerity on this measure, although there is evidence that many countries move rapidly from austerity to fiscal stimulus and back from year to year, rather than there being persistent and prolonged periods of austerity. There is also substantial variation in the scale of austerity, with some countries (e.g. Cyprus, Kuwait, Saudi Arabia) experiencing several years with an AAFI of <-5.

⁹ There is a full discussion on how to classify countries with apparent austerity when the economy is growing or in an economic downturn in the Discussion section of this chapter.

Figure 7.12 - Trends in the Alesina-Ardagna Fiscal Index (AAFI), blue dots indicate values >0 (fiscal stimulus), (1987-2020)

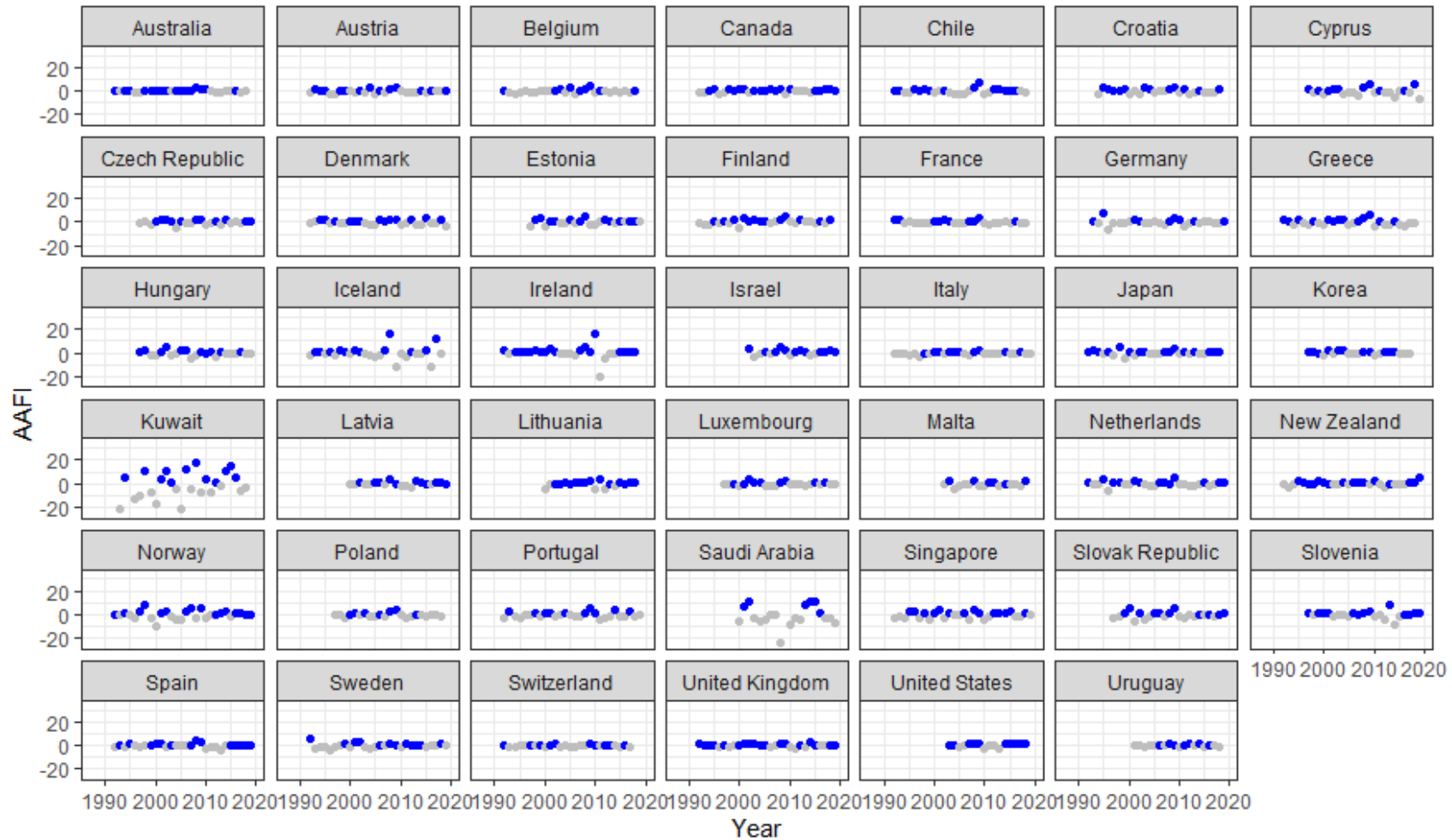
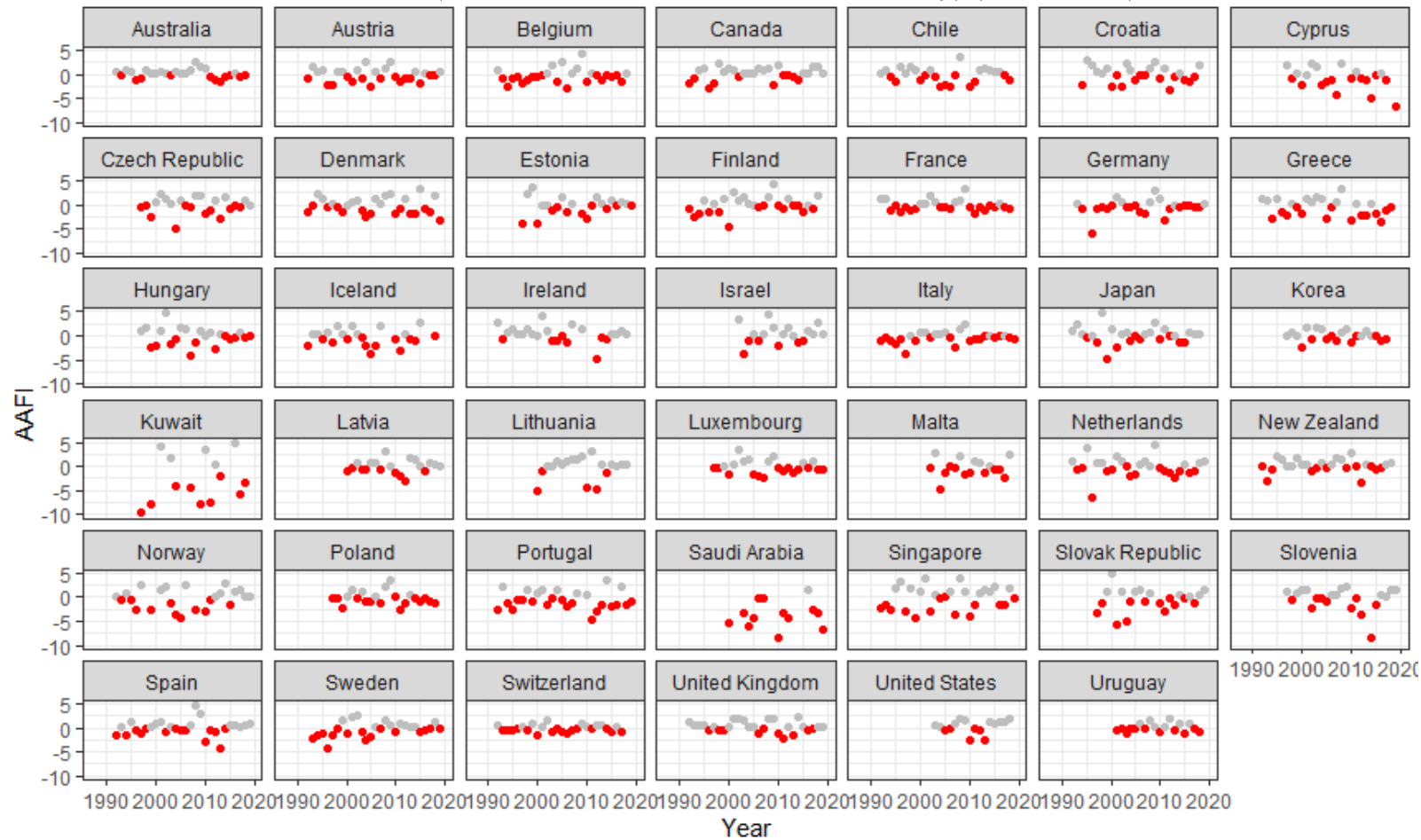


Figure 7.13 - Trends in the Alesina-Ardagna Fiscal Index (AAFI) with shortened y-axis (-10 to +5) and dots coloured red where the AAFI is less than -1.5 (often used as a threshold for austerity) (1987-2020)



Time trends: real Government Expenditure

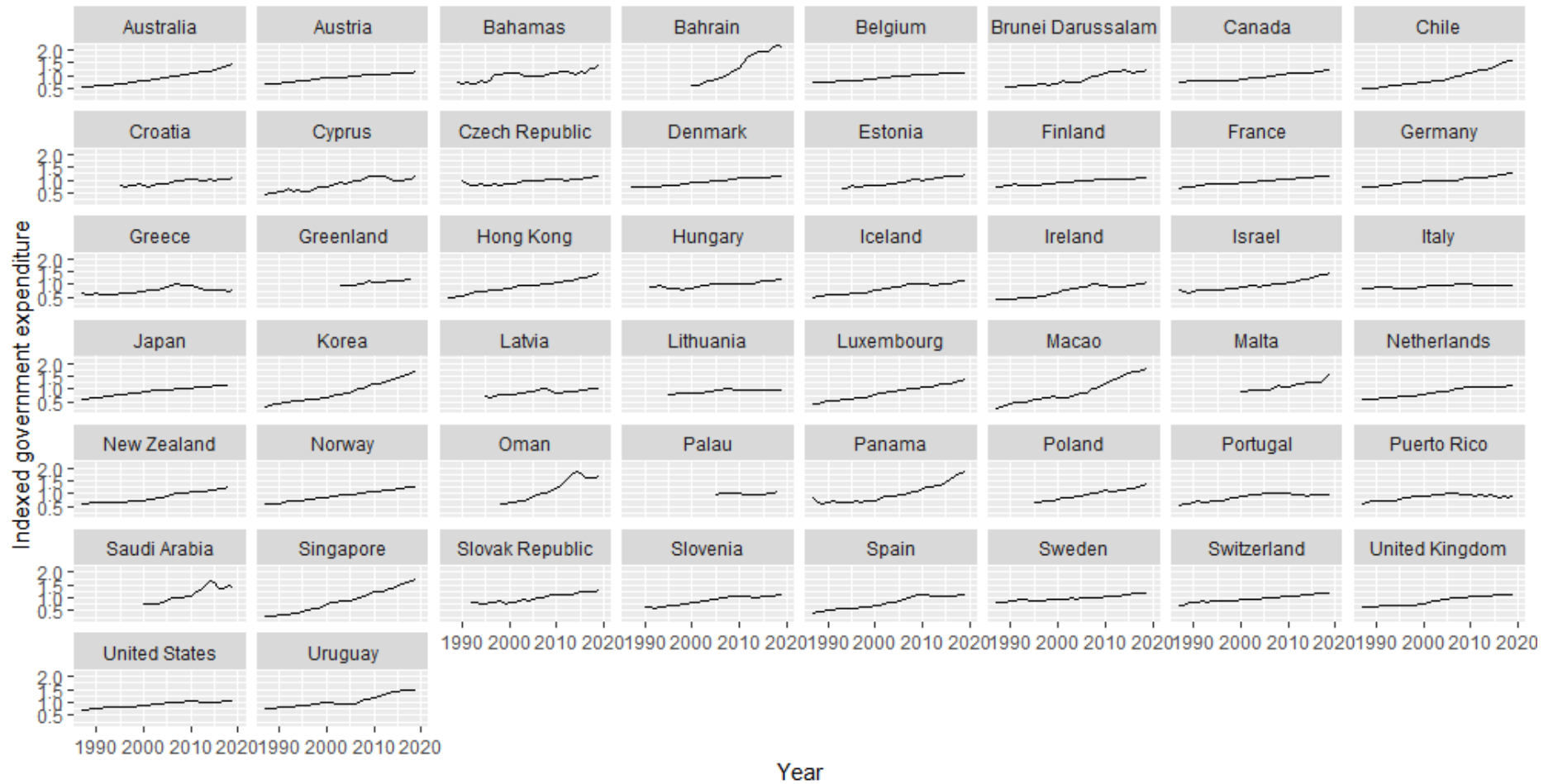
The second measure of austerity, real Government Expenditure, displays a general increase over time across countries, but the difference in values between countries is so varied that the nature of the trends cannot be appreciated on the same scale. This is because the starting levels of expenditure relating to the wealth of the country in question, and the population sizes of the countries, are so different (Figure 7.14).

To allow for changes to be appreciated on the same scale, Figure 7.15 shows the same data but indexed for each country (with available data) to the value in 2007 (chosen as the year prior to the 'Great Recession'). With that visualisation the trends across countries becomes much clearer. A clear increasing trends is seen across almost all countries over time, but with many countries displaying an inflection point after around 2008-2010. The oil-producing Middle-Eastern states (Bahrain, Oman and Saudi Arabia) displayed particularly fast increases, as did Chile, Macao, Korea, Panama and Singapore. Greece was notable for its prolonged period of declining Government Expenditure after around 2009.

Figure 7.14 - Trends in real final Government Expenditure (constant 2010 US\$) (1987-2020)



Figure 7.15 - Indexed trends in real final Government Expenditure (constant 2010 US\$, indexed as 2007 = 1) (1987-2020)



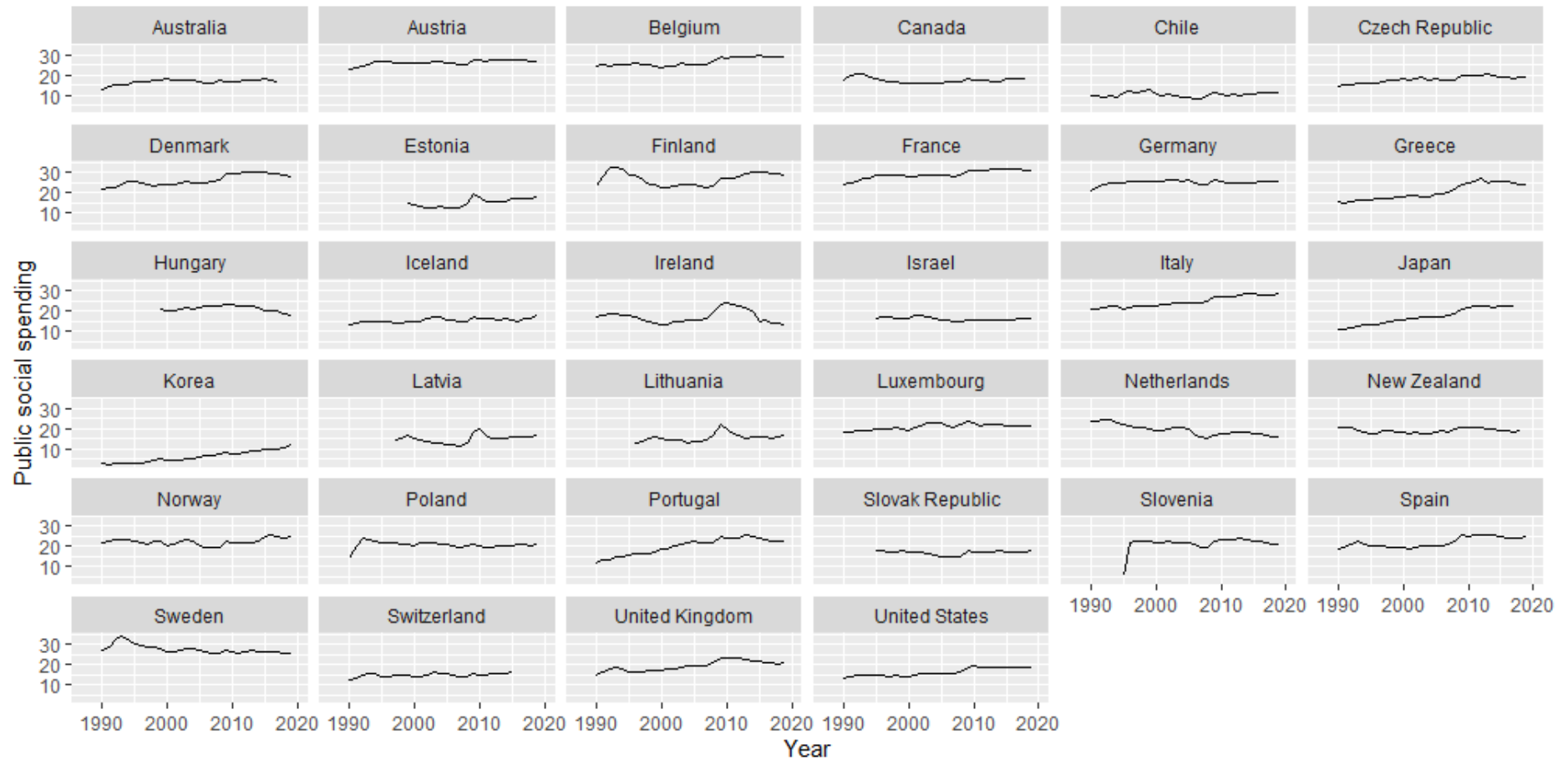
Time trends: Public Social Spending

There were fewer countries for which data on Public Social Spending as a percentage of GDP data were available, compared to 'real final government expenditure' data (Table S7.1). These data demonstrate marked differences between countries in the level of spending, and differences in the trends over time within countries.

Austria, Belgium, Denmark, Finland, France, Germany, Italy and Sweden had higher Public Social Spending on this measure than other countries for most of the period 1987-2019 (Figure 7.15). Some other countries had notably lower spending throughout the time period, including Chile, Estonia, Korea, Switzerland and the USA.

Several countries had slowly increasing trends in Public Social Spending over this time period: Australia, Austria, Belgium, the Czech Republic, Denmark, France, Iceland and Luxembourg. A smaller group of countries had much more rapidly increasing percentages (Greece, Italy, Japan, Korea and Portugal), although these countries generally started from a low base (and in the case of Korea still ended the time series with a lower percentage than most other countries). Notably, the Netherlands and Sweden had declining spending trends across most of the time series, whilst many countries displayed declines after 2010 (Czech Republic, Denmark, Greece, Hungary, Ireland, Luxembourg, New Zealand, Portugal, Slovenia, Spain, and the UK).

Figure 7.15 - Trends in Public Social Spending as a percentage of GDP (1987-2020)



Time trends: Cyclically Adjusted Primary Balance (CAPB)

The CAPB is an estimate of the fiscal balance of a country that would apply under the policies at a particular point in time if output were equal to potential, thereby seeking to remove the effects of additional spending on social security and reduced tax revenues during economic downturns, and excluding interest payments on national debt (IMF, 2016). It is measured as a percentage of GDP. Higher values for CAPB indicate more austere policies, and lower values fiscal stimulus (in the sense that values >0 indicate that government is reducing its debt relative to the point in the business cycle). However, Alesina et al. (2013) have argued that it is less about the absolute value of the CAPB at a point in time that is important, but the change in value. They argue that an *increase* in CAPB of 1.5 percentage points of GDP (rather than an absolute value >0 , or over some other threshold) indicates a period of fiscal consolidation. However, this is not consistent and it has been operationalised in both ways in the past (Perotti, 2013; Alesina et al., 2019b).^h

Figure 7.16 shows the trends in absolute values of CAPB across countries with available data from 1987. The values above zero are shown as blue dots, and those ≤ 0 as grey dots. There are substantial differences in the trends over time and between countries with no clear overall pattern. Some countries show very dramatic changes over time, such as Iceland, Greece, Ireland, Portugal and Spain.

The year-to-year change in CAPB shows a different pattern, but one which is more consistent with the other measures of austerity (Figure 7.17). It is interesting to note the consistent pattern of budget surplus (which can relate to austerity) in countries such as Korea and Italy, and of consistent

^h Note that the AAFI calculation is a measure of the change from year to year, unlike the CAPB. Analysing the annual year in CAPB thereby brings this into line with the approach for AAFI.

budget deficit (stimulus) in some others such as Norway, Japan, the USA and, for most of the time period, the UK.

The expected trends towards austerity in countries such as the UK after 2010 are not clear from these data if the absolute values of CAPB in relation to zero are used as the barometer. If, however, the *change* in CAPB (as suggested by Alesina et al. (2019b)) is instead used to codify more austere periods, the trends in the UK are more similar to the expected values (i.e. there is a period of austerity visible in the UK after 2010 in line with the stated policy objectives of the Conservative/Liberal Democrat coalition government (HM Treasury, 2010) (Figure 7.17).

Figure 7.16 - Trends in Cyclically Adjusted Primary Balance (CAPB) (1987-2020, dots coloured blue if >0)

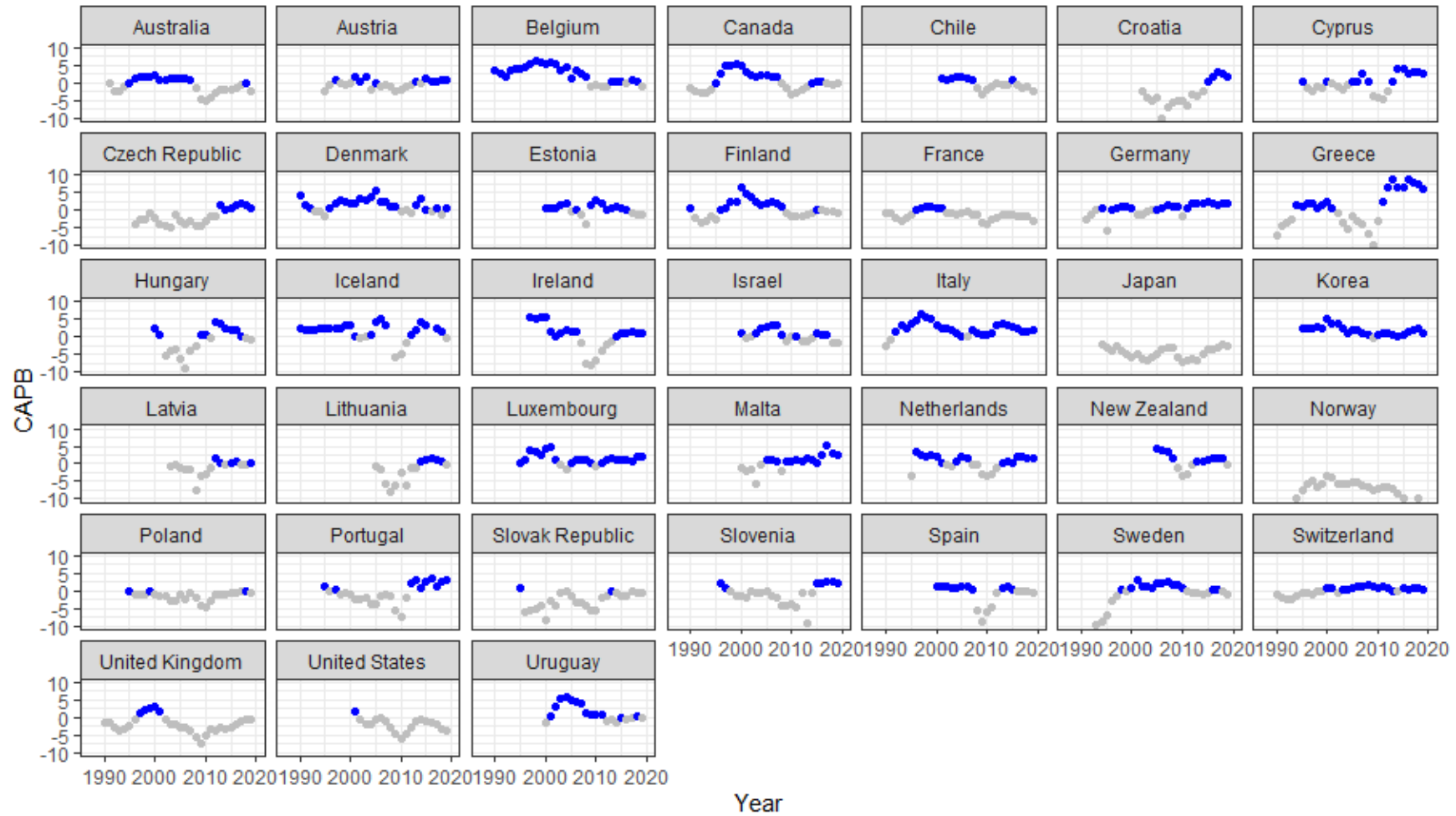
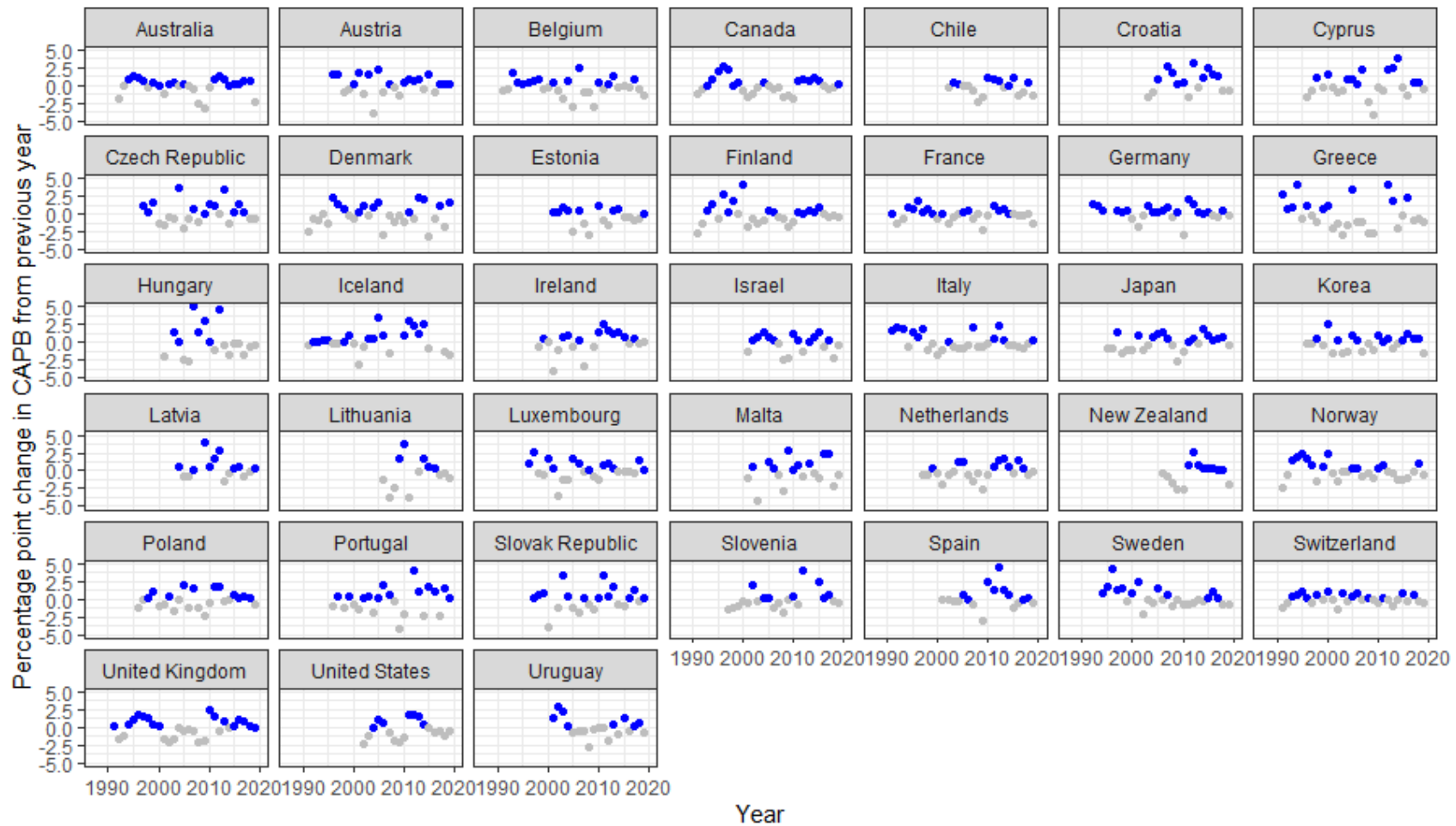


Figure 7.17 - Year-on-year change in Cyclically Adjusted Primary Balance (CAPB) (1987-2020, dots coloured blue if >0)



7.4 How the measures of austerity correspond

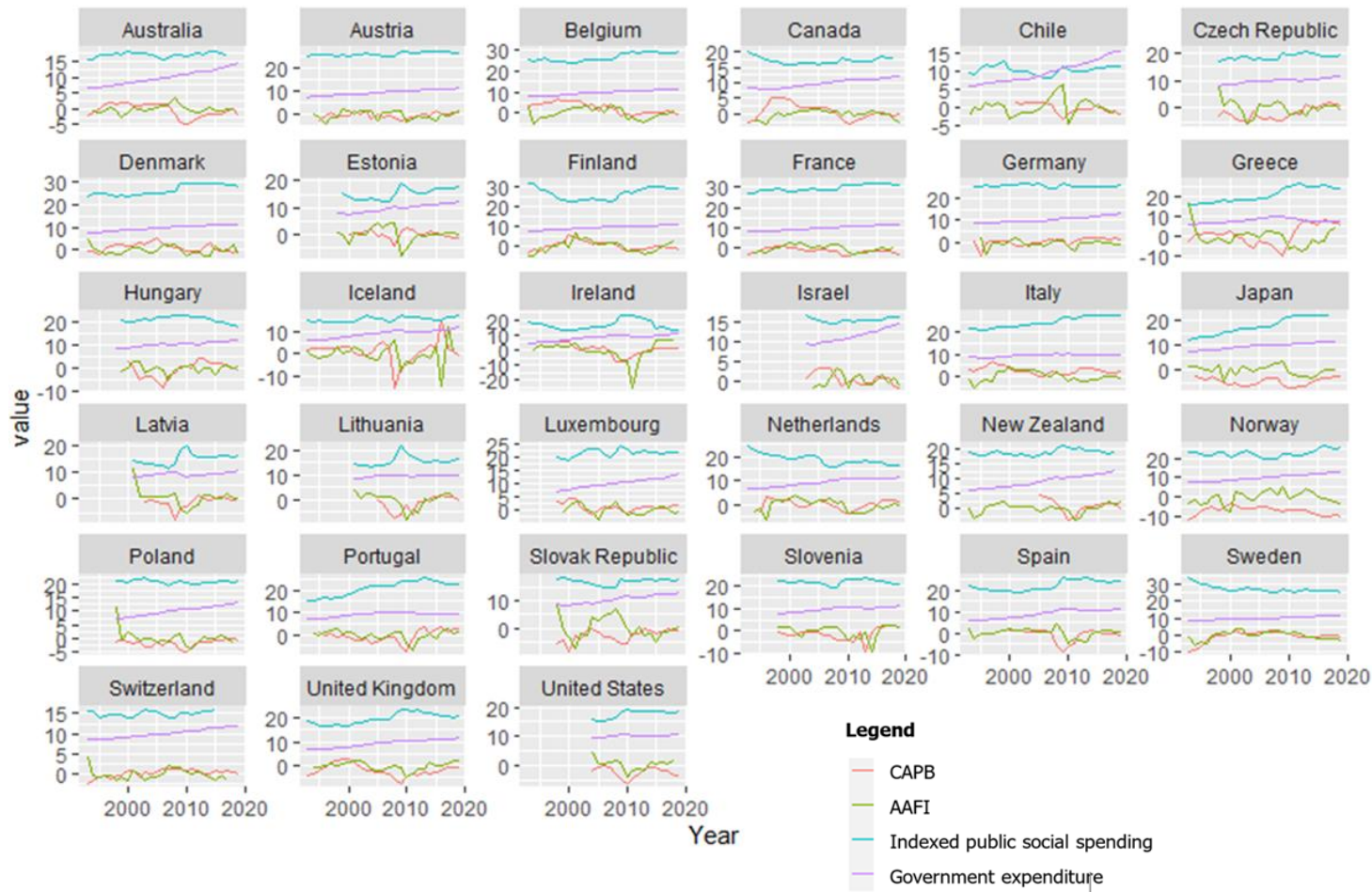
The AAFI was specified in the pre-analytical protocol as the primary austerity exposure variable, with real final Government Expenditure, the CAPB and Public Social Spending as a percentage of GDP as sensitivity analyses (McCartney et al., 2020b). As expected, given the different data that contributes to each, and the variation in the degree to which they seek to adjust for automatic stabilisers in the economy, they show different trends for countries over time.

Figure 7.18 shows the data for all the available countries and time points for all four austerity measures simultaneously. Note that the y-axis scale for this comparison (annotated simply as ‘value’, reflecting the different units for each measure) should be interpreted carefully. The purpose of the comparison is not to look at the absolute values of each measure over time or those absolute values in comparison to one another, but instead to allow a consideration of whether the measures are consistent with one another in their trends and turning points. If all measures of austerity were consistent, we would expect the following changes to occur with the implementation of austerity:

1. The value of AAFI would decrease (with a nominal threshold of less than -1.5 units).
2. CAPB will have increased (with a nominal threshold of an *increase* of 1.5 percentage points).
3. Public Social Spending (or indexed Public Social Spending) would decrease.
4. Government Expenditure would decrease.

Thus, AAFI, Public Social Spending and Government Expenditure decrease with austerity, whilst CAPB increases.

Figure 7.18 - Correspondence between measures of austerity over time (1987-2020)



In general, the AAFI, Public Social Spending and Government Expenditure measures go in the same direction over time, whilst the CAPB generally has an inverse relationship (although the correspondence is less obvious than with the other measures) (Figure 7.18). This is confirmed in Figure S7.50 which shows a scatterplot, linear regression line and R^2 value for the AAFI and CAPB values (measured as year on year change) for each available year and country. The R^2 values are generally low, but the fitted regression lines largely show the expected relationship of high values of AAFI being associated with low values of CAPB (i.e. sloping down from top left to bottom right). Figures S7.51 and S7.52 show the relationships between the AAFI values for each country and year and Public Social Spending and indexed Government Expenditure respectively. Both broadly show the expected relationship, although Public Social Spending has a more consistent relationship across countries.

7.5 Discussion and summary of the chapter

Main findings

This chapter has identified that there is evidence of a worsening in the trends for life expectancy and mortality rates in the 2010s in the datasets used here, but less evidence for a systematic change in trends for lifespan variation. There was consistent evidence across life expectancy and mortality measures for a worsening in trends for Estonia, Latvia and Lithuania during the 1990s, but subsequently improving rapidly.

The segmented regression analyses identified that a majority of countries, for the total populations and females and males separately, had worse trends towards the end of the time series (usually after 2010) when applied to the data from 2000 onwards. This was true for life expectancy, ASMR for all ages, and age-specific ASMRs, but not as clear for lifespan variation. Some countries had very obviously worse trends in the later time periods, including England & Wales, Scotland, the UK overall, and the USA.

The underemployment levels were very different between countries and varied substantially over time. This in part indicates the real differences in the availability of work between countries, but also potentially measurement differences and cultural differences (e.g. around the involvement of women in the labour force and unpaid caring roles). The final outcome measure, household incomes, also showed marked differences between countries in the levels, but more consistently increased over time, with a widespread dip around the time of the Great Recession (2008-2010).

Four measures of austerity exposure have been described. Both the AAFI and CAPB measures broadly showed the expected periods of austerity across countries (Konzelmann et al., 2016; Sawyer, 2012). Government Expenditure had to be indexed (in this case to 2007) to reduce the very substantial variability in levels between countries, but then more clearly corresponded with the other measures. Public social spending and indexed Government Expenditure showed broadly the expected trends across time and countries, but was only weakly correlated to the AAFI measure.

Strengths and limitations

The strengths of the approach to the initial descriptive analyses of the data contained in this chapter include: the pre-analytical protocol setting out the data sources and how the data were to be treated (McCartney et al., 2020b); the internationally harmonised sources of the data; and the triangulation of the results with other sources (see below). However, there are also some limitations. First, as described in Table S7.1 there are limited data availability across countries and time periods for some of the exposures and outcome measures of interest. This is particular true of smaller countries and of the earlier and most recent time periods. There is also a risk that missing data for the most recent years for some countries such as Iceland, Ireland, Italy and Greece (with, for example, life expectancy data only available until 2016, 2017, 2014 and 2013 respectively) represents a systematic bias of less data availability for countries which have

experienced particularly severe recent austerity measures. Indeed, it is even possible that austerity has been partially responsible for delayed reporting of the data through reduced funding of mortality administrative services. This could induce a form of collider bias in the analysis whereby non-reporting of data is associated with austerity, biasing the results towards the null by reducing variation in exposure-outcome relationships within the available dataset.

How this fits with the existing literature

In the protocol for this work (McCartney et al., 2020b), no distinction was made between fiscal contraction (austerity) occurring during an economic downturn or during periods of economic growth. This could be an important omission, as much of the critique of austerity policies in relation to the impact on economic growth have related to the use of fiscal contraction during economic downturns (including periods of recession and of slow economic growth) (Blyth, 2013). There is substantial debate on whether levels of government debt (Konzelmann et al., 2016; Reinhart & Rogoff, 2009; Herdon et al., 2014), fiscal consolidation (austerity), or fiscal stimulus, increase or decrease economic growth (all other things being equal) (Alesina & Ardagna, 2009; Guajardo et al., 2011; Blyth, 2013). There has also been suggestion that the nature of austerity (i.e. tax rises or government expenditure reduction, or a combination of both) also matters to the impact on economic growth (Alesina et al., 2019a).

The two other identified papers to publish annualised data on the AAFI measure categorises countries into three groups for each year: increases of >1.5 points (Fiscal Stimulus); decreases of >1.5 points (Austerity); and changes of between -1.5 and 1.5 (classified as neither Fiscal Stimulus nor Austerity) (Toffoluttia, Suhrcke, 2019; Alesina & Perotti, 1995).

Correspondence with these data indicated similar, although not identical, AAFI values (when categorised in the same manner) for the overlapping

years and countries. The differences are likely to represent the use of different international datasets as the basis for the calculations.

Implications and conclusions

Overall, these data confirm the expected stalled mortality and life expectancy trends, and the periods of austerity across countries. The next chapter (Chapter 8) will use these data to explore the relationship between austerity and the mortality and life expectancy outcomes data, as well as the sensitivity and secondary analyses.

8. To what extent can austerity explain the stalled mortality trends?

8.1 Chapter introduction

This chapter examines the extent to which measures of austerity can explain the public health ‘problem’ of stalled mortality and life expectancy trends. Chapter 7 showed that there was a widespread stalling in the trends of life expectancy, mortality rates at all ages, and mortality rates for some specific age groups in the period from around 2010 onwards across many countries. There is also evidence in many countries that there were periods of austerity implemented following the Great Recession from 2008. Although there is existing evidence associating overall measures of austerity with mortality trends in earlier time periods (Rajmil & Fernández de Sanamed, 2019; Toffoluttia & Suhrcke, 2019; van der Wel et al., 2018), and evidence relating specific aspects of austerity to adverse health trends (Wickham et al., 2020; Taylor-Robinson et al., 2019; Alexiou et al., 2021; Darlingon-Pollock et al., 2021; Wright & Patrick, 2019), there is no study yet published that robustly examines the recent trends and the extent to which these are explained by austerity.

This chapter provides the results for the empirical analyses of the relationship between four measures of austerity and a range of mortality-derived outcomes (as well as on mean household incomes and underemployment rates) to address this knowledge gap. It begins with a brief summary of the methods used (section 8.2). Then follows the results for the relationship between mortality-derived outcomes and AAFI (section 8.3), Government Expenditure (section 8.4), Public Social Spending (section 8.5), and then CAPB (section 8.6). The sensitivity analyses which adjust for potential confounding factors are described in section 8.7, and the analyses which consider underemployment and household incomes as the outcomes of interest in section 8.8. Following this, the additional sensitivity analyses

which restrict the panel to exclude oil-dominated economies (Section 8.9), and to periods of economic downturn (Section 8.10), are presented. Finally, Section 8.11 discusses these results and puts them into context of the current literature.

8.2 Methods summary

A fuller discussion of the methodological approach is provided in Chapter 6. Briefly, an unbalanced panel dataset was assembled for high income countries for which data was available. This dataset has been explored descriptively in Chapter 7. Data were assembled for four measures of austerity (AAFI, indexed real per capita Governmental Expenditure, Public Social Spending as a percentage of GDP, and CAPB), a series of mortality-derived measures for the total, female and male populations of each country (including life expectancy at birth, ASMR, age-specific mortality, and lifespan variation), and on two additional outcomes (time-related unemployment rate and mean household expenditure).

A family of fixed effects panel regression models were fitted for the bilateral relationships between each of the austerity measures and each of the mortality outcome measures. The models were built in R using the ‘plm’ package (Croissant & Millo, 2015), and adapting code from Colonescu (2016). Each model was run for three separate lag periods between the exposures and outcomes (of 0, 2 and 5 years).

The corresponding equation for the regression model is:

$$y_{it} = \alpha_i + B_1x_{1it} + \dots + B_Nx_{Nit} + e_{it}$$

where:

α is the unobserved time-invariant country fixed effect

$i = 1, \dots, N$ represents the country within the panel

$t = 1, \dots, N$ represents the year of the data

y is the mortality outcome

B is the regression coefficient

e is the error term

N is the total number of country-time observations in the panel

x is the austerity measure (AAFI, indexed real per capita Governmental Expenditure, Public Social Spending as a percentage of GDP, and CAPB)

The model estimates, and associated 95% confidence intervals, were then used to create Forest plots for each of the common exposure-outcome relationships. As theorised, fixed effects were confirmed in the panel dataset (Table S8.1).

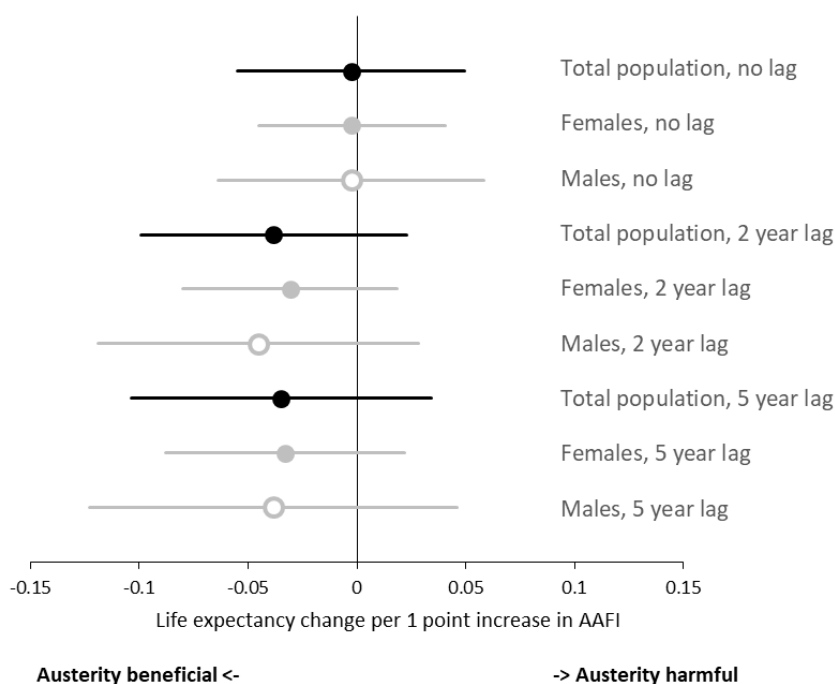
Cross-sectional dependence, whereby a change occurs across the panel at the same time, was detected and probably relates to the simultaneous adoption of austerity across many countries in the panel around 2010-12 (Table S8.2). Serial correlation, where the values in one year are correlated with those in the next year, was also detected, indicating the differences between countries that were theoretically expected (Tables S8.3 and S8.4). Heteroscedasticity - where the standard errors are not constant over time - was detected for the models using Public Social Spending and CAPB. Therefore, standard errors were estimated using a Sandwich estimator. Estimates of the standard error were essentially unchanged using this approach suggesting that this had little impact on the results (Tables S8.5 and S8.6).

The next section presents the results of the models for each of the austerity measures in turn.

8.3 Relationship between AAFI and mortality and life expectancy outcomes

As discussed in Chapter 7, decreases in AAFI represent greater austerity. Figure 8.1 shows that changes in AAFI had little or no change in any total, female or male life expectancy for lag periods 0, 2 or 5 years, with the point estimates all being less than 0.05 years of life expectancy change per unit change in AAFI, and with the 95% confidence intervals all overlapping the line of no effect. For the 2 and 5 year lagged estimates, there were small beneficial impacts of increased austerity, but these estimates were imprecise and any difference from no effect could have been due to chance. The effect sizes were similar for females and males for each of the lag periods.

Figure 8.1 - Relationship between AAFI and total, female and male life expectancy using 0, 2 and 5 year lags between exposure and outcome

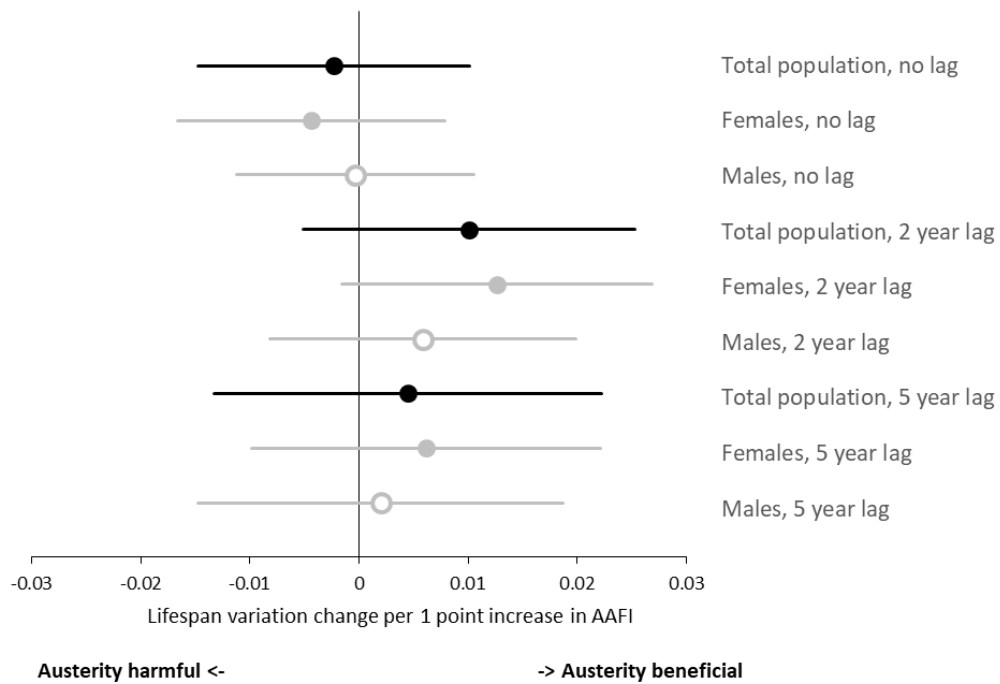


For lifespan variation (Figure 8.2), and age-specific mortality (Figures S8.1-S8.9) the effect estimates for changes in AAFI were similarly close to zero for the total population, females and males, and for each of the three lag

periods between exposure and outcome. The underlying data for all of the AAFI analyses are provided in Tables S8.7 and S8.8.

In summary, the models for the relationship between AAFI and all of the mortality-derived outcomes showed no large or precise effects across any of the lags or population subgroups.

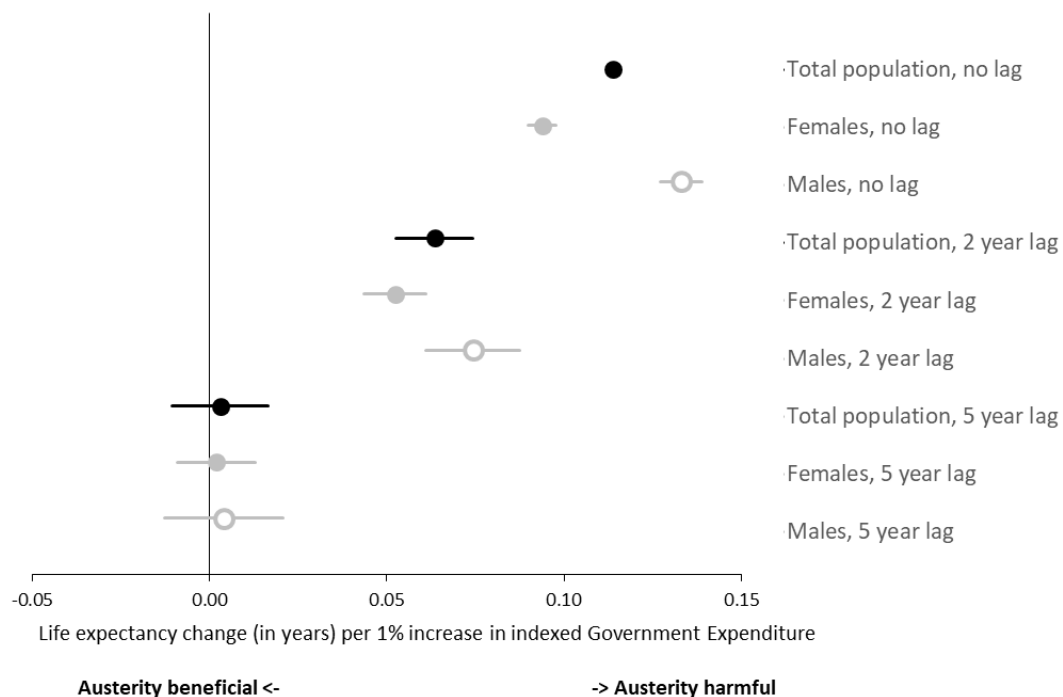
Figure 8.2 - Relationship between AAFI and total, female and male lifespan variation using 0, 2 and 5 year lags between exposure and outcome



8.4 Relationship between indexed real per capita Government Expenditure and mortality and life expectancy outcomes

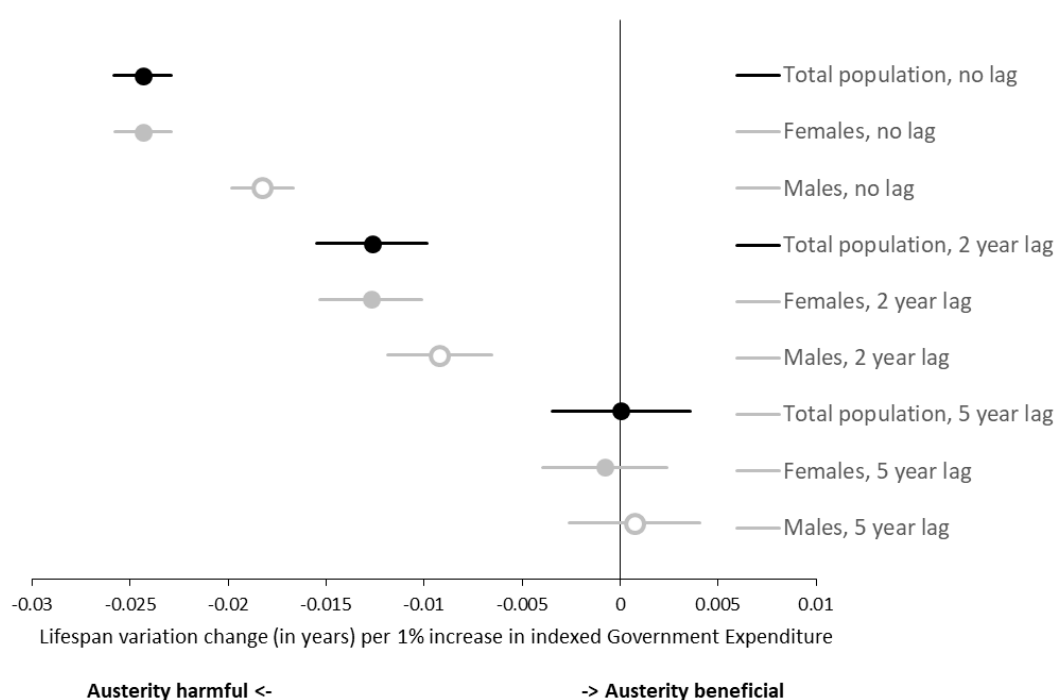
The impact of changes in Government Expenditure are modelled as the impact of a 1% increase in indexed Government Expenditure. In contrast to the results for AAFI, there was a clear relationship between austerity as measured by changes in indexed per capita Government Expenditure and life expectancy (Figure 8.3 and Table S8.9). There was an increase in total life expectancy of 0.11 years and 0.06 years per 1% increase in indexed Government Expenditure with a 0 and 2 year lag respectively. The effect sizes were consistently greater for males than for females. There was no impact of changes in indexed Government Expenditure with a 5 year lag.

Figure 8.3 - Relationship between indexed real per capita Government Expenditure and total, female and male life expectancy using 0, 2 and 5 year lags between exposure and outcome



Very similar harmful impacts of austerity on this measure were present for lifespan variation (Figure 8.4, Table S8.9). Again, the impacts were greatest with zero lag, but in contrast to life expectancy, they were worse for females than males. Note that the total population estimates are similar to those for females rather than sitting between the female and male estimates, relating to lifespan variation being a measure of dispersion rather than an average effect of females and males combined.

Figure 8.4 - Relationship between real per capita Government Expenditure and total, female and male lifespan variation using 0, 2 and 5 year lags between exposure and outcome



The relationship between changes in indexed real Government Expenditure and all-age ASMRs and age-specific mortality are shown in Figures S8.4, S8.5 and S8.6 for 0, 2 and 5 year lag periods respectively, with the underlying data in Tables S8.9 and S8.10. A negative impact of austerity on mortality rates is again evident. A one percentage point increase in indexed per capita Government Expenditure was associated with a decline in ASMR for

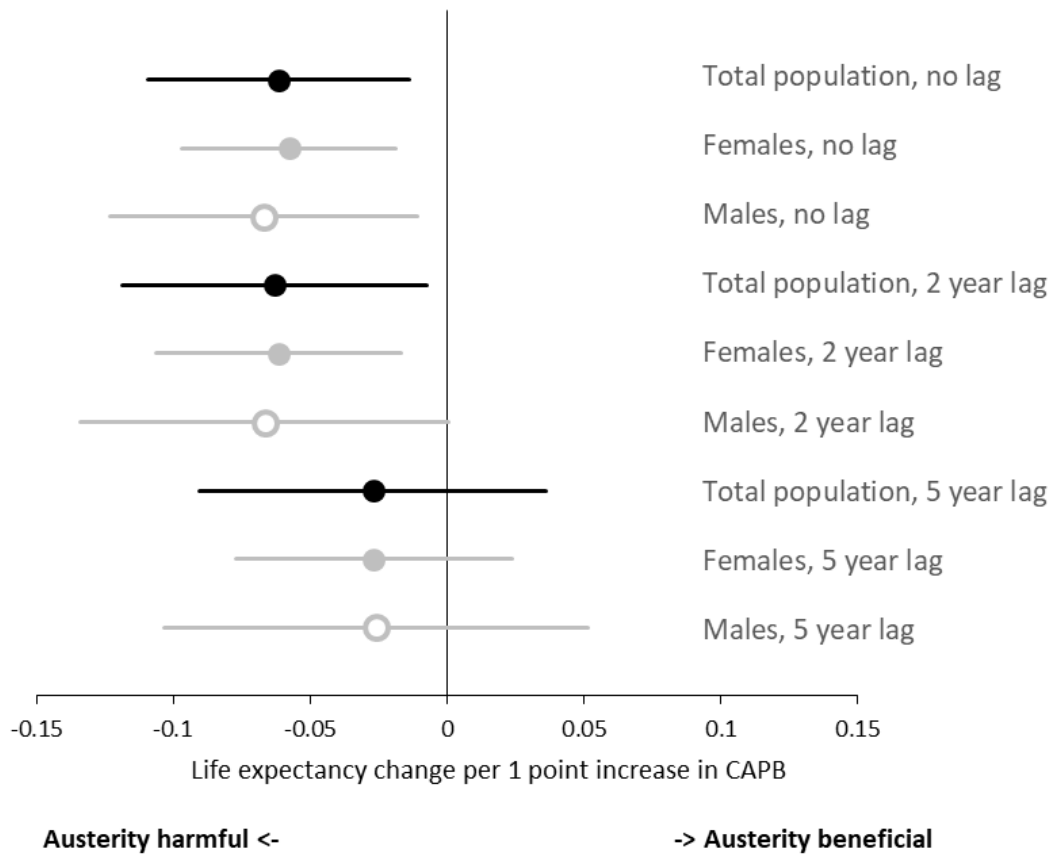
the total population of 10.5 deaths per 100,000 population per year with zero lag and a decline of 6.0 deaths per 100,000 population per year with a two year lag. The effect sizes were larger with increasing age, although this reflects the higher mortality rates for these groups, and for males. For all age groups and for females and males, there was no effect of changes in indexed real Government Expenditure with a 5 year lag between exposure and outcome (Figure S8.6).

8.5 Relationship between Public Social Spending and the mortality and life expectancy outcomes

The relationship between Public Social Spending changes and life expectancy was very similar to the patterns seen for indexed Government Expenditure. Austerity on this measure was again associated with lower life expectancy trends, with the largest effect sizes with a 0 and 2 year lag, and for males (Figure 8.5 and Table S8.5). The effect sizes demonstrated are sizable (e.g. an increase in total life expectancy of 0.37 years with no lag) but this represents a relatively large change in exposure (a one percentage point increase in Public Social Spending). Lifespan variation also worsened, particularly with shorter lag periods, with austerity on this measure (Figure 8.6 and Table S8.11).

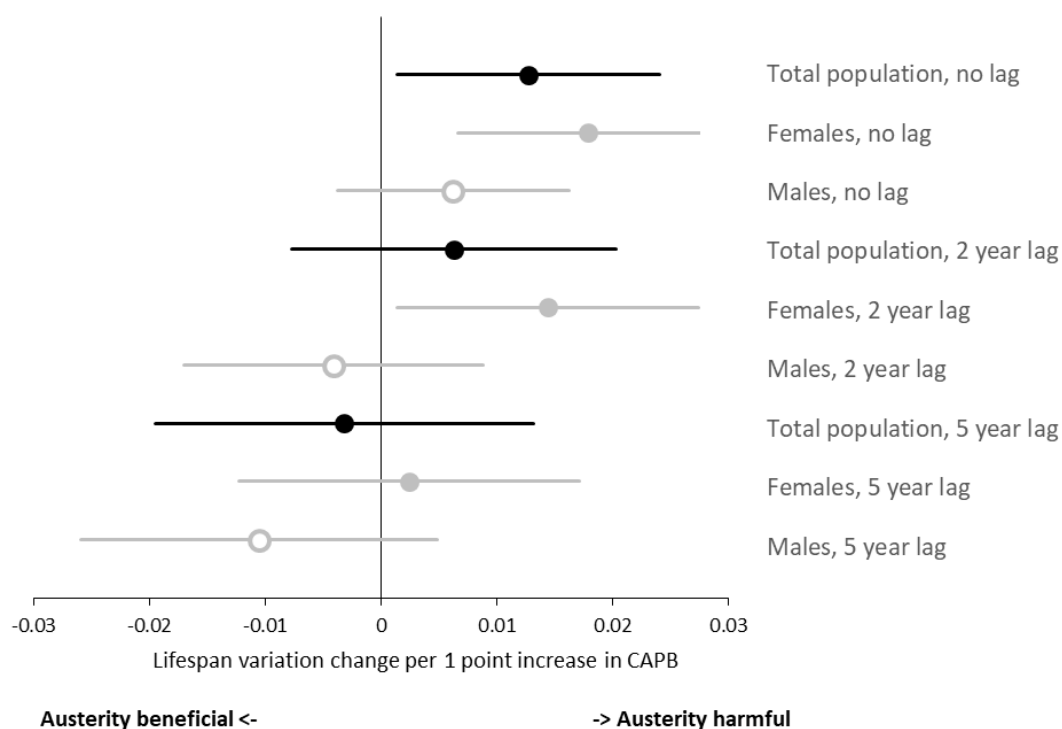
All-age ASMR, and age-specific mortality rates again demonstrated a worsening with austerity on this measure, with greater impacts for males, and with a zero lag (Table S8.12 and Figure S8.13) than for a 2 year lag (Figure S8.8) or 5 year lag (Figure S8.9). The effects were again larger at older ages reflecting the higher mortality rates at increasing age.

Figure 8.7 - Relationship between CAPB and total, female and male life expectancy using 0, 2 and 5 year lags between exposure and outcome



Lifespan variation also worsened with austerity measured by CAPB with no lag for the total population and females, but there was little impact of any changes for other groups or lag periods (Figure 8.8).

Figure 8.8 - Relationship between CAPB and total, female and male lifespan variation using 0, 2 and 5 year lags between exposure and outcome



The changes in ASMR across all ages with austerity measured by CAPB also show negative impacts with an increase of 5.7, 6.3 and 3.7 deaths per 100,000 per year for 0, 2 and 5 year lag periods, for a unit increase in CAPB (Figures S8.10 to S8.12). The impacts for specific age groups were, however, negligible for those aged under 50 years, but were more substantial for the oldest age group (Table S8.14).

8.7 Analyses adjusted for potential confounders and mediators

As specified in the pre-analytical protocol (McCartney et al., 2020b), the theoretical understanding of the relationship between austerity and mortality outcomes includes changes household incomes, GDP and underemployment as likely mediators. As such (as discussed in Chapter 6), adjusting for these variables in the analysis would be over-adjustment,

removing part of the real effect of austerity. However, as there is some uncertainty about their role as mediators, and potential for them to be confounders, a series of sensitivity analyses have been performed with adjustment for each.

The results for the analyses of each of the austerity measures adjusted for mean household incomes are provided in Table S8.15. Adjustment for household incomes made little difference to the relationship between AAFI and life expectancy which had already been close to the null without adjustment (Figure S8.13). Adjustment did substantially reduce the effect size for the relationship between Government Expenditure changes and life expectancy, such that there were no clearly different effects from the null (Figure S8.14). The effect sizes for the relationship between Public Social Spending and life expectancy (Figure S8.15), and between CAPB and life expectancy (Figure S8.16), were however largely preserved, especially with lags of 0 and 2 years (similar to the unadjusted analyses).

The results for the analyses adjusted for real GDP per capita are shown in Table S8.16. After adjustment, there was no relationship between AAFI and life expectancy for females or males, for any of the lag periods (Figure S8.17). For indexed Government Expenditure changes, there was a consistent pattern of austerity being harmful for mortality measures with a reasonably large effect size. However, the estimates were imprecise and the difference from the null could have been due to chance (Figure S8.18). In contrast, there were negative impacts observed in the adjusted analyses for Public Social Spending changes (Figure S8.19) and CAPB (Figure S8.20), which was largest with no lag and declined to no effect with a 5 year lag.

The analyses adjusted for underemployment at baseline are described in Table S8.17. The available data for underemployment was much sparser (given the survey basis of the underlying data) and so the relationships are much less stable than for the other analyses. The results for the relationship between AAFI and life expectancy were close to zero, although in this case

there was a (very small and imprecise) harmful impact of austerity observed (Figure S8.21). The adjusted relationship between changes in Government Expenditure and life expectancy, showed a (implausibly) large negative impact of austerity across all lag periods (Figure S8.22). For changes in adjusted Public Social Spending there was a consistently negative impact of austerity across females and males, and for different lag periods (Figure S8.23), but there was no impact of changes in CAPB observed after adjustment on any of the life expectancy outcomes (Figure S8.24).

8.8 Impact of austerity on underemployment and household incomes

In addition to the impacts of austerity on mortality-derived outcomes, the pre-analytical protocol specified additional analyses considering the impacts on underemployment and mean household incomes. The results for all of these analyses are provided in Table S8.18 and are discussed in turn below.

Across all the measures of austerity (Figures S8.21 to S8.24), there was little or no relationship observed with underemployment using 0, 2 or 5 year lag periods, although there was a suggestion of a negative impact after 5 years with CAPB (Figure S8.24). The effect size estimates were generally small and imprecise, the latter reflecting the sparsity of the underlying underemployment data.

For the relationship with mean household incomes, the estimates across all measures of austerity were very imprecise with no clear direction of effect across the measures, nor any clear patterning with different lag times (Figures S8.21 to S8.24).

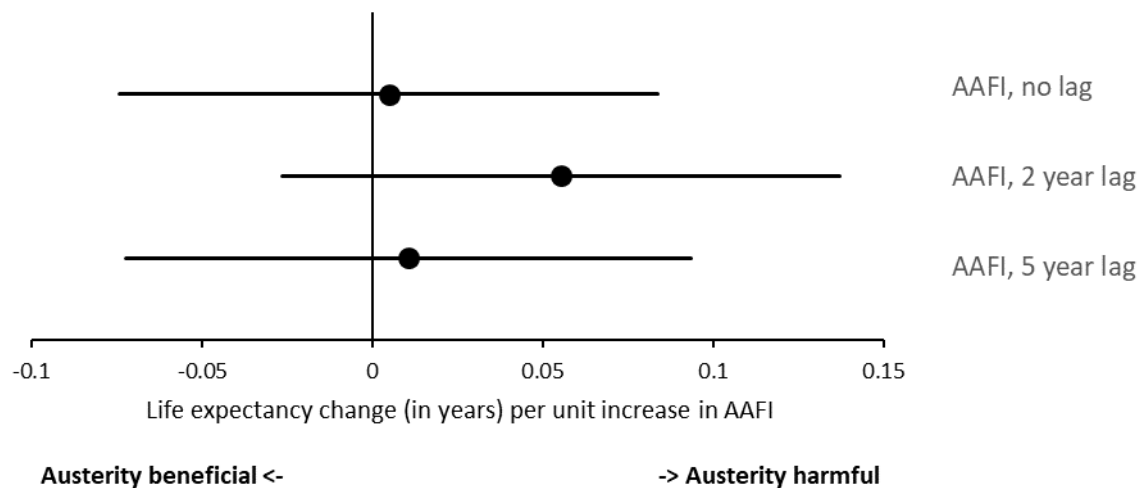
8.9 Sensitivity analysis excluding oil-dominated economies

In recognition of the very different patterning of the measures of austerity in countries whose economies are dominated by oil production (and therefore in which GDP and fiscal balances fluctuate markedly, perhaps reflecting underlying oil and gas prices), as described in Chapters 6 and 7,

an additional sensitivity analysis was undertaken excluding data from Bahrain, Brunei, Kuwait, Oman, Qatar, Saudi Arabia, and the United Arab Emirates. This considered only the relationship between each of the austerity measures and total population life expectancy, but did include analyses for the lags of 0, 2 and 5 years (Table S8.19).

The findings for AAFI are shown in Figure 8.9. As for the unrestricted analysis, the effect size for the relationship between AAFI and total life expectancy is small and imprecise. However, instead of the estimates pointing towards (small, imprecise) beneficial impacts of austerity on this measure (Figure 8.1), these are now all indicating small and imprecise harmful impacts. Thus, the results for AAFI are heavily influenced by the fiscal balance of the countries with economies dominated by oil, and this may indicate that this measure is not as good as hoped at disentangling policy decisions from changes in asset prices and economic cycles.

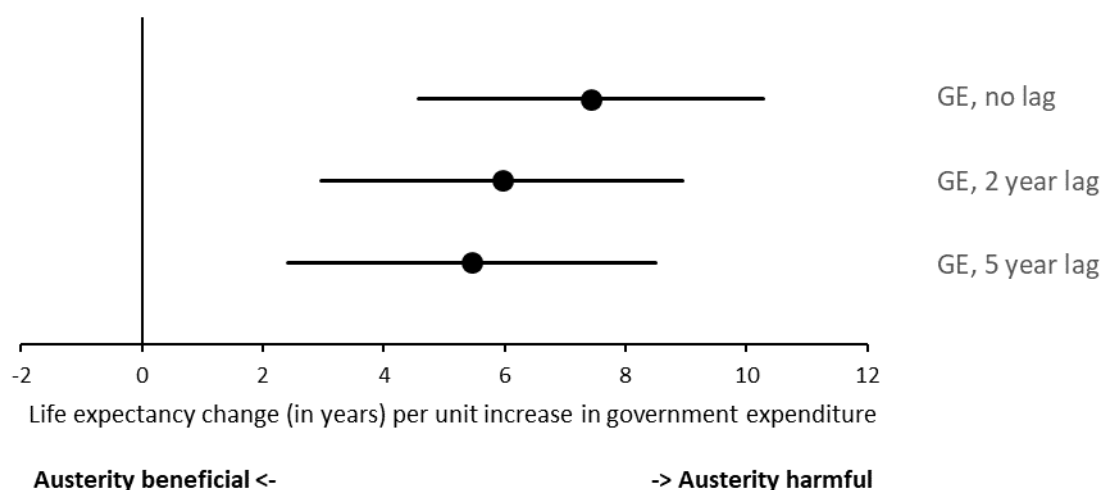
Figure 8.9 - Relationship between AAFI and total population life expectancy, using 0, 2 and 5 year lags between exposure and outcome, excluding oil-dominated economies



The results for the relationship between Governmental Expenditure and life expectancy after restriction are stark (Figure 8.10). Very large negative impacts of austerity are identified across all three lag periods, with the

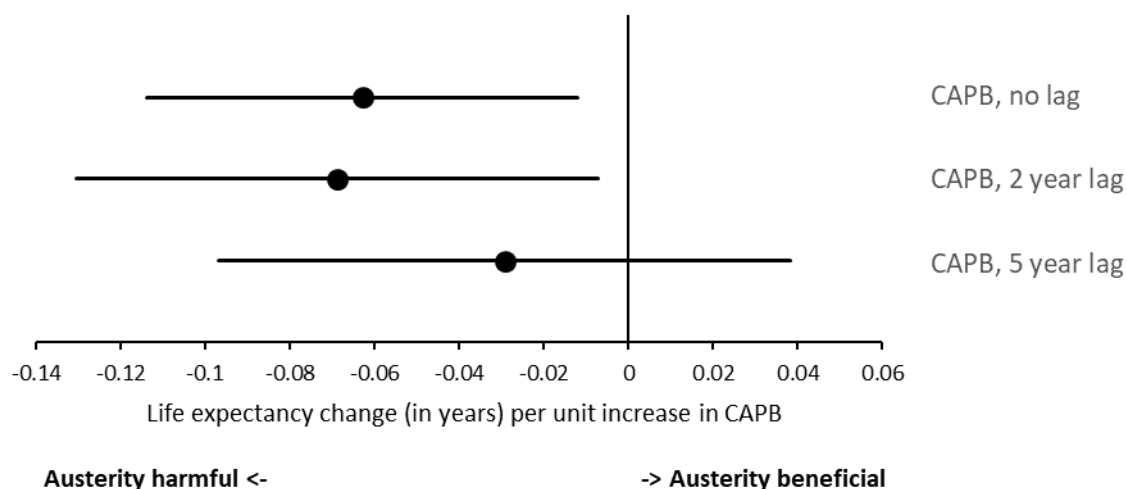
largest effect estimates for the shortest lags. The estimates are imprecise (spanning an effect size of over 6 years in life expectancy change), but because the effect sizes themselves are so large the likelihood of this effect being a result of random chance is negligible. The imprecision of the estimates is likely to be related to a smaller panel of countries being included in the underlying dataset.

Figure 8.10 - Relationship between Government Expenditure (GE) and total population life expectancy, using 0, 2 and 5 year lags between exposure and outcome, excluding oil-dominated economies



The effects of changes in Public Social Spending are consistent with those of Government Expenditure, with negative impacts of austerity again noted, although much smaller (Figure 8.11). There is a more obvious difference in effect size across the three lag periods on this measure, with larger effects for the shorter lags.

Figure 8.12 - Relationship between CAPB and total population life expectancy, using 0, 2 and 5 year lags between exposure and outcome, excluding oil-dominated economies



8.10 Sensitivity analysis restricting exposure periods to post-economic downturns

The final sensitivity analysis restricts the country-time periods in the panel to periods of economic downturn. Economic downturns were defined as the time period in which annual GDP per capita for each country was lower than a previous high point. For example, if GDP per capita (GDPpc) in Country X dropped from \$50,000 in the year 1999 to \$49,000 in the year 2000, but did not rise above \$50,000 again until 2002, the years 2000, 2001 and 2002 would be included in the period defined as an economic downturn, even if GDPpc increased in 2001 compared to 2000, or in 2002 compared to 2001. The purpose of this sensitivity analysis is to attempt to distinguish between austerity implemented during periods of economic growth and downturn, on the basis that there is a marked difference between paying down government debt during periods of growth than during periods of downturn (with the attendant impacts that has on aggregate demand).

The dataset for this analysis is smaller as a result of this restriction, and the estimates generated are consequently generally less precise (Table S8.20).

However, the results are much more consistent across all measures of austerity, showing substantial and statistically significant detrimental impacts on total life expectancy.

Figure 8.13 summarises the relationship for AAFI, where there is a small and imprecise negative impact of austerity, particularly without any lag period. Figure 8.14 shows the relationship for Government Expenditure. This again shows very large estimates (albeit imprecise, but still unlikely to be due to chance), similar to analyses excluding oil-dominated countries. The findings for Public Social Spending again show negative impacts of austerity, except for the 5 year lagged model in which there is a small, but imprecise, positive effect (Figure 8.15). Finally, the relationship for CAPB is shown in Figure 8.16. The estimates here are all imprecise and cross the line of null effect.

Figure 8.13 - Relationship between AAFI and total population life expectancy, using 0, 2 and 5 year lags between exposure and outcome, restricted to economic downturn periods

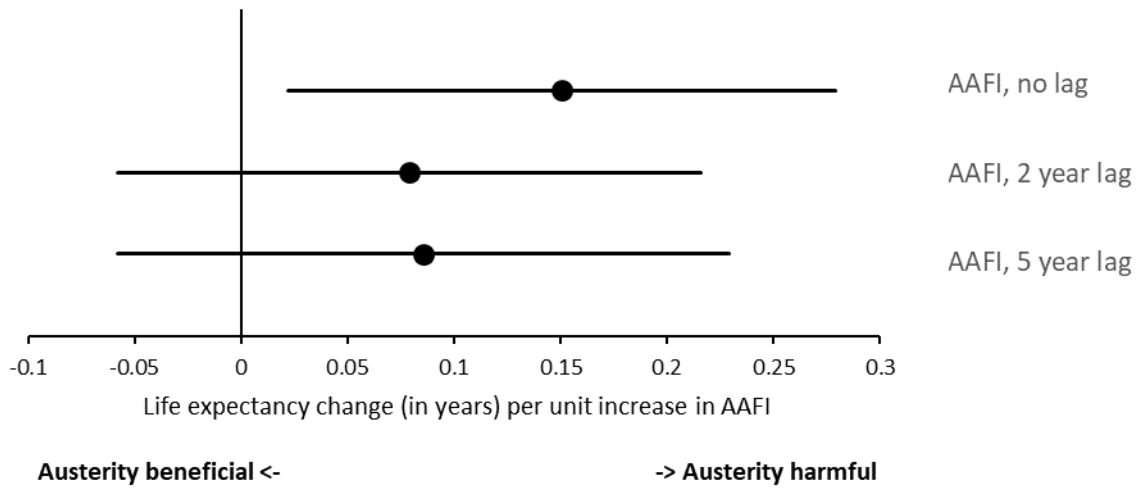


Figure 8.14 - Relationship between Government Expenditure and total population life expectancy, using 0, 2 and 5 year lags between exposure and outcome, restricted to economic downturn periods

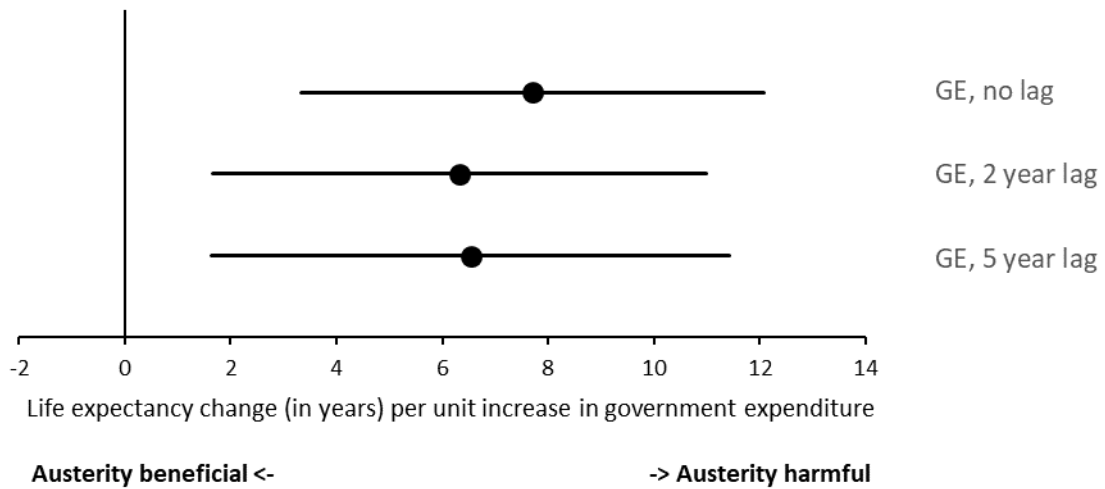


Figure 8.15 - Relationship between Public Social Spending and total population life expectancy, using 0, 2 and 5 year lags between exposure and outcome, restricted to economic downturn periods

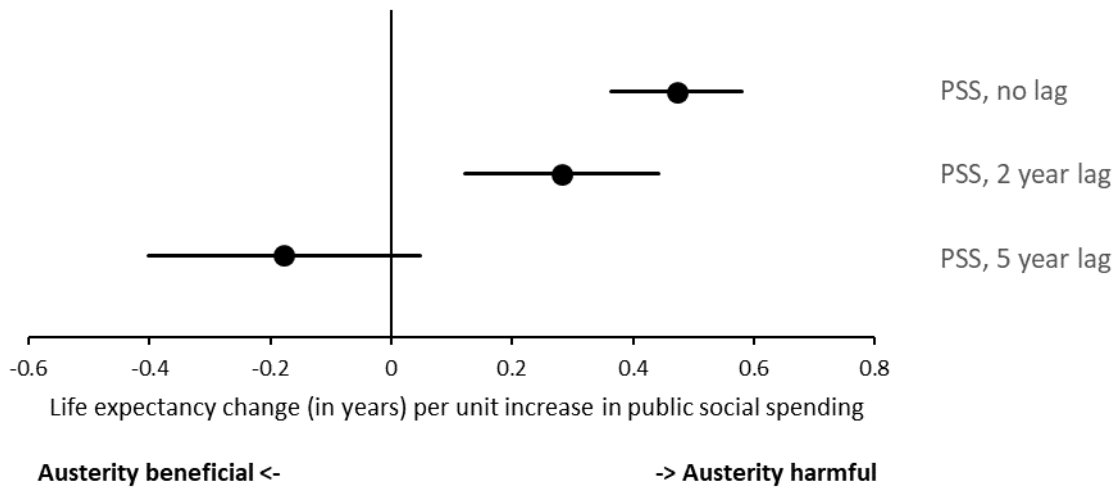
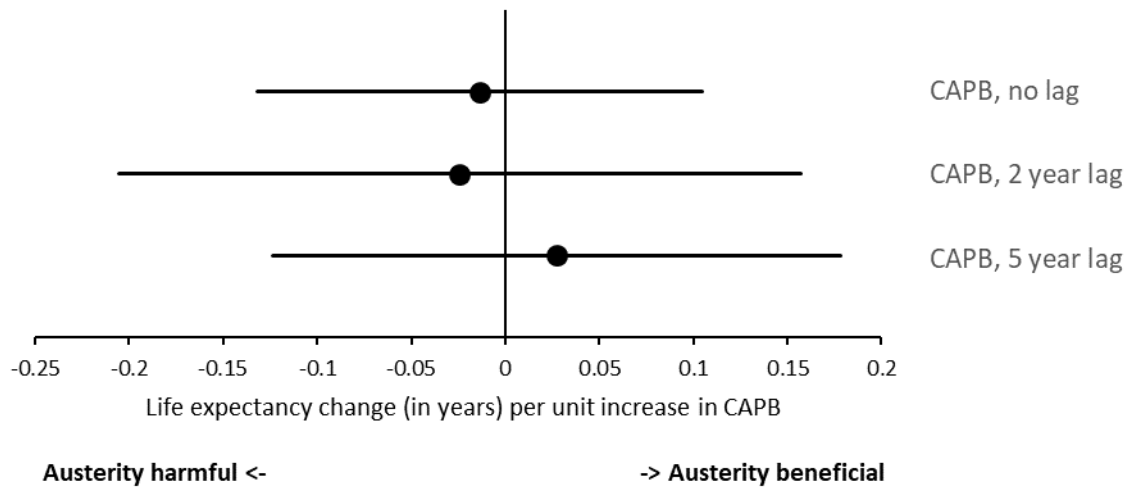


Figure 8.16 - Relationship between CAPB and total population life expectancy, using 0, 2 and 5 year lags between exposure and outcome, restricted to economic downturn periods



8.11 Discussion

This section briefly discusses these results, putting them in context and highlighting the strengths and weaknesses of the approach taken. A fuller discussion of the results across the whole thesis are provided in the next chapter.

The chapter examined the relationship between four measures of austerity (AAFI, Government Expenditure, Public Social Spending and CAPB) and a range of mortality-derived measures (life expectancy, ASMR, age-specific mortality and lifespan variation) for the total population, females and males. The main findings are that for three of the austerity measures (Government Expenditure, Public Social Spending and CAPB) there is evidence that austerity is harmful. The effect sizes are variable, but tend to be largest with shorter lag periods between exposure and outcome, and in many of the models the effects are worse for males. In contrast, the AAFI shows a small, imprecise positive impact of austerity across the mortality measures, but this could have been due to chance and was not significantly different from the null. The imprecision of the estimates for AAFI mean that this positive impact of austerity could be due to random chance, but the consistency across lags and mortality outcomes makes this less likely. A further set of analyses considered the impact of austerity on underemployment and household incomes, but found no large or precise effects.

In addition to the three lag periods used, two other sensitivity analyses were performed. The first restricted the analysis to exclude oil-dominated economies in recognition of the substantial impact variation in oil/gas prices have on those economies and their fiscal balance. This found that restricting in this way created more consistent results of a negative life expectancy impact across all four austerity measures, albeit of very varying scales. The second restricted the analysis to periods of economic downturn. Again, much more consistent results of austerity being harmful to life expectancy

were identified across all austerity measures (although the estimates varied substantially in their size and were generally very imprecise).

The approach taken in this chapter has several important strengths. First, a long panel of data was used (including data between 1987 and 2019), and the time period also included the recent stalling of mortality measures. This meant that there was substantial variation in exposure and outcome measures, avoiding the risks of ubiquity (Pearce, 2011). Second, four different measures of austerity were used, ranging from the more intuitive (Government Expenditure) through to the most sophisticated (AAFI). Although sophisticated, the AAFI revealed itself both in Chapter 7 to be less well correlated with the other measures and to be subject to very short-term instabilities. In this chapter, the AAFI analyses were sensitive to the exclusion of oil-dominated economies, indicating that it may be a less convincing measure of intentional policy change than has been claimed (Alesina, Favero & Giavazzi, 2019a). Third, a series of sensitivity analyses were performed, including changing the time lags between exposure and outcome, adjustments for factors that were likely to be mediators but which could have been confounders (GDP and underemployment), and two panel restrictions (to countries with oil-dominated economies and to periods of economic downturn).

There are however some weaknesses in the approach taken. The diagnostic tests showed several potential issues with the modelling approach used. First, cross-sectional dependence (or contemporaneous correlation) was identified across the panels and models (Hoechle, undated). Second, serial correlation (and associated serially-correlated errors), were identified in the panel and models. This is a common issue in panels with a long time frame but a relatively small number of units of analysis (e.g. in country-level analyses as opposed to panel data for individuals) (Torres-Reyna, 2010). Finally, for two of the four models checked, heteroscedasticity was identified. However, the sandwich estimators for the standard errors (Table S8.6), which controls for heteroscedasticity, had negligible impact on the

size of the standard errors and therefore heteroscedasticity did not impact on the results.

The presence of cross sectional dependence and serial correlation risk residual bias in the estimates generated here (Baltagi et al., 2016). It has been suggested that Monte Carlo simulation modelling can be an approach to estimate their scale, but this form of modelling was outside the scope of this work (Hoechle, undated; Baltagi et al., 2016).

More generally, as shown in Chapter 7, there is something of a disconnect between the measurement of austerity using AAFI in particular, but also to a degree with CAPB, and the popular understanding of austerity (e.g. in the UK austerity has generally been understood as cuts public services and social security benefits). This raises a question of how best to define and measure austerity.

This chapter shows a negative relationship between austerity and mortality which is consistent with several other studies, and in particular during periods of economic downturn. At international level, largely prior to the current period of stalled mortality trends, a negative impact was observed using the AAFI as a measure (Toffoluttia & Suhrcke, 2019). Using mortality data between 2011 and 2015 in Europe, and the CAPB measure of austerity, Rajmil & Fernández de Sanamed (2019) found negative impacts of austerity. However, in their study there was no clear dose-response gradient and the continuous exposure measure was categorised. Within the UK there have been a series of studies considering the health impacts of more specific policy measures than austerity overall, including health and social care spending and local government funding (Loopstra et al., 2016b; Loopstra et al., 2016a; Alexiou et al., 2021; Martin et al., 2021). These have consistently shown negative impacts of austerity although it is difficult to disentangle effects in these sub-national studies because budget cuts were worse in more deprived areas and so there is potential for confounding.

More generally, there is a large body of literature debating the impacts of austerity on a wide range of economic and social outcomes, particularly in the context of the post-2008 financial crash (Konzelmann et al., 2016). Although there remains many advocates of austerity as an effective means of increasing economic growth (Alesina et al., 2019), there seems to be more evidence suggesting that austerity implemented during periods of economic downturn reduces economic growth and prolongs recession, and exacerbates unemployment (Blyth, 2013; Boyer, 2012; Foresti & Marani, 2014; Ostry et al., 2016). Indeed, a recent systematic review of the economics literature in this area (Okeke et al., 2021) suggests that the net impacts are dependent on: the extent to which austerity is operationalised as tax rises or expenditure reductions; the scale of change; the underlying economic conditions (e.g. recession or growth); other policies introduced simultaneously; the duration of the austerity policies; and their design. It is also interesting that contradictory findings for the impact on economic outcomes are found depending on whether periods of austerity are identified narratively or through the CAPB (and equivalents).

The findings described from this thesis support economic policy approaches that avoid austerity, a finding that is more secure for periods of economic downturn and in non-oil-dominated economies, in order to improve health. The implications for policy, practice and future research are discussed in more detail as part of the discussion in Chapter 9 which follows.

9. Discussion and conclusion

9.1 Background

Mortality trends in Scotland, and across many high income countries, displayed substantially slower rates of improvement since around 2012 than in previous decades (ONS, 2018; Fenton et al., 2019a). The causes of these stalled trends have been disputed, and this had led to a lack of clarity in public health explanations and policy recommendations to address this problem (PHE, 2018). In particular, there has been marked debate on whether or not austerity, as a manifestation of political economy, has been a major, or even contributing, factor in driving these trends (Raleigh, 2019; PHE, 2018; Hiam et al., 2018; Raleigh, 2018; Murphy et al., 2019; Hiam et al., 2017; Newton et al., 2017; Baker et al., 2018; Hiam et al., 2021; Newton et al., 2016).

To address the questions raised by this debate, the work of this thesis sought: first to define health, health inequalities and population health; then to summarise and synthesise the theoretical relationships between political economy and population health; to systematically review the existing synthesised evidence of the relationships between political economy and health; to describe the epidemiology of the stalled trends in mortality rates and the trends in measures of austerity (including how these relate to one another); and finally to empirically understand the relationship between austerity measures and mortality-derived outcomes. In doing so, it seeks to provide a contribution towards clarification the role of austerity in explaining the causes of the stalled mortality trends.

9.2 Summary of key contributions of the thesis: Definitions

It is argued in Chapter 2 that health can best be defined as: a structural, functional and emotional state that is compatible with effective life as an

individual and as a member of society. This retains the breadth and multidimensional nature of the early WHO definitions, includes both the individual experience and the functional ability to participate in society, but avoids defining health by its determinants.

Following this, it is argued that health inequalities are best defined as: the systematic, avoidable and unfair differences in health outcomes that can be observed between populations, between social groups within the same population, or as a gradient across a population ranked by social status. This definition is explicit that the differences in health outcomes are systematic and non-random, represent an unfairness or injustice, and can occur between rankable social groups (such as occupational social class) and non-rankable social groups (such as gender or ethnicity). The proposed definition also avoids definition of health inequalities by their causes.

Finally, it is argued that the term 'population health' is best used to: encompass the average, distribution and inequalities in health within a society. As such, it can be used as an overall summary term which incorporates a range of measures of the health of a population.

Considering and defining health terms in this way has not been an esoteric exercise. Within much of the economics literature⁹ health is narrowly defined as an input to the economy: a disembodied and abstracted resource that should be increased and utilised towards the ends of economic growth and accumulation (Hartwig, 2010). To define and operationalise health in this way would mean that changes in mortality and life expectancy trends would only be of concern to the extent that they impact on economic growth metrics. To a lesser extent, some authors have argued that health is largely about the degree to which people can function in society. This can be interpreted in two ways: either as the ability to function in the economy

⁹ This critique is focused on mainstream neoclassical economics rather than *health economics* (a discipline which is principally interested in the relative health impacts of different policies and interventions and their costs), and particularly on the treatment of health as an input rather than outcome within models of the economy.

and to provide labour; or as the ability to participate and be part of society. The latter interpretation is consistent with the definitions here, but misses the experiential elements of health, and the multidimensional aspects of it. The definition proposed here for the first time includes: a multidimensional experience of health; the ability to function and participate in society; health as an individual and population phenomenon; avoids confusion of health as an outcome with its causes; and avoids definition in relation to an ideal and context-free standard.

In a similar way, it is argued that the definition of health inequalities proposed in this thesis for the first time includes specification of them being: systematic and non-random; unfair and unjust; that they are observed between different social groups; and can be observed between categorical or rankable socioeconomic groups, with the latter creating a gradient across society. This proposed definition also avoids the pitfalls of defining health inequalities by its determinants.

The strengths of this contribution to the overall thesis are multiple. First, the definitions proposed are clear, succinct and have a clear rationale for their component parts. Second, they are based on a structured and transparent literature review and critical appraisal that is reproducible by other researchers. The work to define the key terms informed the subsequent systematic review of reviews on the relationship between political economy and health and thus forms a critical and logical step in the argument of the overall thesis.

However, there are limitations to this contribution. First, there are many existing definitions of health and health inequalities, and although, arguably, none are sufficient, there are risks in creating yet another definition. In particular, the lack of a participatory process or inter-agency sponsorship for redefinition means that this work cannot therefore claim to be anything other than the views of the authors. This may therefore create

further disagreement on the best definition of health and lead to a promulgation of inter-disciplinary mismatches in approaches.

Second, although a wide definition of health, including positive and experiential aspects, was defined at the start of the thesis, a lack of data (and time) meant that a much narrower set of measures was used for the empirical analyses, focusing instead on mortality-derived data.

9.3 Summary of key contributions of the thesis: Theory

There are many theoretical theories which seek to provide an explanatory framework for population health. Their heuristic value is important to researchers and policymakers alike, informing how the causes of trends and differences in population health are understood, and thus what research and policy action is taken. The review of these models in Chapter 3 identifies a series of features that have utility from across the available frameworks and models:

- Able to explain average population health and inequalities in health within and between populations;
- Include exposures at all levels from ecological to individuals;
- Incorporates political economy (i.e. social and political processes, institutions and polities which generate differential exposure and outcomes);
- Incorporates historical exposures at individual and societal level;
- Articulates embodiment of exposures (i.e. mechanisms linking exposures to health outcomes);
- Incorporates a clear causal direction and causal pathway; and
- Is simple enough to facilitate widespread use and understanding.

However, as might be expected by the sourcing of features from across models, none incorporate all of these. It is thus argued in Chapter 3 that a new model of political economy and population health can be derived that

can better theorise how these are causally related. This was the theory used to inform the systematic review of reviews in Chapter 4.

There are two key strengths of this new theoretical framework linking political economy and population health. First, it builds on the strengths of existing theoretical models. Second, by identifying the strengths of existing models and designing a new model that incorporates all these aspects, there is a clear rationale for the aspects that have been included or excluded.

However, there are several limitations of the development of the model in Chapter 3. The identification of the theories of population health was not systematic and it is highly likely that other relevant models were therefore not reviewed, and thus facets that could have strengthened the model may have been missed. Furthermore, the identification of models was largely restricted to visual heuristic models rather than theories (with the exception of fundamental cause theory). Relatively new approaches for systematically reviewing the literature for theories have since been developed and could be used in this area of work in the future (Campbell et al., 2014). Finally, some of the critiques made of existing frameworks will be unfair because they were not developed for the purposes of interest here. It is therefore important to recognise that these critiques are largely because of the attempts here to develop a causal theory for political economy and population health, something that few, if any, of the existing theoretical frameworks and models were setting out to do.

9.4 Summary of key contributions of the thesis: political economy and health

Chapter 4 reports a systematic review of reviews of political economy exposures and population health outcomes. There were several contributions from this review. First, that there are few high quality reviews of the population health impacts of political economy (with only 10/58 reviews rated as such). Second, there are some areas of political economy that have no reviews at present. This includes the inter-relationship

between governance, politics, power, macroeconomic policy, public policy and population health. For example, there are no reviews considering the health impacts of patterns of land and capital ownership, or of tax policies. Third, there was sufficient evidence from the included reviews to conclude that follows:

- social democratic welfare states and states with greater public spending seem to have better overall population health, whilst ‘neoliberal’ restructuring seems to be associated with increased health inequalities;
- greater income inequality is associated with lower self-rated health and higher mortality; and
- fair trade policies, extensions to compulsory education provision, microfinance initiatives in low income countries, health and safety policy, improved access to healthcare, and high quality affordable housing are all found to have positive impacts on population health.

Fourth, for several areas there were numerous reviews but none of high quality. This suggests that researchers should focus on producing fewer, but higher quality, reviews in this area.

There are a number of strengths of this review that can be noted. It followed best practice with publication of a protocol in advance of data extraction being undertaken to avoid post-hoc changes of criteria being implemented. The identification of relevant literature was undertaken in a transparent and reproducible manner, with independent, dual and blinded screening of citations and full-texts. All extracted data was checked by another researcher. All included studies were critically appraised and due weight given to the findings of those reviews. This chapter was also peer reviewed as part of the publication process.

However, there were weaknesses to the approach taken. The review sought to cover a wide range of political exposures, and therefore to make the review manageable a review of reviews was undertaken. This means that high

quality primary studies were not sought and, if they hadn't already been included within review studies, they would not have been included. As a result, the sparsity of results in some areas could be due to a lack of reviews rather than a lack of primary research. The second limitation of this type of review relates to the risk of abstraction and de-contextualisation where research results are taken from an existing review rather than the underlying primary studies. This can lead to key messages being warped or misinterpreted due to the distance between the final synthesis and the original studies.

9.5 Summary of key contributions of the thesis: austerity and health

Chapter 7 describes that, in keeping with the literature summarised in Chapter 5, there is evidence of a widespread stalling in mortality trends across high income countries after around 2010. This chapter also describes the trends in four measures of austerity (AAFI, Government Expenditure, Public Social Spending and CAPB) over time. Government Expenditure, Public Social Spending and CAPB show much more similar trends over time than the AAFI measure. This is only partly expected in that, although AAFI attempts to make a more sophisticated adjustment for automatic stabilisers than the CAPB, both should be reasonably aligned because they adjust for changes in unemployment. This malalignment between measures of austerity is a new finding and one that requires further exploration and confirmation.

In exploring the trends in the austerity measures it was also identified that some of the measures have substantially larger swings (i.e. of almost an order of magnitude more) in countries where the economies are more dependent on oil extraction (e.g. the Gulf states). The AAFI and CAPB measures were much less stable than expected pre-analytically, but this reflects that they measure year on year change rather than absolute values of fiscal balance or policy over time.

Chapter 8 shows that austerity, as measured by Government Expenditure, Public Social Spending and CAPB are all harmful to a wide range of mortality-derived measures. In contrast the mortality impact of austerity as measured by AAFI was imprecise but slightly positive. All of these impacts were greatest with no lag time between exposure and outcome, less with a two year lag, and less again (or close to zero) with a five year lag. However, in the sensitivity analyses which restricted the pool of countries to non-oil dominated economies (and thereby reducing the impact of swings in oil prices that were clearly having a large impact on the fiscal balance of oil producing countries), the negative impact of austerity across all four measures was much clearer. Furthermore, restricting the years of interest to those of economic downturns (thereby limiting the definition of austerity to the implementation of fiscal consolidation during or following a recession) had a similar impact of creating a strong signal of negative impacts of austerity on mortality for all austerity measures. Generally, across all analyses, the impacts were worse for males than females, and greater at older than at younger ages.

This is the first time that international analysis has been undertaken of the impact of austerity or mortality-derived measures for the years after 2015, or for more than one measure of austerity. The implication is that austerity measures, particularly when implemented in non-oil dominated economies and in periods of economic downturn, have substantial negative impacts on mortality. This further strengthens the research evidence that austerity policies within the UK are an important cause of the stalled trends, and that austerity has also played a role more widely across high income countries.

There are a number of strengths of the approach taken for these analyses. First, a long time series of data was used including the period of stalled mortality trends across many high income countries. Second, four different measures of austerity were used. Third, a series of sensitivity analyses were performed, including variation in the lag periods, exclusion of oil-dominated countries, and exclusion of time periods outside an economic downturn.

There were weaknesses in the analyses, however. There is the potential that cross-sectional dependence or serial correlation could have biased the results in some of the models, but the direction and magnitude of these biases is unknown. Future analyses could take a different analytical approach to quantifying and reduce these biases, such as using Monte Carlo simulations.

9.6 Implications

Implications of the thesis for policy

The clearest implication of this thesis is that governments who wish to protect the health of their populations, as measured as life expectancy, ASMR, age-specific mortality and lifespan variation, should avoid austerity policies. This is particularly true in periods of economic downturn and for countries whose economies are not dominated by oil extraction. This finding is line with existing evidence at international level which considers the impact austerity across countries, and with the evidence within the UK of the negative impact of specific manifestations of austerity policies. More specifically, austerity is clearly implicated as an important cause of the stalled mortality trends across high income countries since 2010. This should now be recognised as an important cause of the stalled trend by public health agencies, statistical agencies and departments of health.

Implications of the thesis for public health practice

The new definitions created during the work of this thesis for health and health inequalities offer a way of understanding for practitioners that may be more useful than those currently used. These definitions include all relevant aspects of health, clarify what health inequalities are, and avoid some of the problems of alternative approaches. Arguably this has become even more important recently when health inequalities as a term seems to be once again under threat because of the injustices it makes clear (e.g. as

with the creation of the ‘Office for Health Improvement and Disparities’ in England). Having a clear definition with a robust rationale can therefore be an important ballast against those who might deliberately or inadvertently create uncertainty or muddle in the efforts to improve health and reduce health inequalities. Similarly, the theoretical model of political economy and population health, which is supported by the evidence synthesised in the systematic review of reviews, shows the importance of policy, institutions, politics, taxation, etc. for population health. This evidence base and theoretical model can now be used to inform policymaking at all levels, for impact assessments, and for advocacy by public health and other actors in the third sector and political sphere.

Implications of the thesis for future research

Future research could improve on the work of this thesis in several ways, and address some of the research gaps and extension work that follows on from it:

1. Future definitional work could usefully incorporate consensus building approaches to bridge between the scientific contribution and the needs of policymakers and practice (as exemplified by Braveman et al. (2017)). In particular, the involvement of international health bodies such as the WHO would be important in any such work.
2. Future development of theories linking political economy and population health could usefully be based upon a systematic review of the theoretical literature in this area which may reveal further aspects that would enhance the heuristic model developed in Chapter 3. This could also involve a consensus building stage to ensure utility across policy and practice.
3. Future systematic reviews of aspects of political economy and health should focus on their quality so that they do not simply add to the ‘research noise’ in this area.

4. There is scope to undertake analyses of the impact of austerity on population health using a number of alternative methods that could help to triangulate across approaches and strengthen understanding.

This could include:

- a. Qualitatively-derived measures of austerity, such as policy reviews or Qualitative Case Analysis. This may help to isolate the key austerity policies that are most deleterious for health and any contextual dependencies.
- b. Case study analyses of the particular experiences of individual countries, with the potential use of counterfactuals operationalised as synthetic controls or as more simple difference in differences between countries implementing more and less austere policies in relevant time periods.
- c. More sophisticated analyses of the panel data used in this study, to account for serial correlation and cross-sectional dependence, could help to reduce the uncertainties in the effect estimates.
- d. Examining whether the nature of austerity influences the impact on population health: specifically whether austerity due to changes in taxation or government spending is more important, and whether the distributional consequences of austerity is important.
- e. Inclusion of a wider range of health and health inequality outcomes which more fully address the definitions from Chapter 2.
- f. Inclusion of a sensitivity analysis that restricts the panel to non-oil dominated economies and periods of economic downturn simultaneously.
- g. Extension of the analyses to low and middle income countries, both in terms of the identification of stalled mortality and life

expectancy trends, and the relationship between mortality-derived trends to measures of austerity.

9.7 Conclusions

This thesis demonstrates the importance of a wide range of political economy exposures for population health. It is clear that political economy matters for health and that policy, politics, institutions, regulations, taxes, etc. are of utmost importance for all that are concerned about the health of populations.

It is clear that social democratic welfare states and states with greater public spending seem to have better overall population health than countries with different welfare state types and lower public spending. Countries that undertake 'neoliberal' restructuring exacerbate health inequalities for their populations. Income inequality is associated with worse health. However, fair trade policies, extended compulsory education, microfinance initiatives in low income countries, the introduction of health and safety policies, improved healthcare access and the provision of high quality and affordable housing are all good for population health.

It is now also clear that austerity, particularly when implemented in non-oil dominated economies and during economic downturns, is damaging for mortality trends and is an important cause of the stalled mortality trends seen since 2010.

Governments around the world are currently grappling with the multitude of challenges associated with the COVID-19 pandemic. These include the need to make a rapid transition to an ecologically sustainable economy, rebuild economies, and address the stalled mortality trends. The policies that are simultaneously evidenced to be effective for all of these challenges are known, and reflect a need to redesign and re-orientate economies to serve population needs rather than economic growth. This thesis adds evidence for policymakers that supports this transition.

Supplementary material for Chapter 6

Age standardisation

The Human Mortality Database (HMD) provides age stratified mortality rates for 0-1, 1-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85-89, 90-94, 95-99, 100-104, 105-109 and 110+ years strata. However, the European Standard Population (ESP) has an upper age group of 95+ years (Publications Office of the European Union, 2013), creating a mismatch between the age structure of the mortality data and the standard population of choice (Table S6.1).

To resolve this difference an estimate of how the 200 people in the 95+ years upper age stratum in the ESP should be distributed across the HMD breakdown above the age of 95 years was required. The UK Office for National Statistics (ONS) provide detailed estimates of the population sizes for older age groups in the UK, up to 104 years, with an open upper group of 105+ years (ONS, 2019). Using the 2013 UK estimate as a basis for estimating the distribution of the population of 200 persons in the ESP in this upper age strata, the proportion at each single year of age from age 95 to 104 years, and for all those aged 105+ years, was calculated (column (C) in Table S2.2 below). To estimate the proportions at each age from 105 years upwards, the proportionate change in the size of the population with a single year increase in age was calculated from the ONS data (column (D) in Table S2.2 below). The proportion tended towards 0.55 and this was used to divide the 1.38 persons estimated to be aged 105+ years in column (C) in Table S6.2 by multiplying the estimated population size for those aged 104 years (1.16 people) by 0.55 and each subsequent age up to 109 years. This leaves an estimated 0.03 persons aged 110+ years after to be included in the open ended upper age stratum. Summing the number of people at the individual years of age into the 5 year age strata provides a means of expanding the ESP at the older ages to use with the age-specific mortality rates available

from HMD. This provides estimates of populations of for the 95-99, 100-104, 105-109 and 110+ years age strata respectively (Column (D) in Table S6.1.

Table S6.1 - The current ESP and adapted standard population sizes across age strata

(A) ESP age strata (years)	(B) ESP	(C) HMD age strata	(D) Adapted ESP
0-1	1,000	0-1	1,000.00
1-4	4,000	1-4	4,000.00
5-9	5,500	5-9	5,500.00
10-14	5,500	10-14	5,500.00
15-19	5,500	15-19	5,500.00
20-24	6,000	20-24	6,000.00
25-29	6,000	25-29	6,000.00
30-34	6,500	30-34	6,500.00
35-39	7,000	35-39	7,000.00
40-44	7,000	40-44	7,000.00
45-49	7,000	45-49	7,000.00
50-54	7,000	50-54	7,000.00
55-59	6,500	55-59	6,500.00
60-64	6,000	60-64	6,000.00
65-69	5,500	65-69	5,500.00
70-74	5,000	70-74	5,000.00
75-79	4,000	75-79	4,000.00
80-84	2,500	80-84	2,500.00
85-89	1,500	85-89	1,500.00
90-94	800	90-94	800.00
95+	200	95-99	174.32
		100-104	24.30
		105-109	1.35
		110+	0.03
Total	100,000		100,000.00

Table S6.2 - Application of the ONS population estimates to estimate the breakdown of the ESP into the oldest age strata to allow calculation using HMD estimates

(A) Age (years)	(B) UK population estimate , 2013	(C) UK population proportions applied to a population of 200	(D) Proportionat e change in population size with each unit increase in age	(E) Age (years)	(F) Applying proportionate change of 0.55 to 105+ years
95	27,440	54.87			
96	22,170	44.34	0.81		
97	16,970	33.94	0.77		
98	12,360	24.72	0.73		
99	8,230	16.46	0.67		
100	5,330	10.66	0.65		
101	3,260	6.52	0.61		
102	1,920	3.84	0.59		
103	1,060	2.12	0.55		
104	580	1.16	0.55		
105+	690	1.38		105	0.64
				106	0.35
				107	0.19
				108	0.11
				109	0.06
				110+	0.03*

*The number of people in the standard population estimated to be 110+ years is calculated as the difference between the 1.38 persons estimated to be aged 105+ in the standard population (column (C)) and the populations estimated at each age from age 105 to 109 years in column (F).

Supplementary material for Chapter 7

Table S7.1 - Availability of exposures and outcomes data between 1980 and 2019 inclusive

High income countries (sample frame)	Exposures data						Outcomes data			
	Real final Government Expenditure	CAPB	Pubic social spending	Unemployment (needed for AAFI)*	Government expenditure as % of GDP (needed for AAFI)*	Government revenue as % of GDP (needed for AAFI)*	ASMR, age-specific mortality, lifespan variation	Life expectancy	Under-employment**	Household incomes ***
<i>Data downloaded on:</i>	13/2/21	27/4/20	15/12/20	11/12/20	14/1/21	17/1/21	5/12/20	11/12/19	19/12/20	10/01/21
Andorra	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Antigua and Barbuda	2010	n/a	n/a	n/a	n/a	1990-2018	n/a	n/a	2010	2010
Aruba	2010	n/a	n/a	1998-2019	n/a	1995-2018	n/a	n/a	2010	2010
Australia	1980-2019	1991-2019	1990-2017	1980-2019	1991-2019	1990-2018	1980-2018	1980-2016	1980-2019	1980-2019
Austria	1980-2019	1995-2019	1990-2019	1980-2019	1991-2019	1990-2019	1980-2017	1980-2017	2008-2019	1980-2019
Bahamas, The	1989-2019	n/a	n/a	1986-2019	n/a	1990-2019	n/a	n/a	n/a	1989-2019
Bahrain	2000-2019	n/a	n/a	2007-2019	n/a	1990-2018	n/a	n/a	n/a	2000-2019
Barbados	2010	n/a	n/a	1981-2019	n/a	1994-2019	n/a	n/a	2016	2010
Belgium	1980-2019	1990-2019	1990-2019	1980-2019	1991-2019	1990-2018	1980-2018	1980-2017	2018-2019	1980-2019
Bermuda	2010	n/a	n/a	n/a	n/a	n/a	n/a	n/a	2012	2010
British Virgin Islands	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Brunei Darussalam	1989-2019	n/a	n/a	2011-2019	n/a	1990-2019	n/a	n/a	2017-2019	1989-2019
Canada	1980-2019	1990-2019	1990-2018	1980-2019	1991-2019	1990-2019	1980-2016	1980-2016	1997-2019	1980-2019
Cayman Islands	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	2011-2015	n/a
Channel Islands	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Chile	1980-2019	2001-2019	1990-2019	1980-2019	1991-2019	1990-2018	1992-2017	1992-2008	2009-2019	1980-2019
Croatia	1995-2019	2002-2019	1990-2019	1991-2019	1993-2019	1992-2018	2001-2018	2002-2017	2007-2019	1995-2019
Curaçao	n/a	n/a	n/a	1982-2019	n/a	n/a	n/a	n/a	2018	n/a
Cyprus	1980-2019	1995-2019	n/a	1982-2019	1996-2019	1995-2019	n/a	n/a	2010-2019	1980-2019
Czech Republic	1990-2019	1995-2019	1990-2019	1995-2019	1996-2019	1995-2019	1980-2018	1980-2017	2012-2019	1990-2019
Denmark	1980-2019	1990-2019	1990-2019	1980-2019	1991-2019	1990-2019	1980-2019	1980-2016	2018-2019	1980-2019
Estonia	1993-2019	2000-2019	1999-2019	1993-2019	1996-2019	1995-2019	1980-2017	1980-2017	2006-2019	1993-2019
Faeroe Islands	2010	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	2010

High income countries (sample frame)	Exposures data						Outcomes data			
	Real final Government Expenditure	CAPB	Pubic social spending	Unemployment (needed for AAFI)*	Government expenditure as % of GDP (needed for AAFI)*	Government revenue as % of GDP (needed for AAFI)*	ASMR, age-specific mortality, lifespan variation	Life expectancy	Under-employment**	Household incomes ***
Finland	1980-2019	1990-2019	1990-2019	1980-2019	1991-2019	1990-2018	1980-2019	1980-2018	2009-2019	1980-2019
France	1980-2019	1990-2019	1990-2019	1980-2019	1991-2019	1990-2018	1980-2018	1980-2017	2015-2019	1980-2019
French Polynesia	n/a	n/a	n/a		n/a	n/a	n/a	n/a	n/a	n/a
Germany	1980-2019	1991-2019	1990-2019	1980-2019	1992-2019	1991-2019	1990-2017	1990-2017	2012-2019	1980-2019
Gibraltar	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Greece	1980-2019	1990-2019	1990-2019	1980-2019	1991-2019	1990-2018	1981-2017	1981-2013	2010-2019	1980-2019
Greenland	2003-2018	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	2003-2018
Guam	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	2002-2018
Hong Kong SAR, China	1980-2019	n/a	n/a	1980-2019	n/a	n/a	1986-2017	1986-2017	2009-2016	1980-2019
Hungary	1991-2019	2000-2019	1999-2019	1980-2019	1996-2019	1995-2019	1980-2017	1980-2017	2006-2019	1991-2019
Iceland	1980-2019	1990-2019	1990-2019	1980-2019	1991-2019	1990-2018	1980-2018	1980-2016	2001-2019	1990-2019
Ireland	1980-2019	1997-2019	1999-2019	1985-2019	1991-2019	1990-2018	1980-2017	1980-2017	2018-2019	1980-2019
Isle of Man	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Israel	1980-2019	2000-2019	1995-2019	1980-2019	2001-2019	2000-2019	1983-2016	1983-2016	2009-2018	1980-2019
Italy	1980-2019	1990-2019	1990-2019	1980-2019	1991-2019	1990-2019	1980-2017	1980-2014	2006-2019	1980-2019
Japan	1980-2018	1994-2019	1990-2017	1980-2019	1991-2019	1990-2018	1980-2019	1980-2017	2009-2019	1980-2018
Korea, Rep.	1980-2019	1995-2019	1990-2019	1980-2019	1996-2019	1995-2017	2003-2018	2003-2018	2015-2019	1980-2019
Kuwait	2010-2018	n/a	n/a	1981-2018	1991-2019	1990-2019	n/a	1980-2017	n/a	2010-2018
Latvia	1995-2019	2003-2019	1997-2019	1992-2019	1999-2019	1998-2019	1980-2017	1980-2017	2006-2019	1995-2019
Liechtenstein	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Lithuania	1995-2019	2005-2019	1996-2019	1999-2019	1996-2019	1993-2018	1980-2019	1980-2017	2006-2019	1995-2019
Luxembourg	1980-2019	1995-2019	1990-2019	1980-2019	1996-2019	1995-2019	1980-2019	1980-2017	2016-2019	1980-2019
Macao SAR, China	1982-2019	n/a	n/a	2001-2019	n/a	2001-2018	n/a	n/a	2009-2016	1982-2019
Malta	2000-2019	2000-2019	n/a	1983-2019	2001-2019	2000-2018	n/a	n/a	2007-2019	2010
Monaco	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Netherlands	1980-2019	1995-2019	1990-2019	1980-2019	1991-2019	1990-2019	1980-2018	1980-2016	2006-2019	1980-2019
New Caledonia	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
New Zealand	1980-2018	2005-2019	1990-2018	1980-2019	1991-2019	1990-2019	1980-2013	1980-2013	n/a	1980-2018
Northern Mariana Islands	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	2002-2018
Norway	1980-2019	1990-2019	1990-2019	1980-2019	1991-2019	1990-2019	1980-2018	1980-2018	2007-2019	1980-2019
Oman	1998-2019	n/a	n/a	n/a	1991-2019	1990-2018	n/a	n/a	n/a	1998-2019

High income countries (sample frame)	Exposures data						Outcomes data			
	Real final Government Expenditure	CAPB	Pubic social spending	Unemployment (needed for AAFI)*	Government expenditure as % of GDP (needed for AAFI)*	Government revenue as % of GDP (needed for AAFI)*	ASMR, age-specific mortality, lifespan variation	Life expectancy	Under-employment**	Household incomes ***
Palau	2005-2018	n/a	n/a	n/a	n/a	2000-2018	n/a	n/a	n/a	2005-2018
Panama	1980-2019	n/a	n/a	1980-2019	n/a	1994-2018	n/a	n/a	2011-2019	1980-2019
Poland	1995-2019	1995-2019	1990-2019	1990-2019	1996-2019	1995-2018	1980-2018	1980-2016	2013-2019	1995-2019
Portugal	1980-2019	1995-2019	1990-2019	1980-2019	1991-2019	1990-2019	1980-2018	1980-2015	2012-2019	1980-2019
Puerto Rico	1980-2019	n/a	n/a	1980-2019	n/a	n/a	n/a	n/a	n/a	1980-2019
Qatar	2010	n/a	n/a	n/a	1991-2019	1990-2019	n/a	n/a	n/a	2010
San Marino	n/a	n/a	n/a	2001-2019	n/a	2004-2018	n/a	n/a	n/a	n/a
Saudi Arabia	2000-2019	n/a	n/a	1999-2018	1991-2019	1991-2019	n/a	n/a	n/a	2000-2019
Seychelles	2010	n/a	n/a	1995-2019	n/a	1990-2019	n/a	n/a	2015-2017	2010
Singapore	1980-2019	n/a	n/a	1980-2019	1991-2019	1990-2019	n/a	n/a	2009-2019	1980-2019
Sint Maarten (Dutch part)	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Slovak Republic	1992-2019	1995-2019	1995-2019	1993-2019	1996-2019	1995-2019	1980-2017	1980-2017	2012-2019	1993-2019
Slovenia	1990-2019	1996-2019	1995-2019	1992-2019	1996-2019	1995-2019	1983-2017	1983-2017	2006-2019	1990-2019
Spain	1980-2019	2000-2019	1990-2019	1980-2019	1991-2019	1990-2019	1980-2018	1980-2016	2002-2019	1980-2019
St. Kitts and Nevis	n/a	n/a	n/a	n/a	n/a	1990-2018	n/a	n/a	n/a	n/a
St. Martin (French part)	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Sweden	1980-2019	1993-2019	1990-2019	1980-2019	1991-2019	1990-2019	1980-2019	1980-2017	2019	1980-2019
Switzerland	1980-2019	1990-2019	1990-2015	1980-2019	1991-2019	1990-2017	1980-2018	1980-2016	2011-2019	1980-2019
Taiwan, China		n/a	n/a	1980-2019	n/a	1990-2018	1980-2014	1980-2014	n/a	n/a
Trinidad and Tobago	n/a	n/a	n/a	1981-2018	n/a	1990-2019	n/a	n/a	n/a	n/a
Turks and Caicos Islands	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
United Arab Emirates	2011-2019	n/a	n/a	n/a	1992-2019	1990-2018	n/a	n/a	2018-2019	1980-2019
United Kingdom	1980-2019	1990-2019	1990-2019	1980-2019	1991-2019	1990-2019	1980-2018	1980-2016	2009-2019	1980-2019
Northern Ireland	n/a	n/a	n/a	n/a	n/a	n/a	1980-2018	1980-2016	n/a	n/a
Scotland	n/a	n/a	n/a	n/a	n/a	n/a	1980-2018	1980-2016	n/a	n/a
England & Wales	n/a	n/a	n/a	n/a	n/a	n/a	1980-2018	1980-2016	n/a	n/a
United States	1980-2019	2001-2019	1990-2019	1980-2019	2002-2019	2001-2018	1980-2018	1980-2017	1994-2019	1980-2019
Uruguay	1980-2019	2000-2019	n/a	1983-2019	2000-2019	1999-2018	n/a	n/a	1998-2019	1980-2019

High income countries (sample frame)	Exposures data						Outcomes data			
	Real final Government Expenditure	CAPB	Pubic social spending	Unemployment (needed for AAFI)*	Government expenditure as % of GDP (needed for AAFI)*	Government revenue as % of GDP (needed for AAFI)*	ASMR, age-specific mortality, lifespan variation	Life expectancy	Under-employment**	Household incomes ***
Virgin Islands (U.S.)	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n./a	n/a	2002-2017

n/a = data not available

* The IMF provide data up to 2025 as a projection. A cut-off of 2019 was made to avoid projected data.

** There are numerous data marked as unreliable or as breaks in the dataset. Only the data series without changes in methods, and the years with reliable data, have been extracted (and this is the availability noted in the table).

*** Households and Non-profit institutions serving households (NPISHs) Final consumption expenditure per capita (constant 2010 US\$).

Figure S7.1 - Trend in life expectancy at birth for females (1987-2020, note shortened y-axis)

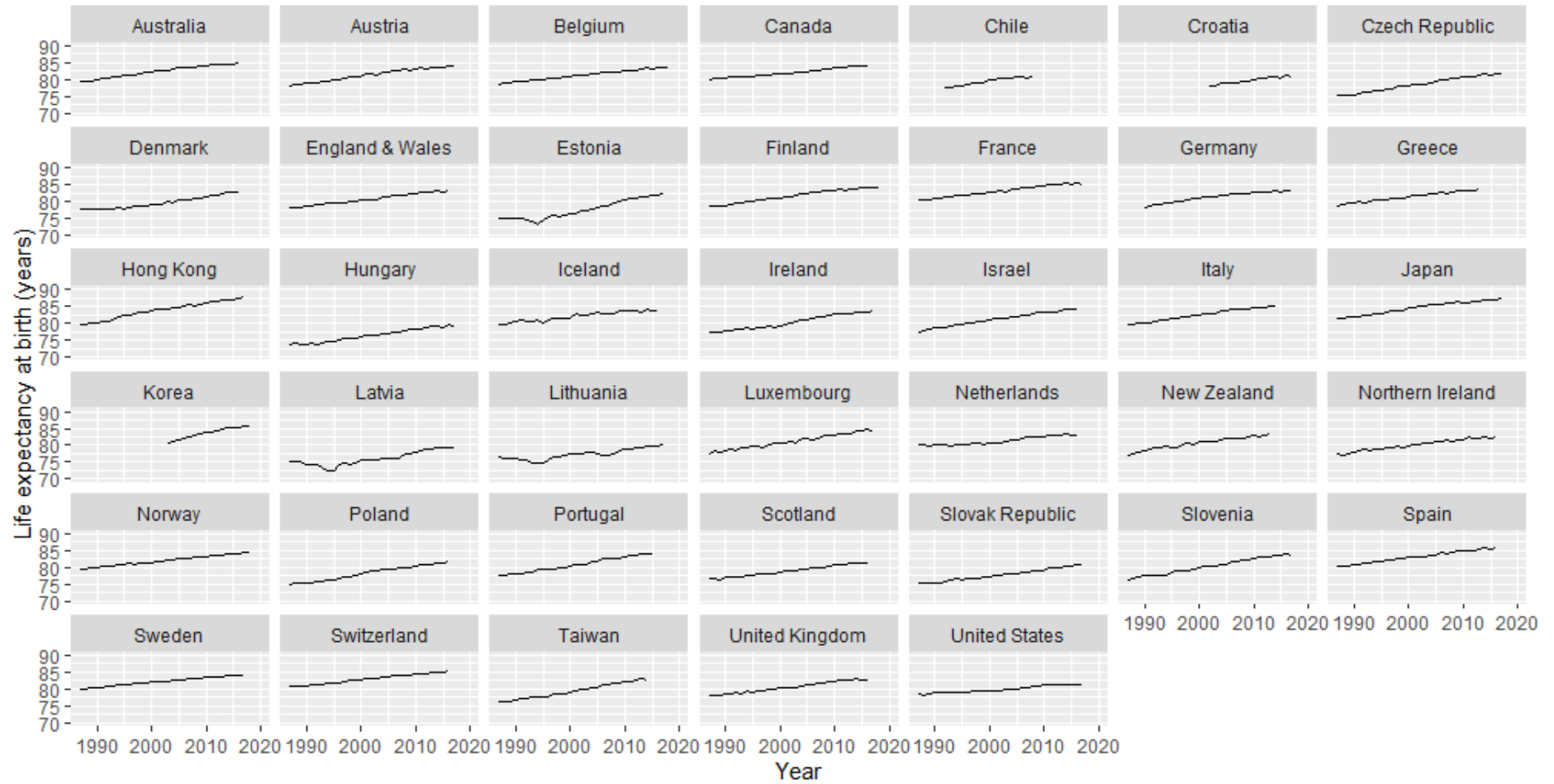


Figure S7.2 - Trend in life expectancy at birth for males (1987-2020, note shortened y-axis)

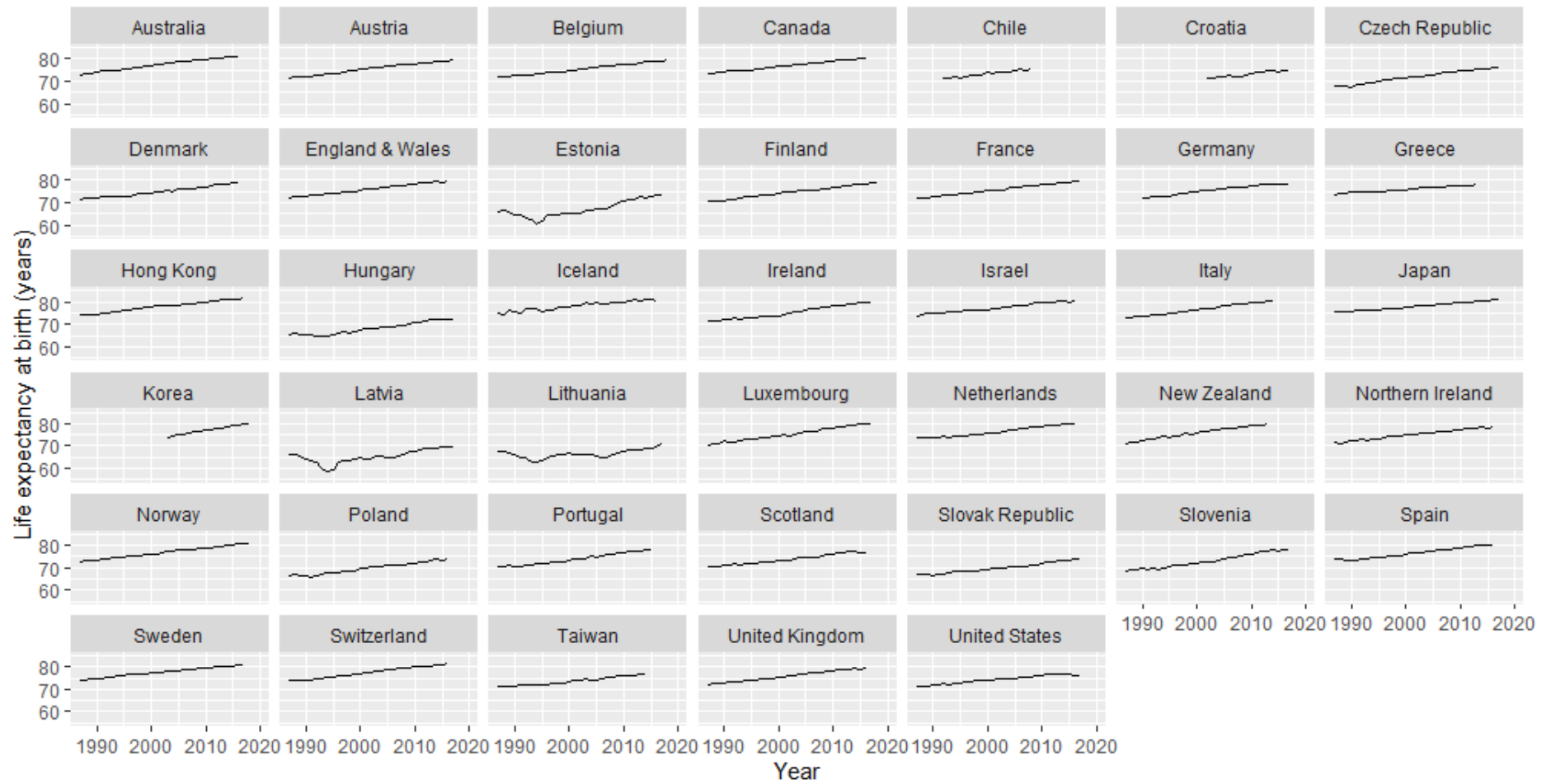


Figure S7.3 - Segmented regression fitted to the life expectancy trends for females (2000-2019, note shortened y-axis, vertical lines indicate breakpoint)

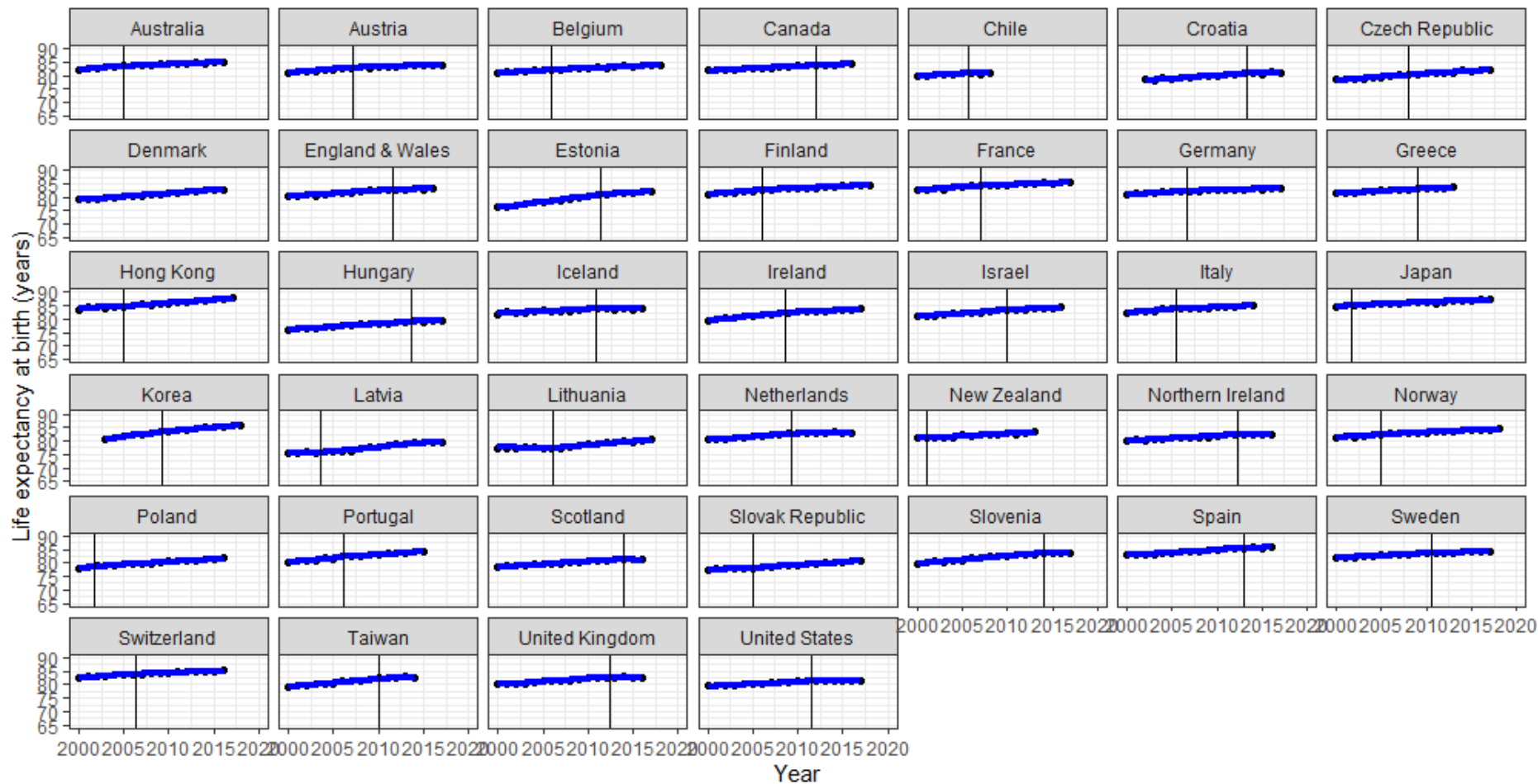


Figure S7.4 - Segmented regression fitted to the life expectancy trends for males (2000-2019, note shortened y-axis, vertical lines indicate breakpoint)

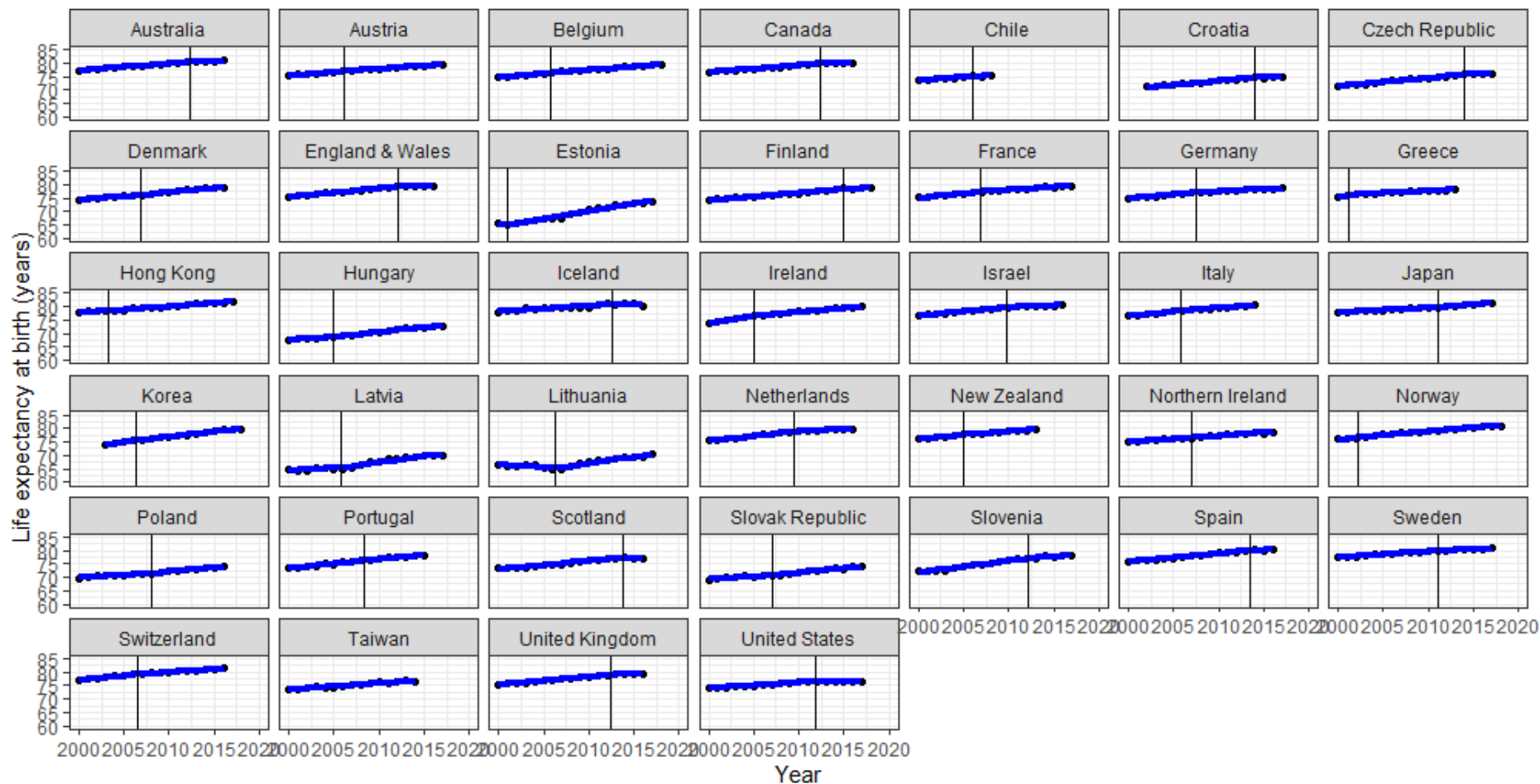


Table S7.2 - Estimated breakpoints in the life expectancy trends between 2000 and 2019, and the regression line slopes for the total population, females and males

Country	Total population			Females			Males		
	Break	Slope 1*	Slope 2*	Break	Slope 1*	Slope 2*	Break	Slope 1*	Slope 2*
Australia	2010.2	0.23	0.13	2005.0	0.24	0.14	2012.4	0.27	0.09
Austria	2006.8	0.26	0.16	2007.1	0.23	0.12	2006.1	0.31	0.21
Belgium	2005.9	0.27	0.19	2006.0	0.20	0.14	2005.8	0.32	0.23
Canada	2012.2	0.21	0.10	2012.0	0.17	0.10	2012.4	0.25	0.10
Chile	2006.0	0.22	0.05	2005.7	0.18	0.05	2006.0	0.25	0.08
Croatia	2013.7	0.26	0.10	2013.3	0.22	0.07	2014.0	0.30	0.13
Czech Rep.	2014.0	0.26	0.13	2014.0	0.24	0.11	2015.8	0.28	-0.04
Denmark	2007.0	0.24	0.29	2014.6	0.25	0.10	2006.8	0.26	0.33
E-&W	2012.2	0.26	0.01	2011.6	0.23	0.03	2012.2	0.30	0.04
Estonia	2013.0	0.51	0.27	2011.4	0.42	0.22	2001.1	-0.44	0.57
Finland	2015.0	0.23	0.11	2006.0	0.27	0.14	2015.0	0.28	0.20
France	2006.9	0.29	0.16	2007.0	0.24	0.11	2006.8	0.33	0.22
Germany	2006.8	0.25	0.13	2006.6	0.21	0.10	2007.4	0.30	0.16
Greece	2001.1	0.45	0.17	2009.0	0.19	0.10	2001.2	0.47	0.17
Hong Kong	2004.5	0.15	0.26	2004.9	0.15	0.25	2003.3	0.12	0.25
Hungary	2005.0	0.23	0.28	2013.7	0.21	0.08	2005.0	0.21	0.35
Iceland	2014.2	0.17	-0.30	2011.0	0.16	-0.01	2014.9	0.20	-0.78
Ireland	2005.5	0.47	0.24	2008.6	0.35	0.15	2005.1	0.53	0.29
Israel	2009.7	0.28	0.14	2009.9	0.24	0.14	2009.7	0.32	0.14
Italy	2005.6	0.30	0.19	2005.6	0.25	0.14	2005.7	0.35	0.24
Japan	2011.8	0.15	0.21	2001.7	0.33	0.14	2011.0	0.17	0.25
Korea	2008.4	0.49	0.33	2009.3	0.45	0.28	2006.4	0.56	0.38
Latvia	2005.5	0.17	0.40	2003.5	0.04	0.31	2005.7	0.20	0.45
Lithuania	2006.2	-0.15	0.41	2006.2	-0.02	0.29	2006.3	-0.23	0.50
Netherlands	2009.4	0.30	0.12	2009.4	0.25	0.08	2009.4	0.36	0.18
New Zealand	2005.0	0.26	0.22	2001.0	-0.22	0.19	2005.0	0.34	0.25
N. Ireland	2013.7	0.21	0.01	2012.3	0.19	-0.01	2007.0	0.22	0.26
Norway	2004.4	0.32	0.21	2005.0	0.24	0.16	2004.0	0.39	0.26
Poland	2008.0	0.21	0.27	2001.7	0.42	0.21	2008.0	0.20	0.32
Portugal	2007.4	0.35	0.25	2006.2	0.33	0.22	2008.4	0.38	0.26
Scotland	2013.8	0.26	-0.17	2014.0	0.20	-0.14	2013.8	0.32	-0.20
Slovak Rep.	2006.8	0.20	0.28	2005.0	0.15	0.22	2007.0	0.22	0.33
Slovenia	2014.0	0.35	0.06	2014.0	0.27	0.05	2014.0	0.43	0.08
Spain	2013.2	0.26	0.10	2013.0	0.20	0.08	2013.5	0.31	0.10
Sweden	2010.7	0.19	0.13	2010.5	0.15	0.10	2011.0	0.22	0.16
Switzerland	2006.4	0.28	0.17	2006.4	0.21	0.13	2006.5	0.34	0.22
Taiwan	NA	0.26	NA	2010.0	0.30	0.16	NA	0.23	NA
UK	2012.3	0.26	0.01	2012.4	0.22	-0.01	2012.3	0.30	0.03
USA	2011.7	0.20	-0.03	2011.5	0.18	0.01	2011.8	0.22	-0.05

*The slopes refer to the rate of change in life expectancy for the period before (Slope 1) and after (Slope 2) the breakpoint, measured in years of life expectancy per year.

Figure S7.5 - Scatterplot of the rate of change in life expectancy for females before and after the breakpoint (2000-2019)

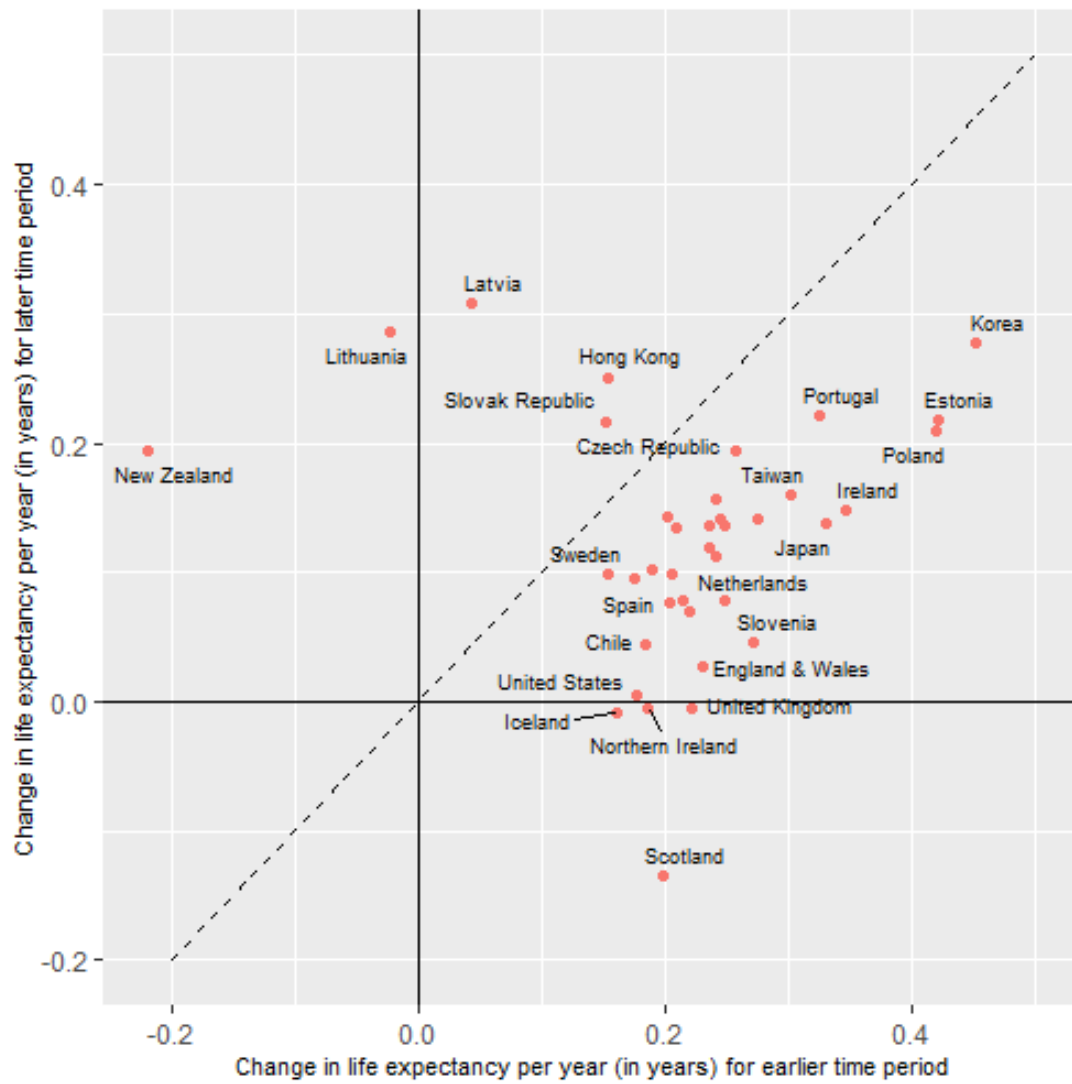


Figure S7.6 - Scatterplot of the rate of change in life expectancy for males before and after the breakpoint (2000-2019)

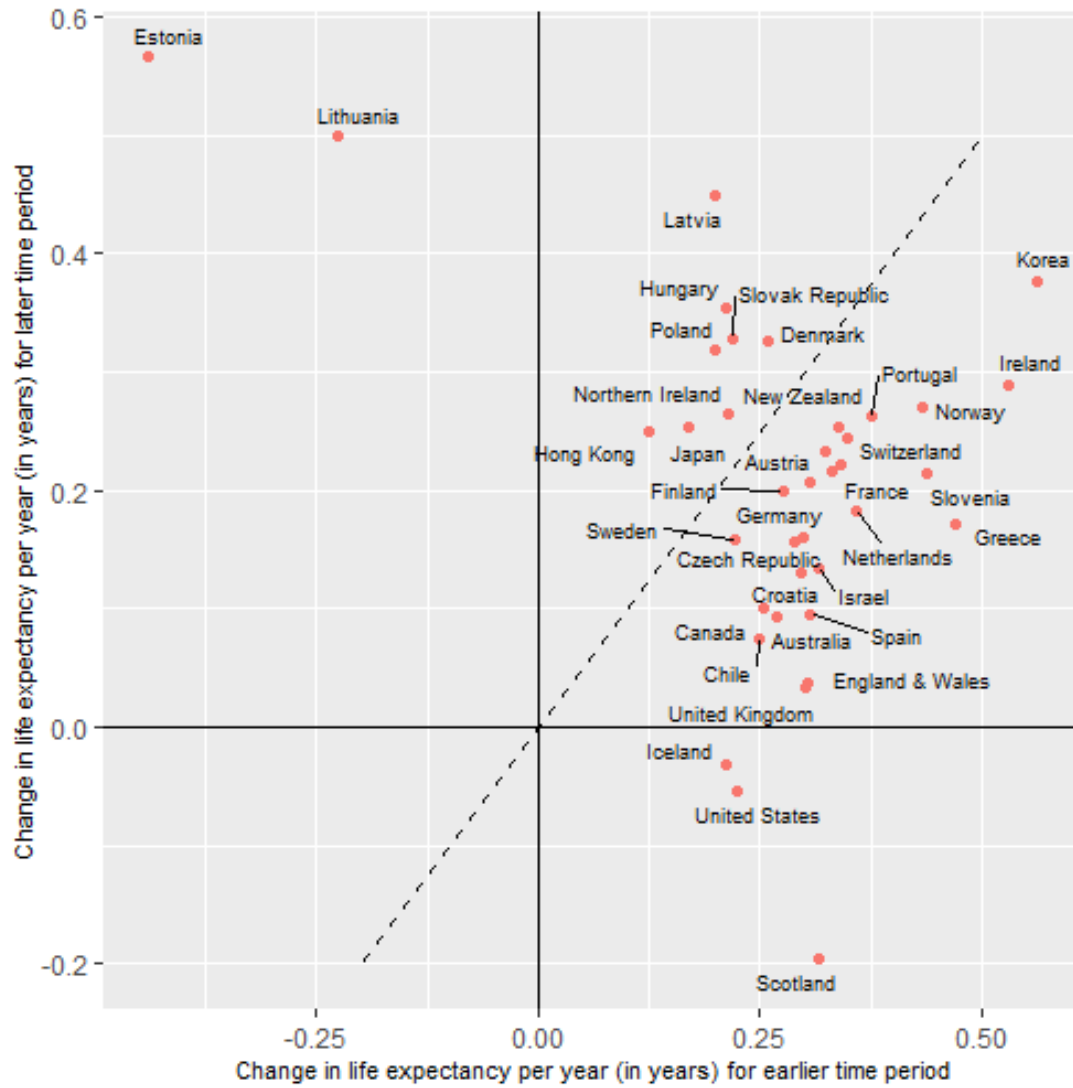


Figure S7.7 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, all ages, for the female population (1987-2018)

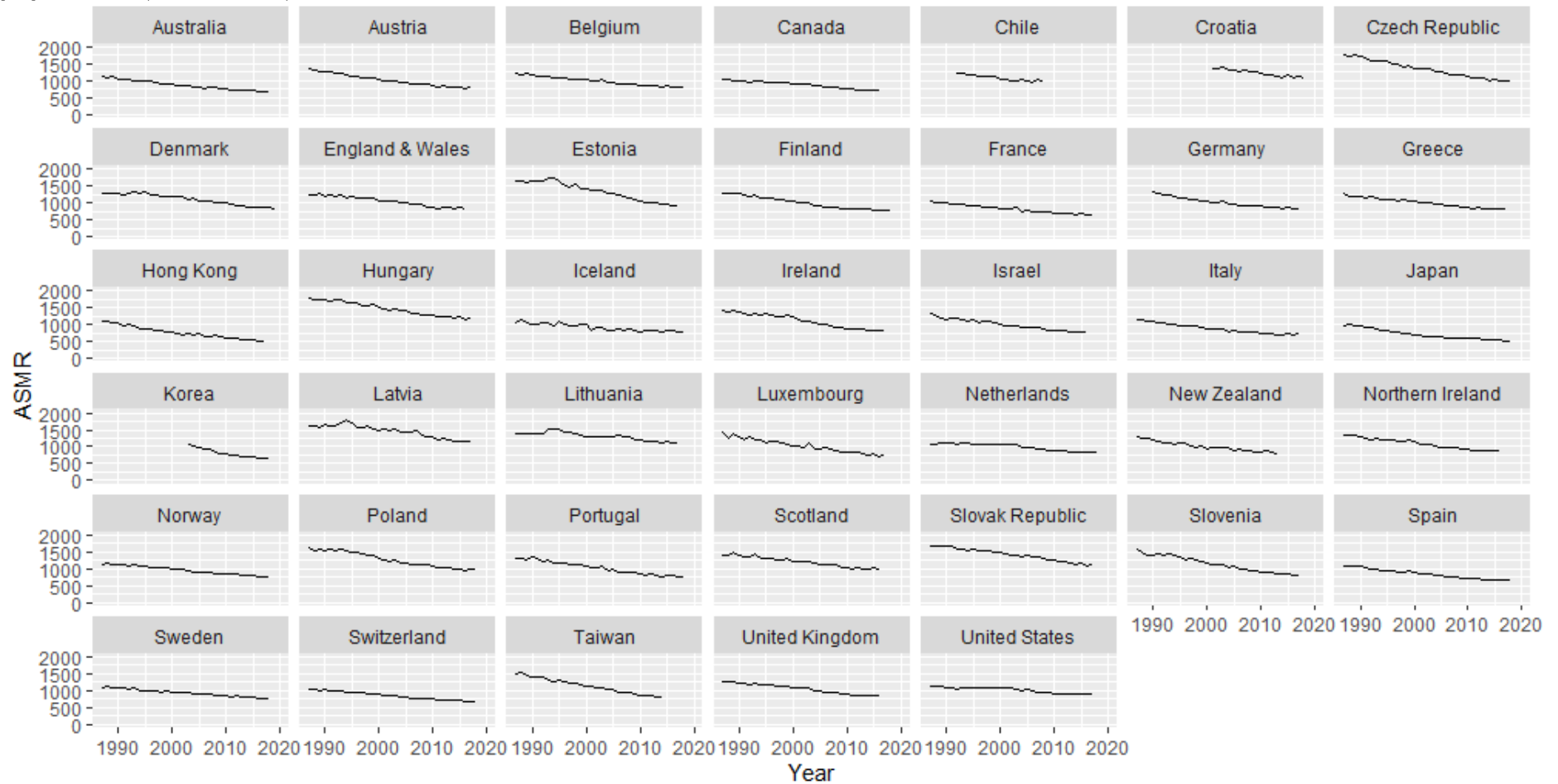


Figure S7.8 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, all ages, for the male population (1987-2018)

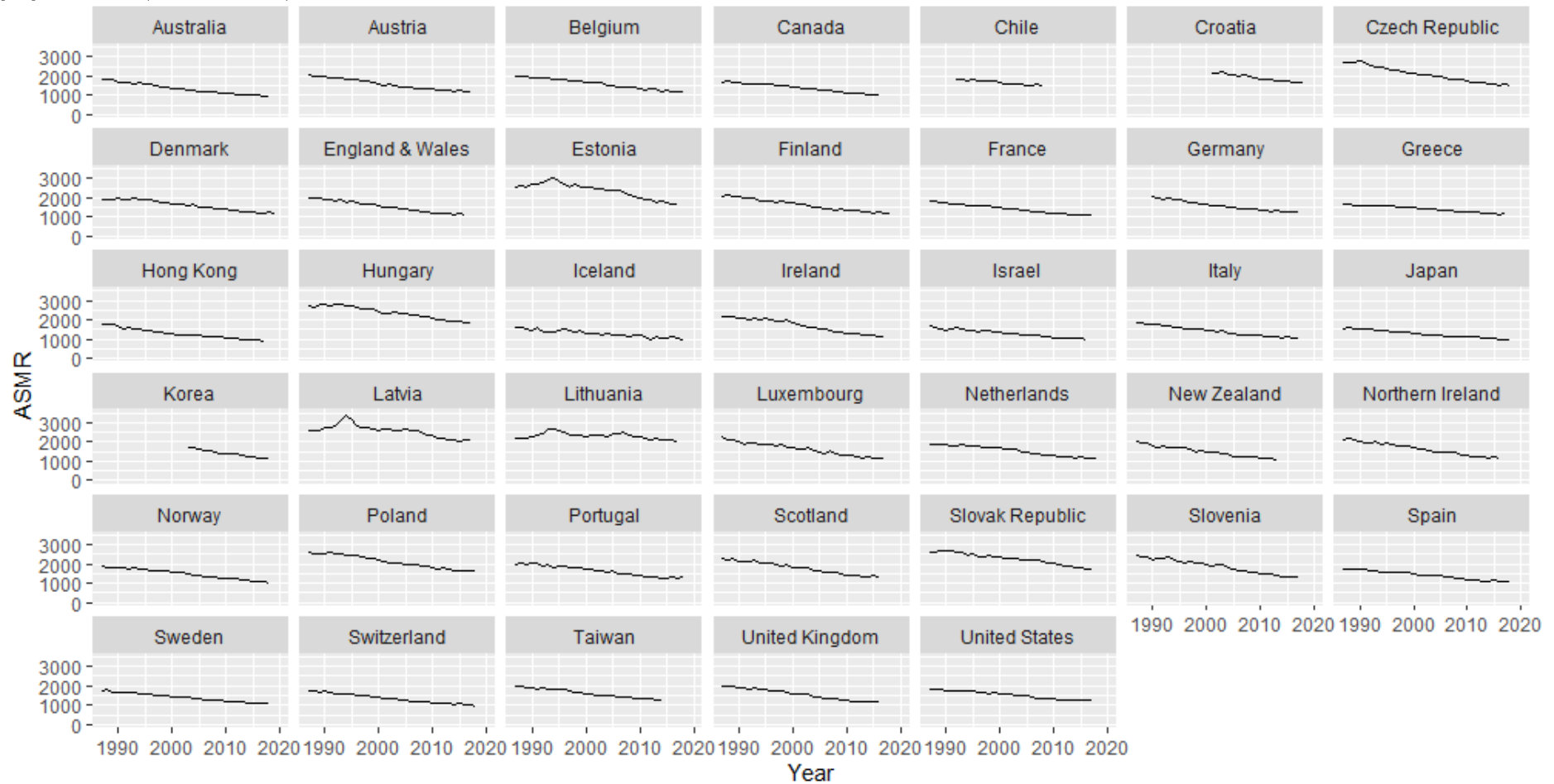


Table S7.3 - Estimated breakpoints in the ASMR trends, between 2000 and 2019, and the regression line slopes for the total population, females and males (all ages)

Country	Total population			Females			Males		
	Break	Slope 1*	Slope 2*	Break	Slope 1*	Slope 2*	Break	Slope 1*	Slope 2*
Australia	2005.0	-23	-14	2005.0	-17	-10	2005.0	-34	-21
Austria	2007.0	-25	-13	2007.2	-21	-10	2006.8	-33	-20
Belgium	2007.0	-25	-16	2010.2	-16	-10	2007.0	-38	-24
Canada	2011.8	-20	-10	2012.0	-15	-8	2011.6	-29	-15
Chile	NA	-13	NA	NA	-10	NA	NA	-18	NA
Croatia	2013.5	-26	-10	2013.0	-22	-9	2013.4	-36	-13
Czech Rep.	2010.9	-33	-19	2011.0	-28	-16	2010.5	-42	-24
Denmark	2014.4	-26	-10	2014.4	-21	-10	2014.3	-34	-11
E&W	2011.0	-27	-4	2011.0	-21	-3	2011.0	-38	-7
Estonia	2013.0	-46	-23	2012.0	-37	-18	2003.2	-20	-61
Finland	2005.7	-35	-14	2006.0	-31	-10	2005.2	-45	-24
France	2007.1	-25	-12	2007.0	-19	-8	2010.7	-31	-15
Germany	2006.4	-22	-11	2006.1	-17	-9	2006.5	-36	-18
Greece	2013.3	-20	2	2013.2	-18	6	2013.6	-24	-4
Hong Kong	2004.7	-11	-20	2004.8	-11	-16	2004.7	-13	-25
Hungary	NA	-26	NA	NA	-19	NA	2005.0	-23	-40
Iceland	2014.0	-12	-3	2010.8	-10	-3	2016.0	-15	-29
Ireland	2008.4	-44	-15	2008.7	-34	-9	2007.8	-60	-25
Israel	2009.8	-22	-11	2010.0	-17	-12	2009.5	-28	-12
Italy	2006.0	-25	-11	2006.0	-19	-8	2006.2	-33	-18
Japan	2001.5	-29	-11	2002.0	-23	-8	2011.1	-15	-20
Korea	2009.0	-48	-25	2009.1	-44	-20	2009.0	-54	-37
Latvia	2005.0	-9	-38	2003.1	1	-28	2005.0	-5	-53
Lithuania	2006.0	12	-30	2006.0	0	-21	2006.3	24	-42
Luxembourg	2015.8	-26	39	NA	-19	NA	2013.0	-41	-9
Netherlands	2009.7	-30	-8	2009.6	-23	-4	2010.5	-42	-14
New Zealand	NA	-19	NA	NA	-13	NA	2005.0	-41	-26
N. Ireland	2011.0	-26	-7	2011.0	-20	-2	2011.2	-38	-15
Norway	2004.6	-32	-17	2004.9	-24	-12	2004.4	-45	-26
Poland	2016.0	-26	13	2016.0	-22	14	2016.0	-34	7
Portugal	2011.0	-29	-9	2011.0	-24	-7	2011.0	-36	-11
Scotland	2011.3	-27	-6	2011.0	-20	-6	2011.8	-40	-5
Slovak Rep.	2006.9	-22	-33	2007.0	-18	-26	2006.5	-25	-46
Slovenia	2010.5	-34	-16	2010.1	-26	-13	2010.3	-52	-26
Spain	2013.0	-21	-6	2013.0	-16	-4	2013.3	-28	-9
Sweden	2009.6	-18	-11	2009.7	-13	-8	2010.6	-25	-16
Switzerland	2006.3	-23	-13	2006.2	-17	-9	2006.7	-32	-19
Taiwan	2009.2	-29	-13	2009.0	-28	-13	2009.3	-29	-10
UK	2011.0	-27	-4	2011.0	-21	-3	2011.1	-38	-7
USA	2010.1	-22	-5	2010.0	-18	-6	2009.8	-31	-9

*The slopes refer to the rate of change in ASMR (per year) for the period before (Slope 1) and after (Slope 2) the breakpoint.

Figure S7.9 - Segmented regression fitted to ASMR trends for females (2000-2019, vertical lines indicate breakpoint)

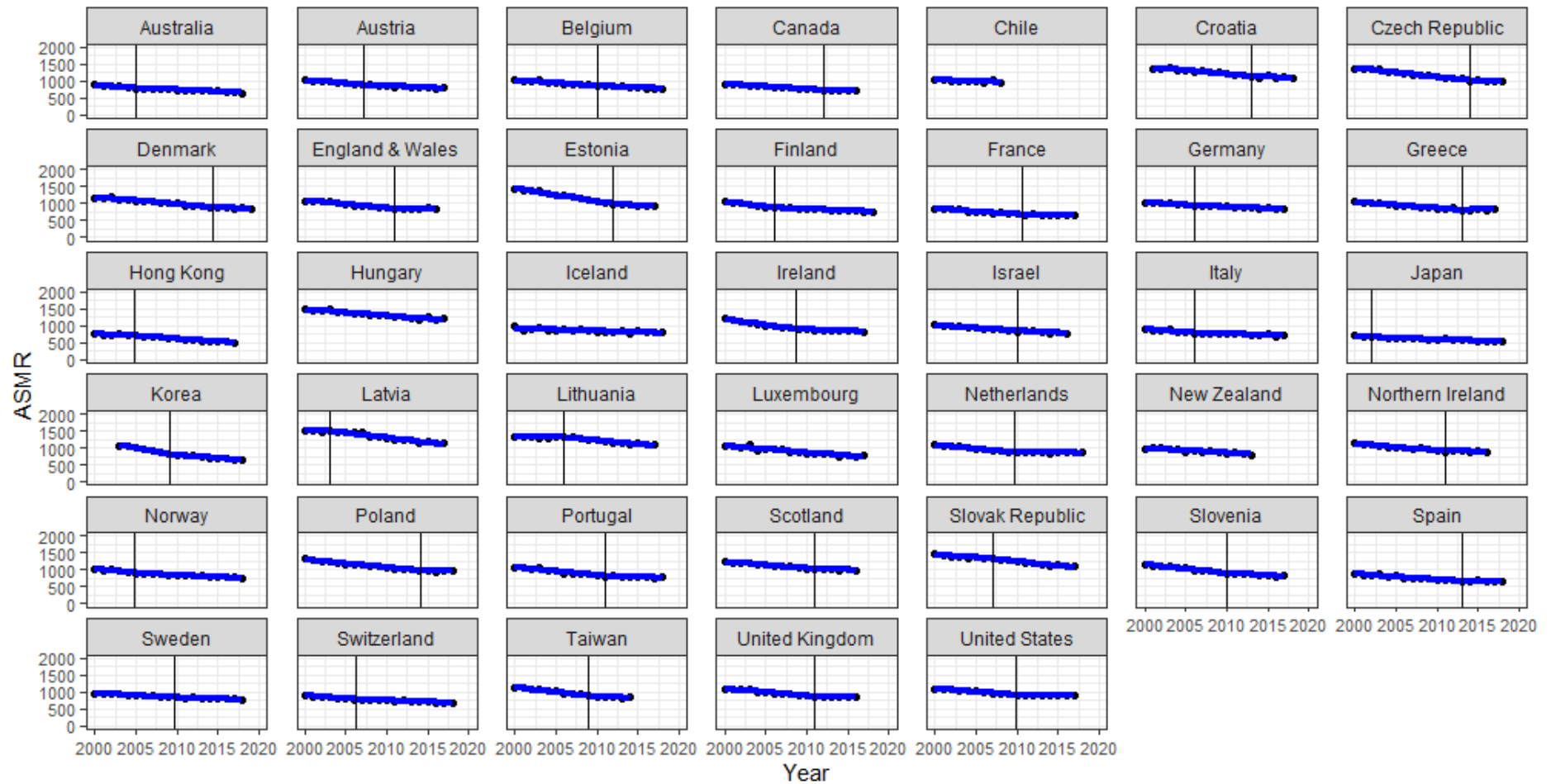


Figure S7.10 - Segmented regression fitted to ASMR trends for males (2000-2019, vertical lines indicate breakpoint)

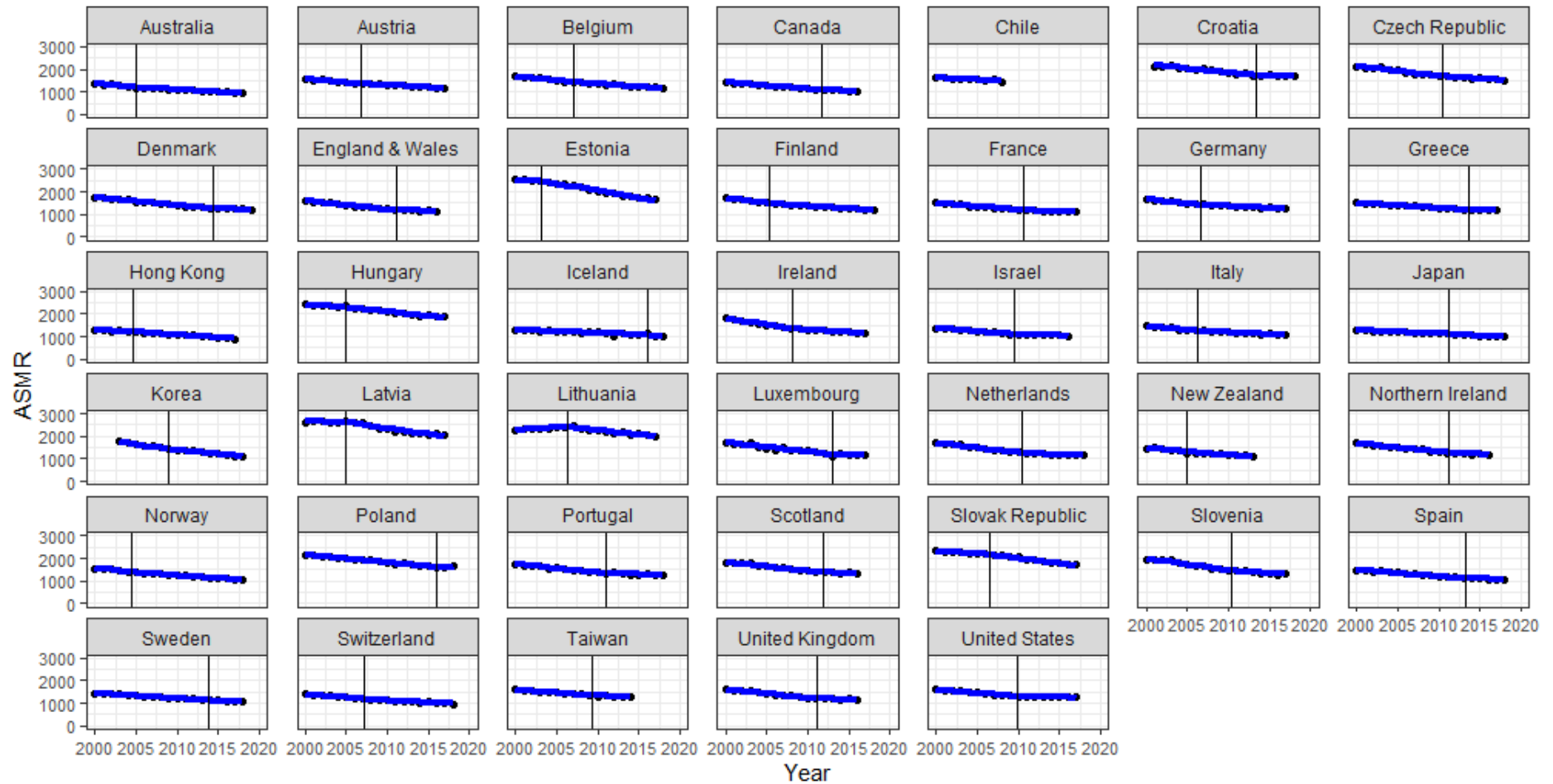


Figure S7.11 - Scatterplot of the rate of change in ASMR for females at all ages before and after the breakpoint (2000-2019)

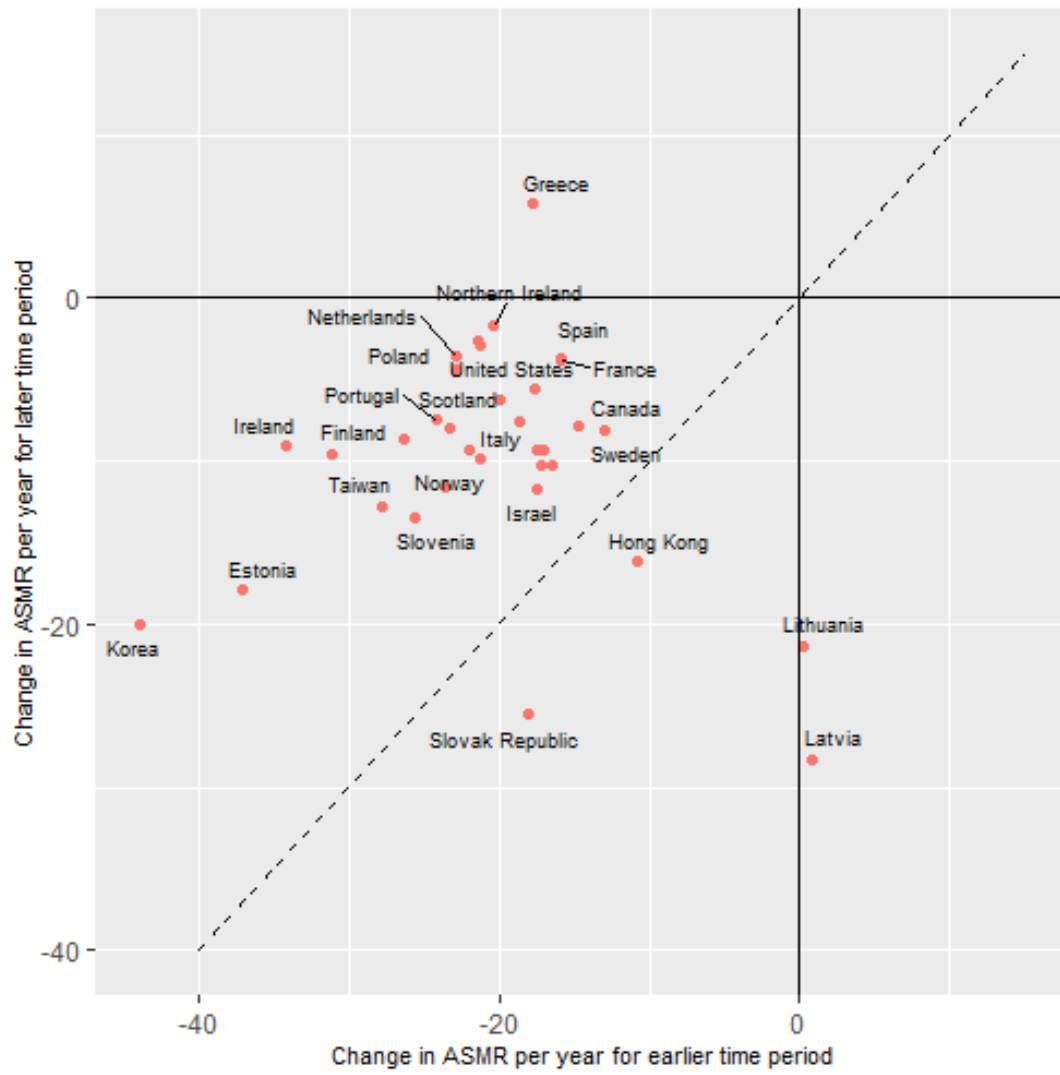


Figure S7.12 - Scatterplot of the rate of change in ASMR for males at all ages before and after the breakpoint (2000-2019)

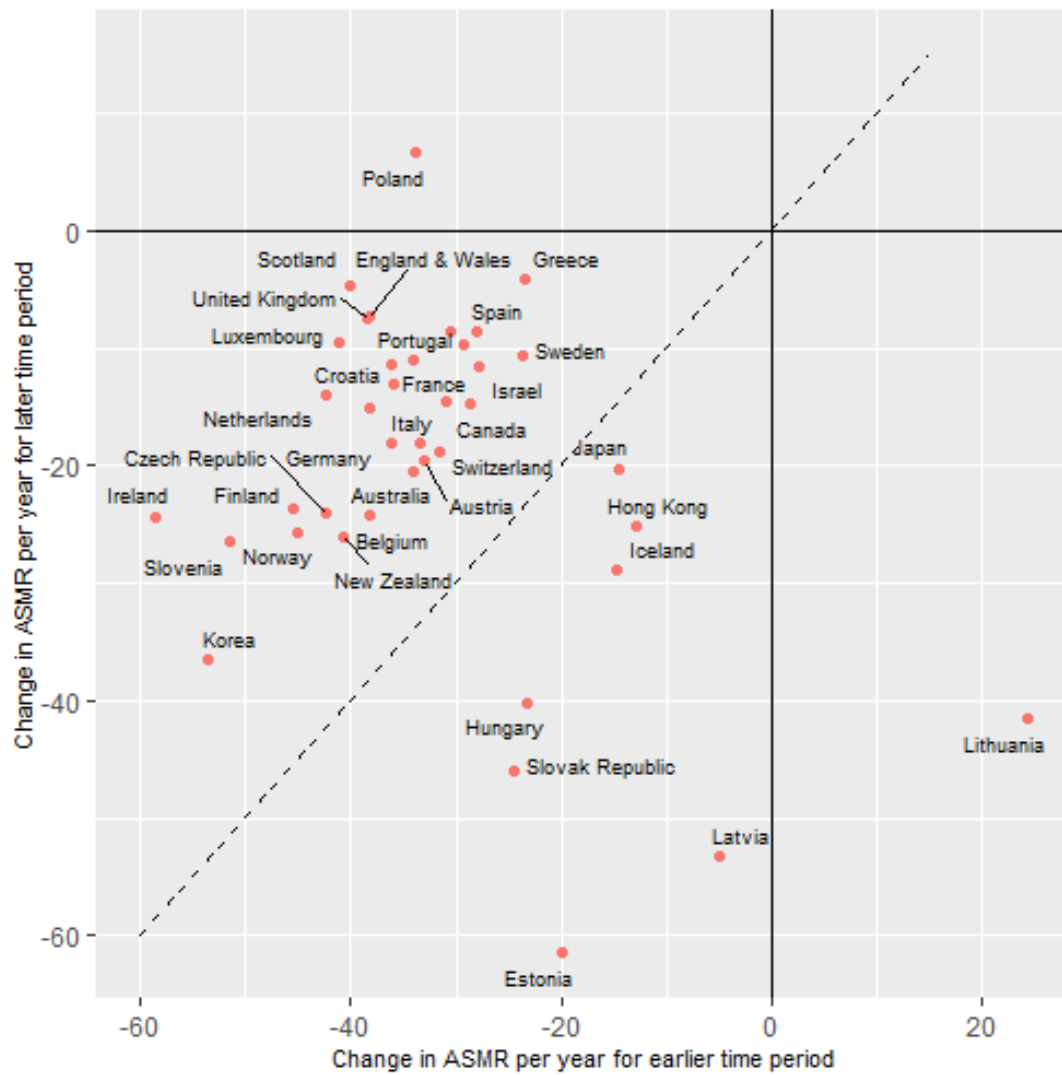


Figure S7.13 - Trend in Infant Mortality Rate (ASMR) per 100,000 population per year, for the total population (1987-2018)

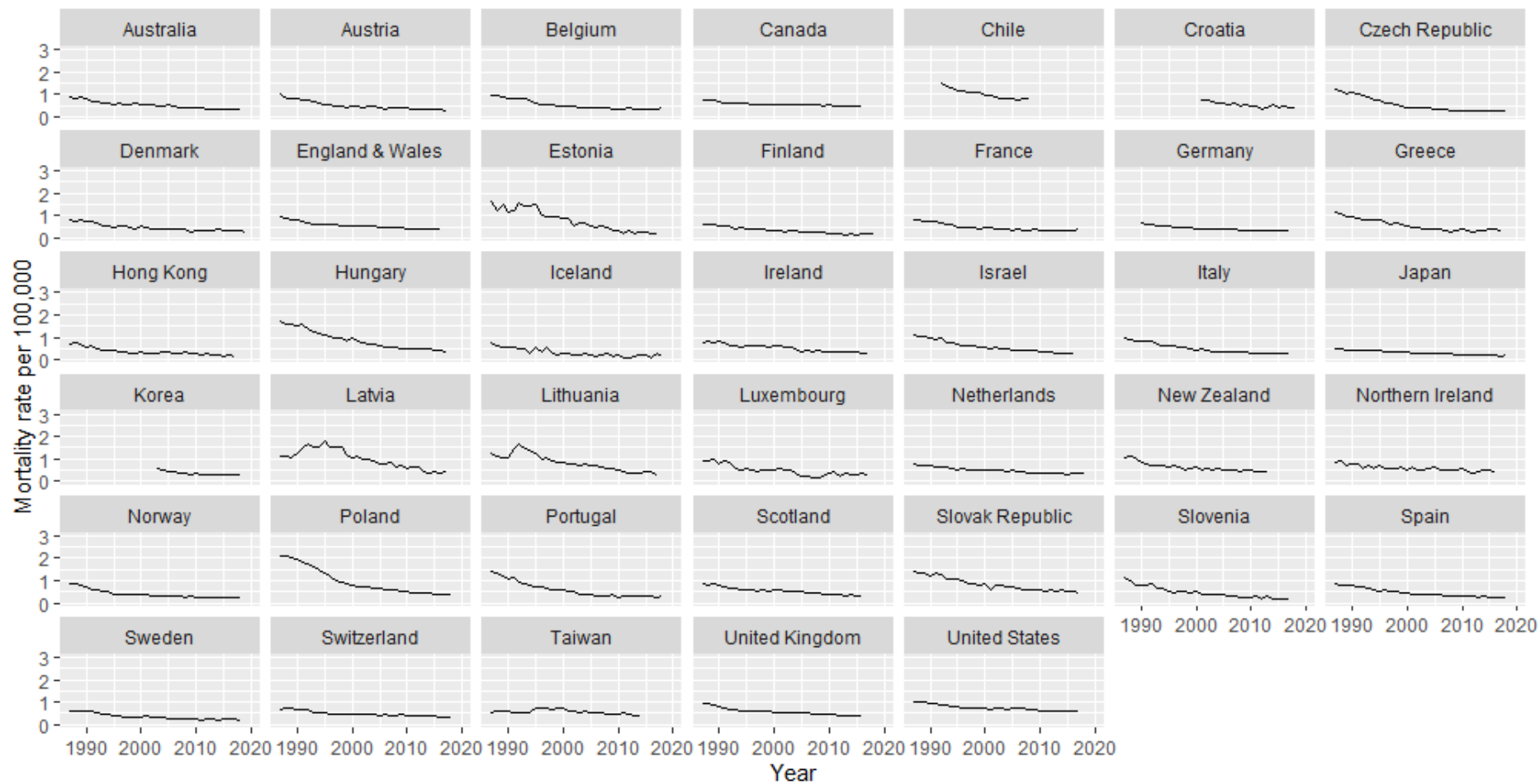


Figure S7.14 - Trend in Infant Mortality Rate (ASMR) per 100,000 population per year, for the female population (1987-2018)

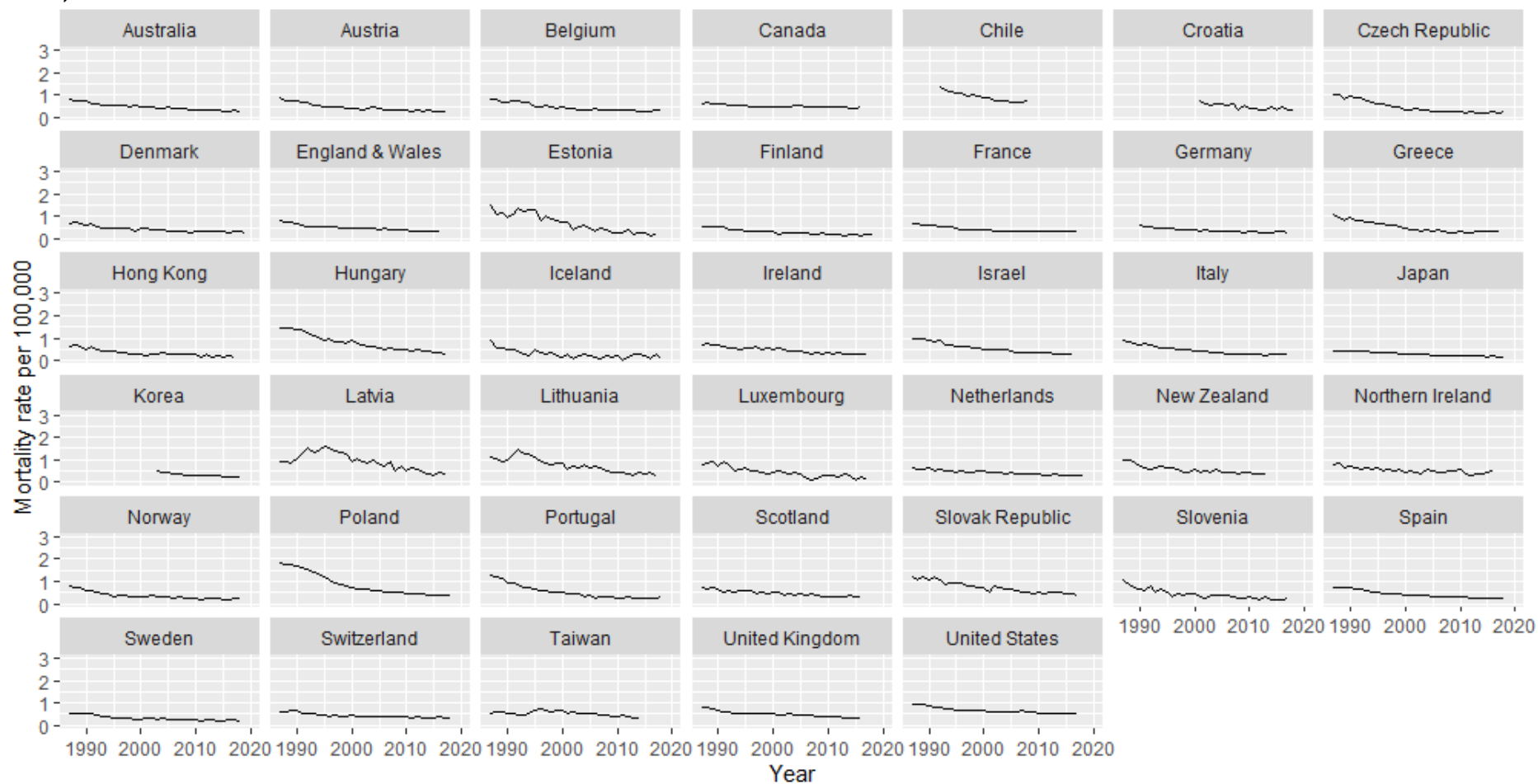


Figure S7.15 - Trend in Infant Mortality Rate (ASMR) per 100,000 population per year, for the male population (1987-2018)

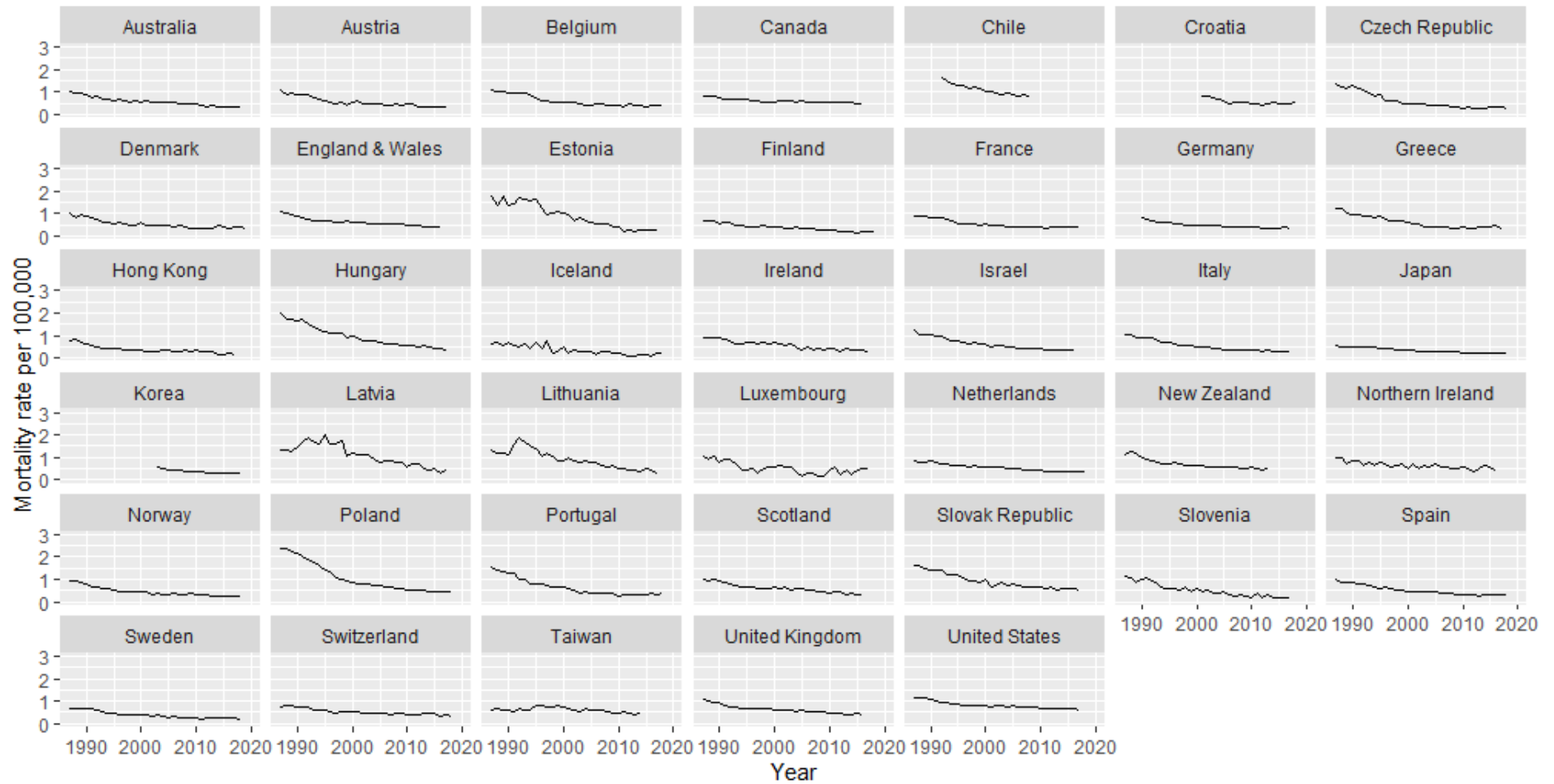


Figure S7.16 - Segmented regression fitted to the IMR trends for the total population (2000-2019, vertical lines indicate breakpoint)

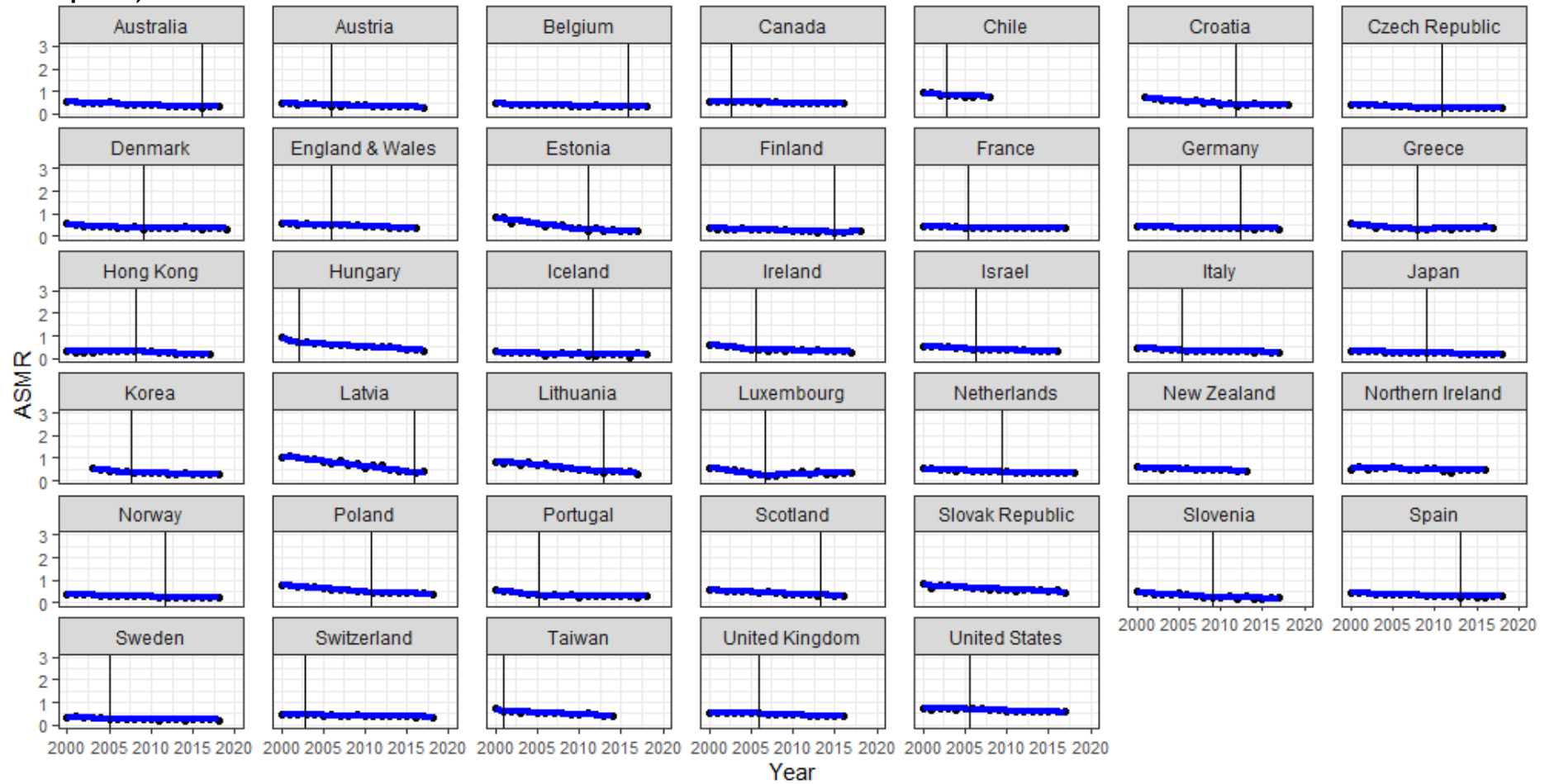


Figure S7.17 - Scatterplot of the rate of change in IMRs for the total population (males and females combined) before and after the breakpoint (2000-2019)

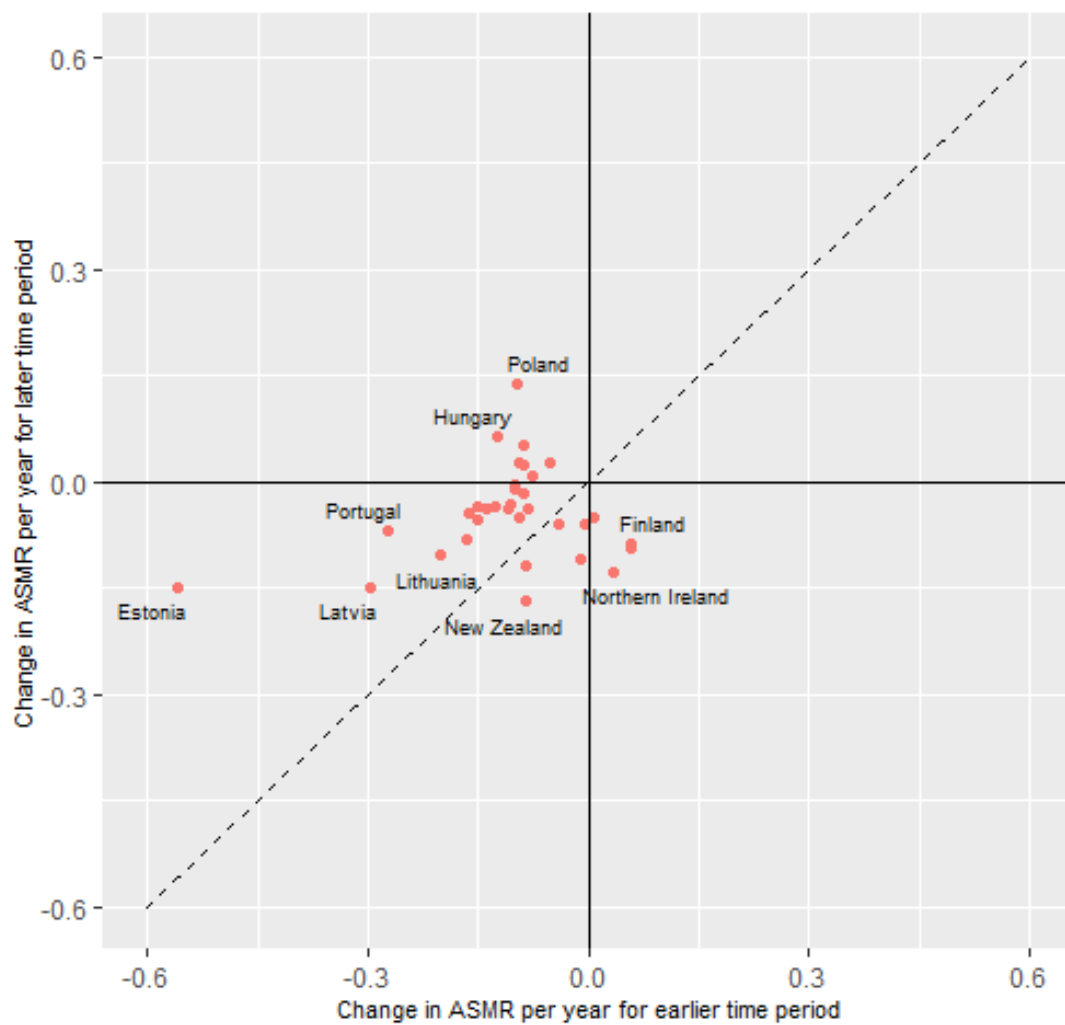


Figure S7.18 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the total population aged 1-14 years (1987-2018)

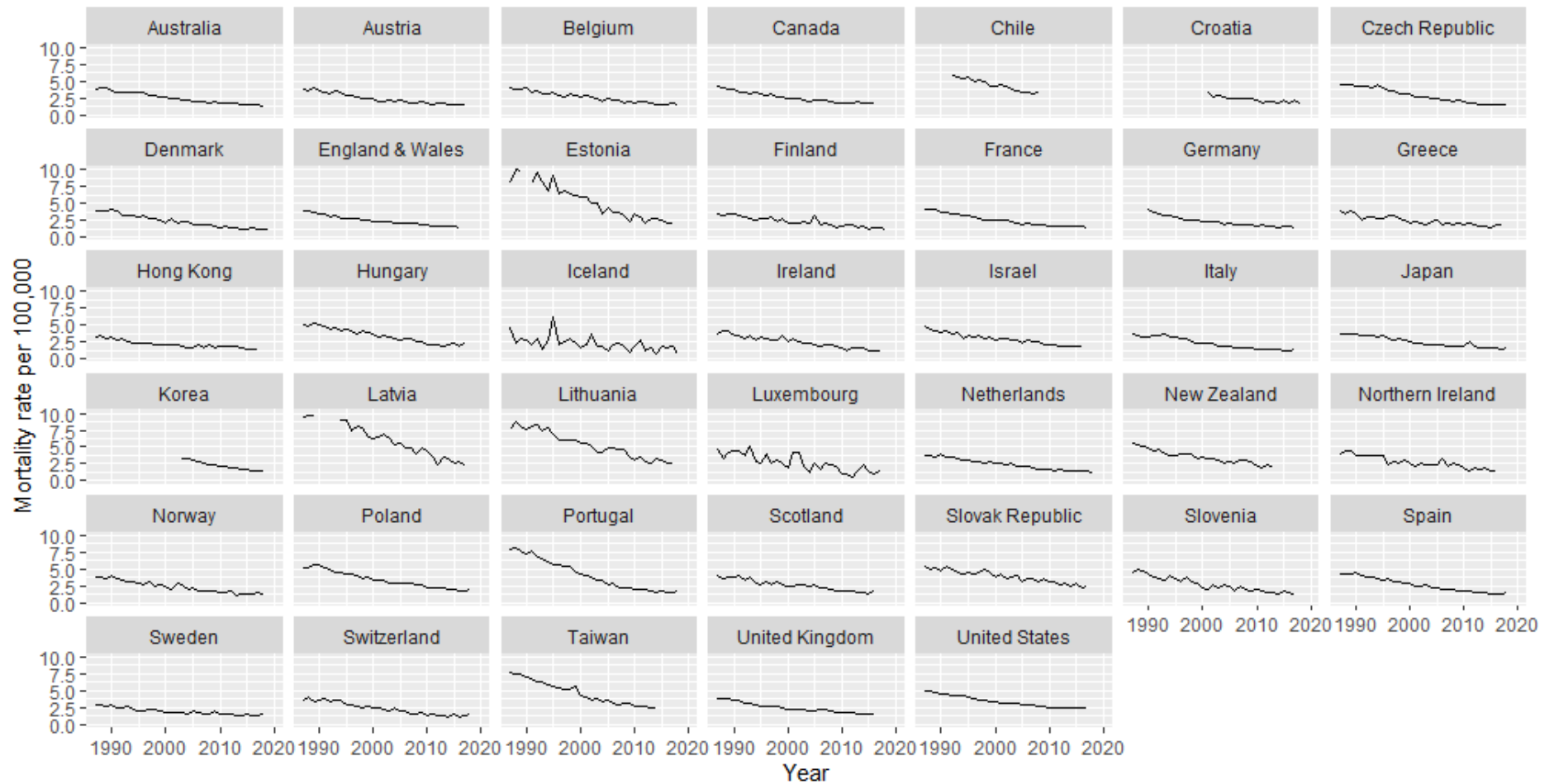


Figure S7.19 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the female population aged 1-14 years (1987-2018)

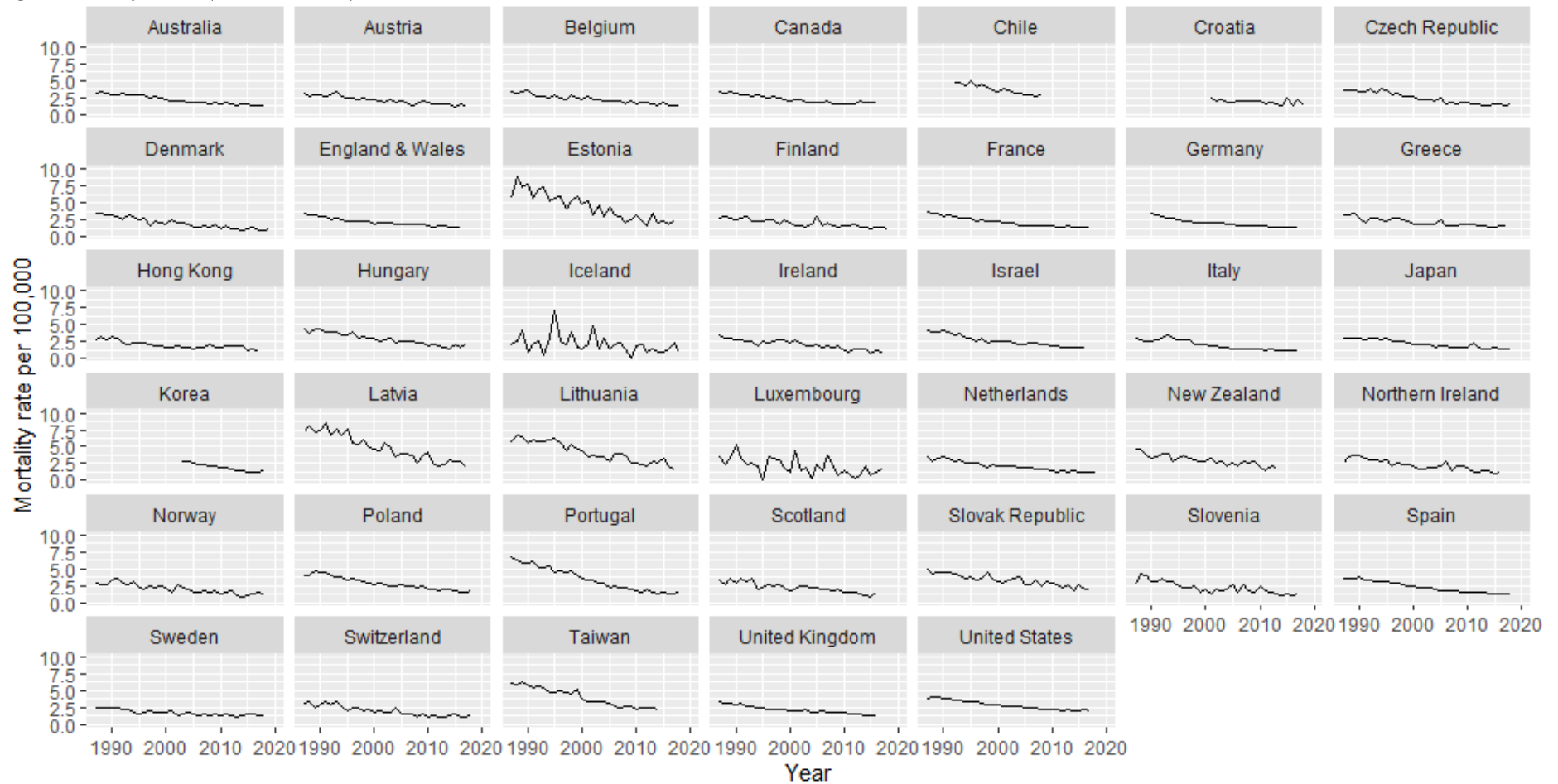


Figure S7.20 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the male population aged 1-14 years (1987-2018)

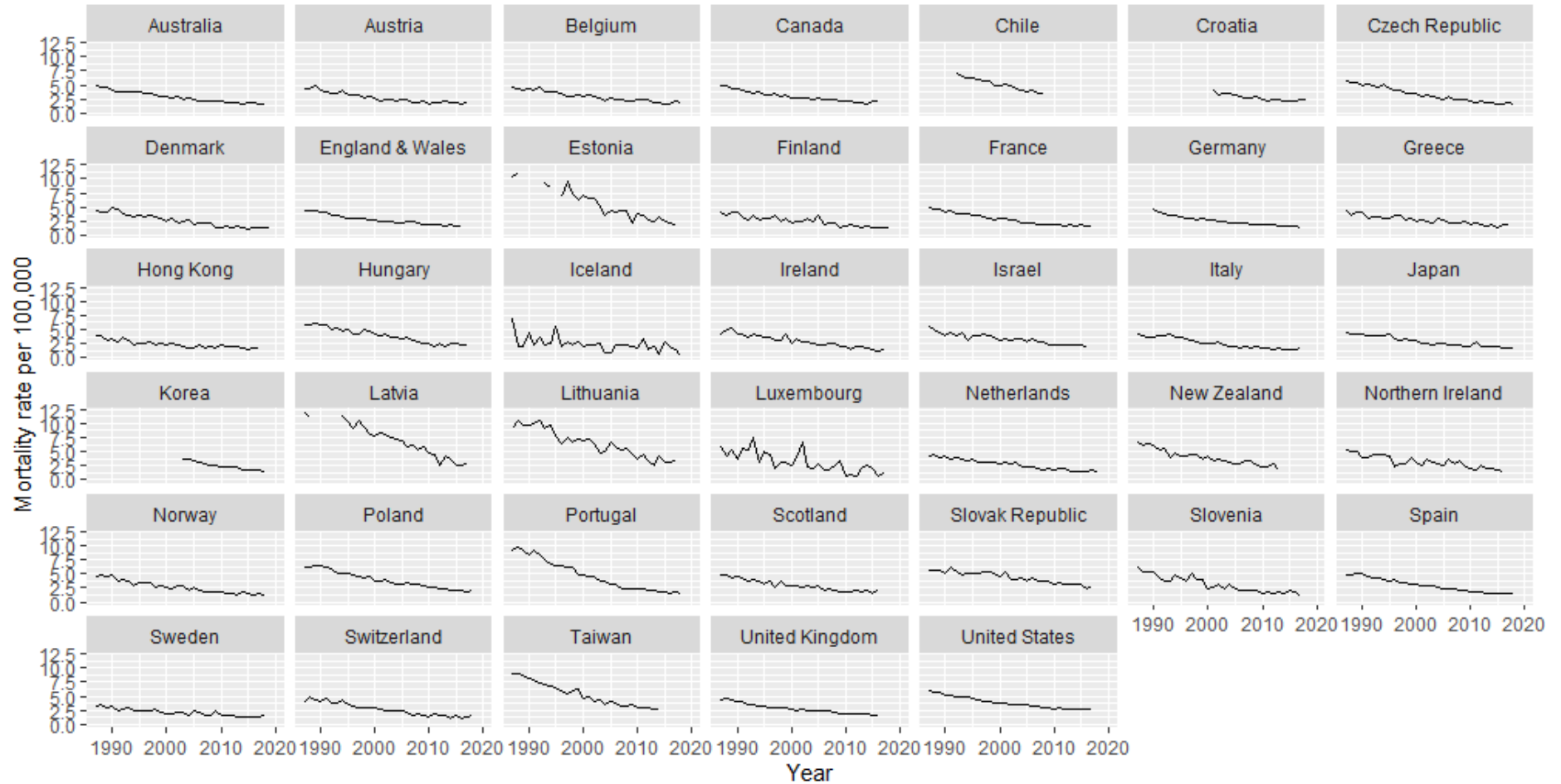


Figure S7.21 - Segmented regression fitted to the Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the total population aged 1-14 years (2000-2019, vertical lines indicate breakpoint)

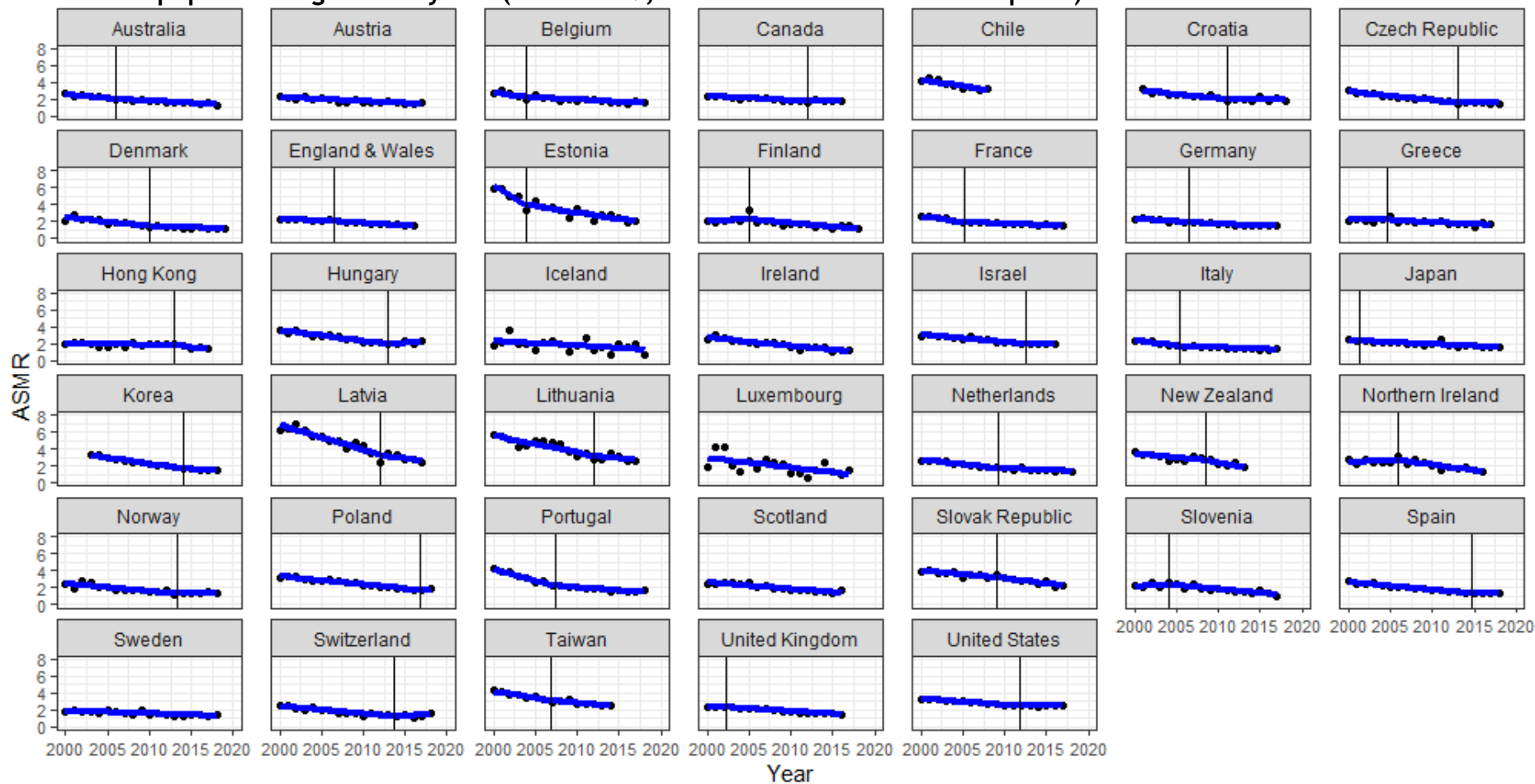


Figure S7.22 - Scatterplot of the rate of change in ASMRs for the total population (males and females combined) aged 1-14 years, before and after the breakpoint (2000-2019)

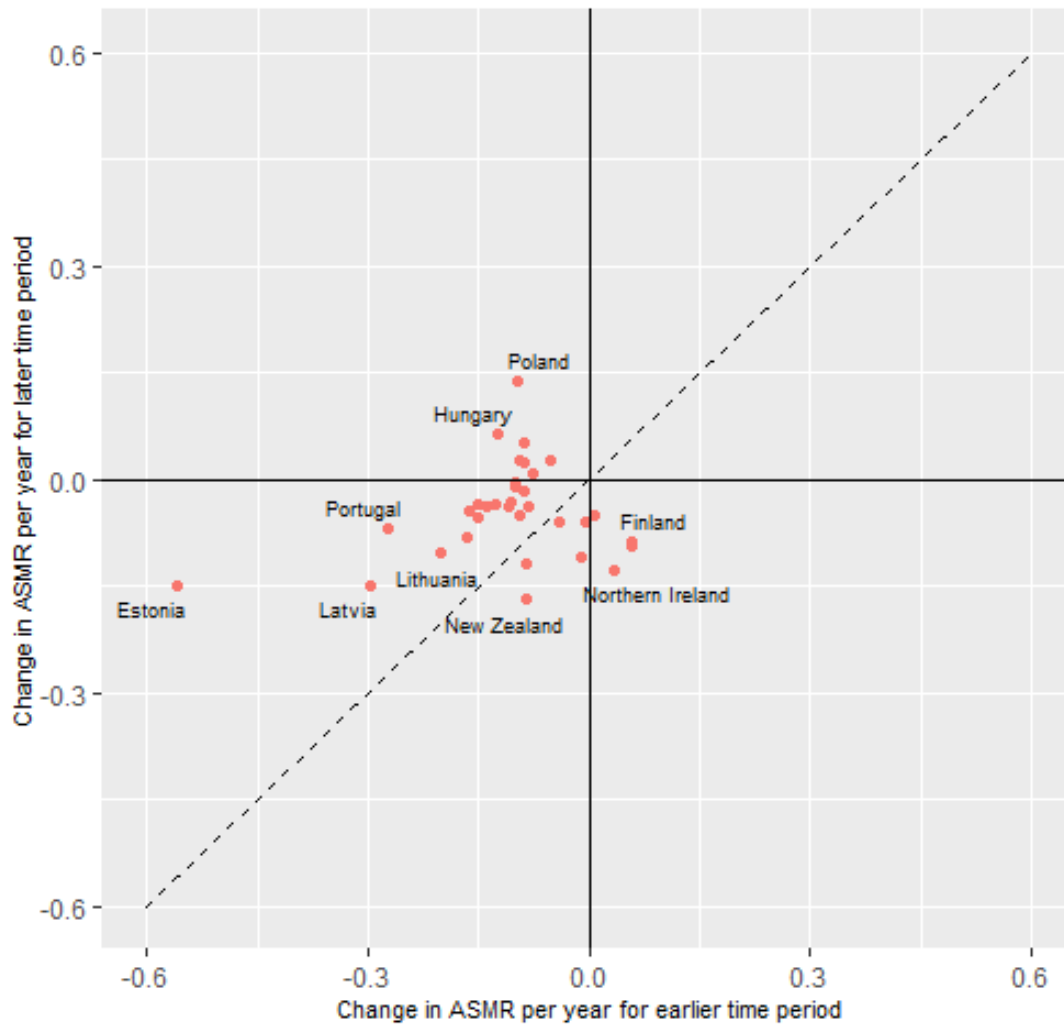


Figure S7.23 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the total population aged 15-29 years (1987-2018)

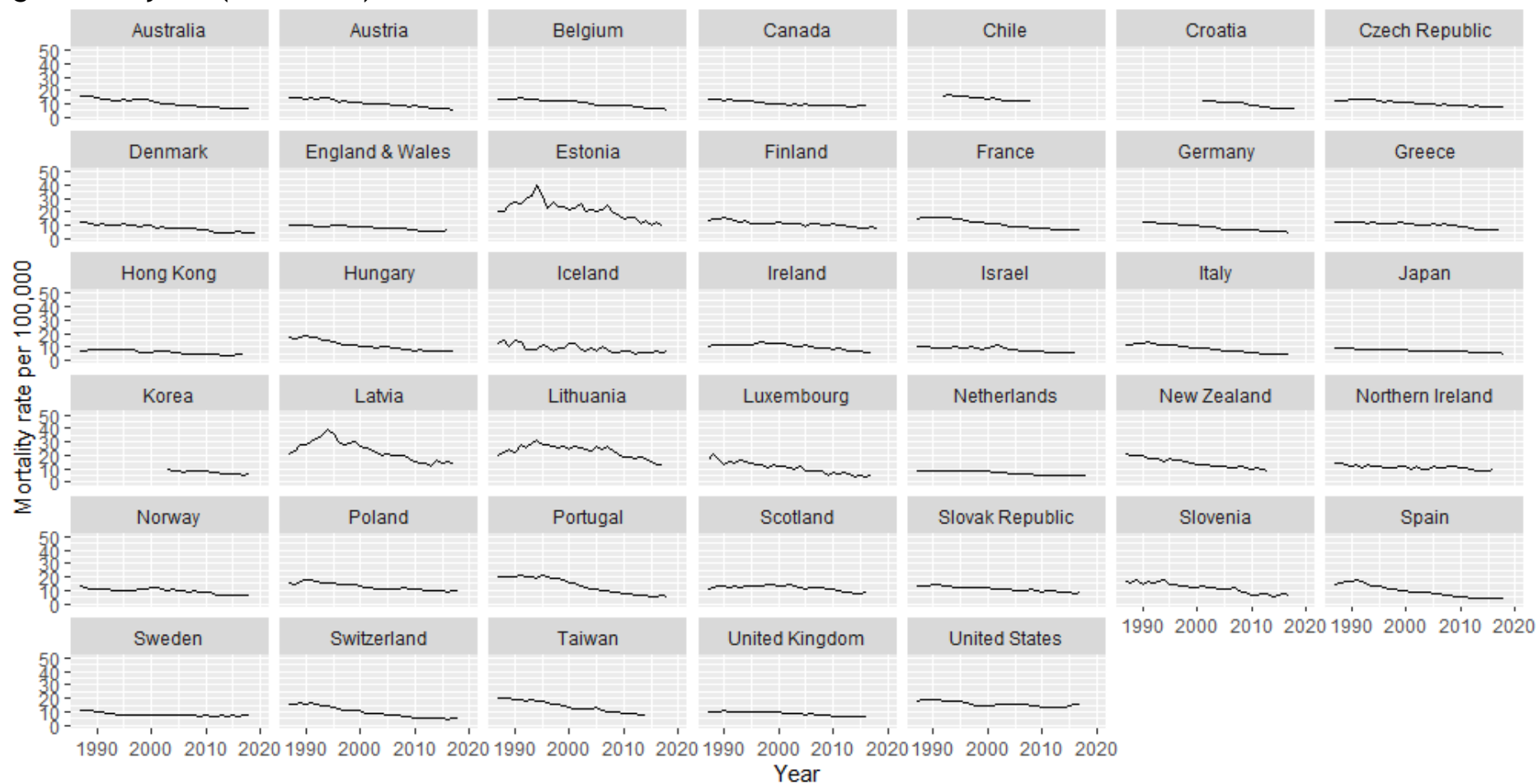


Figure S7.24 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the female population aged 15-29 years (1987-2018)

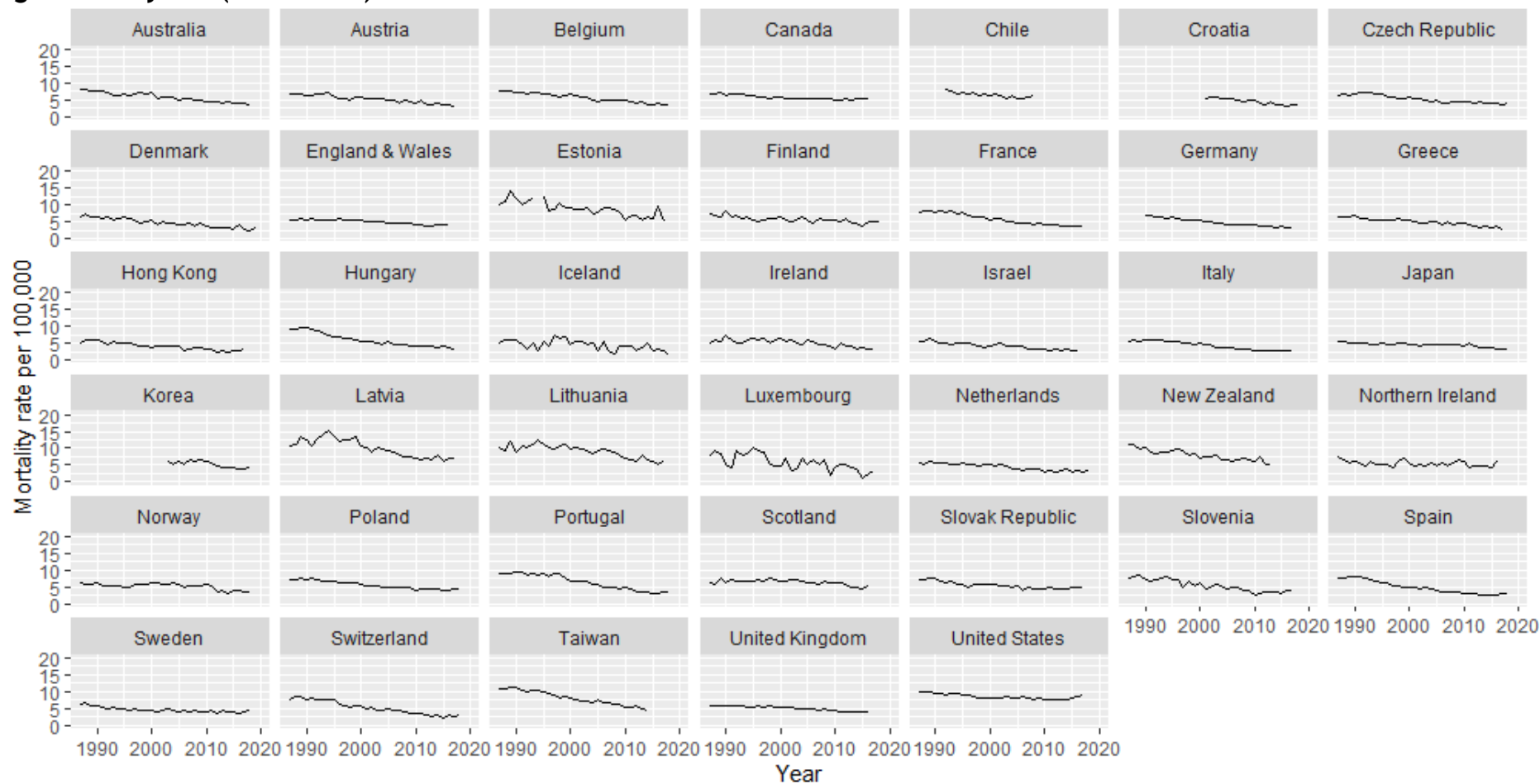


Figure S7.25 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the male population aged 15-29 years (1987-2018)

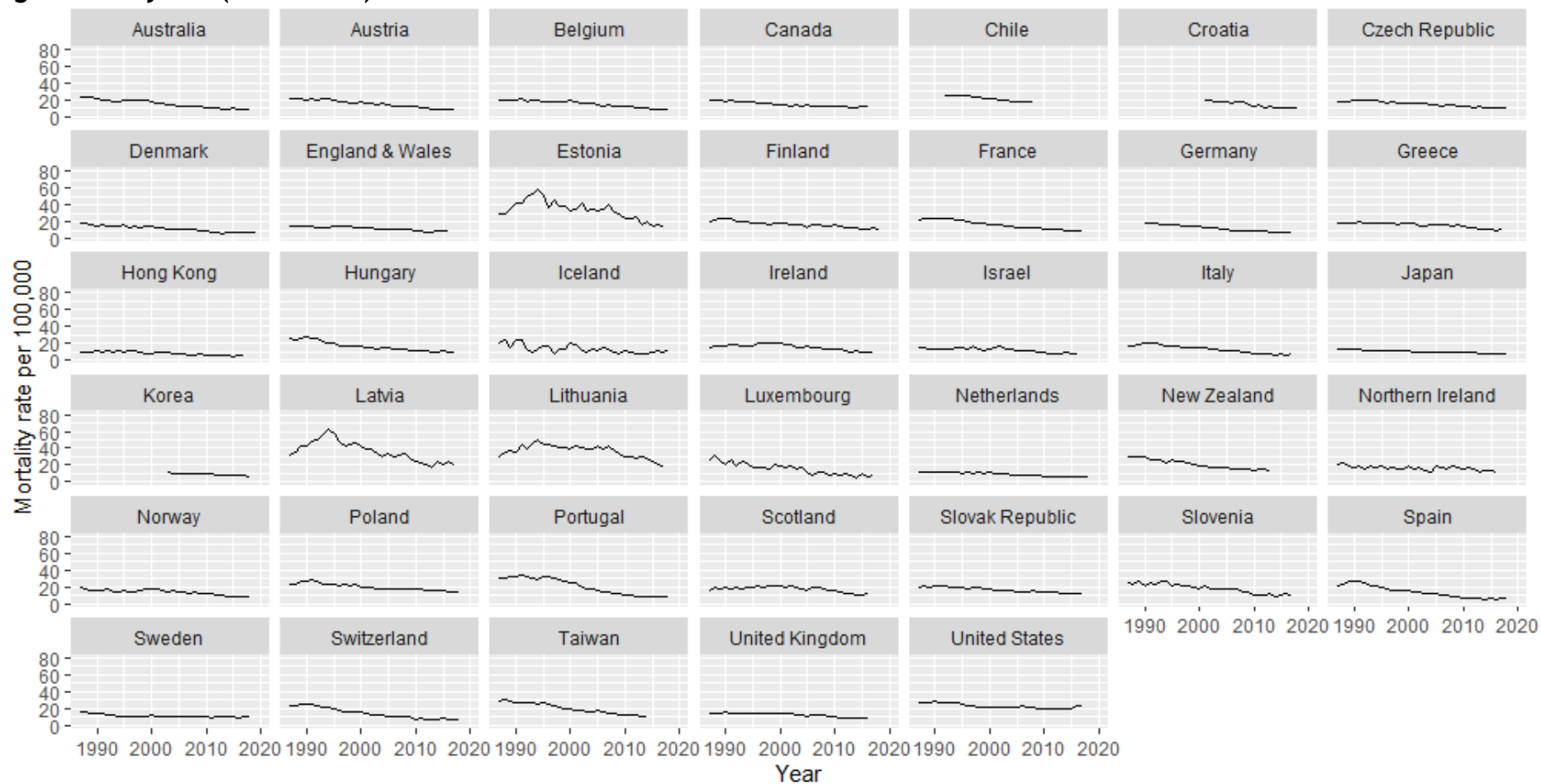


Figure S7.26 - Segmented regression fitted to the Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the total population aged 15-29 years (2000-2019, vertical lines indicate breakpoint)

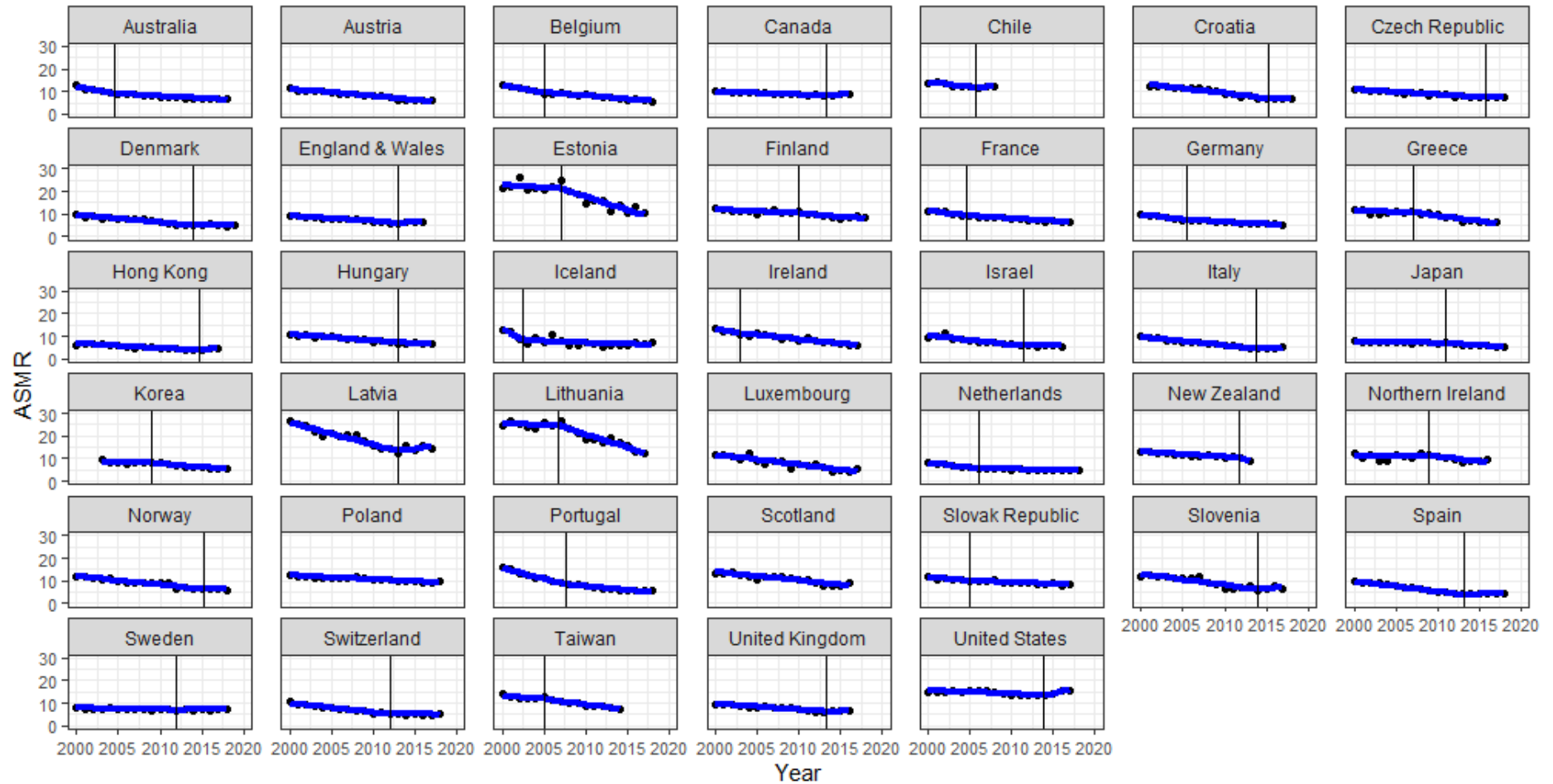


Figure S7.27 - Scatterplot of the rate of change in ASMRs for the total population (males and females combined) aged 15-29 years, before and after the breakpoint (2000-2019)

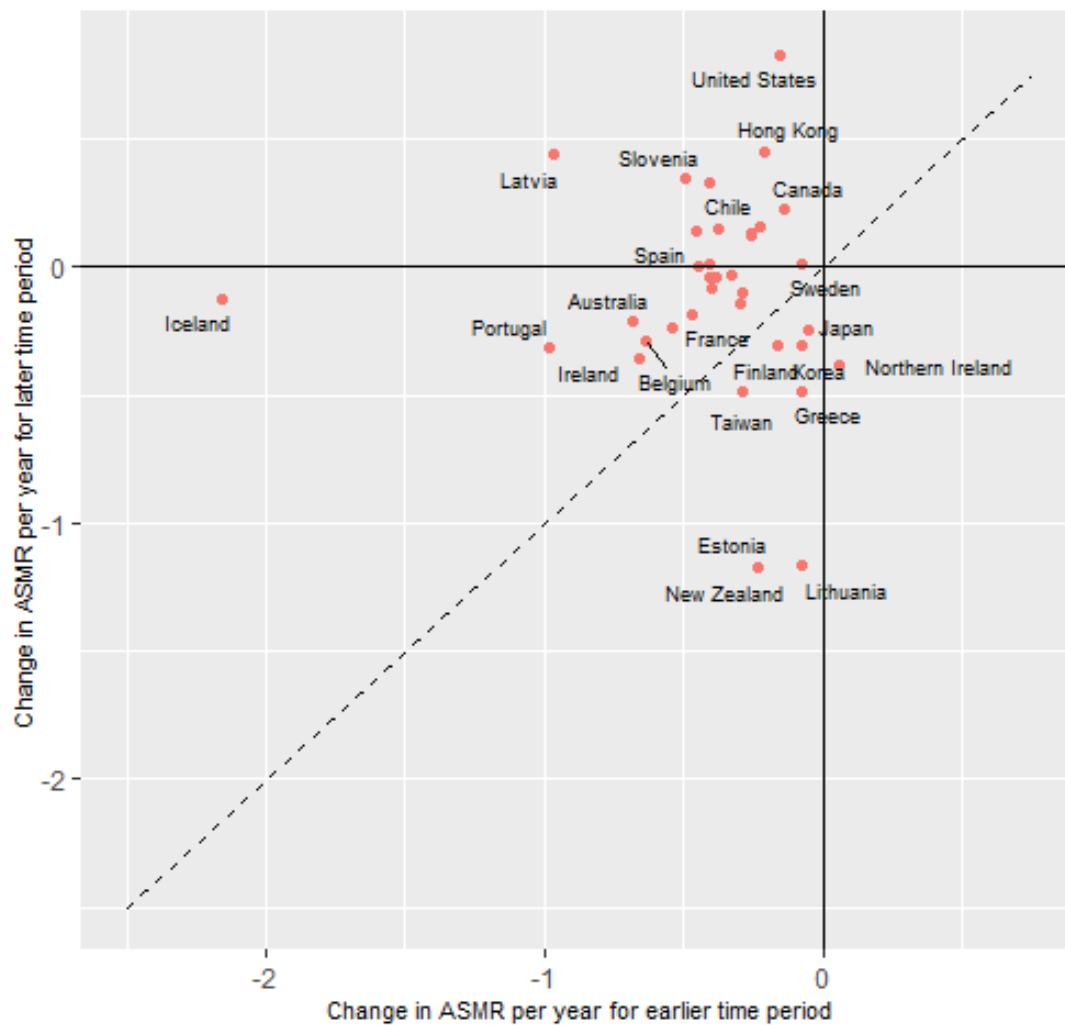


Figure S7.28 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the total population aged 30-49 years (1987-2018)

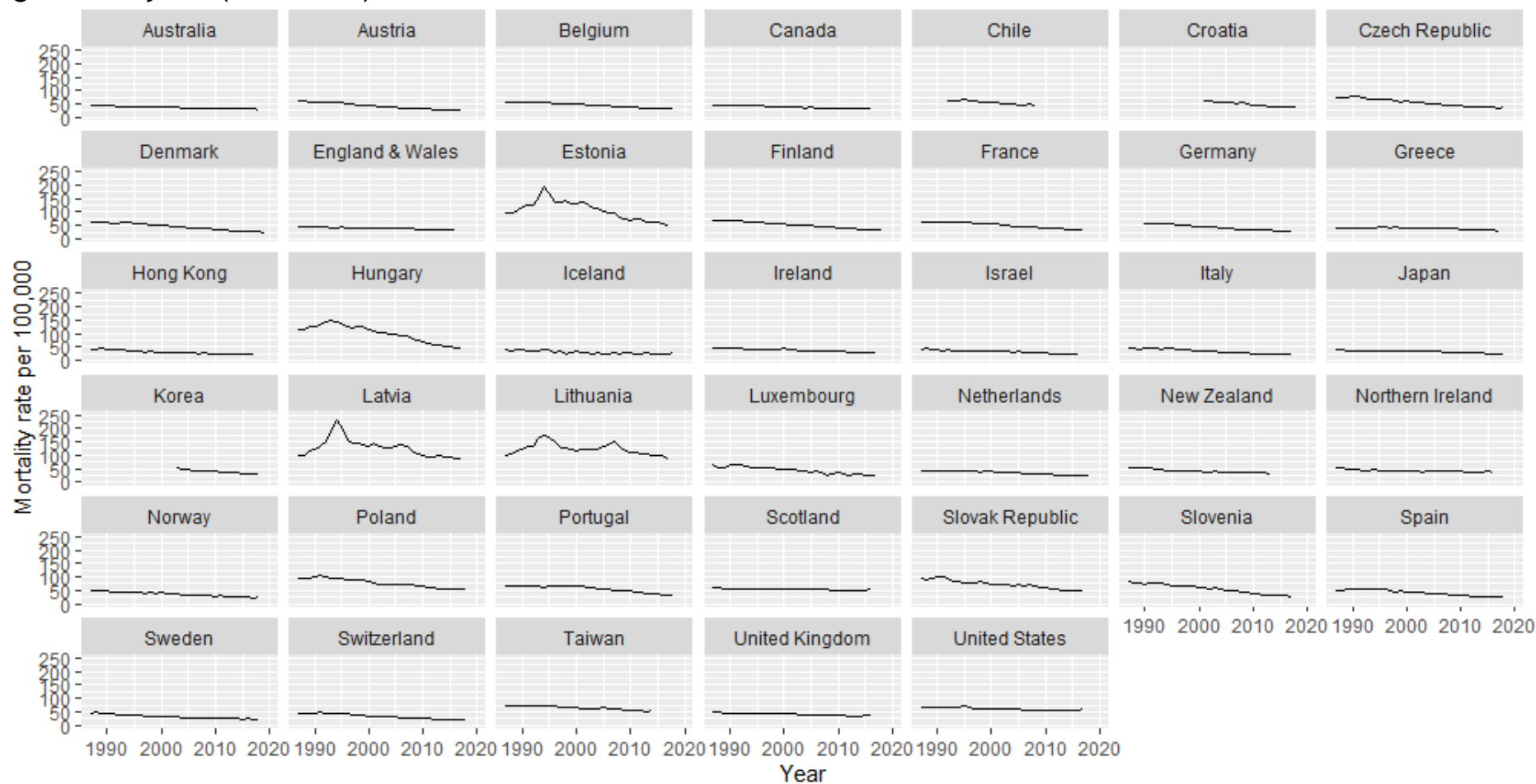


Figure S7.29 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the female population aged 30-49 years (1987-2018)

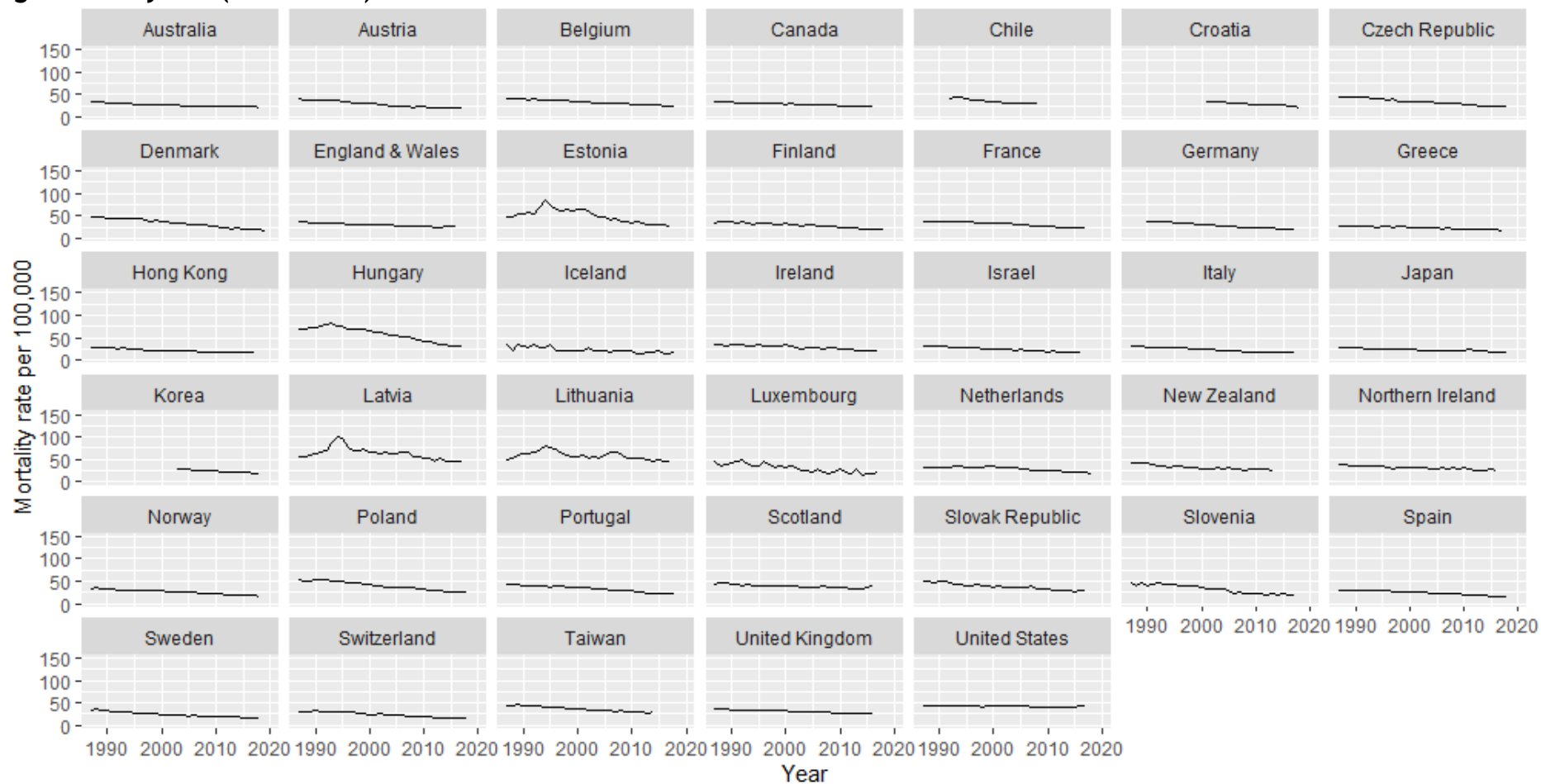


Figure S7.30 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the male population aged 30-49 years (1987-2018)

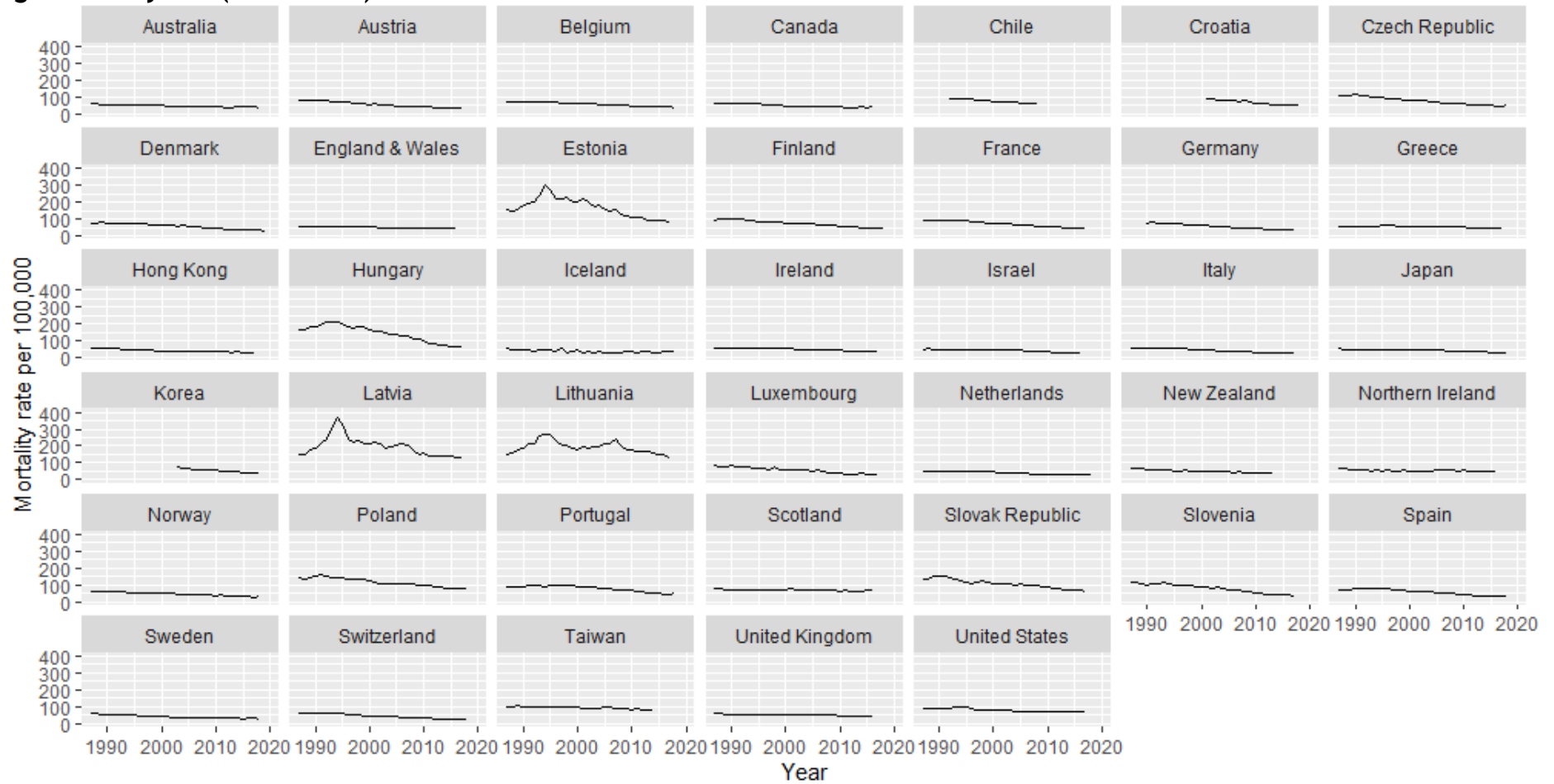


Figure S7.31 - Segmented regression fitted to the Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the total population aged 30-49 years (2000-2019, vertical lines indicate breakpoint)

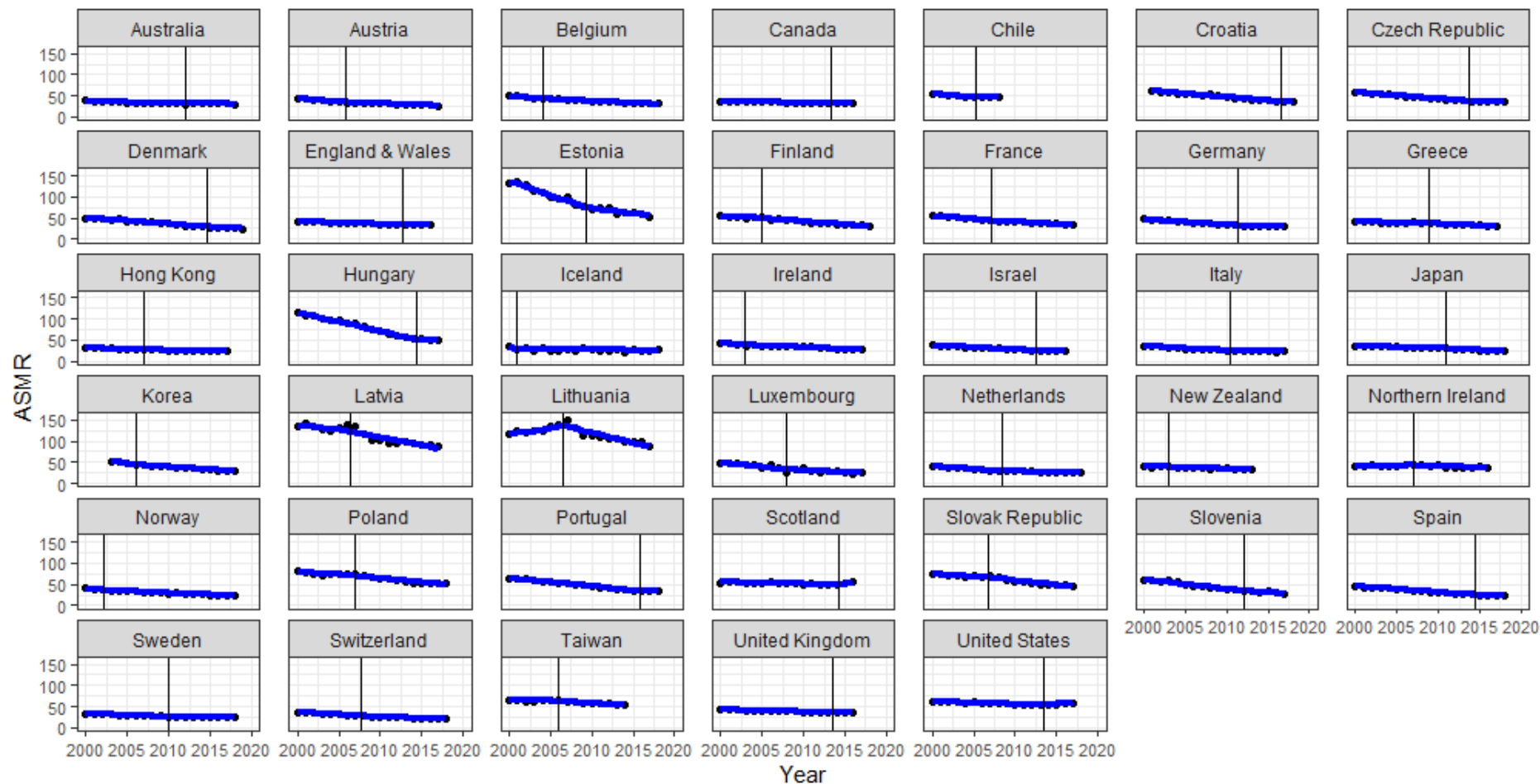


Figure S7.32 - Scatterplot of the rate of change in ASMRs for the total population (males and females combined) aged 30-49 years, before and after the breakpoint (2000-2019)

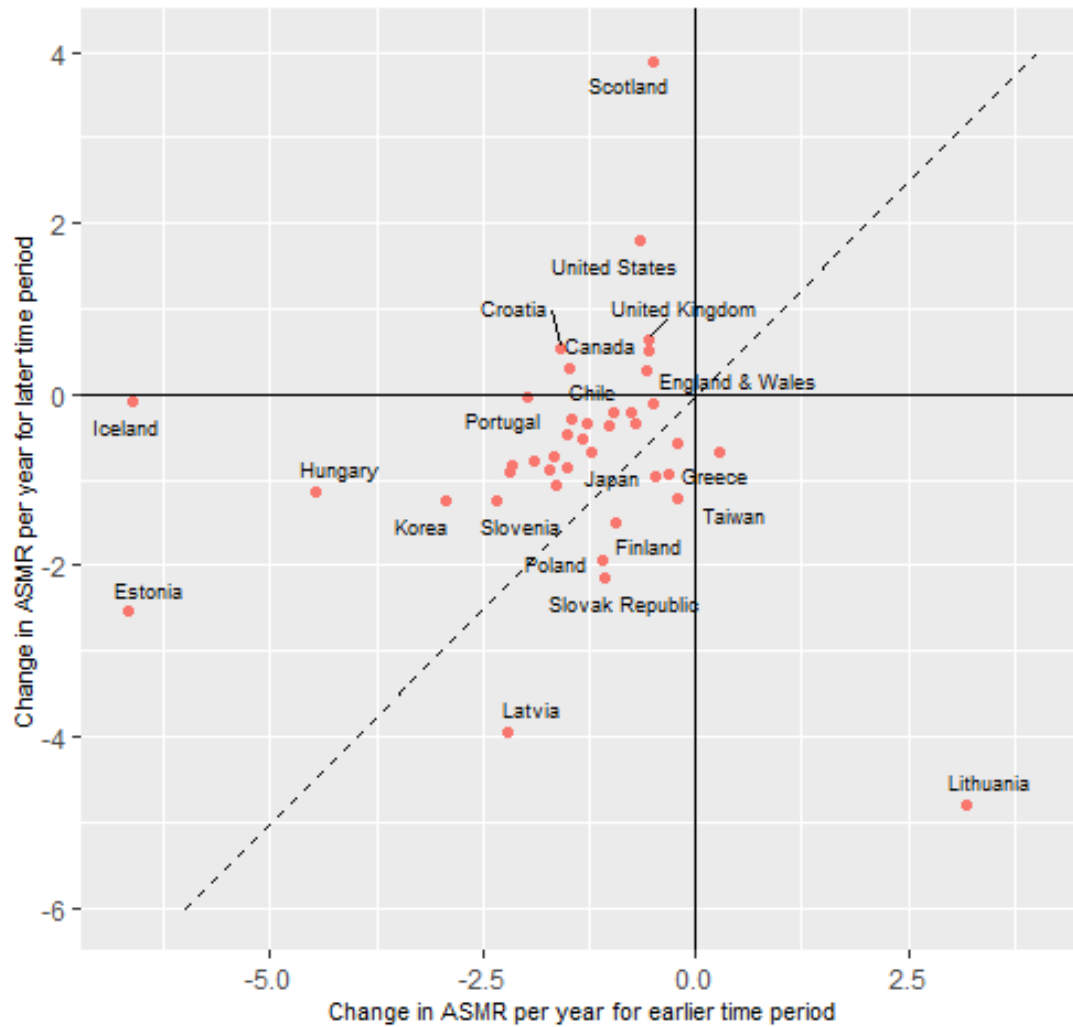


Figure S7.33 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the total population aged 50-69 years (1987-2018)

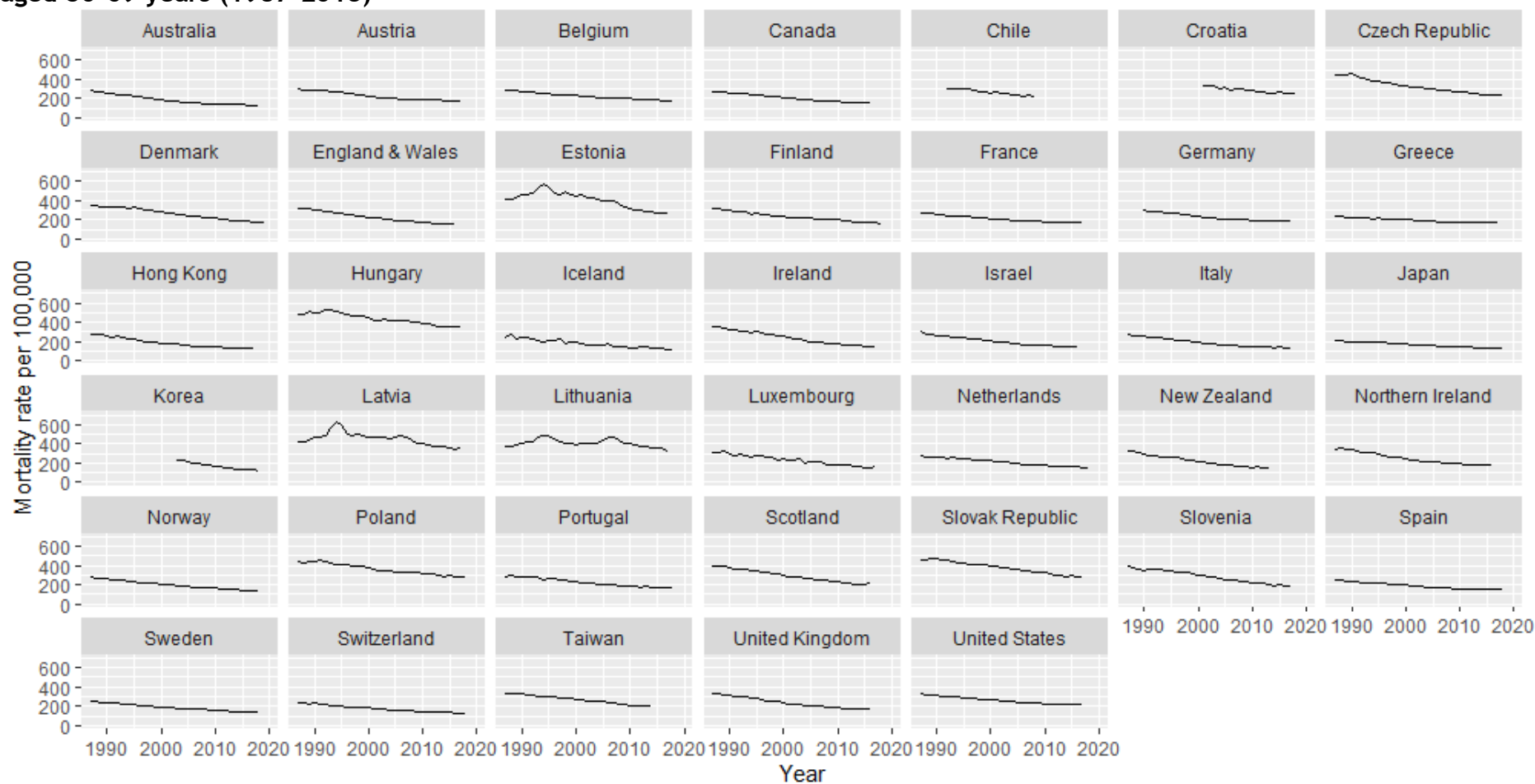


Figure S7.34 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the female population aged 50-69 years (1987-2018)

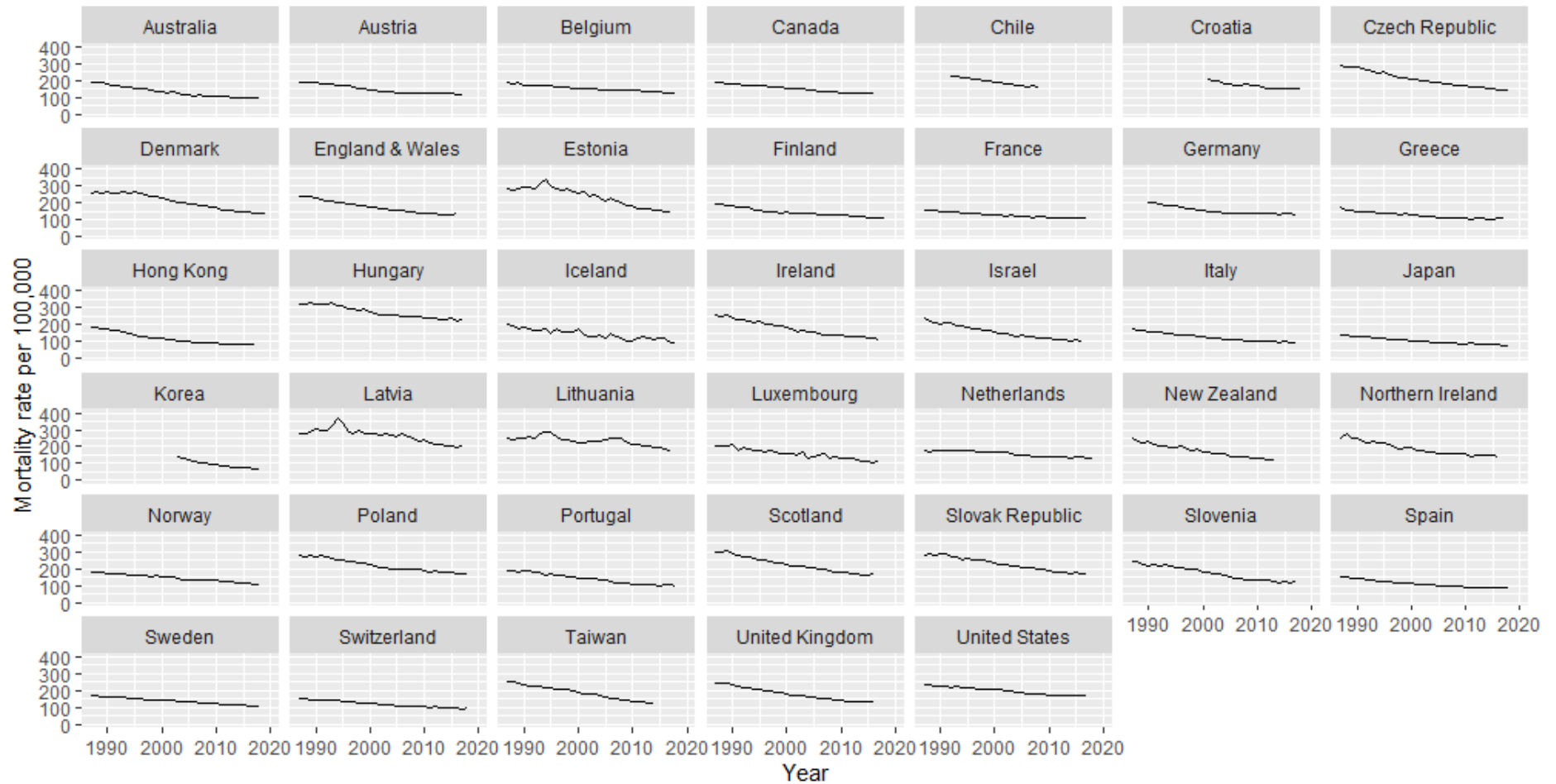


Figure S7.35 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the male population aged 50-69 years (1987-2018)

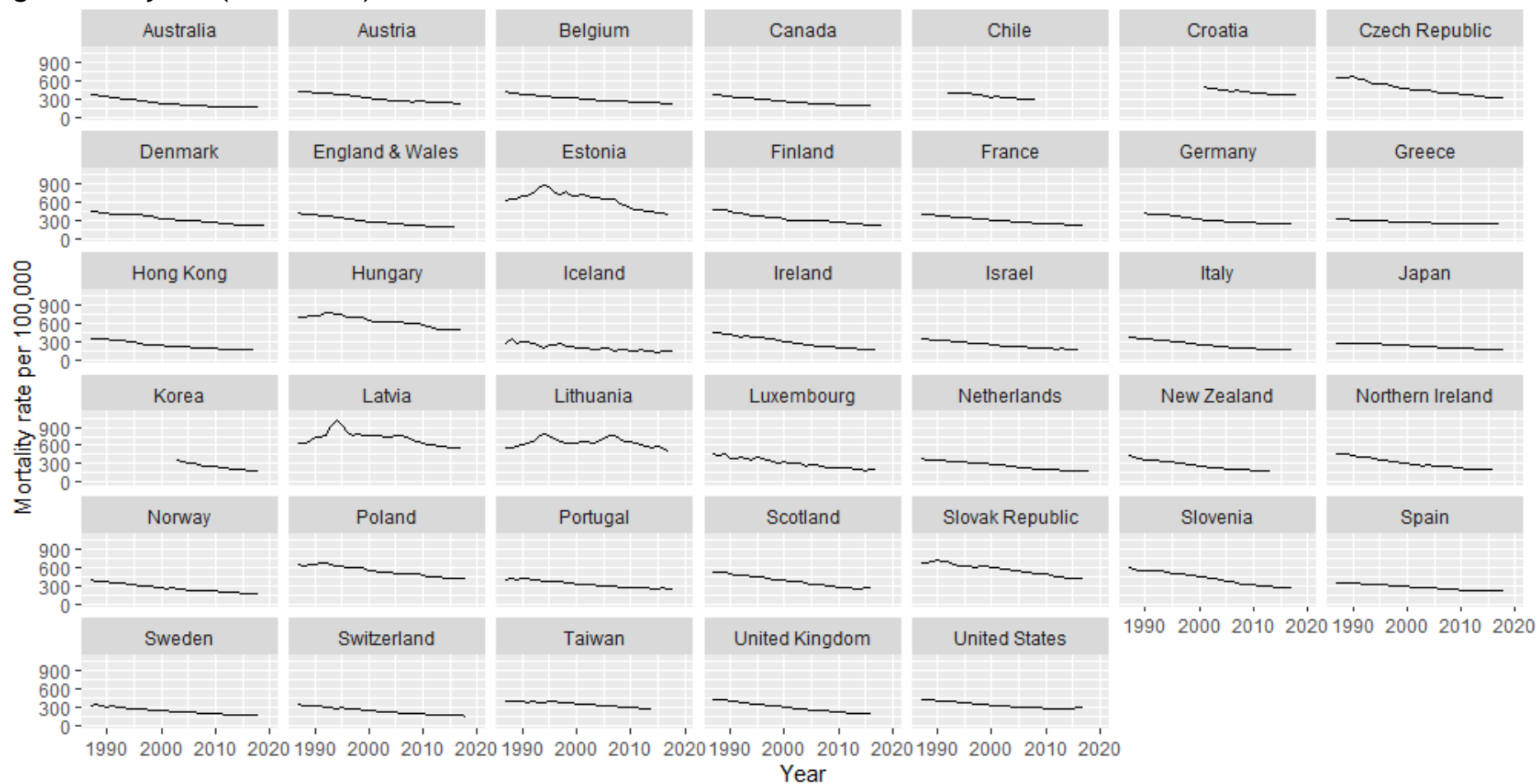


Figure S7.36 - Segmented regression fitted to the Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the total population aged 50-69 years (2000-2019, vertical lines indicate breakpoint)

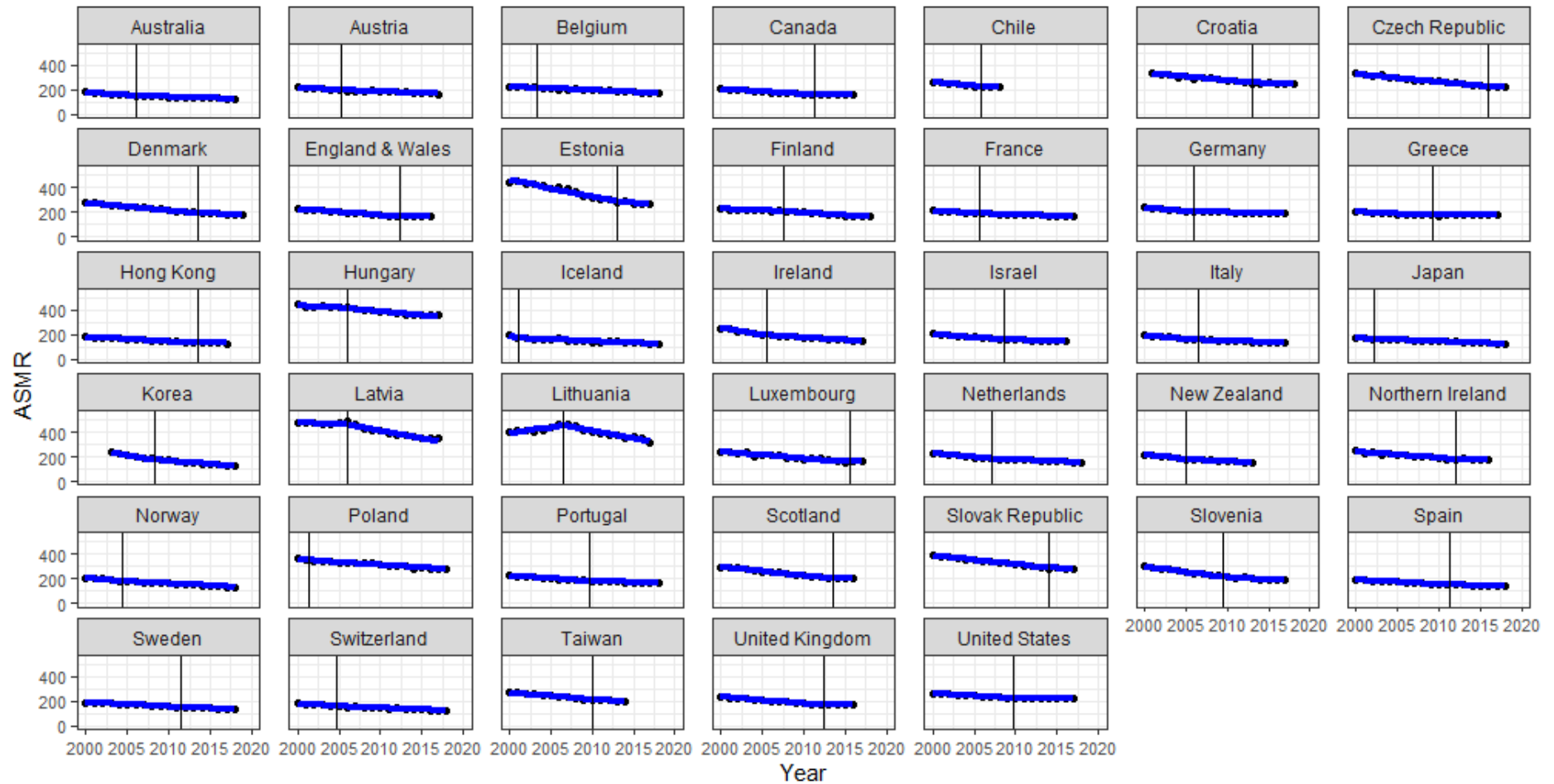


Figure S7.37 - Scatterplot of the rate of change in ASMRs for the total population (males and females combined) aged 50-69 years, before and after the breakpoint (2000-2019)

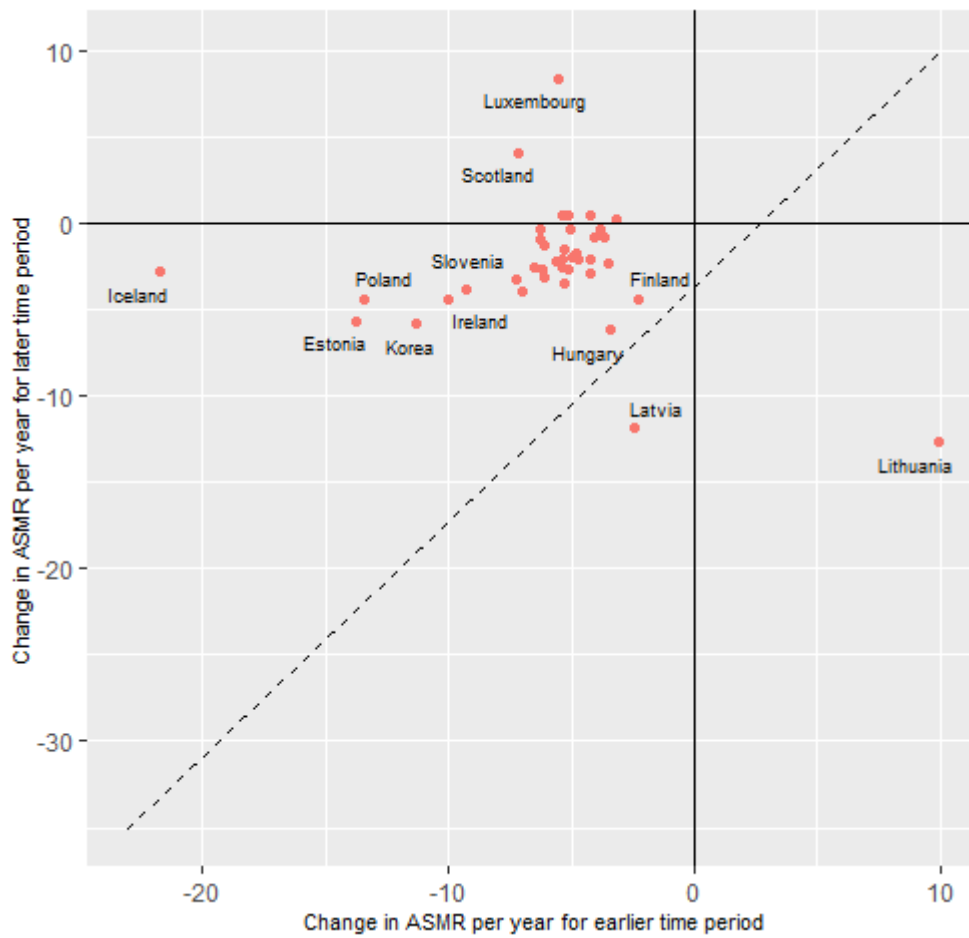


Figure S7.38 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the total population aged 70+ years (1987-2018)

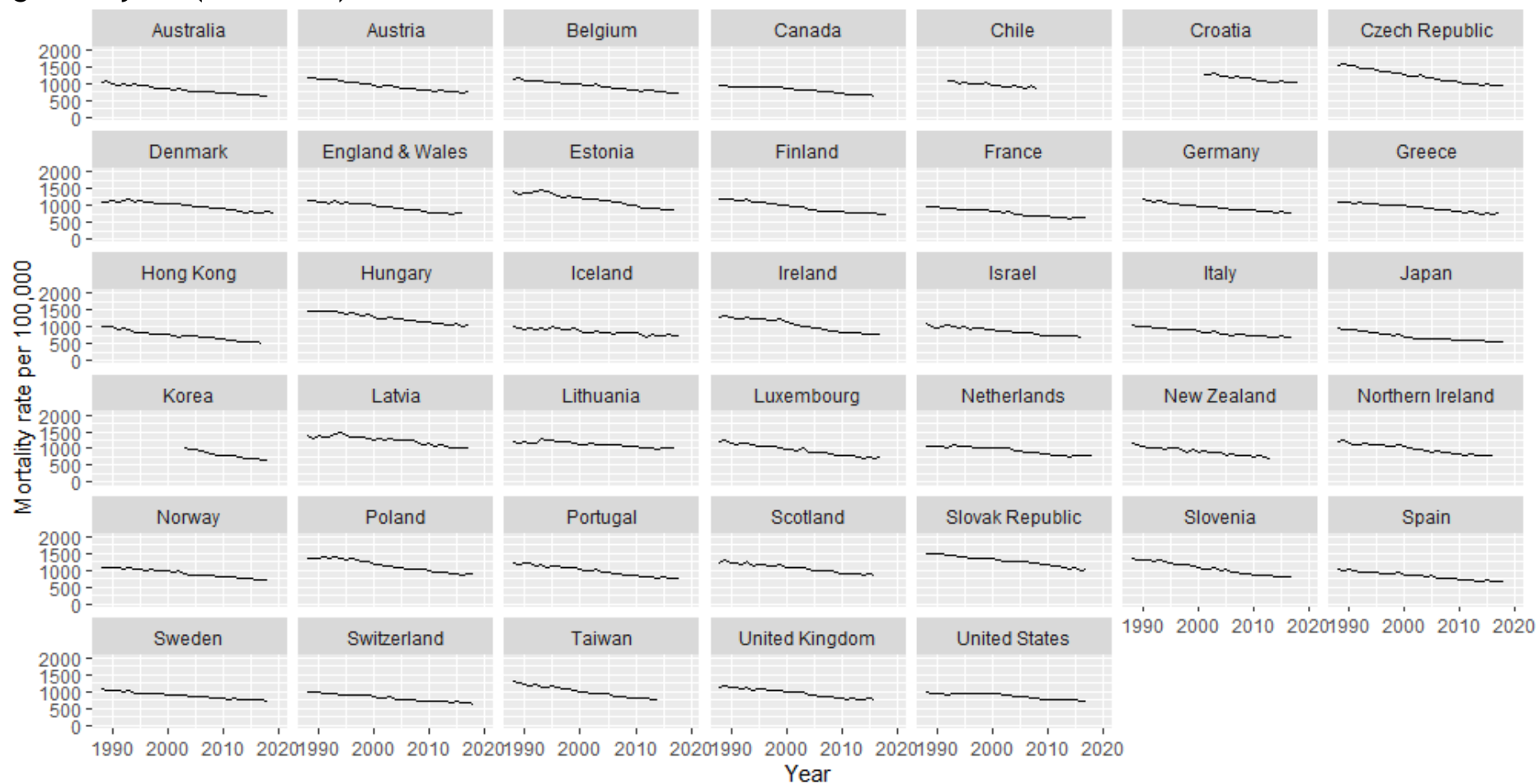


Figure S7.39 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the female population aged 70+ years (1987-2018)

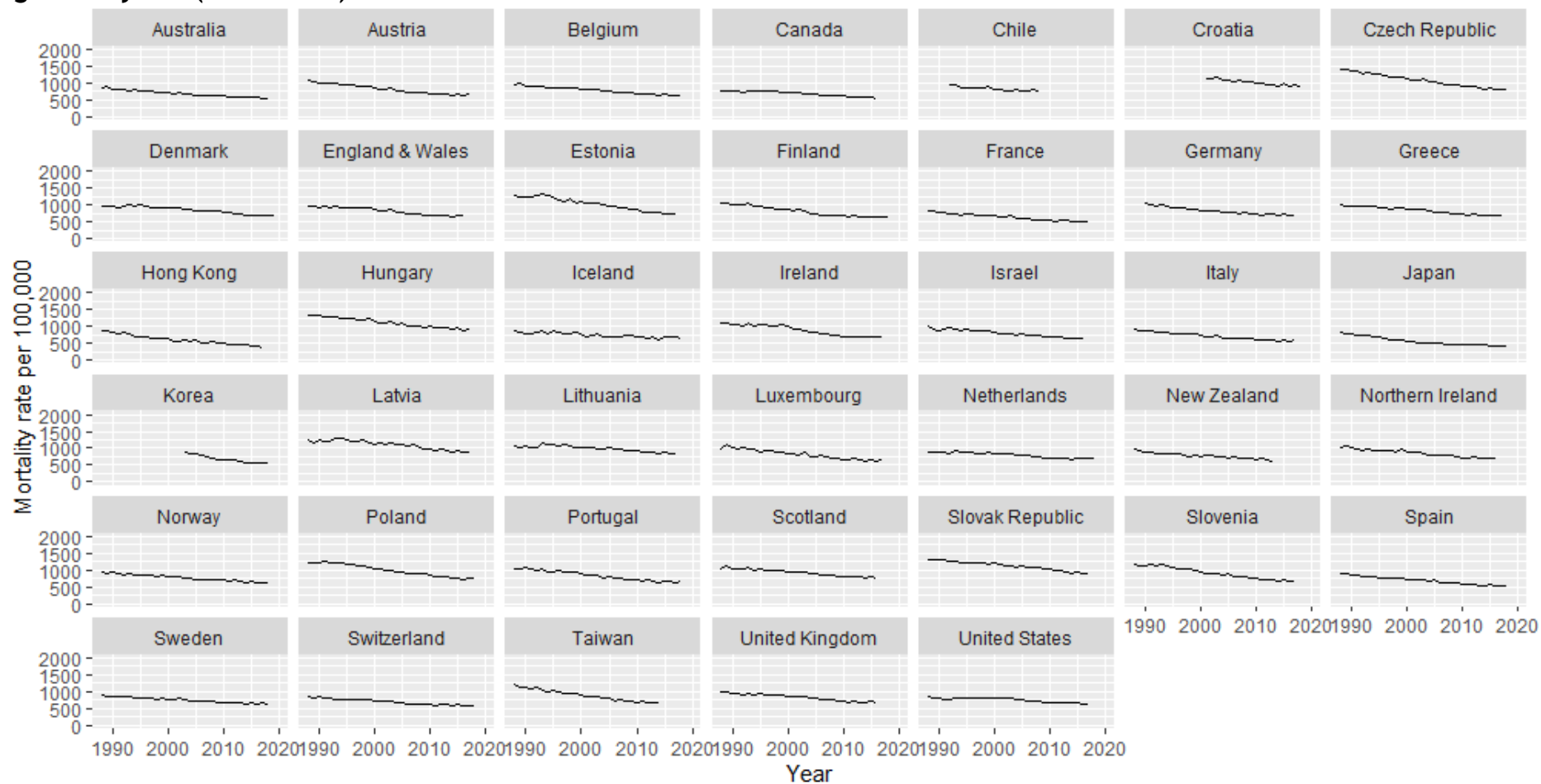


Figure S7.40 - Trend in Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the male population aged 70+ years (1987-2018)

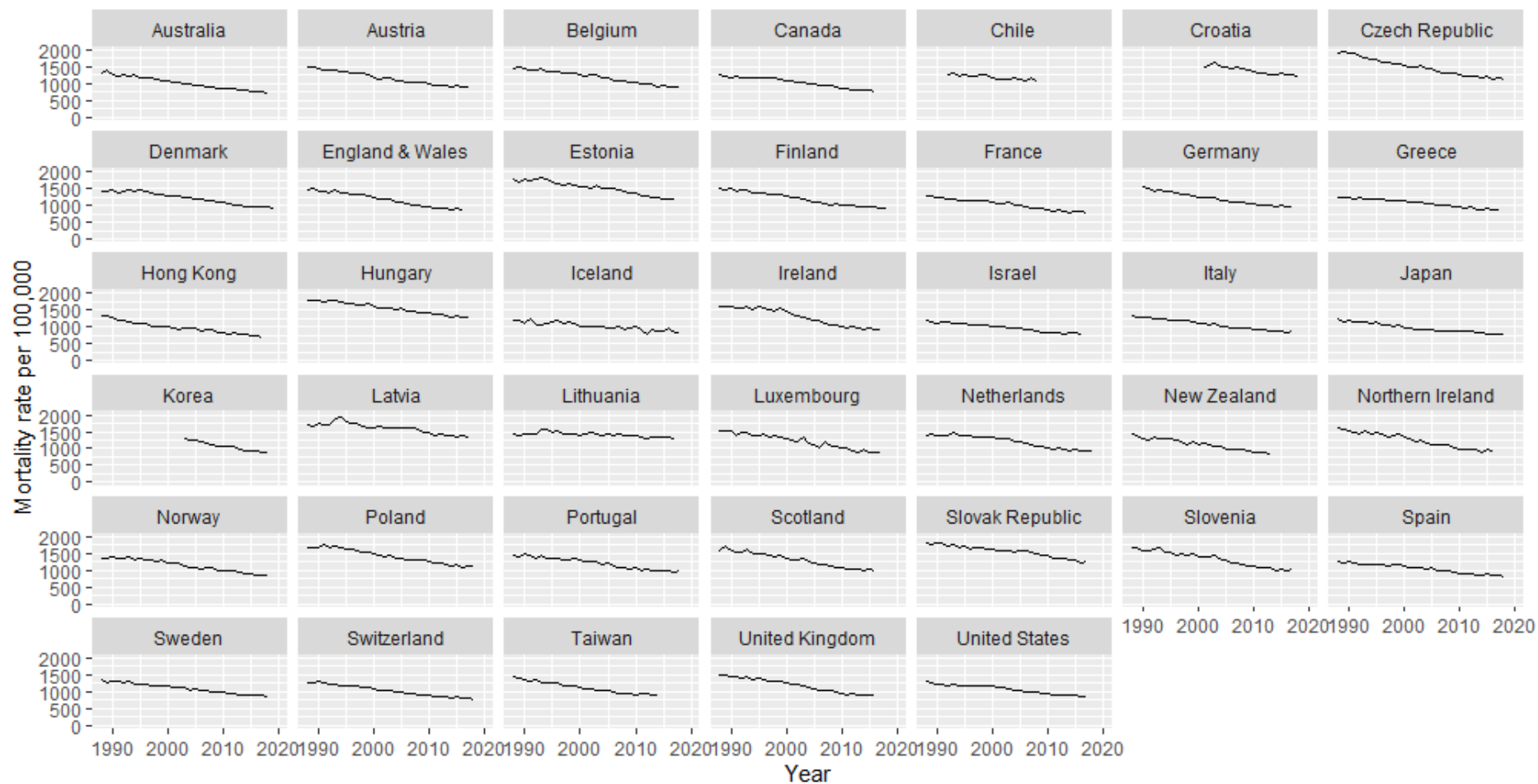


Figure S7.41 - Segmented regression fitted to the Age Standardised Mortality Rate (ASMR) per 100,000 population per year, for the total population aged 70+ years (2000-2019, vertical lines indicate breakpoint)

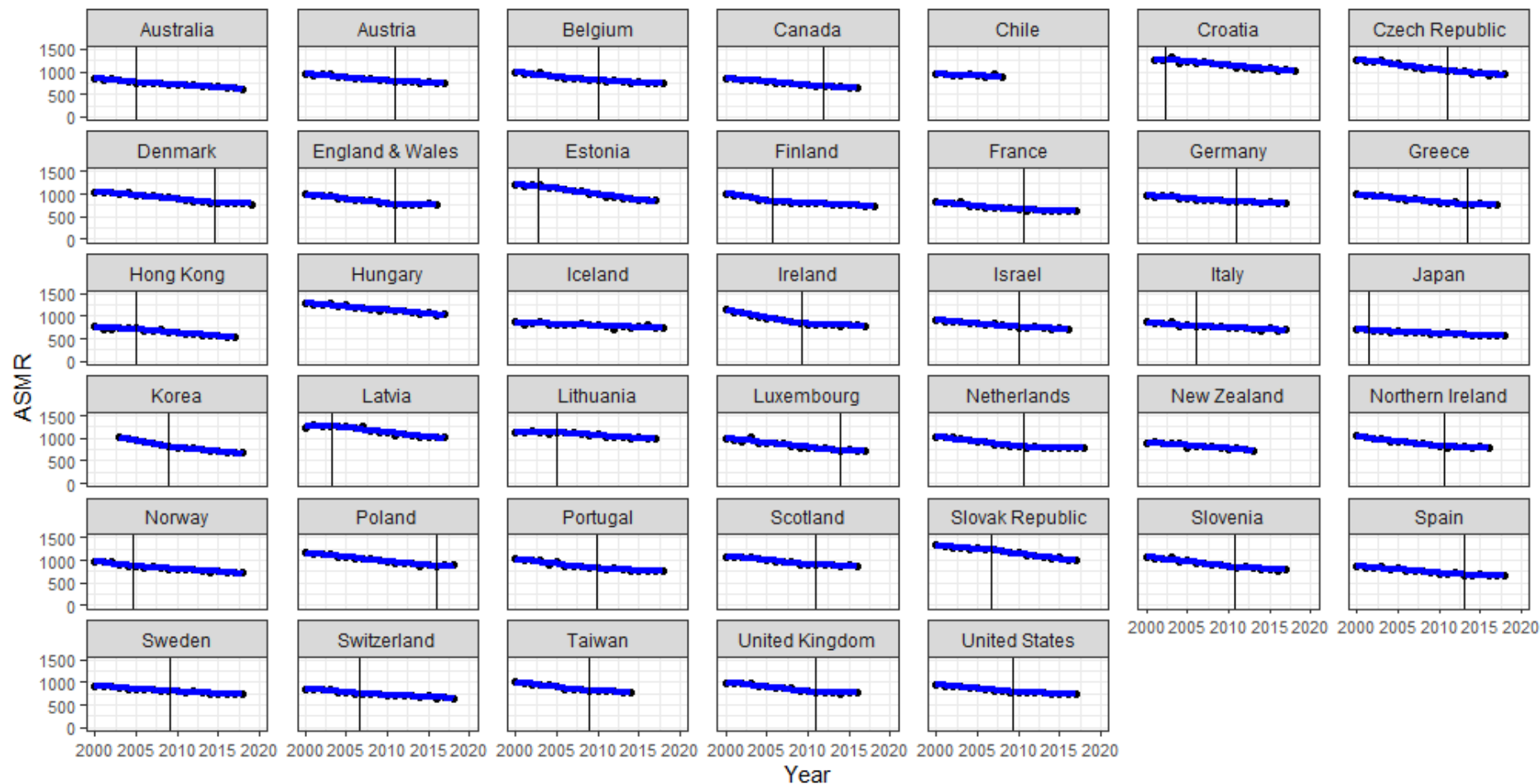


Figure S7.42 - Scatterplot of the rate of change in ASMRs for the total population (males and females combined) aged 70+ years, before and after the breakpoint (2000-2019)

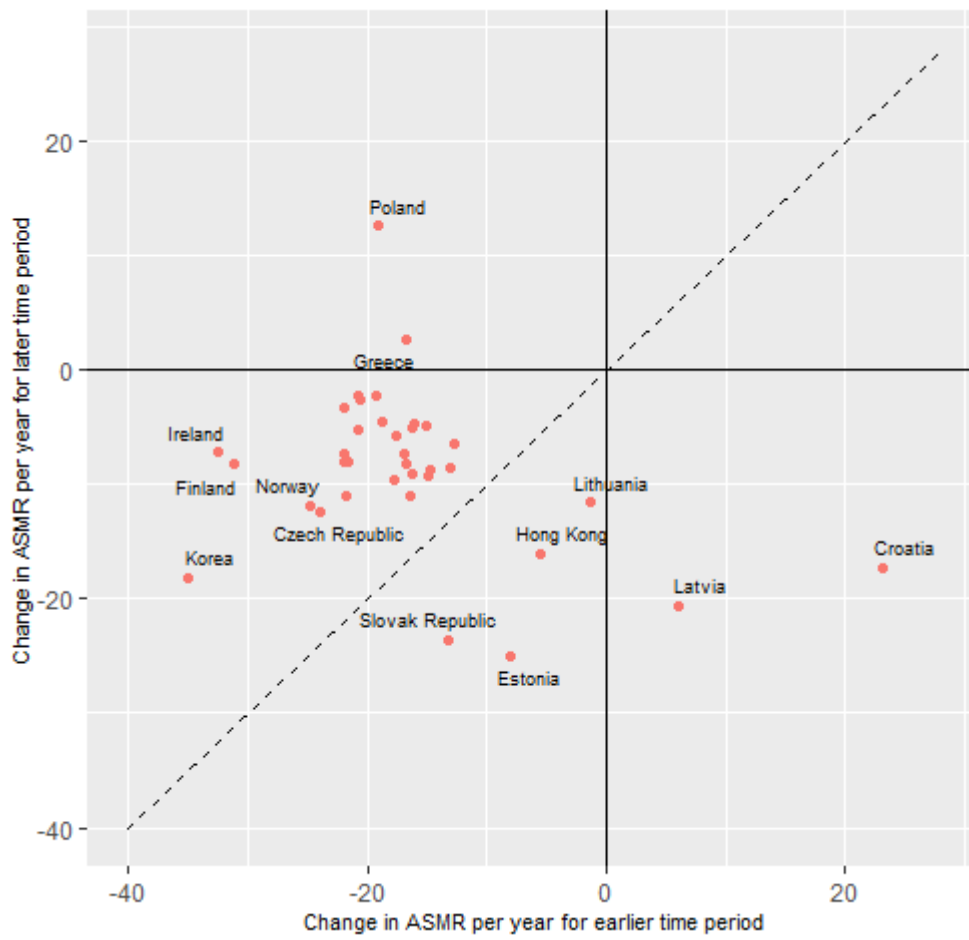


Figure S7.43 - Trend in lifespan variation ($e\ddagger$) for females (1987-2018)

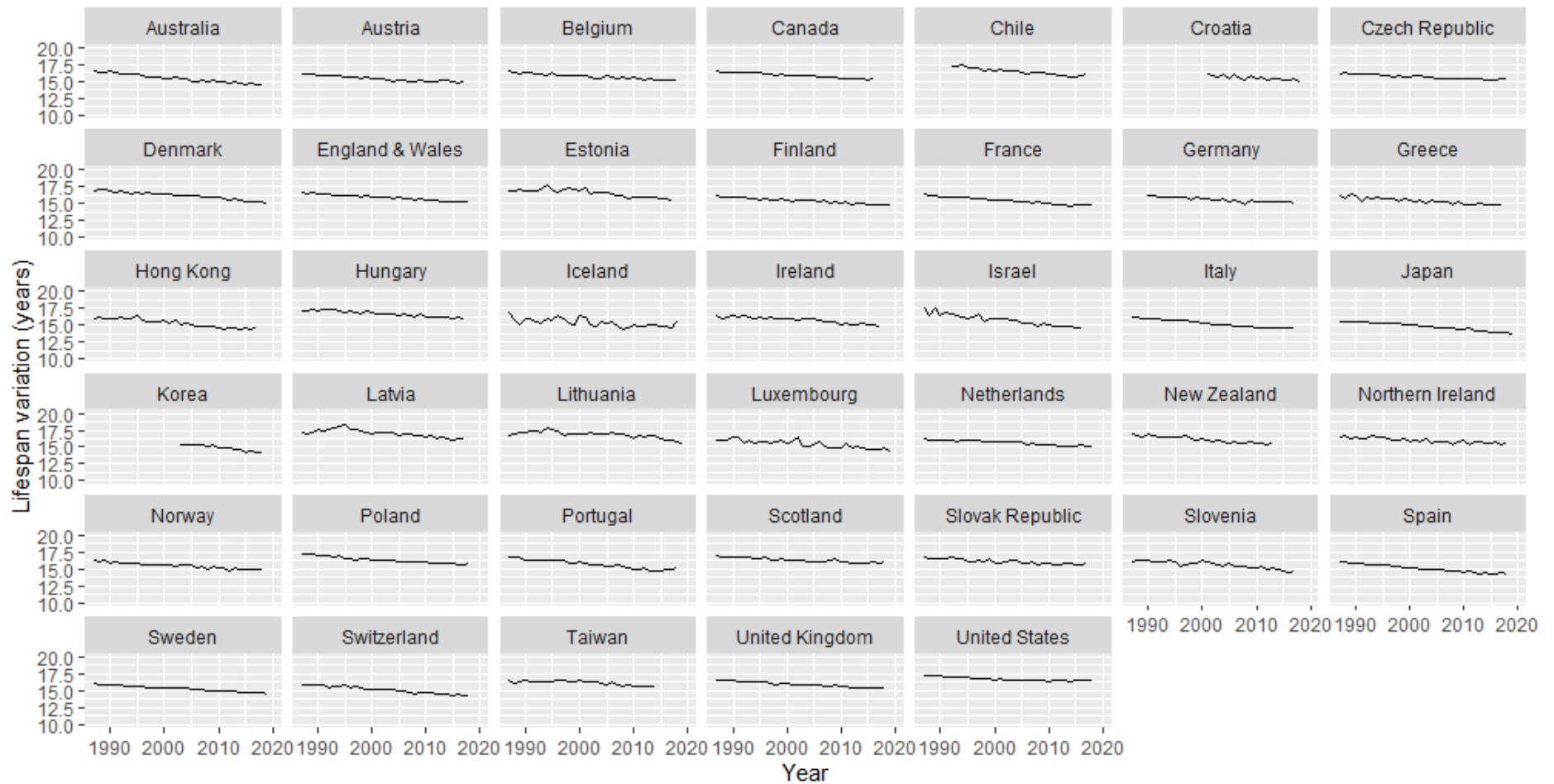


Figure S7.44 - Trend in lifespan variation (e_t) for males (1987-2018)

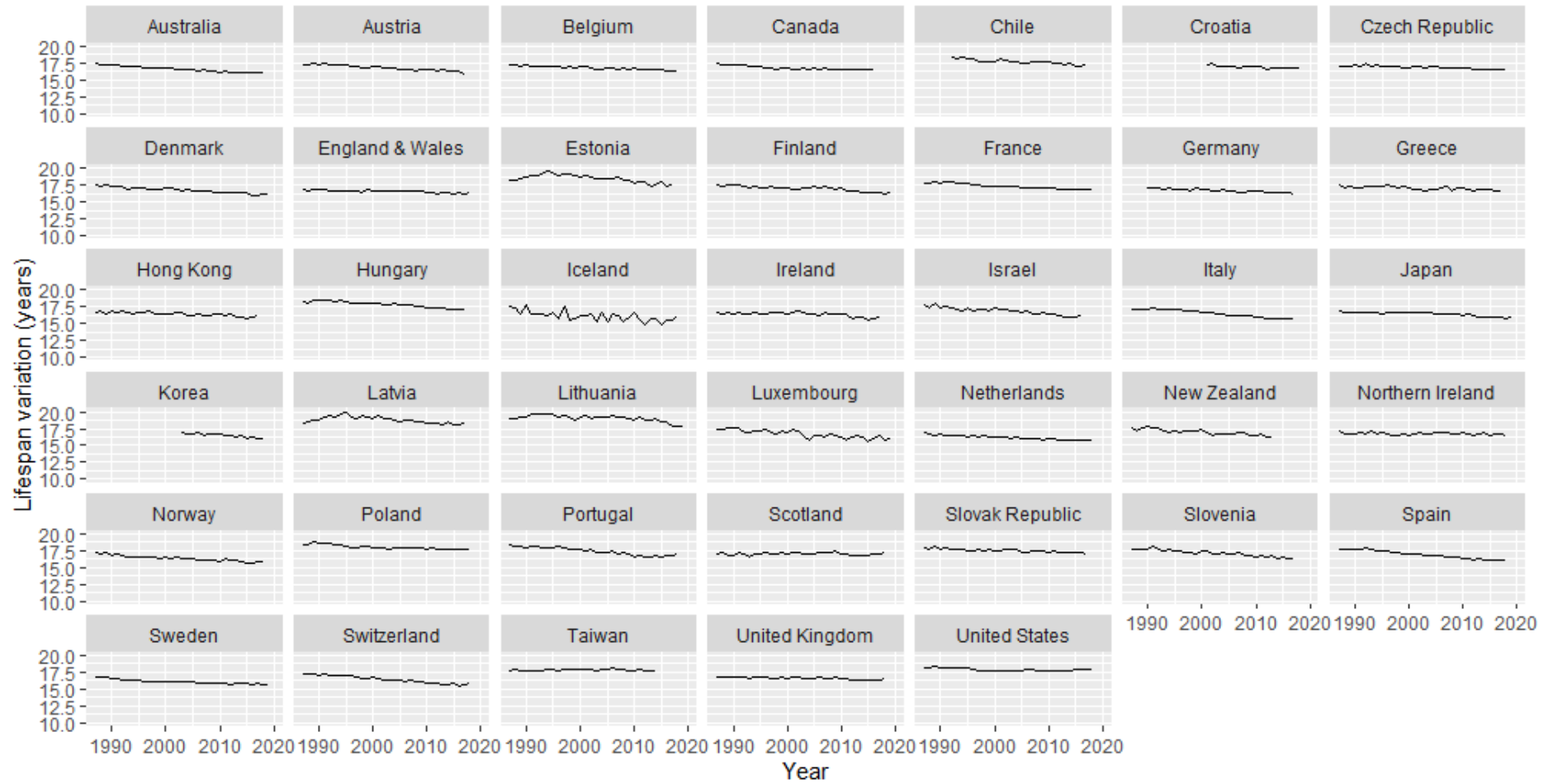


Figure S7.45 - Trends in unemployment rates (1987-2019)

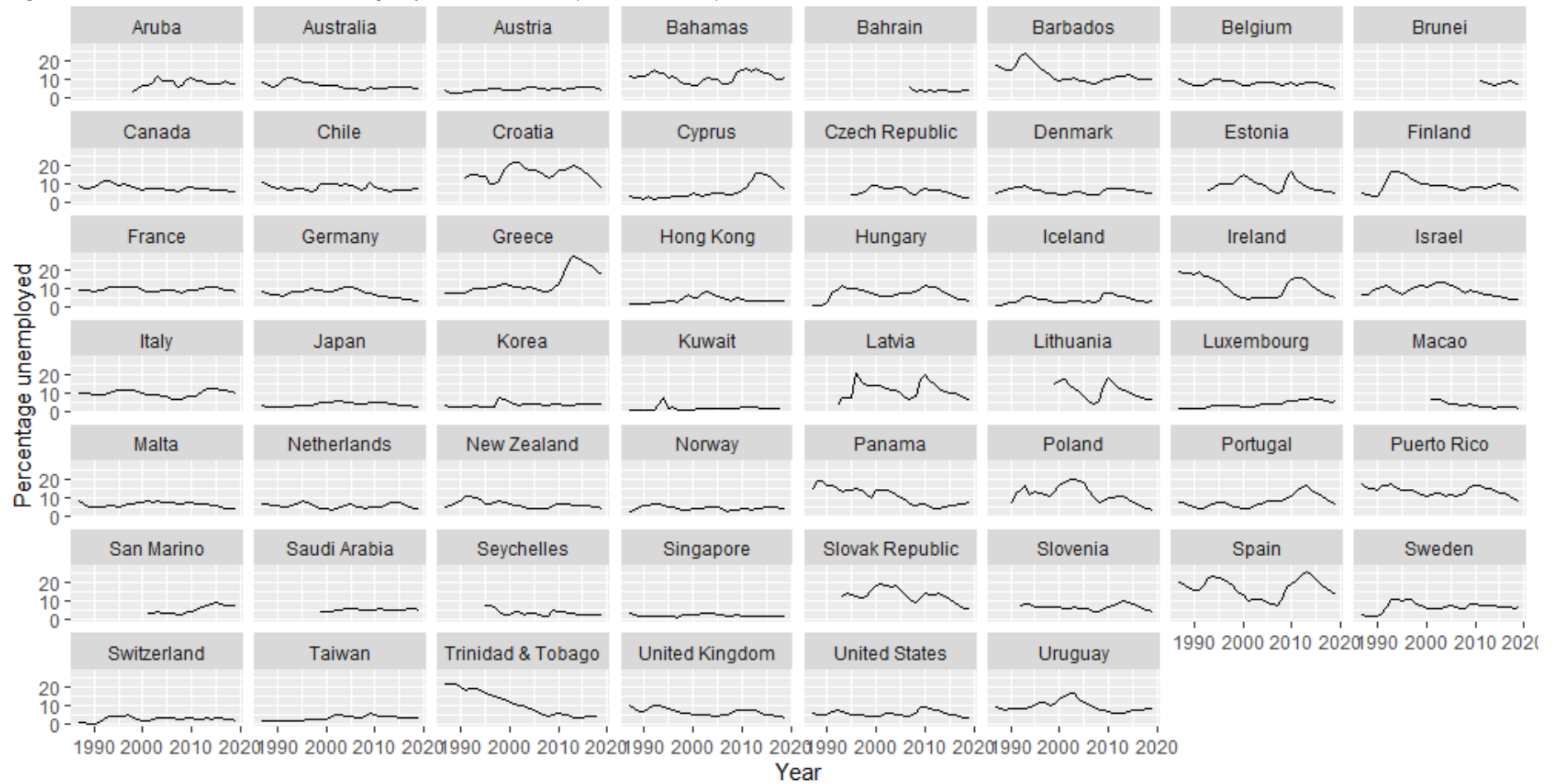


Figure S7.46 - Trends in Government Expenditure as a percentage of GDP (1987-2019)

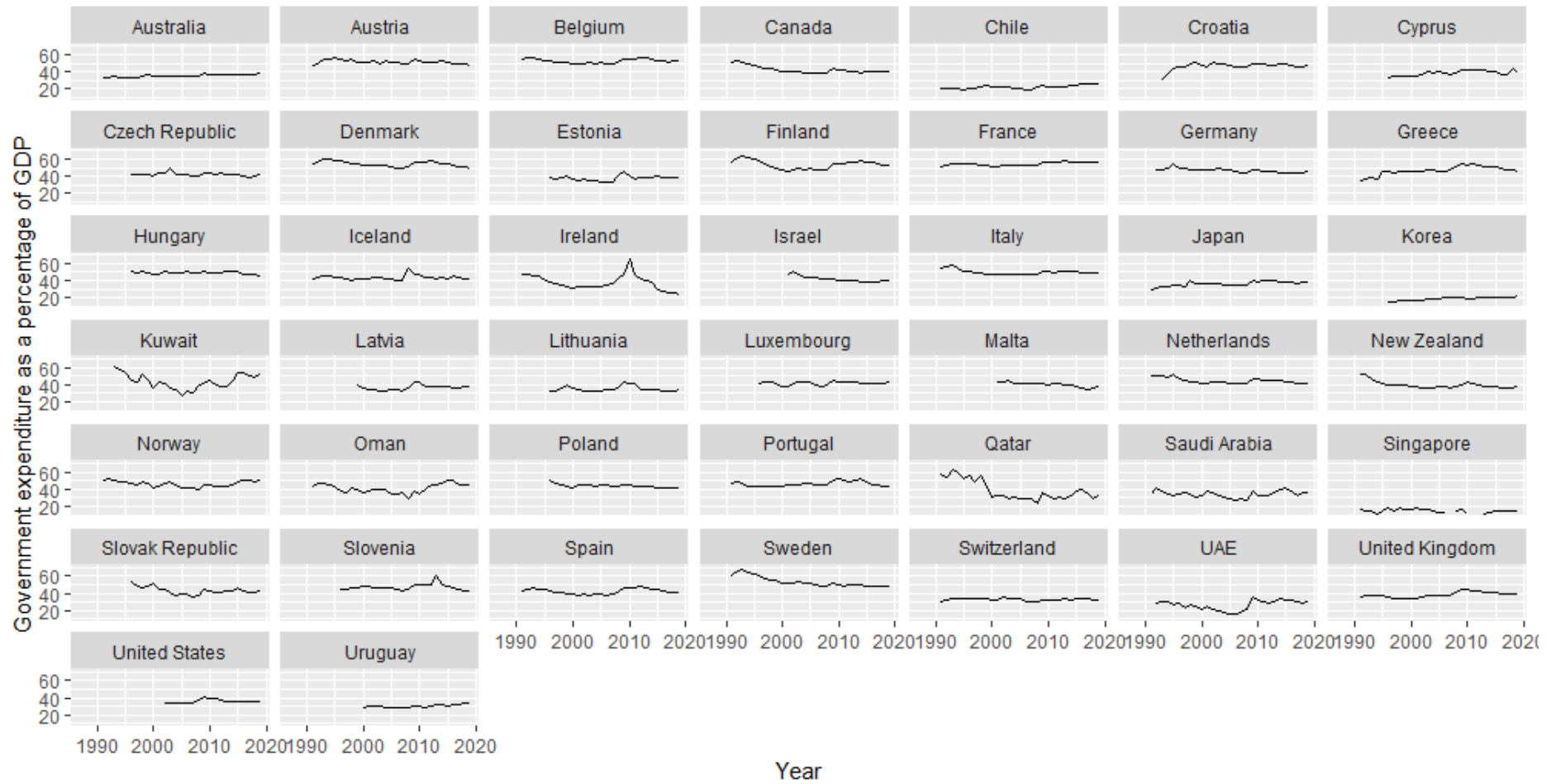


Figure S7.47 - Trends in government revenue as a percentage of GDP (1987-2019)



Figure S7.48 - Predicted government expenditure values as a percentage of GDP (in blue) and actual expenditure (black) (1987-2019)

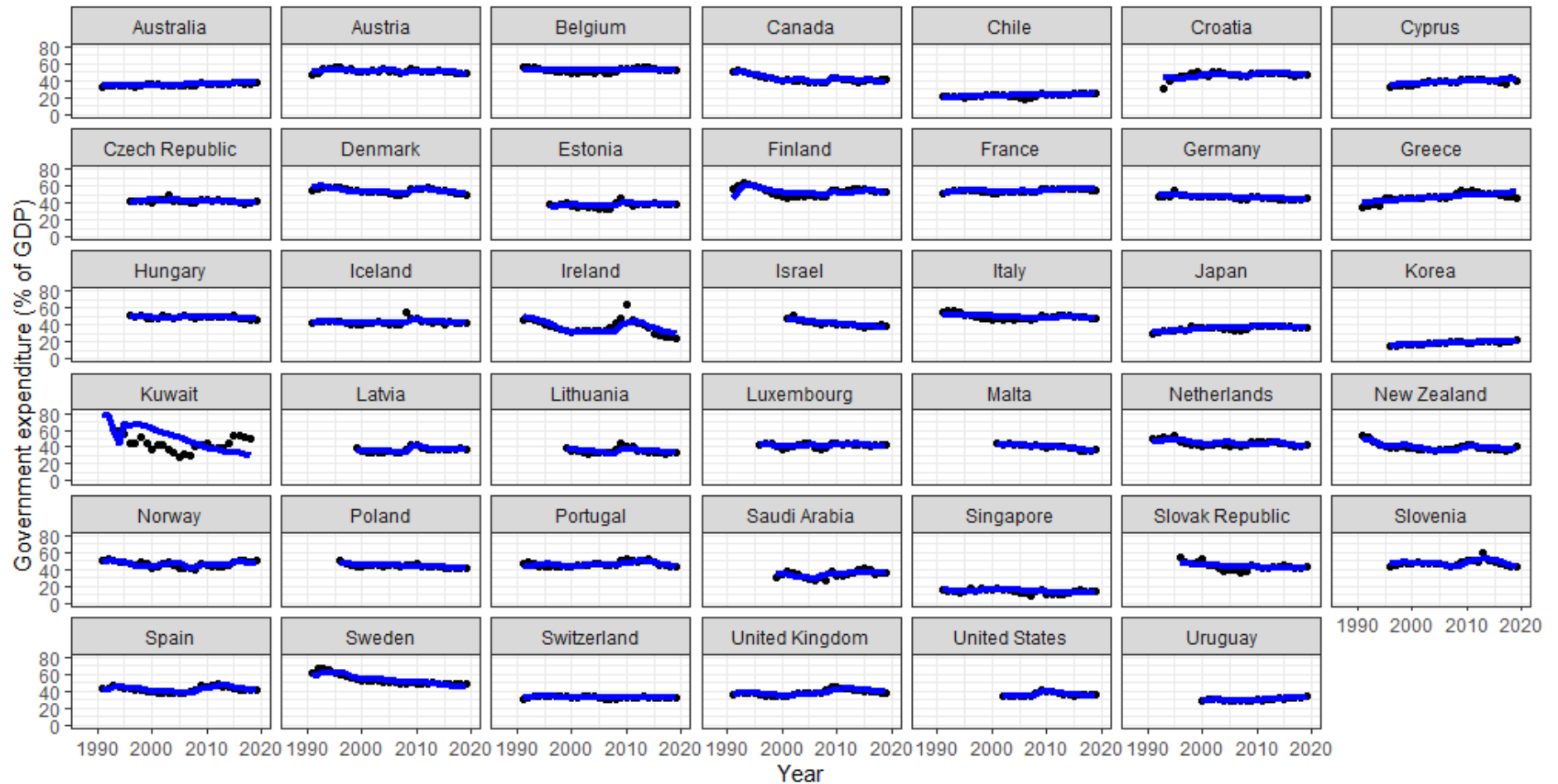


Figure S7.49 - Trends in the Alesina-Ardagna Fiscal Index (AAFI) (1987-2020)

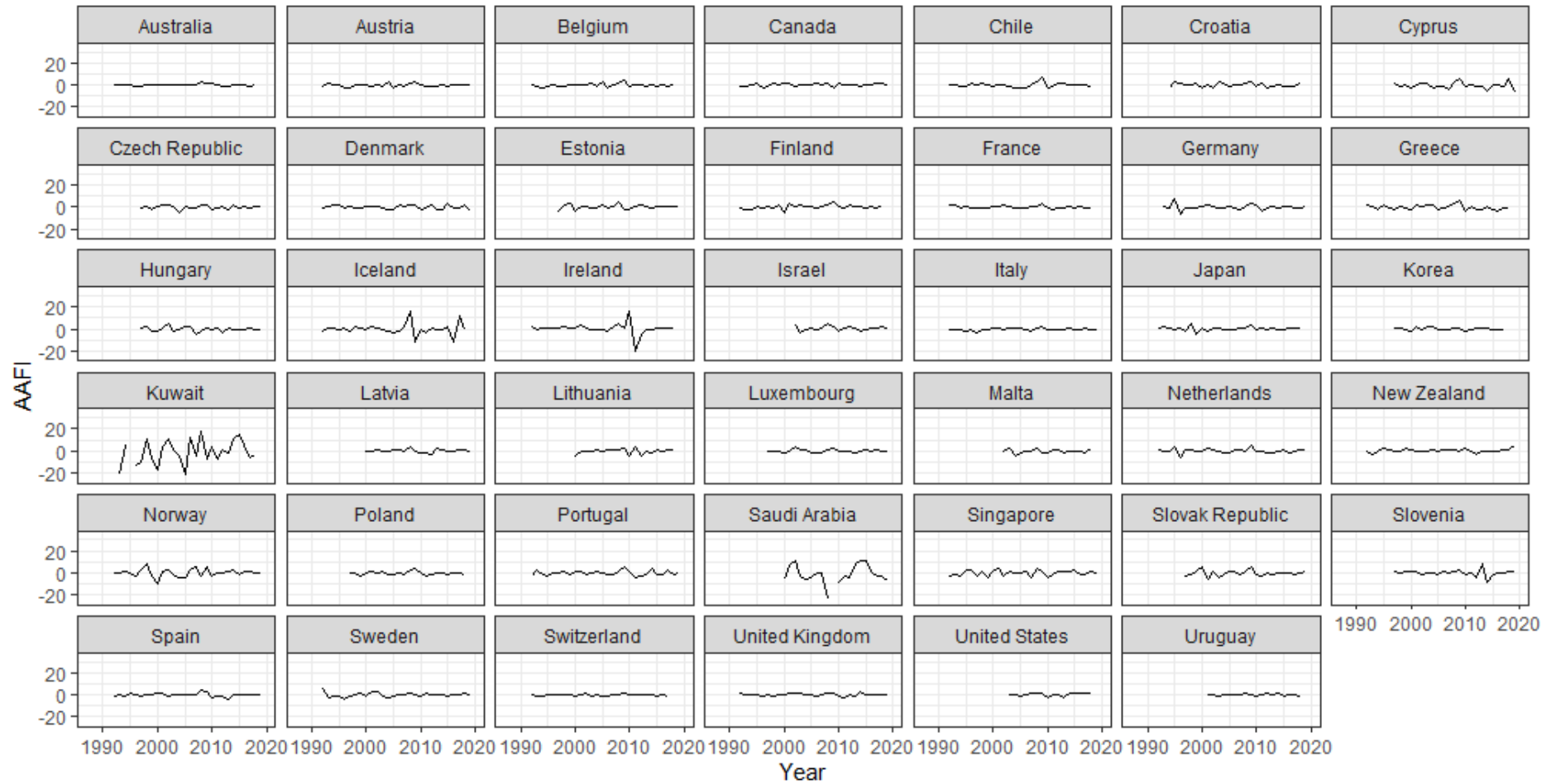


Table S7.5 - AAFI values (Australia to Czech Republic)

	Australia	Austria	Belgium	Canada	Chile	Croatia	Cyprus	Czech Republic
1993	-0.30	1.60	-0.77	-0.83	0.77			
1994	0.74	0.59	-2.47	0.82	-0.57	-2.10		
1995	0.48	0.78	-0.78	1.09	-1.50	2.89		
1996	-1.15	-2.36	-0.50	-2.72	1.41	2.05		
1997	-0.83	-2.10	-1.84	-1.68	0.19	0.56	1.81	-0.43
1998	0.73	0.41	-1.13	2.15	1.63	0.29	-0.97	-0.05
1999	0.20	0.64	-0.34	0.46	0.82	1.19	0.17	-2.48
2000	0.25	-0.32	-0.51	1.27	-1.21	-2.41	-2.03	0.45
2001	0.59	-1.52	-0.30	0.98	-0.28	-0.17	0.03	2.31
2002	0.33	0.74	0.24	-0.65	0.72	-2.57	2.10	1.42
2003	-0.16	-0.94	1.79	0.18	-0.63	2.42	1.67	0.13
2004	0.42	2.65	-1.63	0.23	-2.68	1.02	-2.29	-4.83
2005	0.17	-2.41	2.47	0.34	-2.20	-1.21	-1.61	0.84
2006	0.22	0.48	-2.95	1.14	-2.37	-0.05	-1.06	-0.24
2007	0.86	-0.84	0.21	0.85	-0.03	-0.11	-4.14	-0.33
2008	2.72	1.06	1.18	1.07	3.64	1.34	2.40	1.93
2009	1.66	2.45	4.30	-2.15	6.78	2.56	6.01	1.89
2010	1.11	-0.33	-1.36	1.99	-2.70	-0.77	-0.92	-1.72
2011	-0.46	-1.65	0.29	-0.02	-1.36	1.36	0.68	-1.04
2012	-1.16	-0.73	-0.03	-0.22	1.04	-3.09	-0.80	1.00
2013	-1.39	-0.70	-1.23	-0.47	1.36	-0.55	-1.13	-2.76
2014	-0.45	0.43	-0.08	-1.10	0.85	0.23	-4.97	1.52
2015	-0.13	-1.80	-0.64	0.24	0.67	-1.07	-0.03	-0.72
2016	0.19	0.18	-0.03	0.11	0.40	-1.49	0.27	-0.08
2017	-0.56	-0.30	-1.61	1.65	-0.15	-0.52	-1.23	-0.42
2018	-0.20	-0.20	0.19	1.68	-1.33	1.98	6.38	1.06
2019	-0.30	0.39	-0.77	0.24	0.77		-6.69	0.09

Table S7.5 (continued) - AAFI values (Denmark to Hungary)

	Denmark	Estonia	Finland	France	Germany	Greece	Hungary
1993	-0.13		-2.38	1.40	0.27	1.03	
1994	2.39		-1.92	-1.10	-0.68	-2.96	
1995	1.25		1.00	-0.23	6.94	1.47	
1996	-0.45		-1.53	-1.32	-5.99	-1.50	
1997	0.22	-3.88	0.22	-0.29	-0.77	-2.10	1.06
1998	-0.40	2.32	-1.44	-1.19	-0.32	0.42	1.86
1999	-1.53	3.64	1.37	-0.66	-0.70	-0.27	-2.38
2000	0.07	-3.70	-4.66	0.20	-0.03	-1.89	-2.15
2001	0.47	0.01	2.67	0.34	1.48	1.26	0.97
2002	1.02	0.09	0.92	1.86	0.70	0.46	4.79
2003	-0.98	-1.24	1.75	0.78	-0.37	1.67	-1.71
2004	-2.34	-0.54	0.42	-0.54	-0.48	1.18	-0.77
2005	-1.93	1.62	0.04	-0.27	-0.14	-2.78	1.54
2006	1.20	-1.42	-0.50	-0.87	-1.49	-0.47	1.50
2007	0.10	0.43	-0.24	0.51	-1.66	0.62	-4.24
2008	1.98	5.18	1.65	0.83	0.59	3.33	-1.39
2009	2.28	-1.86	4.41	3.27	2.98	5.39	1.11
2010	-1.86	-2.91	-0.19	-0.37	1.37	-3.23	0.00
2011	-0.79	-0.20	-0.80	-1.69	-3.32	0.29	0.76
2012	1.43	1.80	1.26	-0.41	-0.80	-2.21	-2.91
2013	-1.71	0.16	-0.23	-1.10	0.01	-2.21	0.20
2014	-1.70	-0.64	-0.13	-0.18	-0.50	0.22	-0.01
2015	3.29	0.82	-1.51	-0.32	-0.19	-1.68	-0.84
2016	-0.81	-0.13	0.06	0.15	-0.22	-3.62	-0.34
2017	-1.35	0.52	-0.83	-0.50	-0.26	-0.98	0.57
2018	2.00	0.43	1.91	-0.62	-0.44	-0.35	-0.35
2019	-3.30	-0.14	-2.38	1.40	0.39	1.03	-0.12

Table S7.5 (continued) - AAFI values (Iceland to Latvia)

	Iceland	Ireland	Israel	Italy	Japan	Korea, Republic of	Kuwait	Latvia
1993	0.37	-0.61		-0.38	2.25		-21.89	
1994	0.23	0.75		-0.95	0.35		5.70	
1995	-0.75	1.24		-1.62	-0.19		-27.91	
1996	0.75	0.37		-0.60	0.08		-12.38	
1997	-1.53	0.29		-3.63	-1.39	0.12	-9.54	
1998	2.12	1.52		0.00	4.83	0.62	10.29	
1999	0.25	0.20		-1.20	-4.90	0.00	-7.91	
2000	-0.79	0.11		0.69	1.38	-2.54	-16.46	-1.02
2001	2.00	4.14		0.81	-2.49	1.60	4.03	-0.22
2002	0.28	0.99	3.30	-0.30	0.35	-0.74	10.06	0.90
2003	-0.24	-0.96	-3.64	0.35	0.65	1.74	1.67	-0.57
2004	-2.20	-0.89	-1.12	0.28	-0.88	1.46	-4.18	-0.68
2005	-3.82	-0.13	0.45	0.62	-0.15	-0.75	-21.45	0.67
2006	-1.93	-1.37	-1.17	-0.42	-0.70	-0.17	11.77	0.61
2007	2.08	2.29	0.34	-2.24	0.49	-1.01	-4.43	-0.71
2008	16.38	5.50	4.28	1.20	0.81	0.63	17.25	3.14
2009	-11.38	1.21	1.59	2.51	2.83	1.38	-7.71	0.09
2010	-0.78	16.20	-1.96	-0.91	-0.66	-1.46	3.36	-1.23
2011	-3.10	-20.03	0.31	-0.65	1.19	-0.11	-7.32	-2.04
2012	0.30	-4.84	1.57	-0.75	-0.06	0.16	0.49	-2.91
2013	-0.69	-0.23	0.14	-0.16	0.07	0.89	-2.05	1.91
2014	-0.94	-0.78	-1.44	0.08	-1.25	0.16	11.03	1.54
2015	2.63	0.36	-0.87	-0.37	-1.31	-0.14	15.32	0.19
2016	-11.31	0.32	0.95	-0.14	0.66	-1.14	4.93	-1.02
2017	12.24	1.05	0.20	0.06	0.19	-0.54	-5.60	0.77
2018	-0.06	0.31	2.61	-0.21	0.43		-3.50	0.41
2019	0.37	-0.61	0.46	-0.53	2.25		-21.89	0.06

Table S7.5 (continued) - AAFI values (Lithuania to Poland)

	Lithuania	Luxembourg	Malta	Netherlands	New Zealand	Norway	Poland
1993				-0.55	-2.98	-0.42	
1994				-0.25	-0.77	0.74	
1995				3.80	2.03	-0.50	
1996				-6.35	1.51	-2.56	
1997		-0.30		0.69	0.16	2.53	-0.09
1998		-0.19		0.89	0.14	8.16	-0.28
1999		0.15		-0.86	1.81	-2.65	-2.29
2000	-5.22	-1.80		-0.54	0.48	-10.31	0.28
2001	-0.87	0.24		2.20	0.26	1.37	1.53
2002	0.05	3.33	-0.28	1.10	-0.87	2.23	-0.16
2003	0.13	0.99	2.86	-0.01	-0.15	-1.07	1.27
2004	1.03	1.38	-4.56	-2.15	0.76	-3.64	-0.96
2005	0.29	-1.80	-1.32	-1.61	-0.25	-4.39	-0.91
2006	1.21	-2.07	-0.08	0.34	0.32	2.63	0.10
2007	1.36	-2.30	-0.28	0.94	1.61	5.20	-1.16
2008	1.46	1.03	2.06	0.17	1.34	-2.51	2.07
2009	2.00	2.22	-1.81	4.64	-0.25	5.49	3.51
2010	-4.44	-0.20	-1.27	-0.40	2.77	-3.10	-0.04
2011	3.25	-0.98	1.05	-0.82	-0.10	-0.49	-2.52
2012	-4.79	-0.39	0.58	-1.25	-3.49	0.07	-1.20
2013	0.28	-1.21	-1.42	-2.39	0.27	0.67	0.41
2014	-1.40	-0.65	0.02	-0.87	-0.07	3.01	-0.37
2015	0.35	0.60	-0.79	0.34	-0.76	-1.76	-0.84
2016	0.14	-0.20	-0.74	-1.21	-0.12	1.00	-0.07
2017	0.30	1.06	-2.48	-1.01	0.52	1.48	-0.74
2018	0.32	-0.54	2.47	0.82	0.62	0.12	-1.21
2019		-0.59		1.06	5.10	0.25	

Table S7.5 (continued) - AAFI values (Portugal to Spain)

	Portugal	Saudi Arabia	Singapore	Slovak Republic	Slovenia	Spain
1993	2.22		-1.53			0.33
1994	-1.34		-2.47			-1.40
1995	-2.71		1.97			1.14
1996	-0.55		3.27			-0.47
1997	-0.72		-3.00	-3.39	1.21	-1.20
1998	1.53		1.76	-1.15	-0.69	-0.14
1999	-1.02		-4.25	1.03	0.65	0.25
2000	0.68	-5.27	1.02	4.93	1.53	0.85
2001	1.33	7.09	3.91	-5.54	1.66	1.16
2002	-1.53	11.12	-2.92	1.16	-2.22	-0.69
2003	-0.07	-3.20	0.58	-4.86	-0.35	0.06
2004	1.56	-5.91	-0.20	-0.96	-0.09	-0.01
2005	-0.42	-4.36	-0.01	1.00	-0.90	-0.34
2006	-1.99	-0.23	1.00	1.37	0.62	-0.51
2007	-1.38	-0.08	-3.72	-1.04	0.52	0.40
2008	0.96	-23.23	3.91	0.83	1.91	4.81
2009	5.01	37.79	1.28	5.07	2.29	3.03
2010	0.64	-8.47	-4.04	-1.23	-2.20	-2.85
2011	-4.82	-3.25	-1.74	-2.82	-0.25	-0.61
2012	-2.87	-4.29	0.73	-0.16	-3.63	-0.87
2013	-1.48	7.60	1.53	-1.56	8.74	-4.41
2014	3.42	10.48	1.07	0.48	-8.36	-0.19
2015	-1.96	10.99	2.07	-0.06	-1.67	0.52
2016	-1.63	1.37	-1.45	0.20	0.51	0.50
2017	2.13	-2.76	-1.65	-1.19	0.09	0.03
2018	-1.50	-3.37	1.82	0.52	1.34	0.55
2019	-0.77	-6.63	-0.20	1.62	1.49	0.76

Table S7.5 (continued) - AAFI values (Sweden to Uruguay)

	Sweden	Switzerland	Switzerland	United Kingdom	United States	Uruguay
1993	-2.10	-0.66	-0.66	0.58		
1994	-1.62	-0.65	-0.65	0.49		
1995	-1.28	-0.52	-0.52	0.64		
1996	-4.42	-0.06	-0.06	-0.64		
1997	-1.52	0.10	0.10	0.18		
1998	-0.12	-0.47	-0.47	-0.57		
1999	1.56	0.79	0.79	-0.48		
2000	-1.04	-1.52	-1.52	0.05		
2001	2.30	0.13	0.13	1.78		-0.42
2002	2.63	1.64	1.64	1.93		-0.10
2003	-0.87	-0.95	-0.95	1.57	0.66	-1.20
2004	-2.48	-0.07	-0.07	0.25	0.21	-0.31
2005	-1.70	-0.73	-0.73	0.08	-0.58	-0.30
2006	0.24	-1.32	-1.32	-1.32	-0.30	0.78
2007	-0.11	-0.53	-0.53	-0.11	0.88	-0.12
2008	1.44	-0.22	-0.22	2.02	1.95	1.69
2009	0.38	0.95	0.95	1.94	1.39	0.13
2010	-0.88	0.20	0.20	-1.28	-2.62	-0.87
2011	0.99	-0.06	-0.06	-2.09	-0.29	0.14
2012	0.54	0.44	0.44	0.29	-0.49	2.01
2013	0.34	0.58	0.58	-1.48	-2.43	-0.55
2014	0.28	-0.08	-0.08	2.24	1.26	0.93
2015	-0.97	-0.95	-0.95	0.30	0.85	-1.04
2016	-0.41	0.27	0.27	-0.46	1.30	0.76
2017	-0.16	-0.80	-0.80	-0.10	1.15	-0.22
2018	1.16			0.24	1.96	-0.83
2019	-0.19	-0.66	-0.66	0.26		

Figure S7.50 - Scatterplot of AAFI and change in CAPB for each available year and country (1987-2020), linear regression line (with 95% confidence intervals) and R^2 value. Note high CAPB, and low AAFI, values indicate greater austerity

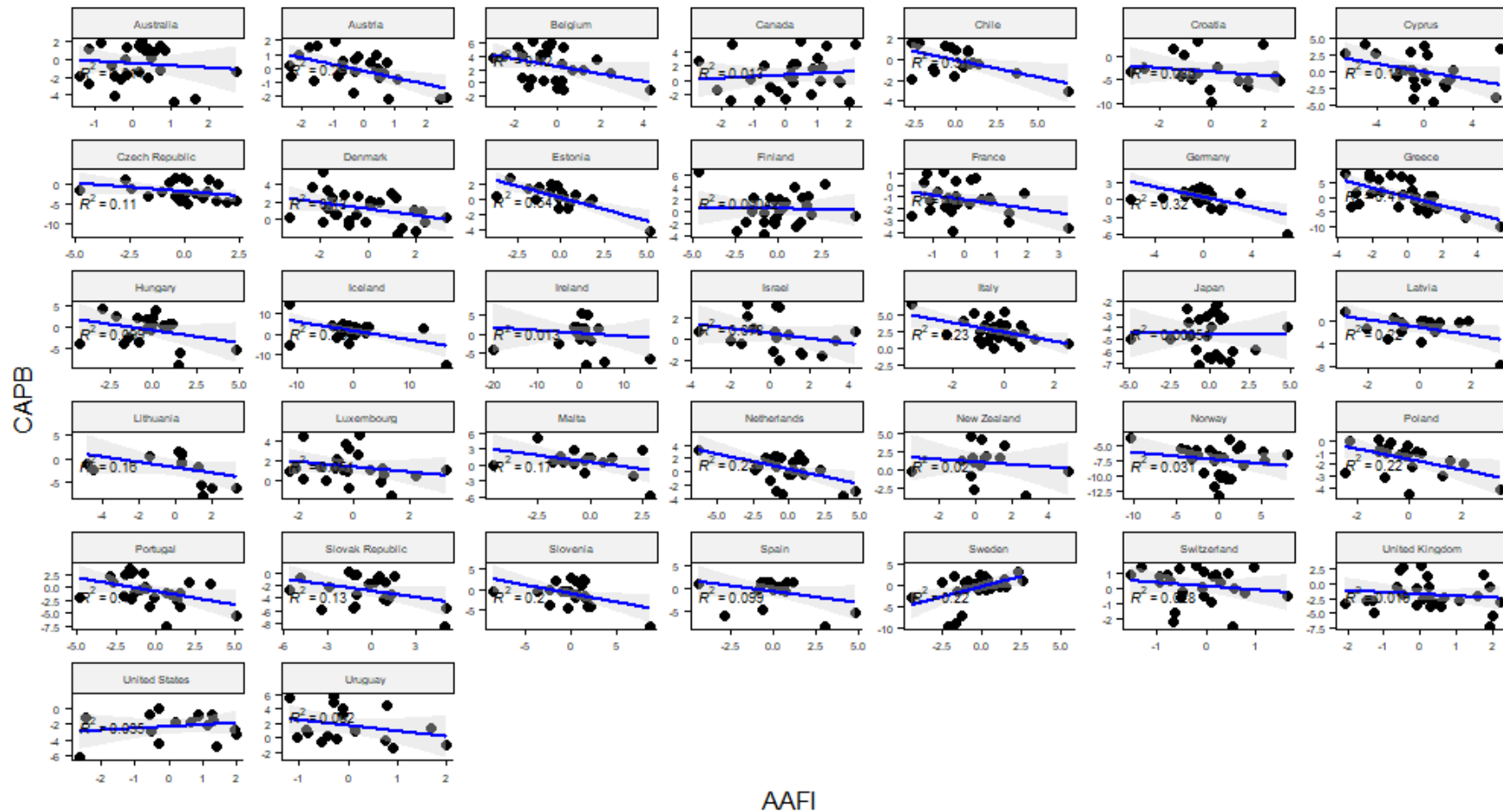


Figure S7.51 - Scatterplot of AAFI and Public Social Spending for each available year and country (1987-2020), linear regression line (with 95% confidence intervals) and R² value. Note low values indicate greater austerity

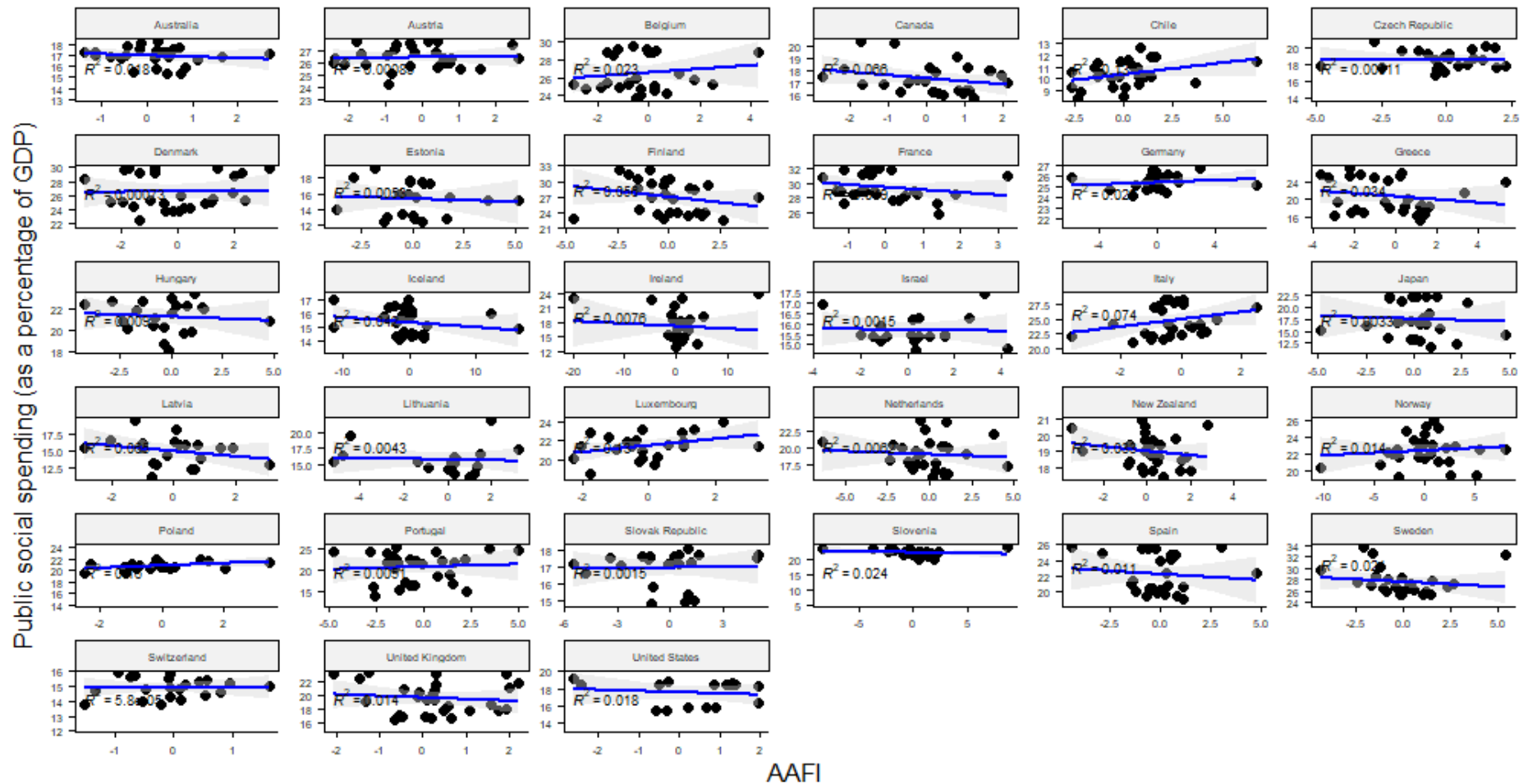
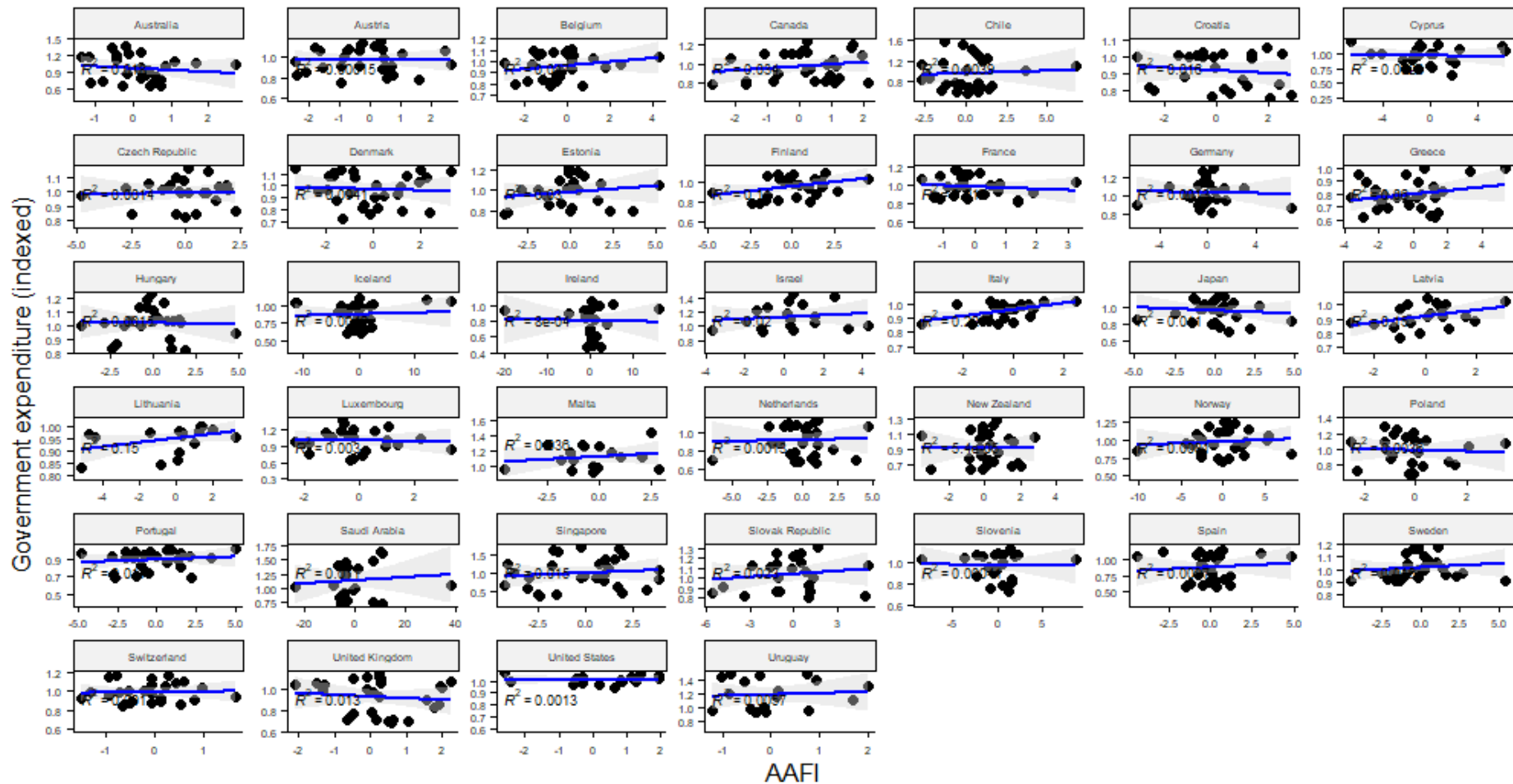


Figure S7.52 - Scatterplot of AAFI and indexed Government Expenditure for each available year and country (1987-2020), linear regression line (with 95% confidence intervals) and R² value. Note low values indicate greater austerity



Supplementary material for Chapter 8

Table S8.1 - Check statistics for model robustness (for model relationships between each austerity measure and total life expectancy, 2 year lag between exposure and outcome), fixed versus random effects

Austerity measure	F test		Hausman test	
	Statistic (p value)	Interpretation	Statistic (p value)	Interpretation
AAFI	26.5 (p = 0)	Fixed effects present	0.007 (p = 0.93)	No fixed effects
Government Expenditure	33.2 (p = 0)	Fixed effects present	6.6 (p = 0.01)	Fixed effects present
Public Social Spending	8.8 (p = 0)	Fixed effects present	22.5 (p = 2x10 ⁻⁶)	Fixed effects present
CAPB	8.3 (p = 0)	Fixed effects present	46.3 (p = 1x10 ⁻¹¹)	Fixed effects present

Table S8.2 - Check statistics for model robustness (for model relationships between each austerity measure and total life expectancy, 2 year lag between exposure and outcome), cross-sectional dependence

Austerity measure	Breusch-Pagan LM test		Pesaran CD test	
	Statistic (p value)	Interpretation	Statistic (p value)	Interpretation
AAFI	3489.9 (p < 2.2e-16)	Cross sectional dependence present	35.1 (p < 2.2e-16)	Cross sectional dependence present
Government Expenditure	2570.7 (p < 2.2e-16)	Cross sectional dependence present	16.0 (p < 2.2e-16)	Cross sectional dependence present
Public Social Spending	2252.3 (p < 2.2e-16)	Cross sectional dependence present	27.3 (p < 2.2e-16)	Cross sectional dependence present
CAPB	2797.9 (p < 2.2e-16)	Cross sectional dependence present	36.3 (p < 2.2e-16)	Cross sectional dependence present

Table S8.3 - Check statistics for model robustness (for model relationships between each austerity measure and total life expectancy, 2 year lag between exposure and outcome), check for serial correlation

Austerity measure	Breusch-Godfrey/Wooldridge test	
	Statistic (p value)	Interpretation
AAFI	341.8 (p < 2.2e-16)	Serial correlation exists
Government Expenditure	291.6 (p < 2.2e-16)	Serial correlation exists
Public Social Spending	239.3 (p < 2.2e-16)	Serial correlation exists
CAPB	266.7 (p < 2.2e-16)	Serial correlation exists

Table S8.4 - Check statistics for model robustness (for model relationships between each austerity measure and total life expectancy, 2 year lag between exposure and outcome), check for unit roots/stationarity

Austerity measure	Dickey-Fuller test	
	Statistic (p value)	Interpretation
Applies across all austerity measures	-6.2068 (p = 0.01)	Non-stationary/ no unit roots present

Table S8.5 - Check statistics for model robustness (for model relationships between each austerity measure and total life expectancy, 2 year lag between exposure and outcome), check for heteroscedasticity

Austerity measure	Breusch-Pagan test	
	Statistic (p value)	Interpretation
AAFI	0.95 p = 0.33	No heteroscedasticity present
Government Expenditure	1.5 (p = 0.22)	No heteroscedasticity present
Public Social Spending	63.0 (p = 2.056e-15)	Heteroscedasticity present
CAPB	27.9 (p = 1.272e-07)	Heteroscedasticity present

Table S8.6 - Robust covariance matrix estimation (Sandwich estimator) of standard errors to control for heteroscedasticity (for model relationships between Public Social Spending/CAPB and total life expectancy, 2 year lag between exposure and outcome)

		Original standard error (H ₀)	H ₁	H ₂	H ₃	H ₄
Assumptions/ data form		No heteroscedasticity	Small samples	Small samples	Small samples with less weight to influential observations	Small samples with influential observations
Austerity measure	Public Social Spending	0.04572277	0.04575719	0.04580554	0.0458888	0.04598595
	CAPB	0.05536675	0.05541373	0.05554813	0.05573308	0.05601229

Table S8.7 - Relationship between changes in AAFI and life expectancy, lifespan variation and all-age age-standardised mortality

Lag between exposure and outcome	0 years				2 years				5 years			
	Estimated change per unit AAFI increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit AAFI increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit AAFI increase	p value	lower 95% CI	Upper 95% CI
Life expectancy (total)	-0.003	0.918	-0.055	0.049	-0.038	0.219	-0.099	0.023	-0.035	0.322	-0.104	0.034
Life expectancy (females)	-0.002	0.912	-0.045	0.040	-0.031	0.221	-0.080	0.018	-0.033	0.239	-0.088	0.022
Life expectancy (males)	-0.003	0.930	-0.064	0.058	-0.045	0.227	-0.119	0.028	-0.038	0.373	-0.123	0.046
Lifespan variation (total)	-0.002	0.716	-0.015	0.010	0.010	0.193	-0.005	0.025	0.005	0.619	-0.013	0.022
Lifespan variation (females)	-0.004	0.487	-0.017	0.008	0.013	0.081	-0.002	0.027	0.006	0.452	-0.010	0.022
Lifespan variation (males)	0.000	0.950	-0.011	0.011	0.006	0.412	-0.008	0.020	0.002	0.816	-0.015	0.019
ASMR (total)	-0.049	0.984	-4.902	4.803	3.787	0.181	-1.751	9.324	3.370	0.288	-2.843	9.584
ASMR (females)	0.085	0.965	-3.705	3.874	3.079	0.161	-1.220	7.379	2.940	0.232	-1.874	7.754
ASMR (males)	-0.375	0.914	-7.170	6.419	5.071	0.207	-2.798	12.939	4.334	0.341	-4.574	13.241

Table S8.8 - Relationship between changes in AAFI and age stratified mortality (standardised within age strata)

Lag between exposure and outcome	0 years				2 years				5 years			
	Estimated change per unit AAFI increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit AAFI increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit AAFI increase	p value	lower 95% CI	Upper 95% CI
ASMR 0-1y (total)	0.000	0.815	-0.003	0.004	0.002	0.434	-0.002	0.006	0.003	0.258	-0.002	0.007
ASMR 0-1y (females)	0.000	0.808	-0.003	0.004	0.001	0.586	-0.003	0.005	0.002	0.256	-0.002	0.006
ASMR 0-1y (males)	0.000	0.832	-0.004	0.005	0.002	0.358	-0.002	0.007	0.003	0.281	-0.002	0.008
ASMR 1-14y (total)	0.006	0.611	-0.017	0.029	0.015	0.220	-0.009	0.039	0.002	0.856	-0.023	0.028
ASMR 1-14y (females)	0.000	0.999	-0.021	0.021	0.009	0.425	-0.013	0.031	0.002	0.837	-0.020	0.025
ASMR 1-14y (males)	0.012	0.420	-0.017	0.040	0.021	0.158	-0.008	0.050	0.002	0.897	-0.029	0.033
ASMR 15-29y (total)	0.015	0.701	-0.062	0.093	0.068	0.112	-0.016	0.152	0.028	0.553	-0.064	0.120
ASMR 15-29y (females)	-0.016	0.360	-0.050	0.018	0.023	0.220	-0.014	0.060	0.021	0.281	-0.017	0.060
ASMR 15-29y (males)	0.045	0.487	-0.081	0.170	0.111	0.108	-0.024	0.246	0.034	0.658	-0.116	0.183
ASMR 30-49y (total)	0.005	0.978	-0.325	0.335	0.221	0.281	-0.180	0.622	0.076	0.755	-0.398	0.550
ASMR 30-49y (females)	0.030	0.742	-0.147	0.207	0.112	0.278	-0.090	0.315	0.098	0.402	-0.131	0.328
ASMR 30-49y (males)	-0.021	0.934	-0.524	0.481	0.334	0.295	-0.290	0.957	0.053	0.889	-0.695	0.802
ASMR 50-69y (total)	0.030	0.957	-1.068	1.128	0.868	0.237	-0.569	2.305	0.439	0.623	-1.309	2.188
ASMR 50-69y (females)	0.053	0.878	-0.617	0.722	0.567	0.182	-0.265	1.398	0.241	0.632	-0.743	1.225
ASMR 50-69y (males)	-0.015	0.986	-1.692	1.663	1.240	0.292	-1.063	3.544	0.682	0.642	-2.190	3.553
ASMR 70+y (total)	-0.110	0.950	-3.516	3.296	2.599	0.175	-1.148	6.345	2.800	0.180	-1.284	6.884
ASMR 70+y (females)	0.014	0.993	-2.931	2.959	2.358	0.161	-0.938	5.654	2.554	0.171	-1.101	6.209
ASMR 70+y (males)	-0.400	0.866	-5.056	4.257	3.343	0.199	-1.747	8.432	3.535	0.208	-1.964	9.034

Figure S8.1 - Relationship between AAFI and mortality (for all ages combined and specific age strata), with no time lag between exposure and outcome

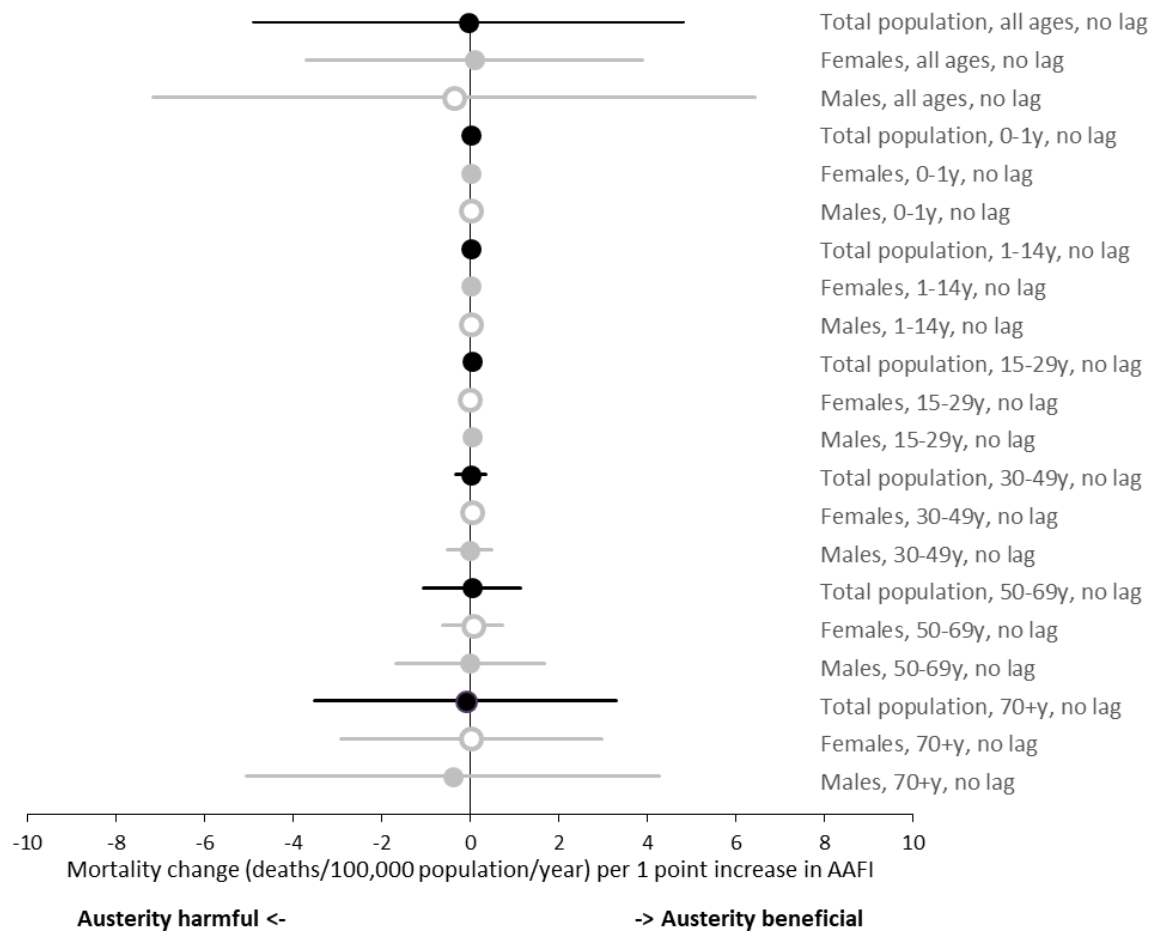


Figure S8.2 - Relationship between AAFI and mortality (for all ages combined and specific age strata), with 2 year time lag between exposure and outcome

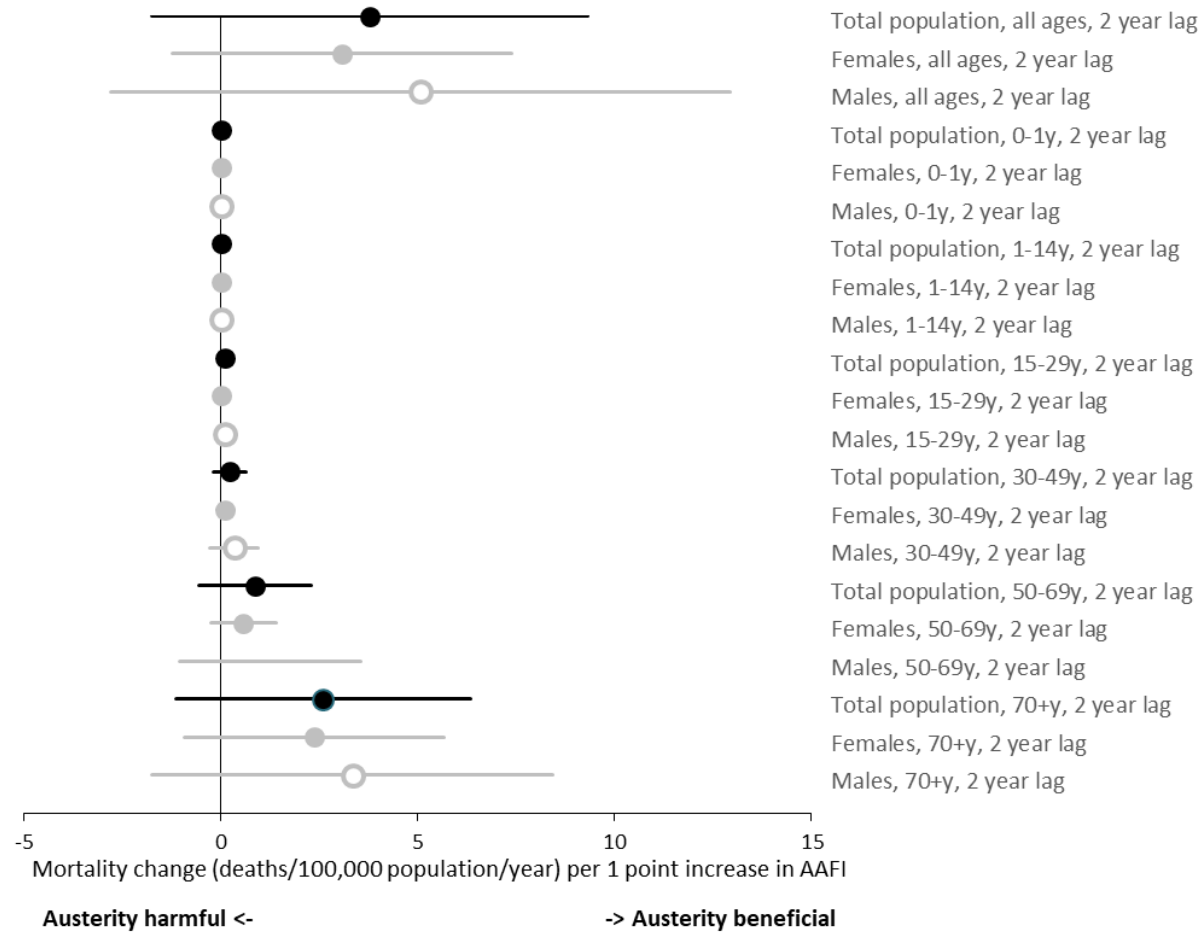


Figure S8.3 - Relationship between AAFI and mortality (for all ages combined and specific age strata), with 5 year time lag between exposure and outcome

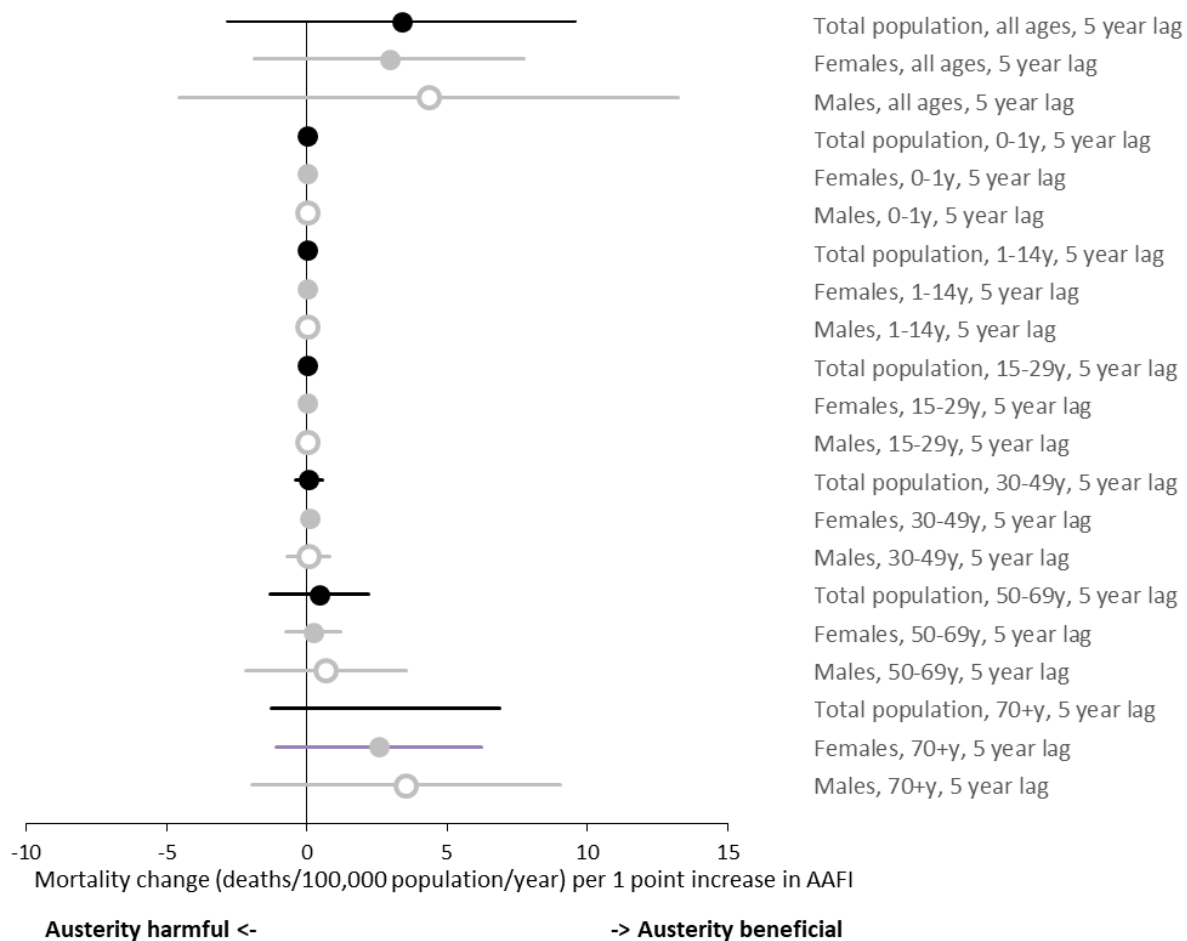


Table S8.9 - Relationship between changes in indexed real Government Expenditure per capita and life expectancy, lifespan variation and all-age age-standardised mortality

Lag between exposure and outcome	0 years				2 years				5 years			
	Estimated change per unit increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit increase	p value	lower 95% CI	Upper 95% CI
Life expectancy (total)	0.114	0.00	0.109	0.119	0.064	0.00	0.053	0.074	0.003	0.66	-0.011	0.017
Life expectancy (females)	0.094	0.00	0.090	0.098	0.052	0.00	0.044	0.061	0.002	0.72	-0.009	0.013
Life expectancy (males)	0.133	0.00	0.127	0.139	0.074	0.00	0.061	0.088	0.004	0.64	-0.013	0.021
Lifespan variation (total)	-0.024	0.00	-0.026	-0.023	-0.013	0.00	-0.015	-0.010	0.000	0.98	-0.003	0.004
Lifespan variation (females)	-0.024	0.00	-0.026	-0.023	-0.013	0.00	-0.015	-0.010	-0.001	0.63	-0.004	0.002
Lifespan variation (males)	-0.018	0.00	-0.020	-0.017	-0.009	0.00	-0.012	-0.007	0.001	0.67	-0.003	0.004
ASMR (total)	-10.5	0.00	-11.0	-10.1	-6.0	0.00	-7.0	-5.0	-0.3	0.61	-1.6	0.9
ASMR (females)	-8.2	0.00	-8.5	-7.8	-4.6	0.00	-5.4	-3.8	-0.2	0.68	-1.2	0.8
ASMR (males)	-14.8	0.00	-15.4	-14.1	-8.4	0.00	-9.8	-7.0	-0.5	0.60	-2.2	1.3

Table S8.10 - Relationship between changes in indexed real Government Expenditure per capita and age stratified mortality (standardised within age strata)

Lag between exposure and outcome	0 years				2 years				5 years			
	Estimated change per unit increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit increase	p value	lower 95% CI	Upper 95% CI
ASMR 0-1y (total)	0.0	0.00	0.0	0.0	0.0	0.00	0.0	0.0	0.0	0.28	0.0	0.0
ASMR 0-1y (females)	0.0	0.00	0.0	0.0	0.0	0.00	0.0	0.0	0.0	0.30	0.0	0.0
ASMR 0-1y (males)	0.0	0.00	0.0	0.0	0.0	0.00	0.0	0.0	0.0	0.28	0.0	0.0
ASMR 1-14y (total)	0.0	0.00	0.0	0.0	0.0	0.00	0.0	0.0	0.0	0.56	0.0	0.0
ASMR 1-14y (females)	0.0	0.00	0.0	0.0	0.0	0.00	0.0	0.0	0.0	0.85	0.0	0.0
ASMR 1-14y (males)	0.0	0.00	0.0	0.0	0.0	0.00	-0.1	0.0	0.0	0.43	0.0	0.0
ASMR 15-29y (total)	-0.1	0.00	-0.1	-0.1	-0.1	0.00	-0.1	-0.1	0.0	0.49	0.0	0.0
ASMR 15-29y (females)	-0.1	0.00	-0.1	-0.1	0.0	0.00	-0.1	-0.1	0.0	0.47	0.0	0.0
ASMR 15-29y (males)	-0.2	0.00	-0.1	-0.1	-0.1	0.00	-0.2	-0.2	0.0	0.51	0.0	0.0
ASMR 30-49y (total)	-0.6	0.00	-0.6	-0.5	-0.3	0.00	-0.4	-0.2	0.0	0.60	-0.1	0.1
ASMR 30-49y (females)	-0.3	0.00	-0.3	-0.3	-0.2	0.00	-0.2	-0.1	0.0	0.90	0.0	0.0
ASMR 30-49y (males)	-0.8	0.00	-0.9	-0.7	-0.4	0.00	-0.5	-0.3	-0.1	0.50	-0.2	0.1
ASMR 50-69y (total)	-2.3	0.00	-2.4	-2.2	-1.3	0.00	-1.6	-1.1	-0.2	0.16	-0.6	0.1
ASMR 50-69y (females)	-1.4	0.00	-1.4	-1.3	-0.8	0.00	-0.9	-0.6	-0.1	0.22	-0.3	0.1
ASMR 50-69y (males)	-3.4	0.00	-3.6	-3.2	-2.0	0.00	-2.5	-1.6	-0.4	0.13	-1.0	0.1
ASMR 70+y (total)	-7.5	0.00	-7.8	-7.1	-4.2	0.00	-4.9	-3.5	0.0	0.91	-0.9	0.8
ASMR 70+y (females)	-6.3	0.00	-6.6	-6.0	-3.5	0.00	-4.1	-3.0	-0.1	0.84	-0.8	0.7
ASMR 70+y (males)	-10.2	0.00	-10.7	-9.8	-5.7	0.00	-6.6	-4.8	0.0	0.98	-1.1	1.1

Figure S8.4 - Relationship between indexed real per capita Government Expenditure and mortality (for all ages combined and specific age strata), with no time lag between exposure and outcome

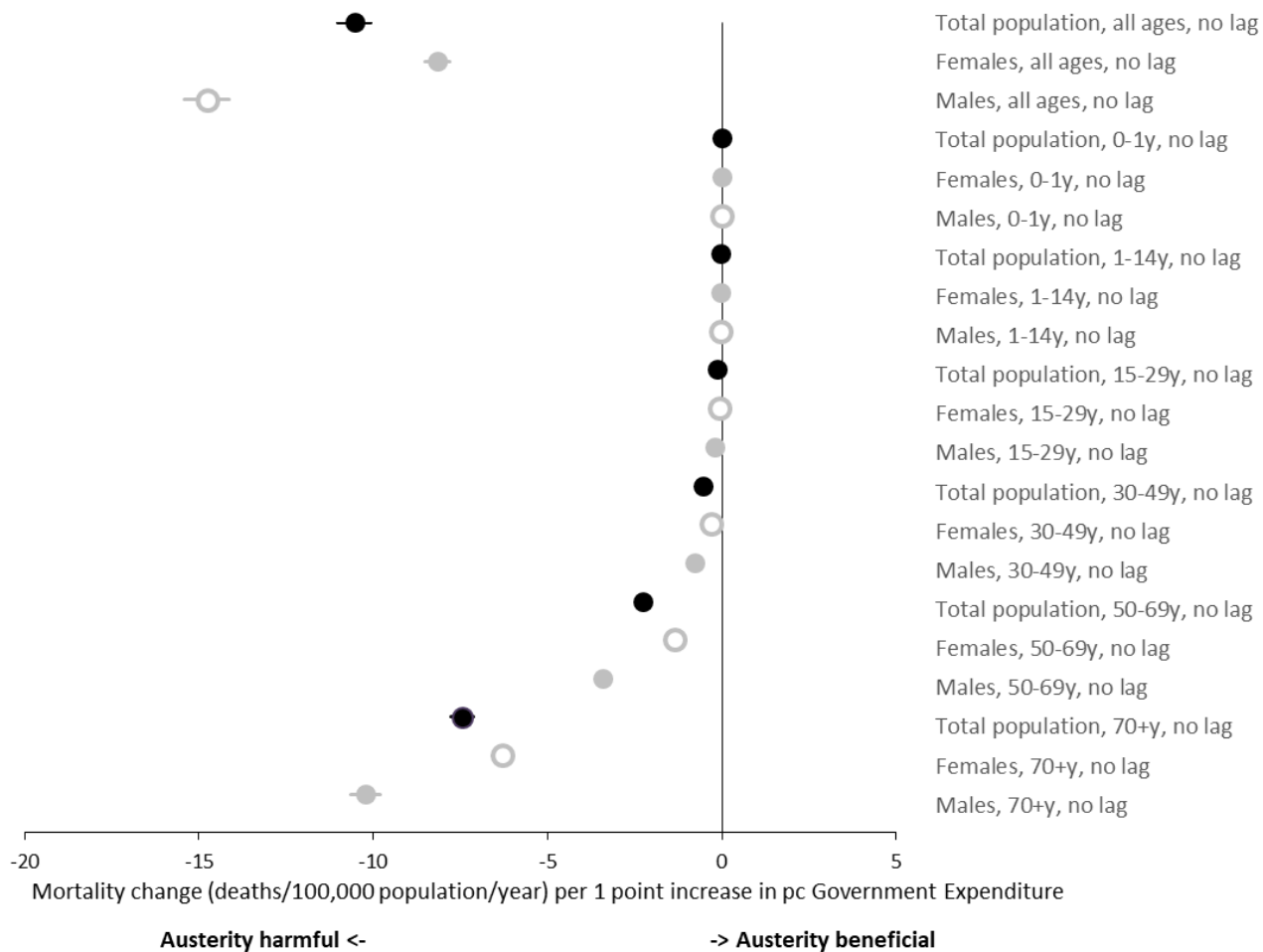


Figure S8.5 - Relationship between indexed real per capita Government Expenditure and mortality (for all ages combined and specific age strata), with 2 year time lag between exposure and outcome

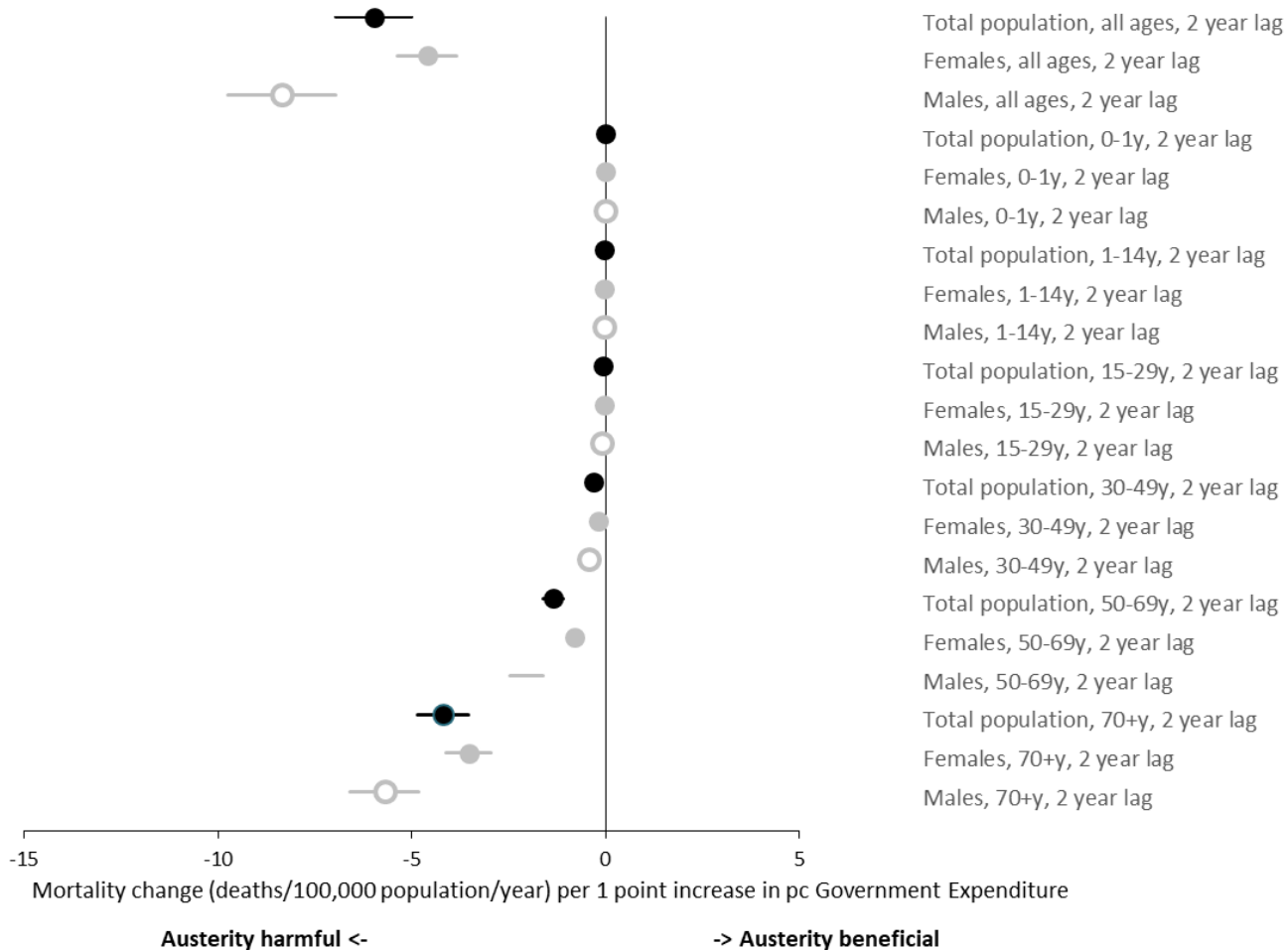


Figure S8.6 - Relationship between indexed real per capita Government Expenditure and mortality (for all ages combined and specific age strata), with 5 year time lag between exposure and outcome

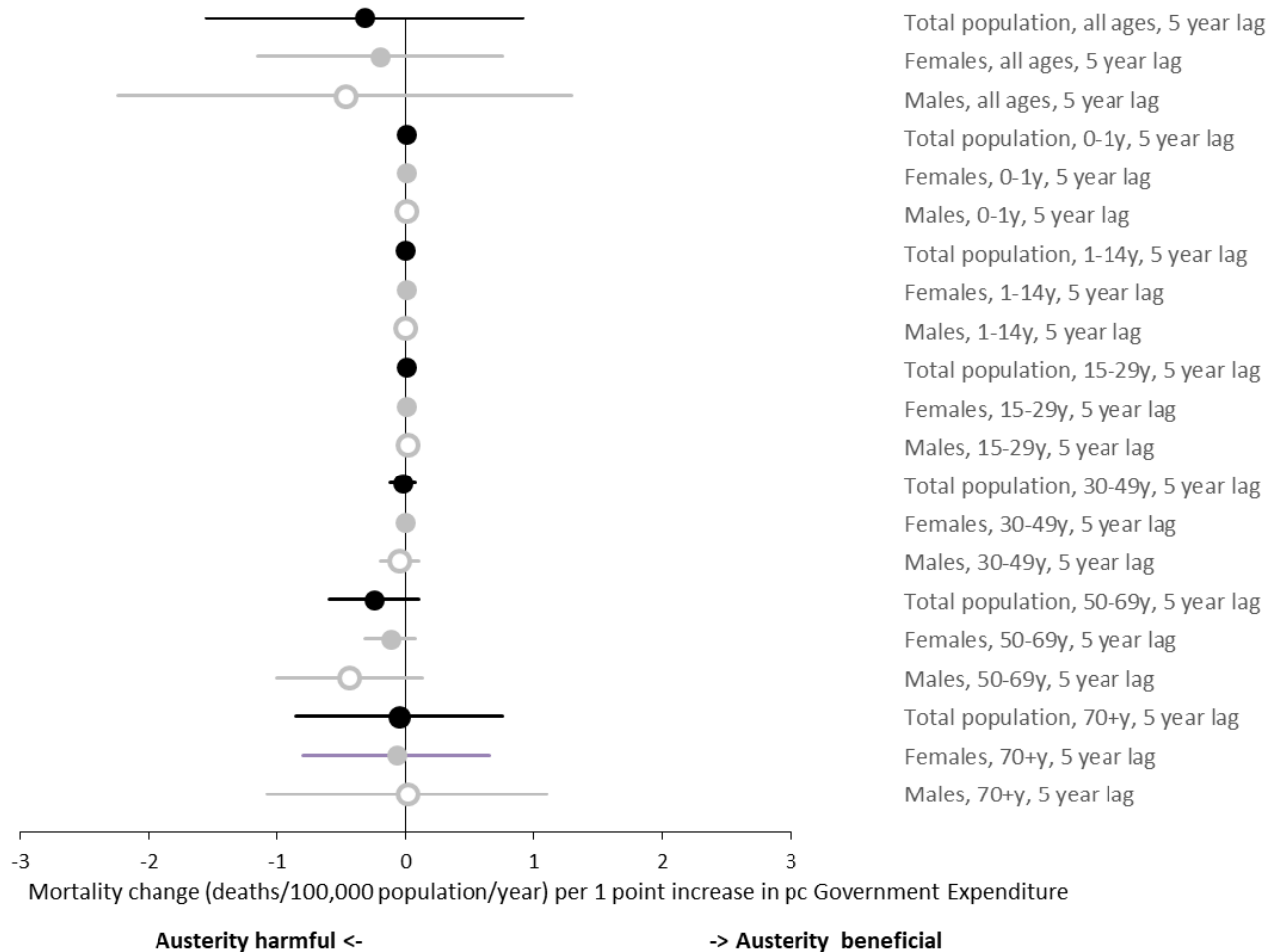


Table S8.11 - Relationship between changes in Public Social Spending and life expectancy, lifespan variation and all-age age-standardised mortality

Lag between exposure and outcome	0 years				2 years				5 years			
	Estimated change per unit increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit increase	p value	lower 95% CI	Upper 95% CI
Life expectancy (total)	0.366	0.000	0.313	0.419	0.275	0.000	0.208	0.342	0.079	0.052	-0.001	0.158
Life expectancy (females)	0.312	0.000	0.268	0.355	0.251	0.000	0.198	0.305	0.099	0.002	0.035	0.162
Life expectancy (males)	0.415	0.000	0.352	0.478	0.296	0.000	0.214	0.377	0.056	0.260	-0.001	0.037
Lifespan variation (total)	-0.085	0.000	-0.097	-0.072	-0.051	0.000	-0.068	-0.034	0.006	0.592	-0.015	0.026
Lifespan variation (females)	-0.085	0.000	-0.098	-0.073	-0.063	0.000	-0.079	-0.048	-0.012	0.186	-0.031	0.006
Lifespan variation (males)	-0.068	0.000	-0.079	-0.056	-0.031	0.000	-0.048	-0.015	0.018	0.067	-0.048	-0.015
ASMR (total)	-32.769	0.000	-37.772	-27.767	-26.657	0.000	-32.733	-20.582	-10.121	0.006	-17.277	-2.966
ASMR (females)	-26.267	0.000	-30.146	-22.388	-22.215	0.000	-26.891	-17.539	-9.891	0.000	-15.415	-4.366
ASMR (males)	-43.895	0.000	-50.976	-36.813	-34.396	0.000	-43.116	-25.676	-10.327	0.050	-20.615	-0.040

Table S8.12 - Relationship between changes in Public Social Spending and age stratified mortality (standardised within age strata)

Lag between exposure and outcome	0 years				2 years				5 years			
	Estimated change per unit increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit increase	p value	lower 95% CI	Upper 95% CI
ASMR 0-1y (total)	-0.024	0.000	-0.028	-0.020	-0.019	0.000	-0.023	-0.015	-0.007	0.007	-0.012	-0.002
ASMR 0-1y (females)	-0.021	< 2.2e-16	-0.024	-0.017	-0.017	0.000	-0.021	-0.013	-0.006	0.019	-0.010	-0.001
ASMR 0-1y (males)	-0.027	< 2.2e-16	-0.032	-0.023	-0.021	0.000	-0.026	-0.016	-0.008	0.005	-0.014	-0.003
ASMR 1-14y (total)	-0.147	< 2.2e-16	-0.171	-0.123	-0.105	0.000	-0.132	-0.079	-0.024	0.113	-0.053	0.006
ASMR 1-14y (females)	-0.118	< 2.2e-16	-0.141	-0.096	-0.085	0.000	-0.110	-0.061	-0.017	0.213	-0.043	0.010
ASMR 1-14y (males)	-0.175	< 2.2e-16	-0.204	-0.145	-0.125	0.000	-0.157	-0.092	-0.030	0.097	-0.065	0.005
ASMR 15-29y (total)	-0.568	< 2.2e-16	-0.647	-0.490	-0.311	0.000	-0.406	-0.217	0.028	0.610	-0.079	0.135
ASMR 15-29y (females)	-0.214	< 2.2e-16	-0.250	-0.178	-0.121	0.000	-0.163	-0.080	0.025	0.281	-0.020	0.070
ASMR 15-29y (males)	-0.911	< 2.2e-16	-1.038	-0.784	-0.495	0.000	-0.647	-0.343	0.030	0.732	-0.143	0.203
ASMR 30-49y (total)	-1.854	< 2.2e-16	-2.207	-1.501	-0.984	0.000	-1.442	-0.526	0.184	0.512	-0.365	0.732
ASMR 30-49y (females)	-1.011	< 2.2e-16	-1.200	-0.822	-0.604	0.000	-0.834	-0.374	0.008	0.951	-0.257	0.274
ASMR 30-49y (males)	-2.737	< 2.2e-16	-3.279	-2.196	-1.369	0.000	-2.084	-0.655	0.401	0.365	-0.465	1.267
ASMR 50-69y (total)	-6.746	< 2.2e-16	-7.903	-5.589	-5.081	0.000	-6.699	-3.463	-1.682	0.103	-3.703	0.338
ASMR 50-69y (females)	-4.349	< 2.2e-16	-5.046	-3.652	-3.595	0.000	-4.518	-2.673	-1.746	0.003	-2.877	-0.615
ASMR 50-69y (males)	-9.661	< 2.2e-16	-11.451	-7.871	-6.769	0.000	-9.386	-4.152	-1.330	0.433	-4.653	1.993
ASMR 70+y (total)	-23.214	< 2.2e-16	-26.717	-19.711	-19.986	< 2.2e-16	-24.042	-15.930	-8.557	0.000	-13.244	-3.870
ASMR 70+y (females)	-20.369	< 2.2e-16	-23.386	-17.352	-17.640	< 2.2e-16	-21.206	-14.074	-8.105	0.000	-12.295	-3.915
ASMR 70+y (males)	-30.140	< 2.2e-16	-34.991	-25.289	-25.429	< 2.2e-16	-30.988	-19.871	-9.315	0.004	-15.647	-2.983

Figure S8.7 - Relationship between Public Social Spending and mortality (for all ages combined and specific age strata), with no time lag between exposure and outcome

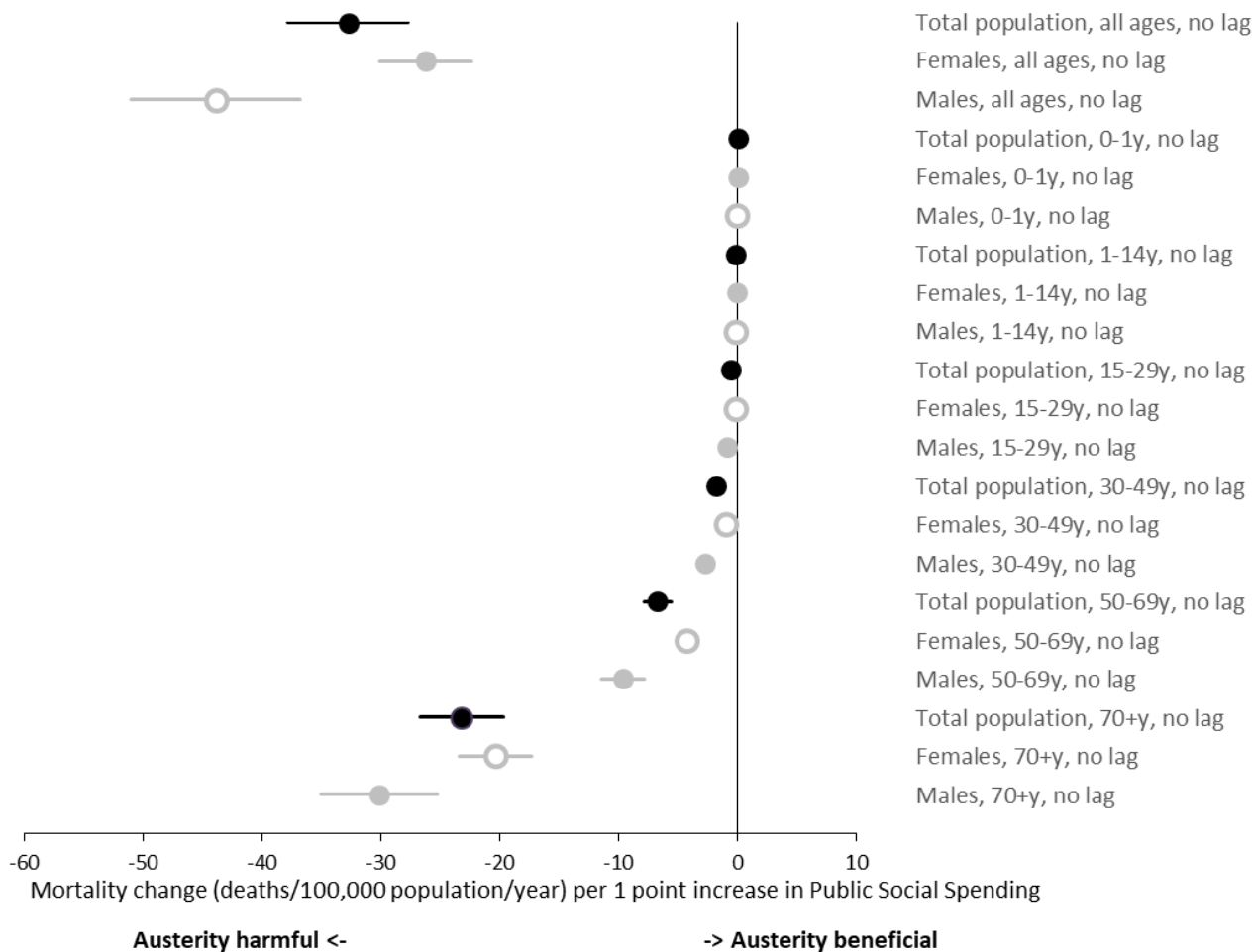


Figure S8.8 - Relationship between Public Social Spending and mortality (for all ages combined and specific age strata), with 2 year time lag between exposure and outcome

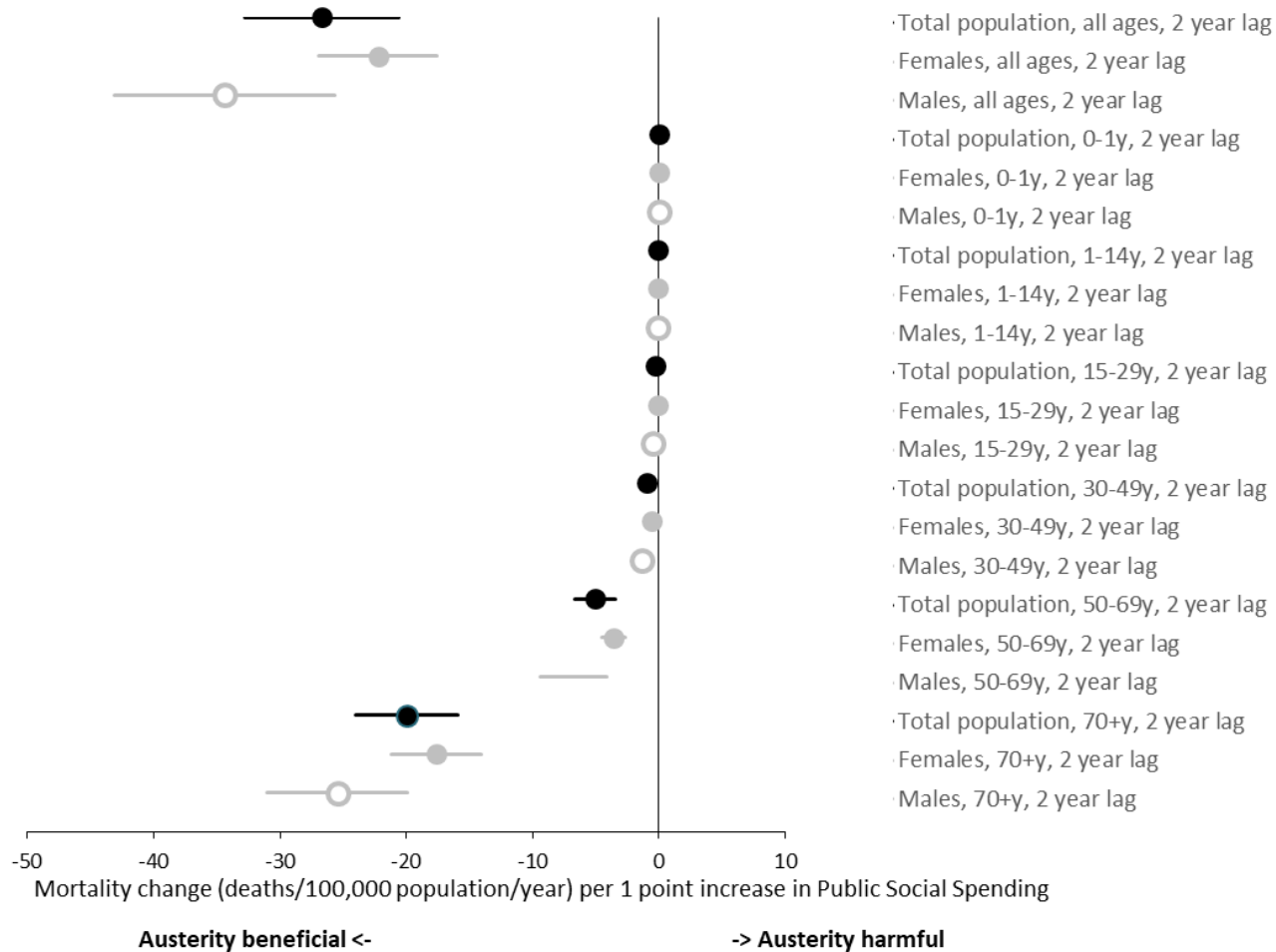


Figure S8.9 - Relationship between Public Social Spending and mortality (for all ages combined and specific age strata), with 5 year time lag between exposure and outcome

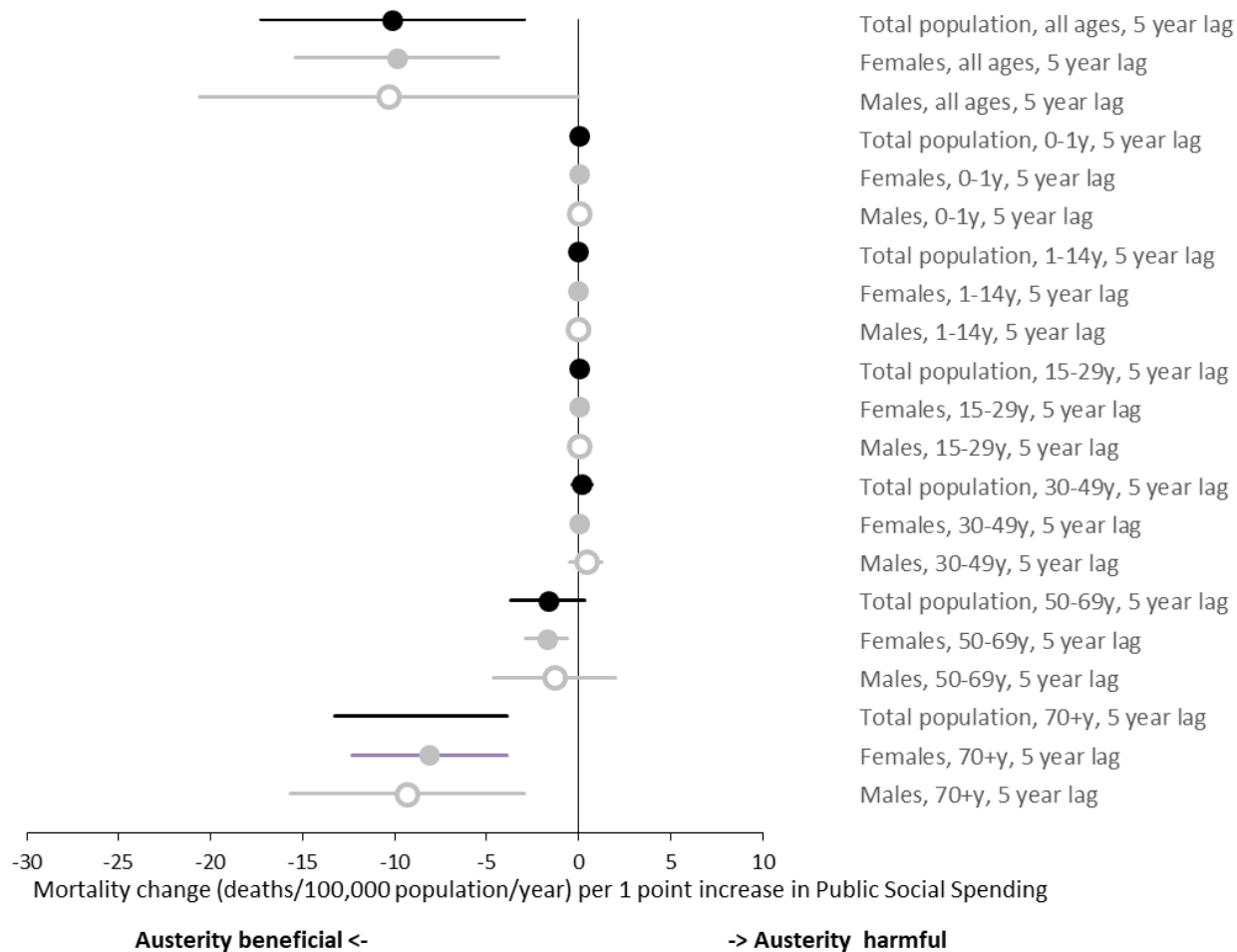


Table S8.13 - Relationship between changes in CAPB and life expectancy, lifespan variation and all-age age-standardised mortality

Lag between exposure and outcome	0 years				2 years				5 years			
	Estimated change per unit increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit increase	p value	lower 95% CI	Upper 95% CI
Life expectancy (total)	-0.061	0.012	-0.109	-0.014	-0.063	0.027	-0.119	-0.007	-0.027	0.402	-0.090	0.036
Life expectancy (females)	-0.058	0.004	-0.097	-0.019	-0.062	0.008	-0.106	-0.017	-0.027	0.300	-0.077	0.024
Life expectancy (males)	-0.067	0.020	-0.123	-0.011	-0.067	0.052	-0.134	0.001	-0.026	0.512	-0.103	0.052
Lifespan variation (total)	0.013	0.029	0.001	0.024	0.006	0.379	-0.008	0.020	-0.003	0.703	-0.020	0.013
Lifespan variation (females)	0.018	0.002	0.007	0.029	0.014	0.030	0.001	0.027	0.002	0.747	-0.012	0.017
Lifespan variation (males)	0.006	0.222	-0.004	0.016	-0.004	0.531	-0.017	0.009	-0.010	0.182	-0.026	0.005
ASMR (total)	5.742	0.011	1.310	10.174	6.336	0.015	1.269	11.403	3.687	0.206	-2.022	9.395
ASMR (females)	4.619	0.009	1.159	8.079	5.025	0.013	1.091	8.959	2.980	0.187	-1.443	7.403
ASMR (males)	8.018	0.012	1.812	14.224	8.992	0.015	1.794	16.191	5.052	0.227	-3.132	13.235

Table S8.14 - Relationship between changes in CAPB and age stratified mortality (standardised within age strata)

Lag between exposure and outcome	0 years				2 years				5 years			
	Estimated change per unit increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit increase	p value	lower 95% CI	upper 95% CI	Estimated change per unit increase	p value	lower 95% CI	Upper 95% CI
ASMR 0-1y (total)	0.003	0.046	0.000	0.007	0.002	0.263	-0.002	0.006	-0.003	0.163	-0.007	0.001
ASMR 0-1y (females)	0.003	0.063	0.000	0.006	0.002	0.155	-0.001	0.006	-0.003	0.128	-0.007	0.001
ASMR 0-1y (males)	0.004	0.048	0.000	0.008	0.002	0.410	-0.002	0.006	-0.003	0.214	-0.008	0.002
ASMR 1-14y (total)	0.017	0.119	-0.004	0.038	0.006	0.568	-0.016	0.029	-0.006	0.605	-0.029	0.017
ASMR 1-14y (females)	0.020	0.040	0.001	0.040	0.011	0.300	-0.009	0.031	-0.007	0.487	-0.028	0.014
ASMR 1-14y (males)	0.014	0.302	-0.012	0.039	0.003	0.854	-0.024	0.029	-0.005	0.730	-0.033	0.023
ASMR 15-29y (total)	0.082	0.025	0.011	0.153	0.021	0.589	-0.056	0.098	-0.045	0.300	-0.130	0.040
ASMR 15-29y (females)	0.041	0.011	0.009	0.072	0.025	0.140	-0.008	0.059	-0.038	0.035	-0.074	-0.003
ASMR 15-29y (males)	0.121	0.039	0.007	0.236	0.017	0.790	-0.107	0.141	-0.052	0.454	-0.190	0.085
ASMR 30-49y (total)	-0.168	0.278	-0.470	0.135	-0.255	0.174	-0.623	0.112	-0.062	0.779	-0.498	0.373
ASMR 30-49y (females)	-0.019	0.816	-0.182	0.143	-0.028	0.769	-0.214	0.158	-0.058	0.589	-0.269	0.153
ASMR 30-49y (males)	-0.328	0.164	-0.788	0.133	-0.509	0.081	-1.081	0.062	-0.085	0.808	-0.773	0.602
ASMR 50-69y (total)	0.758	0.140	-0.248	1.765	0.928	0.168	-0.390	2.247	1.190	0.147	-0.415	2.795
ASMR 50-69y (females)	0.706	0.024	0.094	1.318	0.751	0.054	-0.011	1.514	0.904	0.050	0.002	1.806
ASMR 50-69y (males)	0.618	0.431	-0.921	2.158	0.942	0.383	-1.173	3.058	1.551	0.250	-1.086	4.188
ASMR 70+y (total)	5.018	0.002	1.916	8.120	5.614	0.001	2.197	9.031	2.639	0.169	-1.114	6.392
ASMR 70+y (females)	3.842	0.005	1.155	6.530	4.241	0.006	1.228	7.253	2.209	0.198	-1.151	5.569
ASMR 70+y (males)	7.553	0.001	3.319	11.787	8.522	0.000	3.891	13.153	3.673	0.155	-1.380	8.725

Figure S8.10 - Relationship between CAPB and mortality (for all ages combined and specific age strata), with no time lag between exposure and outcome

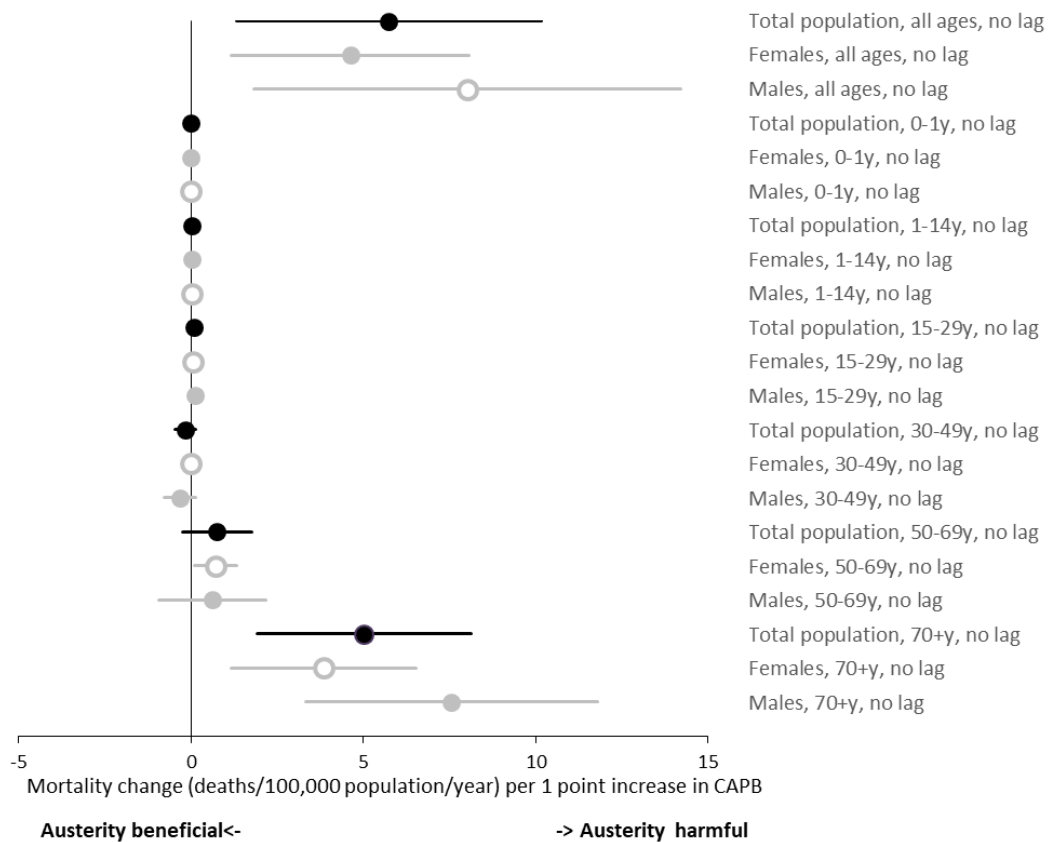


Figure S8.11 - Relationship between CAPB and mortality (for all ages combined and specific age strata), with 2 year time lag between exposure and outcome

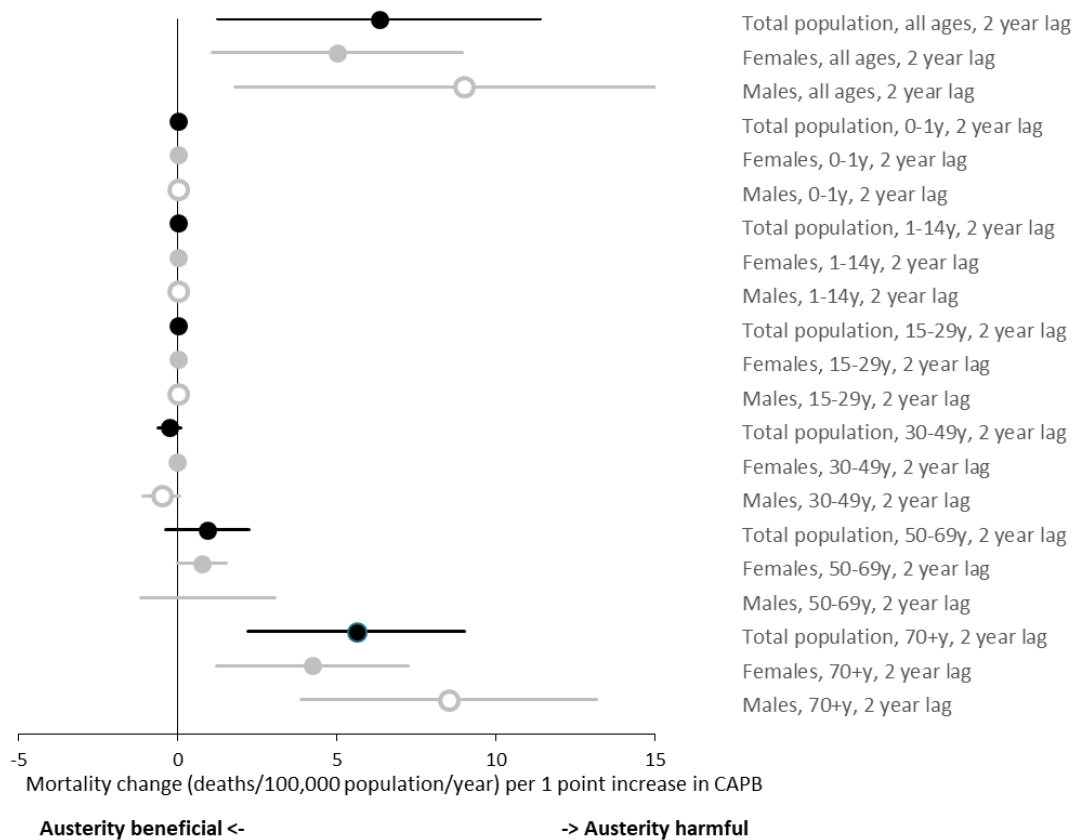


Figure S8.12 - Relationship between CAPB and mortality (for all ages combined and specific age strata), with 5 year time lag between exposure and outcome

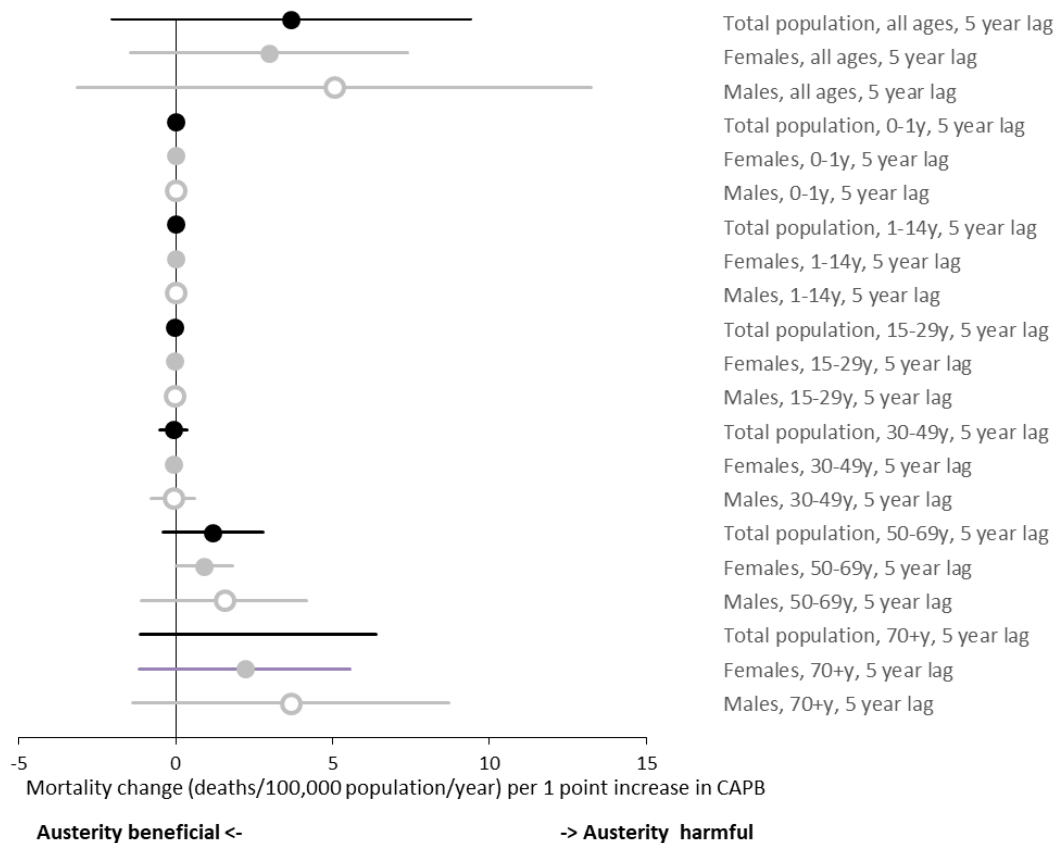


Table S8.15 - Relationship between all measures of austerity and life expectancy, adjusted for baseline mean household incomes

	No lag				2 year lag				5 year lag			
	Estimated change per unit increase	p value	lower CI	upper CI	Estimated change per unit increase	p value	lower CI	upper CI	Estimated change per unit increase	p value	lower CI	upper CI
AAFI												
Life expectancy (total)	-0.02	0.51	-0.09	0.04	-0.04	0.31	-0.12	0.04	-0.01	0.74	-0.10	0.07
Life expectancy (females)	-0.02	0.53	-0.07	0.04	-0.03	0.35	-0.09	0.03	-0.01	0.76	-0.08	0.06
Life expectancy (males)	-0.03	0.53	-0.11	0.06	-0.05	0.31	-0.15	0.05	-0.02	0.71	-0.13	0.09
Government Expenditure												
Life expectancy (total)	0.71	0.52	-1.43	2.85	0.24	0.86	-2.33	2.80	1.15	0.43	-1.72	4.01
Life expectancy (females)	1.20	0.19	-0.59	2.98	0.67	0.53	-1.42	2.76	1.42	0.22	-0.86	3.71
Life expectancy (males)	0.00	1.00	-2.69	2.69	-0.40	0.81	-3.62	2.81	0.74	0.69	-2.87	4.34
Public Social Spending												
Life expectancy (total)	0.07	0.00	0.04	0.10	0.04	0.02	0.01	0.08	0.01	0.61	-0.03	0.05
Life expectancy (females)	0.06	0.00	0.04	0.09	0.05	0.00	0.02	0.08	0.03	0.10	-0.01	0.06
Life expectancy (males)	0.07	0.00	0.03	0.11	0.04	0.10	-0.01	0.08	-0.01	0.78	-0.06	0.04
CAPB												
Life expectancy (total)	0.06	0.01	0.01	0.11	0.04	0.19	-0.02	0.09	-0.02	0.59	-0.08	0.04
Life expectancy (females)	0.02	0.44	-0.02	0.05	0.00	0.99	-0.05	0.04	-0.04	0.13	-0.09	0.01
Life expectancy (males)	0.10	0.00	0.04	0.16	0.07	0.05	0.00	0.14	0.00	0.93	-0.07	0.08

Figure S8.13 - Relationship between AAFI and life expectancy, for 0, 2 and 5 year lags between exposure and outcome, adjusted for mean baseline household incomes

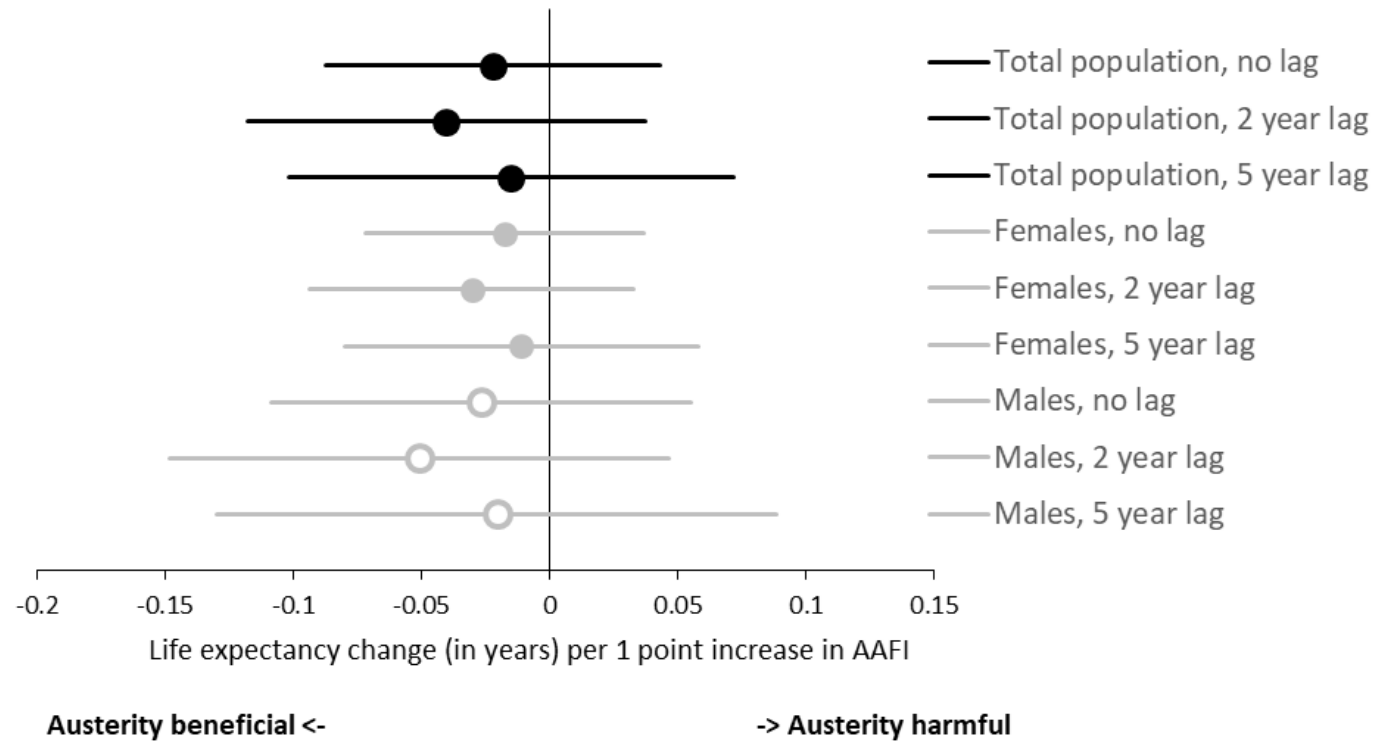


Figure S8.14 - Relationship between indexed Government Expenditure and life expectancy, for 0, 2 and 5 year lags between exposure and outcome, adjusted for mean baseline household incomes

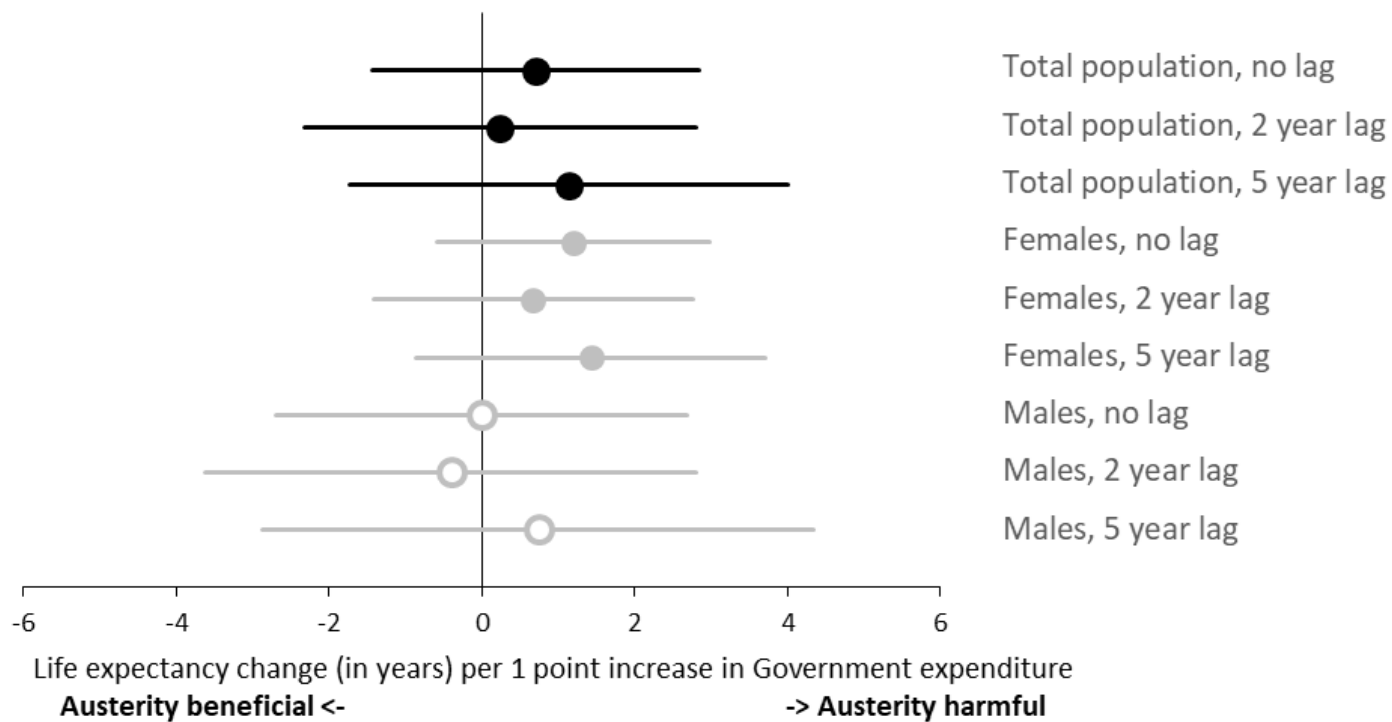


Figure S8.16 - Relationship between CAPB and life expectancy, for 0, 2 and 5 year lags between exposure and outcome, adjusted for mean baseline household incomes

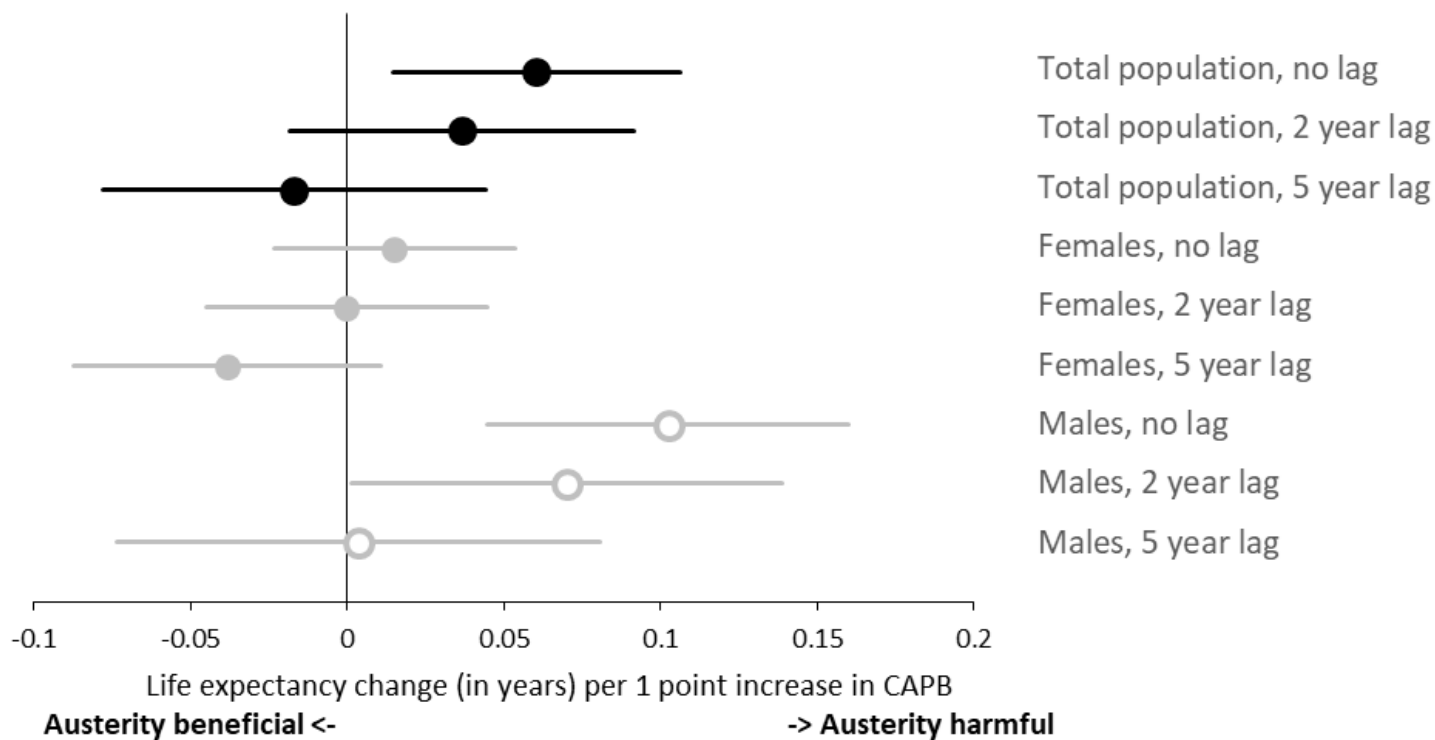


Table S8.16 - Relationship between all measures of austerity and life expectancy, adjusted for baseline real GDP per capita

	No lag				2 year lag				5 year lag			
	Estimated change per unit increase	p value	lower CI	upper CI	Estimated change per unit increase	p value	lower CI	upper CI	Estimated change per unit increase	p value	lower CI	upper CI
AAFI												
Life expectancy (total)	-0.006	0.882	-0.080	0.068	-0.027	0.514	-0.109	0.055	-0.006	0.903	-0.094	0.083
Life expectancy (females)	-0.006	0.854	-0.066	0.055	-0.021	0.530	-0.088	0.045	-0.004	0.901	-0.075	0.066
Life expectancy (males)	-0.005	0.911	-0.098	0.088	-0.034	0.519	-0.137	0.069	-0.008	0.886	-0.120	0.103
Government Expenditure												
Life expectancy (total)	2.070	0.094	-0.352	4.492	1.241	0.369	-1.463	3.945	1.856	0.214	-1.067	4.778
Life expectancy (females)	2.279	0.023	0.312	4.246	1.476	0.185	-0.705	3.657	1.987	0.094	-0.334	4.308
Life expectancy (males)	1.618	0.298	-1.429	4.664	0.789	0.648	-2.598	4.176	1.585	0.399	-2.095	5.264
Public Social Spending												
Life expectancy (total)	0.075	0.000	0.042	0.108	0.048	0.010	0.012	0.085	0.014	0.485	-0.026	0.054
Life expectancy (females)	0.070	0.000	0.044	0.097	0.053	0.001	0.023	0.082	0.029	0.068	-0.002	0.061
Life expectancy (males)	0.079	0.000	0.038	0.120	0.043	0.065	-0.003	0.090	-0.002	0.923	-0.053	0.048
CAPB												
Life expectancy (total)	0.058	0.031	0.005	0.110	0.034	0.252	-0.024	0.092	-0.019	0.557	-0.082	0.044
Life expectancy (females)	0.013	0.543	-0.029	0.056	-0.002	0.935	-0.049	0.045	-0.040	0.120	-0.089	0.010
Life expectancy (males)	0.099	0.003	0.033	0.164	0.067	0.072	-0.006	0.139	0.001	0.983	-0.078	0.080

Figure S8.17 - Relationship between AAFI and life expectancy, for 0, 2 and 5 year lags between exposure and outcome, adjusted for baseline real GDP per capita

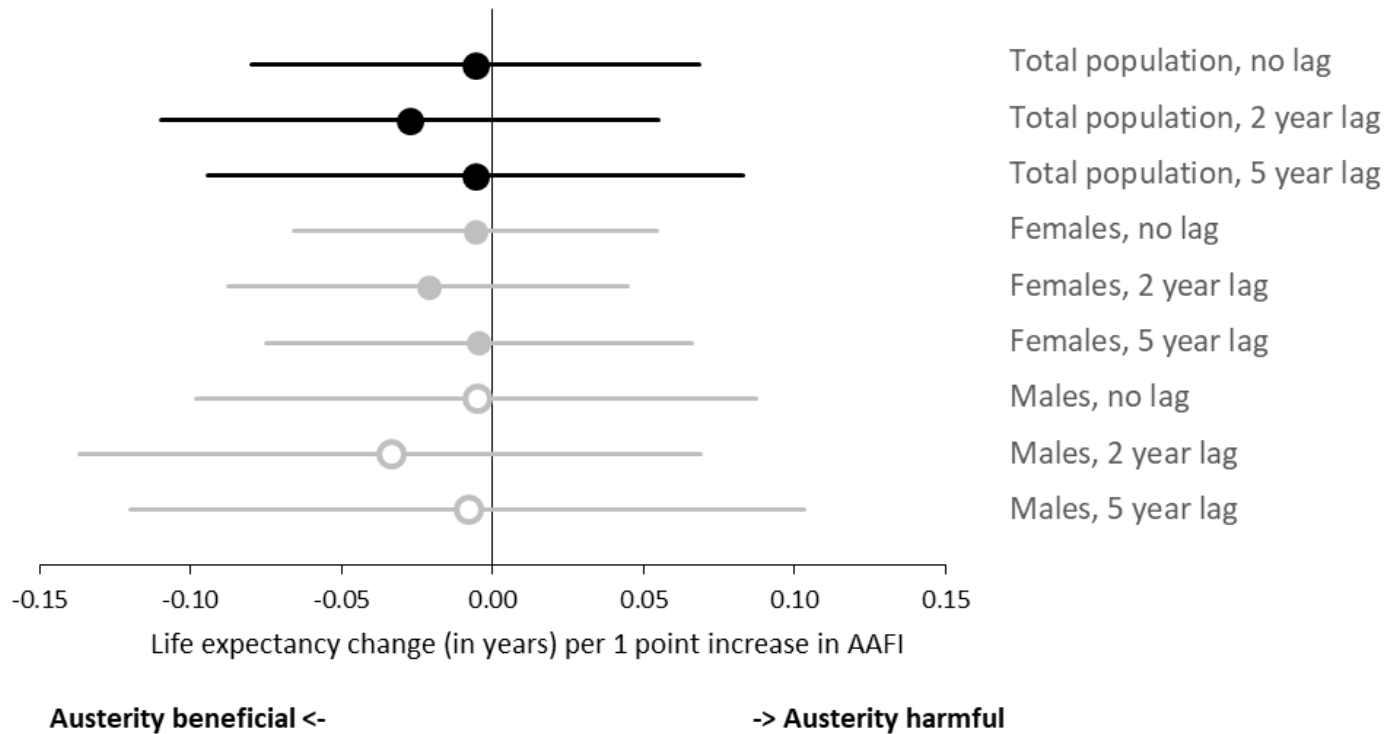


Figure S8.18 - Relationship between indexed Government Expenditure and life expectancy, for 0, 2 and 5 year lags between exposure and outcome, adjusted for baseline real GDP per capita

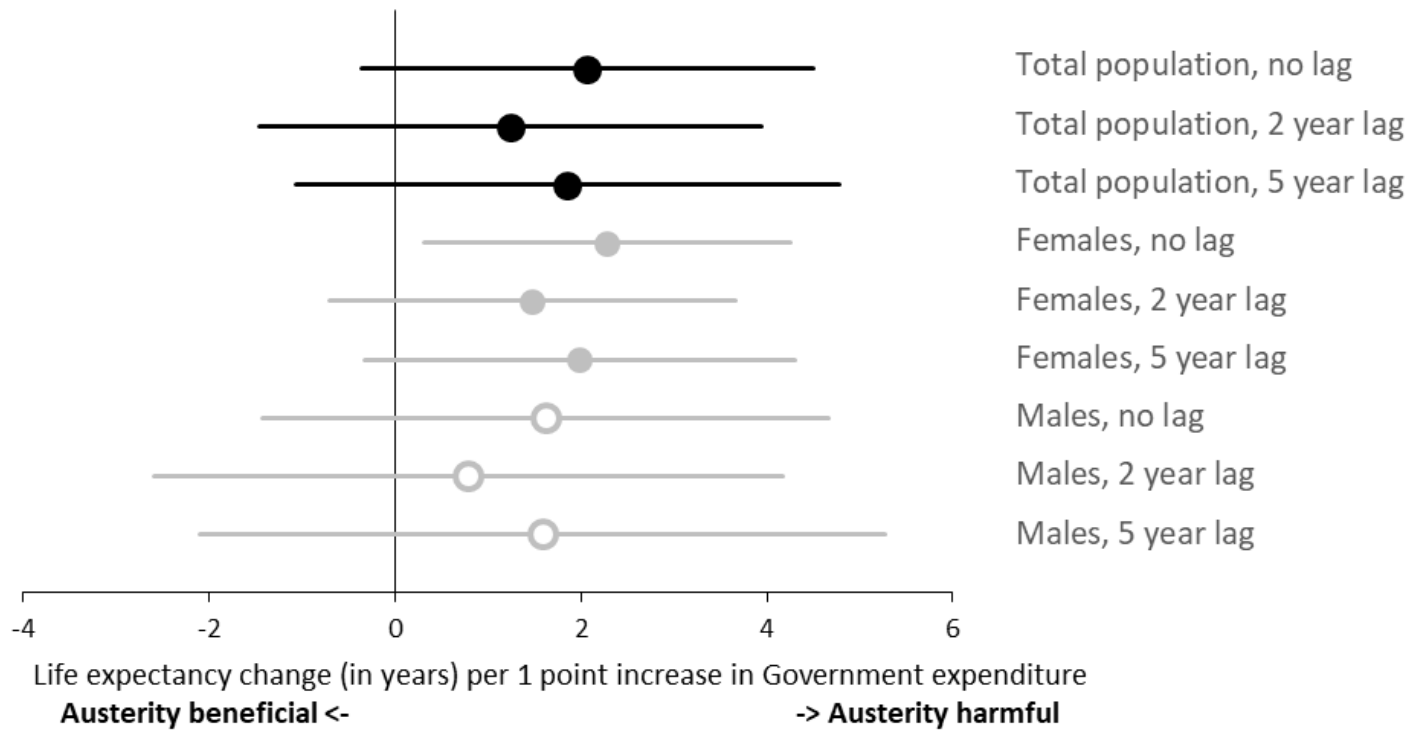


Figure S8.20 - Relationship between CAPB and life expectancy, for 0, 2 and 5 year lags between exposure and outcome, adjusted for baseline real GDP per capita

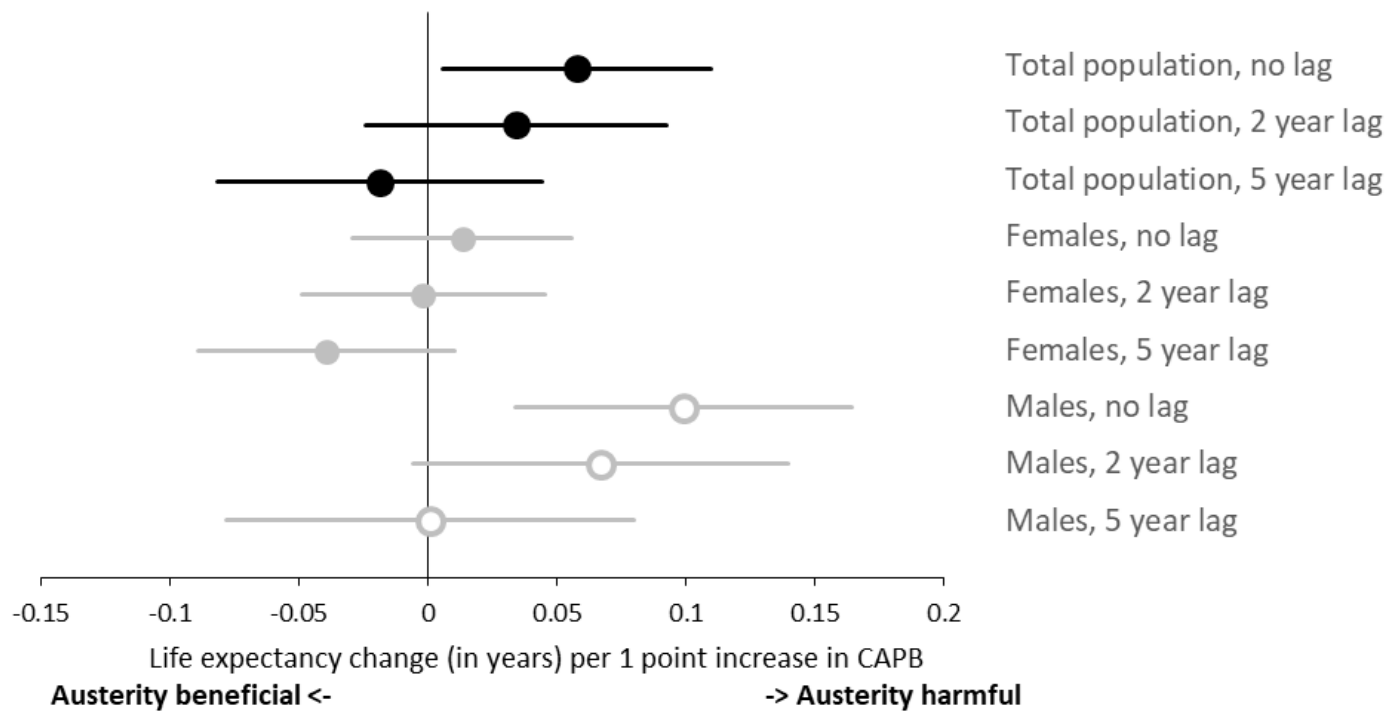


Table S8.17 - Relationship between all measures of austerity and life expectancy, adjusted for baseline underemployment

	No lag				2 year lag				5 year lag			
	Estimated change per unit increase	p value	lower CI	upper CI	Estimated change per unit increase	p value	lower CI	upper CI	Estimated change per unit increase	p value	lower CI	upper CI
AAFI												
Life expectancy (total)	0.08	0.42	-0.12	0.29	0.10	0.34	-0.11	0.32	0.09	0.44	-0.14	0.32
Life expectancy (females)	0.07	0.34	-0.07	0.22	0.08	0.27	-0.07	0.23	0.07	0.38	-0.09	0.23
Life expectancy (males)	0.10	0.49	-0.18	0.38	0.12	0.41	-0.17	0.41	0.10	0.50	-0.20	0.40
Government Expenditure												
Life expectancy (total)	17.42	0.00	12.76	22.07	18.29	0.00	13.48	23.10	18.58	0.00	13.49	23.67
Life expectancy (females)	11.09	0.00	7.75	14.44	11.71	0.00	8.29	15.14	11.92	0.00	8.23	15.61
Life expectancy (males)	24.12	0.00	17.87	30.36	25.08	0.00	18.64	31.52	25.25	0.00	18.55	31.95
Public Social Spending												
Life expectancy (total)	0.17	0.00	0.08	0.26	0.17	0.00	0.08	0.27	0.14	0.01	0.04	0.24
Life expectancy (females)	0.14	0.00	0.07	0.20	0.14	0.00	0.08	0.21	0.11	0.00	0.04	0.18
Life expectancy (males)	0.21	0.00	0.09	0.34	0.21	0.00	0.08	0.34	0.17	0.01	0.04	0.31
CAPB												
Life expectancy (total)	-0.03	0.65	-0.15	0.09	-0.03	0.60	-0.16	0.09	-0.07	0.26	-0.21	0.06
Life expectancy (females)	-0.03	0.43	-0.12	0.05	-0.03	0.44	-0.12	0.05	-0.06	0.19	-0.16	0.03
Life expectancy (males)	-0.03	0.73	-0.19	0.13	-0.04	0.66	-0.21	0.13	-0.09	0.32	-0.26	0.09

Figure S8.21 - Relationship between AAFI and life expectancy, for 0, 2 and 5 year lags between exposure and outcome, adjusted for baseline underemployment

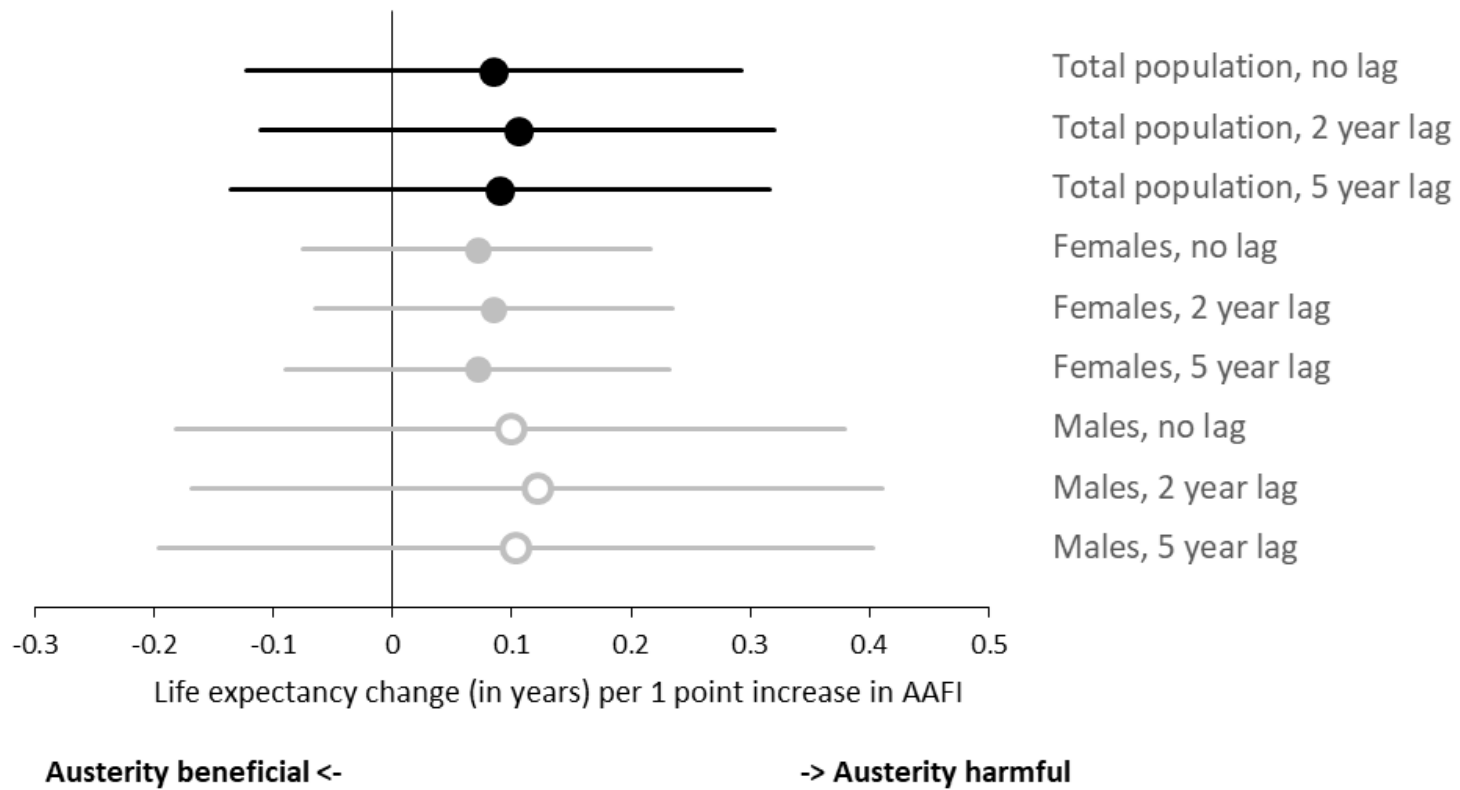


Figure S8.22 - Relationship between indexed Government Expenditure and life expectancy, for 0, 2 and 5 year lags between exposure and outcome, adjusted for baseline underemployment

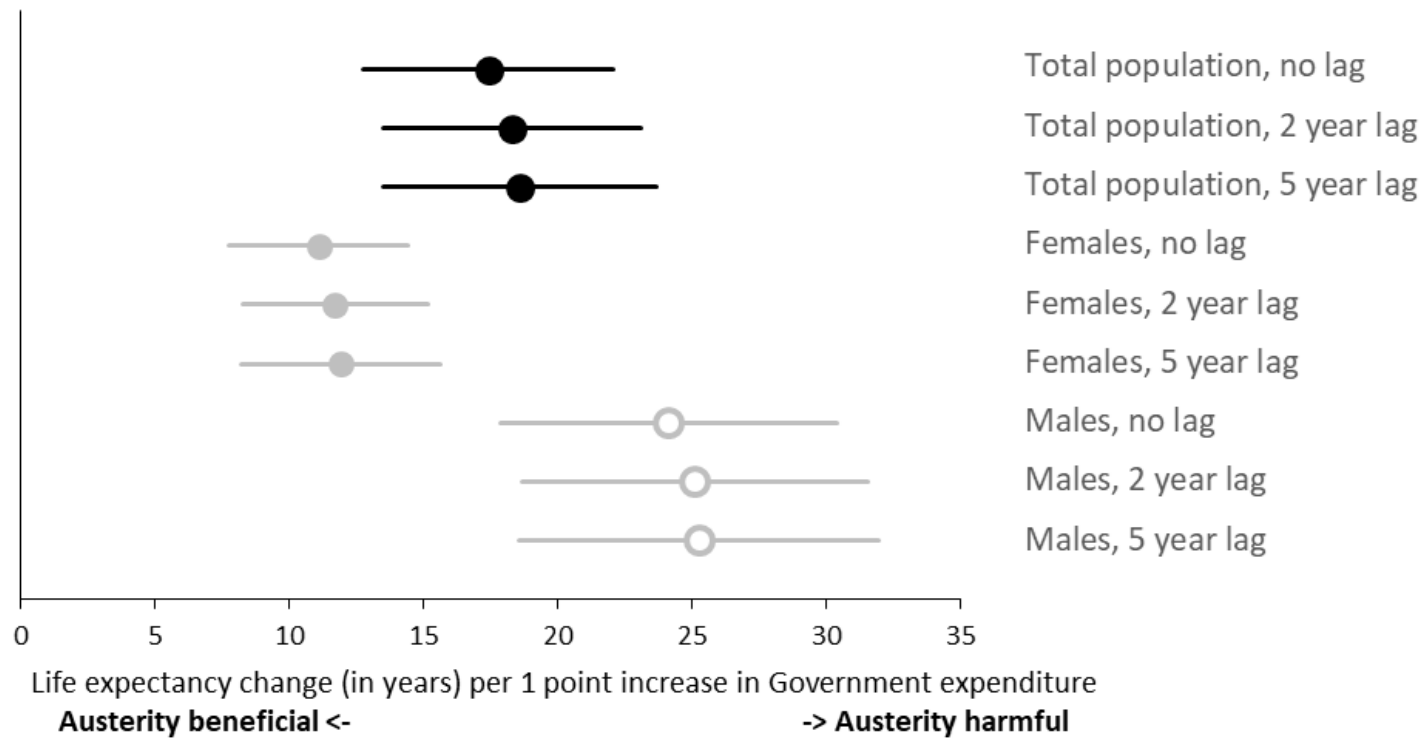


Figure S8.23 - Relationship between Public Social Spending and life expectancy, for 0, 2 and 5 year lags between exposure and outcome, adjusted for baseline underemployment

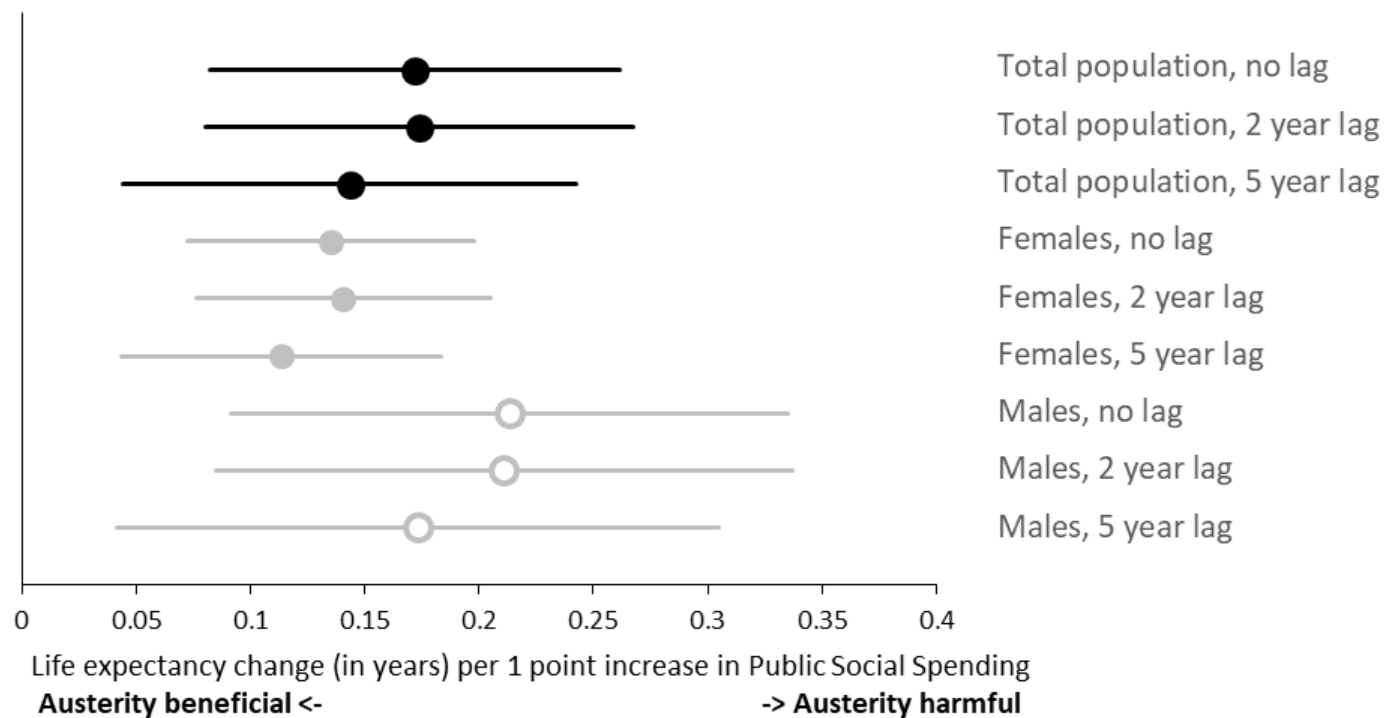


Figure S8.24 - Relationship between CAPB and life expectancy, for 0, 2 and 5 year lags between exposure and outcome, adjusted for mean baseline household incomes

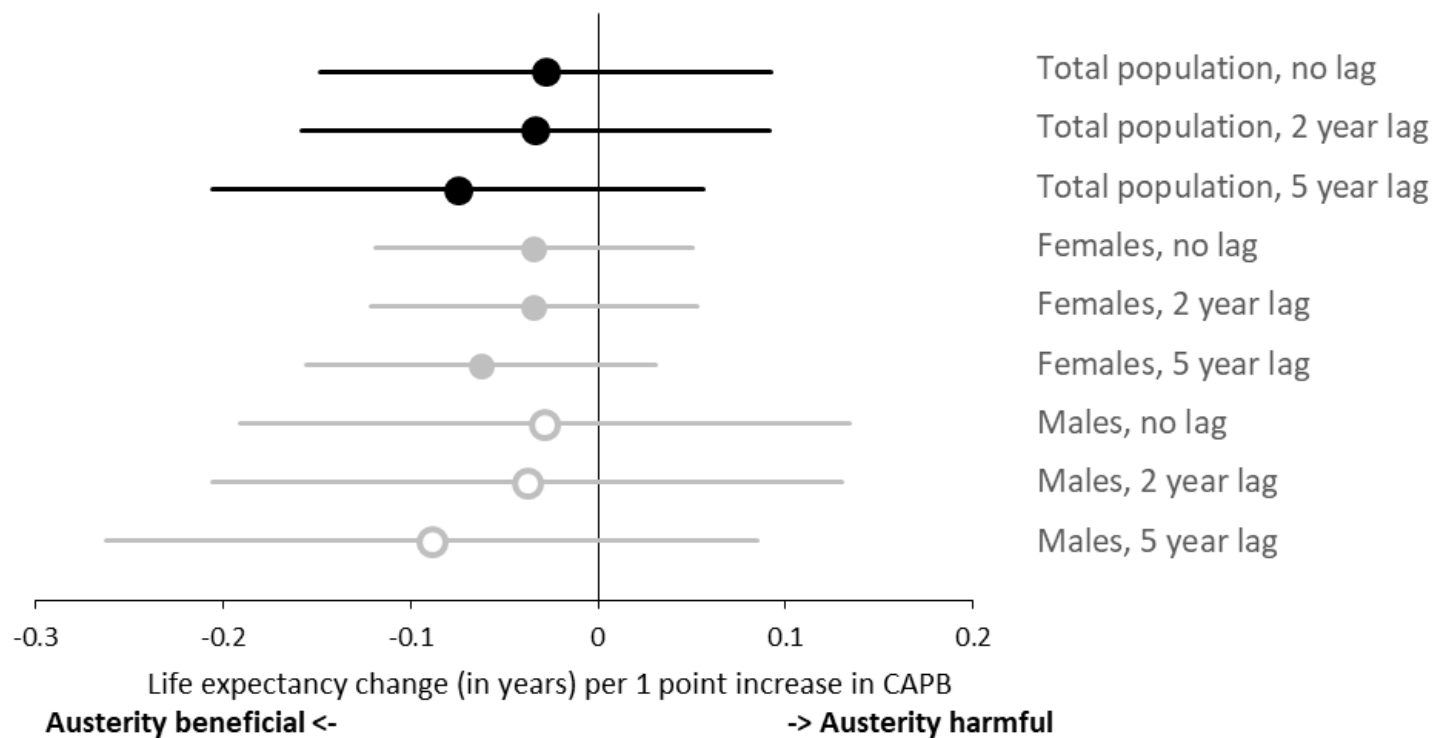


Table S8.18 - Relationship between all measures of austerity and unemployment, and between all measures of austerity and mean household incomes

	No lag				2 year lag				5 year lag			
	Estimated change per unit increase	p value	lower CI	upper CI	Estimated change per unit increase	p value	lower CI	upper CI	Estimated change per unit increase	p value	lower CI	upper CI
AAFI												
Underemployment	-0.03	0.71	-0.16	0.11	0.03	0.73	-0.14	0.21	0.06	0.44	-0.10	0.22
Mean household incomes	-99	0.50	-385	188	-45	0.76	-337	247	-121	0.43	-422	180
Government Expenditure												
Underemployment	-0.65	0.61	-3.15	1.86	-0.48	0.73	-3.15	2.19	1.43	0.41	-1.92	4.79
Mean household incomes	-2,272	0.41	-7,677	3,134	-2,700	0.34	-8,261	2,861	-3,927	0.19	-9,754	1,900
Public Social Spending												
Underemployment	0.00	0.91	-0.09	0.10	0.04	0.50	-0.07	0.15	0.07	0.25	-0.05	0.20
Mean household incomes	-16	1	-207	176	-48	0.63	-242	147	-10	0.93	-220	200
CAPB												
Underemployment	-0.05	0.34	-0.16	0.06	-0.12	0.06	-0.25	0.01	-0.15	0.05	-0.30	0.00
Mean household incomes	-16	0.90	-257	225	-25	0.84	-273	223	8	0.95	-249	265

Figure S8.21 - Relationship between AAFI and underemployment, for 0, 2 and 5 year lags between exposure and outcome

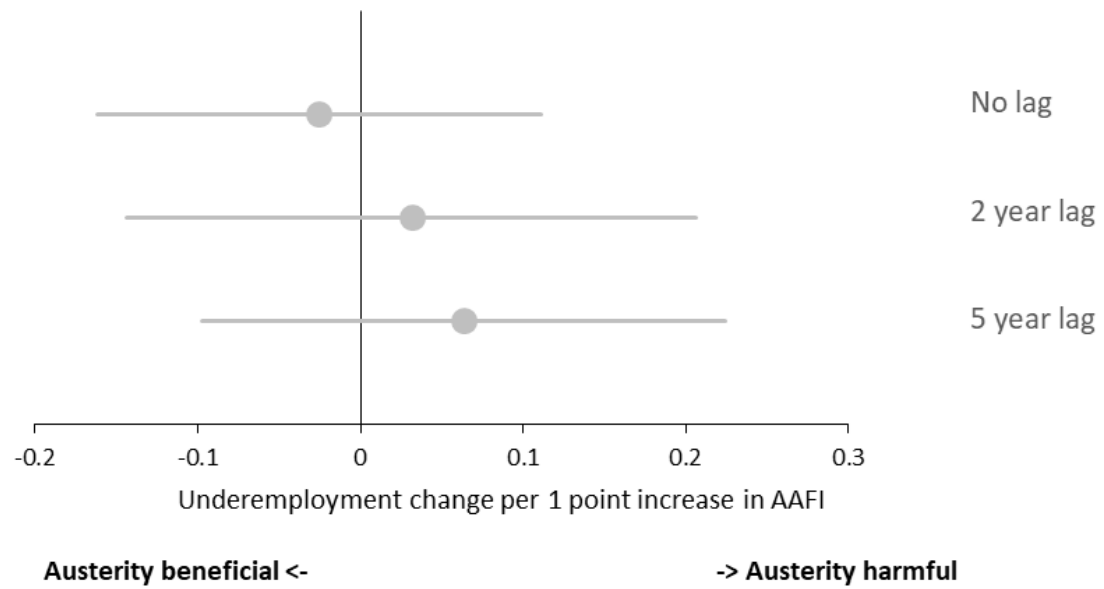


Figure S8.23 - Relationship between Public Social Spending and underemployment, for 0, 2 and 5 year lags between exposure and outcome

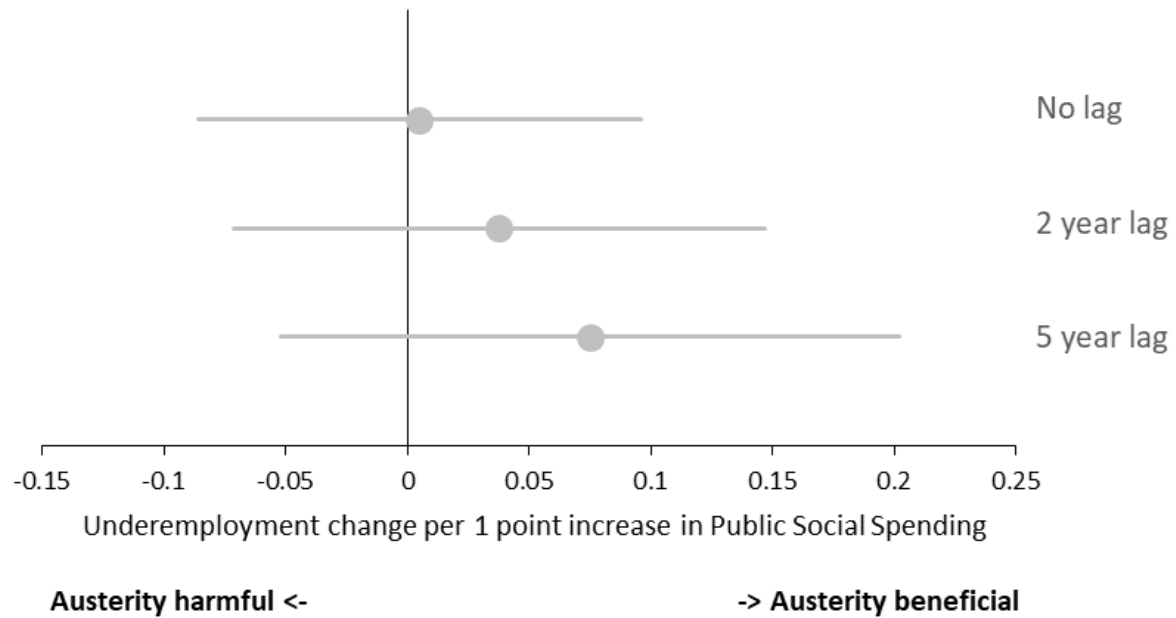


Figure S8.24 - Relationship between CAPB and underemployment, for 0, 2 and 5 year lags between exposure and outcome

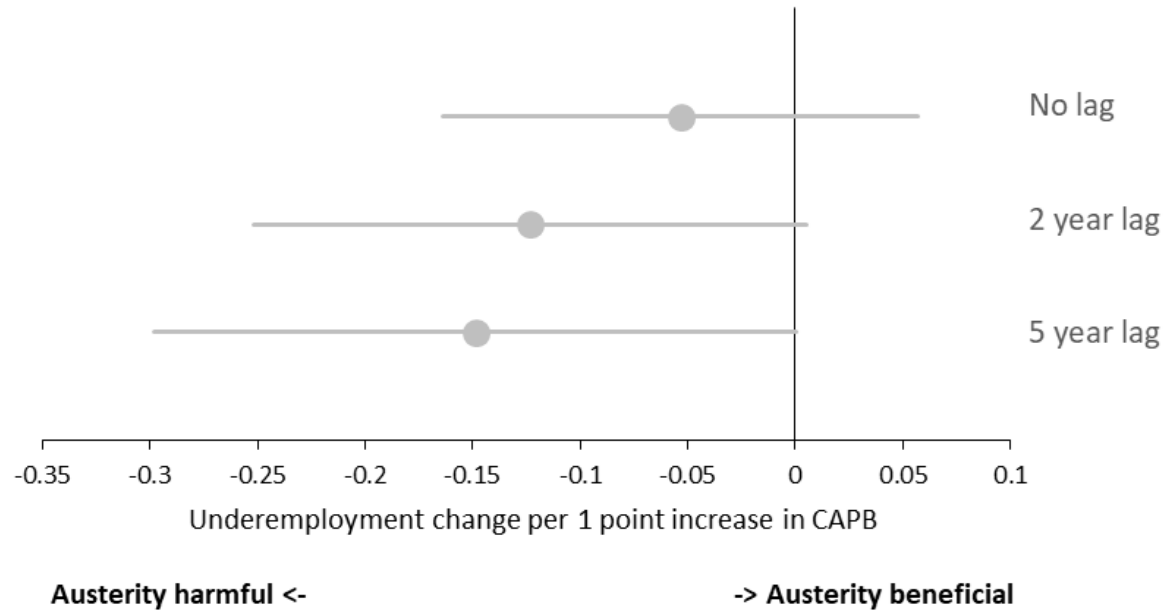


Figure S8.21 - Relationship between AAFI and mean household incomes, for 0, 2 and 5 year lags between exposure and outcome

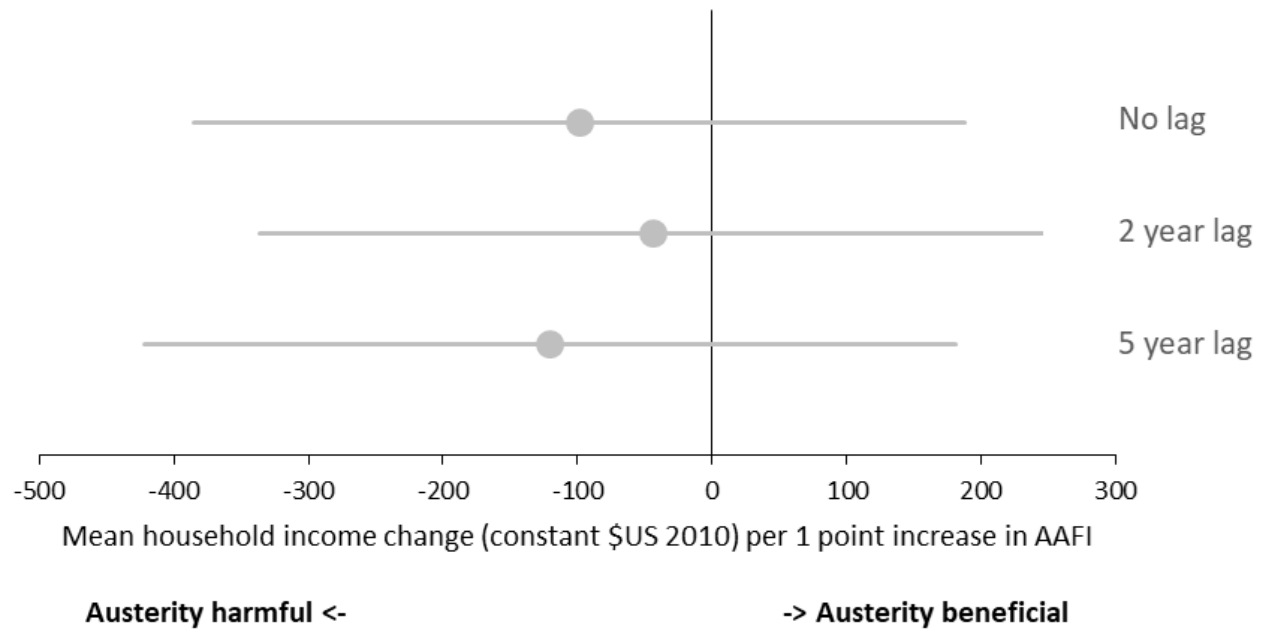


Figure S8.22 - Relationship between indexed Government Expenditure and mean household incomes, for 0, 2 and 5 year lags between exposure and outcome

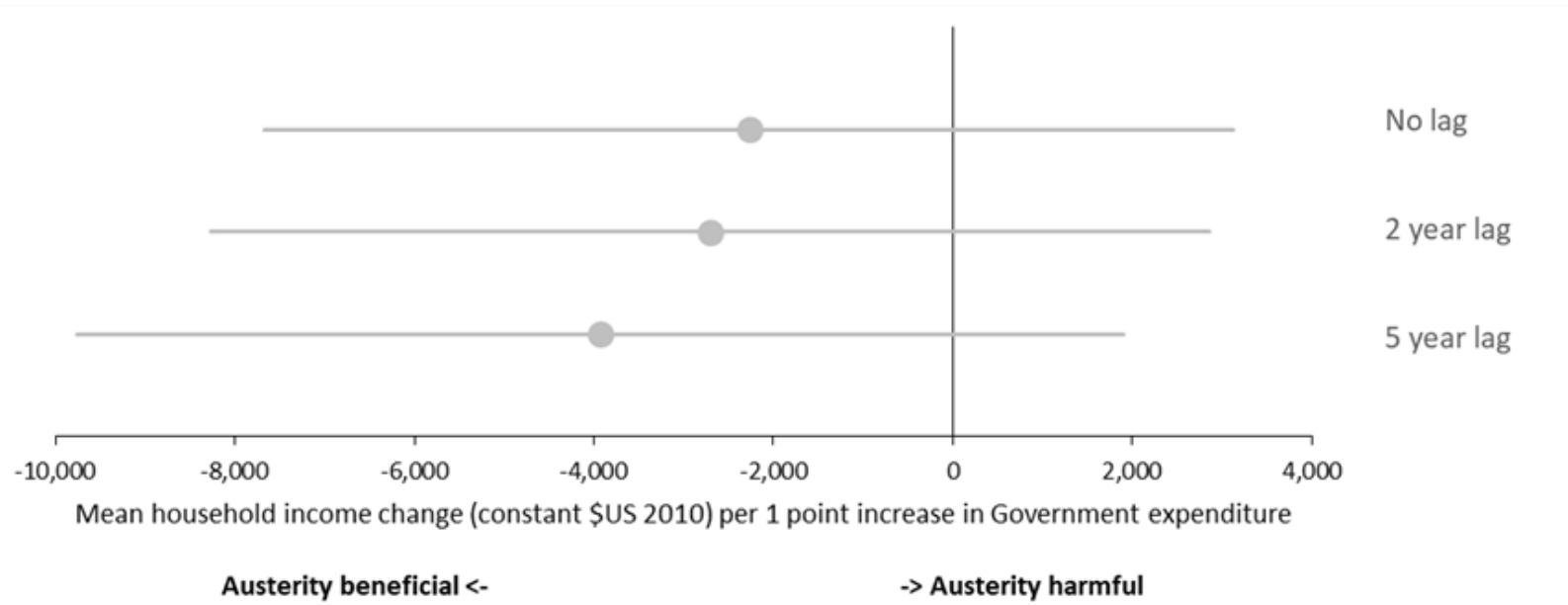


Figure S8.23 - Relationship between Public Social Spending and mean household incomes, for 0, 2 and 5 year lags between exposure and outcome

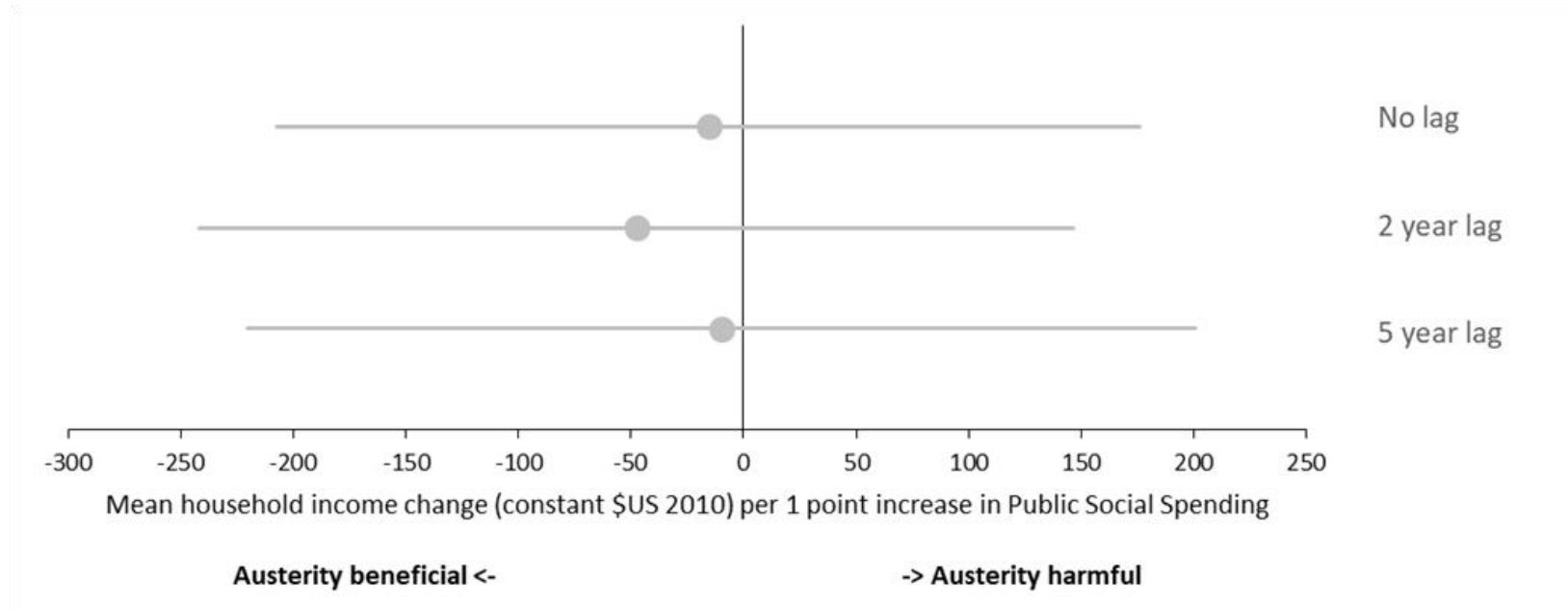


Table S8.19 - Relationship between all measures of austerity and total life expectancy, excluding countries whose economies are oil-dominated

	No lag				2 year lag				5 year lag			
	Estimated change per unit increase	p value	lower CI	upper CI	Estimated change per unit increase	p value	lower CI	upper CI	Estimated change per unit increase	p value	lower CI	upper CI
AAFI	0.005	0.907	-0.074	0.084	0.055	0.186	-0.027	0.137	0.011	0.803	-0.072	0.094
Government Expenditure	7.427	0.000	4.575	10.279	5.958	0.000	2.971	8.945	5.458	0.000	2.421	8.496
Public Social Spending	0.361	< 2.2e-16	0.304	0.418	0.260	0.000	0.188	0.333	0.104	0.017	0.019	0.190
CAPB	-0.063	0.016	-0.114	-0.012	-0.069	0.029	-0.130	-0.007	-0.029	0.396	-0.097	0.038

Table S8.20 - Relationship between all measures of austerity and total life expectancy, restricted to economic downturn years in each country

	No lag				2 year lag				5 year lag			
	Estimated change per unit increase	p value	lower CI	upper CI	Estimated change per unit increase	p value	lower CI	upper CI	Estimated change per unit increase	p value	lower CI	upper CI
AAFI	0.150	0.023	0.022	0.279	0.079	0.261	-0.058	0.216	0.086	0.244	-0.058	0.229
Government Expenditure	7.714	0.001	3.339	12.088	6.324	0.009	1.659	10.989	6.535	0.010	1.631	11.438
Public Social Spending	0.472	0.000	0.363	0.580	0.282	0.001	0.123	0.442	-0.178	0.124	-0.402	0.047
CAPB	-0.014	0.820	-0.132	0.105	-0.024	0.794	-0.206	0.157	0.027	0.724	-0.124	0.178

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