# Social patterning in biomarkers of health, an analysis of health inequalities using Understanding Society: the UK Household Longitudinal Study

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**Abstract**

Health inequalities are known to be prevalent in Britain. Though testing hypothesised pathways between socio-economic position and biological markers of heath, this thesis aims to improve understanding of how socio-economic inequality becomes health inequality and how physiology is affected by socio-economic position.

Using Understanding Society data, access is gained to a range of biomarkers collected cross-sectionally from an adult age range. Methods such as regressions, decompositions and mixed-models are used to identify mediators of SEP's association with grip strength, self-reported type two diabetes, glycated haemoglobin and lung function. The mediators explored are material deprivation and exposures, psychosocial stress and health behaviours. Using retrospective socio-economic position measures, consideration is paid to the timing of disadvantage, while the wide age range enables identification of when inequalities emerge.

Disadvantaged socio-economic position in childhood and adulthood were negatively associated with grip strength, though the gradient does not emerge until mid-adulthood. Health behaviours only slightly mediated this association and childhood socio-economic position continued to be important in adulthood. Support was found for mediation of socio-economic position's association with self-reported type two diabetes, but not with glycated haemoglobin. The mediation was mainly via obesity with no significant mediation through material deprivation, psychosocial stress or health behaviours. Inequalities in lung function were observable at all adult ages and appeared to worsen with increasing age. Material exposures and health behaviours mediated this. Childhood socio-economic position was important in adulthood and moderated the effect of some exposures and health behaviours.

This thesis finds that early disadvantage can have lasting effects. The lack of support for mediation in some outcomes suggests the need to address social inequalities directly, while the identification of mediating mechanisms in other outcomes indicates ways to alleviate these processes.

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**Declarations**

No part of this thesis has been submitted for another degree and all the work in this thesis is original and my own.

# Tables

# Figures

# List of acronyms

| | |
|---|---|
| A level | General Certificate of Education Advanced level |
| AIC | Akaike information criterion |
| BHPS | British Household Panel Survey |
| BMI | Body mass index |
| BWHHS | British Women's Heart and Health Survey |
| $C_6H_6$ | Benzene |
| CO | Carbon Monoxide |
| COPD | Chronic obstructive pulmonary disorder |
| ELSA | English Longitudinal Study of Aging |
| EPIC | European Prospective Investigation of Cancer |
| ETS | Environmental tobacco smoke |
| FEV | Forced expiratory volume |
| $FEV_1$ | Forced expiratory volume in the first second of exhalation |
| $FEV_1$% | Percentage of predicted $FEV_1$ for a person of that age, height, gender and ethnicity |
| FEV1/FVC | The ratio of $FEV_1$ to forced vital capacity |
| FVC | Forced vital capacity |
| GCSE | General Certificate of Secondary Education |
| GHQ | General health questionnaire |
| GP | General practitioner |
| GPS | General population sample |
| $HbA_{1c}$ | Glycated haemoglobin |
| HPA axis | Hypothalamic-pituitary adrenal axis |
| HSE | Health Survey for England |
| IMD | Index of Multiple Deprivation |
| ISCO-88 | International standard classification of occupations |
| JEM | Job exposure matrix |
| kg | Kilogram |
| LTRI | Lower tract respiratory infection |
| MIDUS | Midlife in the United States, A National Longitudinal Study of Health and Well-being |
| MRC | Medical Research Council |
| N | Number of observations |
| NHA | Nurse Health Assessment |
| NHANES | National Health and Nutrition Examination Survey |
| NHS | National Health Service |
| NO2 | Nitrogen dioxide |
| NSHD | National Survey of Health and Development |
| $O_3$ | Ozone |
| OECD | Organisation of Economic Cooperation and Development |
| *p* | Probability of observing result if null hypothesis exists in population |
| PM | Particulate matter |
| PSU | Primary sampling unit |
| P-R2 | Pseudo r-squared |

| | |
|---|---|
| RGSC | Registrar General's Social Class |
| R2 | R-squared |
| SD | Standard deviation |
| SE | Standard error |
| SEP | Socio-economic position |
| SHARE | The Survey of Health, Aging and Retirement in Europe |
| SO | Sulphur dioxide |
| T2D | Type two diabetes |
| UK | United Kingdom |
| UKHLS | United Kingdom Household Longitudinal Study |
| US | United States |
| VOC | Volatile organic compounds |

# Chapter One: Overall introduction

## 1.1 Introduction

Health is influenced by exposures and socio-economic experience across the life course. In Britain, there is evidence of inequalities in both life and healthy life expectancy (Marmot, 2012). Men in the most deprived area had a healthy life expectancy of 54.9 years in 2011-2012 while it was 71.4 years in the least deprived. For women, those in the most deprived area had a healthy life expectancy of 58.3 compared to 69.9 years for those in the least deprived area. Inequalities exist in the prevalence of chronic illnesses such as stroke, arthritis, hypertension and chronic respiratory diseases in England (Dalstra, 2005) as well as in frailty among the elderly (Syddall et al., 2009). Health inequalities have expanded throughout the twentieth century despite the development of the welfare state and the National Health Service (NHS) (Bartley, 2012). Social factors have been shown to be important determinants of health relative to medical care. The social determinants of health have been described as the conditions we live and work in throughout our life course, these are determined by income, wealth and education (Braveman and Gottlieb, 2014). Evidence on the pathways between socio-economic determinants and health outcomes via exposure to hazards, work conditions, stress and behaviours has grown in the preceding decades (Braveman and Gottlieb, 2014).

Through exploring social patterning in biomarkers, this thesis will contribute to the understanding of health inequalities. Three standalone chapters consider how an array of socio-economic characteristics is associated with biomarker outcomes. The roles of explanatory pathways, hypothesised to have a biologically plausible association with the outcome in question, are tested as mediators of the association with socio-economic position (SEP). These include material deprivation and exposures, psychosocial stress and health behaviours. This is valuable as it contributes to knowledge on the mechanisms through which SEP affects health. Identifying these can enable effectively directed policy action to alleviate health inequalities. A life course perspective is used, the importance of SEP at different life stages and trajectories of socio-economic experience are considered through using retrospective measures in cross-sectional analysis. As appropriate to each outcome, critical or sensitive period, social mobility or accumulation theories of the life course are adopted and tested. When inequalities are set and when they emerge is investigated. This enables identification of when intervention to prevent and alleviate health inequalities would be most pertinent.

Much previous research on health inequalities has considered outcomes such as life expectancy, chronic illness and self-reported health. Self-reported health status in particular has been used extensively in large-scale surveys to show socio-economic variation in health. This is limited by biases in self-reporting, misreporting of chronic illnesses is higher among those with disadvantaged SEP, which can result in the underestimation of health inequalities (Mackenbach et al., 1996). General self-reported health does not provide insight into underlying physical conditions or specific health outcomes. These limitations can be overcome by using biomarkers. Biomarkers have been defined by the National Institute of Health Biomarkers Definitions Working Group as objectively measured characteristics that provide indication of biological functioning (2001). Objective measurement prevents bias in misreporting. Biomarkers give insight into decline of physical function and accumulation of stress. They can detect sub clinical poor health as well as the likelihood and beginning of illness (Mayeux, 2004). Due to how biomarkers respond to aging, stress and the socio-economic environment, they contribute to the understanding of how socio-economic experience influences physiology. This enables identification of aspects of biological functioning that are sensitive to specific exposures.

The outcomes explored are grip strength, self-reported Type 2 Diabetes (T2D), glycated haemoglobin ($HbA_{1c}$) and lung function, capturing muscle strength and function, metabolic regulation and respiratory health. These outcomes give insight into discrete aspects of physiological health, for which different exposures and life stages are likely to be important. Grip strength indicates functional capacity, in midlife it is known to be associated with later life frailty as well as morbidity and mortality. T2D is a chronic illness and raised $HbA_{1c}$, depending on the level, can indicate risk or presence of T2D or poor diabetes management. Both are associated with a range of complications, morbidly and increased risk of mortality. Lung function indicates respiratory health and is associated with morbidity and mortality.

To explore the aims of this thesis, it is necessary to have measurement of biomarkers in a representative sample of the population alongside measures that would enable a life course perspective and investigation of explanations for health inequalities. The United Kingdom Household Longitudinal Study (UKHLS) was considered the most appropriate survey for this. Though cross-sectional, the survey collects retrospective measures of SEP from childhood allowing for critical periods, accumulation and SEP trajectories to be explored. The broad scope of the survey means that a range of indicators pertaining to living and working conditions,

deprivation, psychosocial experience and health behaviours were available. Biomarkers have not been collected from a full age range in any other large scale survey in Britain before, this provides an opportunity to investigate whether SEP associations and mediation vary with age as well as when inequalities emerge.

The subsequent sections of the introduction discuss theories of health inequalities and the life course. The overarching conceptual position of the thesis is introduced, as are the empirical chapters. How SEP is conceptualised and measured is explained. Understanding Society: the UK Longitudinal Survey (UKHLS) and the British Household Panel Survey (BHPS), which are used to answer the research questions of this thesis, are described.

## 1.2 Theories of health inequalities

In 1980, the Report of the Working Group on Inequalities in Health, 'The Black Report', concluded that health inequalities in the UK were attributable to social inequalities including income, education, housing, diet, and employment and its conditions rather than due to the NHS (DHSS, 1980). The proposed explanations were artefact, social/natural selection, materialist and cultural/behavioural.

The artefact and selection explanations which attributed health inequalities to how health and social class are measured and to poor health leading to downward mobility respectively were not considered credible explanations (Blane, 1985) and were less prominent in discourse on health inequalities (Bartley, 1998). A review of evidence relating to the selection argument found it was most plausible at entry to the labour market, least probable for children and the elderly, and likely to explain little of the gradient (Blane et al., 1993).

The material explanation encompasses working and living conditions, exposure to environmental hazards and lack of resources (Macintyre, 1997). This was proposed by the Black Report to be the most likely explanation of health inequalities. A systematic review considering material, psychosocial and health behaviour explanations for inequalities in self-reported health found greatest support for the material explanation as measured by living and working conditions, and financial issues (Moor et al., 2016). Researchers have argued that changes in the material conditions of education, health care, welfare provision and work are essential to reduce health inequalities (Lynch et al., 2000).

However, the existence of a socio-economic gradient in health among those with advantaged SEP, as evidenced by the Whitehall studies, indicates that material deprivation alone is not responsible for health inequalities (Marmot and Wilkinson, 2001; Alder and Stewart, 2010). A psychosocial explanation of health inequalities can refer to how stress and a lack of autonomy in work or in life more generally impact negatively on health. Relative deprivation can create insecurity, social anxiety and depression. Psychosocial stress can be conceptualised as being a result of material deprivation as well as the psychosocial burden of relatively low status in a hierarchy.

The cultural/behavioural explanation considered the socio-economic gradient in health a result of differences in diet, smoking, alcohol consumption, exercise and use of preventative health care (Blane, 1985). SEP differences in health behaviours can result from psychosocial issues such as greater exposure to stress for those with disadvantaged SEP and then adapting unhealthy behaviours in response to this (Elstad, 1998; Pampel et al., 2010). Health behaviours can become part of group identity; advantaged SEP groups adopt healthy behaviours as a form of class distinction and behaviours become normalised within SEP groups as social networks reinforce these (Pampel et al., 2010). Advantaged SEP may enable people to better access and utilise health information. Pampel et al (2010) discuss how behaviours cluster, participation in one healthy behaviour can enable and support the uptake of more healthy behaviours just as unhealthy behaviours can encourage more.

It has been argued that focusing on mechanisms between SEP and health neglects the social structure as the fundament cause of health inequality. Unhealthy behaviours may be present among those with disadvantaged SEP but these may indicate other risk factors specific to being at the bottom of a social hierarchy (Blane, 1985). Link and Phelan (1996) argued that the inherent benefits of advantaged SEP in money, knowledge, status, power and connections provides health gains regardless of the mechanisms between SEP and specific health outcomes. For instance, in the past mortality from infectious diseases was higher among disadvantaged groups, now mortality from chronic disease is higher (McCarthy et al., 2013). However, recent findings on inequalities in mortality across eleven European countries over twenty years found a reduction in absolute inequalities in most countries, including England and Wales (treated as one country). The authors attributed this to improvements in population wide behaviours, and in prevention and treatment rather than due to policy on reducing health or social inequality (Mackenbach et al., 2016). They suggested this indicated evidence for the role of

mediators in the reduction in health inequalities though an alternative explanation could be that it was the result of narrower social inequalities earlier in the twentieth century.

More recently, sociologists have added to the debate on health inequalities by claiming that sociological theory of structuration and neo-materialist approaches can contribute to the conceptualisation of health inequalities (Øversveen et al., 2017). Structuration considers both the social structure and mediating mechanisms operating equally as drivers of health inequalities. The neo-materialist approach suggests bringing medical technologies into the framework for investigating health inequalities. Researchers in the US have argued that the role of health services and care in the perpetuation of health inequalities require greater attention in epidemiological research (Kaplan, 2004). While access to health care is provided publically via the NHS in Britain, there is evidence of slight inequalities in the quality of care and patient experience (Cookson et al., 2016).

Researchers have advocated considering explanations for health inequalities as complementing each other and existing in unison rather than as competing explanations, for instance with psychosocial factors being of greater importance than material to those in middle of the gradient (Adler and Stewart, 2010). They can be considered as interdependent and operating via direct or multifaceted mechanisms depending on the health outcome (Benzeval et al., 2014a).

## 1.3 Life course theory and research

Inequalities in adult health may be set in early life, may result from transitions between advantaged and disadvantaged SEP or may accumulate over the life course. The idea of the life course is a relatively recent concept in epidemiology. In sociology, it emerged in the early twentieth century with the use of life histories, trajectories and a longitudinal approach. This emerged with studies on early childhood development, which subsequently gave insights into the impact of changing socio-economic conditions as the children followed in cohort studies grew up through the Great Depression and the Second World War (Elder, 1998). It became more widespread in second half of the twentieth century when birth cohort studies began to mature and sociology moved away from grand theory and towards the trajectories of people's lives.

In epidemiology, patterns in illness on the basis of SEP were explored throughout the twentieth century (Kuh and Ben-Shlomo, 1997). In Britain, research by Barker (1986) on early life nutrition and heart disease using longitudinal area level data was influential for the emergence of life course epidemiology (Ben-Shlomo et al., 2016). Life course effects refer to how health at one age may reflect conditions from conception onwards as well as the current environment one is exposed to (Kawachi et al., 2002). As well as influencing adult health, early life circumstances set socio-economic trajectories in motion (Kawachi et al., 2002). The life course perspective considers individual's transitions between advantaged and disadvantaged SEP as well as the differences between SEP groups. It has been described as an attempt to synthesis various models of disease causation such as adult lifestyle, biological programming and social causation (Kuh et al., 2003).

Life course research in epidemiology has centred around three different models, critical or sensitive periods, accumulation and social mobility. Critical period theories of the life course consider a particular stage in life to be critical for the development of specific health outcomes, within a certain timeframe development is affected by exposures with implications for long-term health (Kuh et al., 2003). Often this refers to early life but it can refer to any stage, for instance, there is evidence of health in adulthood being important for some later life outcomes (Graham, 2002). A sensitive period is similar conceptually to a critical period, it indicates an influential timeframe where development is affected by exposures but the effects are modifiable (Kuh et al., 2003). The instigation of a critical period theory of the life course began at the start of the twentieth century; maternal health and infant health were considered important for health in later life (Kuh and Smith, 1993).

While the critical and sensitive period models suggest that certain stages are more important than others for health outcomes, the accumulation theory of the life course proposes that the effects of exposure to disadvantaged SEP accrue and increase the risk of morbidity and mortality in adult life. Experience of disadvantage in early life increases the likelihood of experiencing disadvantage in later life which adds to health risks (Holland et al., 2000). Though exposures which are not related to SEP may be encountered and add to risk as well. The accumulation perspective takes account of how one experience of disadvantage in life can precipitate another. As people age, the number, duration and severity of exposure to health risks may increase while their ability to repair and withstand exposures declines (Kuh et al., 2003). The effects of accumulation can be additive where exposures increase risk cumulatively or may have a trigger effect where a final exposure sets an increase in

risk. The effects of multiple exposures may also have multiplicative rather than additive effects, such as how exposure to asbestos and smoking interact to result in an increased likelihood of lung cancer greater than their combined risk (Blane et al., 1997).

The social mobility theory of the life course moves from looking at differences between SEP groups to looking at differences in health outcomes on the basis of transitioning between advantaged and disadvantaged SEP (Cable, 2014). Social mobility has been found to constrain the effects of social inequality, where those transitioning from disadvantage to advantage experience less adversity than the SEP they leave but less protection from the SEP they join and vice-versa (Blane, 2006).

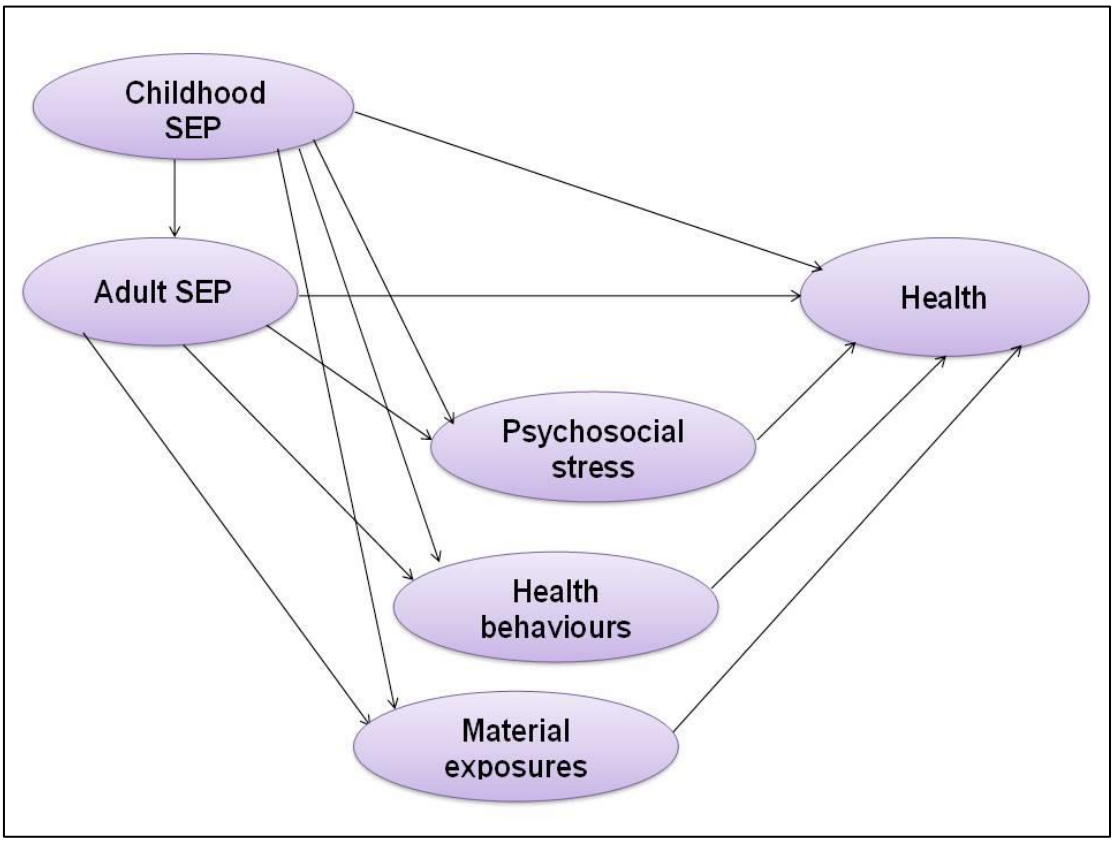
The use of a life course perspective in health research has enabled the development of a broad understanding of social determinants of health. This has developed from exploring differences based on SEP to the implications of when and for how long disadvantage is experienced. It also considers movement between different SEPs. However, there are also limits to the explanatory power of the life course perspective; short term hazards can have immediate impacts and it has been theorised that life course effects become less important in old age when morbidity and mortality are greatest (Blane et al., 2007).

## 1.4 Overview of the research

This section introduces the general aims of the thesis and the objectives and research questions of each chapter. The overarching aim is to explore mediation of SEP's association with health. Mediating pathways explored here include material deprivation and exposures, psychosocial stress and health behaviours. Depending on the evidence from the literature regarding the health outcome in question, mediation of either childhood or adult SEP was focused on. Hypotheses on the importance of pathways between SEP and each outcome were developed based on previous research on mediation as well as research that found direct effects of socially patterned exposures or behaviours. This informed whether a material, psychosocial or behavioural explanation for the inequality or a combination of these was tested as a mediator. Each chapter included additional research questions but this approach is common to all. A general diagram of the conceptual model used in this thesis is shown in Figure 1. This is not a statistical model but represents the conceptual understanding of the relationship between SEP and health. It shows

childhood and adult SEP directly affecting health and affecting psychosocial stress, health behaviours and material exposures. Childhood and adult SEP are depicted as having the same paths to health, though their role and the mediators vary across chapters as specific to the outcome.

Figure 1 General conceptual model of relationship between SEP, mediators and health




The remainder of this section briefly introduces the objectives of each chapter. Grip strength's association with age, and whether this association varies based on SEP, is explored in the second chapter. Social patterning in grip strength has only been explored in cohorts previously, meaning SEP differences in grip strength at different life stages have not been identified. Whether SEP's association with grip strength is mediated by health behaviours is investigated, this has not been examined previously. The chapter considers whether there is evidence to support any of the life course explanations of social patterning in grip strength. A series of models representing SEP trajectories through the life course are compared. This is valuable for understanding inequalities in grip strength as it identifies which socio-economic trajectory is most important for inequalities in grip strength.

The third chapter considers whether material deprivation, psychosocial stress and health behaviours mediate the pathways between SEP and self-reported T2D. This is also done for $HbA_{1c}$ to investigate differences in the pathways between the two outcomes. These may be related to differences in diagnosis and diabetes management, both of these being important to future health outcomes and the development of comorbidities. No research thus far has explored the role of each explanatory pathway as a mediator in unison. This chapter utilises 18 waves of the BHPS to consider long-term exposure to material deprivation, psychosocial stress and poor health behaviours. In decomposing the pathways mediating the SEP association with both outcomes, it allows for understanding of how the association with SEP and the role of mediators may vary between the two outcomes.

The fourth chapter considers mediation of childhood SEP's association with lung function via adult SEP, smoking, physical activity, diet, obesity and occupational exposures. The chapter explores whether disadvantaged SEP in childhood moderates the effect of socially patterned health behaviours and exposures on lung function in adulthood. This could indicate whether childhood is a sensitive period. Exploring moderation is important, as those with disadvantaged childhood SEP will be most likely to encounter these risks. The chapter investigates whether the association between lung function and individual level characteristics vary by household and area and whether indoor air quality and air pollution explain this. This adds to the understanding of the social gradient by bringing the role of house and place into consideration of risk with individual factors. Whether associations between SEP and exposures vary depending on stage of the life course is also explored, adding to knowledge on when social patterning in lung emerges.

## 1.5 Conceptualising socio-economic position (SEP)

The importance of conceptualising SEP in research on health inequalities has been conveyed by numerous authors in this field (Krieger et al., 1997; Bartley, 2004; Galobardes et al., 2006; Braveman et al., 2005; Oakes and Rossi, 2003). There is not necessarily one conceptualisation of SEP which is superior to others, what is important is that the conceptualisation of SEP used is appropriate to the theoretical position of the research and its research questions. Theories of SEP differ between sociologists based on the theory of the social structure that they use. Most theories of the social structure have their basis in Marxian or Weberian theory. SEP can also be conceptualised in a neo-materialist way (Chittleborough et al., 2006) or as

differential access to resources (Oakes and Rossi, 2003). Marxist conceptualisations of SEP understand it as being defined by a person's relationship with the means of production; whether they own means of production or sell their labour to those who own it. This understanding is defined in terms of ownership of means of production and labour, it excludes social status and material resources.

In order to capture both the resources and status elements of SEP, a Weberian conceptualisation is used here. SEP is considered a person's position in the social and economic structure (Bartley, 2004), including class, status and material assets (Berkman and Macintyre, 1996). Material assets and status are hypothesised to underlie the association between SEP and health outcomes through living conditions and deprivation, psychosocial stress and health behaviours. Through focusing on SEP as opposed to poverty, the aim is to examine the mechanisms through which each aspect of SEP may influence health positively or negatively (Adler and Stewart, 2010). The term 'socio-economic position' is used here as opposed to 'socio-economic status' to avoid over-emphasising the role of status over material resources (Krieger et al., 1997).

## 1.6 Measuring adult SEP

There is no single best indicator of SEP in health research, its appropriateness depends on the mechanisms through which socio-economic experience is thought to affect a health outcome (Berkman and Macintyre, 1996). Objectively evaluating how well a SEP indicator works can be difficult, in some literature there has been a propensity to consider measures which produce the greatest differential as the best rather than evaluating how it would plausibly affect a health outcome (Berkman and Macintyre, 1996). There are also differences in reliability, precision and relevance of indicators across different populations. The conceptualisation of SEP as encompassing class, resources and status could be captured by education, occupation or income (Duncan et al., 2002).

Education is used as the primary measure of SEP in this research to capture class, resources and status. Measuring income or wealth could capture resources while occupation affects income and also influences social status. However, education generally precedes these and influences occupation and income (Berkman and Macintyre, 1996; Blane, 1995; Singh-Manoux et al., 2002; Galobardes et al., 2006). Occupation and income could be conceptualised as mediators of the effect of

education. In exploring social patterning in certain health outcomes where material deprivation or work conditions were hypothesised to play a mediating role; measures more closely pertaining to these were included in the analysis.

In comparison to occupation and income, education is relatively stable across the life course, a disadvantage of this is that it does not capture downward social mobility (Liberatos, 1988). Education does not capture the volatility of economic experiences throughout adulthood as income or occupation would (Duncan et al., 2002). Though it helps to avoid reverse causality when considering SEP and health at midlife (Duncan et al., 2002). Income may be more susceptible to this (Galobardes et al., 2006, Duncan et al., 2002), similarly occupation may also be affected by poor health especially at older ages (Duncan et al., 2002). Education can be affected by poor health and SEP in childhood which may in turn affect adult health as well as achieved adult SEP (Galobardes et al., 2006). As education is most often set in early adulthood it is particularly useful when considering health outcomes which have origins in early life (Galobardes et al., 2006). Grip strength and lung function are influenced by early life experience and reach their peak in early adulthood while some studies have found that risk of diabetes is influenced by experience across the life course; education may be a particularly pertinent SEP measure for these outcomes.

Education is the easiest of the main social class indicators to measure; it has low refusal rates, is more likely to be reported accurately and is considered less sensitive (Liberatos, 1988). Sensitivity in reporting income can result in higher non-response (Berkman and Macintyre, 1996). Respondents may also be inclined to misreport their income as higher or as lower than it actually is. Income is most often measured at the household level which does capture sharing of resources but not unequal access to resources which can impact negatively upon women (Duncan et al., 2002). Using measures of income relative to a particular period such as the month or quarter before interview may result in biased results due to seasonal fluctuations in earnings. Income is also less effective as an SEP measure for those who have retired and students. Occupation is inappropriate for those who have never entered in the labour market, students and people who are retired (Chittleborough et al., 2006) and is less appropriate for those who are currently not active in the labour market (Singh-Manoux et al., 2002) while education is universally applicable.

Classifying education by highest level of education attained is done here on the basis that particular levels of achievement are important. Years of education are not

used as this assumes that each year of education contributes equally to SEP (Galobardes et al., 2006). Limitations of highest educational attainment include that it does not capture differences such as whether education was state or private or the type of university a person attended, nor does it capture investments in career which are not formal qualifications (Duncan et al., 2002). Education, as a SEP measure, is influenced by cohort effects (Galobardes et al., 2006, Duncan et al., 2002), educational attainment has increased on average over time and has become more homogeneous (Duncan et al., 2002). However, the addition of age when modelling associations between SEP and health can help control for this.

Using a summary indicator of multiple SEP measures was not considered as this would treat each as though it measured the same feature of SEP and would not distinguish which aspects are related to health outcome (Duncan et al., 2002, Chittleborough et al., 2006). Including education, occupation and income in the model was not considered appropriate as it would be un-parsimonious and it would not represent the temporal ordering between SEP measures. However, in some parts of the research, additional SEP indicators are used as appropriate to the outcome and research question.

Using measures of living conditions such as car ownership, tenure, crowding and amenities were ruled out as primary SEP measures. These measures were considered more distant from the hypothesised causal pathways between SEP and the biomarkers explored here than education, occupation and income. The social processes by which they may be important for health are less clear (Berkman and Macintyre, 1996) and measures such as car ownership and amenities may be related to SEP but they were not considered indicators of SEP in themselves (Galobardes et al., 2006). Wealth was not considered as a potential indicator, though it is particularly relevant for retired populations (Allin et al., 2009), it is less suitable for younger cohorts. Wealth is also difficult to measure as it can be in the form of property or business ownership or assets, many groups either have no wealth or have debt rather than assets.

Area SEP was also ruled out as a primary SEP measure as it would not capture an individual's resources, status or class and there is not homogeneity of individual's SEP within areas. However, much research has indicated an association between area and health. There is potential to enhance understanding of the determinants of health with consideration of what is meant by area and how it plausibly affects health (Tunstall et al., 2004). The socially patterned mechanisms affecting health can be via

social interaction, environmental risks, geography or public services and institutional resources (Galster, 2012). Debate on area has considered whether it is the characteristics of the place or the people comprising a place that determine the association with health, particularly when individual characteristics are aggregated to define area characteristics (Macintyre et al., 2002). The area's environment may be more or less easily negotiated depending on individual's SEP within it, disadvantaged individuals can be more dependent on the resources available in their area (Stafford and Marmot, 2003). As with individual level SEP, a focus of this thesis is understanding the mechanisms through which health inequalities emerge, pathways between area and health were investigated if a biologically plausible mechanism between them was considered likely.

## 1.7 Conceptualising childhood SEP

The life course approach places importance on transitions and critical periods for exposure, it considers how health status at any age reflects not only contemporary conditions but experience from conception onwards and captures the cumulative and dynamic nature of SEP structures and experiences. It could be argued that a single measurement of SEP in adulthood would capture accumulated exposure over the life course, however this was not considered sufficient here to capture the contribution of SEP to the health outcomes being explored. Having SEP measurement at one point in time only would limit the ability of the research to advance understanding of how the influence of SEP varies across life stages. Studies have found that the contribution of circumstances in early life varies greatly between health outcomes and causes of mortality (Galobardes et al., 2004). For the outcomes being explored here, there is evidence in the literature to suggest that early life experience may be particularly important for grip strength and lung function, with some indications of it having a role in T2D. The adversity or protection provided by SEP in childhood may have implications for these health outcomes in the long term if childhood is a critical period for their development. Exploring both childhood and adult SEP has public health importance, it allows for identification of whether both have an independent effect, whether one is of greater importance and whether adult SEP mediates the association between childhood SEP and health. Using SEP measures from different points in the life course can help better understand aetiologies of poor health as well as when risk for certain outcomes is set (Smith et al., 1997). The advantage or disadvantage SEP in childhood provides may set both health and socio-economic trajectories for later life.

## 1.8 Measuring childhood SEP

Given the health outcomes being explored, the conceptualisation of childhood SEP was extended to encompass socially patterned exposures in utero. There is evidence to suggest that prenatal factors influence grip strength and lung function in adulthood (discussed further in Section 2.2.2.1 Grip strength and childhood SEP and Section 4.2.1 Lung function and childhood SEP). As measures pertaining to prenatal exposures and birth weight were not captured in UKHLS, maternal SEP was considered a means of capturing social patterning in the in utero environment as well as childhood SEP. Maternal SEP, as measured by education, has been found to be associated with lower birth weight (Gage et al., 2013; Silvestrin et al., 2013). Maternal SEP may impact on prenatal development through health behaviours, diet, and exposure to stress and to hazards (Reynolds et al., 2013). Obesity, underweight, depression, anxiety, passive and active smoking, not attending ante-natal classes or taking folic acid supplementation, and poor dietary behaviours were all associated with maternal education among pregnant women in DELIVER, a Dutch prospective cohort study (Baron et al., 2015). A study in Ireland found inequalities in preterm delivery and that these were somewhat mediated by material and behavioural factors as well as nutrition (Niedhammer et al., 2012).

Maternal education was considered more suitable than maternal occupation as it is applicable to those who had not entered the labour market, for older cohorts in particular maternal occupation may not be relevant. As a measure related to maternal experience was considered particularly relevant, household or paternal measures were ruled out as they may not capture unequal access to resources within the household, which would be most inclined to affect women negatively. Maternal education is less likely to change after young adulthood than parental occupation or income. Though this may vary for younger cohorts as it has become more common for women to return to education after child bearing (Chittleborough et al., 2006). Using retrospective measures of childhood experience may be more inclined to recall bias, however a study on twins has found high levels of agreement in reporting paternal occupation (Krieger et al., 1998). Retrospective measurement of childhood SEP was moderately associated with directly measured childhood SEP from early life in a cohort (Batty et al., 2005).

## 1.9 Introduction to Understanding Society: the UK Longitudinal Survey (UKHLS)

The UKHLS is a longitudinal survey of approximately 40,000 UK households (Knies, 2014). Wave one began in 2009 and data collection for each wave runs over a two year period. There are multiple components to the sample, it includes a General Population Sample (GPS), Ethnic Minority Boost Sample and as of wave six an Immigrant and Ethnic Minority Boost Sample. The BHPS joined UKHLS in the second wave of UKHLS, which corresponds to the 19$^{th}$ wave of the BHPS. In wave two, a subset of the GPS were invited to participate in the Nurse Health Assessment (NHA). In wave three, the BHPS sample was invited to participate in the NHA.

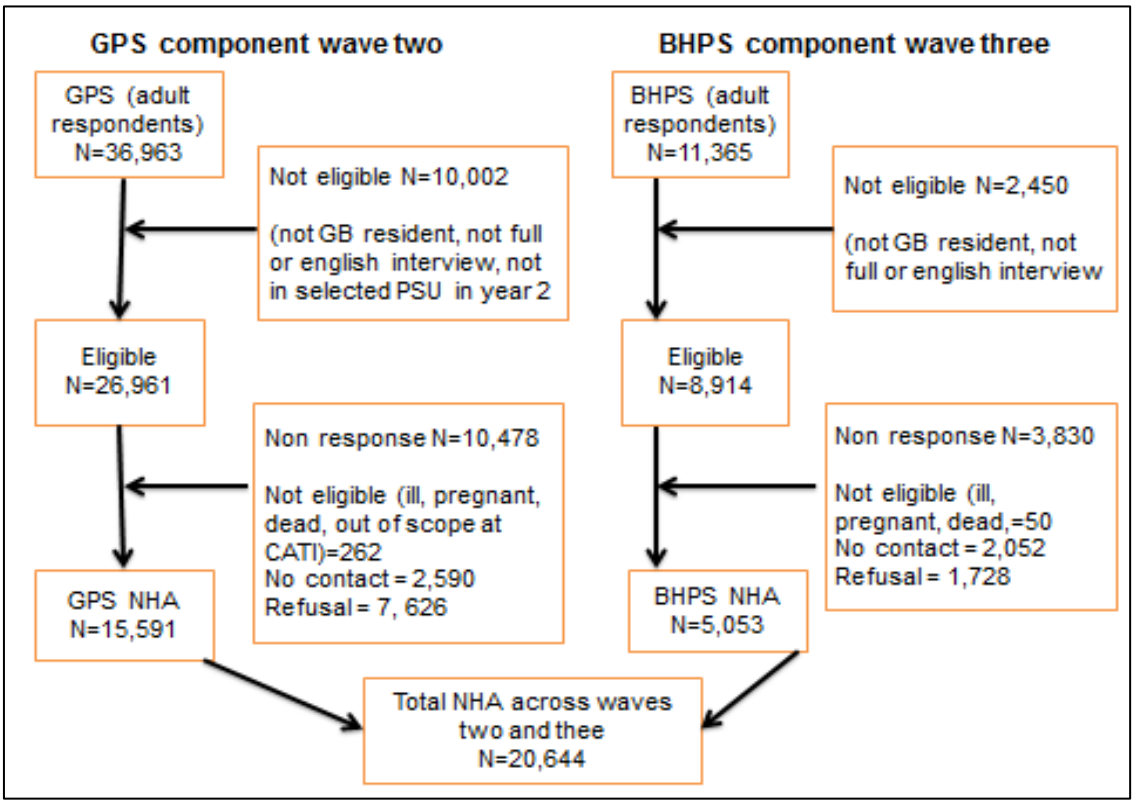
The NHA required a trained nurse to visit respondent's homes and collect a number of anthropic measures. Participants were also invited to provide a blood sample from which more biomarkers were drawn. Approval to collect these was gained from the National Research Ethics Service. To ensure consent was informed, participants were given additional leaflets with information about the measures collected, nurses gave participants time to read this and the consent form and to consider their decision. Oral consent was gained for direct measures such as grip strength while written consent was gained for the blood sample.

The GPS is a stratified, clustered, equal probability sample of residential addresses drawn to a uniform design throughout the whole of the UK (Buck and McFall, 2011). While the GPS includes Britain and Northern Ireland, the NHA was only carried out in Britain. In the first year of wave two, all members of the GPS were invited to participate in the NHA. In the second year of wave two, this was restricted to 81% of the primary sampling units (PSU) in England. Wave two of UKHLS ran from January 2010 to December 2012 with the NHA interviews occurring approximately five months later. In wave two, there were 36,963 adult members of the GPS from which the sample was drawn. The exclusion of those not resident in Britain, those who did not give full interviews, those who had a translated interview and those whose PSU was not selected in year two restricted this to 26,961 who were eligible for participation in NHA. A further 262 individuals were deemed ineligible at follow up; some were deceased and others for reasons such as being ill or pregnant. There were 2,590 with whom no contact was made and a further 7,626 refused to participate. This resulted in 15,591 eligible participants in the NHA, a response rate of 58.6% in wave two.

The BHPS was a longitudinal survey running from 1991. In wave 18 of the BHPS, participants were invited to join UKHLS. Members of the BHPS joined UKHLS in wave two and in wave three adult respondents (n=11, 365) were invited to join the NHA. Wave three ran from January 2011 to December 2013. There were 2,450 from this sample who were ineligible due to being based in Northern Ireland and not doing a full or English language interview. From the remaining 8,914 who were eligible, a further 50 were deemed ineligible due to having passed away or being ill or pregnant. No contact was made with 2,052 members and 1,728 refused to participate. This resulted in 5,053 taking part in the NHA, a response rate of 56.7 (McFall et al., 2013).

Resultantly there were a total of 20,644 participants in the NHA. Figure 2 illustrates response to the NHA in waves two and three.

Figure 2 Response to the NHA in waves two and three of UKHLS



# Chapter Two: A life course approach to exploring social patterning in grip strength

## 2.1 Introduction

Grip strength is an objective measure of functional capacity and is associated with disability, morbidity and mortality. Understanding whether and how socio-economic circumstance affect grip strength has consequences for understanding healthy aging, the development of frailty and health inequalities in later life. Previous studies have focused on grip strength in mid-life, because of its association with health in older age. However, inequalities in many health outcomes emerge in early adulthood and deepen throughout life. Using age-cohort studies may not allow for exploration of when in life such inequalities emerge. Previous research has established that grip strength declines in old age. However, whether this process varies on the basis of socio-economic characteristics has not yet been examined.

The first aim of this study is to explore grip strength's association with age whether this is socially patterned. The second aim is to explore the association between childhood SEP and grip strength and to investigate whether adult SEP and health behaviours mediate this. The third aim is to investigate whether there is evidence to support a critical period (in childhood) theory of the life course as best explaining social patterning in grip strength in comparison to other life course theories.

This chapter begins by reviewing the existing literature on grip strength and SEP. The literature review concludes with an explanation of the conceptual position developed based on the literature and lists the research questions formed. The methodology used by UKHLS, the measures and analytical strategy are described. Results are presented in the fourth section while the fifth section discusses the research findings, their relation to the literature, and strengths and limitations. Consideration is given to future research and policy implications.

## 2.2 Literature Review

The purpose of the literature review is to provide an overview of previous research on grip strength and SEP, to inform the theoretical basis of this chapter and identify where contributions can be made to knowledge in this area. A systematic approach was taken to the literature review, searches were conducted on PubMed, Web of Science and EBSCO using the search terms 'grip strength' and 'socio-economic' as

well as variants of these phrases. Eligible populations were those of any age and based in any location. Publications not in English were excluded. Abstracts were screened and the full texts of 35 papers identified as potentially useful were extracted. When deciding which papers were suitable for inclusion in the literature review, attention was paid to how the sample was selected for the study, whether that sample was representative of the population in question and the sample size. Additional searches were conducted on 'grip strength, frailty, morbidity and mortality' as well as more general searches on health inequalities and the life course. As the role of physical activity and diet emerged as potentially important factors for grip strength from the literature, searches on these were undertaken and their role is discussed.

### 2.2.1 Grip strength and mortality

Grip strength in mid-life is predictive of functional limitation in later life as well as mortality. One study on healthy men aged 45 to 68 years found that grip strength predicted functional limitations and disability among survivors 25 years later (Rantanen et al., 1999). A systematic review of published and unpublished data on objective measures of physical capacity, including grip strength, found that grip strength predicted mortality after adjustment for age, sex and body size (Cooper et al., 2010). Similar findings were obtained using the Danish 1905 cohort study (Oksuzyan et al., 2010).The Hertfordshire Cohort Study found grip strength was associated with an increased number of ageing markers than chronological age (Syddall et al., 2003). Grip strength was found to be associated with mortality independently of physical activity and muscle mass in a study of male participants from the Baltimore Longitudinal Study of Aging, suggesting that grip strength captures aspects of physiology beyond strength (Metter et al., 2002). Similarly, a study based in Britain found that body composition and muscle size did not explain the associations the authors found between grip strength and mortality (Gale et al., 2007). This study also found that the rate of loss of muscle strength was more important than the initial level of grip strength in men younger than 60 years old but strength was more important than the rate of decline in men older than 60 years. Research using the Prospective Urban-Rural Epidemiology (PURE) study found that grip strength better predicted all-cause and cardiovascular mortality than systolic blood pressure, it also predicted myocardial infarction and stroke though the mechanisms through which muscle strength was associated with these is unclear (Sayer et al., 2015). One study which looked at muscle strength (as measured by

grip strength as well as other indicators) and muscle mass as predictors of mortality found that muscle mass did not explain the association between muscle strength and mortality (Newman et al., 2006). The authors suggest that this may be due to the decline of strength being related to hormonal factors such as testosterone and insulin-like growth factor-1. The authors also posit that the decline of motor neurons may lead to a decrease in type-2 muscle fibres with retention of muscle size. A study in Japan using the Adult Health Study (AHS) found that grip strength predicted all-cause mortality in men and women, and heart disease, stroke and pneumonia in men after twenty years follow up (Saski et al., 2007). Few studies have examined grip strength in populations outside of mid-life and old age, but research using the Swedish Military Conscription Register found that grip strength measured in men aged 16 to 25 years was predictive of coronary health disease and stroke in later life (Silventoinen et al., 2009). More research using the same study found that grip strength was associated with all cause (excluding cancer) mortality and cardiovascular mortality independently of body mass index (BMI) and blood pressure (Ortega et al., 2012). Overall, these findings suggest that grip strength is an important determinant of health in old age whether via muscle function directly, or through being associated with other determinants.

### 2.2.2 Grip strength and SEP

#### *2.2.2.1 Grip strength and childhood SEP*

There have been contradictory findings from research on childhood SEP and grip strength. Some studies have found an association between them (Guralnik et al., 2006; Starr and Deary, 2011; Hurst et al., 2013; Murray et al., 2013), while others report no association (Syddall et al., 2009; Birnie et al., 2011; Strand et al., 2011b). Strong associations have been found between birth weight and grip strength (Kuh et al., 2002; Dodds et al., 2010). Kuh et al (2002) found that birth weight predicted mid-life grip strength; a plausible explanation for this is that skeletal size is related to grip strength. Birth weight remained significant even controlling for height and weight in later life; suggesting that prenatal influences on muscle development may last throughout life. Kuh et al (2002) discuss how studies in animals support the hypothesis that the number of muscle fibres is set in utero or in very early infancy. However, some studies suggest that grip strength is affected by development beyond the in-utero period. Dodds et al (2010) examined grip strength in children aged four years of age using the Southampton Women's Survey. The relationship

between birth weight and grip strength became insignificant when adjustment was made for current height and weight. The relationship was attenuated further when absolute and percentage lean mass were adjusted for instead of height and weight. The authors concluded that early influences on muscle growth and development impact on grip strength in children and in adults. These findings suggest that childhood and development in utero may be critical or sensitive periods for the development of grip strength.

#### *2.2.2.2 Grip strength and adult SEP*

Research on adult SEP and grip strength has also produced contradictory results. Disadvantaged adult SEP was associated with chair rise time and standing balance but not with grip strength in research using the Medical Research Council (MRC) National Survey of Health and Development (NSHD) or 1946 Birth Cohort (Kuh et al., 2005). The authors theorised that these measures were more sensitive to SEP than grip strength as chair rise time and standing balance measured neuromuscular speed and control as well as muscle strength. A further study used MRC NSHD to explore when in life a social gradient in disability is set (Strand et al., 2011a). The authors used a life course perspective and included measures of SEP from childhood, early adulthood and adulthood. However, no association between grip strength and SEP measured at any of these life stages was found. It was hypothesised that this could be due to a social gradient in grip strength having not yet emerged in that population who were aged 53 when grip strength was measured. Area deprivation across the life course was not associated with grip strength though it was associated with chair rise time and balance in another study using MRC NSHD (Murray et al., 2013). The authors hypothesised that area deprivation was associated with poorer health behaviours, which may explain the association with chair rise time and balance but that grip strength may be influenced by a different physiological pathway.

In contrast, a number of other studies have found an association between adult SEP and grip strength. An association was found between grip strength and wealth but not between it and income, occupation or education in research using the Survey of Health, Aging and Retirement in Europe (SHARE), which surveys those aged 50 and older (Hairi et al., 2010). The authors questioned whether this finding was due to the increased importance of accumulated wealth in retirement in comparison to the other SEP measures. Syddall et al (2009) found that not having access to a car and not

owning one's home were related to poorer grip strength after adjusting for current and childhood SEP using the Hertfordshire Cohort Study. Researchers using SHARE, the English Longitudinal Study of Aging (ELSA) and the Health and Retirement Study (HRS) in the United States took a different approach. The authors explored whether income inequality was associated with grip strength (De Vries et al., 2012). Historic exposure to income inequality, as measured by the Gini Coefficient of each country between 1960 and 2006, was associated with poorer grip strength among the older population even when controlling for individual SEP (De Vries et al., 2012). Height explained much but not all of this association. Each of these examples could be considered supportive of the materialist explanation of health inequalities. Using the SHARE data, one study which explored how different theories of the life course were associated with grip strength, found that occupational position in mid-life was associated with level of grip strength (though not its decline) for men but not for women (Kröger et al., 2016). The difference in findings on the basis of gender may be due to the differing relevance of occupation as an SEP indicator to older women.

### 2.2.3 Grip strength and health behaviours

#### Physical activity

There is some evidence to suggest that physical activity is socially patterned. A systematic review found that participation in moderate and vigorous physical activity was higher at the advantaged end of the SEP spectrum in comparison to the disadvantaged, but evidence of a gradient across SEP groups was less consistent (Gidlow et al., 2006). Those with disadvantaged SEP consistently report higher rates of obesity and overweight (Howel et al., 2013). Many of the studies reviewed controlled for physical activity as a confounder, however none considered physical activity as a mediator of the association between SEP and grip strength. Physical activity throughout the life stages was found to affect grip strength in later life in research using MRC NSHD (Dodds et al., 2013). This study controlled for SEP rather than considering physical activity as a mediator of it. Previous research on the MRC NSHD when the cohort were aged 53 only found an association between physical activity and grip strength for men (Kuh et al., 2005). Evidence of an association between intense physical activity and grip strength was found for men but not women in research using the Lifestyle Interventions and Independence for

Elders study (Bann et al., 2015). Conversely, an association between leisure time physical activity and grip strength was found in women but not men, in the Hertfordshire Cohort Study (Martin et al., 2008). The age of the sample and definition of physical activity used may be a factor in the difference in findings across studies.

There are only two examples in the literature of research exploring the association between physicality of occupation and grip strength (Liu and Chu, 2006; Russo et al., 2006). Both of these were on small samples of older populations. Russo et al (2006) found a negative association between manual occupation and grip strength while Liu and Chu (2006) found a positive association. Neither of these studies conceptualised manual or physically demanding occupation as an indicator of social class. Eight of the studies reviewed here measured SEP with manual occupation, six of these found no association between this measure and grip strength. It is possible that the physical activity associated with manual occupation is protective against weaker grip strength, which otherwise one may expect to be associated with disadvantaged SEP.

### Diet

Some studies have found indication of a potential association between grip strength and diet. A borderline association, significant at an alpha level of 10%, between plasma carotenoids (an indicator of fruit and vegetable consumption) and grip strength was found in the InCHIANTI (Invecchiare in Chianti or Aging in the Chianti area) study (Lauretani et al., 2008). Adjustment was made for demographic and socio-economic characteristics as well as health behaviours. Significant associations were found between plasma carotenoids and measures of hip and knee strength (Lauretani et al., 2008). This could be seen as indicative of an association between fruit and vegetable consumption and muscle strength. A randomised control trial on a group of 83 healthy older adults found no significant association between high intake of fruit and vegetables (five portions a day) and grip strength though there was a positive non-significant trend towards increased grip strength in the intervention groups (Neville et al., 2013). No significant association was found between fruit and vegetable consumption and grip strength in a study on adolescents (Neville et al., 2014). Similarly, an association between grip strength and diet though this attenuated with the adjustment of other health behaviours in research using Whitehall II (Sabia et al., 2014). The literature suggests there is potentially a weak association between diet and muscle function.

### 2.2.4 Overview of literature on SEP and grip strength

Table 1 presents the findings of the studies on grip strength and SEP discussed thus far. It shows the author and year of the research, the sample size (n), age or its range, the name of the study, and the childhood, early adulthood and adult SEP measures used where applicable. It shows whether a significant association (sig) was found and if so, which SEP measures the association was with as well as whether height (H) and health behaviours (HB) were adjusted for in modelling. Of the 14 papers summarized, seven found a significant association between grip strength and some measure of SEP and two found a significant association with a composite measure of physical function including grip strength. However, it is also apparent that some of these studies used very small and unrepresentative samples. Various combinations of different measures are used as SEP indicators in these studies. Six of these studies use the Register General Social Class (RGSC) classification of occupations, which divides occupations into six classes, but is mainly used to differentiate between manual and non-manual occupations. One other study used elementary occupation as defined by the International Standard Classifications of Occupations (ISCO-88) which largely refers to manual occupations. Using a manual / non-manual SEP measure to explore grip strength may be problematic as the physicality of manual labour may protect against the effect of disadvantaged SEP, for men in particular. Different age groups have been used to investigate SEP and grip strength; this could be a factor in the contradictory findings of these papers. SEP differences in grip strength may deepen or converge over time. The meta-analysis conducted by Birnie at al (2011) had a wide age range through including the Swedish Military Conscription Register as well as cohorts in mid and later life. However, it did not conduct research on participants across all ages within the range of the meta-analysis.

Table 1 Review of literature on SEP and grip strength

| Author & Year | N | Age | Name of Study | Childhood SEP | Early adulthood | Adult SEP | Sig | Sig SEP measure | H | HB |
|---|---|---|---|---|---|---|---|---|---|---|
| **(Rautio et al., 2005)** | 103 | 75; 80; 85 | Evergreen Project | n/a | n/a | Education, income | Y | Borderline for income | N | Y |
| **(Syddall et al., 2009)** | 3225 | 59 to 73 | HCS | Paternal occupation (RGCS) at birth | n/a | Own (for men) or husband's (for women) occupation (RGSC), education, cars, tenure | Y | Cars & tenure | Y | N |
| **(Kuh et al., 2005)** | 2956 | 53 | MRC NSHD | n/a | n/a | Occupation (RGSC) | N | | Y | N |
| **(Strand et al., 2011a)** | 2956 | 53 | MRC NSHD | Maternal education, paternal occupation (RGSC) when 4yrs | Education | Head of household's occupation (RGSC) | N | | Y | N |
| **(Guralnik et al., 2006)** | 2767 | 53 | MRC NSHD | Parental education & occupation (RGSC), housing quality, adequate shoes when 4 yrs. | n/a | Occupation (RGSC) | Y (comp) | Paternal occupation & maternal education | Y | Y |
| **(Murray et al., 2013)** | 2300 | 53 | MRC NSHD | Paternal occupation (RGSC), area deprivation when 4 yrs. | Occupation, education, area deprivation | Occupation (RGSC), area deprivation | N | | Y | N |
| **(Starr et Deary., 2011a)** | 191 | 79, 83 & 87 | Lothian Birth Cohort (1921) | Parental education & occupation (RGSC) when 11yrs | n/a | Occupation (RGSC), education | Y | Adult SEP & father's education | Y | N |
| **(Deeg et al., 1992)** | 240 | 69 to 71 | Koganei Study | n/a | n/a | Occupation, education | Y (comp) | SEP & physical function | N | Y |
| **(Quan et al., 2013)** | 218 | 65+ | HAS | n/a | n/a | Education, income | Y | Income & education & grip strength | Y | Y |
| **(Hairi et al., 2010)** | 27351 | 50+ | (SHARE) | n/a | n/a | Occupation, education, income, wealth | Y | Wealth and grip strength | Y | Y |
| **Russo et al., 2006)** | 364 | (mean 85.9 (4.9) | ilSIRENTE study | n/a | n/a | Occupation (duration & physical stress) | Y | Manual work & grip strength | N | Y |
| **Rantanen et al., 1992)** | 112 | 50 to 60 | n/a | n/a | n/a | Education | N (comp) | | Y | Y |
| **(Birnie et al., 2011)** | 1,061,855 | 18-79 | Multiple | Parental education, paternal occupation; economic environment | n/a | Head of household's occupation, education (where available) | N | | Y | N |
| **(Kröger et al., 2016)** | 5,108 | 65 - 90 | SHARE | Parental occupation (manual) | Occupation (ages 15-35) (manual) | Occupation (36-64) (manual) | Y (for men) | Midlife occupation & grip strength for men | N | N |

### 2.2.5 Conceptual position

Previous research on grip strength and SEP has produced contradictory results using a variety of SEP measures and age groups. However, no existing research has investigated SEP and grip strength in a sample with a full adult age range. Research using older age groups could experience survivor bias. Furthermore, some of the studies reviewed only considered the association between adult SEP and grip strength without considering life course influences or differences in the SEP association depending on life stage. The aim is to contribute to the empirical literature by using a sample with an adult age range, selecting theoretically appropriate SEP measures and using a life course perspective. It is hypothesised that SEP affects the age of grip strength's peak or onset of decline due to socially patterned development in utero and in early childhood. There is some evidence to suggest that grip strength is set at these life stages (Kuh et al., 2002; Dodds et al., 2010). Much previous research has considered body size or height and weight as a confounder however, height and weight are also affected by childhood SEP. Hence, they are not included as confounders in this analysis. The justification for this is elaborated on in the Concepts and Measure Section (2.3.3). Exploring the association between grip strength and age by SEP enables a greater understanding of inequalities in aging through identifying when SEP differences emerge. This is important because if decline begins from a lower base and earlier in the life course then SEP differences may widen with age.

This study assumes a critical period theory of the life course and it is hypothesised that SEP in childhood is primarily important for grip strength. The potential mediating role of adult SEP and health behaviours in adulthood are also investigated. It is posited that a material lack of resources affect diet and thus development in early childhood and that SEP differences in material resources affect physical activity and diet in adulthood, which thus affect grip strength. This allows the extent to which the association between childhood SEP and grip strength is explained by adult SEP and physical activity to be estimated. As material resources are hypothesised to be important here, income is used alongside education as indicators of adult SEP. This contributes to knowledge of which are the important stages of the life course for grip strength's development, and how health behaviours can mediate this. Using separate measures of physical activity through sports and occupation, insight can be provided into whether they have similarly protective effects on grip strength or whether they operate via different mechanisms.

In order to more formally investigate whether there is evidence for the critical period hypothesis, a series of models representing different life course trajectories are compared in order to see which best fits the data. Various researchers have attempted to provide evidence for one life course theory (Holland et al., 2000, Singh-Manoux et al., 2004) however the possibility of different theoretical explanations confounding the results remains a possibility when other models have not been tested (Mishra et al., 2009). The difficulty authors have had in distinguishing between life course models conveys the interrelated and complementary nature of each of these life course models. Mishra et al (2009) developed an approach to compare each life course model to a saturated model. The authors advocated using this approach in testing different models of the life course to avoid assuming one had found evidence for one particular explanation when there may also be evidence for other explanations.

A diagram of the hypothesised pathways between SEP and grip strength is represented in Figure 3. This depicts the environment in utero and childhood SEP as influencing physical growth and development, which influences grip strength. Childhood SEP is shown as indirectly affecting grip strength through the transmission of SEP. The transmission of SEP affects achieved adult SEP and whether one works in a physically active occupation. Through learning socially patterned health behaviours in childhood, adult health behaviours are influenced. Adult SEP, physical activity in occupation and health behaviours; recreational physical activity and diet, are shown as affecting grip strength. Physical activity and diet are shown on one eclipse together for ease of presentation. Measures used to operationalize these pathways are presented in Figure 4 in the Analytical Techniques Section (2.3.4).

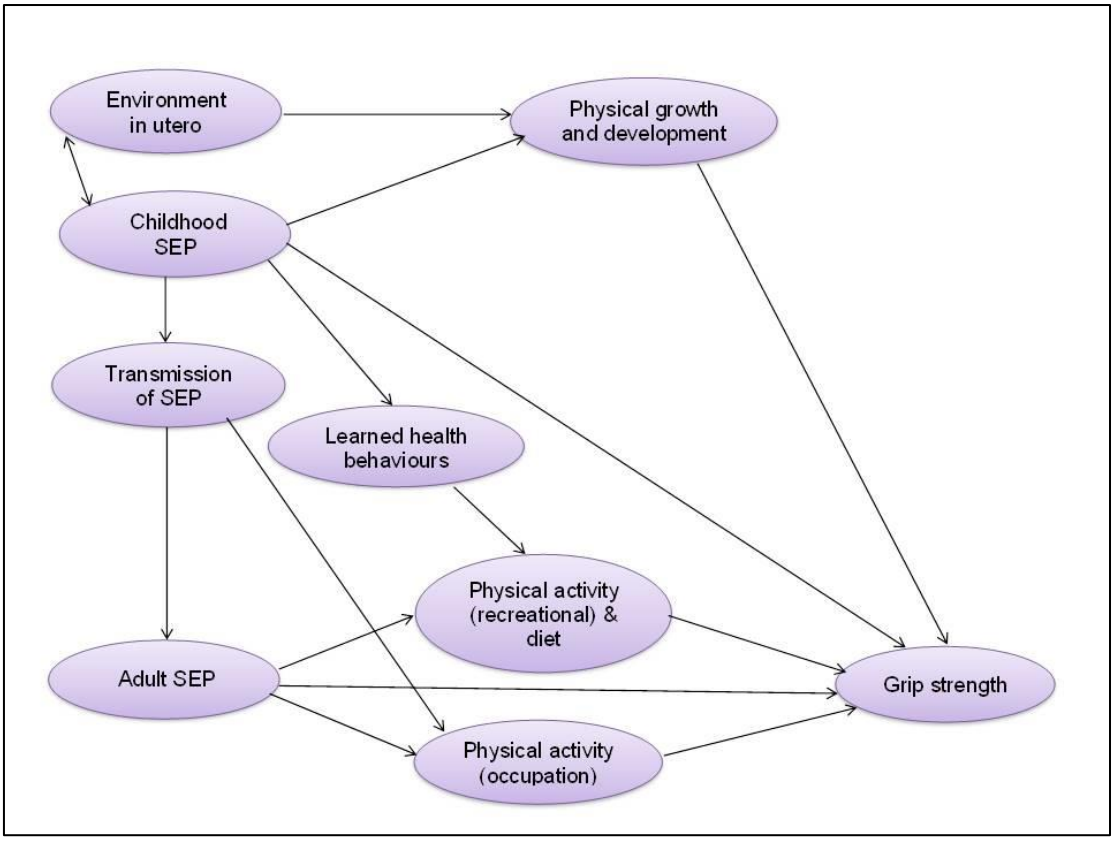
### 2.2.6 Research questions

RQ1: Does the association between age and grip strength differ by SEP?

RQ2: Is childhood SEP associated with adult grip strength? Is this association mediated by adult SEP, health behaviours and physical activity in occupation?

RQ3: Does the (childhood) critical period theory of the life course explain the social patterning in grip strength better than other life course theories?

Figure 3 Path diagram of hypothesised associations between SEP, health behaviours, physical activity and grip strength[1]




[1] Physical activity and diet are represented together for ease of presentation

## 2.3. Methodology

### 2.3.1 UK Household Longitudinal Study

Data used were collected from the UKHLS sample in wave two and from the BHPS sample in wave three. Data were combined and analysed together (the UKHLS and BHPS NHA samples are described in the Introduction to the Understanding Society Section (1.9). Response to the NHA across the two waves was 26,644. Data on SEP and health behaviours were all collected in wave two meaning that there was approximately a one year lag between the main survey and the NHA for the BHPS sample compared to approximately five months for the UKHLS sample. The implications of this are that responses to some variables may have changed over that time for BHPS members however as not all measures were available in wave three, responses were from wave two were used for all. Measures collected in wave two for the BHPS include childhood and adult SEP, as operationalised here these measures should be stable over time. Physical activity, diet and occupation may be more inclined to change though these behaviours are relatively stable over time.

### 2.3.2 Analytical sample

There were 19,292 respondents who provided a valid grip strength measurement. . Respondents with swelling, inflammation, severe pain or a recent injury or surgery in their hands were excluded from grip strength measurement. Nurses were instructed to explain and demonstrate the test to respondents with those who could not follow excluded. In answering the first two research questions, no further exclusions were applied to the sample. In answering the third research question, the analytical sample was limited to those aged older than 22 so that attainment of early adult SEP was complete.

### 2.3.3 Concepts and Measures

This section sets out how all measures were operationalised in this study.

#### Grip strength

Grip strength is commonly measured with a gripometer or dynometer. A Smedley Dynometer was used in UKHLS. Respondents with swelling, inflammation, severe pain or a recent injury or surgery in their hands were excluded from grip strength measurement. Nurses were instructed to explain and demonstrate the test to

respondents, those who could not follow the instructions were excluded. The dynometer was adjusted to fit the respondent's hand. Before testing, respondents were asked to remove any large rings, the dynometer was adjusted to fit the respondent's hand and the measurement arrow was set to zero (as it was after each measurement). Respondents stood up with their arms by their sides and holding the Dynometer with the dial facing outward. One practice measurement was taken with their dominant hand. In order to obtain a valid grip strength measurement, three successful measurements had to be taken from each hand. A trial has shown high levels of test-retest reliability for using one measurement, an average of three or the highest of three (Coldham et al., 2006), the highest grip strength measurement obtained is used here.

### Physical activity

Frequency of participating in mild[2] and moderate[3] sports was ascertained through respondents answering whether they partook in a sport or exercise (including recreational walking) and then being asked how frequently they participated in that sport. A derived variable was created which divided respondents into those who participated in mild or moderate sports at least once a week or exercised less than this. This measure is limited by being self-reported as respondents may misestimate how regularly they are active. The measure does not capture the duration of episodes of physical activity or the intensity of effort made.

Respondents were asked whether they were very, fairly, not very or not at all physically active in their current occupation. This was derived into being 'very and fairly' or 'not very and not at all' physically active. Not being currently employed was included as a dummy to retain those not working in the analysis. Having a physically active occupation is not strictly beneficial for health but it is hypothesised here that it

[2] Mild sports included snooker, pool or billiards; darts; ten-pin bowling; rambling or walking for recreation; shooting; archery; yoga or Pilates; bowls; croquet.

[3] Moderate sports included gym conditioning such as aerobics or keep fit; gymnastics; swimming or diving; cycling, bmx or mountain biking (for recreation), football, rugby or American football; track and field athletics; jogging, cross country and road-running; hill trekking, backpacking or mountaineering; golf (including pitch and putt); boxing; martial arts; water sports (including yachting, dinghy sailing, canoeing, rowing and wind surfing;) horse riding; basketball; netball; volleyball; cricket; hockey; baseball, softball or rounder's; racquet sports such as table tennis, tennis, badminton or squash; ice skating; ski-ing; motor sports; angling or fishing; archery  other sports such as triathlon, fencing, lacrosse, orienteering, curling, Gaelic sports, skateboarding, parachuting , scuba diving or 'anything else'.

may have a beneficial effect on grip strength. As this item was self-reported, it may be subject to respondents over or under estimating the physical demands of their work.

### Diet

Frequency of eating fruit and vegetables was asked in the main questionnaire as part of the module on nutrition. Respondents were asked how many days in a usual week do they eat fruit (not including fruit juice) and how many days on a usual week they eat vegetables (not including potatoes, crisps or chips). They were also asked, how many portions of fruit and vegetables they ate. This was derived into a measure of whether respondents ate at least five portions of fruit and or vegetables a day. As this was self-reported, respondents may potentially not recall dietary intake accurately.

## *Socio-economic position (SEP)*

### Childhood SEP

Maternal education is used to measure childhood SEP. UKHLS collects maternal and paternal occupation as well as father's education. Respondents were asked retrospectively about their parental occupations when they were aged 14 years; these were not used as it was hypothesised that SEP in utero and in early childhood was important for grip strength and maternal education was considered the best measure to capture this. Respondents were asked whether their mother had gone to school at all; left without qualifications; gained some qualifications; achieved higher qualification; or were awarded a degree or higher. As participation in education has widened over time, reported levels of maternal education was lower for older age groups however the inclusion of age in the analysis adjusts for this. Responses of 'do not know' and 'other' were reclassified as missing. The response categories were derived into a dichotomous variable with categories of 'no schooling or qualifications' as disadvantaged childhood SEP and 'some qualifications or post school qualifications as advantaged childhood SEP'. Maternal education was asked of some GPS respondents in wave one and others in wave two, while some members of the BHPS sample responded to maternal education in wave 13 of BHPS. All responses were combined into one variable.

### Adult SEP

Education is used as the primary measure of SEP in this thesis, however this chapter uses both education and income. To answer the third research question of this chapter, relating to different trajectories through the life course, it was necessary to have SEP measures from childhood, early adulthood and adulthood. In order to operationalise this, education and income are treated as early adult and adult SEP respectively. Education is conceptualised as early adulthood SEP since education is completed by early adulthood for the majority of the sample and income is used to measured 'adult' SEP in the life course models. Answering this research question was limited to those aged over 22 years in order for respondents to have completed the attainment of early adult SEP, which was measured with education. The age restriction was used to allow respondents to have completed higher education or to have transitioned into adulthood. Both education and income are used to indicate adult SEP without distinguishing between early adult and adulthood in the first and second research questions.

To measure educational attainment, the derived measure of highest qualification was used which is categorised into degree, other higher qualification, A level, GCSE, other qualification and no qualifications.[4] Education was derived into a dichotomous measure of high education (A level and higher) and low education (GCSE and lower including 'other qualifications' and no qualifications).

Net household income in the month before interview is used to measure income. This income measure is a derived variable using imputed household income and collected income data. There was high non-response to the income questions, circa 20% (Buck and McFall., 2011), using the imputed household income measure reduces missing data to circa 0.3% (Knies, 2014). This was equivalised for household size using the modified Organisation for Economic Cooperation and Development (OECD) equivalence scale. Income was derived into quintiles and a

[4] Degree includes masters and PhD level education, graduate and foundation degrees, graduate membership of a professional institute and PGCE. Other higher degree refers to having a diploma in higher education, nursing or medical qualification or an 'other higher degree', "GNVQ/GSVQ", "NVQ/SVQ – Level 1 – 2". A Level includes baccalaureate, AS Level, Higher Grade/Advanced Higher (Scotland) or (international) certificate of 6th year studies and modern apprenticeships and trade apprenticeships ONC/OND", "BTEC/BEC/TEC/EdExcel/LQL", and "SCOTVEC, SCOTEC or SCOTBEC". GSCE also includes O level. The category of 'other qualification' was created by combining CSE and vocational qualifications such as youth training certificate, "Key Skills", "Basic Skills" and "Entry level qualifications (Wales)" as well as clerical and commercial qualifications and "City and Guilds Certificate" and "Other vocational, technical or professional qualification". No qualifications refer to those without any qualifications.

binary measure of whether respondents were in the lowest income quintile or not was used in the analysis.

### *Covariates*

#### Age

Age was calculated from date of birth and date of interview. The variable 'age confirmed at nurse visit' is used in this analysis, it refers to age at the time of the nurse visit rather than at the time of the main interview.

#### Gender

All analyses are stratified by gender due to gender differences in grip strength.

#### Height and weight

Much of the previous research reviewed adjusts grip strength for height and weight, as they are associated with grip strength (Klum et al., 2012). Height is both socially and biologically determined. Height can be considered an indicator of early childhood nutrition and environmental exposures (Batty et al., 2009). It is known that environment in utero and in early childhood are important predictors for height. It is therefore hypothesised that controlling for height would negate the ability of maternal education to capture socially patterned environment in utero and early childhood development. Weight is associated with grip strength, particularly for men. Weight is also associated with SEP and health behaviours but is not adjusted for here. As a sensitivity check, the analysis was re-estimated controlling for height and weight. The results produced in this sensitivity check were not substantially different.

### 2.3.4 Analytical techniques

All analysis is undertaken using STATA 14 (StataCorp, 2015). Available-case analysis is used to answer research questions one and two, this excludes those who have non-response on a measure needed for one model but that case can be included if they have information in another model. This means that the number of observations varies across models. Complete case-analysis is used answering the third research question, this uses a subsample with no non-response due to this requiring comparison of model fit. Available-case analysis has the advantage of retaining sample size and making use of all available data although in both approaches bias may be introduced if data are not missing completely at random.

Missing data are low (below 2.5%) on each measure aside from maternal education, where it is 14% and for grip strength itself where it was 6.2%.

For maternal education and grip strength, whether demographic, socio-economic and health behaviour characteristics were related to non-response on each measure was explored. For maternal education, non-responders were more likely to be male, younger have disadvantaged adult SEP and poorer health behaviours. For grip strength, non-respondents were more likely to be older, female, to have a long-term illness or disability as well as low education (though no significant association was seen with other indicators of disadvantaged SEP) and poorer health behaviours. This suggests that there has been exclusion of those who are most disadvantaged and who have poorest health, which may result in underestimation of the true association in the population.

Data are weighted using the cross sectional weight for the combined GPS and BHPS NHA samples. The weight adjust for unequal selection probabilities and differential nonresponse to the NHA, this allows results to be generalisable to the population (McFall et al., 2013). However, the generalisability of the results from this analysis is undermined by the high level of non-response on the maternal education measure and to a lesser extent on the grip strength measure. Probability weights are used in linear regressions while analytical weights are used for descriptive statistics.

The aim of investigating whether grip strength's association with age varied by SEP, required specifying the shape of the association between grip strength and age. Scatter plots were initially used to investigate age and grip strength and it was assumed that the association was quadratic. Fractional polynomials were used to test whether a more complex function of age better fitted the association. As this required reducing the simplicity of the models, Aikake's Information Criterion (AIC) was used to examine whether using the functions of age suggested by the fractional polynomial were better than the simpler addition of a quadratic term.

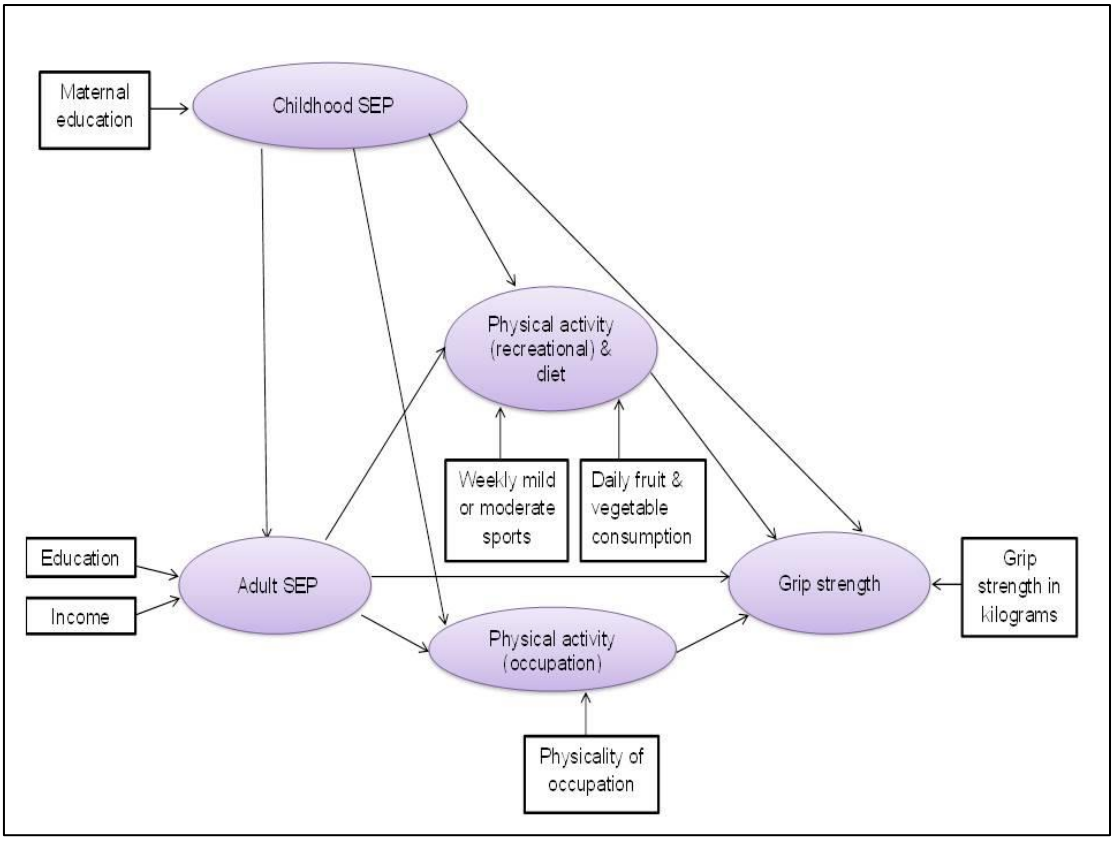
In order to explore whether the association between age and grip strength varied with SEP, regressions between age and grip strength were stratified by advantaged or disadvantaged SEP for the SEP measure in question. Differentiation was used to find the age of highest grip strength for advantaged and disadvantaged SEP groups. This identified the turning point at which grip strength stopped increasing and began decline in the sample. Subsequently, instead of stratifying by SEP, age was regressed on grip strength with an interaction for the SEP measure in question. This allowed comparison of the slopes for advantaged and disadvantaged SEP. The age

measures were centred by age of highest grip strength for advantaged and disadvantaged SEP and the constant was suppressed so coefficients produced by the model showed the predicted mean grip strength for advantaged and disadvantaged SEP groups at the age of highest grip strength. The predicted probabilities from regressions stratified by disadvantaged and advantaged SEP are plotted to illustrate the SEP differences in grip strength's trajectory. These models were stratified by gender.

To explore whether the association between childhood SEP and grip strength was mediated by adult SEP, diet, and physical activity (through sports and occupation), items were added to regressions and how the association with childhood SEP attenuated was assessed. In addition to stratifying by gender, the male and female subsamples were split at the age of grip strength's peak. It was hypothesised that the associations may differ based on whether grip strength was in 'growth' or 'decline'. Thus regressions were run separately for men and women, before and after peak grip strength.

Figure 4 presents an updated depiction of the hypothesised associations presented in Figure 3 where only those pathways that can be measured here are shown with the variables for measurement. The path diagram shows a direct association between childhood SEP, measured by maternal education, and grip strength. Adult SEP, measured by education and income, is shown as a mediator between childhood SEP and grip strength. Health behaviours, recreational physical activity and fruit and vegetable intake, as well as physical activity in one's occupation, are depicted as being influenced by childhood and adult SEP and mediating their association with grip strength. Health behaviours and physical activity through occupation are depicted as mediating the association between adult SEP and grip strength in the diagram but focus here is on how they mediate the association with childhood SEP.

Figure 4 Path diagram of hypothesised associations between SEP, health behaviours, physical activity and grip strength including measures[5]




The third research question explores whether there was evidence to support a critical period model of the life course as best explaining SEP differences in grip strength. In order to test more formally whether a childhood critical period or another theory of the life course best explained the association found between grip strength and SEP, a series of linear regression models representing different theories of the life course are compared. Childhood SEP was measured with maternal education, early adulthood SEP with education and adult SEP with income. To operationalise the critical period models, three regressions were fit including a measure of disadvantaged SEP at each of the three life stages. Whether SEP was advantaged or disadvantaged at other life stages was not restricted, as this should not be important for the outcome given the critical period hypothesis. Social mobility was measured by specifying all possible trajectories between the three periods. To operationalise the accumulation hypothesis, a 'strict' and 'relaxed' version of this were tested. Both strict and relaxed models included SEP measures at each stage of the life course. In the strict model, the coefficients for each SEP measure were

[5] Physical activity and diet are shown together for ease of presentation

constrained to be equal to each other. The basis for this is that theoretically the effect of disadvantaged SEP in each life stage should be equal to one another, when it is experienced should not be relevant. The duration of disadvantaged SEP is hypothesised to be of primary importance. The relaxed model allows the coefficients to vary so that the effect of adversity could vary between life stages.

Each of these models was compared to a saturated model using a likelihood ratio test. The saturated model included all possible permutations of disadvantaged and advantaged SEP at each stage of the life course. A *p*-value greater than 0.05 means that the model is not significantly different to the saturated model and thus it performs just as well as the more complex saturated model. This is considered indicative of support for the life course theory corresponding to that model. A null model containing the age terms without any SEP measures was also tested. For the null model, obtaining a *p* value of greater than 0.05 would indicate that the null model is not significantly different to the saturated model. This implies that the null model performs as well as the saturated model and does not support the hypothesis that SEP is associated with grip strength. These models are stratified by age and gender.

### *Sensitivity checks*

A sensitivity analysis was conducted due to data on income, health behaviours and occupation being collected one wave prior to the NHA for the BHPS sample and in the same wave for the UKHLS sample. First, analysis was rerun including a control variable based on whether respondents were part of the BHPS or UKHLS samples. A further check was undertaken by running the analysis separately for the BHPS and UKHLS subsamples. [6]

[6] Please see Appendix G for a brief overview of results from sensitivity checks.

## 2.4. Results

### 2.4.1 Description of grip strength respondents

Table 2 below shows the socio-demographic profile of grip strength respondents. The mean age of grip strength respondents was 47.7 (standard deviation (SD) 18.6). Non-response to childhood SEP was high at 14%. Among those respondents who provided grip strength measurement, 39.9% reported disadvantaged childhood SEP. There were 49.8% who reported low education and 20.8% were in the lowest income quintile. There were 36.8% of grip strength respondents who reported low levels of recreational physical activity. Twenty-two percent reported low physical activity in their occupation and 41.7% reported not currently being employed. In terms of diet, 78.3% of grip strength respondents did not eat at least five portions of fruit or vegetables a day.

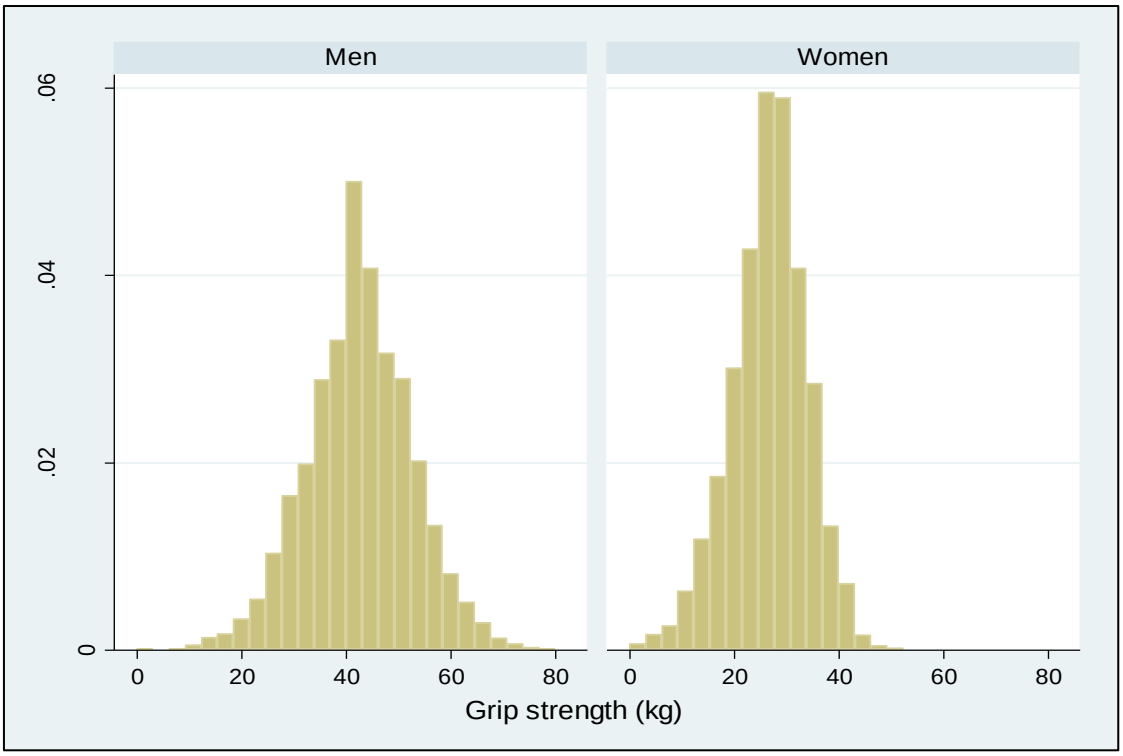
Table 2 Age, gender, socio-economic and health behaviour characteristics of NHA respondents and grip strength respondents

| | **Grip strength respondents** |
|---|---|
| **n** | 19,292 |
| **Age (years)** | |
| **Mean (SD)** | 47.7 (18.6) |
| **Gender** | |
| Female | 55.0% |
| **Childhood SEP** | |
| Disadvantaged childhood SEP | 39.9% |
| Missing | 13.9% |
| **Adult SEP** | |
| Education | |
| GCSE or lower | 49.8% |
| Missing | 1.3% |
| Income | |
| Low income | 20.8% |
| Missing | 0.5% |
| **Physical activity (recreational)** | |
| Low physical activity | 36.8% |
| Missing | 1.0% |
| **Physical activity (occupation)** | |
| Low physical activity | 22.0% |
| No occupation | 41.7% |
| Missing | 0.5% |
| **Diet** | |
| Poor diet | 78.3% |
| Missing | 1.5% |

### 2.4.2 Distribution of grip strength

Figure 5 shows the distribution of grip strength for men and women. For men the mean grip strength was 42.8kg (SD 10.3) ranging from 0kg to 80kg. The distribution was more evenly spread than for women. For women the mean grip strength was 26.7kg (SD 7.1) ranging from zero to 55.[7] Neither distribution was particularly skewed; the median for men was 43.0kg and 27.0kg for women. For women, there is greater clustering around the mean and for men, there is greater variance. There were 1,365 participants (6.6%) with no valid grip strength measurement.

Figure 5 Distribution of grip strength for men and women




[7] Respondents with a grip strength measurement of zero were investigated as this may be considered an implausible measurement. However, of the eight in total who reported highest grip strength measures of zero, seven of these also reported a limited disability or illness and the one who did not was very elderly. Thus these were considered plausible results and retained in the analysis.

### 2.4.3 Does the association between grip strength and age differ by SEP?

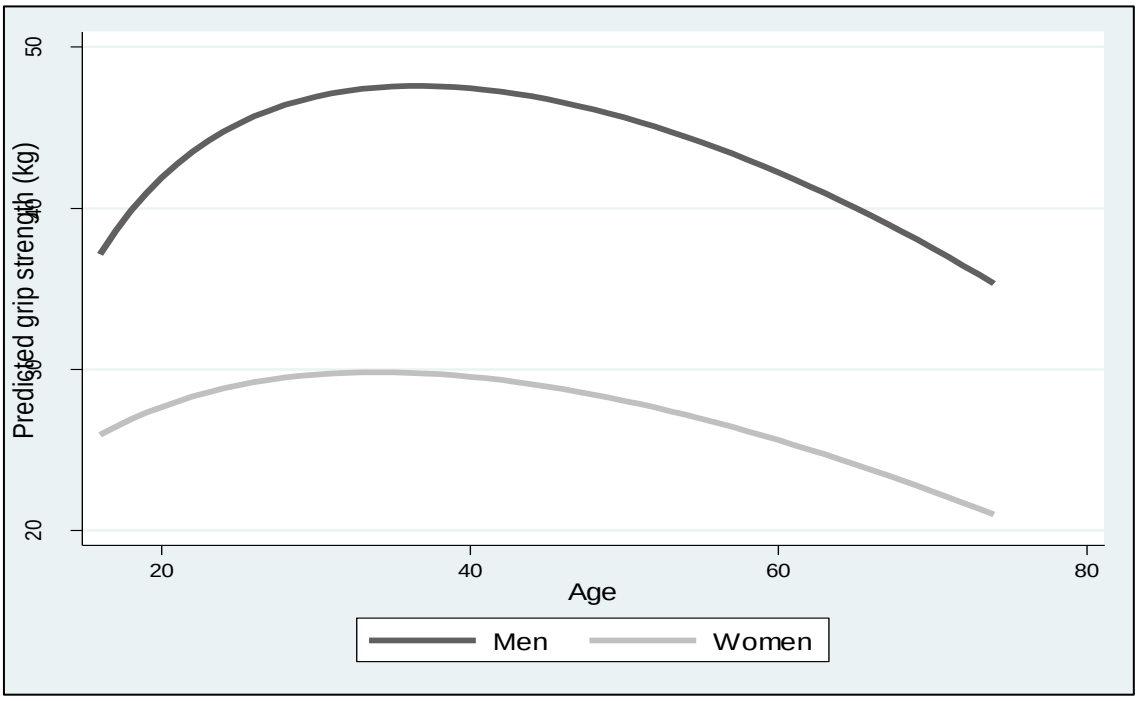
In order to explore whether the association between age and grip strength differed on the basis of SEP, it was necessary to first specify the association between grip strength and age. Using fractional polynomials to assess this association indicated that age to the power of -1 and age to the power of 2 was the best fit for men, and using age to the power 0.5 and age in its linear form was the best for women. These models were compared to simpler quadratic functions using AIC and for both men and women the model suggested by the fractional polynomial had a marginally smaller AIC which indicates a better fitting model. The probability that model with the smaller AIC was a significantly better fit was tested.[8] For both men and women, the model suggested by the fractional polynomial was the better model, so these were used.

#### *Association between grip strength and age*

The predicted probabilities from these regressions are shown on Figure 6; these indicate that the association between age and grip strength is not linear and there is a period of growth, a 'peak' and then decline. As this data are cross sectional, it is not possible to identify whether this is the age at which grip strength was at its highest for those who are now in 'decline' or whether the highs and lows in the distribution of grip strength across the age range is a cohort effect. Nevertheless, the decline of grip strength in midlife has been evidenced in previous studies (Dodds et al., 2014) and it is reasonable to assume that grip strength does begin a 'decline' at some stage in mid-life. The pattern of association between grip strength and age differs for men and women with grip strength being higher among men across the life course. Differentiation was used to identify the age at which grip strength is highest, age 36 for men and age 34 for women. Centring the age terms for age of highest grip strength in the regressions produced a constant or predicted mean grip strength at that age of 29.8kg (29.6kg to 30.0kg) for women and 47.6kg (47.3kg to 47.9kg) for men.

[8] Please see Appendix A for results of model comparison

Figure 6 Predicted grip strength across the life course for men and women




### *Social patterning in the association between grip strength and age*

To answer the first research question, regressions are used to identify whether the association between age and grip strength differed by SEP. Table 3 shows the results derived from using differentiation to identify the age at which grip strength was highest on the basis of whether childhood SEP and adult SEP were advantaged or disadvantaged. Men with disadvantaged childhood SEP are older (approximately half a year) at the time of strongest grip strength than those with advantaged childhood SEP. Those with A level and higher education reached highest grip strength at 37.5 years in comparison to 35.7 years for those with less education. There was no difference in the age of highest grip strength on the basis of income for men. For women, the SEP differences in age of highest grip strength were more pronounced. Women who reported advantaged childhood SEP had highest grip strength at 35.3 years in comparison to 33.3 for those with disadvantaged childhood SEP. The difference in age of highest grip strength was just above two years on the basis of education. Peak grip strength was approximately two and a half years earlier for women with low income.

Table 3 Age of highest grip strength by SEP in men and women

| | Men | |
|---|---|---|
| **SEP measure** | **Advantaged SEP** | **Disadvantaged SEP** |
| **Childhood SEP** | 36.8 years | 37.4 years |
| **Adult SEP** | | |
| **Education** | 37.5 years | 35.7 years |
| | | |
| **Income** | 36.7 years | 36.7 years |
| | | |
| | **Women** | |
| **SEP measure** | **Advantaged SEP** | **Disadvantaged SEP** |
| **Childhood SEP** | 35.3 years | 33.3 years |
| **Adult SEP** | | |
| **Education** | 35.4 years | 33.2 years |
| | | |
| **Income** | 34.5 years | 32.1 years |

Table 4 presents the predicted grip strength at the age of highest grip strength for disadvantaged and advantaged SEP groups. These were produced centring the age terms for the age of highest grip strength and suppressing the constant so coefficients for both disadvantaged and advantaged SEP terms display the predicted grip strength at the age when grip strength is highest. The interaction terms in these regressions were not significant indicating that the associations between age and grip strength were not significantly different on the basis of SEP.[9]

Grip strength was 1.3kg higher for men with advantaged childhood SEP in comparison to those with disadvantaged childhood SEP. The difference on the basis of education was 0.8kg; however, this was in the opposite direction to that hypothesised with those with lower education having higher grip strength. Men with high income had a predicted grip strength of 47.9kg (47.6kg to 48.3kg) at its peak while men with low income had a predicted grip strength of 45.9kg (45.0kg to 46.7kg). For women, advantaged childhood SEP was associated with a grip strength of 30.2kg (29.9kg to 30.5kg) while disadvantaged childhood SEP was associated

[9] Please see Appendix B for the full results from these regressions.

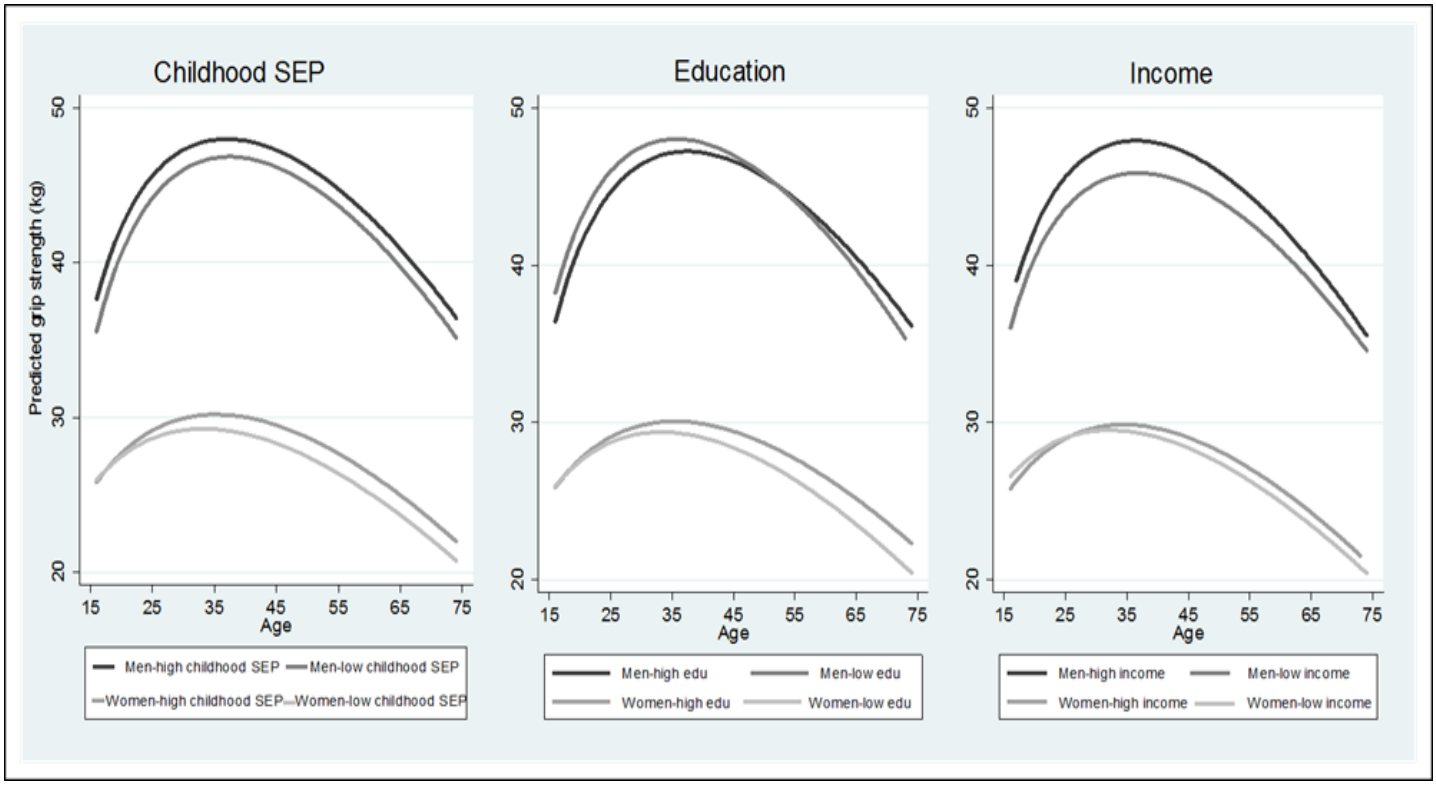
with a predicted peak grip strength of 29.3kg (28.9kg to 29.7kg). Women with high education had a predicted grip strength 0.7kg stronger than those with low education. Women who reported having a low income had slightly lower (0.4kg) predicted grip strength at peak.

Table 4 Predicted grip strength at age of highest grip strength by SEP in men and women

| **SEP measure** | **Men** | |
|---|---|---|
| | **Advantaged SEP** | **Disadvantaged SEP** |
| | Predicted grip strength at peak (95% CI) | Predicted grip strength at peak (95% CI) |
| **Childhood SEP** | 48.0kg (47.5kg to 48.5kg) | 46.8kg (46.2kg to 47.4kg) |
| **Adult SEP** | | |
| **Education** | 47.2kg (46.8kg to 47.7kg) | 48.0kg (47.5kg to 48.5kg) |
| **Income** | 47.9kg (47.6kg to 48.3kg) | 45.9kg (44.9kg to 46.7kg) |
| | | |
| **SEP measure** | **Women** | |
| | **Advantaged SEP** | **Disadvantaged SEP** |
| | Predicted grip strength at peak (95% CI) | Predicted grip strength at peak (95% CI) |
| **Childhood SEP** | 30.2kg (29.9kg to 30.5kg) | 29.3kg (28.9kg to 29.7kg) |
| **Adult SEP** | | |
| **Education** | 30.1kg (29.8kg to 30.3kg) | 29.4kg (29.1kg to 29.7kg) |
| **Income** | 29.9kg (29.7g to 30.1kg) | 29.5kg (29.1kg to 30.0kg) |

Figure 7 presents predicted probabilities for grip strength for men and women, stratified by disadvantaged and advantaged SEP for each measure. An age cut off was not used in the models but the predicted probabilities are only shown up to age 75. For childhood SEP, the difference in grip strength between advantaged and disadvantaged is consistent for men across the life course. For women, the grip strength trajectories diverge after early adulthood. Men with low education have higher predicted grip strength in early adulthood but the trajectory inverts in later life. The differences based on education become steeper at older ages for women. Grip strength peaks at a lower level for men with low income though the trajectories converge in later life. There is less growth and a longer decline for women with low income.

Figure 7 Trajectory of grip strength across the life course by childhood SEP, education and income for men and women



### 2.4.4 Do adult SEP and health behaviours mediate the association between childhood SEP and grip strength?

In order to explore whether the association between childhood SEP and grip strength is mediated by adult SEP and health behaviours, a series of regressions were used to investigate how coefficients are attenuated. Table 5 below presents descriptive statistics on the SEP and health behaviours measures for respondents split at the age of peak grip strength, 37 for men and age 34 for women. Advantaged childhood SEP, high education and high income were all more prevalent for the younger age groups. Slightly more women than men are in the lowest income quintile. Young men have the highest participation in sports and older women have the lowest. Conversely, younger men have the lowest fruit and vegetable intake while older women have the highest. Approximately one fifth of all groups report having a physically inactive job. Older women had the highest proportion not working.

Table 5 Socio-economic and health behaviour characteristics by sex and age

| | Men under 37 | Men 37 & older | Women under 35 | Women 35 & older |
|---|---|---|---|---|
| **n** | 2,076 | 7,006 | 2,481 | 9.095 |
| **Childhood SEP** | | | % | % |
| Disadvantaged childhood SEP | 14.4% | 50.1% | 16.2% | 50.3% |
| Missing | 18.6% | 13.0% | 17.4% | 11.6% |
| **Adult SEP** | | | % | |
| Low education | 40.7% | 50.2% | 37.3% | 57.6% |
| Missing | 4.3% | 0.2% | 3.2% | 0.2% |
| Low income | 19.3% | 19.2% | 23.0% | 21.6% |
| Missing | 1.3% | 0.3% | 1.0% | 0.2% |
| **Physical activity (recreational)** | | | | |
| Low physical activity | 19.0% | 37.9% | 31.6% | 44.4% |
| Missing | 1.5% | 1.0% | 1.0% | 0.8% |
| **Physical activity (occupation)** | | | | |
| Low physical activity | 23.1% | 23.5% | 21.1% | 21.0% |
| No occupation | 27.3% | 42.4% | 37.3% | 48.1% |
| Missing | 1.4% | 0.2% | 1.0% | 0.1% |
| **Diet** | | | | |
| Poor diet | 84.5 | 77.8 | 81.1 | |
| Missing | 3.5 | 1.0 | 2.6 | 0.6 |

### *Childhood SEP, adult SEP, health behaviours and grip strength in men aged less than 37 years*

Table 6 shows results from regressing childhood SEP on grip strength, followed by hypothesised mediators of this association. The table shows the number of observations in each model (n), the R-squared statistic (R2), the coefficient (_b) and the 95% confidence intervals (95% CI). However, no association is found between childhood SEP and grip strength for young men. The addition of adult SEP measures made little difference to this. Low education was positively associated with grip strength, this produced a coefficient (and 95% confidence intervals) of 2.089kg (0.934kg to 3.244kg). Low income was negatively associated with grip strength; those with low income had grip strength 2.024kg (3.481kg to 0.567kg) weaker than the reference group. Adding health behaviours completely attenuated the association seen with low income though the positive association between grip strength and low education remained. Low recreational physical activity was associated with grip strength approximately one and half kilograms weaker. No association was found here between diet and grip strength. A physically active occupation was associated with stronger grip strength; 95% confidence intervals ranged between1.639kg and 4.232kg, while those not working had grip strength between 3.528kg and 0.022kg lower than those in a physically inactive occupation. As the association with education was in the opposite direction to that theorised, whether this was due to manual occupations was investigated and an interaction between low education and physically active occupation was added to the model. As a result, low education becomes insignificant and the significant effect of having a physically active occupation remains though the interaction is insignificant. This indicates that there is no significant positive association with low education where occupation is not also physically active.

Table 6 Attenuation of association between childhood SEP and grip strength by mediators in men aged less than 37 years

| | Childhood SEP | Childhood SEP, education & income | Childhood and adult SEP, health behaviour | Child & adult SEP, health behavior with interaction |
|---|---|---|---|---|
| **n** | 1,373 | 1,330 | 1,309 | 1,309 |
| **R2** | 0.0868 | 0.0953 | 0.1451 | 0.1471 |
| **Childhood SEP** | **_b (95% CI)** | **_b (95% CI)** | **_b (95% CI)** | **_b (95% CI)** |
| **Disadvantaged childhood SEP** | -1.176 (-2.575 to 0.223) | -1.311 (-2.719 to 0.098) | -1.012 (-2.404 to 0.381) | -0.980 (-2.368 to 0.408) |
| **Adult SEP** | | | | |
| Education | | | | |
| Low education | | 2.200*** (1.042 to 3.357) | 1.990*** (0.879 to 3.100) | 1.009 (-0.499 to 2.518) |
| Income | | | | |
| Low income | | -2.024* (-3.481 to -0.567) | -0.597 (-2.028 to 0.833) | -0.601 (-2.029 to 0.827) |
| **Health behaviors** | | | | |
| Physically active (recreational) | | | -1.656* (-3.092 to -0.220) | -1.638* (-3.078 to -0.199) |
| Poor diet | | | -0.774 (-2.313 to 0.765) | -0.768 (-2.310 to 0.774) |
| **Occupation** | | | | |
| Physically active occupation | | | 2.936*** (1.639 to 4.232) | -2.339*** (0.833 to 3.843) |
| Active job * low education | | | | 1.785 (-0.409 to 3.980) |
| No occupation | | | -2.014* (-3.758 to -0.270) | -1.775* (-3.528 to -0.022) |
| Cons | 62.819*** (51.789 to 76.849) | 64.100*** (52.547 to 75.652) | 57.308*** (45.608 to 69.009) | 57.141*** (45.4251 to 68.858) |

* p<.05, ** p<.005, ***p<.001, reference group (ref). Advantaged childhood SEP, high education, above lowest income quintile, low recreational physical activity, being in a physically inactive occupation and good diet

### *Childhood SEP, adult SEP, health behaviours and grip strength in men aged 37 years and older*

As shown in Table 7, there was a significant association between childhood SEP and grip strength for men in the older age group. Those reporting disadvantaged childhood SEP had grip strength 1.143kg (1.712kg and 0.575kg) weaker than those with advantaged childhood SEP. The addition of the adult SEP measures had only a slight attenuating effect on childhood SEP. Low education was not significant but low income was. Being in the lowest income quintile was associated with grip strength 1.330kg (2.047kg to 0.613kg) weaker. The effect of childhood SEP was still significant with the addition of the health behaviours to the model though it was somewhat attenuated, it was now associated with grip strength 0.892kg (1.474kg

and 0.311kg) weaker. Low recreational physical activity was associated with having grip strength between approximately one and two kilograms lower than the reference group. A physically active occupation was positively associated with grip strength.[10] There was no significant association found between having no occupation and grip strength.

Table 7 Attenuation of association between childhood SEP and grip strength by mediators for men aged 37 years and older

| Men aged 37 & older | Childhood SEP | Childhood SEP, education & income | Childhood and adult SEP, health behaviour |
|---|---|---|---|
| **n** | 5,304 | 5.276 | 5,201 |
| **R2** | 0.2864 | 0.2899 | 0.2979 |
| **Childhood SEP** | **_b (95% CI)** | **_b (95% CI)** | **_b (95% CI)** |
| Disadvantaged childhood SEP | -1.143*** (-1.712 to -0.575) | -1.022** (-1.601 to -0.442) | -0.892** (-1.474 to -0.311) |
| **Adult SEP** | | | |
| Education | | | |
| Low education | | -0.161 (-0.694 to 0.372) | -0.184 (-0.722 to 0.354) |
| Income | | | |
| Low income | | -1.330*** (-2.047 to -0.613) | -0.968* (-1.712 to- 0.225) |
| **Health behaviors** | | | |
| Low recreational physical activity | | | -1.607*** (-2.178 to1.036) |
| Poor diet | | | -0.068 (-0.688 to 0.552) |
| **Occupation** | | | |
| Physically active occupation | | | 1.650*** (0.934 to 2.366) |
| No occupation | | | -0.660 (-1.508 to 0.187) |
| Cons | 67.713*** (62.656 to 72.771) | 67.379*** (62.300 to 72.458) | 64.989*** (59.828 to 70.149 |

* p<.05, ** p<.005, ***p<.001, ref. advantaged childhood SEP, high education, above lowest income quintile, low recreational physical activity, being in a physically inactive occupation and good diet

[10] An interaction between low education and physically active occupation was also tested here but the interaction effect was not significant and did not change the main effects.

### *Childhood SEP, adult SEP, health behaviours and grip strength in women aged less than 34 years*

Childhood SEP was not significantly associated with grip strength for women aged less than 34 years. The addition of adult education and income to the model made no substantial difference to the childhood SEP coefficient and neither had significant associations with grip strength. Health behaviours were then added to the model containing each of the SEP measures; this made little difference to the SEP measures. Recreational physical activity and diet were associated with grip strength. Women who participated in low recreational physical activity had grip strength between 1.411kg and 0.045kg weaker than those who were more physically active. Poor diet was associated with grip strength between 1.958kg and 0.126kg weaker than those in the reference category. No association was found between grip strength and either having a physically inactive occupation and not working at all. This is shown in Table 8.

Table 8 Attenuation of association between childhood SEP and grip strength by mediators for women aged less than 34 years

| **Women aged less than 34** | **Childhood SEP** | **Childhood SEP, education & income** | **Childhood and adult SEP, health behaviour** |
|---|---|---|---|
| **n** | 1,681 | 1,625 | 1,605 |
| **R2** | 0.0221 | 0.0214 | 0.0311 |
| **Childhood SEP** | **_b (95% CI)** | **_b (95% CI)** | **_b (95% CI)** |
| Disadvantaged childhood SEP | -0.366 (-1.155 to 0.420) | -0.447 (-1.256 to 0.362) | -0.247 (-1.061 to 0.568) |
| **Adult SEP** | | | |
| Education | | | |
| Low education | | -0.212 (-0.937 to 0.514) | -0.006 (-0.774 to 0.763) |
| Income | | | |
| Low income | | 0.636 (-0.170 to 1.441) | 0.826 (-0.067 to 1.720) |
| **Health behaviors** | | | |
| Low recreational physical activity | | | -0.728* (-1.411 to -0.045 ) |
| Poor diet | | | -1.042* (-1.958 to -0.126 ) |
| **Occupation** | | | |
| Physically active occupation | | | 0.046 (-0.773 to 0.865) |
| No occupation | | | -0.354 (-1.374 to 0.667) |
| Cons | 22.281 (-6.600 to 51.163) | 25.791 (-6.464 to 58.046) | 24.023 (-9.592 to 57.639) |

* p<.05, ** p<.005, ***p<.001, ref. advantaged childhood SEP, high education, above lowest income quintile, low recreational physical activity, being in a physically inactive occupation and good diet

### *Childhood SEP, adult SEP, health behaviours and grip strength in women aged 34 years and older*

Table 9 shows childhood SEP was significantly associated with grip strength in women aged 34 and older. Disadvantaged childhood SEP was associated with grip strength 1.253kg (1.605kg and 0.901kg) weaker than the reference group with advantaged childhood SEP. Introducing adult education and income to the model attenuated this somewhat, disadvantaged childhood SEP was now associated with grip strength 0.939kg (1.300kg to 0.579kg) weaker. Low education was associated with having weaker grip strength; this produced a coefficient of -1.161kg (-1.510kg to -0.812kg). Low income was associated with grip strength ranging between 0.934kg and 0.102kg weaker. Adding health behaviours to the model attenuated childhood SEP; disadvantaged childhood SEP was now associated with having grip strength approximately two thirds of a kilogram weaker. Low recreational physical activity was associated with having grip strength 1.454kg (1.804kg to 1.104kg) weaker than those who were more active. There was a small significant negative effect associated with poor diet. Women who were not working had grip strength between 1.613kg and 0.637kg weaker than those in a physically inactive occupation while no significant association was found with being in an active occupation.

Table 9 Attenuation of association between childhood SEP and grip strength by mediators for women aged 34and older

| **Women aged 34 & older** | **Childhood SEP** | **Childhood SEP, education & income** | **Childhood and adult SEP, health behaviour** |
|---|---|---|---|
| **n** | 6,895 | 6,862 | 6,792 |
| **R2** | 0.2829 | 0.2896 | 0.3002 |
| **Childhood SEP** | **_b (95% CI)** | **_b (95% CI)** | **_b (95% CI)** |
| Disadvantaged childhood SEP | -1.253*** (-1.605 to -0.901) | -0.939*** (-1.300 to -0.579) | -0.684*** (-1.042 to-0.326) |
| **Adult SEP** | | | |
| Education | | | |
| Low education | | -1.161*** (-1.510 to -0.812) | -0.785*** (-1.134 to -0.436) |
| Income | | | |
| Low income | | -0.518* (-0.934 to -0.102) | 0.193 (-0.620 to 0.234) |
| **Health behaviors** | | | |
| Low recreational physical activity | | | -1.454*** (-1.804 to -1.104) |
| Poor diet | | | -0.363* ( -0.698 to 0.028) |
| **Occupation** | | | |
| Physically active occupation | | | 0.169 (-0.257 to 0.595) |
| No occupation | | | -1.125*** (-1.613 to-0.637) |
| Cons | -1.226 (-11.784 to 9.333) | -0.555 (-11.162 to 10.053) | 8.874 (-1.908 to 19.657) |

* p<.05, ** p<.005, ***p<.001, ref. advantaged childhood SEP, high education, above lowest income quintile, low recreational physical activity, being in a physically inactive occupation and good diet

### 2.4.4 Does the (childhood) critical period theory of the life course explain social patterning in grip strength better than other life course theories?

In answering the first and second research questions on grip strength, it was evident that SEP was associated with grip strength after it had peaked. As SEP was not important for grip strength in the younger age groups, the comparison of life course models for this group is not shown in the main results.[11] This section compares a series of models denoting various SEP trajectories through the life course. The proportions of men and women in each possible trajectory and their mean grip strength are shown in Table 10. On these tables, "*0*" denotes experiencing advantaged SEP at that life stage and "*1*" denotes experience of disadvantaged SEP. For the critical period models, SEP was not restricted to being advantaged or disadvantaged outside of that critical period and this is illustrated with a dash; '-'. Table 11 below shows the number (n) and percentage (%) of men aged 37 and older and women aged 34 and older in each possible life course trajectory as well as their mean grip strength and standard deviation (SD). In the critical period trajectories, experience of disadvantaged childhood SEP is prevalent. The social mobility trajectories show all possible permutations, the proportions that had advantaged or disadvantaged SEP all through the life course are shown for interest; these are not included in the model comparisons. Experiencing downward social mobility in early adulthood is the least prevalent response category.

[11] Please see Appendix C for proportions in each life trajectory, mean grip strength and results of model comparisons for men aged less than 37 years and women aged less than 34 years.

Table 10 Frequency and percentage of men aged 37 years and older and women aged 34 years and older in each possible life course trajectory and their mean grip strength

| | Childhood | Early adulthood | Adulthood | Men aged 37 and older | | | Women aged 34 and older | | |
|---|---|---|---|---|---|---|---|---|---|
| **Total n** | | | | 5,276 | | | 6,862 | | |
| **Critical period** | | | | **n (%)** | **Mean (kg)** | **SD** | **n (%)** | **Mean (kg)** | **SD** |
| Disadvantaged in childhood | 1 | - | - | 3,043 (57.7%) | 38.8 | 10.2 | 3,908 (57.0%) | 24.4 | 7.3 |
| Disadvantaged in early adulthood | - | 1 | - | 2,530 (48.0%) | 41.1 | 10.9 | 3,813 (55.6%) | 24.5 | 7.5 |
| Disadvantaged in adulthood | - | - | 1 | 984 (18.7%) | 39.4 | 10.5 | 1,422 (20.7%) | 24.2 | 7.6 |
| **Social mobility** | | | | | | | | | |
| Stable advantaged SEP | 0 | 0 | 0 | 1,272 (24.1%) | 44.7 | 9.8 | 1,590 (23.2%) | 28.9 | 6.5 |
| Early upward mobility | 1 | 0 | 0 | 1,086 (20.6%) | 41.2 | 9.7 | 1,040 (15.2%) | 26.7 | 6.6 |
| Disadvantaged in early adulthood only | 0 | 1 | 0 | 668 (12.7%) | 46.0 | 10.2 | 914 (13.3%) | 27.1 | 7.4 |
| Late downward mobility | 0 | 0 | 1 | 164 (3.1%) | 43.9 | 9.9 | 231 (3.4%) | 28.1 | 7.3 |
| Late upward mobility | 1 | 1 | 0 | 1,263 (24.0%) | 39.8 | 10.5 | 1,894 (27.6%) | 23.9 | 7.3 |
| Advantaged in early adulthood only | 1 | 0 | 1 | 220 (4.2%) | 39.2 | 9.2 | 186 (2.7%) | 25.2 | 7.3 |
| Early downward mobility | 0 | 1 | 1 | 126 (2.4%) | 42.9 | 10.9 | 217(3.2%) | 25.8 | 7.0 |
| **Accumulation** | | | | | | | | | |
| Stable disadvantaged | 1 | 1 | 1 | 471 (8.9%) | 37.0 | 10.5 | 787(11.5%) | 22.3 | 7.4 |

Table 11 shows the coefficients (_b), 95% confidence intervals (95% CI) and results of likelihood ratio tests (lrt) from each of the life course models and the null model compared to the saturated model. For both men and women, the null model was significantly different to the saturated model. The saturated model with all SEP measures explains significantly more variation in grip strength than the null model that just included the age terms. For men, none of the life course models had a *p* value above .05 indicating that none performed as well as the saturated model. Thus, this approach has not found evidence to support any of the life course models as best explaining variation in grip strength for men. For women, the two accumulation models performed as well as the saturated model, the relaxed accumulation model had the highest *p* value, which could be taken as indication that this is the best performing model. This supports the accumulation hypothesis of the life course as best explaining variation in grip strength in women aged 34 and older.

Table 11 Comparison of life course models to saturated model using likelihood ratio test (with model coefficients and 95% confidence intervals) for men aged 37 & older and women aged 34 & older

| | | | | Men aged 37 and older | | Women aged 34 and older | |
|---|---|---|---|---|---|---|---|
| **n** | | | | 4,580 | | 6,034 | |
| | **Childhood** | **Early adulthood** | **Adulthood** | **lrt** | **_b (CI 95%)** | **lrt** | **_b (CI 95%)** |
| **Critical period** | | | | | | | |
| Childhood | 1 | - | - | .000 | -1.153*** (-1.678 to -0.628) | .000 | -1.249*** (-1.538 to -0.859) |
| Early adulthood | - | 1 | - | .000 | -0.449 (-0.929 to 0.031) | .000 | -1.423*** (-1.732 to -1.114) |
| Adulthood | - | - | 1 | .000 | -1.457*** (-2.072 to -0.842) | .000 | -0.775*** (-1.146 to -0.405) |
| **Social mobility** | | | | | | | |
| Early upward mobility | 1 | 0 | 0 | .000 | 0.512 (-0.083 to 1.107) | .000 | -0.680** (0.266 to 1.095 |
| Disadvantaged in early adulthood only | 0 | 1 | 0 | .000 | 1.835*** (1.109 to 2.562) | .000 | 0.103 (-0.336 to 0.543) |
| Late downward mobility | 0 | 0 | 1 | .000 | 0.218 (-1.156to 1.591) | .000 | 0.827* (0.002 to 1.652) |
| Late upward mobility | 1 | 1 | 0 | .000 | -0.692* (-1.259 to -0.125) | .000 | -1.087*** (-1.428 to -0.747) |
| Advantaged in early adulthood only | 1 | 0 | 1 | .000 | -0.991 (-2.184 to 0.202) | .000 | -0.708 (-1.620 to 0.205) |
| Early downward mobility | 0 | 1 | 1 | .000 | -1.198 (-2.756 to 0.361) | .000 | 0.003 (-0.845 to 0.851) |
| **Accumulation** | | | | | | | |
| Accumulation (relaxed) | | | | .000 | | .330 | |
| | 1 | | | | -1.022*** (-1.556 to -0.487) | | -0.939*** (-1.231 to -0.535) |
| | | 1 | | | -0.161 (0.650 to 0.328) | | -1.161*** (-1.479 to -0.843) |
| | | | 1 | | -1.330*** (-1.949 to -0.711) | | -0.518* (-0.889 to -0.147) |
| Accumulation (Strict) | 1 | 1 | 1 | .000 | -0.746*** (-1.019 to -0.473) | .086 | -0.918*** (-1.085 to -0.752) |
| **Null** | | | | .000 | | .000 | |

* p<.05, ** p<.005, ***p<.001

## 2. 5 Discussion

### 2.5.1 Main findings

These results demonstrate that grip strength was at its highest at a younger age and weaker level of strength for men and women on the majority of the SEP measures tested. Before grip strength is at its highest, no evidence of social patterning was found but after grip strength peaks, it becomes socially patterned. The SEP differences in age and level of peak grip strength could be indicative of decline beginning earlier and from a weaker base for disadvantaged SEP groups. This could impact on the capacity for healthy aging for those with disadvantaged SEP. Previous research has found that the rate of decline of grip strength was more important in predicting mortality for men aged less than 60 years than the initial level of strength (Gale et al., 2007). For women, there was greater difference in the age of peak grip strength on the basis of SEP than there was for men. The gender difference in this finding may be explained by men with disadvantaged SEP being more likely to have engaged in manual labour, which could be somewhat protective. It may also imply that the SEP measures used are of differing relevance for men and women or that different mechanisms may underlie the SEP association on the basis of gender.

When considering mediation of the association between childhood SEP and grip strength, childhood SEP continued to be important after the addition of adult SEP and health behaviour measures in the older age groups. There was greater mediation of the association for women suggesting different mechanisms underlying the association by gender. This indicates that adult SEP, health behaviours and physical activity in one's occupation do not explain the association between childhood SEP and grip strength. While this study found no evidence of an association with SEP and grip strength in younger age groups, SEP may be influencing the development of grip strength prior to this. There was greater attenuation of childhood SEP's association with grip strength for women than for men suggesting social or biological processes may differ by gender.

A physically active occupation was found to be protective for men's grip strength but has no association with women's in this study. Physical activity in occupation was not conceptualised as an indicator of SEP in this thesis. In some studies exploring SEP as measured by manual occupation, no association with grip strength was found (Syddall et al., 2009; Kuh et al., 2005; Strand et al., 2011a; Murray et al., 2013). As manual occupation captures physical activity in addition to SEP, it is

possible that this prevents identification of the adverse impact of disadvantaged SEP on grip strength.

This chapter found disadvantaged SEP as measured by education was associated with stronger grip strength in young men, though the inclusion of an interaction between this and physically active occupation resulted in the association becoming insignificant indicating low education had no protective effect for those not in a physically active occupation. In a very specific sample of persons aged over 80 years in Sirente, Italy, it was found that lifetime manual occupation and higher physical stress was associated with lower grip strength (Russo et al., 2006).The positive association between a physically active occupation and grip strength was smaller in the older age group in this thesis. Physically active occupations and manual labour may be beneficial to physical function in early adulthood but may become problematic with aging.

The results from the mediation analysis could be seen as supportive of a critical or sensitive period theory of the life course. However, testing a series of models representing different life course theories did not support this. Comparing the life course models to a saturated model found that the accumulation model performed best for women and there was no support for any life course model as performing best for men. A number of studies have used a structural modelling approach to testing theories of the life course (Mishra et al., 2009, Murray et al., 2011, Robertson, 2014) on various health outcomes although only one study (Kröger et al., 2016) utilised this approach with grip strength. Findings differ between this chapter and the research by Kröger et al (2016) possibly due to the use of different age ranges and conceptualisations of SEP. The study by Kröger et al found that the best performing model for men was low occupational position at midlife and the authors did not find evidence for any life course model for women contradicting the findings of this chapter. The lack of association between grip strength and SEP as operationalised by occupational position may be due to ‘breadwinner’s’ occupation being less proximal to their own experience of SEP. Their approach also did not differentiate between strict and relaxed conceptualisations of the accumulation model or specify social mobility in the same way.

### 2.5.2 Findings in relation to other studies

In exploring age's association with grip strength and whether it varies based on SEP, this chapter builds upon the work of Dodds et al (2014) who examined the trajectory of grip strength across an age range and of researchers who explored social patterning in grip strength (Guralnik et al., 2006; Murray et al., 2013; Starr and Deary, 2011; Hurst et al., 2013; Syddall et al., 2009; De Vries et al., 2012; Kuh et al., 2005; Strand et al., 2011). Previous research has looked at grip strength decline over time in longitudinal data (Granic et al., 2016; Dodds et al., 2014; Deeg et al., 1992), though this has been over a limited follow up period and in a limited age range. Research that has looked at normative data on grip strength has not considered social patterning in the trajectory while research that has explored whether grip strength is socially patterned has not considered whether this varies with age.

Several studies exploring SEP and grip strength included health behaviours in their analysis, (Deeg et al., 1992; Rautio et al., 2005; Guralnik et al., 2006; Quan et al., 2013), however each of these considered health behaviours as a confounder to be controlled for rather than a mediator of the association. Their findings are built upon by exploring the role of adult SEP and health behaviour as mediating the effect of childhood SEP. Another contribution is that previous research used grip strength collected from cohort studies; in this thesis, grip strength was collected from a wide age range. It considers the SEP association and mediation stratified by whether grip strength is before or after its highest point in the age range allowing identification of when associations emerge.

The approach of using model comparisons as a means of finding evidence for a life course theory has been untaken before (Mishra et al, 2009; Murray et al, 2011; Robertson et al, 2014; Kröger et al, 2016). This chapter's exploration of whether there evidence for a life course explanation of social patterning in grip strength is most similar to that of Kröger et al (2016) who use a similar series of model comparisons with grip strength. The research undertaken in this thesis is novel as it tests the life course models both before and after the age of peak grip strength. In the group aged after peak grip strength, age ranges from the early thirties to oldest old, in comparison, the analysis undertaken by Kröger et al (2016) was limited to those aged between 65 and 90. This could be biased by survival bias and underestimate the effects of SEP. Additionally SEP and social mobility transitions were conceptualised and operationalised differently in the study by Kröger et al (2016).

### 2.5.3 Limitations and strengths

While this study has contributed to the literature on SEP and grip strength, there are several limitations pertaining to measurement, generalisability and the inference from these results. The childhood SEP measure used was retrospective and may be more vulnerable to measurement error. However, previous research shows that recall can be effective (Batty et al., 2005). Maternal education was used partially as a proxy for the environment in utero, which was hypothesised to be important for grip strength. While birth weight may have been a better measure to capture the in-utero environment, it is not available in UKHLS. Nevertheless, maternal education was the best available measure to approximate this. The measure of healthy diet utilised in this study was based on fruit and vegetable intake only. Evidence from previous research of an association between diet and grip strength is tenuous, this may be better explored with more detailed measurement of diet such as food diaries. The physical activity measures were also limited by being self-reported.

The UKHLS is a household based survey meaning that those who reside in care homes or other institutions were not reached by this survey. Those who are sick and elderly are more likely to have been missed by data collection and would be likely to have weak grip strength. Thus, this study may be limited by the exclusion of these populations though this limitation would apply to most large-scale collections of grip strength data. The sample used in UKHLS was representative of the British household population and using the NHA weight for the combined GPS and BHPS samples allows the results from this analysis to be generalised to the household population of Britain. However, the generalisability of these results is undermined by the large non-response to maternal education. The profile of non-respondents suggested that experience of socio-economic disadvantage and poor health was overrepresented among them. Excluding those with the most extreme experiences of disadvantage and poor health may result in underestimation.

Cross sectional data restricts the assumptions that can be made about grip strength's growth, peak and decline across the life course. The trajectory of individual's grip strength across their life course is not modelled here. The ideal way to measure this would be with repeated measures of grip strength at regular intervals, however such data are not available. Repeated measures could allow the addition of baseline controls and identification of change.

Testing different models of the life course necessitated reducing each hypothesised life course pathway into a binary variable, which may have over simplified the

theories being empirically tested. This analysis did not control for illness, which could affect grip strength. Though as frailty and illness, particularly in old age, are socially patterned, this could underestimate the importance of SEP.

This study benefitted from the unique opportunity of being able to use grip strength measurement from a representative sample of the British population over 16 years of age. The association between SEP and grip strength has not been explored across an adult age range before. This research fits with other explorations of the association between grip strength and SEP in the literature; it builds on the findings of these studies by adding a full age range and a life course perspective. This study also benefited from comparing a series of structural models; had this not been done, it may have concluded from the results to the first two research questions that late adulthood was a critical period for SEP affecting grip strength. Through using UKHLS and retrospectively collected childhood SEP measures as well as adult SEP measures, a life course perspective could be used with cross sectional data.

### 2.5.4 Future research

Longitudinal research with repeated measures of grip strength could help verify and further understand the process of growth and decline of grip strength. Research into whether certain behaviours mediate the onset of decline would be beneficial for understanding whether individuals can offset the effects of disadvantage. Further research on grip strength with more detailed and nuanced measures from childhood could explore the mechanisms through which childhood SEP affects grip strength's growth and decline. More extensive health behaviour measures, such as participation in sports or exercise from multiple stages in the life course may also help understand social patterning in grip strength. Further research into the biological processes through which childhood SEP could affect grip strength would contribute to the understanding of these findings.

### 2.5.5 Policy recommendations

Grip strength is an important determinant of frailty, morbidity and mortality, its maintenance is important for health and the ability to live independently in old age. In the context of an aging and increasingly sedentary population, interventions to ensure people, particularly in midlife, are undertaking activities to maintain and slow the decline of their grip strength may help the population to age more healthily. Participating in physical activity was associated with a positive effect on grip strength for both men and women. Participation in sports was particularly low in women aged over the age of 34, though it was one of the strongest predictors of grip strength for this group. Current UK Government recommendations are for 150 minutes of physical activity or 75 minutes of vigorous physical activity a week. Knowledge of these recommendations has been found to be low (Knox et al., 2015a; Morton et al., 2016) and socially patterned (Knox et al., 2015a). Government guidelines also include a recommendation to undertake muscle-strengthening activities twice a week. The studies reviewed (Knox et al., 2015a; Morton et al., 2016) did not measure survey participants knowledge of muscle strengthening activity though it is probable that it is also low. Promotion of recommendations related to building or maintaining muscle strength alongside those to be more active generally may be useful to alleviate the decline of grip strength and inequalities in this. As the association between childhood SEP and grip strength persisted with adjustment for health behaviours, it indicates the need to alleviate social patterning in grip strength through policies to decrease health inequalities and social inequalities more broadly.

# Chapter Three: Mediation of the association between SEP and type two diabetes

## 3.1. Introduction

Rates of diabetes are increasing with estimates suggesting that the prevalence of diabetes among adults doubled globally between the 1980s and 2010s (Bachmann et al., 2003). In Britain, it was estimated that in 2013 6% of the adult population had been diagnosed with diabetes, 90% of these diagnoses were Type Two Diabetes (T2D) and this rate is also increasing annually (Gatineau et al., 2014).

T2D occurs when the body becomes resistant to the insulin produced by the pancreas or the pancreas does not produce enough insulin to maintain a normal level of blood glucose. It develops most often in adulthood (after the age of 40 for people of a white ethnicity). Its prevalence varies by age, weight, ethnicity and genetics. T2D is now mainly diagnosed by measuring glycated haemoglobin ($HbA_{1c}$) (John, 2012). $HbA_{1c}$ indicates the level of red blood cells that joined with glucose over the preceding eight to 12 weeks. T2D is diagnosed by having $HbA_{1c}$ raised above 6.5% (48 mmol/mol). When $HbA_{1c}$ is not high enough to be determined as T2D but is above the normal level, between 5.5% and 5.9% depending on the definition used, it is often termed prediabetes and considered an indication of being likely to develop T2D. Type One Diabetes (T1D) is mainly determined by genetics.

T2D is a chronic illness, which can be managed through diet and physical activity to maintain a healthy weight as well as through medication. Poorly managed diabetes is associated with complications and morbidity including increased risk of microvascular complications such as retinopathy (blindness), neuropathy (nerve disorder), diabetes nephropathy (kidney disease), cataracts, heart failure and amputations as well as increased risk of mortality (Romao and Roth, 2008). Prescribing related to diabetes accounted for almost 10% of the total cost of prescribing in England in 2012/13 (Gatineau et al., 2014).

Incidence and prevalence of both diagnosed T2D and raised $HbA_{1c}$ have been found to be socially patterned, however less is understood about the pathways and mechanisms through which socio-economic experience affects these. This study looks at both objectively measured $HbA_{1c}$ and self-reported T2D as outcomes. $HbA_{1c}$ is used here to encompass prediabetes, undiagnosed and diagnosed diabetes and badly managed diabetes. Presence of T2D could be ascertained by self-report, reported medication usage and by $HbA_{1c}$, badly managed diabetes could include

self-reported T2D with elevated $HbA_{1c}$. While all these measures indicate presence of the diabetic outcomes, SEP may have different pathways to $HbA_{1c}$ and self-reported T2D due to SEP differences in achieving a correct diagnosis, in prediabetes and in diabetes management. Previous research has proposed material deprivation, psychosocial stress and health behaviours as explanations for inequalities in diabetic outcomes. Here, the role of each of these explanations is considered simultaneously, which is a novel approach. Understanding these mechanisms contributes to a better understanding of inequalities in T2D and $HbA_{1c}$.

This chapter begins by reviewing previous research on T2D and $HbA_{1c}$, the literature review concludes with the conceptual position, a path diagram and the specific research questions to be addressed. The methodology section introduces the BHPS, which is used to answer the research questions of this chapter. The concepts and measures are explained, attrition in the BHPS is considered and the statistical techniques used are described. The results section describes the analytical sample and presents the findings to the research questions. The discussion considers the main findings, how they relate to other studies, strengths and limitations, opportunities for further research and policy implications.

## 3.2 Literature review

The aim of the literature review is to show whether there is evidence in the literature of social patterning in T2D and $HbA_{1c}$. This section first discusses evidence of a link between SEP in childhood and adulthood and T2D and $HbA_{1c}$. Reviewing the literature, evidence emerged for material, psychosocial and health behaviour explanations as plausible explanations of this. Subsequently, research with specific focus on diabetic outcomes and material deprivation (food insecurity in particular), psychosocial stress and health behaviours is reviewed. Lastly, the value of considering both T2D and $HbA_{1c}$ is considered. This section describes how prediabetes is a risk to health worthy of investigation, and considers socially patterning in diabetes diagnosis and management.

### 3.2.1 T2D, $HbA_{1c}$ and SEP

#### *3.2.1.1. T2D, $HbA_{1c}$ and childhood SEP*

Foetal environment, birth weight, childhood SEP and socially patterned growth in childhood are associated with one another and may affect the development of diabetic outcomes. In research exploring childhood SEP and T2D and $HbA_{1c,}$ some have found evidence of an association. Childhood SEP had indirect effects on raised $HbA_{1c}$ and T2D through physical activity and waist circumference in a study using the Midlife in the US (MIDUS) survey (Tsenkova et al., 2014). Research using the British Women's Heart and Health Study (BWHHS) found that, in addition to low birth weight, childhood SEP and short leg length were associated with insulin resistance (Lawlor et al., 2003). Similarly, leg length and leg height ratio were associated with insulin sensitivity and beta cell function even with adjustment for own and parental SEP, comorbidity, weight and waist circumference in research using the Prospective Metabolism and Islet Cell Evaluation study (Johnston et al., 2013). The association of leg length with childhood nutrition suggests that nutrition and growth may be a pathway through which childhood SEP affects the development of insulin resistance. This could be indicative of a material explanation for inequalities in diabetic outcomes. In another study using the BWHHS, Lawlor et al (2007) found an initial association between childhood SEP and diagnosed diabetes, this attenuated with adjustment for adult health behaviours (Lawlor et al., 2007). Research on childhood SEP and T2D has indicated that growth and nutrition in childhood may influence this in conjunction with adult SEP and health behaviours.

#### *3.2.1.2 T2D, $HbA_{1c}$ and adult SEP*

Many studies have found an association between adult SEP and prevalence and incidence of T2D (Ross et al., 2010; Dinca-Panaitescu et al., 2011; Espelt et al., 2012; Maier et al., 2013) and raised $HbA_{1c}$ (Larsson et al., 1999; Bachmann et al., 2003; Thomas et al., 2007; Bardenheier et al., 2013; Mainous et al., 2014; Tsenkova et al., 2014). In much of these studies, the mediating role of obesity and physical activity has been investigated with some finding evidence for this. An association was found between SEP and T2D in a study in Spain, this was mediated by obesity and physical activity for women but not for men (Espelt et al., 2012). Increasing socio-economic inequality was associated with increased diagnosed T2D in women in research using the Health Survey for England (HSE) (Imkampe and Gulliford, 2011). In men, a weak association was attenuated by risk factors including BMI,

waist to hip ratio and physical activity. While the authors interpreted this as there not being an SEP association for men, another explanation could be that socially patterned health behaviours mediated it. Both individual SEP and area deprivation were associated with T2D in a German study, this did not attenuate with adjustment for obesity and physical activity (Maier et al., 2014).

Studies using $HbA_{1c}$ rather than reported T2D as an outcome have had similar findings. Greater prevalence of $HbA_{1c}$ above the normal range was found among those with disadvantaged SEP in research using the 1958 Birth Cohort Study (Thomas et al., 2007). An association between disadvantaged SEP and prediabetes was found in the HSE though the results were not consistent over time (Mainous et al., 2014). A study in the US on people aged over 50 years found that diet and physical activity mediated SEP's association with prediabetes as measured by $HbA_{1c}$ (Bardenheier et al., 2013). These studies indicate evidence of an association between SEP and both T2D and $HbA_{1c}$ with varying evidence of mediation via physical activity, diet and obesity.

However, fewer studies have considered psychosocial stress as well as health behaviours as mediators of this association. One study reviewed found obesity, physical activity and depressive symptoms mediated childhood and adult's SEP's association with $HbA_{1c}$ in MIDUS (Tsenkova et al., 2014). The association between childhood SEP, as measured by paternal occupation, and T2D completely attenuated with the inclusion of adult SEP and risk factors such as physical activity, BMI and psychosocial factors in research using the Stockholm Diabetes Prevention Programme, (Agardh et al., 2007).

These findings suggest that SEP is predictive of T2D and $HbA_{1c,}$ some of the studies reviewed considered the role of mediators, in particular health behaviours, in explaining the association. None considered a material explanation as a potential mediating pathway and few considered psychosocial explanations. The next three sections of this literature review consider the associations between material deprivation, psychosocial stress and health behaviours and diabetic outcomes.

### 3.2.2 Material pathway

Income or resources are important for health inequalities as they are required to afford the necessities of a healthy lifestyle (Pampel et al., 2010), while the absence of resources may contribute to psychosocial stress. Low income is considered an

important part of the pathway to diagnosed T2D and raised $HbA_{1c}$, it limits resources for a healthy diet and other beneficial health behaviours (Kamphuis et al., 2006, Campbell et al., 2014). Research that has focused particularly on low income found it was associated with both prevalence and incidence of T2D in research using the Canadian National Health Survey (Dinca-Panaitescu et al., 2011, Dinca-Panaitescu et al., 2012). Low income may also affect propensity to experience psychosocial stress, which in turn could affect the propensity to develop T2D. The biological pathway between psychosocial stress and T2D is discussed in Section 3.2.3 Psychosocial Pathway.

The experience of inadequate or insufficient food among households in developed countries has been conceptualised as food deprivation, food poverty or food insecurity. Food poverty has been defined as the inability to have an adequate and nutritious diet due to affordability or accessibility (Dowler, 1998). Wider definitions may include reference to affordability of activities considered a social norm such as eating out (Friel, 2004). Food security means that people have consistent access to sufficient, safe and nutritious food to meet their dietary needs and food preferences for an active and healthy life with confidence for the immediate and long-term future (FAO, 1996). Much research on diabetes and food insecurity in the United States (Seligman et al., 2012; Berkowitz et al., 2013; Berkowitz et al., 2014; Gucciardi et al., 2014; Sattler et al., 2014) has utilised the development of a food insecurity scale by the United Sates Department of Agriculture. This scale was developed to capture this particular dimension of poverty. While income poor households are most likely to experience food insecurity, some income poor households can be food secure while some households above the poverty line experience food insecurity.

Previous research has found food insecurity to be associated with both T2D and raised $HbA_{1c}$. Severe food insecurity was associated with T2D even with adjustment for physical activity and BMI in research using waves 1999 to 2002 of the National Health and Nutritional Examination Survey (NHANES). The authors hypothesised that increased consumption of calorie dense and nutritionally poor foods was a factor in this (Seligman et al., 2007). Similar research using the NHANES (1999 to 2004 waves) also found an association between diabetes and severe food insecurity (Seligman et al., 2010). A systematic review, also in the US, concluded that interventions to reduce material deprivation in regards to food may help prevent diabetes (Barnard et al., 2015). Having $HbA_{1c}$ at prediabetes levels was found to be more prevalent among those who were food insecure in the NHANES (Ding et al., 2014). One study that considered pre-diabetic insulin resistance found that this was

higher in those who were food insecure, and persisted even among those who were of a normal weight (Liu et al., 2015).

The mechanisms through which food insecurity affects the likelihood of developing T2D are similar to how poor diet would affect this. Diets with lower intake of nutrients provided by fruits and vegetables had a non-significant trend with higher $HbA_{1c}$ with adjustment for BMI and SEP in a study using MRC NSHD (Prynne et al., 2009). Increases in energy consumed and some micronutrients provided by milk and meat were significantly associated with raised $HbA_{1c}$ in Prynne et al's (2009) research. Reduced fruit and vegetable intake was found to be associated with higher average $HbA_{1c}$ in research using the European Prospective Investigation of Cancer (EPIC)-Norfolk study (Sargeant et al., 2001). A different investigation using EPIC-Norfolk, found higher fat intake was associated with higher $HbA_{1c}$ (Harding et al., 2001). Both studies adjusted their analysis for other dietary factors, BMI, waist-hip ratio and physical activity. Regularity of meal times was associated with metabolic syndrome, which is a risk factor for T2D even with adjustment for energy intake, physical activity and SEP in research using MRC NSHD (Pot et al., 2014). The evidence for lower intake of healthy food being associated with higher $HbA_{1c}$ may suggest that it is not just the presence of consuming too much or unhealthy foods or obesity that affects propensity to T2D. The importance of regularity of meal times suggest that consistent access to food, which is a feature of food insecurity, could affect this. Associations found between reduced fruit and vegetable intake and $HbA_{1c}$ suggest that specific dietary patterns related to unaffordability and inaccessibility of food may affect $HbA_{1c}$ and propensity to development T2D.

### 3.2.3 Psychosocial pathway

Psychosocial stress includes exposure to stress, a person's coping capacity in response to it and their social support (Chida and Hamer, 2008). It has been proposed that disadvantaged SEP results in greater psychosocial demands with less resources to tackle them (Adler and Snibbe, 2003). This section reviews previous research on whether psychosocial stress is associated with diabetic outcomes. Psychosocial stress is hypothesised to affect the development of T2D via the hormonal response to stress, which causes dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis (Kyrou and Tsigos, 2007). Additionally, a circular relationship between obesity, stress and T2D has been suggested whereby stress causes dysregulation of the HPA axis, which then leads to accumulation of visceral

fat, which in turn leads to further dysregulation of HPA axis (Kyrou and Tsigos, 2007). Research on psychosocial factors and T2D and $HbA_{1c}$ has had contradictory findings, indicating that certain forms of stress may be associated with developing diabetic outcomes.

Exposure to stressful life events have been associated with the development of chronic diseases including T2D (Mason et al., 2013; Renzaho et al., 2014) and metabolic syndrome (Raikkonen et al., 2007, Yamamoto et al., 2011) and with undiagnosed T2D (Mooy et al., 2000). One study in Australia considered the association between impaired glucose metabolism and both stressful life events and perceived stress. An independent effect for perceived stress was found in women but not men, and no clear association was found with actual stressful life events (Williams et al., 2013), suggesting that perceived stress may be more important than actual stressful events. Psychological distress as measured by the General Health Questionnaire (GHQ) was associated with incidence of T2D in longitudinal research using the BHPS (Mommersteeg et al., 2012). However, the authors did not adjust for SEP or BMI in their analysis.

No association was found between GHQ and incidence of T2D in the Whitehall II (Kumari et al., 2004) but a socio-economic gradient in incidence of T2D was found, as was an association with effort-reward imbalance at work for men. Similar results were found in a study on male industrial workers in Germany, work stress as measured by effort-reward imbalance was associated with prediabetes as well as diabetes (Li et al., 2013). Some studies have explored other forms of stress such as job strain. One such study, which focused on women only, did not find an association between the two after adjustment for BMI and physical activity (Kroenke et al., 2007). An association between job strain and T2D was found in other studies even with adjustment for SEP, stress from factors outside of work, diet physical activity and BMI (Toshihiro et al., 2008; Heraclides et al., 2009). These findings suggest that some aspects of psychosocial stress relating to autonomy and control in the work place may be associated with T2D.

In a literature review, Chida and Hamer (2008) found that adverse psychosocial factors (using a variety of definitions from the literature) were associated with poor diabetes control but not with incident T2D. Emotional distress related to having diabetes itself has been found to be associated with poorer diabetic control as measured by $HbA_{1c}$ (Strandberg et al., 2014). Similarly, psychosocial measures related to satisfaction with diabetes treatment and problems with diabetes were

related to $HbA_{1c}$ but wellbeing, self-esteem, social support and self-efficacy were not in a study on patients with T2D (Nozaki et al., 2009). These studies show potential links between stress affecting $HbA_{1c}$ and the stress of having diabetes itself affecting diabetes management. Psychosocial stress may also indirectly affect propensity to T2D and raised $HbA_{1c}$ via health behaviours.

### 3.2.4 Health behaviour pathway

There is some evidence to indicate that disadvantaged SEP is associated with physical activity and obesity. Numerous studies have found that being physically active is protective against the development of T2D and raised $HbA_{1c}$. Physical activity has been found to be protective against T2D with adjustment for diet and BMI but that it was least protective for those who had a high genetic risk (Klimentidis et al., 2014). Moderate levels of physical activity, as measured by steps walked per day, had a protective effect against developing T2D in a study on a population with high obesity (Fretts et al., 2012). Other studies have shown that high levels of physical fitness have been associated with lower prevalence of T2D (Sieverdes et al., 2010). These findings persisted with adjustment for other factors including BMI, family diabetes history, smoking and alcohol consumption. One longitudinal analysis using the National Longitudinal Study of Adolescent Health found a link between physical activity, but not sedentary behaviour, and increased risk of T2D with adjustment for other lifestyle factors and social class (Lee, 2014).

Little association was found between both time spent watching television and physical activity and $HbA_{1c}$ for those who were not obese in a study using the MRC NSHD (Power et al., 2014). However, there was a stronger association between these and $HbA_{1c}$ for those who were already obese (Power et al., 2014). Covariates such as social class and diet were adjusted for but not psychosocial factors. Being physically active was associated with lower $HbA_{1c}$ in adults with a high risk of T2D (over age 40 and with a BMI above 30) in the NHANES. The impact of physical activity on $HbA_{1c}$ was greater among those with lower overall levels of physical activity (Gay et al., 2016). This suggests that the benefits of physical activity in preventing T2D and in maintaining healthy $HbA_{1c}$ may be increased for those who are at the greatest risk. Several studies included obesity in their modelling, suggesting that physical activity may be independently associated with T2D as well as indirectly via obesity.

Obesity is a well-established cause of T2D. Insulin secretion and action can be disrupted in fat laden cells (Romao and Roth, 2008). Fat tissue releases leptin, this affects appetite and metabolism, adiponectin which increases sensitivity to insulin and resistin antagonising the effects of insulin. It has been hypothesised that leptin is important in mediating the link between obesity and T2D (Girard, 1997). The distribution of fat is also important, studies have found that excess visceral fat is a greater risk for T2D than subcutaneous fat (Cederberg et al., 2015).

Several studies exploring SEP and T2D found that the association between the two attenuated with the addition of an obesity measure to the model suggesting obesity mediates this association. Both childhood and adult SEP's association with T2D attenuated completely with adjustment for BMI and hip-to-waist ratio in research using the BWHHS (Lawlor et al., 2007). However, the association between financial wealth and prevalent T2D persisted with adjustment for other SEP measures, physical activity and BMI in a study using ELSA (Tanaka et al., 2012). The association between wealth and incident T2D, rather than prevalence, attenuated with the addition of physical activity and BMI in the same research. Obesity has been included as a mediator or confounder in many studies on T2D and $HbA_{1c}$ however, the extent to which it mediates the SEP association has not been formally decomposed in previous research.

The literature shows that SEP is associated with T2D and $HbA_{1c}$. There has been much research considering mediation of this and which has found varying evidence of how psychosocial stress and health behaviours in particular mediate the SEP association. Research on the role of material factors such as food insecurity in diabetic outcomes has focused on its direct effect rather than as mediating SEP in conjunction with other explanations for inequality.

### 3.2.5 Self-reported T2D and $HbA_{1c}$

The literature reviewed has discussed social patterning in self-reported T2D and $HbA_{1c}$ as well as their associations with material deprivation, psychosocial stress and health behaviours. It is hypothesised that these associations and the pathways between SEP and self-reported diagnosed T2D and objectively measured raised $HbA_{1c}$ may vary due to social and biological differences in the outcomes. Using $HbA_{1c}$ allows those who are pre-diabetic, have undiagnosed diabetes and badly managed diabetes to be captured in the outcome. This section discusses how prediabetes is biologically different to diabetes, the health implications of prediabetes

and how there may be social patterning in the diagnosis of T2D and management of diabetes.

#### *3.2.5.1 Prediabetes*

$HbA_{1c}$ is used for the diagnosis of T2D, an $HbA_{1c}$ measurement of 6.5% or above indicates T2D is present. However, $HbA_{1c}$ becomes raised prior to reaching diabetic levels and this stage can be defined as prediabetes. Prediabetes can indicate likelihood of developing diabetes but is a physiologically different state to the presence of other risk factors such as metabolic syndrome and family history of diabetes. There are health implications associated with being in a pre-diabetic state independent of its association with the development of T2D. For these reasons, the presence of prediabetes warrants study. There are no clear symptoms associated with prediabetes so patients are unaware of it and thus may not seek health care or manage the condition.

Between 5% and 10% of those with prediabetes become diabetic, though this may vary depending on the demographic profile of the population and how diabetes is measured. One UK based study found that between 55% and 80% of people with impaired fasting glucose at baseline had normal fasting glucose at a ten-year follow up though other studies have reported lower conversion rates (Tabák et al., 2012). Insulin sensitivity was present as much as 13 years before the onset of diabetes in the Whitehall II, with a steeper rise in the five years before development, the authors suggest that decreased beta-cell function is already present in the pre-diabetic stage (Tabák et al., 2009).

The experience of being ‘pre-diabetic’ itself can have implications for health. In healthy people, blood glucose is very precisely regulated, it is hypothesised that small fluctuations can affect the feedback loops between glucose and insulin which maintain homeostasis. There are indications that prediabetes, in itself not just via its progression to diabetes, may be related to complications traditionally associated with diabetes.  One study by the Diabetes Prevention Programme Research Group found that people with impaired glucose tolerance and no history of diagnosed diabetes had higher risk of retinopathy consistent with diabetic retinopathy (2007). A review in this area found some studies had found associations between prediabetes and retinopathy while others had not (Nguyen et al., 2007).

Prediabetes is linked to nephropathy and chronic kidney disease based on measurements of urinary albumin exertion rate and estimated glomerular filtration rate. Data from the NHANES 1999-2006 showed markers related to nephropathy

were associated with impaired fasting glucose (Tabák et al., 2012). A study on a small sample of patients with prediabetes, diabetes and a control group used electro-diagnostic testing of distal sensory nerves found substantially higher rates of neuropathy among those with prediabetes in comparison to the control group (Duksal et al., 2016). There is some evidence to suggest that prediabetes is associated with decreased heart rate variability and decreased postural change in heart rate. Cross sectional studies also indicate that there is an excess prevalence of coronary disease in those with glucose which is raised but not at diabetic levels (Tabák et al., 2012).

The capacity to reverse blood glucose from pre-diabetic levels to normal and potentially to reverse some of the damage caused in this stage demonstrates the potential health benefits of identification of prediabetes. A study on mice found that the pre-diabetic stage was associated with peripheral nerve functional abnormalities, the authors also found that these changes could be reversed by using dietary control to return to normal glucose levels (Obrosova et al., 2007). This shows that there is health risks associated with prediabetes but also that this damage may be reversed through early identification and life style change. Another study on humans found that reversal from prediabetes to normal glucose levels, even if only temporary, was associated with a reduced risk of developing diabetes in the future (Tabák et al., 2012). The implications of the risk of developing diabetes in the pre-diabetic period as well as the health risks associated with raised glucose independent of subsequent development of T2D indicate that identification and treatment at this stage is crucial.

#### *3.2.5.2 Social patterning in diagnosis of T2D*

Previous research exploring whether SEP is associated with undiagnosed T2D has had mixed findings. Disadvantaged SEP was associated with undiagnosed diabetes in women as well as with impaired glucose tolerance in men in a German study (Rathmann et al., 2005). However, no association was found between undiagnosed diabetes and SEP in researching using the NHANES III (Wilder et al., 2005). Though this study did find an association between ethnicity and undiagnosed diabetes, ethnicity and SEP are related to one another particularly in the US. Neither was any evidence of an association between undiagnosed diabetes and SEP found in the BWHHS (Lawlor et al., 2007). However, there is substantial research to suggest social patterning in treatment and diagnosis more generally.

Patients with advantaged SEP were more likely to have a diagnostic test ordered at a GP visit but were less likely to receive a prescription than patients with

disadvantaged SEP in an Australian study (Scott et al., 1996). This persisted with control for health status though the association inverted in old age. This suggests that SEP is associated with GP decision-making and the authors found that age and gender also influenced this. One plausible explanation suggested by the authors was that GPs responded to what they perceived to be the patient's expectation from the visit for instance that taking a blood sample for a diagnostic test was an indicator of good care. That patients with disadvantaged SEP may be more diffident and not express their preferences was also posited by the authors. This implies that patients with disadvantaged SEP may not have the causes for their ailments explored in the same way as patients with advantaged SEP, as diagnostic testing is required in order to identify the presence of diabetes.

The quality of communication between doctor and patient has been explored as a potential mechanism through which health inequalities are perpetuated. Quality of communication is required for the identification of symptoms, building knowledge of a patient's health problem and then ensuring the patient knows how to manage and treat it. Patients with disadvantaged SEP received a more directive and less participatory consulting style, were less involved in treatment decisions, had lower control over communication, and received less diagnostic and treatment information and more physical examination (Mercer et al., 2007). The review found that patients with advantaged SEP were more adept at providing unprompted information and active communication and they received more explanations even when not requested. One study discussed in Mercer et al's (2007) review found that in terms of health behaviours, doctors focused more on diet and physical activity with advantaged SEP patients and on smoking with disadvantaged SEP patients. Overall, the consequences of SEP patterning in communication between patients and doctors indicate that there may be SEP differences in identification of the causes of ill health and in patient's receipt of information.

One study explored doctor's perceptions of patients on the basis of their ethnicity and SEP and found that patients who were black and had disadvantaged SEP were judged more negatively by doctors (van Ryn and Burke, 2000). Patients with disadvantaged SEP were judged more negatively in terms of personality, intelligence, compliance, and health behaviour with adjustment for patient and doctor demographics and patient personality characteristics. The authors concluded that this was consequential, as other studies have shown that doctor attitudes to patients influence their behaviour and treatment decisions.

#### *3.2.5.3 Social patterning in diabetes management*

Inequalities have been found in glycaemic control among diabetics indicating an association between SEP and poorly managed T2D (Larsson et al., 1999; Bachmann et al., 2003). Evidence was found of a gradient in $HbA_{1c}$ control in HSE data from 1998 to 2004 (Millett et al., 2007). There was only one measurement of $HbA_{1c}$ available for analysis at the time, longitudinal analysis of other outcomes suggested that improvements in diabetes management were not equally distributed. Similar findings were reported from a whole population longitudinal study of Lower Area Super Output Areas (LSOAs) in England between 2004/2005 and 2011/2012. Glycaemic control, as well as other indicators of management, was found in this study to have improved overall but inequalities persisted (Fleetcroft et al., 2017). A systematic review showed that inequalities in $HbA_{1c}$ were consistently found on the basis of individual SEP and area deprivation (Grintsova et al., 2014). Research in the US has found associations between food insecurity and glycaemic control, suggesting consistent ability to afford and access healthy food is a factor in diabetes management (Berkowitz et al., 2013). One qualitative study explored factors that impacted on ability to manage diabetes among diabetics with disadvantaged SEP. The participants discussed challenges such eating healthy regular meals being constrained by shift work, partaking in physical activity being limited by safety concerns in their neighbourhoods and lack of money impacting on the ability to have a healthy lifestyle (Steyl and Philips, 2014).

### 3.2.6 Conceptual position

T2D and $HbA_{1c}$ have been found to be associated with material factors, psychosocial stress and health behaviour as well as with SEP. None of the research reviewed has considered all of these pathways together which is where this research makes a novel contribution. Additionally, two outcomes are explored in order to assess if mediation is different between them.

The position taken in this research is that disadvantaged SEP increases propensity to experience material deprivation; in particular low income and food insecurity are hypothesised to mediate SEP's association with T2D and $HbA_{1c}$ via limiting resources and resulting in an inadequate, innutritious and inconsistent diet. Food insecurity is conceptualised differently here to diet as a health behaviour as the mechanism causing it is hypothesised to be entirely material. Disadvantaged SEP is hypothesised to result in greater exposure to stressful events with fewer resources to

respond positively to it and thus result in greater psychosocial stress. Health behaviours vary on the basis of SEP, some of the association between disadvantaged SEP and T2D and $HbA_{1c}$ is hypothesised to be due to SEP differences in physical activity. Physical activity is required to regulate $HbA_{1c}$ and to maintain an appropriate weight. Obesity affects glucose metabolism as well as insulin release and action, which can lead to dysregulation of $HbA_{1c}$ and increased likelihood of developing T2D. Obesity is hypothesised to be affected by physical activity and diet as well as the other mediating pathways. It is hypothesised that each of these mediating pathways may also affect one another. Experience of limited resources may constrain participation in health behaviours, alongside food insecurity, it may also create psychosocial stress. Experience of psychosocial stress may affect participation in health behaviours and obesity through HPA dysregulation. In the reviewed literature, either material deprivation, psychosocial stress or health behaviours are often focused on as directly affecting T2D or $HbA_{1c}$ or as attenuating the effect of SEP on these outcomes. However, none of the research reviewed has decomposed the effect of SEP via all of these explanatory pathways.

T2D and $HbA_{1c}$ are both modelled as outcomes here. $HbA_{1c}$ captures those with diagnosed diabetes as well as prediabetes and undiagnosed diabetes. It is hypothesised that the importance of each mediating pathway between SEP and these outcomes may differ between T2D and $HbA_{1c}$ due to social patterning in the diagnosis of T2D, prevalence of prediabetes and management of diabetes.

Figure 8 shows the hypothesised pathways between SEP, material factors psychosocial stress, health behaviours and both T2D and $HbA_{1c}$. The diagram shows a direct effect between childhood SEP and adult SEP through the transmission of SEP as well as a direct pathway between childhood SEP and the diabetic outcomes. The literature reviewed shows that childhood SEP may be important for the development of T2D and raised $HbA_{1c}$ in later life. It is hypothesised in this thesis that though childhood SEP may be associated with these, the risk is not set through experience in utero or childhood development. It is posited that the development of raised $HbA_{1c}$ and T2D in adulthood are influenced more by proximal exposures than by distal SEP in childhood.

### 3.2.7 Research questions

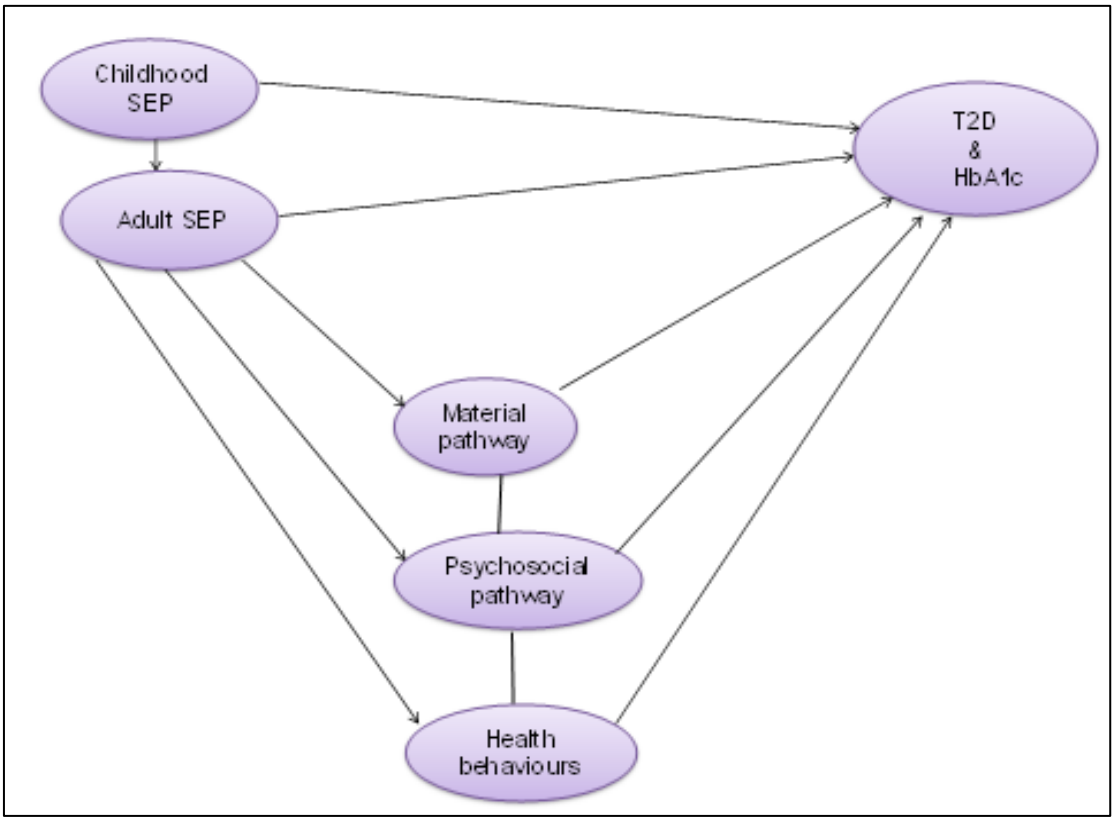
RQ 1. Is disadvantaged adult SEP associated with reported diagnosis of T2D and with raised $HbA_{1c}$?

RQ2. Are there direct effects between the material, psychosocial and health behaviour pathways and T2D and $HbA_{1c}$?

RQ3. Are SEP's associations with T2D and raised $HbA_{1c}$ mediated by the material, psychosocial and health behaviours pathways and by obesity?

RQ4. Does the role of the material, psychosocial and health behaviour pathway vary between T2D and $HbA_{1c}$.

Figure 8 Path diagram of hypothesised associations between SEP via material, psychosocial and health behaviour pathways and diabetic outcomes



## 3.3. Methodology

This section discusses the BHPS and the UKHLS, the analytical sample with T2D and $HbA_{1c}$ as outcomes, the measures used, attrition in the BHPS and its implications and the modelling techniques used.

### 3.3.1 The British Household Panel Survey (BHPS)

The BHPS was selected to answer the research questions as it collected measures of SEP and of each explanatory pathways hypothesised to mediate the association over a long time period. It provided data collected over 20 waves allowing for temporal ordering of the measures of interest. The BHPS collected self-reported diabetes annually while $HbA_{1c}$ was collected from a subset of the BHPS in its 20$^{th}$ wave. The BHPS ran for 18 waves between 1991 and 2009 when it merged with UKHLS. Wave one began with a sample of 8,167 nationally representative households selected from the Postcode Address File, interviews were attempted at all private addresses within the sample. Five thousand and fifty households responded, a response rate of 74%. Those who took part became Original Sample Members (OSM), and in subsequent waves, the sample was all adults in all households where one member was resident in a household interviewed in wave one. In 1999, the Scottish and Welsh samples were extended from approximately 500 households each to 1500 households in each to allow for independent analysis of each country. In wave 11, a Northern Ireland sample was added; 1,900 households were recruited. The survey was conducted by an interviewer with a household and individual questionnaire as well as a self-completion questionnaire of more sensitive questions. Telephone questionnaires were used when attempts at face-to-face interview failed (Brice et al., 2010).

The BHPS joined UKHLS in its second wave, which corresponds to wave 19 of BHPS. In wave three of UKHLS, the BHPS sample was invited to participate in the NHA, which included a blood test, this was undertaken approximately five months after the main interview beginning in June 2011 (Benzeval et al., 2014b). The NHA is described in more detail in the Introduction to Understanding Society Section (1.9). This included measurement of $HbA_{1c,}$ which is glycated haemoglobin. Of 8,914 BHPS sample members eligible to participate in the NHA, 5,053 took part in the Nurse visit and a blood sample was successfully taken from 3,342 of these (McFall et al., 2013).

### 3.3.2 Analytical sample with T2D as an outcome

Initially, 33,015 respondents were present at least once in any wave of the BHPS. A flow chart showing the removal of ineligible participants is shown in Figure 9.

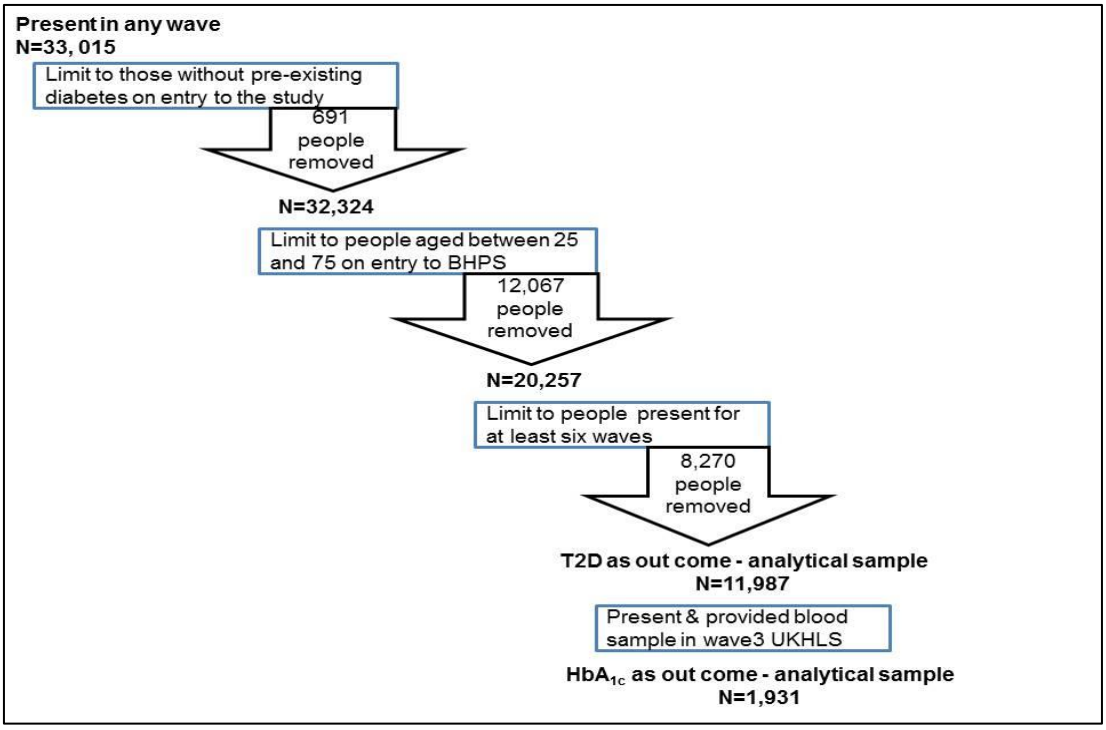
The first exclusion applied was to remove those who reported having diabetes in the first wave in which they entered the sample. This was to ensure that the outcome was limited to the development of diabetes and this resulted in the removal of 691 people from the sample. To restrict the outcome to the development of T2D rather than T1D, people who were aged less than 25 on entry to the BHPS were removed from the analysis, the majority of cases of T1D are diagnosed in childhood and in puberty though it can occur or be diagnosed at any age (Haller et al, 2005). This may result in excluding a small number of people who develop T2D before the age of 25 and may potentially misclassify a small number of people who develop or are not diagnosed as having T1D until after age 25. This removed 10,664 participants. The majority of diagnoses of T1D occur in teenage years or early adulthood and T1D only accounts for 10% of all diabetes.

Those who were aged older than 75 on entry to the sample were also excluded which removed 1,403 from the sample, as diagnoses of T2D decline after midlife (Kirkman et al, 2012). The two age restrictions removed a total 12,067 participants from the sample. In order to be able to measure a period of exposure, the sample was limited to those who were present in the BHPS for at least six waves. These means that some members of the analytical sample could have joined in wave one and left in wave six, some may have been present for any six waves and others may have joined in wave 12 and remained until wave 18. This cut off was selected to allow exposures to SEP and each of the pathways to be captured by the BHPS. This removed a further 8,270 from the sample. As a result, there were 11,987 respondents left in the analytical sample. Each of whom did not have diabetes on entry, were aged between 25 and 75 on entry and were present in the BHPS for at least six waves.

Of the BHPS members who interviewed in wave 18, 79.4% completed the UKHLS wave two interview and 1.3% did so by proxy interview. Interview rates were lower for those who were aged under 30, students, men, the unemployed, private renters, those in poor health and those who expected to move in the next twelve months. There was little variation by education or income (Lynn et al., 2012). Of the 8,914 eligible BHPS sample, there were 56.7% (5,053) who participated in the NHA and 66.1% (3,342) of these provided a blood sample and there were 3,000 with a valid

$HbA_{1c}$ measurement. With the prior exclusions in reference to age and number of waves applied to the sample, there were 1,931 with $HbA_{1c}$ as an outcome in this analytical sample.

Figure 9 Removal of ineligible participants to form the analytical sample for analysis of T2D



A breakdown of the number of waves members of the analytical sample were present in the survey for is shown in Table 12 below. The minimum six waves respondents had to be observed for could be any non-consecutive six waves between waves one and 18.

Table 12 Number of waves in BHPS for the analytical sample with T2D as outcome

| Number of waves present | Frequency | Percentage |
|---|---|---|
| **n** | 11,987 | |
| **Eighteen** | 3,450 | 28.8% |
| **Seventeen** | 551 | 4.6% |
| **Sixteen** | 321 | 2.7% |
| **Fifteen** | 266 | 2.2% |
| **Fourteen** | 234 | 2.0% |
| **Thirteen** | 270 | 2.3% |
| **Twelve** | 257 | 2.1% |
| **Eleven** | 261 | 2.2% |
| **Ten** | 2,327 | 19.4% |
| **Nine** | 790 | 6.6% |
| **Eight** | 1,899 | 15.8% |
| **Seven** | 713 | 6.0% |
| **Six** | 648 | 5.4% |

### 3.3.3 Analytical sample with $HbA_{1c}$ as outcome

Of the 11,987 members of the BHPS for whom incidence of T2D was modelled, 6,566 entered UKHLS in its second wave. Subsequently, 3,092 participated in the NHA in wave three of UKHLS and 2,186 provided a blood sample. There were 1,931 samples of $HbA_{1c}$ successfully obtained from this sample. The number of waves this analytical sample were present in the BHPS is shown in Table 13.

Table 13 Number of waves in the BHPS for the analytical sample with $HbA_{1c}$ as outcome

| Number of waves present | Frequency | Percentage |
|---|---|---|
| **n** | 1,931 | |
| **Eighteen** | 981 | 50.8% |
| **Seventeen** | 102 | 5.3% |
| **Sixteen** | 31 | 1.6% |
| **Fifteen** | 20 | 1.0% |
| **Fourteen** | 20 | 1.0% |
| **Thirteen** | 19 | 1.0% |
| **Twelve** | 14 | 0.7% |
| **Eleven** | 17 | 0.9% |
| **Ten** | 561 | 29.1% |
| **Nine** | 110 | 5.7% |
| **Eight** | 33 | 1.7% |
| **Seven** | 18 | 0.9% |
| **Six** | 5 | 0.3% |

### 3.3.4 Concepts and Measures

#### Type 2 Diabetes

Each year in BHPS, participants were asked, "*Do you have any of the health problems or disabilities listed on this card? Exclude temporary conditions*". The list of illnesses included diabetes but it did not specify whether it is T1D or T2D and did not make specific reference to the condition being diagnosed by a doctor or health professional. It did specify that temporary conditions should be excluded which should prevent misclassification of those who may have had gestational diabetes. Those with diabetes at the first wave they enter BHPS are excluded and incidence T2D is defined as a new report of diabetes by a respondent. The outcome modelled is whether diabetes was reported in any wave after a respondent's first wave in the BHPS.

#### $HbA_{1c}$

$HbA_{1c}$ or glycated haemoglobin is a measure of red blood cells or haemoglobin proteins that join with glucose in the blood to become 'glycated'. Measuring $HbA_{1c}$ gives an indication of blood sugar levels over the preceding eight to 12 weeks. The World Health Organisation has recommended that $HbA_{1c}$ be used to diagnose diabetes mellitus, an $HbA_{1c}$ of 6.5% (48 mmol/mol) or greater is indicative of the presence of diabetes. For people without diabetes, $HbA_{1c}$ of 4%-5.9% (20-41mmol/mol) is normal and for people with diabetes, an $HbA_{1c}$ level of 6.5% or lower is considered good control. Several studies have found that increased risk of T2D is present in those with $HbA_{1c}$ greater than 5.5% (Ko et al., 1998, Edelman et al., 2004, Inoue et al., 2008, Cohen et al., 2010). Those with $HbA_{1c}$ above 5.5% were classified as having impaired glucose tolerance for this study. Thus, the $HbA_{1c}$ measure captures those at risk of developing diabetes as well as those who have already developed it and may be managing it well or badly. It had been intended that this measure could be used to identify prediabetes, undiagnosed diabetes, and well and badly managed diabetes to explore each of these outcomes separately however, the available sample was not sufficient to allow this. Instead, a dichotomous measure of $HbA_{1c}$ above or below 5.5% was used.

$HbA_{1C}$ is affected by other factors that may result in decreased $HbA_{1c}$ measurement, these include having chronic liver disease, taking aspirin and anti-inflammatory medications. Artificially low measurements may also be found in those with high levels of triglycerides (Benzeval et al., 2014b). The analysis shown does not adjust for these measures.

### Treatment of time/waves in derived variables

It is hypothesised that the greater amount of time spent in adversity on each of the pathways captured; material, psychosocial and health behaviour; the greater the impact will be on propensity to develop T2D. For each explanatory pathway, a baseline measure was captured in the wave respondents first entered the survey and subsequently the proportion of time observed they spent in adversity was measured. For each pathway after baseline measurement, respondents' number of reported incidents of adversity in each path was summed and derived into the proportion of time spent in adversity out of the number of waves they were present. Those who did not have data for at least three waves were reclassified as missing and excluded from the analysis, resulting in a loss of 1,940 observations from the final T2D model.

### Socio-economic position – highest educational attainment

Education is measured using the highest qualification obtained at time of entry to the BHPS. Capturing SEP at entry to the survey, using a measure that is most pertinent to early adulthood and is time invariant may also help prevent reverse causality. A small proportion of respondents change highest qualification during the observation period, however their highest qualification on entry is used. As respondents are aged 25 upon entry, the long-term exposure to their SEP experience prior to returning to education may be better captured by their educational level upon entry. Educational attainment is dichotomised into high and low; high education refers to A level and higher and low refers to GCSE level and lower.

### Material pathway - low income

Income is measured with net household income in the month before interview. This is a derived variable using imputed household income and collected income data. Varying proportions of income may be imputed for different households depending on the fullness of the data they provided. After wave one, labour income from previous waves where the respondent was in the same job were used for imputation. Non-labour income variables are imputed solely using data collected at that wave. This was equivalised for household size using the modified OECD equivalence scale.

Income was derived into a binary measure of whether respondents were in the bottom quintile of income in each wave. As this was based on the distribution of income within waves, no adjustment was made for inflation. The baseline measure of income uses whether income was in the lowest quintile or not in the wave

respondents entered the survey. For subsequent measurement of income, the proportion of time respondents spent in 'low' income was used. Low income was derived into a dichotomous measure of whether a respondent spent more than one third of the time they are observed in the BHPS in the lowest income quintile.

### Material pathway - food insecurity

Expenditure on food is used to measure food insecurity.[12] Expenditure on food does not capture the quality of the food purchased or coping mechanisms such as eating with relatives to avoid spending on food. However, this measure is objective and avoids positive response bias. Expenditure on food is asked in BHPS as "*Please look at this card and tell me approximately how much your household spends each week on food and groceries? Include all food, bread, milk, soft drinks etc.; exclude pet food, alcohol, cigarettes and meals out*", responses were captured in ordinal categories of expenditure in waves two to 18 but was continuous in wave one.

Responses from waves two onwards were first transformed into a continuous measure, spending bands were transformed into continuous variables by taking the top of the range for each spending category. Spending bands were of £10 up to £100, and then in bands of £20 with the highest band £160 or more which was treated as £180. Weekly spend was transformed into monthly expenditure and this was then equalised for household size. A binary measure of low spend on food was created by recoding those in the bottom three deciles of food expenditure as being food insecure in each wave. A question on how much was spent on eating out was introduced to BHPS in wave seven. In each wave after this, those who were classified as food insecure but who also spend more than the median on eating out were reclassified as food secure. For earlier waves, this means the measure of food insecurity was less robust. For the baseline measurement, being in the bottom three deciles of food expenditure in the first wave of entry to BHPS was used. Subsequently, spending at least one third of the time observed or more in food insecurity was used as a binary measure of exposure to food insecurity.

[12] Please see Appendix D for more details on how this measure of food insecurity was created.

### Psychosocial pathway – General Health Questionnaire (GHQ)

Ideally, measuring psychosocial stress would capture exposure to stress, a person's coping capacity in response to this and their social support. However, the 12-item GHQ has been found to be effective in measuring tendency towards psychological distress (Goldberg et al., 1997, Goldberg et al., 1998). The GHQ asks a range of questions on psychological distress, it is limited in that it refers to respondents 'recent' experience thus it may not capture prolonged or intermittent episodes of psychological distress. Responses are in the form of a four point Likert scale, responses of one and two are recoded to zero while responses of three and four are recoded to one for the 12-item scale. These are then summed resulting in a scale from zero (the lowest distress score) to 12 (the highest distress score). The questions relate to concentration, loss of sleep, feeling that oneself is useful, capable of making decisions, constantly under strain, has problems overcoming difficulties, enjoys day-to-day activities, is able to face problems, is unhappy or depressed, losing confidence, has self-worth and general happiness. Thresholds used to indicate caseness vary, however it has been recommended to use a threshold of either two or three when the mean score in a sample is between 1.85 and 2.7. Thus, a threshold of three or more is used here (Goldberg et al., 1997, Goldberg et al., 1998). A limit of the measure is that experience of the symptoms of T2D such as fatigue prior to its diagnosis or being reported in the BHPS may impact on response to this.

A baseline measure of GHQ is used, whether respondents reported caseness or not in their first wave in the BHPS. Subsequently a binary measure of whether respondents spent one third or more of the time for which they were observed in the BHPS reporting caseness is then used.

### Health behaviour pathway – physical activity

The BHPS asked respondents in waves six, eight, 10, 12, 14, 16 and 18 how frequently they played sports, or went walking or swimming. Reference is made specifically to these activities; other forms of physical activity are not mentioned. Respondents could answer that they went at least once a week, at least once a month, several times a year, once a year or less or never/ almost never. Responses were dichotomised into at least once a week or less. This approach has been previously used in research (Popham and Mitchell, 2006). The first reported response for this is used as the baseline measure. Whether respondents spent at least two thirds of the time for which they were observed reporting low physical activity was used as a binary measure of exposure to low physical activity.

### Obesity pathway – Body Mass Index

Height and weight were collected by self-report in waves 14 and 16 of BHPS. These were derived to make a measure of BMI. There is high non-response to this question. There was no baseline collection of height and weight. Due to the large non-response to this item, BMI collected in wave 14 is used in the first instance, however if those who did not respond in wave 14 did in wave 16, their response from wave 16 is used. There were 138 members of the analytical sample with a measurement from wave 16. BMI was then classified into whether respondents were obese or not. Those with a BMI of 30 and higher were classified as obese. Those categorised as non-obese included respondents were overweight as well as a very small number of respondents who were underweight. A small number of respondents (eight) with implausibly low BMI measurements were recoded as missing.

As BMI was first collected in wave 14 and the analysis is restricted to those who were present in at least six waves of BHPS, it means that some members may have dropped out of the BHPS prior to measurement of obesity. This was also an issue for maternal education and ethnicity, which were first collected at 13. A dummy measure for those who dropped out of the sample prior to wave 14 was included as a control measure in the analysis to retain these (1,318) cases.

## *Covariates*

The following measures are included as controls in the models as they are known to be associated with the development of T2D and with $HbA_{1c}$.

### Childhood SEP – maternal education

For childhood SEP, it is thought that nutrition in utero and in childhood may affect propensity to T2D. Childhood SEP is used as a control variation while mediation between adult SEP and the outcomes is focused on. Maternal education is treated as a dichotomous control variable for childhood SEP in the model. Advantaged childhood SEP was classified as reporting that maternal education was some or post school qualifications, and disadvantaged childhood SEP was reporting no school or having left school without qualifications. As respondents were asked this initially in wave 13, some of the analytical sample had left the BHPS before this was asked. As with obesity, a dummy measure for those who dropped out of the sample prior to collection of maternal education was included to retain these cases.

#### Age

Respondents' age at the time where they first enter the survey is used as a control measure as propensity to T2D increases with age. Participants must be aged between 25 and 75 years in their year of entry to the survey.

#### Ethnicity

Respondents were asked their ethnic background from wave 13 onwards in the BHPS. Ethnicity was included as a dichotomous control in the modelling, as rates of T2D are higher in some ethnic groups. Ethnicity was derived into white ethnicity or black and minority ethnicity. A dummy measure for drop out prior to wave 13 allowed the retention of cases that left before ethnicity data were collected.

#### Gender

Respondent's gender is included in the model, as T2D is more prevalent among men and known to occur at lower levels of BMI (Logue et al., 2011). It was not hypothesised that the mechanisms between SEP and the outcomes would act differently based on gender so the analysis was not stratified by gender, nor were interactions with gender included.

#### Number of years in the BHPS

The number of waves respondents have been observed in BHPS for is also included in the models as a continuous measure. All respondents were present for a minimum of six waves. It was hypothesised that attrition maybe socially patterned and that the likelihood of observing T2D occur would increase with time spent in the BHPS.

### 3.3.5 Attrition in the BHPS

In the second wave of BHPS, 654 households refused to participate. These refusals were reviewed and where it was deemed that households might be persuaded to re-join the survey, up to three attempts were made to convert them (Brice et al., 2010). There were 354 (54%) of the households who refused at wave two were contacted to participate again at wave three. If a household refused to participate, the reasons for refusal were gathered and interviewers decided whether to attempt to convert the household. Refusals were categorised into three categories of weak, strong or conclusive. Weak refusals were reasons such as finding the survey boring and strong refusals were reasons including caring responsibilities or illness, in both cases attempts were made to convert these households. Conclusive reasons were where respondents made it clear they did not want to be contacted again, in these cases,

judgement was made as to whether to try to encourage the household to participate again, two consecutive conclusive refusals were accepted.

Members can also leave the survey through a terminating event such as moving into an institution, which moves them out of the population of interest. Cases that move and cannot be traced, or refusals were used creating weights, those with terminating events were not used, as they were no longer eligible. The analysis is this chapter is not weighted, as using weights would result in substantial loss of sample size. Longitudinal weights are specific to the sample in final wave observed which would be wave 18 in this chapter. However, much of the analytical sample dropped out before wave 18. Weights were not used analysing T2D or $HbA_{1c}$ to retain sample size. This means that data are not representative which limits the generalisability of the findings.

Attrition was highest between waves one and two, 11% of the OSM were not re-contacted. However, attrition has been low subsequently, generally less than 5% between each wave. Demographic factors associated with survey non-response include age; those who are younger and more mobile are less likely to respond and more likely to attrite while the elderly may not respond or leave due to ill health. Single person households have higher risk of non-response due to it being less likely for a household member to be home. Men are more likely to non-respond than woman. Being in any form of employment is associated with non-response due to time spent outside of the home. Poor health affects response through bringing respondents outside of their home to be cared for and being less willing to participate when they are found at home.

Previous research on the BHPS has found that those reporting less satisfaction with their health in wave one were more likely to become non-respondents. This implies that initial poor health predicts eventual non-response, however self-reported poor health may precede diagnosis with a chronic illness, which has implications for analysing incidence of T2D as an outcome. Previous research has also found a curvilinear association between SEP and participation with those at the more advantaged and disadvantaged extremes less likely to participate (Uhrig, 2008).

Analysis on predictors of attrition between waves one and 14 of the BHPS found that health items including reported physical health problems, GHQ, smoking status and disability did not predict non-response, non-contact and refusal. Self-reported poor and very poor health was associated with non-contact though not non-response relative to those reporting excellent health. This was hypothesised to be due to those

with poor health possibly having to move to institutions or being less likely to be at home due to the pursuit of care. The same research found that higher education was associated with fewer refusals to participate (no association was found with non-contact), low income was not associated with attrition through missing income was. This implies that estimates produced in this chapter may be biased, as those with lower education and poorer self-reported health are more likely to leave the sample.

### 3.3.6 Statistical Techniques

In order to answer the research questions, it was necessary to establish first whether there was a link between SEP and the diabetic outcomes and then to look at how much of this association was mediated by the explanatory pathways.

As the outcome variables, T2D and raised $HbA_{1c}$, were binary measures, logistic regression was used in the first instance to model the association between SEP and the outcomes with adjustment for age, gender, ethnicity, maternal education and number of waves in the BHPS. Logistic regression was also used to explore whether there were direct effects between each of the explanatory pathways and each outcome. Available-case analysis is used throughout. While this has the advantage of retaining sample size and making use of all available data, it limits comparability across models where the number of observations changes with the addition of mediators. Another limitation is via non-response bias. Non-response is high on maternal education and obesity. Characteristics related to having missing data on these two variables was explored here. Having missing data on maternal education was associated with low income, food insecurity and being male, it was not associated with either outcome. Not providing a response to height and weight was associated with low education, being female and having raised $HbA_{1c}$ and it had a borderline ($p$<.1) association with T2D. This implies that there has been exclusion of those at risk of diabetic outcomes and the analysis may under estimate associations as a result.

In order to measure the indirect effects of the specified pathways in explaining these associations, it was necessary to select a method that would allow for identification of indirect effects with binary measures. For this reason, a decomposition method developed by Breen, Karlson and Holm was selected (Breen et al., 2013). This was done using the STATA command *khb (*Kohler et al., 2011). In undertaking the analysis of indirect effects, the intention was to decompose the total effect of the independent measure (SEP in this case) into a direct effect and an indirect effect.

The indirect effect is the proportion of the total effect of SEP that is mediated by the explanatory pathway being tested. The direct effect is the impact of SEP on the outcome, which is not mediated by the measured pathways. Those it may be mediated by unobserved factors.

In modelling binary outcomes, the total effect does not decompose into direct and indirect effects in the same way as in linear models. If the model was fit without the pathway mediators, it would have a larger residual standard deviation than a model with the mediators because the model with the mediators will explain more variation in the latent outcome. Thus including the mediators would result in changes to the coefficient of the direct effect (SEP in this case) because of rescaling. Comparing the coefficients across logit models with and without the mediator variables does not simply reflect confounding but also rescaling and changes in the fit of the error to the assumed functional form. This means it is necessary to use an approach that holds the scale constant and the fit of the error to the assumed logistic distribution. The *khb* technique allows the total effect to be decomposed into direct and indirect effect and additionally allows multiple binary mediators to be used in the model.

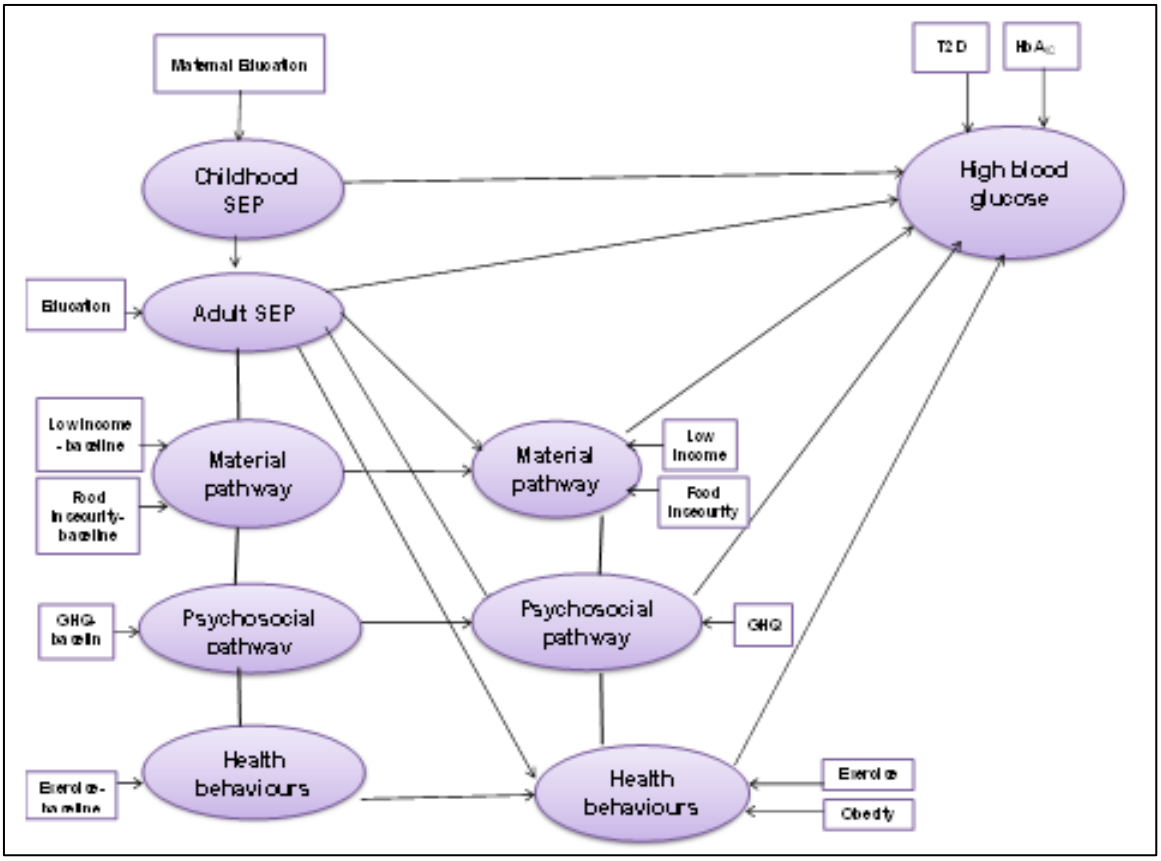
In modelling the pathways, baseline measures of each mediator, where available, are first tested as mediators of the association. After this, they are included as control measures in order to model the mediation by subsequent experience on these pathways. Age, gender, maternal education and entry to BHPS are also included as control measures. The indirect effect of each pathway is modelled individually on the SEP and T2D association, then each pathway is considered in unison. The diagram in Figure 10 shows these hypothesised pathways, their measures and their baselines. For simplicity in the diagram, obesity is depicted as part of the health behaviours pathway. Obesity is not conceptualised as a health behaviour but as a being influenced by each pathway and mediating the SEP association. It is also assumed that each pathway may affect each other though this is not depicted.

### *Sensitivity checks*

The analysis on T2D was rerun on the sample for which $HbA_{1c}$ was measured. The analysis was rerun without the inclusion of sample members who dropped out prior to wave 14 who were retained with a dummy measure. Analysis of $HbA_{1c}$ was rerun

with control for long-term liver disease, taking aspirin, taking anti-inflammatories and for triglycerides. [13]

Figure 10 Path diagram of hypothesised associations between SEP via material, psychosocial and health behaviour pathways and diabetic outcomes including measures




[13] Please see Appendix G for a brief overview of results from sensitivity checks

## 3.4. Results

### 3.4.1 Description of the analytical sample with T2D as outcome

Table 14 describes the analytical sample with T2D as outcome. Slightly less than half the sample, 45.9%, are male. The mean age on entry is 45.2 (13.9) years. Only 4.8% of the sample reported being of black or minority ethnicity (BME). There was 57.8% of the sample reporting disadvantaged SEP as measured by education and 51.6% reporting disadvantaged childhood SEP, responses to these vary by age with lower education being more prevalent among older members of the sample. There was 11.6% of the sample who did not respond to childhood SEP. At their first observation in the BHPS, 15.4% of the sample reported low income and 26.7% reported food insecurity. There were 34.5% and 38.0% who reported low income and food insecurity for more than one third of the time they were observed in the BHPS. The psychosocial pathway was captured by the GHQ, psychological distress was reported by 23.1% on their entry to the BHPS and 26.9% reported experiencing this for more than one third of the time they were observed. Forty-four percent of the sample reported low physical activity on their first observation in the BHPS and 29.2% reported this for two thirds of the time they were observed. Obesity was reported by 14.8% of the sample. There was high non-response to this measure at 11.5%. There were 4.6% of the sample who reported developing T2D.

Table 14 Demographic, socio-economic, material, psychosocial, health behaviour and obesity characteristics for analytical sample with T2D as outcome

| n=11,987 | | Percentage / mean (SD) at baseline | Measures captured throughout observation period | | Percentage |
|---|---|---|---|---|---|
| **Gender** | Female | 54.1% | | | |
| **Age** | Age at entry | 45.2 (13.9) | | | |
| **Ethnicity** | BME | 4.8% | | | |
| | Missing | 5.8% | | | |
| **Childhood SEP** | Disadvantaged childhood SEP | 51.6% | | | |
| | Missing | 11.6% | | | |
| **Adult SEP** | Disadvantaged adult SEP | 57.8% | | | |
| | Missing | 1.6% | | | |
| **Material pathway** | Low income | 15.4% | Low income | More than one third of time observed spent in low income | 34.5% |
| | Missing | 14.0% | | Missing | 1.4% |
| | | | | | |
| | Food insecurity | 26.7% | Food insecurity | More than one third of time observed spent in food insecurity | 38.0% |
| | Missing | 3.0% | | Missing | 0.0% |
| | | | | | |
| **Psychosocial pathway** | Psychological distress | 23.1% | Psychological distress | More than one third of time observed spent in psychological distress | 26.9% |
| | Missing | 6.5% | | Missing | 0.0% |
| | | | | | |
| **Health behaviour** | Low physical activity | 44.0% | Physical activity | More than two thirds of time observed spent with low physical activity | 29.2% |
| | Missing | 5.0% | | Missing | 0.2% |
| | | | | | |
| **Obesity** | | | Obesity (wave14) | Obese | 14.8% |
| | | | | Missing | 11.5% |
| **Outcome** | | | | | |
| **Development of T2D** | T2D | 4.6% | | | |
| | Missing | 0.6% | | | |

### 3.4.2 Description of analytical sample with $HbA_{1c}$ as outcome

Table 15 describes the analytical sample with $HbA_{1c}$ as outcome in regards to the key variables used. Slightly less than half the sample, 48.0%, are male. The mean age on entry is 42.8 (12.1) years. The majority of the sample reported a white ethnicity and 7.4% reported being of black or minority ethnicity (BME). Disadvantaged childhood SEP was reported by 60.7% of the sample and 9.3% of the sample had no response to this. There were 42.3% reporting disadvantaged SEP in adulthood. There was 16.6% of the sample reporting low income in their first time being observed in the BHPS and 30.8% reported food insecurity. There were 19.5% and 28.6% who reported low income and food insecurity for more than one third of the time they were observed in the BHPS. Psychological distress as captured by the GHQ was reported by 16.6% on their entry to the BHPS and 29.2% reported experiencing this for more than one third of the time they were observed. Low physical activity was reported by 48.0% of the sample on their first observation in the BHPS and 26.2% reported this for two thirds of the time they were observed. Obesity was reported by 16.8% of the sample. Non-response to this measure was much lower than for the T2D analytical sample at 3.8%. HbA1c above 5.5% was reported by 55.8% of the sample.

Table 15 Demographic, socio-economic, material, psychosocial and health behaviour characteristics for analytical sample with $HbA_{1c}$ as outcome

| n=1,931 | | Percentage / mean (SD) at baseline | Measures captured throughout observation period | | Percentage |
|---|---|---|---|---|---|
| **Gender** | Female | 54.5% | | | |
| **Age** | Age at entry | 43.7 (12.1) | | | |
| **Ethnicity** | BME | 5.8% | | | |
| | Missing | 2.3% | | | |
| **Childhood SEP** | Disadvantaged childhood SEP | 57.6% | | | |
| | Missing | 9.3% | | | |
| **Adult SEP** | Disadvantaged adult SEP | 48.6% | | | |
| | Missing | 0.5% | | | |
| **Material pathway** | Low income | 17.9% | Low income | More than one third of time observed spent in low income | 27.3% |
| | Missing | 10.3% | | Missing | 3.8% |
| | | | | | |
| | Food insecurity | 27.5% | Food insecurity | More than one third of time observed spent in food insecurity | 31.4% |
| | Missing | 2.6% | | Missing | 0.4% |
| | | | | | |
| **Psychosocial pathway** | Psychological distress | 18.1% | Psychological distress | More than one third of time observed spent in psychological distress | 24.6% |
| | Missing | 3.1% | | Missing | 0% |
| | | | | | |
| **Health behaviour** | Low physical activity | 44.9% | Physical activity | More than one third of time observed spent in low physical activity | 23.0% |
| | Missing | 1.6% | | Missing | 0.2% |
| | | | | | |
| **Obesity** | | | Obesity (wave 14) | Obese | 17.1% |
| | | | | Missing | 5.0% |
| **Outcome** | | | | | |
| **$HbA_{1c}$** | Above 5.5% | 48.5% | | | |
| | Missing | 0.0% | | | |

### 3.4.3 Direct effects of SEP and pathways on T2D and $HbA_{1c}$

The aim of this study is to explore mediation of the association between SEP and T2D and $HbA_{1c}$. Logistic regression was used to confirm that there was an association between them. The direct effects between each of the pathway measures at baseline and subsequently in the BHPS on T2D were also modelled with logistic regression. Table 16 shows the odds ratios (OR), 95% confidence intervals (95% CI), number of observations in the model (n) and the pseudo R-squared statistic (P-R2) for each model of T2D and $HbA_{1c}$. There was a significant association between SEP and T2D. The risk of developing T2D over the observation period was higher for people with disadvantaged SEP, they were approximately 1.5 times more likely to develop this than their advantaged counterparts. For the material pathway, neither low income at baseline or throughout the observation period was associated with likelihood of developing T2D, and nor was food insecurity. Having a high GHQ score was associated with greater likelihood of developing T2D than having a low GHQ score throughout the observation period. Psychological distress at baseline was just below the threshold of statistical significance, confidence intervals ranged between 0.999 and 1.602. Throughout the observation period, those reporting psychological distress were 1.742 (1.418 to 2.139) times more likely to develop T2D. Those who reported doing low physical activity in their initial observation and throughout the BHPS had significantly higher incidence of T2D. The strongest association was seen between obesity and T2D, those who were obese were approximately four times more likely (3.617 to 5.369) to develop T2D.

Table 16 Direct effects of SEP and each mediator on T2D

| | | Measurement at baseline | | | | Measurement throughout observation period | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | **OR** | **95% CI** | **n** | **P-R2** | **OR** | **95% CI** | **n** | **P-R2** |
| **SEP** | Disadvantaged SEP | 1.514 *** | (1.223 to 1.875) | 10,275 | .061 | | | | |
| **Pathways** | | | | | | | | | |
| **Material** | Low income | 1.139 | (0.912 to 1.420) | 9,070 | .059 | 1.177 | (0.963 to 1.437) | 9,648 | .058 |
| | Food insecurity | 1.071 | (0.859 to 1.336) | 10,150 | .056 | 0.857 | (0.678 to 1.084) | 10,366 | .057 |
| **Psychosocial** | Psychological distress | 1.265 | (0.999 to 1.602) | 9,831 | .057 | 1.742 *** | (1.418 to 2.139) | 10,248 | .063 |
| **Health behaviour** | Low physical activity | 1.762 *** | (1.457 to 2.131) | 10,043 | .065 | 1.584 *** | (1.304 to 1.925) | 9,994 | .059 |
| | Obesity | | | | | 4.406 *** | (3.617 to 5.369) | 9,557 | .119 |

*p<.05, **p<.005, ***p<.001 models adjusted for age, gender, ethnicity, childhood SEP and number of waves in the BHPS

Table 17 shows the direct effects of disadvantaged SEP and each pathway measure on $HbA_{1c}$. Having disadvantaged SEP was associated with greater likelihood of having high $HbA_{1c}$. In the material pathway, neither low income nor food insecurity at baseline or through the observation period was associated with greater likelihood of having raised $HbA_{1c}$. In the psychosocial pathway, neither psychological distress at baseline or during the observation period was associated with $HbA_{1c}$. In regards to the health behaviour pathway, both low physical activity at baseline and throughout the observation period were associated with $HbA_{1c}$. Each was associated with being approximately 1.3 times more likely to have raised $HbA_{1c}$. Obesity had a strong direct effect on $HbA_{1c}$, those who were obese were 1.856 (1.424 to 2.419) times more likely to have raised $HbA_{1c}$.

Table 17 Direct effects of SEP and each mediator on $HbA_{1c}$

| | | Measurement at baseline | | | | Measurement throughout observation period | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | **OR** | **95% CI** | **n** | **P-R2** | **OR** | **95% CI** | **n** | **P-R2** |
| **SEP** | Low education | 1.336 * | (1.081 to 1,651) | 1,743 | .084 | | | | |
| **Pathways** | | | | | | | | | |
| **Material** | Low income | 1.170 | (0.890 to 1.538) | 1,574 | .086 | 1.313 | (1.043 to 1.654) | 1,696 | .085 |
| | Food insecurity | 0.980 | (0.778 to 1.234) | 1,709 | .081 | 1.042 | (0.833 to 1.305) | 1,751 | .080 |
| **Psychosocial** | Psychosocial distress | 1.151 | (0.887 to 1.498) | 1,697 | .080 | 1.180 | (0.933 to 1.492) | 1,751 | .080 |
| **Health behaviour** | Low physical activity | 1.320 * | (1.078 to 1.616) | 1,730 | .084 | 1.344 * | (1.060 to 1.704) | 1,750 | .082 |
| | Obesity | | | | | 1.856 *** | (1.424 to 2.419) | 1,682 | .088 |

***p<.05, **p<.005, ***p<.001, models adjusted for age, gender, ethnicity, maternal education and number of waves in the BHPS**

### 3.4.3 Mediation of the association between T2D and $HbA_{1c}$ and SEP by the pathways at baseline measurement

Before looking at the mediating effect of each pathway on SEP's association with T2D and $HbA_{1c}$, baseline measures of each pathway were added simultaneously to the model. They capture the experience of each pathway at the first point respondents enter the study and are indicative of experience of that pathway up to the first observation period. The results shown in Table 18 indicate the total effect of SEP, the direct effect of SEP and the indirect effect. The indirect effect refers to the proportion of the total effect of SEP explained by the baseline measure of the pathways. The number of observations in the model (n), the pseudo r-squared statistic, the odds ratios (OR) for the total, direct and indirect effect as well as their standard error (SE) and 95% confidence intervals (95% CI) are shown.

Table 18 shows that the total and direct effects were significant in the T2D model. The total effect of SEP was 1.487 (1.177 to 1.880) and the direct effect of SEP was only slightly less than the total effect, 1.435 (1.131 to 1.820). The indirect effect of the pathway measures at baseline was not significant.

In the $HbA_{1c}$ model, the total effect was significant producing an odds ratio of 1.404 (1.116 to 1.766). The direct effect of SEP on $HbA_{1c}$ was 1.380 (1.095 to 1.740). The indirect effect of the pathway measures at baseline was not significant indicating that these did not explain a significant proportion of the association between SEP and $HbA_{1c}$.

Table 18 Decomposition of SEP's association with T2D and $HbA_{1c}$ via mediating pathways at baseline

| | | T2D n=8,427 P-R2=.07 | | | $HbA_{1c}$ n=1,502 P-R2=.10 | | |
|---|---|---|---|---|---|---|---|
| | **Measure** | **OR** | **SE** | **95% CI** | **0R** | **SE** | **95% CI** |
| **Total effect** | SEP & pathways at baseline | 1.487 ** | 0.178 | (1.177 to 1.880) | 1.404 ** | 0.165 | (1.116 to 1.766) |
| **Direct effect** | SEP | 1.435 * | 0.174 | (1.131 to 1.820) | 1.380 * | 0.163 | (1.095 to 1.740) |
| **Indirect effect** | Pathways at baseline | 1.037 | 0.023 | (0.993 to 1.082) | 1.017 | 0.017 | (0.983 to 1.052) |

***p<.05, **p<.005, ***p<.001, models adjusted for age, gender, ethnicity, childhood SEP and number of waves in the BHPS**

Though insignificant, the indirect effect of each pathway at baseline accounted for 9.0% of the total effect of SEP on T2D. The role of mediators in the indirect effect is broken down in Table 19 showing their coefficient, standard error and the percentage of the indirect effect they explained. The majority of the indirect effect was explained by low physical activity, 7.7% of the total effect of SEP on T2D was mediated by low physical activity at baseline. The effect of low income at baseline was in the opposite to hypothesised. The psychosocial distress pathway mediated an insignificant 1.6% of the total effect. The proportion of the indirect effect explained by food insecurity and was negligible.

The indirect effect of these measures on the association between SEP and $HbA_{1c}$ was not significant and they attenuated the total effect by 5%. The material and health behaviour pathways contributed the majority of this, income being important within the material pathway.

Table 19 Separation of indirect effects in mediating pathways at baseline measurement for T2D and $HbA_{1c}$

| | | T2D | | | $HbA_{1c}$ | | |
|---|---|---|---|---|---|---|---|
| **Pathway** | **Measure** | **_b** | **SE** | **Contribution %** | **_b** | **SE** | **Contribution %** |
| **Material factors** | Food insecurity | 0.003 | 0.007 | 0.6% | -0.002 | 0.004 | -0.6% |
| | Low income | -0.004 | 0.019 | -0.9% | 0.008 | 0.015 | 2.4% |
| **Psychosocial** | Psychosocial distress | 0.006 | 0.004 | 1.6% | 0.003 | 0.004 | 0.8% |
| **Health behaviour** | Low physical activity | 0.030 | 0.009 | 7.7% | 0.008 | 0.009 | 2.4% |
| **Percentage mediation of the total effect** | | | | 9.0% | | | 5.0% |

### 3.4.4 Mediation by material pathway

The two aspects of the material pathway, which were theorised to mediate the association between SEP and T2D and $HbA_{1c}$, were income and food insecurity and the indirect effect of these was considered simultaneously. As the number of observations varies between models, the total effect is slightly different to the previous model. The indirect effect of these in the T2D model was not significant. SEP continued to have a significant direct effect on T2D, which was only marginally smaller than the total effect. The direct effect of SEP on T2D was 1.442 (1.134 to 1.835) and the indirect effect was 1.011 (0.975 to 1.048). The material pathway also did not mediate a significant proportion of the total effect of SEP on $HbA_{1c}$. The direct effect of SEP on $HbA_{1c}$ was still significant, 1.349 (1.062 to 1.712) was only slightly smaller than the total effect. These results are shown in Table 20.

Table 20 Decomposition of SEP's association with T2D and $HbA_{1c}$ via the material pathway

| | | T2D n=8,232 P-R2=.07 | | | $HbA_{1c}$ n=1,492 P-R2=.10 | | |
|---|---|---|---|---|---|---|---|
| | **Measure** | **OR** | **SE** | **95% CI** | **OR** | **SE** | **95% CI** |
| **Total effect** | SEP & material pathway | 1.442 ** | 0.177 | (1.134 to 1.835) | 1.372 * | 0.163 | (1.087 to 1.732) |
| **Direct effect** | SEP | 1.426 * * | 0.178 | (1.117 to 1.812) | 1.349 * | 0.164 | (1.062 to 1.712) |
| **Indirect effect** | Material pathway | 1.011 | 0.019 | (0.975 to 1.048) | 1.017 | 0.027 | (0.966 to 1.070) |

*p<.05, **p<.005, ***p<.001, models adjusted for age, gender, ethnicity, childhood SEP, number of years in the BHPS and pathways at baseline

Though insignificant, the material pathway explained 3.1% of the total effect of SEP on T2D, food insecurity made no substantial contribution to this indirect effect. The material pathway did not contribute significantly to explaining the association between SEP and $HbA_{1c}$, but did mediate 5.3% of the total effect. Similarly to the decomposition on T2D, this was explained by low income. This is shown in Table 21.

Table 21 Separation of indirect effect in the material pathway for T2D and $HbA_{1c}$

| | | T2D | | | $HbA_{1c}$ | | |
|---|---|---|---|---|---|---|---|
| **Pathway** | **Measure** | **_b** | **SE** | **Contribution %** | **_b** | **SE** | **Contribution %** |
| **Material factors** | Food insecurity | 0.001 | 0.003 | 0.1% | -0.002 | 0.004 | -0.6% |
| | Low income | 0.011 | 0.018 | 3.0% | 0.019 | 0.025 | 5.9% |
| **Percentage mediation of the total effect** | | | | 3.1% | | | 5.3% |

### 3.4.5 Mediation by the psychosocial pathway

Table 22 below shows the decomposition of the total effect of SEP into its direct effect and indirect effect via psychological distress. The total effect of SEP on T2D was 1.435 (1.130 to 1.823) and the direct effect of SEP only marginally different. There was no significant mediation of the total effect by the psychosocial pathway, it accounted for a negligible proportion of the total effect. The decomposition on $HbA_{1C}$ produced similar results; the indirect effect was not significant and the indirect effect was in the opposite direction to that hypothesised

Table 22 Decomposition of SEP's association with T2D and $HbA_{1c}$ via the psychosocial pathway

| | | T2d n=8,407 P-R2=.07 | | | $HbA_{1c}$ n=1,502 P-R2=.10 | | |
|---|---|---|---|---|---|---|---|
| | **Measure** | **OR** | **SE** | **95% CI** | **OR** | **SE** | **95% CI** |
| **Total effect** | SEP & psychosocial pathway | 1.435 ** | 0.175 | (1.130 to 1.823) | 1.380 * | 0.163 | (1.095 to 1.740) |
| **Direct effect** | SEP | 1.434 ** | 0.175 | (1.129 to 1.821) | 1.382 * | 0.163 | (1.096 to 1.742) |
| **Indirect effect** | Psychosocial | 1.001 | 0.005 | (0.992 to 1.010) | 0.999 | 0.003 | (0.993 to 1.004) |
| **Percentage mediation of the total effect** | | 0.2% | | | -0.4% | | |

*p<.05, **p<.005, ***p<.001, models adjusted for age, gender, ethnicity, childhood SEP, number of years in the BHPS and pathways at baseline

### 3.4.6 Mediation by health behaviour pathway

The indirect effect of health behaviours on the association between SEP and T2D was not significant. The total effect of the model was 1.442 (1.136 to 1.831) and the direct effect of SEP attenuated very slightly in the decomposition to 1.430 (1.126 to 1.815). The indirect effect was just below statistical significance, confidence intervals ranged between 0.999 and 1.019. The health behaviour pathway also had a similar indirect effect on the association between SEP and $HbA_{1c}$. The total effect of this association was 1.378 (1.093 to 1.737). Low physical activity was not a significant mediator of the association between SEP and $HbA_{1c}$. Similarly to the decomposition on T2D, the indirect effect was insignificant, it ranged from just to below one up to 1.009. This is shown in Table 23.

Table 23 Decomposition of SEP's association with T2D and $HbA_{1c}$ via the health behaviour pathway

| | | T2D n=8,166 P-R2=.07 | | | $HbA_{1c}$ n=1,501 P-R2=.10 | | |
|---|---|---|---|---|---|---|---|
| | **Measure** | **OR** | **SE** | **95 % CI** | **OR** | **SE** | **95 % CI** |
| **Total effect** | SEP & health behaviour pathway | 1.442 * | 0.176 | (1.136 to 1.831) | 1.378 * | 0.163 | (1.093 to 1.737) |
| **Direct effect** | SEP | 1.430 * | 0.174 | (1.126 to 1.815) | 1.375 * | 0.163 | (1.091 to 1.734) |
| **Indirect effect** | Health behavior pathway | 1.009 | 0.005 | (0.999 to 1.019) | 1.002 | 0.004 | (0.995 to 1.009) |
| **Percentage mediation of the total effect** | | 2.4% | | | 0.5% | | |

*p<.05, **p<.005, ***p<.001, models adjusted for age, gender, ethnicity, childhood SEP, number of years in the BHPS and pathways at baseline

### 3.4.7 Mediation by obesity pathway

Table 24 decomposes SEP's association with T2D and $HbA_{1c}$ into the proportions attributable directly to SEP and to obesity. For the decomposition of T2D, the total effect was smaller than in previous models as there was a drop in observations due to non-response to height and weight measurement. The direct effect of SEP was attenuated to the null by obesity. Obesity mediated much of the association between SEP and T2D, it explained 22.0% of the total effect. For $HbA_{1c}$, the indirect effect was not significant though it attenuated the total effect by 3.2%. The direct effect of SEP was 1.403 (1.107 to 1.779) while the total effect was 1.419 (1.119 to 1.779).

Table 24 Decomposition of SEP's association with T2D and $HbA_{1c}$ via obesity

| | | T2D n=7,792 P-R2=.13 | | | $HbA_{1c}$ n=1,444 P-R2=.10 | | |
|---|---|---|---|---|---|---|---|
| | **Measure** | **OR** | **SE** | **95 % CI** | **OR** | **SE** | **95 % CI** |
| **Total effect** | SEP & obesity | 1.336 * | 0.172 | (1.038 to 1.719) | 1.419 ** | 0.172 | (1.119 to 1.799) |
| **Direct effect** | SEP | 1.254 | 0.162 | (0.974 to 1.614) | 1.403 * | 0.170 | (1.107 to 1.779) |
| **Indirect effect** | Obesity | 1.066 *** | 0.015 | (1.036 to 1.096) | 1.011 | 0.013 | (0.985 to 1.038) |
| **Percentage mediation of the total effect** | | 22.0% | | | | 3.2% | |

*p<.05, **p<.005, ***p<.001, models adjusted for age, gender, ethnicity, childhood SEP, number of years in the BHPS and pathways at baseline

### 3.4.8 Mediation by all pathways

The results in Table 25 show that the total effect of SEP on T2D was significant in the full model. The direct effect of SEP on T2D was insignificant with the addition of all of the mediating pathways to the model and the indirect effect was significant. For $HbA_{1c}$, the direct effect of SEP on $HbA_{1c}$ was still significant with the addition of all the explanatory pathways to the model. The direct effect of SEP on $HbA_{1c}$ was 1.372 (1.108 to 1.751). The indirect effect was insignificant and accounted for 1.027 (0.969 to 1.088) of the total effect.

Table 25 Decomposition of SEP's association with T2D and $HbA_{1c}$ via all mediating pathways

| | | T2D n=7,359 P-R2=.13 | | | $HbA_{1c}$ n=1,436 P-R2=.10 | | |
|---|---|---|---|---|---|---|---|
| | **Measure** | **OR** | **SE** | **95% CI** | **OR** | **SE** | **95% CI** |
| **Total effect** | SEP & all pathways | 1.335 * | 0.175 | (1.032 to 1.725) | 1.409 * | 0.171 | (1.110 to 1.789) |
| **Direct effect** | SEP | 1.250 | 0.167 | (0.962 to 1.623) | 1.372 * | 0.171 | (1.108 to 1.751) |
| **Indirect effect** | All pathways | 1.068 * | 0.027 | (1.017 to 1.122) | 1.027 | 0.031 | (0.969 to 1.088) |

*p<.05, **p<.005, ***p<.001, models adjusted for age, gender, ethnicity, childhood SEP, number of years in the BHPS and pathways at baseline

The proportion of the total effect mediated by the pathways was 23.8%. Table 26 shows the contribution of each measure to the indirect effect. Neither measure of material deprivation contributed significantly to this, income contributed negatively to explaining the total effect. Similarly, the psychosocial pathway was not a significant mediator. Physical activity made a very modest, insignificant contribution to explaining the total effect. The total effect was largely explained through obesity. Obesity was responsible for 23.4% of the indirect effect and was the only significant mediator.

The indirect measures explained 7.7% of the total effect of SEP on $HbA_{1c}$. Unlike the decomposition for T2D, low income was an important mediator though it did not make a statistically significant contribution to the indirect effect. Obesity was also somewhat important, but not statically significant, as a mediator.

Table 26 Separation of indirect effects in all mediating pathways for T2D and $HbA_{1c}$

| | | T2D | | | $HbA_{1c}$ | | |
|---|---|---|---|---|---|---|---|
| **Pathway** | Measure | _b | SE | Contribution % | _b | SE | Contribution % |
| **Material** | Food insecurity | 0.001 | 0.002 | 0.3% | -0.003 | 0.005 | -0.9% |
| | Low income | -0.004 | 0.020 | -1.5% | 0.017 | 0.025 | 4.8% |
| **Psychosocial** | Psychosocial distress | 0.001 | 0.004 | -0.2% | -0.001 | 0.002 | -0.1% |
| **Health behavior** | Low physical activity | 0.005 | 0.005 | 1.8% | 0.001 | 0.003 | 0.2% |
| **Obesity** | Obese | 0.067 | 0.015 | 23.4% | 0.012 | 0.013 | 3.6% |
| **Percentage mediation of the total effect** | | | | 23.8% | | | 7.7% |

## 3.5. Discussion

### 3.5.1 Main findings

Evidence was found of an association between SEP with both T2D and $HbA_{1c}$. For T2D, the direct effect of SEP on T2D became insignificant with the addition of all mediating pathways though only obesity significantly contributed to this. The indirect effect explained approximately 23% of the total effect of SEP in T2D. The importance of obesity may be due to it being more biologically associated with T2D than the other factors. Although the other pathways did not explain significant proportions of the association between SEP and T2D, low physical activity and psychosocial distress did have significant direct effects on T2D. Physical activity and psychosocial stress are related to propensity to develop T2D but they are not important mediators of the association between SEP and T2D. The material pathway was not significant as a mediator nor did it have a significant direct effect.

For $HbA_{1c}$, the direct effect of SEP was still significant with the inclusion of all explanatory pathways and the indirect effect, which explained approximately 8% of the total SEP association, was not significant. None of the pathway measures had a significant mediating effect, SEP continued to have a significant direct effect and low income was the strongest mediator suggesting material factors may be more important for this outcome. The difference in findings between T2D and $HbA_{1c}$ may reflect capturing those with prediabetes, SEP differences in gaining a diagnosis of T2D and managing it after diagnosis. Previous research has found SEP disparities in communication with physicians (Verlinde et al., 2012), which could influence gaining a diagnosis and managing the condition. Low income may be important for managing diabetes due to the increased costs of healthy and specialised food. Physical activity and obesity did have significant direct effects on $HbA_{1c}$ without being important mediators of the SEP association. Psychological distress did not have a significant direct or indirect effect.

Previous findings in this area which explored SEP, T2D and $HbA_{1c}$ have been built upon by formally decomposing the association between SEP and T2D and $HbA_{1c}$ into material, psychosocial and behavioural pathways. The aim of this has been to explore how socio-economic experience leads to physiological change.

### 3.5.2 Findings in relation to other studies

Much research has focused on SEP's association with T2D and $HbA_{1c}$ controlling for some of these explanatory pathways however, no research thus far has explored the role of each pathway as a mediator in unison. Many studies have considered health behaviours as a confounder or mediator in exploring the association between SEP and T2D and $HbA_{1c}$ (Lapidus et al, 2008; Tanaka et al, 2012; Dinca-Panaitescu et al, 2011; Lawlor et al, 2007; Anderson et al, 2008; Maier et al, 2014; Bardenheier et al, 2013; Power et al, 2014). Some previous research considered both health behaviours and psychosocial experience as either mediators or confounders (Agardh et al, 2007; Tsenkova et al 2014; Kumari et al 2004; Kroenke et al; 2007; Heraclides et al, 2009).

However, no other research in this area has considered material deprivation and food insecurity in particular as part of this, as a separate pathway between SEP, diabetes, and $HbA_{1c}$. This is an important contribution as it allows for exploration of how financial deprivation and food insecurity may be specific mechanisms between SEP and diabetic outcomes. Previous research has found an association between food insecurity and diabetes and $HbA_{1c}$ (Seligman et al, 2007; Seligman et al, 2010; Heerman et al, 2015; Bernard et al, 2015). Food insecurity is a specific type of poverty, which may affect propensity to diabetes in ways that disadvantaged SEP in general, or other forms of poverty may not. However, research that has examined food insecurity and diabetes has focused on its direct effect on diabetes and not on its role as a mediator between SEP and diabetes nor has it been considered in conjunction with other pathways. Considering the role of food insecurity without these other pathways could result in overstating its importance.

Additionally mediation between of the SEP association is explored here in a more formal way than much previous research considering attenuation. Some previous research has conducted mediation analysis in this area; (Walker et al, 2015; Demakakos et al, 2012; Espelt et al, 2012; Maty et al, 2008; Lidfeldt et al, 2007; Best et al, 2005). The analysis in this chapter is most similar to that of Tsenkova et al (2014). In that study, a mediation analysis was used to consider the role of waist circumference, physical activity and depressive symptoms were as pathways between socio-economic disadvantage and raised $HbA_{1c}$ (prediabetes). This is built upon here by including a material deprivation pathway. This chapter also benefitted from using longitudinal data; SEP was captured prior to the measurement of mediators and baseline measures of the mediators were included in the model. This

allowed for more reliable exploration of the temporal and directional patterns of association between SEP, mediators and T2D and $HbA_{1c}$.

Another contribution of the chapter is that it explores mediation on two outcomes, self-reported incidence of T2D and raised $HbA_{1c}$. Having $HbA_{1c}$ above 6.5% is indication of having diabetes, and $HbA_{1c}$ that is raised but not at diabetic levels is suggests risk of developing T2D. In decomposing the pathways mediating the SEP association with both outcomes, it allows for understanding of how the association with SEP and the role of mediators may vary between the two outcomes. This is important as differences in the pathways between the two outcomes may be related to differences in diagnosis and ability to manage to diagnosis, both of these being important to future health outcomes and the development of comorbidities.

### 3.5.3 Limitations and strengths

This study has several limitations pertaining to measurement of concepts and the sample available for some parts of the analysis.

The main limitation to the contribution of this chapter is that only a small subset of the sample was present and participated in biomarker collection. The results from the pathway analysis on both outcomes are not directly comparable. It was decided to use an unbalanced panel for modelling the T2D analytical sample in order to utilise the rich data collected in the BHPS, which limits their comparability with those who were present for $HbA_{1c}$ collection.

The analysis of both T2D and $HbA_{1c}$ is not generalisable to the general population due to attrition from the BHPS, previous research has shown associations between health and education with attrition in BHPS (Uhrig, 2008) and the data were not weighted. However, using weights would have resulted in the loss of a substantial proportion of the sample.

The main aims of the chapter were to decompose the SEP inequality in T2D and $HbA_{1c}$ and to consider whether the mediators between the two outcomes were different. The ability to do this was limited, the small number of observations with $HbA_{1c}$ as an outcome meant differences between prediabetes, undiagnosed diabetes, and well and badly managed diabetes could not be investigated. The differences in the two analytical samples means the difference in findings needs to be interpreted with caution, sensitivity analysis on these did not support the comparability of the samples. Having sufficient observations to consider the

differences between prediabetes, undiagnosed diabetes, well and badly managed diabetes in these data would require a cross sectional approach in UKHLS which would prevent the use of baseline measures or observation of long periods of exposure to adversity as available from the BHPS. Analysis of T2D as outcome using a balanced sample directly comparable with HbA1c would be inefficient and require excluding approximately 5,000 cases. Though in terms of the aim to decompose the inequality, three mediating pathways were tested in unison using an appropriate decomposition method.

Food insecurity was measured via expenditure on food, this does not capture the dimensions of food insecurity hypothesised to be associated with blood glucose directly. A more sensitive food insecurity measure may identify an association between T2D and $HbA_{1c}$ with food insecurity. The psychosocial pathway was measured by the GHQ, which captures tendency towards psychological distress (Goldberg et al., 1997). However, indicators of distress on the scale are asked in the context of whether they were felt 'recently' which might not capture persistent psychosocial distress. This measure does not capture exposure to stress, a person's coping capacity and their social support; measures of these would allow psychosocial stress to be more fully measured.

There was high non-response to the height and weight measures. These were captured by self-report which is not as reliable as objective measurement. BMI was asked late in the observation period, waves 14 and 16. It was not possible to use a baseline measure or use the proportion of time respondents spent in that condition as a mediator thus long term exposure was not captured. The measure of physical activity was limited by only being collected between every second wave between waves six and 18 and was further limited as it only collected sports, walking and swimming as activities. For each of the pathways explored, the use of binary measures of one or two indicators of that pathway may oversimplify long-term experience of material deprivation, psychosocial stress and health behaviours.

The analysis of $HbA_{1c}$ did not adjust for use of aspirin or anti-inflammatories or for high triglycerides however, a sensitivity test including these did not result in associations changing. No measure was collected for family history of diabetes, thus it was not possible to adjust for this.

The strengths of this research pertain to the simultaneous testing of different hypotheses of inequalities in diabetes outcomes. The research identified theoretical support for each hypothesis from the literature and for theoretically plausible

mechanisms through which each explanatory pathway could impact upon propensity to prediabetes, diabetes and badly managed diabetes. This approach of decomposing the effect of SEP into material deprivation, including food insecurity in particular, psychosocial stress and health behaviours has not been undertaken previously with T2D or $HbA_{1c}$. Through looking at each pathway in unison, it prevents confounding by unmeasured pathways. SEP's associations with T2D and $HbA_{1c}$ were decomposed using a method suitable for binary outcomes. Some previous research has considered how the association between SEP and T2D attenuated with the addition of mediators but without using a decomposition technique appropriate for considering indirect effects with binary measures. Through using longitudinal data and using an SEP measure set in early adulthood, it prevents reverse causality through T2D or raised $HbA_{1c}$ affecting SEP. This study benefitted from a large sample size for the analysis of T2D which was observed for between six and 18 waves.

### 3.5.4 Further research

While this research did answer its research questions, it also raises questions regarding the difference in findings between T2D and $HbA_{1c}$. Further research in $HbA_{1c}$ with a larger sample size comparing self-reported diagnosed T2D and objectively measured T2D through $HbA_{1c}$ would be useful to confirm these findings. Research utilising $HbA_{1c}$ from the large sample available in UKHLS could also explore whether there are differences mediating the SEP association of different diabetic outcomes cross-sectionally. Further research using broader measures of food insecurity and psychosocial stress may provide more insight into these pathways. Food insecurity may be better measured with questions pertaining the affordability of and access to food. A single measure of obesity was used in this research; a study with repeated measures of obesity may better measure long-term exposure. This research included ethnicity as a control measure, a very small proportion of the sample reported being of a black or minority ethnicity. This prevented undertaking a stratified analysis or using interactions to ethnicity in order to test if the associations measured differed by ethnicity, which could be explored in future research.

### 3.5.5 Policy recommendations

Inequalities in incidence of T2D and raised $HbA_{1c}$ were found in this research and obesity was an important mediator of the association with T2D. Targeting reduction in obesity among those with disadvantaged SEP could help alleviate inequality in T2D. Government efforts to reduce obesity have focused on promotion of undertaking 150 minutes of physical activity (or 75 of vigorous activity) and healthy eating such as consuming five portions of fruit a day and awareness of food labelling. However, the value of these forms of health communication alone may be limited. There is evidence to suggest that how individuals perceive their own levels of physical activity can be overestimated, and the relevance of Government guidelines to oneself can be perceived differently depending on how one views their own physical activity and health relative to those around them (Knox et al., 2015b). Knowledge of Government guidelines was not associated with undertaking the recommended level of physical activity in one study (Knox et al., 2015b). The authors recommended that campaigns to promote physical activity focus on subjective norms, portraying being physically active as something that is widely prevalent, relatable and normalised as a means to increase activity levels. Similarly, knowledge on recommended levels of physical activity was not associated with daily physical activity in a study on adolescents (Best et al., 2017). The authors suggested focusing on promoting how enjoyable physical activity and sports are rather than the health benefits. The promotion of guidelines and health benefits in relation to physical activity, diet and obesity could benefit from additionally innovative forms of communication to promote behaviour change.

# Chapter Four: Mediation, moderation and variation in the relationship between SEP and lung function

## 4.1 Introduction

Lung function is an important biomarker of respiratory health and is associated with morbidity and mortality (Hole et al., 1996; Baughman et al., 2012). Social patterning in lung function has been established by previous research however, there remains much to be understood in explaining why this social patterning exists. Understanding the pathways between SEP and lung function could aid the development of public health policy to alleviate these inequalities and help individuals to manage their risk of respiratory problems. COPD affects 4% of the population aged over 40 years, inequalities are known in its prevalence and it accounted for 5.3% of UK deaths in 2012 (Snell et al., 2016). Much previous research on inequalities in lung function has considered health behaviours, exposures, household air quality and local area air quality separately. Using UKHLS, how health behaviours, obesity and occupational exposures mediate social inequalities in lung function simultaneously is considered, this has not been done before. Furthermore, the research explores whether experience of disadvantaged childhood SEP impacts on other exposures encountered in adulthood. The role of household and area in social patterning of lung function is investigated, research to date has not considered the role of household, area as well as individual level exposures in lung function inequalities. Additionally, whether these associations differ depending on the stage of the life course is considered. UKHLS captured lung function as well as health behaviours and socio-economic data needed to answer the research questions from a representative sample of the British adult population. It was easily linked to area data providing indicators of air pollution and household level information. Using UKHLS allows each aim to be investigated.

This chapter begins with a review of the literature on lung function and SEP, focusing on research that has considered mediation between them. How household and area SEP affect lung function through air quality is discussed. The literature review finishes by summarising the conceptual position of this chapter, it shows a path diagram of the hypothesised associations and presents the research questions. Following this, the methodology discusses the analytical sample and the measures and techniques used. The fourth section presents the results from the analysis and the fifth section discusses the findings, their contribution, and considers their strengths, limitations, policy implications and opportunities for future research.

## 4.2 Literature review

Lung function indicates how well a person's lungs work. Various measures are used which can indicate how much air a person can take into their lungs, how much air they can blow out and how quickly. Lung function indicates how well the lungs bring oxygen into the blood, remove carbon dioxide and how strong the breathing muscles are. Weak lung function impairs the body's ability to get the oxygen it needs to create energy. It is thought to peak in the early twenties and then declines with aging (Sharma and Goodwin, 2006). Lung function is known to be lower in people with disadvantaged SEP (Hegewald and Crapo, 2007; McFadden et al., 2009; Ramsay et al., 2011; Gray et al., 2013). Previously, much of this difference was assumed to be the result of differences in smoking. More recently, research has considered exposure to environmental and occupational hazards, childhood and adult SEP and health behaviours.

The literature review discusses the association between childhood and adult SEP and lung function. In reviewing the literature, research that considered mediators and socially patterned exposures was focused on. The mediators which evidence has been found on in the literature included health behaviours such as smoking, physical activity and diet, as well as obesity and occupational exposures. The importance of socio-economically patterned exposures via environmental tobacco smoke (ETS) and air pollution also emerged from the literature review. Additionally, research that has considered how these associations may change with age is discussed. The literature review discusses childhood and adult SEP and then discusses evidence for the pathway between each mediator and lung function. The literature review finishes by presenting the conceptual position developed based on previous research, presents the hypotheses as path diagrams and the particular research questions are listed.

### 4.2.1 Lung function and childhood SEP

Disadvantaged childhood SEP has been found to be associated with lower lung function in children (Demissie et al., 1996; Hegewald and Crapo; 2007 Bui et al., 2017) and may impact on achieved adult lung function. There is evidence that the environment in utero may be important for lung function both in infancy and in later life. Birth weight, which is indicative of in utero environment, has been found to be associated with lung function (Lawlor et al., 2005, Orfei et al., 2008). Disadvantaged SEP is associated with prematurity and lower birth weight, it is hypothesised that this

occurs via exposures such as stress, poverty, housing, diet and education (Reynolds et al., 2013). Maternal cigarette smoking is an important mediator of the association between SEP and birth weight (Kramer et al., 2000), and this is particularly pertinent for lung function. Lung function has been found to be associated with maternal smoking, diet and exposure to air pollution. Maternal smoking and maternal diet are socially patterned (Delpisheh et al., 2006b). Exposure to nicotine via maternal smoking is associated with smaller lungs, smaller alveoli and increased risk of bronchial hyper-responsiveness (Stocks and Sonnappa, 2013). Maternal diet can moderate the effects of smoking and air pollution through the presence or absence of antioxidants and also through affecting birth weight (Stocks and Sonnappa, 2013). Exposure to air pollution in utero has been associated with decreased lung function in neonates; the hypothesised mechanism between them is via oxidative stress and inflammation in the mother's airways affecting the blood-air barrier resulting in reduced placental blood flow and decreased transfer of nutrients (Latzin et al., 2009). A study in children at age four and a half found that maternal exposure to air pollution in pregnancy was associated with lower lung function at this age (Morales et al., 2015). Socially patterned maternal smoking, diet and exposure to air pollution impact on foetal development and initiate social patterning in lung function in utero.

Evidence has been found to suggest that the impact of low birth weight can be alleviated by successful growth in early childhood. One study found that lung function, measured in early adulthood, was improved among low birth weight children who had catch up growth between birth and age five (Suresh et al., 2015). Diet and exposure to ETS mediate the association between SEP and lung function for children (North and Emmett, 2000, Delpisheh et al., 2006a). Associations have been found between lung volume and breast feeding, post-natal nutritional intake and intake of vitamins A, D and E (Stocks and Sonnappa, 2013). Childhood exposure to ETS is associated with increased risk of lower tract respiratory infection (LTRI) (Stocks and Sonnappa, 2013) and decreased lung function in children (Beyer et al., 2009; Brunst et al., 2012; Guerra et al., 2013; Stocks and Sonnappa, 2013). The effects of this continue to adulthood, the impact of smoking is aggravated (Beyer et al., 2009) and smokers in early adulthood who were also exposed to ETS as children have steeper lung function decline than those without ETS exposure (Guerra et al., 2013). A study on school children found that exposure to air pollution from traffic was related to worse lung function, stratifying the analysis by SEP showed that the effect of air pollution was worse for those with disadvantaged SEP

(Cakmak et al., 2016). This suggests increased susceptibility to the effects of air pollution for children with disadvantaged SEP.

Research using the Newcastle Thousand Families Study found that in adults, childhood factors including birth weight, breast feeding and LTRI were significant predictors of adult lung function as well as adult smoking status (Tennant et al., 2008). The association between childhood SEP and lung function fully attenuated with the addition of these factors. One study on the 1958 National Childhood Development Study (NCDS) found financial adversity in childhood's association with adult lung function was mediated by housing deprivation, continuation of disadvantage and smoking. The authors suggested that childhood may be more important as a social rather than biological mediator due to the strong pathways between childhood SEP and adult SEP and smoking status (Bartley et al., 2012).

The socio-economically patterned exposures encountered by children affect their lungs in childhood and continue to impact into adulthood. The experience of disadvantaged childhood SEP may indirectly influence lung function in later life through transmission of SEP and influencing adult health behaviours.

### 4.2.2 Lung function and adult SEP

Several studies have identified an association between adult SEP and lung function. One longitudinal study of British men found that decline of lung function over a twenty year period was socially patterned, the difference in decline was greatest comparing those with disadvantaged SEP in both childhood and adulthood with those who were advantaged at both times (Ramsay et al., 2011). Adjustment was made for obesity and pack years smoking though whether these attenuated the association was not explored. In this study, it was hypothesised that occupational exposures, housing environment and air pollution as well as factors from early life may be contributing to the SEP gradient but the authors did not have the measures to test this (Ramsay et al., 2011). Lower education and 'low occupational status' were associated with steeper lung function decline over a nine year period in men but not women in the Hordaland County Cohort Study (Johannessen et al., 2010). Adjustment was made for smoking and occupational exposures though their mediating effect was not explored. The study did test whether smoking and occupational exposures moderated the effect of disadvantaged SEP and found a significant interaction between smoking and low income for men. Research using the

EPIC-Norfolk cohort study found that for men cigarette smoking explained much of the SEP gradient in Forced Expiratory Volume in the first second ($FEV_1$), and for women it explained less of the relationship (McFadden et al., 2009). Diet was also important in this study; plasma level vitamin C, an indicator of fruit and vegetable intake, attenuated the association between SEP and $FEV_1$, as did physical activity for men but not women. Living in a deprived area explained a small amount of the association and was also independently associated with $FEV_1$. Area deprivation was measured with the Townsend index and the authors hypothesised that this may have captured variation in air quality. Workplace exposures or household level air quality were not considered here. Using the Scottish Health Surveys, a study considered whether the SEP gradient in lung function and mediators of this differed by age group. It found that for young people childhood exposure to hazards and smoking were the most important mediators while environmental factors were more important for older age groups (Gray et al., 2013). A systematic review which included various different ways of measuring lung function and of conceptualising SEP consistently found evidence of an association between SEP and lung function (Hegewald and Crapo, 2007).

The literature indicates evidence of an association between SEP and lung function, several studies reviewed considered both the direct effects of and mediation via smoking, physical activity diet, obesity, occupation and area factors. None of the studies on adult groups considered the role of household exposures in addition to these factors though several of the studies on children did consider household exposures via ETS. Based on the literature, this thesis hypothesises an association between childhood SEP and lung function mediated by socially patterned exposures and health behaviours. These exposures and health behaviours are smoking, physical activity, diet, obesity and occupational exposures. The subsequent part of this section considers how SEP is associated with these mediators and how they in turn may affect lung function.

#### *4.2.2.1 Smoking*

Smoking is more prevalent among people with disadvantaged SEP in Britain (Townsend et al., 1994; Lahelma et al., 2016). In the past, smoking was more common among advantaged SEP groups but as cigarette smoking became more widespread and its health effects began to be known, advantaged SEP groups were the first to stop smoking (Pampel, 2005). Approximately 21% of the British population smoke (Gilmore et al., 2015). There has been a decline in the proportion of the population who smoke however, smoking is still more prevalent among

disadvantaged SEP groups (Gilmore et al., 2015). Smoking in adulthood is associated with decreased lung function (Macleod et al., 2015). Lung function is affected via several mechanisms. Smoking exposes the lungs to free radicals and oxidative stress which accelerate aging of the lungs and cause inflammation (Ozguner et al., 2005). Oxidative stress from smoking can result in DNA damage (Asami et al., 1997). Smoking reduces forced vital capacity (FVC) which is the amount of air a person can blow out. This has been hypothesised to be due to reduced strength of respiratory muscles via the introduction of free radicals to the vascular system reducing respiratory muscle blood supply (Tantisuwat and Thaveeratitham, 2014).

#### *4.2.2.2 Physical activity*

Evidence that physical activity is associated with SEP is less clear than the other health behaviours considered here. A systematic review found that levels of leisure time activity and moderate or vigorous physical activity were higher for those with the most advantaged SEP compared to the least advantaged but evidence of a gradient across SEP groups was less consistent (Gidlow et al., 2006). People with disadvantaged SEP are more likely to be physically active at work (Macintyre and Mutrie, 2004). Though as the levels of physical activity in occupations is declining over time, this may adversely affect the health of disadvantaged SEP groups (Stamatakis et al., 2007). SEP differences in leisure time physical activity have been explained by disadvantaged SEP men having more physically demanding occupations and disadvantaged SEP women having higher job strain, both of which are associated with lower physical activity (Makinen et al., 2010). The costs associated with physical activity and time constraints of shift work as well as reliance on more unhealthy means of weight control have also been proposed as reasons for the SEP differences in physical activity in women (Williams et al., 2011).

Physical activity can be protective for smokers, active smokers have a less steep decline of lung function that those who are inactive. The authors posited that this occurs through the anti-inflammatory effects of physical activity (Garcia-Aymerich et al., 2007). Exercise has been found to be associated with higher lung function and less steep decline in non-smokers (Nystad et al., 2006). Similarly to how physical activity can be protective against the oxidative stress of smoking, the same protective mechanism could apply to the lung function of those exposed to other oxidative hazards such as air pollution and occupational exposures.

#### *4.2.2.3 Diet*

As with the other health behaviours, diet is socially patterned (Maguire and Monsivais, 2015). People with disadvantaged SEP consume more meat and meat products, fats, sugars and preserves and less fresh vegetables, fruit and higher fibre products (James et al., 1997). SEP differences have been found in dietary knowledge which is thought to explain some of the social differential in diet (Turrell and Kavanagh, 2006, Beydoun and Wang, 2008). Material factors are likely to contribute to the differences with research on diets finding a correlation between healthy eating and food cost (Ryden and Hagfors, 2011). Low income can also affect the ability of disadvantaged SEP households to access affordable foods through restricting ability to travel to retail outlets (James et al., 1997).

Diet may have an indirect effect on lung function via obesity, and a direct link has been suggested through the consumption of anti-oxidants. This is particularly important for those who experience greater oxidative stress via smoking and environmental exposures. Anti-oxidant intake was found to be protective against steeper decline of lung function for current and former smokers (Bentley et al., 2012). Vitamin D has a protective effect on lung function for smokers (Lange et al., 2012). There is a theorised mechanism between dietary fat and lung function through fat triggering innate immune activation, inflammation and oxidative stress (Wood et al., 2010). This suggests that both intake of fruit and avoidance of excess dietary fat may be important for lung function.

#### *4.2.2.4 Occupational exposures*

Exposure to dusts, gases and chemical fumes are associated with lung function. Continued exposure to occupational dusts and fumes was associated with decline in $FEV_1$ over a five year period in participants with early COPD in the Lung Health Study (Harber et al., 2007). A systematic literature review on occupational exposures and COPD supported a causal association between the two through reviewing population based and industry specific studies as well as studies explicitly on organic and inorganic dust exposure (Omland et al., 2014). A study in France explored exposures using three differently constructed job exposure matrixes (JEM) all of which aimed to capture exposure to dusts, gases and fumes. Exposure as measured by all three JEM was associated with lower lung function (Le Moual et al., 1995). The study did not survey those in manual occupations; the authors suggest that the association could have been greater with the inclusion of those in manual occupations. It has been proposed that a healthy worker effect may also lead to the effects of occupational exposures being under reported (Rushton, 2006). Another

study which used a JEM to look at the effect of exposure to vapour, gases, dusts and fumes on lung function did not find an association (Zock et al., 2001). The authors hypothesised that this was due to the young age of the sample (aged 20 to 44) and that the effects of these exposures may become apparent later in the life course.

Particular occupations have higher risks of exposure to dusts, gases and chemical fumes. Agricultural workers may encounter gases and organic dusts, textile workers can be exposed to harmful dusts and those using welding processes can encounter chemical, physical and radiation risks (Rushton, 2006). Mechanics and service technicians can be exposed to fumes as well as gases and metal dust, construction workers may encounter organic and inorganic dusts, gases and fumes. The main mechanism through which occupational exposures impact on lung function are through chronic inflammation, changes to small airways (bronchitis) and loss of lung parenchyma (emphysema) due to oxidants, these can lead to cell injury through pro-inflammatory factors (Hnizdo and Vallyathan, 2003).

#### *4.2.2.5 Obesity*

Obesity in Britain varies by SEP; those with disadvantaged SEP often report higher rates of obesity and overweight (Evans et al., 2000; Howel et al., 2013). This is consistent using BMI and waist circumference. Inequalities in obesity are prevalent in other rich countries and are also stronger among women than men (Devaux and Sassi, 2013). SEP in childhood and in adulthood have been found to be associated with BMI in British adults (Power et al., 2007). Recent research found that underweight as well as obesity were both be more prevalent among those who are unemployed (Hughes and Kumari, 2017).

Obesity is thought to affect lung function through how fat is deposited around the lungs and through inflammation. $FEV_1$ and FVC are minimally affected by obesity unless a person is morbidly obese though both decrease with increasing obesity (Parameswaran et al., 2006). Bariatric surgery reverses these negative effects suggesting that obesity is directly important for lung function (Brazzale et al., 2015). This is supported by studies where obesity as measured by waist to hip ratio was associated with lung function after adjusting for physical activity (Canoy et al., 2004; Lin et al., 2006; Steele et al., 2009). It has been suggested that the association is due to the increased systemic inflammation caused by obesity (Thyagarajan et al., 2008). The deposition of fat in the chest wall, abdomen and upper airway has been found to reduce lung volume (Brazzale et al., 2015). This suggests that abdominal obesity, as well as being obese in itself, is problematic for lung function.

### 4.2.3 Household environmental tobacco smoke and SEP

Indoor air quality can affect lung function. It is determined by pollutants such as second hand tobacco smoke or ETS, radon, nitrogen dioxide ($NO_2$) and carbon monoxide (CO) and particulate matter (PM) which are produced by cooking ($NO_2$ and CO also from heating) and volatile organic compounds (VOC) and ozone ($O_3$) from cleaning products and paint. Indoor air pollutants can cause or worsen respiratory diseases, cause a decline in respiratory function and create sensitivity to allergens (Franchi et al., 2006). ETS is focused on here due it being socially patterned (Bolte et al., 2010). Children from disadvantaged SEP households had higher levels of cotinine (a marker of exposure to nicotine) in a study of schoolchildren in Liverpool (Delpisheh et al., 2006b).

Exposure to ETS has been associated with respiratory symptoms (Delpisheh et al., 2006a) and decreased lung function in children (Beyer et al., 2009; Brunst et al., 2012; Guerra et al., 2013; Stocks & Sonnappa, 2013). Similar findings have also been reported in adults, exposure to ETS was associated with self reported respiratory symptoms in a study which compared bar workers before and after the introduction of the smoke-free legislation (Ayres et al., 2009). A small but significant decrease in lung function has been found for non-smoking adults who had ETS expsoure as measured by higher cotinine levels but not as measured by living with a smoker (Carey et al., 1999). A study on a small sample in Scotland found that lung function was adversely affected by being exposed to ETS in the workplace but not at home (Chen et al., 2001). This suggests that the effects of ETS may vary depending on the environment or concentration of ETS.

### 4.2.4 Air pollution and SEP

Air pollution is the presence of harmful particulates and gases, including $NO_2$, PM, sulphur dioxide ($SO_2$), benzene ($C_6H_6$), $O_3$ and CO. There has been some variation in findings from research exploring SEP, air pollutants and lung function. Findings have differed depending on the measure of air pollution used and the age of those surveyed. Certain measures of air pollution are more likely to be found in deprived areas in the UK (Pye et al., 2001). Previous research on air pollution, SEP and respiratory health (Wheeler and Ben-Shlomo, 2005) modelled the effect of SEP on the odds of being in a 'poor' air quality category with separate models for urban and rural areas. They found that in urban areas, disadvantaged SEP households were more likely to live in areas of poor air quality but that this did not persist in rural

areas. Air quality had a positive association with lung function ($FEV_1$), the social gradients in lung function were not attenuated by air quality; both had a significant but modest effect. There was also indication of an interaction between disadvantaged SEP and poor air quality for men. It was posited by the authors and by previous research (Jerrett et al., 2004), that the un-attenuated association between SEP and $FEV_{1,}$ was due to material deprivation creating increased susceptibility to detrimental effects of air pollution, greater exposure to workplace pollutants and disadvantaged SEP being associated with lower mobility and lower error in exposure assessment.

Air pollution is greatest in urban areas and close to major roads and lowest in rural areas. In urban areas, deprivation is associated with higher air pollution but in rural areas, those with disadvantaged SEP live in remote areas with lower air pollution and advantaged SEP groups live in commuter areas and closer to main roads (Wheeler, 2004). Depending on the air quality indicator used, there may be differences in the social patterning of air pollution across urban and rural areas. Proximity to land fill sites was greater for those with disadvantaged SEP in rural areas, land fill sites have linked to respiratory illness (Mataloni et al., 2016). Discrete aspects of air pollution affect lung function in different ways. $SO_2$ causes constriction of the airways in the lungs, particularly affecting people with asthma and pre-existing COPD. It also contributes to the ill health effects caused by PM. Both long and short-term exposure to PM, particles with a diameter of either below 10 micrometres (µm) (PM10) or below 2.5µm (PM2.5), are associated with respiratory and cardiovascular illness and mortality. Long-term exposure to PM has been found to be associated with mortality and respiratory related morbidity. PM is thought to cause inflammation though whether this is the exact mechanism at work is still being explored (Scapellato and Lotti, 2007). $NO_2$ is associated with inflammation of the airways and can enhance the response of allergens in people who are sensitive to them; long-term exposure may affect lung function and respiratory symptoms. Air pollution causes oxidative stress via the production of free radicals (Moller et al., 2014, Miller, 2014). One study in the USA considered levels of pollutants, $NO_2$, PM2.5 and $O_3$, within the levels set by Environmental Protection Agency and found exposure within these levels was still associated with lower lung function (Rice et al., 2013).

### 4.2.5 Age

Previous research has found age variation in health behaviours and exposure's relationships with lung function. In one study, the role of mediators varied with age, whereby social inequality in lung function widened with age and these trends persisted for those who had never smoked (Gray et al., 2013). This may indicate that the detrimental effects of environmental exposures accumulate over the life course or that the elderly are more susceptible to their effects. It has also been found that the elderly, children and those with comorbidities have greater vulnerability to the adverse health effects of smoking (Makri and Stilianakis, 2008).

In addition to sensitivity to different hazards changing with age, exposure to hazards and participation in health behaviours also change with age. Smoking is more prevalent among young adults and decreases after middle age, concurrently the proportion of former smokers in the population increases with age (Marston et al., 2014). Level and type of physical activity undertaken vary with age. Research using the HSE found that the proportion of people undertaking at least 30 minutes of physical activity at least five days a week decreased with age (Belanger et al., 2011). The study also found that people aged 16 to 24 were more likely to do exercise or fitness activities while people aged 65 and older were more likely to be physically active through domestic activities. Despite declining physical activity, diet is healthier in older age groups. Older people consume more fruit and vegetables than younger people (Swan, 2004). Fruit and vegetable consumption was lowest among young adults (19 to 24 years) and highest in those aged 50 to 64 years. The association between obesity and age is different again. Obesity is most prevalent in midlife, it is lower in old age and lowest among young adults (Swan, 2004). Exposures to hazards for lung function via occupation also differ with age. Depending on cohort and age, female participation in the labour market varies. For older cohorts, those in occupations with exposure to pollutants may have experienced greater hazards than younger cohorts who will have benefited from improved protection in the work place. Depending on age, those who have retired could be further removed from exposures or their impact may worsen through accumulation across the life course.
Participation in health behaviours and exposure to hazards differ by age and their effects vary by age, which may be a result of accumulation of risks over the life course, increased susceptibility or both of these factors.

### 4.2.6 Conceptual position

The literature shows that SEP, health behaviours, obesity, occupational exposures, and indoor and outdoor air quality are associated with lung function. It is hypothesised here that the association between SEP and lung function is mediated by health behaviours, obesity and occupational exposures and that disadvantaged SEP in childhood creates a greater susceptibility to the adverse effects of these. The literature shows that exposures related to disadvantaged SEP in childhood can have an adverse effect on lungs. Maternal smoking, maternal diet, exposure to air pollution and to ETS are associated with smaller lungs, smaller alveoli, low birth weight, increased risk of bronchial hyper-responsiveness and LTRI and poorer lung function in childhood itself. It is posited that these socially patterned exposures in childhood create a biological susceptibility to socially patterned hazards to lung function in adulthood. Disadvantaged SEP in childhood is also associated with disadvantaged SEP in adulthood and thus greater exposure to hazards, which may have a greater effect than on those with advantaged SEP. These hypothesised associations are shown in Figure 11.

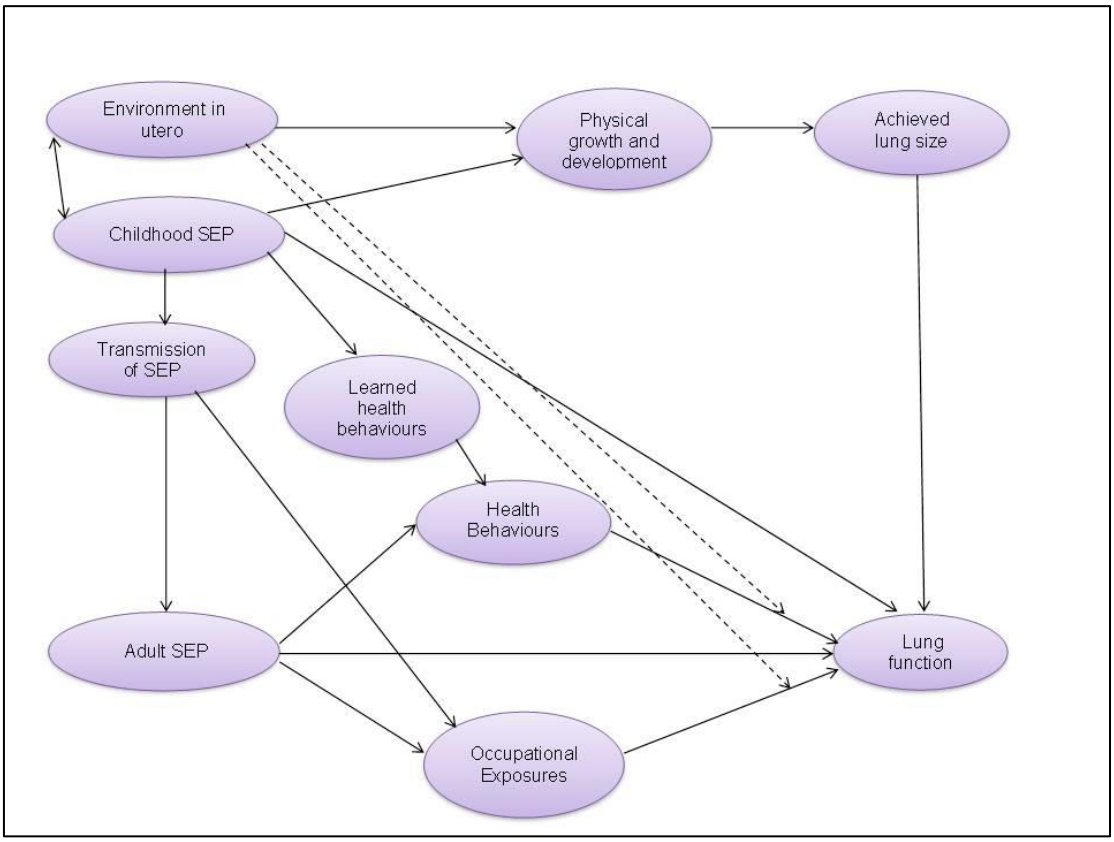
The diagram in Figure 11 represents the conceptual position on the individual level, subsequent diagrams build upon this to show how household and area are assumed to be associated with lung function. Figure 11 shows a diagram of the hypothesised associations between socially patterned exposures and lung function. The starting point here is the environment in utero and childhood SEP, these are thought to affect lung function via socially patterned growth and development initiating in utero and continuing until early adulthood. Childhood SEP is also hypothesised to indirectly affect lung function through the transmission of SEP. This influences achieved adult SEP and the occupational exposures that come with this as well as via health behaviours learned through the childhood social environment, which persist into adulthood. Adult SEP is hypothesised to indirectly affect lung function via occupational exposures to materials, which are hazardous to the lungs and through socially patterned health behaviours.

As discussed, smoking has been shown to be harmful to lung function. There is evidence to suggest that physical activity is protective against the oxidative effects of smoking and other exposures, and that diet (consumption of fruit and avoidance of dietary fats) can be beneficial to lung function through consumption of anti-oxidants and avoidance of inflammation. Occupational exposures affect lungs via chronic inflammation, changes to small airways (bronchitis) and loss of lung parenchyma (emphysema) due to oxidants, leading to cell injury through pro-inflammatory factors.

Obesity, central obesity in particular, has been posited by previous research as being important via inflammation and deposition of fat in the chest wall, abdomen and upper airway reducing lung volume. Whether childhood association with lung function persist with adjustment for hypothesised indirect pathways between them is also tested.

It is hypothesised here that people may become more susceptible to exposures as they age which could be due to this being a sensitive period or due to accumulation of exposures across the life course taking affect while lung function declines; this is not shown in the diagram. Updated versions of these path diagrams showing the variables used to measure each concept (where available) are shown in the Techniques Section (4.3.3.)

Figure 11 Path diagram of hypothesised associations between individual SEP, health behaviours and exposures and lung function



Individual level exposures are conceptualised as being nested within households within areas. Figure 12 extends the associations shown in Figure 11; the associations shown in Figure 11 are depicted as ‘associations measured at the individual level’. Socially patterned ETS within households is hypothesised to have a direct effect on lung function and the associations measured on the individual level are hypothesised to vary by household. ETS is assumed to affect lung function through the same mechanism as smoking. This thesis posits that having additional risks in one’s home increases susceptibility to the adversity experienced via individual SEP, health behaviours, obesity and occupational exposures. Conversely, those who do not experience socially patterned exposures within their homes may be better protected from adversity.

Figure 12 Path diagram of hypothesised associations between individual characteristics, household exposures and lung function

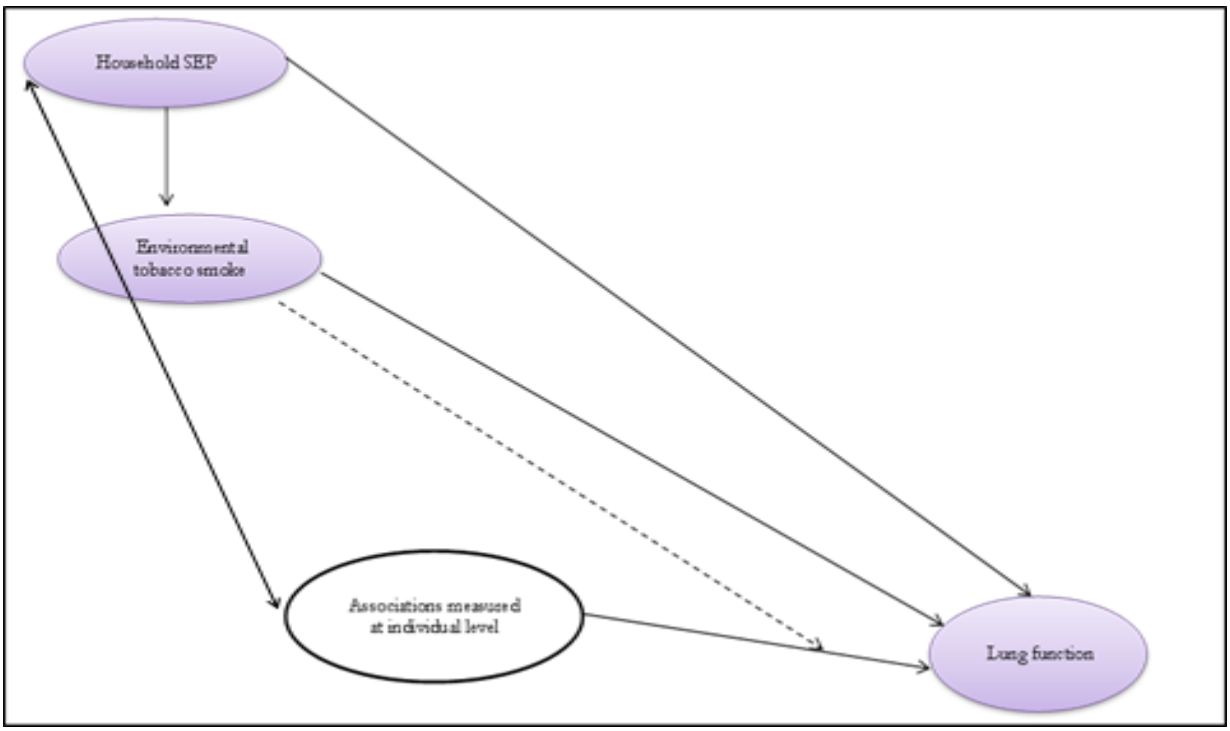
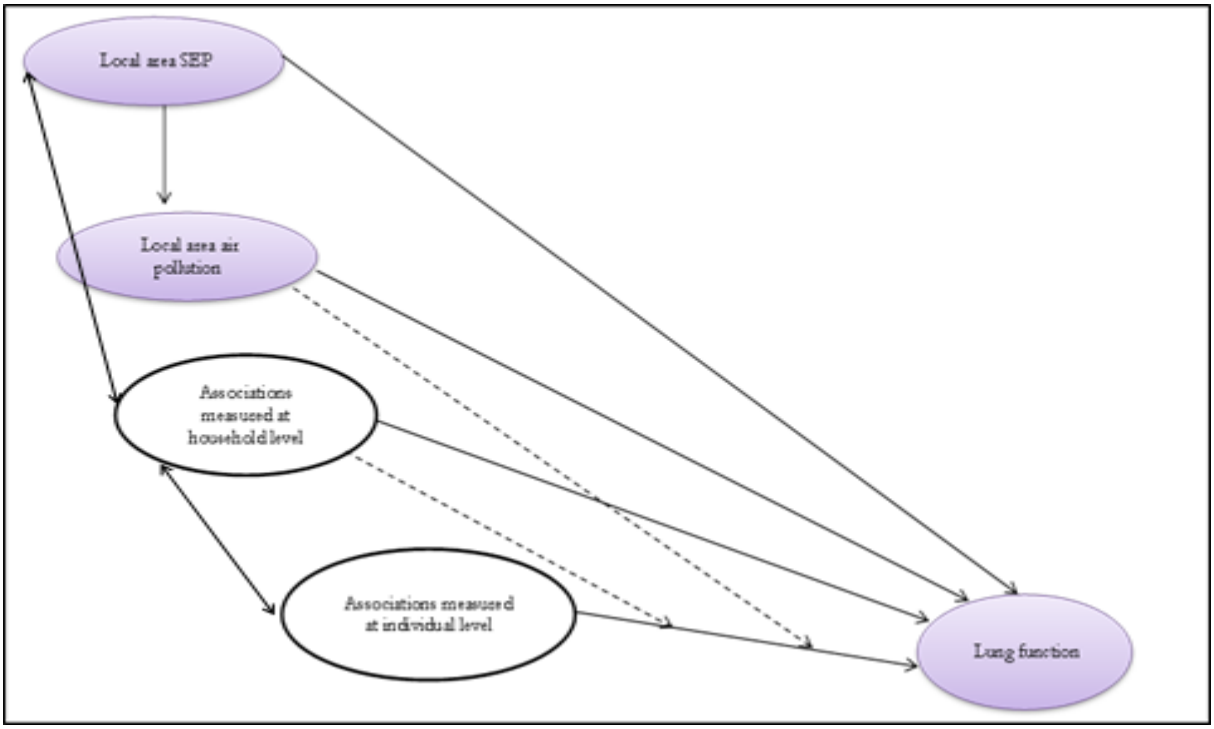


Figure 13 extends the path diagrams to add the area level to the hypothesised associations. The household level paths depicted in Figure 12 are now depicted as one eclipse. It is hypothesised that socially patterned exposure to air pollution within areas adversely affects lung function.

Figure 13 Path diagram of hypothesised associations between individual characteristics, household exposures, area exposures and lung function




Through using UKHLS, it is possible to access measures of SEP, health behaviours, occupational exposures and both household and area air quality. Previous research in this area has not considered exposures on each of these three levels in unison on adults. Previous research attempting to explain SEP differences in lung function via health behaviours is built upon by adding occupational exposures as well as area and household exposures. A novel approach is taken by considering how the effects of exposures on the individual level may vary based on household.

### 4.2.8 Research questions

1) Do adult SEP, health behaviours (i.e. smoking, physical activity, diet), obesity and occupational exposures mediate the association between childhood SEP and lung function?

   - Does childhood SEP have an association with lung function?
   - Does childhood SEP have an association with adult SEP, health behaviours, obesity and occupational exposures?
   - Do adult SEP, health behaviours, obesity and occupational exposure have an association with lung function?

2) Are the effects of smoking, physical activity, diet, obesity and occupational exposure moderated by childhood SEP?

3) Does lung function cluster within the household and area one lives in?

4) Does the association between lung function and individual level characteristics vary on the basis household?

5) Do these associations between lung function and SEP, health behaviours and occupational exposures differ depending on age?

## 4.3. Methodology

### 4.3.1 United Kingdom Household Longitudinal Study

Data used were collected from the UKHLS sample in wave two and from the BHPS sample in wave three, data from the samples were combined and analysed together (N=20,644). This is described in the Introduction to the Understanding Society Section (1.9). Data on SEP, health behaviours, occupation, household and area were all collected in wave two meaning that there was approximately a lag of one year and five months between the main survey and the NHA for the BHPS sample compared to approximately five months for the UKHLS sample. The implications of this are that responses to some variables may have changed over that time for BHPS members however as not all measures were available in wave three, responses were from wave two were used for all.

### 4.3.2 Analytical Sample

The analysis undertaken in this section is restricted to men aged over 25 years and women over 20 years in order to restrict the analysis to those whose lungs had already fully developed (Sharma and Goodwin, 2006). The analysis was further restricted to those who were resident in England; thus a further 1,568 who were resident in Scotland were removed as well as 1,392 Welsh residents. Analysis was restricted to English residents, as air pollution data were only available for England using the Index of Multiple Deprivation (IMD) for 2010.This left an analytical sample of 16,328.

### 4.3.2 Concepts and Measures

This section explains how lung function, childhood and adult SEP, health behaviours, obesity and occupational exposures are conceptualised and measured. How household and area are defined, as well as measurement of SEP and air quality within them, is described.

#### Lung function

Lung function can be measured in various ways. Forced expiratory volume (FEV) is the amount of air a person exhale, the amount of air they can blow out in the first second is called $FEV_1$ and the total amount of air exhaled is the forced vital capacity (FVC). The ratio of $FEV_1$ to FVC ($FEV_1$/FVC) is also used as a measure to test for

airflow limitation. In England and Wales, the electronic NDD Easy On-PC spirometer was used to measure lung function. It changes the energy in the breath to ultrasound and measures this. It linked directly to the nurses' laptop and recorded a range of parameters. Respondents who were pregnant, had abdominal or chest surgery in the last three months, had a heart attack in the last three months, detached retina or eye or ear surgery in the past three months, admitted to hospital with a heart complaint in the preceding month, had a resting pulse rate of more than 120 beats per minute or were currently taking medication to treat tuberculosis were excluded from measurement (McFall et al., 2013). Nurses conducted a calibration check on each day the device was used. The nurse demonstrated to participants how to use the device beginning with a full inhalation and then an exhalation that is maintained as long as possible. The nurse also shows participants a computer animation of a boy blowing up a balloon that pops as they complete their exhalation. The procedure is done seated in an upright-seated position with both feet on the floor. The nurse verbally encourages participants while doing the test. They breathe in as deeply as possible and then immediately blow the air out as hard and quickly as they can until there is no more air left in the lungs. If done incorrectly, the programme gives the nurse feedback on how to instruct the participant to do it better. This continues until a Grade of A for quality was achieved. Grade B was also acceptable if the nurse was concerned for their safety or thought the participant was too tired or unwilling (McFall et al., 2013). Grade A for quality required participants to produce two highest FVC and $FEV_1$ measurements within 100ml of each other, Grade B required the two highest measurements to be within 150ml, grade C requires a range of 200ml between measures and grade D is anything beyond this range or just one measure. Measurements with a poor quality Grade where included in this analysis, a Grade A for quality was only achieved by 51.3% of participants who provided a lung function measurement.

$FEV_1$ was used to indicate lung function in this analysis. Normal ranges for lung function including $FEV_1$ are dependent on age, height, gender and ethnicity. For this reason, $FEV_1$ was transformed using guidelines from the Global Lung Function Initiative into a percentage of the expected $FEV_1$ ($FEV_1$%) for a person of that age, height, gender and ethnicity (Quanjer et al., 2012). The reference values developed by the Global Lung Function Initiative were taken from a healthy non-smoking population. Values that are 80% to 120% of the predicted values are considered 'normal', the measure is treated as a linear variable in this analysis. This standardisation of $FEV_1$ included adjustment for height, which is associated with both

childhood SEP and lung function. This may remove some of the influence of disadvantaged childhood SEP. However as leg rather than trunk length is more influenced by childhood circumstances (Li et al., 2007, Gunnell et al., 1998) and as trunk height is more deterministic of lung function, the adjustment for height is included. Nevertheless, this may result in some underestimation of the association with childhood SEP.

Other measures of lung function include FVC and $FEV_1$/FVC. $FEV_1$ is the most widely used parameter to measure the mechanical properties of the lungs (Gildea and McCarthy, 2010). $FEV_1$ is more reproducible than FVC especially in airflow obstruction, $FEV_1$ is important in measuring the severity of the COPD though it can be less sensitive in picking up mild obstruction (Pierce et al., 2005). $FEV_1$/FVC was not considered a useful measure for the purposes of this analysis as if both FVC and $FEV_1$ are reduced as in restrictive lung diseases and lung defects then a normal $FEV_1$/FVC result is produced. FVC measures the maximum volume of air expelled from the lungs, it may be reduced due to lung compliance, chest deformity, muscle weakness and airways obstruction (in COPD). However, reduced FVC can occur despite a normal or increased lung volume, the overall accuracy of FVC for restriction is about 60%, (Gildea and McCarthy, 2010). A limitation of both FVC and $FEV_1$ is that participants must make an effort to obtain the measurement; differences in measured lung function may reflect some difference in exertion.

### Childhood SEP

Maternal education is used to measure childhood SEP. How this was measured in UKHLS and operationalised in this study is described in more detail in the Chapter 2, Concepts and Measures Section (2.3.3). Responses of ‘do not know’ and ‘other’ were reclassified as missing. The response categories were derived into a binary measure where those who reported that their mother had ‘no schooling or qualifications’ were classified as having disadvantaged childhood SEP. Those who reported that their mother had ‘some qualifications’ or ‘post school qualifications’ were classified as having advantaged childhood SEP. The measure is limited by being collected retrospectively and not directly capturing the exposures that were hypothesised to be important for lung function.

### Adult SEP

Education was used to measure adult SEP. Data collection and operationalisation of this measure is described in more detail in the Chapter 2, Concepts and Measures Section (2.3.3). This was derived into a binary measure with those who obtained

GCSE level education, 'other' qualification or no qualifications being classified as having disadvantaged SEP. Those who reported having degree level or higher, another higher qualification or A- level education were classified as having advantaged SEP.

### Smoking status

Respondents were asked in wave two of UKHLS whether they had ever smoked cigarettes and those who responded positively were asked if they still smoked cigarettes at the time of interview. Responses to these were derived into a measure that classified respondents as current smokers, former smokers and never smokers. This measure does not capture how many cigarettes daily or for how long current smokers smoke or former smokers smoked. The measure is further limited for BHPS sample members as their smoking status may have changed between wave two of UKHLS, when smoking status was collected, and wave three, when lung function was measured.

### Physical activity

Physical activity was measured via whether respondents participated in mild and moderate sports or exercise (including recreational walking) and how frequently they partook in this. The sports and exercises are listed in the Chapter 2, Concepts and Measures Section (2.3.3), the same measurement is used here. A derived variable was created which divided respondents into those who participated in mild or moderate sports at least once a week or exercised less than this. This measure does not include physical activity through housework or in one's occupation, physical activity levels may have changed for BHPS sample members between waves.

### Obesity

Obesity was measured using waist circumference. Those who had a stomach surgery such as colostomy or ileostomy, were pregnant or unable to stand were excluded from measurement. In the NHA, two measures were taken by the nurse and if these differed by more than three centimetres a third was taken. The mean of all valid waist measurements was used in this analysis. It was derived into a binary measure of 'very high' waist circumference and normal or high waist circumference. Very high waist circumference is defined as 102cm (40.2inches) or greater for a man and 88cm (34.7inches) or greater for a woman. This definition was selected as these measures of waist circumference are associated with substantially increased risk of metabolic complications in Caucasians (World Health Organisation, 2008). Waist circumference rather than other measures of obesity were used as the distribution of

fat has been shown to be important for lung function as centrally distributed fat directly affects lung function (Brazzale et al., 2015).

### Diet

Consumption of fruit was used to measure diet due to the evidence discussed in the literature review suggesting that anti-oxidant intake was important for lung function. Fruit was used rather than vegetables as fruit contains more anti-oxidants (Carlsen et al., 2010). Frequency of eating fruit was asked in wave two of UKHLS in the module on nutrition. Respondents were asked how many days in a usual week they eat fruit, including tinned, frozen, dried and fresh fruit and not including fruit juice. This was derived into a binary measure of those who ate fruit daily and those who ate fruit less than daily. The measure is limited as it does not capture the number of portions or quality of fruit consumed and diet may have changed between waves for members of the BHPS.

### Occupational exposures

Certain occupations involve exposure to dusts, gases or fumes that may be detrimental to lung function. In order to capture the risks associated with these, a job exposure matrix (JEM) which has been designed to assess risk factors relating to the development of COPD was used in this analysis (Sadhra et al., 2016). The JEM was selected as it focuses particularly on exposure to vapours, gases, dusts, fumes, fibres and mists. This scored occupation titles from the standard occupational classification in the year 2000 (SOC2000), using the four digit classification, on exposure to these. This was linked to SOC2000 classification of occupations in UKHLS and was then derived into a binary measure of whether participants experienced exposure to any of these in their current or last occupation. If respondents were not currently employed but had ever worked before, they were asked their last occupation. While this allows the inclusion of respondents who are unemployed, carers, students, retired and those who are ill or disabled, a limitation of the measure is that it less relevant for those who only spent a short amount of time in that occupation or have been long absent from it. A small proportion who had never worked were excluded.

### Age

As $FEV_1$% is used as the outcome here, $FEV_1$ is already adjusted for age. Age is used in this analysis to capture different stages of the life course in answering the fifth research question. Age was calculated from date of birth and date of interview. The variable ‘age confirmed at nurse visit’ is used in this analysis and refers to age

at the time of the nurse visit rather than at the time of the main interview. Entry to the analytical sample was limited to women who were aged at least 20 years and to men aged at least 25 years in order to limit the sample to those whose lungs had matured. Age was then classified into three categories, early adulthood, mid-life and old age. Early adulthood was defined as between ages 20 and 39, mid-life was between ages 40 and 64 and old age was classified as being 65 and older. These classifications were used to preserve the numbers that could be used in the analysis and to fit with social and biological changes over the life course.

### *Household exposures*

Sample members were classified as being within households based on their household identifier number in wave two.

#### Household SEP

Household tenure was used to measure household SEP. As it was hypothesised that household would affect lung function via socially patterned ETS, household tenure was included as a control for household SEP. Households were asked whether they owned their home outright or with a mortgage, rented from a Local Authority or Housing Association or employer or privately rented (either furnished or unfurnished) or 'other' rented. This was derived into a binary measure, household SEP was classified as disadvantaged when tenure was rented from a Local Authority or Housing Association. All other forms of tenure were classified as advantaged SEP.

#### Environmental Tobacco Smoke (ETS)

It was hypothesised that living in an environment with exposure to second hand smoke from cigarette smoking would affect lung function. There is no direct measure of this in UKHLS. Using the household identification measure attached to all members, a variable was created which identified households that included a smoker and households without any smokers. There are no questions in the survey about whether smokers smoke indoors or not, and this limited measure is used as a proxy to capture ETS.

### *Area exposures*

Lower Area Super Output Area (LSOA) was used to classify area. LSOA is defined as an area with a population between 1,000 and 3,000 individuals and between 400 and 1,200 households. The size of the area in terms of space varies based on the size of the population.

#### Area SEP

Area SEP was hypothesised to affect lung function through air pollution. Modelled without control for area SEP, air pollution could capture area SEP and other aspects of the environment which could inflate estimates, thus a measure of area SEP was included as a control. The income domain of the IMD was used to measure area SEP. The overall deprivation score on the IMD was not used as the air pollution measure used here forms part of this and additionally sex and age adjusted morbidity, disability and mortality is used for the overall IMD. The income domain of the IMD was measured by summing five indicators; adults and children in Income Support families, in income based Jobseeker Allowance families, in Pension Credit (Guarantee) families, in Child Tax Credit families whose equalised income (Excluding housing benefits) is below 60% of median before housing costs and asylum seekers in receipt of subsistence or accommodation support. The combined count of income deprived individuals per LSOA forms the numerator of an income deprivation rate, this is expressed as a proportion of the total LSOA population (McLennan et al., 2011). A binary measure of whether an area was above the mean level of deprivation was used.

#### Air pollution

The IMD for England in 2010 captures air pollution as part of the 'living environment' domain. The living environment domain includes measures of $NO_2$, PM, $SO_2$ and $C_6H_6$, each of these is a modelled estimate of the concentration of each pollutant obtained on a 1km grid from the UK National Air Quality Archive (now the UK Air Information Resource) (Department of Communities and Local Government, 2011). $NO_2$, $C_6H_6$ and PM are related to a standard value defined by the UK's National Air Strategy and $SO_2$ is related to the standard value defined by the World Health Organisation. Having a higher concentration of a pollutant beyond the standard value is deemed a risk to health or ecosystems. If grid points for emission estimates were not available within one LSOA, data from the nearest available point was used. The level of each pollutant was divided by its standard value so that a value of one was equal to the limit set, a value above one meant the level of that pollutant was higher than the limit and below one meant it was lower. These were then summed to create

an overall measure, potentially this could range from zero to infinity but in practice, values beyond four were unlikely (Department of Communities and Local Government, 2011). For example, one LSOA had a $NO_2$ measure of 1.25, a PM measure of 0.6, an $SO_2$ measure of 0.007 and a $C_6H_6$ measure of 0.06; this resulted in an air quality score 1.97. This was derived into a binary measure of whether an area was above the mean level of air pollution. The measure does not include ozone ($O_3$) which is a pollutant for which there is strong evidence of adverse health effects. It also does not measure Carbon Monoxide (CO), which can impair the transport of oxygen in the blood, nor does it include atmospheric lead. A limitation of this measure is that there is likely to be variation of air pollution within LSOA, measures of chemicals on the basis of an area are unlikely to be equally distributed within that area (Wheeler, 2004).

#### Location

Whether respondents lived in urban or rural areas was included in the analysis due to the association between this and air pollution. This dichotomous measure was derived from the National Statistics Rural and Urban Classification of Output Areas. Urban was defined as being in a settlement with a population of 10,000 or more, and rural was ascribed to addresses in settlements with a smaller population than this.

### 4.3.3 Techniques

Regression analysis is used to explore the association between $FEV_1$% and SEP, health behaviours, obesity and occupational exposures using Stata 14. The approach taken to testing for mediation was that proposed by Baron and Kenny (Baron and Kenny, 1986). Initially, whether there was an association between each mediator and the independent variable, childhood SEP, was established with logistic regression. Then whether there was an association between childhood SEP and lung function was tested with linear regression. Linear regressions were also used to test whether there was an association between each mediator: adult SEP, smoking, physical activity, diet, obesity and occupation exposure with lung function. Subsequently, each mediator was added sequentially to a model in order to assess how the coefficients of the independent variables attenuated with the addition of each mediator. The effect of adjusting for each mediator on the independent variable is expressed as its percentage change.

In order to see whether the effect of having disadvantaged SEP in childhood moderated the effect of health behaviours, obesity and occupational exposures, a

model with interactions between each of these and childhood SEP was fitted (Baron and Kenny, 1986). If the interactions were significant, this was taken as evidence of the impact of each of these as independent variables differing due to disadvantaged childhood SEP.

It was hypothesised that household and area were important for lung function and that associations captured on the individual level would vary depending on the household and the area a person lived in. A multilevel approach was undertaken in order to estimate how much variation there was in lung function on the basis of household and area as well as whether the associations with individually measured characteristics varied on these levels. Mixed models were used, initially a random intercept model was fit to measure variation in lung function was explained by household and area (Rabe-Hesketh and Skrondal, 2008). The random intercept model allows the intercept or constant to vary for on the basis of household and area. This model included the measures from the individual level and a likelihood ratio test were used to assess whether the addition of random intercepts for household and area improved the fit of the model compared to the single level regression. The measures captured at the household and area level were then added, whether these explained variation at these levels was assessed and a likelihood ratio test compared its fit with that of the previous model.

In the random intercepts model, variation was allowed in the constant but the coefficients for each parameter were assumed to have the same effect across households and areas. In order to assess if there was variation in these based on household, a model with random effects on the household level was fit. The coefficients, or effect of each variable, were now allowed to vary on the basis of households. If significant variation was found, it would indicate that the effect of individual characteristics, behaviours and exposures on lung function differ depending on the household one lives in. A likelihood ratio test was used to compare whether the addition of random effects improved the fit of the model in comparison to the random intercepts model.

In order to see whether the effect of SEP, health behaviours, obesity and occupational exposures were differed by life stage; the main part of the analysis was rerun with stratification by age. Three age groups were used, those aged between 20 and 39, 40 and 64 and aged 65 and older. The coefficients produced by these models were compared using the Wald chi square test for seemingly unrelated estimation in order to test if associations for significantly different across age groups.

Item non-response is treated as missing throughout, available case analysis is used and there is no imputation of missing values. While this allowed for efficiency by using all available data, it limits the comparability of estimates across models where the number of observations varies. This treatment of missing data requires that non-response on the outcome and explanatory variables are missing at random and treating observed cases as a random sample of whole sample (Pigott, 2001). With low levels of missing data, there is greater likelihood that it is missing at random. In this study, there are particularly high levels of non-response to maternal education and to lung function measurement. Those who did provide a response to maternal education were more likely to report low education and unhealthier behaviours in regards to smoking, physical activity and diet. They were also younger and more likely to have encountered occupational exposures. Those who did not provide lung function measurement were more likely to report low education, low physical activity and to be obese. They were also more likely to be older, to have a long-standing illness or disability and to report emphysema. As with the other chapters, the exclusion of those who may be the most disadvantaged and may have the poorest health may mean that estimates are biased and the associations between SEP and lung function found here could be weaker than exist in the population. The data are weighted to population level so that the sample should be representative of the English household population. Probability weights are used throughout.

### *Sensitivity checks*

Due to the BHPS sample participating in the NHA in wave three and having had data on health behaviours and occupation collected in wave two, analysis was reran including a control variable based on whether respondents were part of the BHPS or UKHLS samples and ran separately for the BHPS and UKHLS subsamples. Lung function is treated as a linear measure. Analyses were rerun using a binary outcome, those with a $FEV_1$% below 80% as having low $FEV_1$%. As poor health and particularly poor lung function may affect the ability to take part in lung function measurement, a further sensitivity test was ran using a binary measure of those with low lung function incorporating those who did not provide a lung function measure. The analysis was rerun split for by 'Grade A' quality lung function measurement and all other levels of quality. [14]

[14] Please see Appendix G for a brief overview of results from sensitivity checks

Figure 14 shows an updated version of the concepts and associations shown in Figure 11, this shows how these concepts are measured in the analysis and removes those which cannot be measured here.

Figure 14 Path diagram of hypothesised associations between individual SEP, health behaviours and exposures and lung function including measures

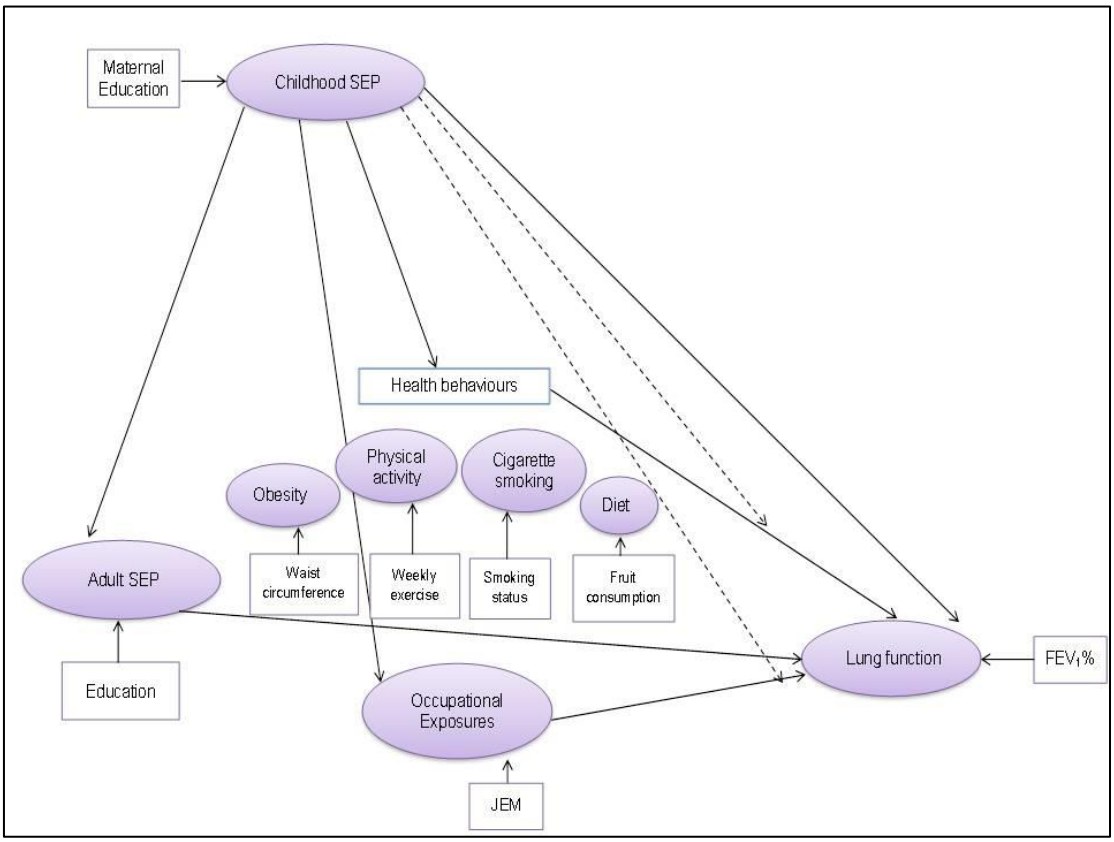
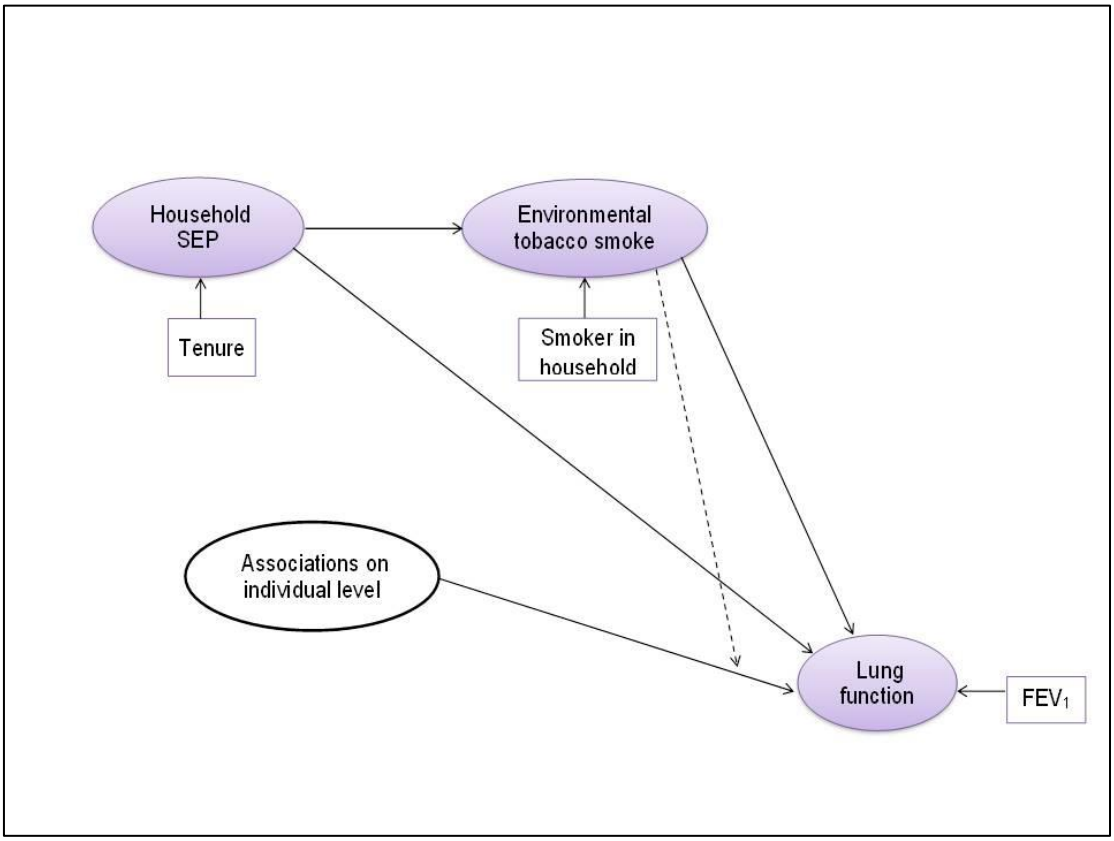


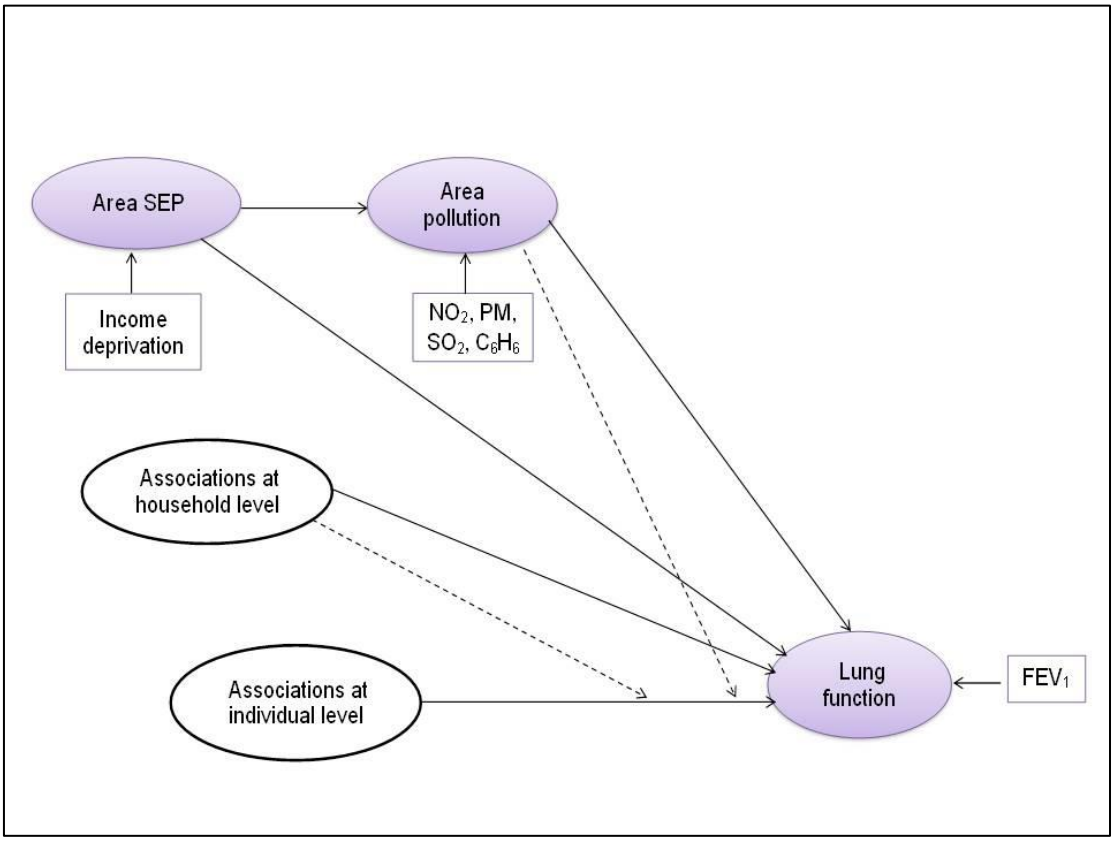
Figure 15 shows the measures used to capture concepts at the household level, the associations depicted at the individual level in Figure 14 are represented with a single eclipse. At the household level, whether another member of the household smokes was used as a proxy measure of ETS. Household tenure was used to measure household SEP. The dashed line indicates variation in individually measured associations on the basis of household. It is also hypothesised that there variation in the mean level of lung function based on household, for simplicity, this is not depicted in the diagram.

Figure 15 Path diagram of hypothesised associations between individual characteristics, household exposures and lung function including measures



At the area level, $NO_2$, PM, $SO_2$ and $C_6H_6$ are used to measure air pollution and income deprivation is used for area SEP. Figure 16 below encompasses the associations depicted in Figure 14, those measured at the individual level, and in Figure 15, those measured at the household level. For simplicity, these are depicted as one eclipse each. The dashed line indicates variation in the individually measured associations based on both household and area. Again, the mean level of lung function is hypothesised to vary by household and area but this is not shown on the diagram below.

Figure 16 Path diagram of hypothesised associations between individual characteristics, household exposures, area exposures and lung function including measures



## 4.4 Results

### 4.4.1 Description of the analytical sample

Table 27 shows the profile of the analytical sample. There were slightly more women than men, 56.9%. The mean age was 50.5 (standard deviation (SD) 17.2) ranging from 20 to 102. Forty percent of the analytical sample reported disadvantaged childhood SEP and 46.5% reporting disadvantaged adult SEP. Among the health behaviours, 20.4% reported being a current smoker while 38.8% had smoked in the past. In regards to physical activity, 39.1% reported participating in mild or moderate sports less than weekly, which indicated low physical activity. Fifty-two percent reported eating fruit less than daily indicating poor diet for the purposes of this research. A very high waist circumference, indicating obesity, was reported by 44.1%. There were 38.8% who were in occupations where they encountered exposures associated with poorer lung function. In terms of the households people live in, 26.8% who lived in a household including at least one smoker, which was used as a proxy indicator of ETS. Seventeen percent of the analytical sample rented from the council (a Local Authority or Housing Association), indicating disadvantaged household SEP. For the area measures, area income deprivation ranged between 0.01 and 0.65 with a mean of 0.14 (0.11). Higher scores indicated greater income deprivation within an LSOA. This is treated as a binary measure where being in an area with above mean income deprivation is treated as disadvantaged SEP, 37.6% were in an area with above mean income deprivation. Air pollution was measured within LSOAs and this ranged from 0.36 to 2.14 with a mean of 0.95 (SD 0.25). Again, being in an area with above mean air pollution was used to indicate the presence of air pollution. There were 37.7% in areas with above mean levels of pollution.

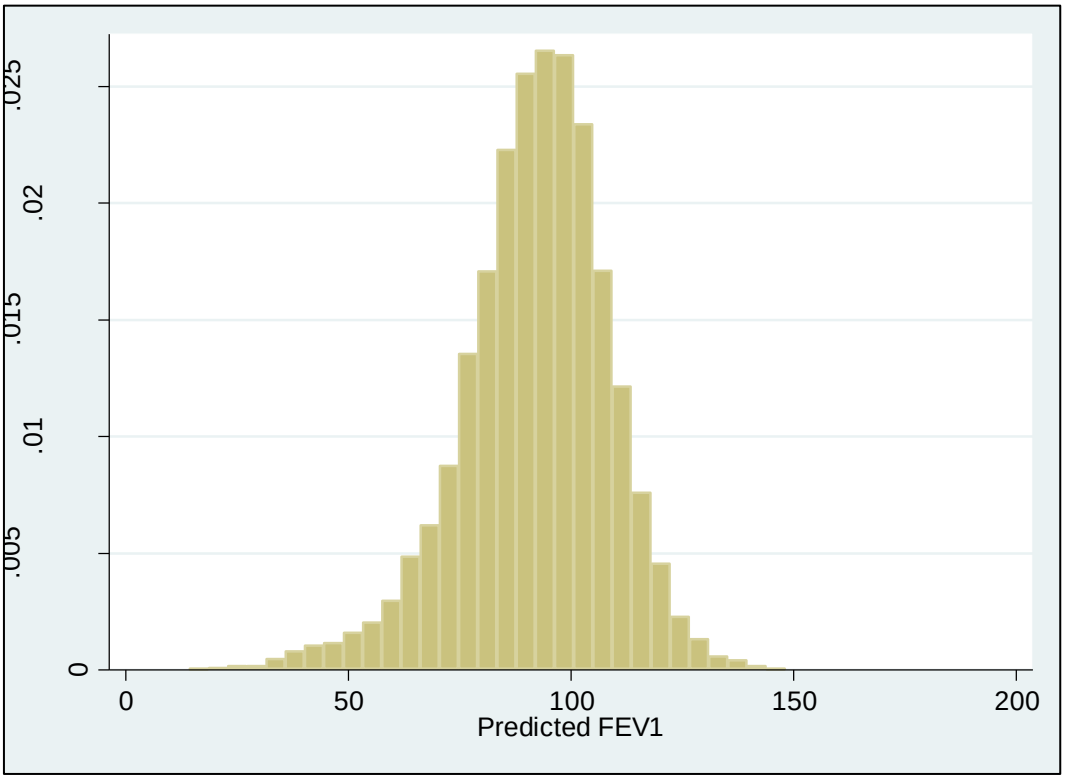
Table 27 Demographic, socio-economic, health behaviours characteristics and exposures for analytical sample with $FEV_1$% as outcome

| **Available sample n=16,328** | |
|---|---|
| **Gender** | Percentage |
| Female | 56.9% |
| **Age in years (standard deviation)** | 50.5 (17.2) |
| **Childhood SEP** | |
| Disadvantaged childhood SEP | 42.7% |
| Missing | 12.2% |
| **Adult SEP** | |
| Disadvantaged adult SEP | 46.5% |
| Missing | 0.2% |
| **Health behaviours** | |
| **Smoking** | |
| Smoker | 20.4% |
| Former smoker | 38.9% |
| Never smoked | 40.6% |
| Missing | 0.1% |
| **Physical activity** | |
| Low physical activity | 39.8% |
| Missing | 0.7% |
| **Obesity** | |
| Obese | 44.1% |
| Missing | 1.9% |
| **Diet** | |
| Poor diet | 52.0% |
| Missing | 0.3% |
| **Occupation** | |
| Occupational exposures | 38.8% |
| Missing | 9.1% |
| **Household** | |
| **Indoor air quality** | |
| ETS | 26.8% |
| Missing | 0.2% |
| **Household SEP** | |
| Disadvantaged household SEP | 17.0% |
| Missing | 0.2% |
| **Area (LSOA)** | |
| **Air pollution** | |
| High air pollution | 37.7% |
| Missing | 0.1% |
| **Area SEP** | |
| Disadvantaged area SEP | 37.6% |
| Missing | 0.1% |
| **Urban or rural location** | |
| Urban | 79.6% |
| Missing | 0.0% |

### *Lung function*

$FEV_1$% was captured for 14,195 (86.2%) of the sample. The mean $FEV_1$% was 92.0% (SD 16.5). This ranged from 14.4% to 164.4% and the median was 93.1%. The 5th percentile was 63.0% and the 95th was 116.5%. Due to the extreme range of values, characteristics of those at the extreme ends of the distribution were investigated. The majority of those with $FEV_1$% below 60% had disadvantaged SEP, poor health behaviours and greater reported of long-standing illness or disability, the converse was true of those with $FEV_1$% above 120%. These cases were retained. Figure 17 below shows the distribution of $FEV_1$%.

Figure 17 Distribution of $FEV_1$%



### 4.4.2 Is the association between childhood SEP and lung function mediated by adult SEP, health behaviours, obesity and occupational exposures?

The first research question addressed is whether childhood SEP's association with lung function is mediated by adult SEP, health behaviours, obesity and occupational exposures. In order to do this, it was necessary to establish that childhood SEP had an association with lung function, that it had an association with each of the mediators and that each of the mediators had an association with lung function. Associations between childhood SEP with lung function and with each mediator as well as between each mediator and lung function were found.[15]

Table 28 shows attenuation of the association between lung function and childhood SEP and with the addition of each mediator to the model. The number of observations (n), r-squared (R2), coefficient (_b) and 95% confidence intervals (95% CI) are shown for each model as well as the percentage change in the childhood SEP coefficient with the addition of each mediator. Figure 18 shows a plot of these coefficients to illustrate this attenuation. Childhood SEP was regressed first, before sequentially entering adult SEP, smoking status, physical activity; obesity, diet and occupational exposure to the model.

Disadvantaged childhood SEP was associated with having lung function 4.307% (5.016% to 3.714%) lower than those with advantaged childhood SEP. The addition of adult SEP to the model resulted in the childhood SEP coefficient decreasing to -3.290% (-3.960% to -2.620%) or by 23.6%. Next smoking status was added, being a current smoker was associated with much poorer lung function compared to never-smokers, their $FEV_1$% was 5.550% (6.450% and 4.651%) lower. Being a former smoker was associated with slightly a slightly lower $FEV_1$% measurement; -0.865% (-1.572% to -0.159%). The addition of smoking did not attenuate the effect of disadvantaged childhood SEP, the coefficient increased in size by 10%. Physical activity was added next, low physical activity was associated with $FEV_1$% 4.001% (4.697% to 3.305%) lower than those in the reference category. This attenuated the coefficient for childhood SEP by 14.8%. Obesity was added next, it was associated with -3.730% (-4.380% to -3.079%) lower $FEV_1$%, attenuating the effect of disadvantaged childhood SEP by 10.9%. Subsequently, poor diet was added to the model and no association between this and lung function was found. Whether people encountered occupational exposures was added last. This in itself was associated

[15] Please see Appendix E for these results.

with having $FEV_1$% approximately 1% (-1.715% to -0.365%) lower than those in the reference group. Occupational exposures attenuated the effect of childhood SEP by 7.5%.

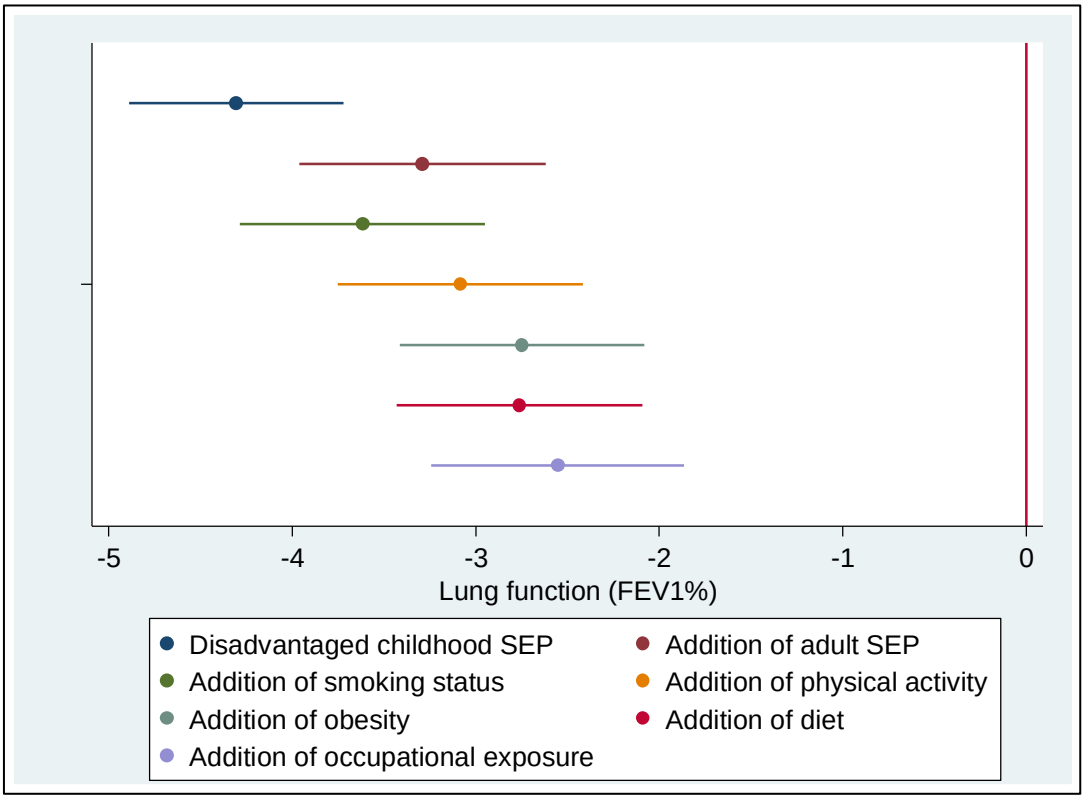
Table 28 Attenuation of association between childhood SEP and $FEV_1$% by mediators

| | Childhood SEP | | Adult SEP | | Smoking behaviour | | Physical activity | | Central obesity | | Diet | | Occupational exposures | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **N** | 12,330 | | 11,557 | | 11,549 | | 11,483 | | 11,388 | | 11,388 | | 10,437 | |
| **R2** | 0.017 | | 0.028 | | 0.043 | | 0.056 | | 0.067 | | 0.067 | | 0.068 | |
| **Childhood SEP** | **_b** | **95% CI** | **_b** | **95% CI** | **_b** | **95% CI** | **_b** | **95% CI** | **_b** | **95% CI** | **_b** | **95% CI** | **_b** | **95% CI** |
| **Disadvantaged childhood SEP** | -4.307 *** | (-5.016 to -3.714) | -3.29 *** | (-3.960 to -2.620) | -3.618 *** | (-4.287 to -2.949) | -3.084 *** | (-3.752 to -2.416) | -2.748 *** | (-3.414 to -2.081) | -2.762 *** | (-3.430 to -2.093) | -2.554 *** | (-3.244 to 1.864) |
| **Attenuation of childhood SEP** | | | -23.6% | | 10.0% | | -14.8% | | -10.9% | | 0.01% | | -7.5% | |
| **Adult SEP** | | | | | | | | | | | | | | |
| **Disadvantaged adult SEP** | | | -3.556 *** | (-4.239 to -2.872) | -2.898 *** | (-3.590 to -2.207) | -2.226 *** | (-2.914 to -1.537) | -1.826 *** | (-2.511 to -1.140) | -1.809 *** | (-2.496 to -1.122) | -1.539 *** | (-2.246 to -1.864) |
| **Health behaviours** | | | | | | | | | | | | | | |
| **Smoker** | | | | | -5.550 *** | (-6.450 to -4.651) | -5.214 *** | (-6.110 to -4.318) | -5.311 *** | (-6.204 to -4.419) | -5.245 *** | (-6.152 to -4.339) | -5.200 *** | (-6.122 to -4.269) |
| **Former smoker** | | | | | -0.865 * | (-1.572 to - 0.159) | -0.944 * | | -0.731 * | (-1.432 to -0.031) | -0.729 * | (-1.430 to -0.029) | -0.746 * | (1.461 to -0.031) |
| **Low physical activity** | | | | | | | -4.001 *** | (-4.697 to -3.305) | -3.49 *** | (-4.184 to -2.797) | -3.471 *** | (-4.170 to -2.772) | -3.258 *** | (-3.983 to -2.533) |
| **Obesity** | | | | | | | | | | | | | | |
| **Obese** | | | | | | | | | -3.73 *** | (-4.380 to -3.079) | -3.732 *** | (-4.383 to -3.081) | -3.693 *** | (-4.359 to -3.028) |
| **Poor diet** | | | | | | | | | | | -3.732 | (-0.930 to 0.372) | -0.330 | (-0.995 to 0.336) |
| **Occupation** | | | | | | | | | | | | | | |
| **Occupational exposure** | | | | | | | | | | | | | -1.040 ** | (-1.715 to -0.365) |
| **Cons** | 94.609 *** | (94.199 to 95.019) | 95.283 *** | (94.847 to 95.720 | 96.582 *** | (96.012 to 97.153) | 97.531 *** | (96.942 to 98.120) | 98.574 *** | (97.952 to 99.197) | 98.697 *** | (98.011 to 99.382) | 99.004 *** | (98.288 to 99.720) |

*p<.05, **p<.005, ***p<.001

Figure 18 illustrates the change in the coefficient for disadvantaged childhood SEP shown in Table 27. The addition of adult SEP is associated with the most substantial attenuation. The change seen with the addition of the smoking (current and former) coefficients was in the opposite direction to that hypothesised. Both physical activity and obesity were associated with a considerable proportion of attenuation. There was no attenuation with the addition of poor diet. While occupational exposures resulted in the childhood SEP coefficient attenuating by 7.5%. The coefficient produced by disadvantaged childhood SEP diminished from -4.307% to -2.554% once all measures had been added to the model. The effect of disadvantaged childhood SEP on lung function was mediated by adult SEP, physical activity, obesity and occupational exposures. The significant association between disadvantaged childhood SEP and lung function remained with the addition of these.

Figure 18 Attenuation of childhood SEP coefficients with addition of each mediator



### 4.4.3 Does childhood SEP moderate the effects of health behaviours, obesity and occupational exposures?

In order to explore whether disadvantaged childhood SEP results in increased susceptibility to the detrimental effect of harmful exposures on the lungs, each health behaviour, obesity and occupational exposure were interacted with childhood SEP to ascertain if there were significant multiplicative effects. Results are presented for main and interaction effects in Table 29.

#### *Main effects*

The main effect for disadvantaged childhood SEP was not significant once each interaction was added to the model. Disadvantaged adult SEP was associated with having $FEV_1$% 1.586% (2.295% to 0.877%) lower than those in the reference group. The main effect of smoking was significantly associated with $FEV_1$%, it was 2.640% (3.755% to 1.525%) lower than the reference group. The main effect of being a former smoker was not significant in this model. The main effect for low physical activity was significant and it was associated with having $FEV_1$% 2.857% (3.798% to 1.916%) lower. Being obese was associated with having $FEV_1$% -3.503% (-4.344% to -2.663%) lower than the reference group. No evidence was found to support an association between poor diet and lung function. For occupational exposures, the main effect of being exposed to hazards to lung function was not significant.

#### *Interaction effects*

The interaction between being a smoker and disadvantaged childhood SEP was significant; this was associated with $FEV_1$% 5.952% (7.842% to 4.063%) lower than the main effect of being a smoker implying that the effect of smoking is worse for those with disadvantaged childhood SEP. The interaction between disadvantaged childhood SEP and being a former smoker was also significant, though the main effect was not. This was associated with having $FEV_1$% 2.438% (3.888% to 0.988%) lower. There was no significant interaction effect between disadvantaged childhood SEP and low physical activity. The interaction for poor diet and disadvantaged childhood SEP was not significant, nor was the interaction between obesity and disadvantaged childhood SEP. Although the main effect of occupational exposure was not significant, the interaction between this and disadvantaged childhood SEP was associated with having $FEV_1$% 1.699% (3.062% to 0.336%) lower than the reference group.

Table 29 Interaction between disadvantaged childhood SEP and health behaviours and exposures

| N=10,437 R2=.074 | _b | 95% CI |
|---|---|---|
| **Main effects** | | |
| **Childhood SEP** | | |
| Disadvantaged childhood SEP | 0.565 | (-0.833 to 1.964) |
| **Adult SEP** | | |
| Disadvantaged adult SEP | -1.586 *** | (-2.294 to -0.877) |
| **Health behaviours** | | |
| Smoker | -2.640 *** | (-3.755 to -1.525) |
| Former smoker | 0.424 | (-0.475 to 1.322) |
| Low physical activity | -2.857 *** | (-3.798 to -1.916) |
| Poor diet | -0.553 | (-1.377 to 0.272) |
| **Obesity** | | |
| Obese | -3.503 *** | (-4.344 to -2.663) |
| **Occupation** | | |
| Occupational exposure | -0.190 | (-1.054 to 0.675) |
| | | |
| **Interactions** | | |
| Smoker * disadvantaged childhood SEP | -5.952 *** | (-7.842 to -4.063) |
| Former smoker * disadvantaged childhood SEP | -2.438 ** | (-3.888 to -0.988) |
| Low physical activity * disadvantaged childhood SEP | -1.125 | (-2.563 to 0.314) |
| Obese * disadvantaged childhood SEP | -0.453 | (-1.792 to 0.887) |
| Poor diet * disadvantaged childhood SEP | 0.553 | (-1.377 to 0.272) |
| Occupational exposure * disadvantaged childhood SEP | -1.699 * | (-3.062 to -0.336) |
| Cons | 97.609 *** | (96.748 to 98.471) |

***p<.05, **p<.005, ***p<.001, Reference group: advantaged childhood SEP, advantaged adult SEP, never smoked, physically active, good diet, not obese, no occupational exposures**

### 4.4.4 Does lung function vary on the basis of household and area?

The next stage of the analysis was to consider whether household and area were associated with lung function, thus answering the third research question. Individuals were nested within households within areas both of which contain socially patterned exposures. It was hypothesised that both household and area were important for lung function and that some of the individual level variation in lung function would be explained by their household and area.

Initially a random intercept for both household and area was added to a model with the SEP, health behaviour, obesity and occupational exposures on the individual level and without any explanatory variables at the household and area levels. This allowed for variation in the constant, the average level of lung function, on the basis of household and area. The results of this are shown in Table 30. The table shows the coefficient and 95% confidence intervals for each variable as well as the variance estimate, its standard error (SE) and 95% confidence intervals for the household and area levels.

A likelihood ratio test compared the fit of the random intercept model to an ordinary linear model and found the random intercept model improved the fit; chi2(3)= 196.21, $p$<.001. At the area level, the variance was 7.496 (SE 4.932) explaining 2.9% of variation, which was not a signification proportion of variation in lung function. The variance at household level was 43.232 (SE 6.930) this explains 18.7% of the variation in lung function. The majority of variation (78.4%) was at the individual level.

The explanatory variables measured at the individual level differed slightly to the single level regression as the multilevel approach accounts for the non-independence of observations clustered within households and areas. The association with disadvantaged childhood SEP remained unchanged though disadvantaged adult SEP attenuated very slightly. The coefficients for smoking and being a former smoker were slightly larger. While the coefficients produced by the measures of obesity, physical activity, diet and occupational exposures were broadly equivalent.

This shows that there is significant variation in average lung function based on the household one lives in. Area however, was not associated with significant variation in average lung function.

Table 30 Mixed model of $FEV_1$% with individual level predictors and random intercepts for household and area

| | | |
|---|---|---|
| **Fixed** | **_b** | **(95% CI)** |
| **Childhood SEP** | | |
| Disadvantaged childhood SEP | -2.597 *** | (-3.260 to -1.934) |
| **Adult SEP** | | |
| Disadvantaged adult SEP | -1.385 *** | (-2.076 to -0.695) |
| **Health behaviours** | | |
| Smoker | -5.551 *** | (-6.467 to -4.634) |
| Former smoker | -1.039 ** | (-1.732 to -0.346) |
| Low physical activity | -3.171 *** | (-3.871 to -2.470) |
| Poor diet | -0.441 | (-1.090 to 0.207) |
| **Obesity** | | |
| Obese | -3.733 *** | (-4.382 to -3.085) |
| **Occupation** | | |
| Occupational exposures | -1.110 ** | (-1.768 to -0.453) |
| Cons | 99.235 *** | (98.541 to 99.929) |
| | | |
| **Random Intercepts** | **Estimate (SE)** | **95 %CI** |
| Variance at area level (cons) | 7.496 (4.932) | (2.065 to 27.219) |
| | 2.9% | |
| Variance at household level (cons) | 43.232 (6.930) | (31.577 to 59.191) |
| | 18.7% | |
| Residual (individual) variance | 189.439 (5.749) | (178.500 to 201.047 |
| **Likelihood ratio test with linear model** | | chi2(3)= 196.21*** |

N=11,057, LSOA=6,260, mean n in LSOA=1.8, households=8,333, mean n in household=1.3, *p<.05, **p<.005, ***p<.001

Table 31 shows the results of a model with random intercepts for household and area with explanatory variables measured at household and area level added to the fixed part of the model in order to test whether they were association with lung function and whether they would attenuate the variation at household and area level.

Disadvantaged household SEP was associated with $FEV_1$% 2.215% (3.394% to 1.036%) less than the reference group. The coefficient produced for ETS was not significant. The area level measures: income deprivation, poor air quality and being in an urban area, were all associated with lower lung function. Income deprivation was not significantly associated with 0.757% (1.527% to 0.013%) lower $FEV_1$%. Living an area with poor air quality was associated with $FEV_1$% 1.369% (2.107% to 0.630%) lower than those in areas with better air quality. Living in an urban area was also associated with having significantly lower $FEV_1$%. The addition of these measures was associated with slight attenuation of some of the variables measured at individual level.

The variation at area level was 5.617 (SE 4.744), this was 1.9% of the total variation and this did not explain a significant proportion of variation. The household level variation was 43.182 (SE 6.762) or 18.9% of the total variation. At the area level, the unexplained variance reduced by 1.0% from the model without measures of income deprivation, air quality and urban or rural environment. The level of variation explained at the household level did not change substantially. A likelihood ratio test showed that the addition explanatory variables measured at household and area level was a significant improvement to the model, chi2(5)=242.95, $p$<.001.

This suggests that although area deprivation, air pollution and household SEP are associated with lung function, they do not explain the variation that exists in lung function based on household.

Table 31 Mixed model of $FEV_1$% with individual, household and area level predictors and random intercepts for household and area

| | | |
|---|---|---|
| **Fixed** | **_b** | **95% CI** |
| **Childhood SEP** | | |
| Disadvantaged childhood SEP | -2.560 *** | (-3.224 to -1.897 ) |
| **Adult SEP** | | |
| Disadvantaged adult SEP | -1.151 ** | (-1.846 to -0.455) |
| **Health behaviours** | | |
| Smoker | -4.839 *** | (-6.380 to -3.300) |
| Former smoker | -1.058 ** | (-1.746 to -0.369) |
| Low physical activity | -2.870 *** | (-3.567 to -2.172) |
| Poor diet | -0.269 | (-0.918 to 0.380) |
| **Obesity** | | |
| Obese | -3.639 *** | (-4.289 to -2.990) |
| **Occupation** | | |
| Occupational exposures | -0.971 ** | (-1.633 to -0.310) |
| **Household** | | |
| Disadvantaged household SEP | -2.215 *** | (-3.394 to -1.036) |
| ETS | -0.208 | (-1.557 to 1.141) |
| **Area** | | |
| Disadvantaged area SEP | -0.757 | (-1.527 to 0.013 |
| Air pollution | -2.392 *** | (-4.112 to -0.673) |
| Urban area | -1.031 * | (-1.806 to 0.256) |
| **Cons** | 108.437 *** | (106.413 to 110.461) |
| | | |
| **Random Intercepts** | Estimate (SE) | 95% CI |
| Variance at area level (cons) | 5.617 (4.744) | (1.073 to 29.402) |
| | 1.9% | |
| Variance at household level (cons) | 43.182 (6.762) | (31.770 to 58.693) |
| | 18.9% | |
| Residual (individual) variance | 189.518 (5.795) | (178.658 to 201.038) |
| **Comparison to model without household and area variables with likelihood ratio test** | | chi2(5)=242.95*** |

N=11,038, LSOA=6,247, mean n in LSOA=1.8, households=8,317, mean n in household=1.3, *p<.05, **p<.005 ***p<.001

### 4.4. 5 Do the effects of SEP, health behaviours and occupational exposure vary on the basis of household?

Table 32 shows the results of the mixed model with random effects at the household level and a random intercept at the area level. The table shows the fixed coefficients and 95% confidence intervals as well as variation in these estimated by the random part of the model, the standard error for these estimates and their 95% confidence intervals.

The fixed part of the model was largely similar to the random intercepts model, in the main coefficients were only slightly attenuated. However, the coefficient for area deprivation, which was not significant in the random intercepts model, now had a significant coefficient. The coefficient for air pollution did attenuate substantially though the urban measure, which was also on the area level, did not change meaningfully. The variation in the effect of childhood SEP based on household was significant; 37.345 (SE 8.225). For adult SEP, there was also significant variation in its effect on lung function based on household, producing a variance estimate of 16.334 (SE 8.165). The variation in the effect of being a smoker and a former smoker was also significant. There was a variance estimate for low physical activity on lung function, this had an estimate of 50.895 (SE 9.152). As there was no significant association between lung function and diet, this was not added to the random effects specification. There was significant variation in the effect of occupational exposures on the basis of household at 31.939 (SE 8.341).

The variation remaining at the area level was 4.635 (SE 4.755) and not significant. The amount of residual variation at the individual level was reduced from the random intercepts model to 151.335 (SE 6.551) in this model. A likelihood ratio test was used to compare this model to the random intercepts model and it was a significantly better fit, chi2(7)=280.75, $p<.001$.

This indicates that the effect of disadvantaged SEP, poor health behaviours, obesity and occupational exposures vary significantly on the basis of the household one lives in.

Table 32 Mixed model of $FEV_1$% with individual, household and area level predictors, random intercepts for household and area, and random effects on household level

| | Fixed | | Random | |
|---|---|---|---|---|
| | _b | 95% CI | Estimate (standard error) | 95% CI |
| **Childhood SEP** | | | | |
| Disadvantaged childhood SEP | -2.395 *** | (-3.047 to -1.743) | 37.345 (8.225) | (24.252 to 57.506) |
| **Adult SEP** | | | | |
| Disadvantaged adult SEP | -1.016 ** | (-1.697 to -0.336) | 16.334 (8.165) | (6.132 to 43.509) |
| **Health behaviours** | | | | |
| Smoker | -4.658 *** | (-6.183 to -3.132) | 23.933 (9.614) | (10.891 to 52.591) |
| Former smoker | -0.878 * | (-1.545 to -0.212) | 16.533 (7.069) | (7.152 to 38.219) |
| Low physical activity | -2.798 *** | (-3.482 to -2.114) | 50.895 (9.152) | (35.774 to 72.408) |
| Poor diet | -0.465 | (-1.089 to 0.077) | | |
| **Obesity** | | | | |
| Obese | -3.503 *** | (-4.134 to -2.873) | 17.858 (8.386) | (7.114 to 44.829) |
| **Occupation** | | | | |
| Occupational exposure | -0.845 ** | (-1.490 to -0.200) | 31.939 (8.341) | (19.287 to 52.887) |
| **Household** | | | | |
| Disadvantaged household SEP | -1.614 ** | (-2.737 to -0.491) | | |
| ETS | -0.224 | (-1.551 to 1.102) | | |
| **Area** | | | | |
| Disadvantaged area SEP | -0.836 * | (-1.586 to -0.860) | | |
| Air pollution | -1.418 *** | (-2.134 to -0.703) | | |
| Urban area | -1.140 * | (-1.897 to -0.384) | | |
| **Cons** | 102.476 *** | (101.033 to 103.920) | 8.722 (6.297) | (2.119 to 35.901) |
| **Random Intercept** | | | | |
| Variance at area level (cons) | 4.635 (4.755) | (0.621 to 34.616) | | |
| Residual (individual) variance | 151.335 (6.551) | | (139.025 to 164.735) | |
| **Comparison with random intercept model using likelihood ratio test** | | | chi2(7)=280.75*** | |

N=11,057, LSOA=6,260, mean n in LSOA=1.8, households=8,333, mean n in household=1.3, *p<.05, **p<.005, ***p<.001

### 4.4.6 Do the effects of SEP, health behaviours, obesity and occupational exposure differ on the basis of age?

The last research question was to explore whether and how the associations between SEP and socially patterned hazards differed by age. Differences by age were hypothesised to be due to the cumulative effects of disadvantage and unhealthy behaviours over the life course or due to changes in sensitivity to some exposures at different ages. This section considers the association between SEP and lung function for three different age groups; those aged 20 to 39, 40 to 64 and 65 and older. These groupings were selected in the interest of having sufficient observations for subgroup analysis as well as capturing early adulthood, midlife and post retirement.

#### *4.4.6.1 Descriptive statistics stratified by age*

Table 33 presents the descriptive statistics stratified by age for each of the key explanatory variables of interest. It is apparent that disadvantaged childhood and adult SEP are more prevalent among the older age groups probably reflecting changes over time in school leaving patterns. Smoking is less common in the older age groups while being a former smoker is more common. Low physical activity is least common among the youngest age group and most common among the oldest while the reverse is true for poor diet. Reporting having a current or last occupation in a role with exposures associated with COPD was reported by slightly less of the younger age group; 34.9% in comparison to 40.9% and 39.8% in the 40 to 64 and 65 and older categories respectively.

Table 33 Demographic, socio-economic, health behaviours and exposures for each life stage

| | 20 to 39 | 40 to 64 | 65 and older |
|---|---|---|---|
| **N** | 3,986 | 8,020 | 4,322 |
| **Childhood SEP** | | | |
| Disadvantaged childhood SEP | 18.8% | 45.1% | 68.6% |
| Missing | 13.4% | 11.5% | 12.2% |
| **Adult SEP** | | | |
| **Disadvantaged adult SEP** | 31.0% | 46.4% | 66.8% |
| **Missing** | 0.4% | 0.2% | 0.2% |
| **Health behaviours** | | | |
| **Smoker** | 26.8% | 21.9% | 9.5% |
| **Former smoker** | 30.7% | 37.0% | 53.2% |
| **Missing** | 0.1% | 0.1% | 0.1% |
| **Low physical activity** | 32.3% | 38.4% | 52.3% |
| **Missing** | 0.2% | 0.1% | 2.7% |
| **Poor diet** | 62.0% | 52.5% | 38.5% |
| **Missing** | 0.1% | 0.1% | 0.1% |
| **Central obesity** | 29.3% | 48.1% | 55.5% |
| **Missing** | 1.2% | 1.6% | 3.5% |
| **Occupation** | | | |
| **Exposures** | 34.9% | 40.9% | 39.8% |
| **Missing** | 7.7% | 4.9% | 18.3% |

### *4.4.6.2 SEP, health behaviours, occupational exposure and $FEV_1$% at different life stages*

Table 34 shows the results of childhood SEP regressed on $FEV_1$% for each age group. Childhood SEP was significantly associated with lung function for all age groups. For the youngest age group, disadvantaged childhood SEP is associated with having lower $FEV_1$% than those with advantaged childhood SEP. Their $FEV_1$% was 2.823% (4.165% to 1.482%) lower. For those aged between 40 and 64 years, disadvantaged childhood SEP was associated with a 3.204% (4.071% to 2.337%) reduction in $FEV_1$%. For those aged 65 and older, it was associated having $FEV_1$% 4.630% (6.587% to 2.674%) lower. A Wald chi square test was used to compare the coefficients between models. No significant differences between the disadvantaged

childhood SEP coefficients were identified suggesting the effect of childhood disadvantage did not vary significantly between the three age groups.

Table 34 Association between childhood SEP with $FEV_1$% for different age groups

| | Age 20 to 39 | | Age 40 to 64 | | Age 65+ | | Significantly different coefficients |
|---|---|---|---|---|---|---|---|
| | N=3,039, R2=.0084 | | N=6,291, R2=.010 | | N=3,000, R2=.0091 | | |
| | _b | 95% CI | _b | 95% CI | _b | 95% CI | |
| **Childhood SEP** | | | | | | | |
| **Disadvantaged childhood SEP** | -2.823 ** | (-4.165 to -1.482) | -3.204 *** | (-4.071 to -2.337) | -4.630 *** | (-6.587 to -2.674) | Chi(2)=2.72, *p*=.256 |
| **Cons** | 94.832 *** | (94.248 to 95.415) | 94.115 *** | (93.546 to 94.683) | 92.289 *** | (113.072 to 130.552) | |

***p<.05, **p<.005, ***p<.001**

As with the analysis for all ages, whether childhood SEP moderated the association each health behaviour, obesity, occupational exposure and was tested. However, in this case the interaction effects were mostly insignificant. The only significant interaction effect was for those with disadvantaged childhood SEP who were also smokers in the midlife and the oldest age group. It is thought that the numbers in each group became too small for precise estimation with the division into age groups.[16]

Subsequently each of the health behaviours and occupational exposure as well as the SEP measures were regressed on $FEV_1$%, this is shown in Table 35.

For those aged 20 to 39, much of the association between childhood SEP and $FEV_1$% was attenuated, the coefficient decreased by 37.4% though it continued to be significantly associated with lower $FEV_1$%. Low physical activity and being obese were associated with decreased lung function. Unexpectedly being a former smoker was associated with higher lung function than never smokers. The effect of smoking was not significant. This may be partially explained by this group having a particularly healthy lifestyle and this group will have stopped smoking in early adulthood. Having a poor diet had no significant association, nor did occupational exposures for this age group.

[16] These results are shown in Appendix F.

For people aged between 40 and 64 years, the association between childhood SEP and $FEV_1$% attenuated by 44.8% but remained statistically significant. Being a smoker was associated with having $FEV_1$% 7.769% (-9.072% to -6.467%) lower than the reference group, while being a former smoker was not associated with any difference in $FEV_1$%. Low physical activity and obesity were again associated with worse $FEV_1$%, -2.089 (-3.070% to -1.107%) and -4.010 (-4.878% to -3.141%) respectively. Poor diet was not significantly associated with lung function for this age group. Being in an occupation with exposures to hazards related to COPD was associated with $FEV_1$% approximately 1% lower than the reference group.

In the oldest age group, the association with childhood SEP was completely attenuated once the mediators were introduced to the model. Smoking was associated with a strong negative effect on $FEV_1$%, $FEV_1$% being 15.851% (19.150% to 12.553%) weaker for those who smoked compared to those who never smoked. Being a former smoker was associated with having $FEV_1$% 3.379 (5.314% to 1.443%) weaker. The effect of low physical activity and obesity were worse for this group than they were for the younger age groups being association with approximately 8.963% and 5.223% poorer lung function respectively. Being in, or previously having been in, an occupation with hazardous exposures for lung function was associated with having $FEV_1$% 2.508% (4.338% to 0.679%) lower than the reference group.

Smoking had the greatest difference in effect between the age groups. The effect of smoking became progressively worse with age. Although it was insignificant in the youngest age groups, it was associated with an approximate 16% decrease in $FEV_1$% for the oldest age group. Similarly, being a former smoker was only negatively associated with lung function for the oldest age group. The effect of low physical activity and obesity also worsened with age.

Table 35 Attenuation of association between childhood SEP and FEV1% by mediators at different ages

| | Aged 20 to 39 | | Aged 40 to 64 | | Aged 65 and older | | Significantly different coefficients |
|---|---|---|---|---|---|---|---|
| | n=2,491, R2=.0243 | | n=5,349, R2=.0828 | | n=2,200, R2=.1188 | | |
| | _b | 95% CI | _b | 95% CI | _b | 95% CI | |
| **Childhood SEP** | | | | | | | |
| **Disadvantaged childhood SEP** | -1.766 * | (-3.103 to -0.430) | -1.770 *** | (-2.659 to -0.881) | -2.062 | (-4.271 to 0.147) | Chi2(2)=0.08 |
| **Adult SEP** | | | | | | | |
| **Disadvantaged adult SEP** | -0.703 | (-1.952 to 0.545) | -1.586 ** | (-2.517 to -0.655) | -1.678 | (-3.575 to 0.220) | Chi2(2)=.1.02 |
| **Health behaviours** | | | | | | | |
| **Smoker** | -0.061 | (-1.420 to 1.298) | -7.769 *** | (-9.072 to -6.467) | -15.851 *** | (-19.150 to -12.553) | Chi2(2)=99.57 *** |
| **Former smoker** | 1.414 * | (0.184 to 2.645) | -0.552 | (-1.479 to 0.375) | -3.379 *** | (-5.314 to -1.443) | Chi2(2)=17.13 *** |
| **Low physical activity** | -1.874 * | (-3.040 to -0.868) | -2.089 *** | (-3.070 to -1.107) | -7.093 *** | (-8.963 to -5.223) | Chi2(2)=31.07 *** |
| **Obesity** | -2.062 ** | (-3.256 to -0.868) | -4.010 *** | (-4.878 to -3.141) | -4.450 *** | (-6.283 to -2.616) | Chi2(2)=5.91 |
| **Poor diet** | -0.912 | (-2.001 to 0.178) | -0.406 | (-1.287 to 0.475) | 0.252 | (-1.726 to 2.230) | Chi2(2)=1.09 |
| **Occupation** | | | | | | | |
| **Occupational exposures** | 0.020 | (-1.097 to 1.138) | -0.923 * | (-1.818 to -0.028) | -2.508 * | (-4.338 to -0.679) | Chi2(2)=5.57 |
| **Cons** | 96.134 *** | (94.895 to 97.373) | 99.204 *** | (98.282 to 100.126) | 101.662 *** | (99.148 to 104.175) | |

***p<.05, **p<.005, ***p<.001**

**Reference group: advantaged childhood SEP, advantaged adult SEP, non-smoker, physically active, good diet, non-obese, no exposures to COPD hazards in current or previous occupation**

## 4.5 Discussion

### 4.5.1 Main findings

The analysis shows childhood and adult SEP, health behaviours, obesity and occupational exposures are significantly associated with lung function. Much of the effect of disadvantaged childhood SEP is mediated by adult SEP, physical activity and occupational exposures. The association between childhood SEP and lung function was attenuated, though a significant association remained, suggesting the behaviours and exposures measured here do not fully explain the association. This suggests that childhood may be a critical period for lung function in later life or that there are other mediators of the association. Unexpectedly, smoking did not attenuate the association between childhood SEP and lung function but smoking did have a significant effect on lung function. This chapter did not find that diet attenuated SEP's association with lung function, it only had a small significant direct effect in an unadjusted model. Other studies have found diet had a greater effect (Bentley et al., 2012; Lange et al., 2012). However, these studies had a stronger focus on diet and smoking with better measures of anti-oxidant intake and no control for SEP.

It was found here that disadvantaged childhood SEP had significant interactions with hazards to lung function in adulthood, with being a current or former smoker and with occupational exposures but there was no significant interactions with physical activity, obesity or diet. This could be indicative of sensitivity to direct hazards to lung function being set in childhood. The mechanisms through which both, smoking and occupational exposures, affect lung function are mainly via oxidative stress and inflammation. Physical activity and diet can protect against these through having an anti-inflammatory effect but these did not interact with childhood SEP nor did obesity. Susceptibility to hazards is greater for those with disadvantaged childhood SEP but the protective effect of healthy behaviours is no different. Those with advantaged childhood SEP were not as vulnerable to the effects of occupational exposures or having formerly smoked. This is an important finding as it implies those with disadvantaged childhood SEP who are more likely, due to the intergenerational transmission of SEP to be disadvantaged in adulthood, are also more susceptible to the effects of socially patterned exposures.

Previous research on inequalities in lung function is complemented by considering how lung function and its association with individual level characteristics vary by household and area. A significant proportion of variation in lung function was

explained by household. This indicates that the average level of lung function varies across households, though neither the addition of a household SEP measure nor ETS to the model explained this. This suggests that other features of the household environment are important for lung function. The addition of random effects to the multilevel model showed that the effect of SEP, health behaviours, obesity and occupational exposures varied based on household. In particular, childhood SEP, physical activity and occupational exposures showed the greatest variation. This implies that aspects of the household environment, not observed here, may be moderating these associations, aggravating the effect of exposures or providing a protective environment from them. Other socially patterned household hazards, which were not measured here, may affect lung function. Possibly exposures that are not socially patterned such as PM, CO and $NO_2$ from cooking and heating have socially patterned effects due to increased susceptibility to hazards. The small variation in lung function at the area level was an unexpected finding, though the inclusion of a level for household has been found to attenuate area variation in analysis of health outcomes (Chandola et al., 2005). Evidence was still found to suggest that air pollution was important for lung function as it had a significant association in the fixed part of the model.

A significant association with the proxy measure of ETS used here was not observed. Previous research considering the effect of ETS on lung function has had contradictory findings depending on the measure used. Working in an environment with exposure to ETS and high levels of cotinine was found to be associated with lung function but living with a smoker was not (Carey et al., 1999, Chen et al., 2001), the findings on living with a smoker from this thesis supports these results.

The effects of disadvantage, health behaviours, obesity and occupational exposures varied somewhat depending on one's stage in the life course, though only significantly for being a smoker or former smoker. For each of these their negative effect on lung function became greater with age and was greatest for the oldest age group. This could be due to duration of the exposure or greater sensitivity in old age to it. Though not significantly different to the other age groups, a larger coefficient was produced for occupational exposures than observed in the younger age group. As this age group are mainly retired, the remaining effect could indicate accumulation of exposure.

### 4.5.2 Findings in relation to other studies

The findings from this chapter are mainly supported by the literature. The importance of childhood SEP for lung function in adulthood is supported by findings that show socially patterned exposures in childhood affect lung size, alveoli size and bronchial hyper-responsiveness (Stocks and Sonnappa, 2013). Childhood SEP continued to be significantly associated with lung function in this thesis with adjustment for all measured mediators. Previous research has explored the role of health behaviours as mediators between SEP and lung function (Gray et al 2013; Ramsay et al., 2011; McFadden et al., 2009; Schikowski et al., 2008) and has found that the SEP association persisted after adding mediators to the model. This chapter has added to the understanding of the role of mediators through considering occupational exposures as well as health behaviours and obesity, which had not been done before.

A novel contribution to the literature was made by expanding upon the work of previous research which considered how interactions between childhood and adult SEP affect lung function (Ramsay et al., 2011) but not between childhood SEP and adult health behaviours, obesity and occupational exposures. In exploring the moderating effect socially patterned exposures from childhood, previous research (Beyer et al., 2009; Guerra et al., 2013) has found that exposure to ETS in childhood is associated with an aggravated effect of smoking (Beyer et al., 2009) and with steeper lung function decline in smokers (Guerra et al., 2013). This chapter supports the findings of Beyer et al (2009) and Guerra et al (2013) suggesting that socially patterned exposures in childhood moderate the effect of smoking in adulthood. Their work is built upon by also testing whether physical activity, diet, obesity and occupational exposures were moderated by childhood SEP.

Previous research has found that lung function is associated with household exposures (Delpisheh et al., 2008; Beyer et al., 2009; Brunst et al., 2012; Guerra et al., 2013; Stocks & Sonnappa, 2013) as well as area exposures (Wheeler & Ben-Shlomo, 2005; Jerrett et al., 2005; McFadden et al., 2009). This research builds upon previous findings by considering the role of both area and household. One study did consider household as well as area exposures in conjunction with individual risk factors, the sample was limited to school children only and did not use a multi-level approach (Cakmak et al., 2016). This research is therefore unique; it represents the first empirical investigation to include measures of individual, household and area exposures.

The chapter builds upon previous research that has explored whether there is a differential association between SEP and lung function by age (Tabák et al., 2009; Gray et al., 2013). It is most similar to that of Gray et al (2013) in that it explores the association between SEP and lung function, how this is explained by health behaviours and whether this differs in different stages of the life course. The research by Gray et al (2013) is built upon by adding measures of diet and occupational exposures as explanatory variables. This is an important contribution to the literature as both diet and occupational exposures are important mediators of SEP's association with lung function, and both vary by age. This could indicate that these exposures are of greater importance at different stages in the life course. The research by Gray et al (2013) found that social inequalities in lung function widened with age. In unadjusted models, a larger coefficient for disadvantaged childhood SEP was seen in the oldest age group. The childhood SEP association in the oldest age group was fully explained by the mediators in this chapter. This analysis also found that occupational exposures were important in later life, no association was seen in the ages 20 to 39 age group. This supports previous research by Zock et al (2001) where no association with occupational exposures was found in a sample aged 20 to 44 years.

### 4.5.3 Limitations and strengths

This study has several limitations pertaining to measurement and the sample used. Many of the measures used were operationalised as binary measures, which may oversimplify experience of health behaviours, obesity, occupational exposure and SEP. The measure of exposure to ETS was a proxy based on whether one was living with a smoker or not. There was no direct measure of whether smokers smoked indoors or if participants were exposed to ETS in other locations. Childhood SEP was also measured crudely. As childhood SEP appeared to be an important determinant of lung function, it would be interesting to consider more detailed measures from childhood such as maternal smoking, exposure to ETS or history of LTRI. This could not be considered here, as these exposures are not captured in UKHLS. The air pollution measure used was based on modelled estimates from 2008 and these may have differed from the actual air pollution at the time of the NHA. The measure also does not capture the extent to which people were exposed to this air pollution or whether they were mobile outside of their LSOA and the modes of transport used. The analysis presented here was restricted to England and thus the results may not be representative of the wider British population. Given the cross

sectional nature of this study, it is not possible to ascertain whether poorer lung function could influence participation in physical activity.

As the sample used in UKHLS was representative of the British household population, those who are resident in institutions such as homes for the elderly or hospitals were not included in this study meaning that those who could be likely to have low $FEV_1$% were excluded. This study did not exclude sample members with comorbidities likely to impair their lung function or make adjustments for illness or use of medications.

The study benefits from a large weighted, sample of the English population and from having a broad range of measures for SEP, health behaviours, occupation, household and area level exposures. However, the generalisability of associations found here is limited by non-response to the lung function measure and to maternal education. Examining the socio-demographic and health profiles of non-respondents on each of these suggests that those who are disadvantaged and in poor health are more likely to be have been excluded from the analysis which may under estimate associations.

### 4.5.4 Further research

The findings are supportive of the hypotheses which have been tested here although further research is needed to fully understand the mechanisms through which SEP affects lung function. The association between childhood SEP and $FEV_1$ is not fully explained by the health behaviours and occupational exposures, childhood SEP appears to be an important determinant of adult lung function. Further research could help understand the mechanisms underpinning this. In this analysis, the information available on childhood experience is limited and maternal education is used as a proxy to capture disadvantage and childhood exposures. In order to disentangle the specific aspects of childhood SEP which are important for lung function it would be useful to capture measures such as birth weight, parental smoking, areas lived in throughout the life course and experience of respiratory infections. These measures were not available in UKHLS but longitudinal and cohort studies may be particularly useful to investigate this.

When considering the effect of household, an association was found with household tenure and lung function though not with ETS. ETS was measured by a proxy indicator of whether there was a smoker living in the household without

measurement of whether they smoked within the house. A more direct measure may have been more effective. Further investigation of the household environment would be useful as the measure of ETS used here may not have been effective due to being a proxy and further measures could help better understand how the household environment affects lung function.

### 4.5.5 Policy implications

The effect of smoking, being a former smoker and occupational exposures are worse for those with disadvantaged childhood SEP suggesting that those most likely to encounter these exposures are at greater risk to their effects. Policy to alleviate this inequality requires intervention via change of behaviours for smoking and via health and safety legislation for occupational exposures. The mediation analysis showed that while exposures in adulthood explain some of the association between childhood disadvantage and $FEV_1$%, childhood SEP continued to have a direct effect. This suggests that childhood may be a critical or sensitive period for lung function exposures. Interventions to alleviate socially patterned exposures in childhood may help improve respiratory health in later life. Disadvantaged childhood SEP also increases vulnerability to certain exposures in adulthood. This may be important for considering limits for hazards to lung function in occupational settings or in the environment as these are often based on the population average risk rather than the risk posed to those who are more vulnerable.  Encouraging and enabling participation in health behaviours and avoidance of risks such as smoking and obesity could potentially help alleviate some of the social inequality in $FEV_1$%.

# Chapter Five: Overall discussion

This section reiterates the main findings, considers how the choice of SEP measures may influence the findings, considers the implications for policy, and the potential for further research and concludes the thesis.

## 5.1 Main findings

This thesis set out to explore how socio-economic inequalities become health inequalities. It aimed to further our understanding of how SEP affects physiology. To do this, biomarkers were used as health indicators rather than self-reported health, morbidity or mortality. Biomarkers respond to stress and aging, and they can detect poor health before illness manifests. Using biomarkers allowed identification of minor inequalities in grip strength and lung function in mid and early adulthood respectively. The SEP differences were greater in the late adulthood for both biomarkers. These findings indicate that inequalities in these areas emerge earlier in adulthood then when problematically lower levels of grip strength or lung function are evident or when frailty or chronic illness emerge. Using biomarkers to answer the questions of this thesis has contributed to a richer understanding of how socio-economic inequalities become health inequalities.

The thesis aimed to contribute to existing knowledge on the mechanisms through which SEP affects health. Measures of material deprivation and exposures, psychosocial stress and health behaviours were used to test hypothesised pathways between SEP and the health outcomes. The importance of these pathways varied between the three biomarkers and one self-reported health measure used. This thesis also used measures of SEP from childhood and adulthood to gain insights into whether different life stages or trajectories through the life course are important for the health outcomes in question. As the findings indicate different life stages as well as different pathways are important for discrete outcomes, it indicates how socio-economic disadvantage affects distinct aspects of health through a variety of co-existing pathways and mechanisms. Each empirical chapter of this thesis produced distinct findings for each discrete biological outcome which are discussed in the remainder of this section.

For grip strength, inequality did not emerge until mid-adulthood. There was evidence of minor mediation of the association with SEP through physical activity. Thus, we do not find strong support for health behaviours as an explanation of inequalities in grip

strength. Whether there was support for any theory of the life course in explaining social patterning in grip strength was also tested. Here evidence was found to support the accumulation theory of the life course as best explaining social patterning in grip strength for women but not for men. The importance of childhood SEP was apparent, it was not fully attenuated by adult SEP or health behaviours.

In exploring whether there was support for a material, psychosocial or behavioural explanation for social patterning in T2D and $HbA_{1c}$, there was little indication to suggest these explanations were important mediators of these outcomes. The most important mediator was obesity, the role of this being both socially related to SEP as well as being more biologically related to diabetes. The difference in findings between T2D and $HbA_{1c}$ is interpreted with caution due to differences in samples used. Nevertheless, the findings suggest that different factors may mediate the associations.

Evidence was found to support material and behavioural explanations of inequalities in lung function. Physical activity and occupational exposures mediated the association between childhood SEP and lung function, though these mediators did not explain the entire gradient. The importance of childhood SEP as a predictor of adult lung function could indicate support for a critical or sensitive period theory of the life course where lung function is set or influenced by disadvantage in childhood. The household environment was important for lung function though the mechanisms through which this occurred were not observed. Area was not associated with lung function though air pollution was associated with weaker lung function. There was some evidence to suggest that susceptibility to exposures increased with age or their effect accumulates over the life course.

## 5.2 SEP measurement

SEP was conceptualised in this thesis as encompassing class, resources and status. Education was used as the primary measure to capture these though occupation and income could also have been suitable measures (Duncan et al., 2012). Education was chosen for both conceptual and practical reasons and its use as SEP measure has a range of advantages and disadvantages.

SEP measures may pertain more or less closely to particular domains of SEP. Income is more closely related to resources than occupation or education while occupation is more closely related to class. A disadvantage of using education to

measure SEP is that it is more removed from class, resources and status than occupation and income which education is a determinant of. SEP measures are not interchangeable, each affects health through different mechanisms and the associations found with health could differ had an alternative SEP measure been used (Geyer et al., 2006).

The experience of disadvantage and how SEP may affect health is likely to vary depending on whether a person experiences disadvantage in their resources, class, status or all of these domains. Having a low income in combination with a high status occupation would affect health through different mechanisms to having an adequate income in a low status occupation. Resources may impact more closely on aspects of health sensitive to material deprivation while status could impact more on stress. Relating this to the findings of this thesis, T2D and $HbA_{1c}$ may have been more closely related to occupation as an SEP measure, as previous research found autonomy and control at work were related to T2D and prediabetes (Kumari et al., 2004; Li et al., 2013; Kroenke et al., 2007; Toshihiro et al., 2008; Heraclides et al., 2009).

In addition to SEP measures being associated with health via discrete mechanisms, they may also be more closely related particular life stages. Education is related to early adulthood and can be pertinent to outcomes set in early life (Galobardes et al., 2006). This was considered particularly suitable for grip strength and lung function. Education is also more likely to be constant than income and occupation. While it does not capture short term changes in SEP, it may be more reflective of long standing SEP which could help capture accumulated exposures. This was considered applicable for the research questions explored in this thesis.

Some of the advantages of using education include that it has low refusal rates, particularly compared to income which may be perceived as sensitive (Liberatos, 1988; Berkman and MacIntyre, 1996). It is applicable to all while occupation and income may be less relevant to those who are economically inactive (Chittleborough et al., 2006). Disadvantages of using education to measure SEP include it being subject to cohort effects as educational attainment has increased over time and what constitutes low education is not consistent across age groups. The value of educational attainment may differ depending on the demographic characteristics of an individual, previous research has shown discrimination against ethnic minorities in entry to the labour market and earnings (Rafferty, 2012). Being over-qualified for one’s position in the labour market may also come about due to economic

downturns. Inconsistency of position across education, occupation and income such as being under-employed is also related to aspects of health (Peter et al., 2007).

Using education to measure SEP brought practical and some conceptual advantages. While education was fitting in terms of being more related to early life SEP, the main disadvantage was that it is more distal from class, resources and status than income and occupation. This is a limitation to how well education as an SEP measure captures these domains and has implications for understanding the mechanisms through which SEP affects health.

## 5.3 Implications of findings for policy

This thesis finds evidence of social inequalities in the three biomarkers and the self-reported outcome explored, suggesting that changes to alleviate socio-economic inequalities and socially patterned mediators are required to reduce health inequalities.

### 5.3.1 Grip strength

The findings indicated that childhood SEP was important for grip strength and the evidence for only slight mediation via adult health behaviours implies this inequality is not easily modifiable in adulthood. Those with disadvantaged SEP were found to reach peak grip strength at an earlier age with a lower level of strength, suggesting those with disadvantaged SEP become frail earlier in old age. This has consequences for government policy on extending retirement age. The state retirement age for women is currently being gradually increased from 60 to 65 years. As of April 2017, it is 63 years and nine months and will be extended to 64 years and six months in April 2018. Greater evidence of inequalities in grip strength was found among women than men in this research. This is problematic as women with disadvantaged SEP are likely to develop weak levels of grip strength at a younger age and are more likely to be in physically demanding occupation than those with advantaged SEP. While female dominated disadvantaged SEP sectors such as caring, cleaning or catering may not be as strenuous as male dominated manual occupations, they are nevertheless physically demanding. This may affect the ability of women with disadvantaged SEP to continue to work in old age.

Current government policy aims to increase the retirement age for both men and women to age 66 by 2020 and to 67 between 2026 and 2028. Life expectancy at

birth was 78.4 for men and 82.4 for women in 2009-11, however their disability free life expectancy was 64 years and 64.8 years respectively. Inequalities in the onset of physical disability and frailty in conjunction with SEP differences in the physicality of work means that those with disadvantaged SEP will be least able to continue working in old age and will be least able to negate the financial implications of stopping work before reaching state pension age. Socio-economic differences in the ability to continue working and the physical demands of occupations that are not equally distributed in the population, should be addressed in pension policy through exceptions to the minimum retirement age for those in physically stressful occupations and for those experiencing physical limitations to prevent the further exacerbation of health inequalities.

### 5.3.2 Diabetes

Inequalities in incidence of T2D and raised $HbA_{1c}$ were found in this research. The Department of Health has set standards for diabetes care in the National Service Framework for Diabetes. While these guidelines acknowledge the social inequality in prevalence of T2D and in outcomes for diabetics, specific actions to reduce this inequality are not proffered. Inequalities can be reduced by targeting prevention, diagnosis and management. Identification of prediabetes provides an opportunity for behavioural interventions to prevent progression to T2D. Including socio-economic characteristics as risk factors with demographic characteristics could aid the identification of prediabetes and allow for timely treatment of T2D. Inequalities in diagnosis could be reduced via training on how prejudices based on SEP can affect patient assessment and decision-making on whether to use diagnostic tests. This study did not find evidence of material, psychosocial or health behaviour mediators being important in explaining the social inequality in T2D or $HbA_{1c}$ though obesity was an important mediator. Targeting reduction in obesity among those with disadvantaged SEP could help alleviate inequality in T2D.

### 5.3.3 Lung function

Working in an occupation with exposures to dusts, fumes and gases was significantly associated with weaker lung function and contributed to the inequality in lung function in this research. Limits on maximum allowable pollutants are set by Control of Substances Hazardous to Health legislation. Workplaces are subject to inspections to determine whether they met the requirements set by the regulations.

The findings indicate that current regulations may not be sufficient to prevent damage to lung function or that they are not adequately enforced. In the analysis stratified by age, the effect of occupational exposures was significant for those aged 40 to 65, this group are likely to be have been exposed since the introduction of current regulations. Occupational exposures were not significant for those aged less than 40 though this may indicate that the effect of exposure accumulates or that sensitivity intensifies with increasing age. The effect was also significant for those aged 65 and older, though this group have mainly exited from the labour market. Disadvantaged childhood SEP modified the effect of occupational exposures increased risk to their harmful effects among those more likely to be exposed to them. The findings imply that either the limits set by these regulations should be revised or that inspections and enforcement should be improved. Similarly, the findings on air pollution show that legal limits are not met in some areas and that exposure to air pollution is associated with weaker lung function. The limits set are defined by the UK's National Air Strategy and the WHO (for SO2) and are based on the level required to present a risk to health or ecosystems. However, susceptibility to the risks presented by exposures is not equally distributed in the population. Although variation in the effect of air pollution was not explored in this thesis, we find disadvantaged childhood SEP moderates the effects of smoking and occupational exposure. Limits set based on the level at which exposures have a deteriorating effect on health on average in the population fail to take into account that increased susceptibility of those with disadvantaged SEP.

## 5.4 Future research

Based on the findings from this thesis, some further research questions have emerged. The importance of childhood SEP was evident for lung function and grip strength. Further research using studies with more detail on childhood could explore the mechanism between SEP in childhood and these outcomes. Given the indication of there being different pathways between SEP and self-reported diagnosed T2D and raised $HbA_{1c}$, further investigation of the different mechanisms between SEP and prediabetes, undiagnosed diabetes, well and badly managed diabetes would enable greater understanding how SEP affects each of these outcomes. For prediabetes, it would enable understanding of mechanisms at a stage where intervention to prevent the development of T2D would be most pertinent. In the analysis of lung function, a moderating effect was found between childhood SEP

and individual level exposures. This analysis could be extended to explore whether the increased susceptibility found with individual level exposures exists for area level exposures too.

## 5.5 Conclusion

This thesis has contributed to the knowledge on health inequalities through investigating social patterning in biomarkers. It has enhanced the understanding of how SEP affects physiology through testing mediating pathways between SEP and the health outcomes explored, in some places finding evidence of a mechanism through which SEP is associated with the outcome. Identification of these mechanisms is important to enable the alleviation of health inequalities through modifying socially patterned behaviours, exposures and stressors. The persistence of the SEP differential in health outcomes, even when mediating pathways have been taken into account, points to the need to address social inequalities directly. The thesis examined when in life exposure to disadvantaged SEP was most consequential for the health outcomes explored. This aids our understanding of when in life people are most susceptible to the effects of disadvantaged SEP, enabling identification of when policy intervention may be most effective to reduce health inequalities. The thesis found support for different theories of the life course depending on the outcome explored. The variation in support for different theories of health inequalities and the life course found here convey how multiple pathways and mechanisms operate to affect different aspects of health.

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# Appendix A Comparison of quadratic and fractional polynomial models

Using a fractional polynomial indicated that age to the power of -1 and age to the power of two was the most appropriate approach for men and using age to the power -0.5 and linear age was the best approach for women. As this required reducing the simplicity of the models and making the intercepts produced more difficult to interpret, AIC was used to examine whether using the functions of age suggested by the fractional polynomial was the best approach. A smaller AIC indicates a better fitting model. In each case the model suggested by the fractional polynomial had a marginally smaller AIC. In order to compare the AICs; an approach suggested by Burnham & Anderson (2002) was used to test the probability that the model with a smaller AIC was significantly better. This involved producing a p value.[17] For both men and women, the terms produced by the fractional polynomial models were a better fit. This is shown in Table 36.

Table 36 Comparison between quadratic and fractional polynomial models for men and women

| | | Men | | Women |
|---|---|---|---|---|
| **Men** | Age & age^2 | Age^-1 & age^2 | Age & age^2 | Age^0.5 & age^1 |
| **AIC** | 46596.46 | 46534.48 | 53381.17 | 53332.42 |
| **P** | p<.001 | | p<.001 | |

[17] $p$=exp($AIC_{fp}$-$AIC_{quad}$)/2

# Appendix B Models producing highest grip strength

These models were produced by using age terms centred to the age of highest grip strength specific to whether SEP was disadvantaged or advantaged to obtain an estimate for grip strength at that age. The results for men are shown on Table 37 and for women are on Table 38.

Table 37 Age regressed on grip strength with interactions between age terms and SEP for men

| Men | N | R2 | Coefficient | Lower 95% CI | Higher 95% CI |
|---|---|---|---|---|---|
| **Childhood SEP** | 6,677 | 0.9587 | | | |
| Advantaged | | | 48.025*** | 47.544 | 48.506 |
| Disadvantaged | | | 46.825*** | 46.237 | 47.413 |
| Age^-1 | | | -453.338*** | -529.507 | -377.168 |
| Age^2 | | | -0.004*** | -0.005 | -0.004 |
| Age ^-1* Disadvantaged childhood | | | 21.279 | -68.125 | 110.682 |
| Age ^2 * Disadvantaged childhood SEP | | | 1.082e-6 | -0.001 | 0.001 |
| **Adult SEP** | | | | | |
| **Education** | 7,799 | 0.9582 | | | |
| High education | | | 47.236*** | 46.790 | 47.681 |
| Low education | | | 48.025*** | 47.513 | 48.537 |
| Age^-1 | | | -432.048*** | -481.159 | -382.938 |
| Age^2 | | | -0.004*** | -0.004 | -0.004 |
| Age^-1 * low education | | | 15.803 | -52.782 | 84.388 |
| Age^2 * low education | | | -0.001* | -0.001 | 9.46e-4 |
| **Income** | 7,811 | 0.9581 | | | |
| High income | | | 47.910*** | 47.546 | 48.274 |
| Low income | | | 45.850*** | 44.984 | 46.717 |
| Age^-1 | | | -405.474*** | -481.464 | -329.485 |
| Age^2 | | | -0.004*** | -0.005 | -0.004 |
| Age^-1 * low income | | | 24.421 | -108.596 | 59.755 |
| Age^2 * low income | | | 3.46e-3 | 1.29e-3 | 8.21e-3 |

*p<.05, **p<.005, ***p<.001

Table 38 Age regressed on grip strength with interactions between age terms and SEP for women

| Women | N | R2 | Coefficient | Lower 95% CI | Higher 95% CI |
|---|---|---|---|---|---|
| **Childhood SEP** | 8,576 | 0.9502 | | | |
| Advantaged childhood SEP | | | 30.190*** | 29.930 | 30.451 |
| Disadvantaged childhood SEP | | | 29.270*** | 28.866 | 29.673 |
| Age | | | -1.159*** | -1.269 | -1.049 |
| Age^.5 | | | 13.773*** | 12.336 | 15.210 |
| Age * Disadvantaged childhood SEP | | | 0.101 | -0.062 | 0.263 |
| Age^.5 * Disadvantaged childhood SEP | | | -1.568 | -3.816 | 0.679 |
| **Adult SEP** | | | | | |
| **Education** | 9,824 | 0.9494 | | | |
| High education | | | 30.081*** | 29.832 | 30.331 |
| Low education | | | 29.408*** | 29.101 | 29.715 |
| Age | | | -1.102*** | -1.193 | -1.010 |
| Age^.5 | | | 13.076*** | 11.591 | 14.561 |
| Age * low education | | | -0.397 | -2.351 | 1.557 |
| Age ^.5 * low education | | | -0.003 | -0.146 | 0.141 |
| **Income** | 9,848 | 0.9490 | | | |
| High income | | | 29.872*** | 29.658 | 30.085 |
| Low income | | | 29.520*** | 29.068 | 29.972 |
| Age linear | | | -1.163*** | -1.242 | -1.083 |
| Age^.5 | | | 11.943*** | 10.031 | 13.855 |
| Age * low income | | | 0.109 | -0.050 | 0.269 |
| Age ^.5 * low income | | | -1.707 | -3.901 | 0.488 |

*p<.05, **p<.005, ***p<.001

# Appendix C Life course tests for men aged less than 37 and women aged less than 35

Table 39 below shows the number and percentage of men aged between 23 and 36 years and women aged between 23 and 33 years in each possible life course trajectory as well as the mean grip strength for those in that trajectory. The number of observations in these age categories is quite small and some of the cell sizes below are too small for reliable analysis. The mode life course trajectory for men and women in these age groups is 'stable high' SEP. The 'early upward' social mobility trajectory is the least common.

Table 39 Frequency and percentage of men aged less than 37 years and women aged less than 34 years in each possible life course trajectory and their mean grip strength

| | Childhood | Early adulthood | Adulthood | Men: 23-36 n=1,011 | Mean (kg) | SD | Women: 23-34 n=1,200 | Mean (kg) | SD |
|---|---|---|---|---|---|---|---|---|---|
| **Critical period** | | | | | | | | | |
| Disadvantaged in childhood | 1 | - | - | 193 (19.1%) | 45.6 | 9.4 | 260 (21.7%) | 28.9 | 6.2 |
| Disadvantaged in early adulthood | - | 1 | - | 406 (40.1%) | 48.0 | 9.8 | 409 (34.1%) | 29.2 | 6.4 |
| Disadvantaged in adulthood | - | - | 1 | 166 (16.4%) | 44.7 | 10.2 | 258 (21.5%) | 29.6 | 6.3 |
| **Social mobility** | | | | | | | | | |
| Stable advantaged SEP | 0 | 0 | 0 | 456 (45.0%) | 46.2 | 9.4 | 580 (48.4%) | 29.0 | 5.7 |
| Early upward mobility | 1 | 0 | 0 | 70 (7.0%) | 45.4 | 8.6 | 89 (7.5%) | 28.7 | 5.3 |
| Disadvantaged in early adulthood only | 0 | 1 | 0 | 245 (24.2%) | 48.6 | 9.9 | 199 (16.6%) | 29.4 | 6.4 |
| Late downward mobility | 0 | 0 | 1 | 66 (6.6%) | 42.8 | 10.2 | 94 (7.8%) | 29.6 | 6.1 |
| Late upward mobility | 1 | 1 | 0 | 75 (7.4%) | 47.1 | 9.2 | 72 (6.0%) | 28.1 | 6.6 |
| Advantaged in early adulthood only | 1 | 0 | 1 | 13 (1.4%) | 39.5 | 6.8 | 26 (2.2%) | 29.9 | 8.1 |
| Early downward mobility | 0 | 1 | 1 | 51 (5.1%) | 48.1 | 9.3 | 64 (5.4%) | 29.2 | 6.1 |
| **Accumulation** | | | | | | | | | |
| Stable disadvantaged | 1 | 1 | 1 | 33 (3.3%) | 45.3 | 11.4 | 72 (6.1%) | 29.7 | 6.2 |

Table 40 below shows the results of likelihood ratio tests for each of the models for men and women between the age of 23 and 39 and 23 and 33 respectively. For men, the null model performed significantly differently to the saturated indicating that SEP did add to the fit of the models. The accumulation model had the highest p-value and low education was associated with higher grip strength while low income was associated with lower grip strength. For women, the null model also produced a fit as good as the saturated model indicating that the addition of the SEP measures did not improve the fit and that SEP did not add to the explanation of variation in grip strength.

Table 40 Coefficients (95% confidence intervals) and p values from likelihood ratio tests for each model of the life course tested with a saturated model for men aged 23 to 36 and women aged 23 to 33

| | | | | **Men aged 23 to 36 n=1,015** | | **Women aged 23 to 33 n=1,200** | |
|---|---|---|---|---|---|---|---|
| | Childhood | Early adulthood | Adulthood | Likelihood ratio test | _b (CI 95%) | Likelihood ratio test | _b (CI 95%) |
| **Critical period** | | | | | | | |
| Childhood | 1 | - | - | .000 | -1.093 (-2.604to 0.416) | .493 | -0335 (-1.161to 0.490) |
| Early adulthood | - | 1 | - | .021 | 2.487*** (1.287 to 3.687) | .442 | 0.168 (-0.547 to 0.883) |
| Adulthood | - | - | 1 | .000 | -1.916* (-3.530 to 0.303) | .750 | 0.677 (-0.149to 1.502) |
| **Social mobility** | | | | | | | |
| Early upward mobility | 1 | 0 | 0 | .000 | -1.513 (-3.841 to 0.815) | .502 | -0.627 (-1.921 to 0.667) |
| Low in early adulthood only | 0 | 1 | 0 | .009 | 2.648*** (1.127 to 4.024) | .492 | 0.370 (-0.542 to 1.281) |
| Late downward mobility | 0 | 0 | 1 | .000 | -3.421* (-5.828 to -1.014) | .548 | 0.666 (-0.594 to 1.927) |
| Late upward mobility | 1 | 1 | 0 | .000 | 0.948 (-1.318 to 3.214) | .724 | -1.121 (-2.545 to 0.302) |
| High in early adulthood only | 1 | 0 | 1 | .000 | -7.411** (-12.510 to -2.311) | .461 | 0.723 (-1.594to 3.040) |
| Early downward mobility | 0 | 1 | 1 | .000 | 1.729 (-0.962to 4.420) | .437 | 0.311 (-1.195 to 1.816) |
| **Accumulation (relaxed)** | | | | .468 | | .747 | |
| Childhood | 1 | | | | -1.463 (-2.980 to 0.055) | | -0.758 (-1.401 to 0.325) |
| Early adulthood | | 1 | | | 2.839*** (1.624 to 4.054) | | 0.141 (-0.606 to 0.888) |
| Adulthood | | | 1 | | -2.147* (-3.758 to -0.535) | | 0.758 (-0.100 to 1.616) |
| **Accumulation** | | | | | | | |
| Accumulation (Strict) | 1 | 1 | 1 | .000 | 0.262 (-0.470 to 0.994) | .714 | 0.117 (-0.259 to 0.493) |
| **Null** | | | | .000 | | .534 | |

* p<.05, ** p<.005, ***p<.000

# Appendix D Using food expenditure as a measure of food insecurity

In order to justify using food expenditure as an indicator of food insecurity; it was necessary to ensure that the measure captured a dimension of material deprivation specific to food and distinguishable from low income or other forms of deprivation. In order to do this, a number of ways of measuring food insecurity were considered. The available measures in the BHPS pertained to food expenditure and two food deprivation items.

### *Food expenditure and deprivation*

There are two deprivation measure related to food asked in BHPS from waves six onwards; whether respondents can afford to eat meat every second day and whether respondents can afford to provide a meal for visitors once a month. Across waves, these are reported to be unaffordable by 1.8% and 4.7% of respondents respectively. Each of these measures were considered problematic as a measure of food insecurity conceptually as affordability of meat every second day would not be relevant to vegetarians and providing a meal for visitors could be considered more relevant as a social participation measure. However, these were used to help validate the food expenditure approach.

Table 41 shows mean spending for respondents reporting deprivation and those not reporting deprivation for each item in the index of deprivation. Mean expenditure on groceries is lowest for those reporting being unable to afford meat every second day followed by those unable to provide a meal to visitors once a month. This suggests that spending on groceries is associated with food deprivation more specifically that other forms of deprivation. For some of the deprivation items such as replacing old furniture the difference in grocery spending between those reporting that form of deprivation and not is negligible and those reporting being unable to afford an annual holiday have a slightly higher mean spend on groceries.

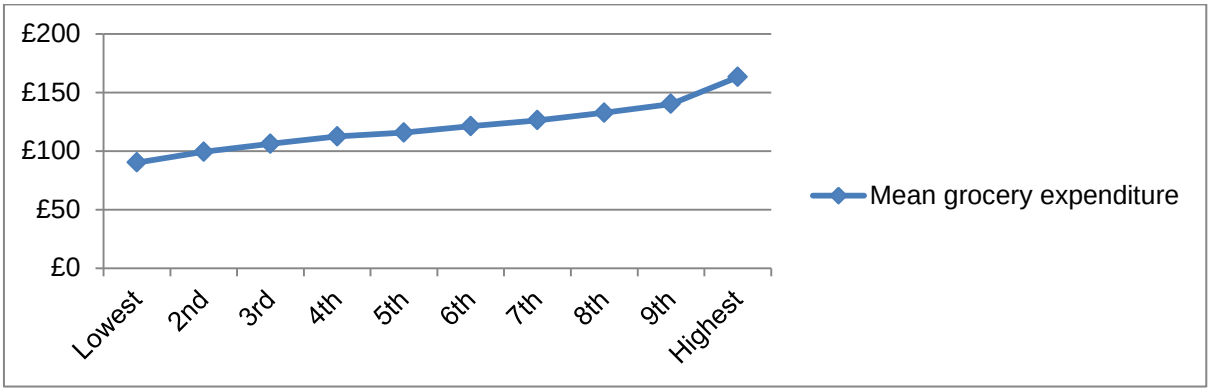
Table 41 Mean expenditure on groceries by each deprivation measure

| | Meat | Social meal | Warm home | Annual holiday | Replace furniture | New clothes | Two pairs shoes | Decorate home | Contents insurance |
|---|---|---|---|---|---|---|---|---|---|
| **No deprivation** | £126.19 | £126.88 | £125.66 | £125.13 | £125.89 | £125.97 | £137.75 | £137.76 | £138.24 |
| **Reported deprivation** | £92.95 | £100.37 | £112.56 | £129.76 | £121.88 | £113.30 | £124.18 | £115.17 | £126.07 |

### *Food expenditure and income*

The association between grocery expenditure and income was considered; it was expected that there would be some association between them. Income and grocery expenditure were significantly correlated with one another; with a correlation coefficient of 0.3. This suggests a weak relationship and also indicating the two are associated but still capture different concepts. Figure 19 shows mean grocery expenditure by income decile.

Figure 19 Mean grocery expenditure by income decile



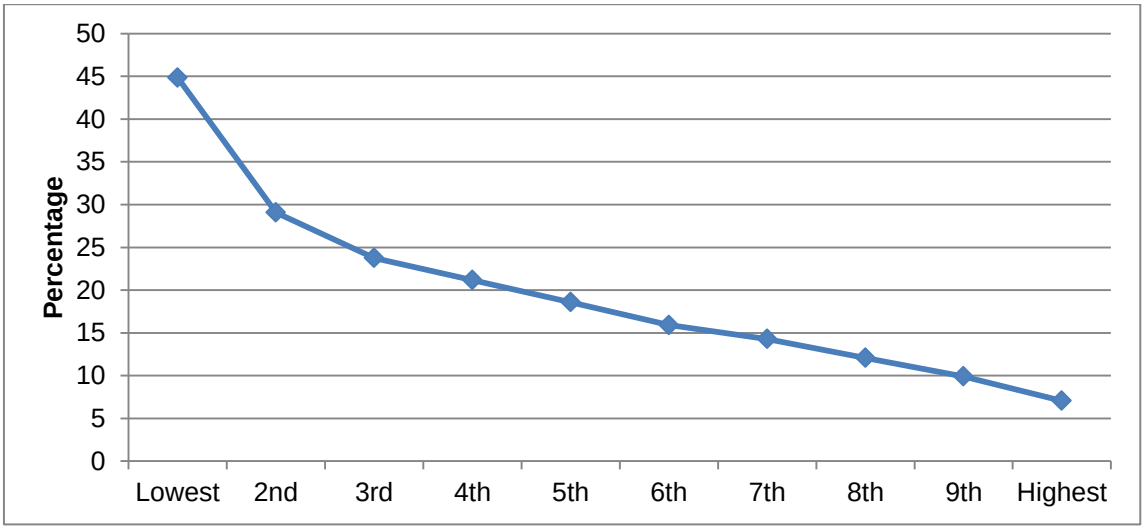

### *Operationalisation of food expenditure*

Having established that grocery expenditure was not collinear with income and that low grocery expenditure was more closely related to food deprivation that other deprivation items; different ways of using grocery expenditure as food insecurity measures were examined. These related to using a cut off on the basis of the typical cost of a healthy basket of food, using a cut off for the proportion of income which grocery expenditure accounted for and using a cut off of relatively low expenditure on groceries.

The suggested cost of an adequate basket of food used for the minimum income standard for Britain was first developed by the Joseph Rowntree Foundation in 2008. This was considered as an indicator of minimum expenditure on food with adjustment for inflation so it could be used as a cut off for previous waves of BHPS. However, this measure was considered to be not sufficiently discriminating in identifying people experiencing food insecurity. The minimum suggested cost of food for a single person in 2008 was £40.34; the median spend in the BHPS this year for a single person household was £39; this would require considering approximately half the sample as having insufficient expenditure on groceries.

Spending a large proportion of one's income on food is considered in some literature (Friel et al., 2004) to be indicative of food insecurity. Figure 20 shows the proportion of income spend on groceries for each income decile; the proportion of income spent in highest in the lowest income decile and lowest in the highest income decile. The mean proportion of income spent on groceries was 15.5% (SD 11) and the median was 12.6%. Proportion of income spent on groceries was also correlated with income with a correlation coefficient of -.5 suggesting a moderate level of correlation. It was decided not to use proportion of income spent on groceries as it was highly correlated with income and conceptually for low earners choosing the spend a large proportion of income on groceries, it could be considered avoidance of food insecurity while potentially experiencing other forms of deprivation. Mean spend on groceries for those who spent 15% of their income or more of groceries was greater than the sample mean.

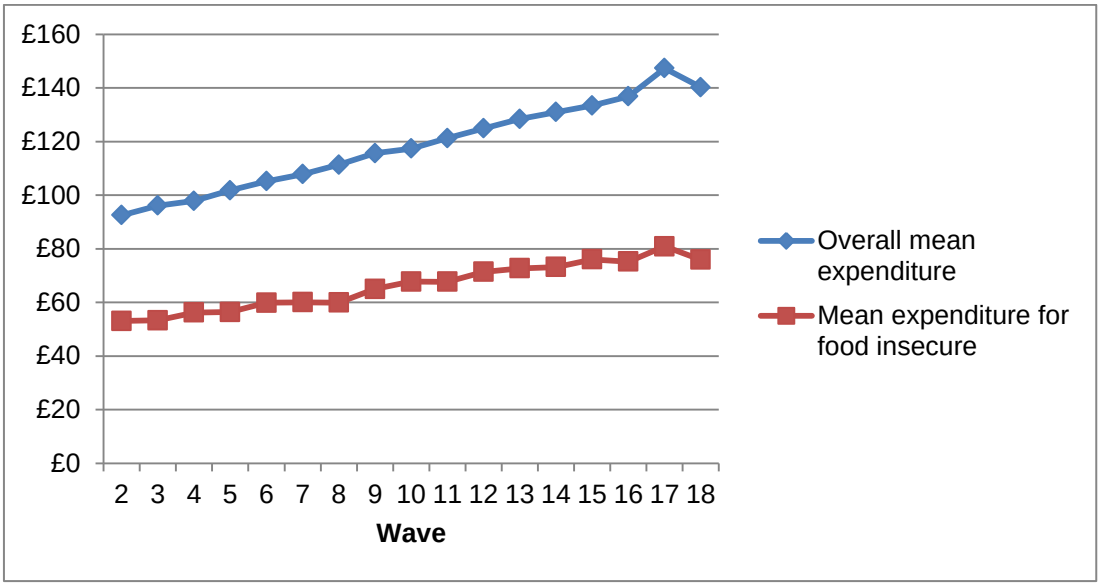
Figure 20 Percentage of income spent on groceries by income decile (in wave 18 of BHPS)




The next approach considered was low expenditure on groceries. The equivalised monthly expenditure on groceries was transformed into deciles. The prevalence of reported deprivation on the food items as well as the other forms of deprivation was examined for each decile. The reported deprivation of not being able to afford meat every second day and not being to afford a meal for visitors once a month was higher than in the first three deciles of grocery spending than the overall sample prevalence. Reported deprivation on the other items was higher in the lower grocery expenditure deciles though the gradient was not as pronounced as for the food

deprivation items. For this reason, it was decided to use being in the bottom three deciles of food expenditure as a measure of food insecurity. Figure 21 below shows the mean expenditure for whole sample for each wave and the mean expenditure for food insecure for each wave.[18]

Figure 21 Mean grocery expenditure for BHPS sample and for food insecure across waves




### *Limitations of using food expenditure to measure food insecurity*

The measurement of food insecurity here is limited by being restricted to the measures available in the BHPS. Ideally, measurement would capture dimensions of food insecurity such as affordability and accessibility, whether the diet achieved was adequate, safe and nutritious, and whether it was consistently affordable. In 1995, the United States Department of Agriculture developed an 18 item scale to measure household food insecurity. Items such as being worried about food running out, not being able to afford balanced meals, and going hungry. These items were selected from 30 items which were trialled. Ideally, developing a measure of food insecurity would require an approach such as this to ensure it was appropriate to capturing food insecurity in the British context.

The measure created here does not capture whether respondents are unable to access food or issues which may restrict food security such storage or ability to

[18] The Living Costs and Food Survey found that in 2009 (i.e. wave 18 for BHPS); household expenditure fell for the first time since the introduction of its current system in 2001.

prepare food. The nutritional adequacy of the food obtained is not assessed. It is not possible to ascertain if there have been inequalities in the sharing of household resources, previous research has found that children's food intake is prioritised by women in low income households who restrict their own diet to provide more for them (Coakley, 2001).

# Appendix E Verification of associations for mediation analysis

This section first confirms that childhood SEP is associated with lung function, then that childhood SEP is associated with each mediator and finally whether each mediator is associated with lung function. Each table shows the coefficient or odds ratios as appropriate, 95% confidence intervals, number of observations and r-squared or pseudo r-squared.

## *Is childhood SEP associated with lung function?*

Table 42 shows the results of disadvantaged childhood SEP being regressed on $FEV_1$%, those reporting disadvantaged childhood SEP had a $FEV_1$% approximately 4.3% (-5.016% to -3.714%) lower than those reporting advantaged childhood SEP.

Table 42 Direct effect of childhood SEP on predicted $FEV_1$%

| | _b | 95% CI | N | R2 |
|---|---|---|---|---|
| **Childhood SEP** | | | 12,330 | .011 |
| Disadvantaged childhood SEP | -4.307 *** | (-5.016 to -3.714) | | |
| Cons | 94.609 *** | (94.199 to 95.019) | | |

*p<.05, **p<.005, ***p<.001

## *Is childhood SEP and associated with each mediator?*

In this section, results from logistic regressions with disadvantaged childhood SEP as predictor of each mediator are presented. Table 43 below shows that childhood SEP is significantly associated with adult SEP.

Table 43 Direct effect of childhood SEP on adult SEP

| | 0R | 95% CI | N | P-R2 |
|---|---|---|---|---|
| **Childhood SEP** | | | 13,310 | .074 |
| Disadvantaged childhood SEP | 3.723 *** | (3.440 to 4.030) | | |
| Cons | 0.415 *** | (0.392 to 0.441) | | |

***p<.05, **p<.005, ***p<.001**

Table 44 shows that disadvantaged childhood SEP was negatively associated with being a current smoker but positively associated with being a former smoker. This may be due to the greater prevalence of disadvantaged SEP among older age groups who are less likely to be smokers and more likely to be former smokers.

Table 44 Direct effect of childhood SEP on being a smoker and a former smoker

| | OR | 95% CI | N | P-R2 |
|---|---|---|---|---|
| **Smoker** | | | 13,333 | .001 |
| **Childhood SEP** | | | | |
| Disadvantaged childhood SEP | 0.868 ** | (0787 to 0.957) | | |
| Cons | 0.260 | (-0.243 to 0.279) | | |
| **Former smoker** | **OR** | **95% CI** | N | P-R2 |
| **Childhood SEP** | | | 13,333 | .001 |
| Disadvantaged childhood SEP | 1.121 ** | (1.040 to 1.210) | | |
| Cons | 0.611 | (0.589 to 0.646) | | |

*p<.05, **p<.005, ***p<.001

Table 45 below shows that having disadvantaged childhood SEP was associated with being circa twice as likely to report low physical activity in adulthood.

Table 45 Direct effect of childhood SEP on low physical activity

| | OR | 95% CI | N | P-R2 |
|---|---|---|---|---|
| **Childhood SEP** | | | 13,250 | .026 |
| Disadvantaged childhood SEP | 2.169 *** | (2.004 to 2.347) | | |
| Cons | 0.437 *** | (0.411 to 0.463) | | |

*p<.05, **p<.005, ***p<.001

Table 46 shows that rates of obesity were higher for those with disadvantaged SEP in childhood; 1.788 (1.657 to 1.930).

Table 46 Direct effect of childhood SEP on obesity

| | OR | 95% CI | N | P-R2 |
|---|---|---|---|---|
| **Childhood SEP** | | | 13,092 | .015 |
| Disadvantaged childhood SEP | 1.788 *** | (1.657 to 1.930). | | |
| Cons | 0.602 *** | (0.569 to 0.636) | | |

*p<.05, **p<.005, ***p<.001

Table 47 shows that there was no association between disadvantaged SEP in childhood and diet.

Table 47 Direct effect of childhood SEP on diet

| | OR | 95% CI | N | P-R2 |
|---|---|---|---|---|
| **Childhood SEP** | | | 13,092 | .015 |
| Disadvantaged childhood SEP | 0.935 | (0.868 to 1.007) | | |
| Cons | 1.082 *** | (1.025 to 1.142) | | |

*p<.05, **p<.005, ***p<.001

Table 48 shows that disadvantaged SEP in childhood was positively associated with being reporting occupational exposures in one's occupation.

Table 48 Direct effect of childhood SEP on occupational exposures

| | OR | 95% CI | N | P-R2 |
|---|---|---|---|---|
| **Childhood SEP** | | | 12,114 | .009 |
| Disadvantaged childhood SEP | 1.581 *** | (1.460 to 1713.) | | |
| Cons | 0.573 *** | (0.540 to 0.607) | | |

*p<.05, **p<.005, ***p<.001

### *Is each mediator associated with lung function?*

Adult SEP, each health behaviour and exposure were regressed on $FEV_1$% to establish if the associations between these described in the literature were also found here. The results of these are shown in Table 49. Being a current smoker was associated with having a $FEV_1$% approximately 4.5% (5.152% to 3.915%) lower than people who never smoked. While those who smoked in the past had a $FEV_1$% between 1.878% and 0.526% lower than those who never smoked. Not participating in mild or moderate sports at least weekly was associated with having $FEV_1$% 5.411% to (6.063% to 4.759%) lower than those who exercised more than this. Being obese was also significantly associated with lower $FEV_1$%, those with a very high waist circumference had $FEV_1$% close to 5% lower (5.399% to 4.170%) than those with a healthy or high waist circumference. Not eating fruit daily was associated with an $FEV_1$% measurement between 2.282% and 1.060% lower than those eating fruit daily. Those whose current or last occupation had exposures to hazards for lung function as measured by the JEM had a $FEV_1$% between 3.288% to 1.995% lower.

Table 49 Direct effects of mediators on $FEV_1$%

| | _b | 95% CI | N | R2 |
|---|---|---|---|---|
| **Adult SEP (ref. advantaged adult SEP)** | | | 13,126 | .029 |
| Disadvantaged adult SEP | -4.534 *** | (-5.152 to -3.915) | | |
| Cons | 93.998 *** | (93.621 to 94.374) | | |
| | | | | |
| **Smoking (ref. never smoked)** | | | 13,150 | .020 |
| Smoker | -6.154 *** | (-6.991 to -5.3186) | | |
| Former smoker | -1.202 | (-1.878 to -0.526) | | |
| Cons | 93.702 *** | (93.235 to 94.170) | | |
| | | | | |
| **Physical activity (ref. high physical activity)** | | | 13,073 | .026 |
| Low physical activity | -5.411 *** | (-6.063 to -4.759) | | |
| Cons | 94.085 *** | (93.736 to 94.435) | | |
| | | | | |
| **Obesity (ref. non-obese)** | | | 13,054 | .021 |
| Obese | -4.784 *** | (-5.399 to -4.170) | | |
| Cons | 94.093 *** | (93.702 to 94.484) | | |
| | | | | |
| **Diet (ref. healthy diet)** | | | 13,148 | .026 |
| Poor diet | -1.671 *** | (-2.282 to -1.060) | | |
| Cons | 92.852 *** | (92.411 to 93.293) | | |
| | | | | |
| **Occupation (ref.no occupation exposures)** | | | 12,005 | .016 |
| Occupational exposure | -2.642 ** | (-3.288 to -1.995) | | |
| Cons | 93.300 *** | (92.918 to 93.682) | | |

*p<.05, **p<.005, ***p<.001

# Appendix F Does the moderating effect of childhood SEP on health behaviours and exposures differ on the basis of age?

Table 50 shows whether childhood SEP moderated the effects of health behaviour and exposures by age. Interaction effects were mainly insignificant aside from disadvantaged childhood SEP by smokers in the midlife and the oldest age group. The numbers in each group may be too small for precise estimation and comparison.

Table 50 Interaction between disadvantaged childhood SEP and health behaviours and exposures for each age group

| | **Aged 20 to 39 years** | | **Aged 40 to 64 years** | | **Aged 65 years and older** | |
|---|---|---|---|---|---|---|
| **N and R2** | n=2,491, R2=.0266 | | n=5,349, R2=.0856 | | n=2,200, R2=.1100 | |
| | _b | 95% CI | _b | 95% CI | _b | 95% CI |
| **Main effects** | | | | | | |
| **Childhood SEP** | | | | | | |
| Disadvantaged childhood SEP | -1.840 | (-4.666 to 0.986) | -0.303 | (-2.552 to 1.494) | -1.548 | (-6.235 to 3.140) |
| **Adult SEP** | | | | | | |
| Disadvantaged adult SEP | -0.679 | (-1.926 to 0.568) | -1.620 ** | (-2.100 to -0.687) | -1.647 | (-3.553 to 0.258) |
| **Health behaviours** | | | | | | |
| Smoker | 0.016 | (-1.491 to 1.524) | -5.893 *** | (-7.618 to -4.168) | -13.041 *** | (-19.174 to -6.908) |
| Former smoker | 1.070 | (-0.307 to 2.447) | 0.076 | (-1.265 to 1.113) | -0.305 | (-3.463 to 4.073) |
| Low physical activity | -1.911 ** | (-3.217 to -0.606) | -2.587 *** | (-3.937 to -1.237) | -6.798 ** | (-10.925 to -2.670) |
| Poor diet | -1.049 | (-2.277 to 0.178) | -0.013 | (-1.154 to 1.129) | -0.885 | (-4.855 to 3.086) |
| **Obesity** | | | | | | |
| Obese | -1.818 * | (-3.132 to -0.504) | -4.250 *** | (-5.382 to -3.117) | -6.201 ** | (-10.015 to -2.387) |
| **Occupation** | | | | | | |
| Occupational exposures | 0.339 | (-0.921 to 1.599) | -0.264 | (-1.462 to 0.985) | -3.895 | (-7.831 to 0.41) |
| **Interactions** | | | | | | |
| Smoker * disadvantaged childhood SEP | -0.414 | (-3.811 to 2.983) | -3.545 * | (-6.085 to -1.005) | -3.682 * | (-10.918 to 3.554) |
| Former smoker * disadvantaged childhood SEP | 2.320 | (-0.719 to 5.359) | -0.937 | (-2.797 to 0.923) | -4.848* | (-9.233 to -0.463) |
| Low physical activity * disadvantaged childhood SEP | 0.278 | (-2.556 to 3.110) | 0.931 | (-1.016 to 2.878) | -0.418 | (-5.072 to 4.237) |
| Poor diet * disadvantaged childhood SEP | 0.818 | (-1.779 to 3.415) | -0.787 | (-2.559 to 0.985) | 0.885 | (-4.855 to 6.031) |
| Central obesity * disadvantaged childhood SEP | -1.069 | (-4.129 to 1.991) | 0.396 | (-1.345 to 2.136) | 2.289 | (-2.053 to 6.625) |
| Exposures* disadvantaged childhood SEP | -1.778 | (-4.502 to 0.947) | -1.235 | (-3.011 to 0.542) | 1.836 | (-2.631 to 6.302) |
| Cons | 96.136 *** | (94.778 to 97.495) | 98.497 *** | (97.383 to 99.611) | 101.211 *** | (97.061 to 105.361) |

*p<.05, **p<.005, ***p<.001

# Appendix G Sensitivity checks

### *Chapter two: sensitivity checks*

Due to some data (on income, health behaviours and occupation) being collected one wave prior to the NHA for the BHPS sample and in the same wave for the UKHLS sample, some sensitivity checks were conducted. First, analysis was reran including a control variable based on whether respondents were part of the BHPS or UKHLS samples. This is not result in any change to the results. A further check was undertaken by running the analysis separately for the BHPS and UKHLS subsamples. The results for the UKHLS subsample were broadly the same as those found for the whole sample. The results found for the BHPS subsample were broadly similar but associations were slightly weaker though this may be as a result of losing power through having less observations.

### *Chapter three: sensitivity checks*

The analysis on T2D was reran on the sample for whom $HbA_{1c}$ was measured. The association between SEP and T2D attenuated completely with the addition of demographic covariates. Thus comparing the decomposition of T2D and $HbA_{1c}$ in the same analytical sample was not pursued beyond this. The loss of power due to the reduced sample size may have been a factor in this, however the finding does not support the comparability of the T2D and $HbA_{1c}$ analytical samples. Analyses were rerun without the use of the dummy measure, which retained 1,318 cases who dropped out of the BHPS prior to the collection of maternal education, ethnicity and obesity. This made no difference to the analytical sample for $HbA_{1c}$ as these cases would not have been present at this stage of the survey. For the analysis of T2D, no substantial difference to findings was observed. The analysis of $HbA_{1c}$ with adjustment for long term liver disease, taking aspirin, taking anti-inflammatories and for triglycerides produced associations consistent with the unadjusted analysis.

### *Chapter four: sensitivity checks*

Due to the BHPS sample participating in the NHA in wave three and having had data on health behaviours and occupation collected in wave two, sensitivity checks were conducted to see if this affected associations. Analysis was reran including a control variable based on whether respondents were part of the BHPS or UKHLS samples. This is not result in any change to the results. The analysis was also ran separately for the BHPS and UKHLS subsamples. The results for the UKHLS subsample were broadly the same as those found overall. The results found for the BHPS subsample

were broadly similar; some associations had attenuated though this may be due to the reduction in the number of observations.

The analysis on lung function treats it as a linear measure, analyses were rerun using a binary outcome which classified those with a $FEV_1$% below 80% as having low $FEV_1$%. Below 80% of $FEV_1$% was used as it indicates below normal lung function and can indicate COPD. The associations found were broadly in line with those found in the linear models. However, no significant association was found with the job exposure matrix suggesting that only slightly lower $FEV_1$% was associated with this measure. Another difference was that in the younger age group, none of the socio-economic, health behaviour, obesity or occupational exposures were significant.

As poor health and particularly poor lung function may affect the ability to take part in lung function measurement, a further sensitivity test was ran using a binary measure of those with low lung function incorporating those who did not provide a lung function measure. The associations found were supportive of the findings from the linear models.

Lastly, the analysis was split for those who achieved a ‘Grade A’ quality lung function measure and those with any level of quality below those. Results were not substantively different throughout except for the occupational exposures measure where those with a Grade A measurement had a stronger association with occupational exposure and those with a level of quality below this did not have a significant association with occupational exposures.