

# Life Course Social Inequalities in Body Composition: Understanding the Role of Child Behavioural and Emotional Problems and the Environment

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# Declaration

I, Charis Bridger Staats confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

# Acknowledgments

*Dedicated to my sister Tilly – this year has been challenging in ways no one expected, and yet you have shown strength and resilience beyond your years. Thank you for being there for me despite it all.*

*I promise it will get easier.*

Firstly, I would like to start with a huge thank you to my supervisors, Rebecca Hardy, Yvonne Kelly and Rebecca Lacey, who without, this would not have been possible. Thank you for your continued support, guidance and supervision over the last 4 years, and for fostering a learning environment where I have felt comfortable to express and discuss ideas freely. I want to especially thank you for the last six months in the run-up to submitting, which have not been easy for anyone – I truly appreciate that you have all found the time to provide valuable feedback and to discuss my work, despite your own high workloads and challenges faced because of the ongoing pandemic.

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Given the life course approach adopted in this PhD, it would be difficult to skate over the important early life influences and support provided by family from a young age. No doubt the early engagement and genuine interest in my education from my family has impacted my approach to academia and learning throughout my life so far and influenced the direction my academic interests have taken.

In particular, my Mum, Vicki, who works as a GP Practice Manager has certainly influenced my interest in population health and has been passionate about righting the wrongs in the world since I can remember. Her interest in the health and inequalities that face the patients at her GP practice, an area in Bristol with some of the highest levels of deprivation, has undoubtedly influenced the direction my interests have taken in health inequalities. More importantly, my Mum has always been my biggest champion and often celebrated my achievements more than I have myself - thank you for always being there for me.

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# Abstract

Despite life course inequalities in body mass index (BMI) being well studied, less is known about inequalities in body composition. There is also a lack of understanding regarding the role psychological characteristics, and the environment in which people live, play in the development of inequalities.

Firstly, a systematic review was conducted to summarise the evidence on the association between socioeconomic position (SEP) and body composition. Secondly, data from the Millennium Cohort Study (MCS), a cohort of 19,517 children born in 2000/2, were used. Growth curve models were applied to investigate inequalities in trajectories of fat mass index (FMI), fat-free mass index (FFMI) and their ratio (FM:FFM). Structural equation modelling (SEM) was used to investigate mediators (internalising and externalising symptoms) of associations between SEP with body composition and BMI in adolescence, and to test moderation of mediating paths by environmental conditions (area-deprivation, green space density, fast-food and domestic garden density).

The systematic review found lower SEP related to higher fat mass (FM) and less consistent evidence of lower SEP related to lower fat-free mass (FFM) in high-income countries. Inequalities were more common among children compared to adults, and more common in females compared to males, except for inequalities in FFM in childhood. In the MCS those in more disadvantaged SEP in infancy had higher FMI and FM:FFM age 7, and inequalities widened to age 17.

Social inequalities in FMI and FM:FFM at age 17 were mediated by internalising symptoms, and for FFMI and BMI internalising and externalising symptoms jointly mediated associations. For FMI, FM:FFM and BMI – the mediation through internalising symptoms was greatest among those living in the least green and most deprived areas.

In order to improve childhood and adolescent body composition, early life disadvantage should be addressed and improvement of the environment in disadvantaged areas considered.

# Impact Statement

Obesity is a major public health priority that has been acknowledged globally since 1997. Similarly, mental health in children is acknowledged as another increasingly important public health challenge, with the World Health Organisation estimating that one in four individuals will suffer from mental health problems in their lifetime. Increases in both BMI and adverse mental health during the COVID-19 pandemic were reported across the UK population. In November 2021, the National Child Measurement Programme in England reported that obesity prevalence was 4% higher among children in primary schools during the pandemic (2020-21), than in the year preceding the pandemic (2019-20). Both increasing poverty and worsening mental health were highlighted as being likely important factors behind this observation.

The findings of this thesis highlight links between socioeconomic disadvantage, mental health and obesity that are potentially important for understanding the observed trends witnessed before, as well as during, the COVID-19 pandemic. It is possible that during the COVID-19 pandemic, the widening of inequalities demonstrated in this thesis, accelerated. This PhD therefore highlights an important need to address inequalities, in order to improve both mental health and body composition. The novel finding that green spaces and area deprivation level may moderate associations between mental health and body composition, have particular relevance in light of the national lock downs that have been imposed over 2020 and 2021 in the UK, where the environment in which people live has become increasingly important. In particular, the findings from this thesis highlight the need to improve environments in the most disadvantaged areas, which may have an important impact in reducing inequalities in body composition.

This thesis demonstrates that inequalities exist for body composition as well as BMI. In particular, although modest, evidence of inequalities in fat-free mass (FFM) were highlighted, and the changing fat mass (FM) to FFM ratio where inequalities widened into adolescence, have important implications for health across the life course. This thesis calls attention to the need to investigate FFM as well as FM in future research, and to explore how associations with disadvantage may vary with age and cohort. This is particularly important for life course research in ageing, where peaks in FFM in early adulthood are related to FFM in later life, and FFM in older ages is related cardio-metabolic disease and physical capability.

The findings in this thesis have been communicated to academic researchers and disseminated to a wider audience in different ways. Work has been presented at national and international conferences and therefore has contributed to academic discourse, especially around the importance of measuring body composition including FFM. Work from this PhD has also been published in well-respected peer-reviewed journals (four papers to date). Findings have been reported in the national media (e.g. Daily Mail, Evening Standard) and other local news outlets. As such the work from this PhD has had a wider reach than just an academic community and has made the public more aware of inequalities in body composition.

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# List of Abbreviations

<b><i>Abbreviation</i></b>	
<i>ALSPAC</i>	Avon Longitudinal Study of Parents and Children
<i>ASM</i>	Appendicular Skeletal Muscle
<i>ASM%</i>	Appendicular Skeletal Muscle Percentage
<i>ASMI</i>	Appendicular Skeletal Muscle Index
<i>BCS</i>	British Cohort Study
<i>BIA</i>	Bioelectrical Impedance Analysis
<i>BMI</i>	Body Mass Index
<i>CFA</i>	Confirmatory Factor Analysis
<i>CT</i>	Computed Tomography
<i>DXA</i>	Dual-Energy X-Ray Absorptiometry
<i>EFA</i>	Exploratory Factor Analysis
<i>FFM</i>	Fat-Free Mass
<i>FFM%</i>	Fat-Free Mass Percentage
<i>FFMI</i>	Fat-Free Mass Index
<i>FM</i>	Fat Mass
<i>FM%</i>	Fat Mass Percentage
<i>FM:FFM</i>	Fat Mass to Fat-Free Mass Ratio
<i>FMI</i>	Fat Mass Index
<i>HIC</i>	High-Income Country
<i>IMD</i>	Index of Multiple Deprivation
<i>LM</i>	Lean Mass
<i>LM%</i>	Lean Mass Percentage
<i>LMI</i>	Lean Mass Index
<i>LSOA</i>	Lower Layer Super Output Areas
<i>MCS</i>	Millennium Cohort Study
<i>MIC</i>	Middle-Income Country
<i>MRI</i>	Magnetic Resonance Imaging
<i>NCDS</i>	National Child Development Study
<i>NSHD</i>	National Survey of Health and Development
<i>NSSEC</i>	The National Statistics Socioeconomic classification
<i>NVQ</i>	National Vocational Qualification
<i>SEM</i>	Structural Equation Modelling
<i>SEP</i>	Socioeconomic Position
<i>WC</i>	Waist Circumference
<i>WHR</i>	Waist-To-Hip Ratio
<i>WHtR</i>	Waist-To-Height Ratio

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# Chapter 1 Introduction

This thesis aims to generate a greater understanding of life course social inequalities in body composition, in particular focusing on how inequalities in infancy and early life relate to body composition across adolescence. Social inequalities in health are defined as “systematic differences in health between different socioeconomic groups within a society” that are largely considered unacceptable due to their “socially produced” and “potentially avoidable” nature [1]. This thesis builds on previous work conducted in the British Birth Cohorts that have examined changing inequalities in body mass index (BMI) over time and demonstrated greater evidence of inequalities among those born more recently.

The British Birth Cohorts are a series of four nationally representative cohorts born 12 to 30 years apart. The first cohort is the Medical Research Council (MRC) National Survey of Health and Development (NSHD) made up of individuals born in one week in March in 1946. This is followed by the National Child Development Study (NCDS) made up of individuals born in a single week in March 1958 and the British Cohort Study (BCS) consisting of individuals born in a single week in April 1970. Finally, the Millennium Cohort Study (MCS), where participants were recruited at 9 months, comprises individuals born across a 16-month period starting from September 2000 to early 2002. These four cohorts are known collectively as the “British Birth Cohorts” and are referred to throughout this thesis.

This thesis aims to expand knowledge of inequalities in body size by using body composition rather than the more commonly studied BMI. This thesis uses the MCS to describe the extent of inequalities in body composition and to investigate how they develop in a contemporary cohort of UK adolescents. In particular, this thesis has been influenced by the secular changes observed in BMI inequalities across the British Birth Cohorts and has developed hypotheses relating to the importance of the environment in emerging inequalities. Similarly, this thesis has drawn on work in the British Birth Cohorts around trends in mental health and how they relate to BMI, and generated hypotheses around potential interactions and contributions of both environmental and psychological determinants.

This chapter introduces the research by describing contemporary trends in obesity and consequential costs of the high prevalence observed. The importance of the environment and mental health is also highlighted. Fat mass (FM) as a component of body composition is introduced as an alternative measure of obesity to BMI, along with fat-free mass (FFM) as a lesser studied component of body composition that captures lean tissue. Measurement issues and clinical significance of both are discussed. Finally, an outline of the thesis is provided.

## 1.1 Obesity and Health Costs

Obesity has been a significant public health challenge recognised globally since 1997 [2]. Obesity is a condition characterised by excess adiposity that increases risk of metabolic disease [3]. Excess adiposity, and as such obesity, is most frequently defined through BMI, a measure of weight (kg)

divided by height (m) squared. A BMI of 25kg/m<sup>2</sup> or above is typically classed as overweight, whilst a BMI of 30kg/m<sup>2</sup> is considered obese. Other anthropometric measures such as waist circumference (WC), waist-to-hip ratio (WHR), and waist-to-height ratio (WHtR) estimate the amount of adiposity stored centrally and, in the case of WHR and WHtR, indexed to other components of body size. However, FM may more accurately measure adiposity, which can be measured either indirectly through skinfold thickness or directly through body composition scanners, such as bioelectrical impedance analysis (BIA) or dual-energy X-ray absorptiometry (DXA).

Obesity defined by BMI and other anthropometric measures (WC and WHR) is associated with increased risk of mortality and morbidity, cardiovascular disease, diabetes and cancer [4], decreased mobility [5], reduced responses to medicines [4], and decreases in mental health and wellbeing [6-8]. Obesity in childhood is associated with adverse consequences across the life course and is a predictor of obesity, measured by BMI, in adulthood [9]. The societal costs of obesity are high. In England, over 10,000 hospital admissions were directly attributable to obesity in 2017/18 [10], and the estimated cost to the UK National Health Service (NHS) between 2014 and 2015 was £6.1 billion [11].

Considerable research and policy efforts have focused on tackling obesity in the UK, as seen in the landmark 2007 Foresight Report [12]. There has been a particular focus on addressing childhood obesity, as demonstrated by the 2016 “Childhood obesity Plan: a Plan for action”, which resulted in the 2018 Soft Drinks Levy being introduced [13]. In 2021, the Parliamentary Office of Science and Technology (POST) produced a POSTnote on the current state of childhood obesity and the policies that could be adopted to address obesity in childhood [14]. This is in recognition of the fact that adolescence and childhood are key points in the life course where obesity develops. In 2019-20, 21% of children leaving primary school in the UK were estimated to be obese, and 35% were either overweight or obese [14], with tracking of weight being demonstrated from childhood to adolescence. Over this time, behaviours that are important for obesity development also emerge, meaning childhood and adolescence may be an important point in the life course to intervene [15].

### 1.1.1 Trends Over Time

Surges in overweight and obesity were observed worldwide in the 1980s [16]. Review of global trends in BMI using pooled data from 1,698 population-based studies found global age-standardised mean BMI increased from 1975 to 2014, rising from 21.7 kg/m<sup>2</sup> in men and 22.1 kg/m<sup>2</sup> in women to 24.2 kg/m<sup>2</sup> and 24.4 kg/m<sup>2</sup>, respectively [17]. The prevalence of obesity in 2014 was over three times that reported in 1975 for men, and over twice the prevalence for women [17]. In children, pooled analysis of 2,416 population-based measurement studies found that global age-standardised obesity had risen from 0.7% in girls and 0.9% in boys in 1975 to 5.6% and 7.8% in 2016 respectively [18].

BMI is known to increase as people age, and then declines [19]. Globally, peak levels of obesity are found in those aged 50-60 in high-income countries, and 40-50 in middle and low-income countries [20]. In the UK, peak BMI was reported at age 71 and peak WC at age 80 for an older cohort of adults, with declines observed thereafter [19].

Obesity prevalence varies according to the global region and development level of the country [16-18, 20]. There was almost an 11kg/m<sup>2</sup> and 8kg/m<sup>2</sup> difference in BMI for women and men, respectively, between the regions with the lowest (central Africa for men and South Asia for both men and women) and highest (Polynesia and Micronesia) mean BMI in 2014 [17]. Amongst children, there is evidence that increases in BMI have started to plateau in North-western European countries and high-income English-speaking countries [18]. In middle and low-income countries there are rural-urban divides, with higher levels of obesity in urban areas, although females have higher levels in both settings than males [20].

The prevalence of overweight and obesity in the UK has been increasing since the 1980s across all ages. The Health Survey for England (HSE) reported that in 2019, 28% of adults in the UK were obese and 64% of the adult population were overweight or obese, with rates steadily increasing since 1993 [21]. Among children there have been stark increases in obesity between 1995 and 2004 [22, 23]. Increasing trends in mean BMI since the 1980s have been observed across all generations. Those born earlier in the 20<sup>th</sup> century have seen increases in obesity across the 1980s [24]. More recent generations are becoming obese at younger ages, therefore, spending more of their lifetime overweight and obese. By the age of 10, the likelihood of being overweight or obese, measured by BMI, was two to three-fold higher in cohorts born post-1980 than those born prior [24].

Obesity, as measured by BMI, follows a social gradient. Those living in the most disadvantaged circumstances are more likely to be overweight than their more advantaged counterparts. In the UK this is true of both adults [25] and children [26], highlighting a need to understand how and when in the life course such inequalities emerge. Understanding the unequal burden of obesity according to socioeconomic position (SEP) is important. Public health interventions aimed at reducing obesity that do not consider inequalities may have little impact on reducing inequalities or may inadvertently increase them [27]. In the UK, a comparison of inequalities in BMI across the British Birth Cohorts demonstrated there were no inequalities in BMI in childhood before the 1980s [28], and that inequalities in adult cohorts emerged around the same time [29].

## 1.2 Impact of the Environment

As the time in which obesity has emerged spans only the last five decades, increases in obesity are unlikely to be the result of genetic mutations and more likely reflect changes to our environment [30, 31]. This is supported by observed increases in obesity that have coincided with changes in our environment and ways of life [32]. Our environment, including the physical, social and cultural aspects, has often been heralded as a major determinant of the obesity crisis.

Arguably, as the environment has such a fundamental impact on our behaviour, if elements of the environment that promote obesity are not adequately addressed, preventions and treatments aimed at changing behaviour or educating individuals are likely to have limited impact [30]. Even where biology (i.e genetics) does have an important role in explaining contemporary variation in body size, it is often explained through “gene-environment” interactions [33]. As a result, the environment, particularly “obesogenic” aspects of the environment, has come under scrutiny as an explanation for

modern rises in obesity across the globe. The “obesogenic environment” was a term first defined by Egger and Swinburn in 1997 [30] as “the sum of influences that the surroundings, opportunities, or conditions of life have on promoting obesity in individuals or populations” [34], and has since been used to understand the elements of the environment that promote obesity.

### 1.3 Prevalence and Trends in Mental Health

Alongside increasing levels of obesity, the last century has seen increases in mental health problems, especially among adolescents. This is partly explained by increased recognition and reporting of mental health conditions but is also thought to reflect genuine secular changes [35, 36]. In the UK, a comparison between the 1970 birth cohort and the 2002/2003 Health Survey for England found twice as many reports of depression and anxiety in adolescents in the later-born cohort [37]. Comparison of the 1970 cohort with the 1958 NCDS and the 1999 British Child and Mental Health Survey also identified substantial increases in adolescent conduct problems over the 25 years [38]. Mental health disorders have been linked to an increased risk of obesity, with both conditions often co-presenting [6-8]. In the 1946 cohort, evidence of the link between mental health and obesity in childhood and adolescence was less clear [39] than in more recent born cohorts [40-42], indicating a possible secular change in the association.

### 1.4 Interactions between the Environment and Mental Health

Characteristics of the environment, such as access to green spaces, have been shown to improve mental health and wellbeing [43-45], including among adolescents [46]. Environmental conditions have also been shown to influence obesity relevant health behaviours such as engagement in physical activity [47, 48] and consumption of energy-dense foods [49]. The same health behaviours are associated with psychological disorders and wellbeing and are hypothesised to be on the pathway linking mental disorders and obesity [50]. It is therefore possible that environmental conditions interact with mental state to change how psychological characteristics manifest, or the health behaviours adopted as a consequence of mental state.

Children living in disadvantaged settings are typically at greater risk of social emotional problems [51]. Both obesity and mental health problems co-present with disadvantage [52]; mental health may sit on the pathway between SEP and obesity. Therefore, this mediating effect may vary dependent on the environment in which adolescents live.

### 1.5 Challenges of Measurement

Measures of body composition, such as FM and FFM, are relatively understudied compared with BMI. BMI is a good predictor of health outcomes at the population level [53] with high specificity [54] and is regarded as one of the easiest measurements to obtain as it only relies on height and weight. However, weight is made up of the mass of fat, bone, muscle and other tissues. As BMI is non-discriminant in estimating adiposity, there is no distinction between different bodily components such as FM and FFM that contribute to weight. As such, heterogeneity in obesity phenotypes is not sufficiently captured by BMI, and BMI may under or overestimate adiposity and the related health risk in an individual.

BMI has low sensitivity in measuring cases of obesity, with a systematic review and meta-analysis finding that half of adults classified as having excess adiposity measured by body fat percentage were not identified as obese using BMI [55]. Comparison of BMI to FM index (FMI) found BMI to underestimate the association between SEP and obesity in children [56]. The issues with BMI sensitivity are particularly notable when taking into account global variations in body shape, resulting in overall conservative estimates of adiposity. This, in turn, has consequences for the use of BMI as a risk factor in predicting disease, with the need for lower BMI cut-offs to be adopted to capture disease risk among different ethnic groups [57].

### 1.5.1 Why Use Body Composition?

Body composition breaks the body down into chemical or anatomically distinct compartments [58], that have distinct and independent associations with health outcomes. Whilst a lot of focus has been given to the importance of FM, less has been given to muscle mass and FFM. However, there has been a move to understand obesity in the context of both FM and FFM, with increased phenotyping of obesity and consideration of functional body composition [59]. For example, there has been greater recognition of conditions such as sarcopenic obesity in relation to ageing, characterised by low muscle mass and function, and excess adiposity, and the unique challenges this presents for health [60]. Additionally, there has been recognition of phenotypes such as “normal weight obesity” characterised by a “normal” BMI (BMI 18.5 – 24.9 kg/m<sup>2</sup>) but high levels of FM [61], and “metabolically healthy obesity” characterised by high BMI (BMI >30 kg/m<sup>2</sup>), lower visceral fat and high muscle mass with metabolic abnormalities absent [62].

Although BMI is correlated with both FM and FFM [59], there is still large variability in body composition phenotype among individuals with the same BMI, particularly in the ratio of fat to muscle [60]. There has been a move to understand obesity through criteria based on bodily components such as both FM and FFM, the ratio of fat to muscle, and their metabolic function and relation to disease risk [59].

### 1.5.2 Measuring Body Composition

Body composition can be measured in multiple ways, but the most common methods when assessing total body composition for research purposes are DXA and BIA. The gold standard for measuring body composition is through DXA, which uses the attenuation of two X-ray beams of differing energy levels to investigate the distribution and location of body composition [63]. The method allows accurate separation of the body into three distinct components: FM, lean mass, and bone mass [64]. In particular, as bone is excluded from the measure of lean mass, body composition measured by DXA provides a better measure of muscle compared to BIA [58]. Additionally, DXA is more fit for purpose in clinical and research settings compared to other techniques such as Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) scans, which are expensive, require technical expertise, and emit radiation in the case of CT scans [58].

However, obtaining measurements with DXA is still expensive compared to BIA. Therefore, BIA, which uses an electrical current to measure the body’s resistance [65], is often used as an alternative



in large population studies [66]. Instead of using a three-component model, BIA uses a two-component model that separates the body into FM and FFM. The FFM that is estimated includes both lean and bone mass. The method of BIA is unable to distinguish these two components unless population equations are applied that utilise the raw impedance measures [67]. However, such equations are highly specific to the population, reference method and BIA device used, with the recommendation being to develop specific prediction equations validated against a reference method in populations where this has not previously been done [67]. Therefore, it is usual to just use FFM which includes both bone and lean mass.

### 1.5.3 Fat-Free Measures

Although FM is relatively easy to measure and interpret, the composition of FFM is not. There is ongoing confusion in the literature relating to the terminology used to describe fat-free measures [68]. Here I provide the correct definitions which I use throughout the thesis, which are defined in Table 1-1.

FFM is total body weight minus FM, whilst lean body mass (LBM) is a fat-free measure equivalent to FFM plus essential fats found in the nervous system, cell membranes and bone marrow [69]. The difference between FFM and LBM is approximately 2-10% [68]. Lean mass (LM) is equivalent to FFM minus bone mass, and more closely captures what is colloquially thought of as “muscle” [58]. LBM and FFM are most often measured through BIA, because the method is unable to separate bone mass and lean tissue mass, whilst LM is most often measured through DXA because it uses the three-compartment model [58]. Appendicular Skeletal Muscle (ASM) is often included as a total body fat-free measure, as muscle mass in the limbs represents 75% of total skeletal muscle mass (SMM) [58, 70]. From here on, the term ‘Fat-free Mass (FFM)’ refers to the body composition measure that is weight minus fat and essential fats, whilst the term ‘fat-free measures’ is a collective term for all non-fat measures.

### 1.5.4 Indexing

Body composition measures can be presented in a number of ways. All measures start from the raw measure, which is the weight of the particular bodily component. This can then be converted into a percentage measure (typically as a percentage of total weight), a ratio measure (compared to a different component of body composition) or indexed to height (Table 1-1). Typically, in the body composition literature, percentage FM (FM%) has been used as a measure of adiposity. However, a number of issues exist with using FM% [71]. The main criticism is that FM% does not appropriately adjust for body size as it does not account for height, and therefore FM% remains correlated with height. Further, FM% cannot be interpreted independently of FFM, as FM plus FFM make up total body weight. FM% will therefore be 100% minus FFM% i.e., if FM% is 25% then FFM% will be 75%. Additionally, because total body weight includes FM when calculating FM%, FM is included in both the numerator and denominator. As a result, substantial changes in FM result in only minor changes in FM% [72]. As such indexing for height has been suggested, and the traditional formula used for BMI of indexing to height squared has been adopted.

**Table 1-1.** Description and Abbreviations of Direct Body Composition Measures used in this Thesis

<b>Body Composition Measures Used</b>	<b>Abbreviations</b>	<b>Description</b>
<b>Fat Mass Measures</b>		
<b>Raw Measures</b>		
Fat Mass (kg) (or Body Fat (kg))	FM (or BF)	Total mass of fat measured using BIA/DXA or other Sometimes used interchangeably with body fat in papers.
<b>Indexed and Percentage Measures</b>		
Fat Mass Percentage (or Body Fat Percentage)	FM% (or BF%)	Total fat mass as a percentage of total body mass: $FM\% = (fat\ mass\ (kg) / total\ body\ mass\ (kg)) \times 100$ Sometimes used interchangeably with BF%.
Fat Mass Index (kg/m <sup>2</sup> )	FMI	Total mass of fat indexed to height squared: $FMI = fat\ mass\ (kg) / height\ (m)^2$
<b>Fat-Free Measures</b>		
<b>Raw Measures</b>		
Fat-Free Mass (kg)	FFM	The difference between total body weight and FM. $FFM = total\ body\ mass\ (kg) - fat\ mass\ (kg)$ Includes bone mass. Most frequently calculated from BIA which is not able to separate bone from total body mass.
Lean Body Mass (kg)	LBM	LBM is similar to FFM, but also includes additional essential fat found in the nervous system, cell membranes and bone marrow, not captured by FFM [69]. $LBM = FFM\ (kg) + essential\ fats$ LBM is often considered synonymously, and used interchangeably, with FFM. Differences between LBM and FFM are around 2-10% [68].
Dry Lean Mass (kg)	DLM	Dry lean mass is equivalent to LBM, but without body water. $DLM = LBM - body\ water$ DLM includes bone mass.
Lean Mass (kg)	LM	Total body mass excluding fat and bone tissue *. $LM = total\ body\ mass\ (kg) - (fat\ mass\ (kg) + bone\ mass\ (kg))$ Most frequently calculated through DXA as able to distinguish bone mass. "Lean mass", if not measured by DXA, is more likely referring to LBM or FFM. Where the term lean body mass is used in relation to a measurement using DXA, it is included as LM as it is probable that it excludes bone, unless explicitly stated otherwise.
Appendicular Skeletal Muscle (kg)	ASM	Muscle mass of the four limbs without fat and bone tissue. Appendicular skeletal muscle represents 75% of total skeletal muscle [58, 70]. In some cases, ASM is used interchangeably with muscle mass (MM) or appendicular MM.
<b>Indexed and Percentage Measures</b>		
Fat-Free Mass Percentage	FFM%	Total fat-free mass as a percentage of total body mass: $FFM\% = (FFM\ (kg) / total\ body\ mass\ (kg)) \times 100$
Fat-Free Mass Index (kg/m <sup>2</sup> )	FFMI	Total fat-free mass indexed to height squared: $FFMI = FFM\ (kg) / Height\ (m)^2$

Lean Mass Percentage	LM%	Total lean mass as a percentage of total body mass: $LM\% = (LM \text{ (kg)} / \text{total body mass (kg)}) \times 100$
Lean Mass Index (kg/m <sup>2</sup> )	LMI	Total lean mass indexed to height squared: $LMI = LM \text{ (kg)} / \text{Height (m)}^2$
Appendicular Skeletal Muscle Percentage	ASM%	Appendicular Skeletal Muscle as a percentage of total body mass: $ASM\% = (ASM \text{ (kg)}) / \text{total body mass (kg)} \times 100$
Appendicular Skeletal Muscle Index (kg/m <sup>2</sup> )	ASMI	Total appendicular skeletal muscle indexed to height squared: $ASMI = ASM \text{ (kg)} / \text{Height (m)}^2$

**Table 1-1 Footnotes.** In some cases, measures indexed to height<sup>2</sup> may be calculated with an alternative power due to the high correlation of the indexed measure with height. \*Variations in the way LM may be calculated: exclusion of the head from estimates due to high proportion of bone meaning estimates of soft tissue are less accurate; participants observe fasting conditions and take no exercise prior to measurement.

### 1.5.5 Reference Values

Traditionally, obesity has been defined as the level of excess adiposity associated with increased disease risk. For FMI, there are no internationally agreed standard cut-offs that indicate obesity, but reference values were developed in The National Health and Nutrition Examination Survey in the USA and a FMI of < 3 kg/m<sup>2</sup> is taken to represent under fat, 3 kg/m<sup>2</sup> to 6 kg/m<sup>2</sup> for normal fat, 6 kg/m<sup>2</sup> to 9 kg/m<sup>2</sup> for excess fat (equivalent to overweight), and > 9 kg/m<sup>2</sup> as obese [73]. However, it is worth noting that different reference values have been developed in different populations.

For FMI in children and adolescents, different approaches have been taken as it is difficult to estimate disease risk among younger populations due to the small numbers of individuals presenting with metabolic diseases. Some papers adopt the 90<sup>th</sup> percentile [74], in line with the way obesity in children is measured by BMI using the international obesity task-force cut-off [75]. Others, including research conducted in the Avon Longitudinal Study of Parents and Children (ALSPAC), a regional birth cohort in the Southwest of England, has adopted a lower cut-off of the 75<sup>th</sup> percentile [76, 77]. A review of articles conducted in Mexican paediatric samples proposed FMI cut-offs of 5.1 kg/m<sup>2</sup> and 6.1 kg/m<sup>2</sup> at 6 years of age, 7.4 kg/m<sup>2</sup> and 10.3 kg/m<sup>2</sup> at 12 years of age, and 10.3 kg/m<sup>2</sup> and 13.1 kg/m<sup>2</sup> at age 19 years of age indicating obesity in boys and girls respectively [78]. However, these are likely to differ to a predominantly white population in the UK or Europe, and therefore continuous measures rather than cut-offs are adopted in this thesis.

### 1.5.6 Clinical Importance of Body Composition

Body composition is related to health outcomes independently of BMI [71]. A higher proportion of fat-to-lean mass [79] and greater central visceral fat [80-82] are associated with increased cardiovascular disease risk. Total and proportion of FM is associated with cardiovascular and metabolic disease, with higher central adiposity and android-to-gynoid FM ratio also implicated in increased risk [81-84]. Greater FM is associated with decreased muscle quality, which is more important for functional outcomes than muscle mass [85]. Additionally, FMI has been found to be a better screening tool for predicting metabolic syndrome than BMI or FM% [71].

Research conducted in the ALSPAC found LM associated with lower atherogenic traits [86]. In particular rapid increase in muscle during adolescence in males confers benefits to cardiovascular health in early adulthood [86]. Increasing muscle volume and strength may be important for other aspects of health in later life, such as physical capability. In particular, muscle development in late adolescence and early adulthood is important for maintenance of later life physical capability and reduced risk of all-cause mortality [87].

Among women, a decline in physical functioning has been found to be dependent on baseline values of physical functioning, with greater rates of decline in early life and mid-life among individuals with lower baseline functioning [88]. Higher FFM and relative muscle mass also have a protective role in the development of insulin resistance, having the potential to delay the onset of diabetes and other metabolic conditions [89, 90].

## 1.6 Thesis Outline

This thesis aims to generate an understanding of the relation between SEP and body composition that will inform knowledge of the potential health consequences of inequalities in adolescent obesity. In particular, this thesis is interested in understanding inequalities among children and adolescents in the UK, building on previous work examining similar associations for BMI. It aims to understand the extent to which emotional and behavioural problems in childhood and adolescence explain social inequalities in body composition, and whether environmental conditions moderate these pathways.

This thesis is structured into four parts, each consisting of multiple chapters. The first section consists of the introduction, literature review and methods. Following the introduction chapter, Chapter 2 provides a literature review that outlines the different theoretical approaches adopted in this thesis, namely a life course approach and social determinants approach. Chapter 2 also provides definitions and theoretical background of key measures, presents relevant literature on the topic, and sets out the framework on which the rest of this thesis is based. Chapter 3 is the aims and objectives and sets out the hypotheses that are tested. Chapter 4 is the methods chapter and introduces the data set and key measures.

The second part of this thesis aims to understand the extent of life course inequalities in body composition, both globally and in the UK. This is achieved firstly through a systematic review of the literature linking SEP and body composition in both adults and children across the globe (Chapter 5). Chapter 5 adopts a wide inclusion criterion in order to understand how inequalities have changed over time and how this varies between countries. This provides a broad understanding of the extent of body composition inequalities globally and provides the context in which they likely developed in high income countries across generations and across the life course. The focus is then narrowed to children and adolescents in the UK for subsequent secondary data analysis. This narrower focus is achieved through a growth curve analysis of inequalities in body composition using the UK MCS (Chapter 6) to identify if and when inequalities in body composition emerge, and the extent and direction of these inequalities. By looking at SEP in infancy, Chapter 6 adopts a life course approach that investigates the long-term impact of early life inequalities on body composition and allows

identification of when is the best time to intervene to prevent inequalities in body composition. Importantly, Chapter 6 considers both family-level SEP and area-level SEP in infancy, to begin to untangle the separate effect of environmental determinants of inequalities.

The third section of this thesis aims to investigate a possible explanation underlying inequalities in body composition through internalising and externalising symptoms (measured through the Strengths and Difficulties Questionnaire (SDQ)) and environmental characteristics. Chapter 7 aims first to demonstrate the robustness of the SDQ scale for measuring internalising and externalising symptoms through exploratory and confirmatory factor analysis, as internalising and externalising symptoms are hypothesised mediators in the relationship between SEP and body composition. Finally, in Chapter 8 these mediating pathways are tested using Structural Equation Modelling (SEM), and a moderated mediation model is tested. The interaction between environmental conditions is explored, to see if the extent of mediation depends on characteristics of the environment (the level of deprivation, green spaces, domestic gardens and fast-food density of an area) in which individuals live.

The final part of this thesis is the Discussion, Appendices and Bibliography. Chapter 9 is the discussion that summarises and provides critical comments on the findings, discusses the methodological challenges of this thesis, comments on policy implications, and concludes this thesis.

# Chapter 2 Literature Review

This chapter provides a review of the literature relevant to the aims of this thesis. Firstly, the different theoretical underpinnings that are important to the study of social inequalities are discussed, along with other theoretical approaches that have influenced the work. Next, consideration is given to the different ways SEP can be measured. This is followed by a review of the literature exploring inequalities in BMI, and a discussion of secular trends in body composition. The key mediators and moderators adopted in this thesis, that are hypothesised to explain the link between SEP and body composition, are then defined. This is followed by a critical discussion of relevant hypotheses and empirical literature relating to the mediating and moderating variables.

## 2.1 Theoretical Frameworks

The research in this thesis is influenced by several theoretical approaches. Most notably this thesis adopts a life course approach to the study of social inequalities through exploring the way in which inequalities develop. Additionally, there is application of a social determinants of disease framework that conceptualises the role of SEP on health. This thesis also takes ideas from affective state frameworks in health psychology, to understand how environments may modify association between psychological state and body composition. Finally, obesity is viewed as being influenced by the obesogenic environment, which falls under a socio-ecological approach.

### 2.1.1 A Life Course Approach

A life course approach to epidemiology seeks to understand health trajectories across life by adopting a multidisciplinary understanding of the factors that shape development, including social and physical exposures, that occur early in the life course [91, 92]. Importantly, the life course approach recognises that both social and biological factors are important for human health, and that these independently, cumulatively and interactively impact development of disease across life [91]. The life course approach does not neglect the importance of behavioural risk factors (i.e. diet and exercise) that occur in adulthood but explores these alongside early life influences to understand how and why they may come about [91]. Although the life course approach originated from a desire to understand adult chronic disease outcomes through early life exposures, more recently there is recognition that life course studies can be used to understand outcomes in childhood and adolescence also. Ultimately the life course approach aims to identify when in the life course interventions may be most effective in improving lifelong health, with recognition that development of disease occurs much earlier than the visible onset [93].

A life course approach to social inequalities investigates how inequalities develop over the life course and how socially patterned exposure across life result in social inequalities in adult health. In particular, disadvantage in early life shapes child development through influencing the material, physical, and psychosocial conditions experienced [94].

The life course approach adopts a number of different models that can help conceptualise the causal pathways between social circumstances and body composition. The accumulation of risk model, which was developed from Riley's concept of insult accumulation [95], assumes that negative or positive exposures accumulate over the life course. These lifetime exposures can either be independent or clustered [91]. Clustered models are particularly relevant when thinking about social determinants of disease, as the multiple exposures – such as environmental conditions, health behaviours, material circumstances – are often related to, or clustered within, socioeconomic circumstances.

An alternate but related model, the chain of risk model, stipulates that disadvantage at one point in the life course is related to risk factors in another, and that each factor in the chain can be independently and additively related to the outcome [91, 92]. Chains of risk can be formed of both social risk factors, therefore a “social chain of risk”, or formed of biological and psychological risk factors (“biological and psychological chains of risk”), or a mix. Chain of risk models involve mechanisms, such as mediators and moderators, that explain how pathways form between the exposure and outcome over the life course [91].

The life course approach is interested in critical periods of development where exposures at that time, but not at any other time, may have lifelong impacts. Traditionally, critical periods are conceptualised to be early in the life course during infancy when development is rapid, and relate to biological programming or latency effects [96]. However, recent work has highlighted how adolescence can also be conceptualised as a critical period in the life course, as second to infancy, adolescence is the period with the most rapid physiological development [15]. Adolescence is also an important period socially when individuals mature from children into adults and gain greater independence. It is an important period for psychological development, and also the period in which patterns of health behaviours develop and are maintained.

A more recent focus in life course epidemiology is an interest in life course trajectories of measures of health, which considers patterns of change in measures of body function and structure [91]. Many functional trajectories are defined by rate of development, the timing and degree of peak function, and the rate of decline in functioning [97].

### 2.1.2 Social Determinants Approach

The social determinants of health approach originated from the observation that many health outcomes follow a social gradient, with dramatic differences in health status between the most and least advantaged in society, and incremental and proportional differences for all those between [98, 99]. As such social structures must be important for understanding population health. The approach recognises that health is social as well as biological and aims to examine the non-medical reasons that influence population health [100]. These non-medical reasons include both social circumstances at the individual level, but also broader social, environmental and political conditions, and the structures that maintain social positions for individuals within society [101]. A key feature of the social

determinants approach is that in order to address health inequalities, the barriers to control over one's life and material deprivation, need to be removed so that individuals' health needs can be met [98].

The social determinants of health framework assumes traditional behavioural risk factors for disease, such as smoking, drinking, diet and exercise can be considered the causes of disease, but takes a step back to ask what are the "causes of the causes?" [98]. It is the social and environmental conditions in society that are framed as the "causes of the causes" [98, 101, 102]. In this sense, behavioural risk factors are on the pathway between socioeconomic risk factors and health outcomes. Thus, clear parallels can be drawn between the social determinants of health approach and the life course approaches chain of risk and accumulation of risk models.

The social determinants framework also explains gradients in health through material and psychosocial pathways. Material conditions include living and working conditions, as well as the potential to consume. Material circumstances impact health through access and availability to food and services and through the living conditions individuals experience on a day-to-day basis [101]. Psychosocial pathways focus on the subjective experiences of individuals, and the consequences of social disadvantage for wellbeing [98]. It is assumed that such states of disadvantage or relative deprivation create conditions of chronic stress, and, in turn, can trigger mental illness or impact physical health. These conditions of stress and poor psychological functioning then have further consequences for physical health.

### 2.1.3 Affective States and the Affect Regulation Model

Affect is a broad characterisation of emotions and feelings that influence the ways in which individuals interact with their surroundings and opportunities. Affect can either be negative or positive, characterised by negative emotions such as anger and sadness or by positive emotions such as joy and enthusiasm, respectively [103]. Negative and positive affective states are considered independent of each other, in that they are considered separate constructs as opposed to being different ends of the same continuum. Regular experience of negative affect is associated with psychological conditions such as anxiety and depression. Depression is associated with high negative affect and low positive affect, and anxiety is associated with high negative affect and average or high positive affect [103]. However, affective states also vary based on circumstances and situations, can change daily, and therefore can be influenced by the conditions experienced by individuals.

In the health behaviour literature, affective states can influence behaviour change and the decision-making process through affect regulation models. Those who experience positive affective states tend to adopt better coping strategies, engage in positive health behaviours, and be more likely to have long-term adherence to such behaviours [104]. Those who have negative affective experiences tend to adopt negative coping strategies and engage in maladaptive health behaviours such as bingeing and eating unhealthy food to regulate the negative states [105]. In this sense, the affective state model relates to the social determinants of health model that hypothesises psychosocial pathways between socioeconomic disadvantage and health outcomes.



The affective state model can be used to explain these psychosocial pathways, by assuming the psychosocial impacts of disadvantage increase negative affect which in turn increases the propensity to engage in damaging health behaviours. In the context of obesity, the relationship with psychological symptoms can be understood through emotional eating or binge eating to reduce or regulate negative affect states [106, 107]. Additionally, negative affect states may also increase engagement in risk behaviours by increasing reward sensitivity and impairing decision-making capacities [107]. It has been demonstrated that positive affective states result in increased enjoyment from health behaviours such as healthy eating and exercise, and therefore can encourage a positive feedback loop to engage with such behaviours in the future [104].

#### 2.1.4 Socio-Ecological Models and the Obesogenic Environment

The obesogenic environment and research that tries to understand environmental impacts on obesity are routed in socio-ecological models [31]. The initial model proposed by Egger and Swinburne categorised the environment in two ways. Firstly, into the macro (effecting population prevalence of obesity) and the micro (effecting individual likelihood of obesity) environments. Secondly, they split the environment into the physical, economic and sociocultural environments [30]. The initial model of the obesogenic environment was later refined into the Analysis Grid for Environments Linked to Obesity (ANGELO) framework, and the addition of the “political” environment was included [33].

In the ANGELO framework, the physical environment refers to “what is available” [33] and is one of the most frequently researched elements of the environment in relation to obesity [108, 109]. The physical environment refers to tangible aspects of our environment, such as availability and type of food and outlets, access and opportunity to engage in leisure, and the built environment that promotes or discourages physical activity. However, Swinburne and Egger stress that the physical environment also refers to less tangible elements, such as training opportunities, nutrition and exercise expertise, technological innovations, and information [33].

The sociocultural environment refers to attitudes, values and beliefs held within the community or society that make up the social and cultural norms. These can occur within micro-environments such as in the house, school or workplace, or in macro-environments, where mass media and celebrities may have important influence. The economic environment refers both to the costs related to food and physical activity in the environment, but also refers to income, education, occupations and SEP [33]. Finally, the political environment refers to the rules and regulations placed on food and activity. This can also occur at the micro-level (i.e in schools or households) or at the macro-level, where governments or local councils may make decisions, such as preservation of green spaces, the amount of food advertisement aimed at young people, regulations on food products, and town planning [33]. As such, social, economic, and political elements of the environment, including SEP, are a fundamental part of the obesogenic environment. However, it is these parts of the environment that are considerably understudied in comparison to the physical environment [108, 109].

## 2.2 Social Inequalities

Before exploring inequalities in BMI and obesity, it is important to be clear about what frequently used measures of SEP indicate. Many interrelated SEP measures exist, with some of the most commonly used in high-income countries being education, income and occupation. Education captures aspects of earlier life SEP and is a determinant of later life SEP such as occupation and income [110].

Pathways from education to health outcomes are conceptualised through knowledge assets and increased health literacy. In the context of obesity, this may be the awareness of the negative health consequences of excess weight gain and how to prevent it. Income is a measure of material resources [110] and can increase access to resources indirectly and directly, such as through access to healthier food options, leisure centres and health services. Income also determines where individuals live, which indirectly influences access to resources through geographic location.

Occupation captures different aspects of SEP, including education, income and material resources. The relation between occupation and health outcomes can be explained in part by these other factors, as well as social standing, networks and through working conditions [110]. Highly sedentary jobs will have different impacts on obesity outcomes compared to manual occupations. Similarly, long working hours will affect opportunities to engage in activities outside of work.

## 2.3 Social Inequalities in BMI and Body Composition

There have been no systematic reviews that have previously considered the link between SEP and body composition. However, the link between SEP and obesity measured by BMI has been extensively reviewed and is discussed here. Sobal and Stunkard (1989) were the first to systematically review the literature on BMI. They examined 144 papers published from the 1960s to the mid-1980s, and found strong and consistent associations between more disadvantaged SEP and greater prevalence of obesity among women in high-income countries [111]. The association in children and men was less clear [111]. In low- and middle-income countries they reported consistent associations between more advantaged SEP and higher levels of obesity in all groups.

McLaren's (2007) update to this review supported a higher number of inverse associations, where advantaged SEP was associated with lower levels of obesity, in high-income countries, and a greater number of positive associations, where advantaged SEP was associated with higher levels of obesity, among low and middle-income countries [112]. McLaren found associations differed based on the SEP measure used, with education, area-level indicators, occupation and composite measures presenting strong inverse associations for women in high-income countries. McLaren found the differences between women in lower and higher income countries to be less drastic than previously reported [112].

Systematic reviews focused on low-income countries indicate predominantly positive associations across groups, with socioeconomic advantage typically associated with higher BMI [113]. In middle-income countries there is evidence of mixed associations in men and inverse associations in women, with socioeconomic advantage associated with lower BMI [113]. As the gross national product increases, the burden of obesity shifts to those of a less advantaged position within society, which

affects women first [114]. This gender difference is thought to result from the greater pressures of weight related ideals faced by women, which are easier to maintain in a position of advantage [115].

### 2.3.1 Childhood Socioeconomic Position and Obesity Across the Life Course

The cross-sectional link between SEP and BMI in high-income countries has been well established, but studies have highlighted that childhood SEP may have a continuing effect on adult obesity. This relates to a life course approach to inequalities, as previously discussed in the theoretical frameworks.

Parsons et al. (1999) reviewed the associations between early life SEP and risk of being overweight in adulthood, and found those of the most disadvantaged SEP in early life to experience an increased risk of being overweight in adulthood [116]. Senese et al.'s (2009) update found consistent associations between childhood SEP and obesity in adulthood in females, but a less consistent pattern among males [117]. Inequalities were found to increase with age and across time. Inverse associations were found in 64% of studies conducted in females before 1950, with approximately half of the studies included consisting of participants born before this time, and 74% after. For males, this was 22% before and 33% after [117].

### 2.3.2 Inequalities and Life Course Inequalities in BMI: UK Context

Disadvantaged SEP has been found to be associated with higher levels of obesity in systematic reviews for children in the UK [26], whilst systematic reviews conducted among adults in the UK found disadvantaged SEP to be associated with increased adult obesity [25]. In adults, associations tended to be stronger in women than in men [25].

Research from the British Birth Cohorts have shown childhood SEP is associated with adult BMI in both males and females, although more strongly in females [29]. Those born in the 1970 (BCS) cohort, demonstrate larger inequalities in BMI by adult SEP at the same age in adulthood, compared to those at the same age in earlier born cohorts. Inequalities for those born in the 1946 cohort (NSHD) were lower still than those born in the 1958 cohort (NCDS) [29]. Looking at BMI inequalities in childhood, those born in the 1970 cohort and earlier showed no or only small inequalities in childhood, whilst the MCS cohort born in 2000/2 showed inequalities in BMI from a young age [28]. Additionally there was widening of the inequality, by income, in obesity between the ages of 5 and 11 in the 2000/2 born cohort [118]. Children who were in the lowest fifth of household income had double the risk of being obese by the age of five when compared to those in the highest fifth. By the age of 11, the same children were three times more likely to be obese [118].

### 2.3.3 Trends in Body Composition

BMI and body composition measures are related, and there is evidence of secular changes in the association. Increases in BMI at younger ages observed among more recent cohorts are likely due to increased adiposity as opposed to increasing LM [24]. Evidence from serial data in the US Fels Longitudinal Study shows increases in FM%, WHtR and BMI among boys and girls aged 8 to 18 between 1960 and 1999 [119]. Among boys, these increases in BMI were shown to reflect increases in adiposity, not FFM; whilst in girls, this did reflect an increase in FFM [120]. A study in England

using repeated cross-sectional data between 1998 and 2014 found evidence of decreasing muscle strength among children, in association with decreasing physical activity [121].

Indirect evidence suggests increases in BMI in adolescence and adulthood are associated with acquisition of fat. Infant growth patterns in the 1946 NSHD cohort were associated with body composition outcomes in later life. Although higher birth weight and weight gains in infancy were associated with higher lean mass at age 60-64, greater gains in BMI in childhood and adolescence were associated with greater android-to-gynoid fat and fat-to-lean ratios also measured at age 60-64 [122]. In Finland, individuals born between 1934 and 1944 had a higher FMI age 56-70 if they experienced more rapid BMI increases between the ages of 2 and 11 [123]. Both cohorts also found higher BMI and faster increases across childhood to be associated with greater muscle in older age [85, 123]. However, the quality of muscle was lower at age 60-64 for those with greater gains in BMI from age 15 to 60-64 in the 1946 cohort [85]. There was no accompanying increase in muscle strength measured by grip strength, indicating poorer muscle quality in later life with greater BMI in adolescence and BMI gains across the life course [85].

#### 2.3.4 Inequalities in Body Composition

Although the number of studies investigating inequalities in body composition is substantial, it is still smaller than the number investigating the association for BMI. Few studies test the association between SEP and body composition as a primary research aim, with a larger number of studies including SEP in models as a covariate or tested as part of secondary analysis. While a large number of systematic reviews have been carried out on social inequalities in BMI, the literature for body composition has not been systematically reviewed. If inequalities in body composition exist and the effects of adiposity on health have previously been underestimated when using BMI alone, this is a major public health concern. This is supported by research that finds BMI underestimates the educational gradient in adiposity among Dutch children compare to FMI [56]. In the body composition literature, a lot of focus has been given to the importance of FM or adiposity, whilst less has been given to muscle and FFM. This is an important but understudied area, with little known about inequalities in fat-free measures. Therefore, the first aim of this thesis is to review the literature to identify if inequalities in FM reflect those seen for BMI, and to review the evidence on inequalities in FFM (Chapter 5).

#### 2.4 Hypothesised Mediators and Moderators

Internalising and externalising symptoms are potential mediators of the association between SEP and body composition. It is assumed that disadvantage increases the likelihood of exhibiting internalising and externalising problem behaviours in childhood and adolescence, and this, in turn, increases the risk of unhealthy body composition. In order for these symptoms to be a potential mediator, they need to be patterned by SEP and associated with obesity. As such, evidence for these associations is explored below.

It is thought that in more obesogenic areas, mediation through internalising and externalising symptoms is more pronounced, due to stronger associations between symptoms and body

composition. This section outlines possible mediating paths, moderating effects, and interactions between the two. A moderating effect is explored by investigating characteristics of the environment thought to capture obesogenic environments and exploring how they interact with psychological symptoms.

### 2.4.1 Adolescent Mental Health: Internalising and Externalising Symptoms

Internalising and externalising symptoms are concepts used to understand psychopathology and mental health among children. They are considered types of emotional and behavioural difficulties in childhood. As such internalising and externalising symptoms are often referred to as emotional and behavioural problems, respectively. The two distinct sets of behaviours were originally developed through clinical classification systems but have more recently been validated as different behavioural traits in children, and have distinct predictive properties. Although internalising and externalising symptoms are not mental disorders or illnesses in and of themselves, they are often precursors to mental disorders in later life.

Externalising symptoms are those that are directed outward from the individual and are considered disruptive. Typically, externalising symptoms are characterised by impulsivity, lower self-regulation and worse inhibitory control [124] and associated with conditions such as attention deficit hyperactivity disorder (ADHD) [106]. In the literature, externalising symptoms are often referred to as problem behaviours or discussed in terms of hyperactivity and conduct disorders.

Internalising problems are directed inwards to the individual, and emotional difficulties are internalised. Internalising symptoms are categorised by emotional dysregulation and peer problems, and are predictive of and related to conditions such as depression and anxiety among others [106]. In the literature, internalising symptoms are often referred to as emotional problems. For both sets of symptoms the terms “disorder” and “problems” are often used interchangeably [125].

#### 2.4.1.1 *Social Inequalities in Internalising and Externalising Symptoms*

Systematic reviews [126, 127] and studies in populations across Europe [128, 129] find support for higher psychological and emotional distress among both adults and adolescents experiencing greater social disadvantage. In children and adolescents, a review of 55 papers found overwhelming evidence that early life disadvantage is related to increased mental health problems, with those children experiencing disadvantage being 2-3 times more likely to develop mental health problems [51]. Persistent disadvantage was more strongly related to mental health problems, and associations were generally stronger in childhood compared to adolescence, and in relation to income and parental education compared to parental occupation [51].

Many dimensions of SEP relate to internalising and externalising symptoms. Both neighbourhood and family poverty were shown to be related to emotional and behavioural problem trajectories in the MCS [130, 131]. Not only have individual-level and neighbourhood-level disadvantage been shown to relate to internalising and externalising symptoms, but school poverty as measured by number of students on free school meals has been related to both sets of symptoms concurrently in the MCS [131, 132], and longitudinal associations were observed for emotional symptoms. Gender moderated

associations between school poverty and emotional and behavioural symptoms, with girls having a steeper increase in internalising symptoms as a result of school poverty [132].

#### *2.4.1.2 Relation of Internalising and Externalising Symptoms with Obesity*

Internalising and externalising symptoms have been found to relate to obesity. Psychological characteristics and constructs relevant to internalising and externalising symptoms co-present with obesity in cross-sectional, community-based and clinical studies [6-8]. Systematic reviews [116, 133, 134] have highlighted the importance of psychological factors in earlier life and weight in adulthood. Longitudinal research in the USA showed emotional and behavioural self-regulation difficulties, including reduced inhibitory control and increased reward sensitivity at 2 years of age, to be related to obesity among children aged 5.5 years [135]. Further research in the USA, analysing a prospective cohort of mothers and children found externalising problem behaviours to be associated with increased probability of obesity in both boys and girls [124].

The Health Survey for England found that both internalising and externalising factors were associated with obesity in children and adolescents [136]. Longitudinal studies have provided further support for the association between internalising factors and obesity, with a study of 6-17 year olds finding greater incidence of being overweight and having a higher BMI 10-15 years later among those experiencing major depression, when compared to a healthy control group [137]. These results have also been supported in nationally representative samples, where baseline depressive mood has been found to be predictive of worsening obesity, and increased weight among those adolescents who were not obese at baseline [138].

In the MCS, poor emotional regulation at age 3 was associated with an increased risk of obesity at age 11 [42]. There's some evidence that such associations may begin at a young age, with modest evidence of an association between externalising behaviours at 24 months and BMI from ages 2-12 in the National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development [139]. Associations between emotional and behavioural problems and obesity from the ages of 3 and 5 are observed in the MCS [41].

#### *2.4.1.3 Mediating Pathways*

There has been little research that has looked at mediating paths for social inequalities in body composition through mental health. A paper conducted in adults demonstrated that depressive scores mediated the inverse relationship between SEP and WC and WHR, but not trunk fat measured by DXA, in white American women [140]. Depressed mood was found to be related to muscle mass among adolescent girls in South Korea [141], but there was no investigation of whether depressed mood mediated any associations between SEP and muscle mass.

More research has investigated mediating paths for BMI and other anthropometric measures. Evidence supports the pathway between psychological symptoms and obesity through eating as a regulatory mechanism for emotions [106, 142-144]. Experimental studies have also created the conditions of disadvantage and found individuals were more likely to consume calories from snacks compared to those that were put in conditions that made them feel wealthy [145]. A study of 150

participants in the Northwest of England found evidence that those from more disadvantaged social positions had greater psychological stress, resulting in increased emotional eating and higher BMI [146]. The pathways between SEP, psychological stress, emotive eating and increased BMI has been tested on a few occasions, and studies systematically reviewed [147]. Of 14 studies looking at the links between social position, stress and body mass index, those of more advantaged social position had lower stress levels and lower body weight, which was explained through healthier dietary patterns [147].

#### 2.4.2 The Physical and Social Environment

A number of different measures of the environment relate to the obesogenic environment. Those considered in this thesis are green spaces, domestic gardens, fast-food density and area deprivation (measured through the index of multiple deprivation (IMD)). These measures of the environment are often conceptualised as influencing obesity risk through: having access and opportunity to exercise; access to healthy or unhealthy food; access to services; and, more broadly, access to social and cultural opportunities provided by the local environment that relate to health behaviours and coping strategies. Research on the obesogenic environment has often adopted single and physical elements of the environment, such as the number of fast-food outlets and access to green spaces. These aspects are easier to conceptualise causal pathways for and to design public health interventions for.

The IMD can be considered in addition to physical measures of the environment, as area deprivation is an understudied aspect of the obesogenic environment. Area deprivation may provide further insights into the impact of sociocultural elements of the environment. Conceptual models have highlighted how neighbourhood deprivation relates to obesity through both the built environment and the social environment [148].

There are a number of challenges in measuring the environment in obesity focused research. Studies that have attempted to focus on a single measure of the physical environment have often found mixed results, likely because environments have both positive and negative elements that are not captured by a single measure. Additionally, individuals are not static and exposure to different environments, even on a day-to-day basis, may confound associations [149]. Environments in which people live also change over time, with evidence that this has happened dramatically since the 1980s [150, 151]. Therefore, environmental measures are often prone to high levels of confounding by other environmental characteristics. This is something that is highlighted in the Foresight report as a key barrier when using single measures of the obesogenic environment [109].

The IMD touches on several understudied areas relating to the social and cultural environment, that may influence obesity through social norms, networks and peer behaviours influence [148]. Additionally, area-level SEP measures are considered good proxies of local environment conditions given the close association with physical elements of the environment [151-159]. For example, the IMD in the UK has been shown to correlate with fast-food density [160]. It is likely that the IMD also captures aspects of the environment less routinely collected, such as local services, and gives a more holistic assessment of both the positive and negative elements of the environment. The relation of

area-level SEP measures with obesity often reflects differences in the local environment and less individual SEP. In Spain, the association between area-level SEP and obesity was better explained by the number of sporting facilities than by household SEP or risk behaviours [161]. In UK Biobank, social deprivation (Townsend Deprivation Index) was found to best capture aspects of the obesogenic environment that were responsible for increased obesity among genetically susceptible adults [162]. Therefore, by adopting multiple measures of the environment in this thesis, and by using broader definitions of the obesogenic environment that incorporate deprivation, some of the issues highlighted can start to be addressed.

#### *2.4.2.1 Social Inequalities in the Environment*

Physical aspects of the environment are socially distributed. In England [152], Scotland [153] Australia [155] and the USA [154], it has been shown that there are more fast-food outlets in deprived or low-income areas. The number of outlets was particularly high around primary and secondary schools in disadvantaged areas in Australia [155]. The increase in number of fast-food outlets opening between 1990 and 2008 was also highest in the most deprived areas compared to the least in the UK [151], and food advertising space was also larger [156]. In the USA, areas of more disadvantaged SEP had reduced access to sports facilities [159]. In Spain [157] and China [158], distance to green spaces was greater in more deprived areas, and those spaces were found to be of lower quality.

#### *2.4.2.2 Association of Environment with Obesity*

Multiple aspects of the environment have been linked to obesity. Higher ratios of fast-food restaurants and convenience stores to grocery stores [163] and higher density of fast-food outlets in proximity to where people live [49, 152] was associated with increased intake of takeaway food and risk of obesity. Cross-sectional research in the UK using objective metrics of the food environment found that greater exposure to takeaway foods in either the home or work environment, or when commuting increased intake of these foods [49]. Those who had the greatest exposure, measured by a greater density of takeaway food outlets in the environment, were more likely to be obese with a dose-response relationship in the association [49]. Another study in England found similar results among children, with the number of food outlets serving unhealthy food increasing the risk of obesity, and those areas with other types of food outlets decreasing the risk of obesity [152]. Exposure to healthy stores and food outlets has been found to be associated with improved dietary quality scores among children [164].

A systematic review of green spaces and obesity found that the majority of papers demonstrated positive or weak associations between greater objectively measured greenspace access and obesity related health indicators, such as physical activity and BMI [165]. Associations were inconsistent, with results varying by age, SEP and how green space access was measured (i.e. distance to home, count within given area, percentage green space in an area). Another systematic review found green spaces had a greater benefit for general physical health among individuals from disadvantaged SEP [166]. In China, distance to green spaces was related to incidence of disease, measured by heart



disease, hypertension and chronic pneumonia, with a shorter distance to high-quality parks associated with improved outcomes [158].

Systematic reviews have linked other aspects of the built environment, in particular access to sports facilities, to obesity over the life course. A review by Papas et al. (2007) which focused on how aspects of the built environment relates to physical activity, found 85% of included studies reported an association between some aspect of the built environment and obesity [167]. Dunton et al. (2009) found adolescent obesity to be associated with aspects of the built environment such as access to sports facilities, urban/rural areas and urban sprawl [168]. Other reviews found that, among individuals of disadvantaged SEP and ethnic minorities, lack of access to exercise facilities, supermarkets and experience of reduced safety were associated with higher BMI or poor weight-related health behaviours [169].

Conceptual models have highlighted how neighbourhood deprivation relates to obesity through both the built environment and the social environment that captures social capital, collective efficacy, and crime [148]. Neighbourhood social environments may also relate to obesity through physical activity levels, with a positive perception of social environments increasing the likelihood of engagement in physical activity [148]. Changes to behaviour may occur directly by restricting access to facilities, or indirectly through greater exposure to cues in the environment, resulting in unconscious changes to feelings of satiety, hunger and food preferences [150]. Health behaviours are one of the main pathways linking the environment to obesity, supported by increased intake with greater exposure to fast-foods [49] and higher levels of physical activity in greener areas [47, 48]. Those living in closer proximity to urban green spaces are more likely to meet recommended guidelines for physical activity [170].

#### *2.4.2.3 Environmental and Psychological Interactions*

Both environmental and psychological determinants have been explored as mediators of social inequalities in obesity. There has been limited research, however, investigating individual psychology and environmental exposures together to explain inequalities in obesity. A systematic review has looked at both pathways between SEP and obesity and found the combined effect of environmental and psychological factors to result in greater mediation than a single pathway [171]. However, it is likely that the two sets of factors also interact.

Elements of the obesogenic environment accentuate, and moderate other factors related to the individual, such as genetic risk or adherence to behaviour interventions [162, 172]. It is therefore possible that emotional and behavioural problems may also be influenced by environmental conditions, which provide environmental cues that moderate coping strategies. It is hypothesised that the mediating effect of internalising and externalising symptoms is moderated by characteristics of the environment, such that the mediation is greater in a more obesogenic environment.

That mental health can be influenced by environmental conditions has been previously demonstrated, especially in relation to the positive impacts of green spaces [43-46]. Previous research has demonstrated that green spaces improve mental wellbeing and reduce mental distress [173, 174].

Among individuals who move to greener areas, mental health, as measured by the General Health Questionnaire (GHQ), improves, whilst among those who move to less green areas it worsens [175]. In the MCS, neighbourhood green spaces have been related to lower emotional and behavioural problems in childhood (ages 3-7), and the positive effect of green spaces was particularly noted in poor children in urban settings [176].

A number of theories have been proposed to explain the benefits of green space and natural environments for mental health. The attention restoration theory (ART) first proposed by Kaplan (1989), suggests improvements to concentration and reductions in mental fatigue can be achieved through interactions with natural environments [177]. In particular, increased time looking at or watching nature, or spending time in nature, is likely to offer opportunities for mental restoration [177]. Similarly, the stress reduction theory, proposed by Ulrich (1981), suggests that experiencing nature can reduce feelings of stress and increase positive emotions, such as pleasure and feelings of calm [178, 179]. Green spaces, especially in urban settings, may therefore provide an opportunity for mental restoration, reduce stress and increase the abundance of positive emotions.

Improved mental health is related to increased engagement with positive health behaviours such as healthy eating and exercise. Both physical activity and spending time in natural environments can increase positive affective states [180], whilst green spaces are related to increased engagement in physical activity [47, 48]. Therefore, positive elements of the environment may simultaneously improve mental wellbeing, alongside providing cues to engage in healthy behaviours. In areas where green space access is higher, social inequalities in health have been found to be smaller than those with poorer green space access [181].

On the other hand, negative environments may result in worsening mental health. Social and economic deprivation of areas has been shown to be related to children's problem behaviours in the MCS. Aspects of deprivation such as the income, education and employment levels of people living in an area were related to childhood emotional and behavioural problems, with higher deprivation across domains being associated with a greater presentation of problems [182]. Chronic stress from being in a less advantaged social position and in an environment of deprivation may contribute to behavioural and emotional problems in children, which in turn may increase the likelihood of negative affect regulation. Area-level measures of deprivation also likely capture the cultural and social environments in which people live, and through this they also capture the social norms, social networks and peer behaviours in the environment that influence diet and physical activity of adolescents [148].

In the USA, higher fast-food outlet density has been demonstrated to be associated with worse psychological profiles [183]. Increased consumption of food was also found among individuals with higher mean negative affect [184]. However, higher snacking in response to access to fast-food was moderated by daily variability in negative affect, with weaker associations with higher variability [184]. Negative affective states can be related to both restricted and excess eating, with the latter more common among those who are already obese [185]. It is possible that excess eating as a regulation mechanism of affective states is more common with environmental cues, with meta-analysis

previously demonstrating moderated behaviours in response to affective state in the presence of environmental cues [107].

Differences in the relation between emotional and behavioural symptoms and obesity across generations further highlights likely environmental interactions. In the MRC NSHD which follows cohort members born in 1946, females with internalising symptoms in childhood had the lowest BMI aged 15 [39]. At age 15 the NSHD cohort were experiencing the end of post-war food rationing, having limited ability to regulate their emotions through overeating. In midlife, when the cohort was living in a more obesogenic environment, females who had experienced internalising symptoms in childhood had faster rates of increase in BMI, and higher BMI at age 53 compared to those with no childhood symptoms. In contrast, in the MCS where children were born in 2000/2 and have lived their whole life in an obesogenic environment, depression and low self-esteem were associated with obesity at age 11 [40]. Emotional regulation and behavioural problems in earlier childhood were also found to be associated with obesity [41, 42].

## 2.5 Summary

This review has drawn on expansive literature covering inequalities in obesity and highlights a number of important frameworks and theories that can be adopted to understand inequalities in obesity. However, a number of gaps were highlighted. Much of the research conducted on inequalities in obesity relies on anthropometric measures such as BMI, therefore highlighting a need for a greater understanding of the relationship between SEP and body composition. Reviews of inequalities in anthropometric measures have demonstrated inequalities in obesity that have increased in recent generations. There is a gap in the literature to systematically review inequalities in body composition.

This literature review has also identified gaps in knowledge relating to the role area-level measures of SEP play in inequalities in body composition, and to understand whether the effects of area-level measures are different to those of individual-level SEP. Additionally, the life course approach highlights the need to understand trajectories in health outcomes. There is a gap to explore inequalities in trajectories of body composition in relation to both area- and individual-level disadvantage in early life, and to understand when in the life course inequalities first emerge.

This review has brought together several different perspectives to explore inequalities in body composition. Much previous work investigating mediating factors between SEP and adiposity has focused on health behaviours, such as physical activity and diet. There is a gap in knowledge regarding the role that emotional and behavioural problems in children and adolescents play in mediating associations between SEP and body composition. This literature review finds evidence to support associations between SEP and internalising and externalising symptoms, whereby those in disadvantaged socioeconomic circumstances have a greater risk of emotional and behavioural problems. The literature review also demonstrates associations between both sets of symptoms and obesity, with the relationship explained through affect regulation models. A plausible mediating pathway is highlighted from SEP to body composition, through internalising and externalising symptoms. However, the mediating path has not previously been directly tested.

Finally, the literature review explored the importance of the obesogenic environment in the development of obesity. A gap was highlighted emphasising the need to look at area-level deprivation as an indicator of social and cultural environments alongside measures of the physical environment. Furthermore, the literature review finds supporting evidence of interactions between environmental characteristics and mental state but highlighted a lack of understanding about how this relates to body composition. The literature reviewed demonstrated how elements of the environment both may simultaneously influence affective state but also the cues and opportunities that allow people to regulate their affective state. However, there is a gap in understanding this in a broader sense of inequalities in body composition, that is addressed in this thesis.

# Chapter 3 Aims and Objectives

The overall aim of this PhD is to understand life course inequalities in body composition and the role of the relationship between the individual and the environment – measured through area deprivation, fast-food density, density of green space and domestic gardens – in the manifestation of these inequalities. This PhD aims to achieve this in two ways: firstly, by describing the state of inequalities in body composition; secondly, by conducting analytic epidemiology to explore the relation of individual psychological characteristics and the environment.

## 3.1 Objective One: Systematically Review the Literature on Socioeconomic Inequalities in Body Composition

The first objective of my PhD is to summarise the evidence linking SEP and obesity using directly measured body composition in place of more traditional anthropometric measures. By conducting a systematic review of the literature, I aim to test the following hypotheses:

**Hypothesis 1:** *There are social inequalities in fat mass (FM) and fat-free mass (FFM), with less advantaged SEP being related to higher FM and lower FFM in high-income countries, and associations in the opposite direction in middle- and low-income countries.*

**Hypothesis 2:** *Social inequalities in FM and FFM vary between generations, with greater evidence of inequalities in FM and FFM among those born more recently.*

**Hypothesis 3:** *Less advantaged SEP is related to higher FM and lower FFM more frequently in females compared to males in HICs.*

## 3.2 Objective Two: Assess the Degree of Socioeconomic Inequality in Directly Measured Body Composition in Childhood and Adolescence using a Contemporary UK Cohort.

The second objective of my PhD is to better understand the current inequalities in body composition in the UK using a contemporary nationally representative cohort of adolescents. In particular, by using early life SEP to understand life course inequalities in trajectories of body composition in the UK Millennium Cohort Study (MCS), I aim to test the following hypotheses:

**Hypothesis 4:** *Disadvantaged SEP in infancy is related to higher FM, lower FFM and a higher FM:FFM ratio among children and adolescents in the UK at age 7, and these inequalities widen from age 7 through adolescence to age 17.*

**Hypothesis 5:** *Inequalities in FM, FFM and FM:FFM are greater in females compared to males.*

**Hypothesis 6:** *Area-level inequalities in body composition trajectories are observed, and are not explained by inequalities in family-level SEP.*

### 3.3 Objective Three: Assess the Appropriateness of using the Strengths and Difficulties Questionnaire to Measure Internalising and Externalising Symptoms as Mediators, in a Contemporary Cohort of Children in the UK

The third objective of my PhD is to assess the appropriateness of using the Strengths and Difficulties questionnaires (SDQ) in the MCS to measure internalising and externalising symptoms in further analysis, in particular mediation analysis. I also seek to demonstrate that internalising and externalising symptoms are appropriate to use in multi-group analysis by demonstrating the similarity in which constructs are measured. By conducting confirmatory factor analysis, I aim to test the following hypothesis:

**Hypothesis 7:** *The Strength and Difficulties Questionnaire captures internalising and externalising latent traits in a UK population of adolescents and the latent factors are appropriate to use as mediators in further analysis.*

**Hypothesis 8:** *The latent constructs of internalising and externalising symptoms are measured in the same way in boys and girls, and across different environmental conditions (i.e high and low deprivation, high and low green space, domestic garden and fast-food outlet density).*

### 3.4 Objective Four: Assess the Mediating Role of Internalising and Externalising Symptoms in Understanding Inequalities in Body Composition and Body Mass Index, and to Explore Moderating Effects by the Environment.

The fourth objective of my PhD is to assess the role that internalising and externalising symptoms play in the development of social inequalities in body composition (and BMI) among adolescents in the UK, and to investigate if this mediating role differs dependent on environmental conditions. Using the MCS, I aim to test the following hypotheses:

**Hypothesis 9:** *Social inequalities in body composition (and BMI) among children and adolescents in the UK are mediated by internalising and externalising symptoms, so that those in disadvantaged circumstances exhibit higher levels of these symptoms, which in turn are related to less healthy body composition (higher FM and lower FFM).*

**Hypothesis 10:** *Environmental characteristics modify the mediating effect of internalising and externalising symptoms among children and adolescents in the UK, such that those experiencing a more obesogenic environment show greater mediation by internalising and externalising symptoms, such that the association between symptoms and less healthy body composition is strongest in the most obesogenic areas.*

# Chapter 4 Methodology

This Chapter outlines the dataset and methods used in this thesis. The Millennium Cohort Study (MCS) was used, as it represents a contemporary cohort of children and captures the lives of young people in the UK today. The MCS is the fourth nationally representative cohort that has followed children from birth, or near birth, across their lifetime. As outlined in Chapter 1, the other three cohorts have followed children born in 1946, 1958 and 1970 whilst the MCS has followed children born in 2000 to 2002. The MCS, along with the other British Birth Cohorts, allow understanding of the lives of people today, and are particularly well placed to examine changing trends between generations.

A large wealth of data have been collected in the MCS since infancy, making it well suited to answer social and biomedical questions. The MCS is one of few nationally representative cohort studies that has repeated measures of body composition, allowing change in inequalities of body composition to be modelled. This therefore allows a life course approach to be adopted in understanding how and when inequalities emerge. Further, the MCS has consistent and detailed measures of the environment in which participants live, something that is traditionally difficult to collect for cohort studies and is lacking in the other British Birth Cohorts during childhood and adolescence. Finally, psychological assessments have been collected using a validated measurement scale throughout childhood in the MCS, allowing internalising and externalising symptoms to be measured and used in analysis. For these reasons, the MCS is used to address the hypotheses set out in Chapter 3.

This Chapter provides an introduction to the dataset, describes the variables adopted and the distribution of the sample by key variables. This chapter also discusses challenges of missing data and provides a brief introduction to statistical methods that have relevance across the remaining chapters.

## 4.1 Introduction to the Dataset: The Millennium Cohort Study

The MCS is an on-going observational cohort study that follows the lives of children born at the turn of the Millennium [186]. The MCS is a multidisciplinary study with extensive measures of child health, development, social circumstances as well as having collected biological samples, accelerometry and time use diaries.

The MCS sample is a nationally representative sample of the UK population taken from all four countries of the UK: England, Wales, Scotland and Northern Ireland. Cohort members were recruited from a sample of children born in a 16-month period at the start of the millennium, in part to take into account seasonality [187]. In England and Wales, cohort members were eligible for recruitment if they were born between 1<sup>st</sup> of September 2000 and 31<sup>st</sup> August 2001, whilst in Scotland and Northern Ireland the sample was drawn from cohort members born between 24<sup>th</sup> November 2000 and 11<sup>th</sup> January 2002.

At the first follow-up, or “sweep”, conducted in 2001, children were recruited to the sample when they were 9 months of age if they were eligible for the government’s child benefit scheme. At the time of

recruitment, the scheme had almost universal coverage, with the exception of a small subset of children, such as those seeking asylum [186]. A total of 18,551 families with children born between 2000 and 2002 in the UK were recruited [186]. Because of the inclusion of 256 set of twins and 10 sets of triplets, the number of children included at 9 months was 18,818 [187]. At the second sweep when cohort members were age 3, recruitment of 692 new eligible families occurred, bringing the total number of families to 19,243 and children to 19,517.

As of 2021, a total of seven sweeps have taken place. The first was at nine months, followed by sweeps at age 3, 5, 7, 11, 14 years and the most recent at age 17 where 10,757 children (10,625 families) took part.

#### 4.1.1 Stratification Characteristics

The MCS was designed to be both representative of the UK population, and to provide usable data on sub-groups of children [188]. These sub-groups included children from each of the four countries in the UK (England, Scotland, Wales and Northern Ireland), children living in advantaged and disadvantaged circumstances, and children of ethnic minorities. The MCS therefore adopted methods of random selection in areas of the UK stratified by the above criteria [188].

Ideally, stratification of disadvantaged and ethnic minority children would have been done at the individual level, but this information was only available at the electoral ward level. Therefore, in England, electoral wards were stratified into “ethnic minority”, “disadvantaged” and “advantaged”, whilst in the remaining countries of the UK only the advantaged and disadvantaged strata were used [188]. This was because of the small number of ethnic minority groups in Wales, Scotland and Northern Ireland, which at the time was equivalent to 1% of the population [187].

The ethnic stratum was determined as those wards where more than 30% of the children living in the ward were an ethnic minority according to the 1991 census. The disadvantaged stratum was determined as those, other than the ethnic minority stratum, that were rated among the 25% poorest wards according to the Child Poverty Index for England and Wales [189]. The advantaged stratum was all other wards not included in the ethnic or disadvantaged stratum. Because both the ethnic stratum and disadvantaged stratum were area-level measures, the grouping of strata may not accurately represent the situation of households living in that stratum. For example, it was demonstrated in 1998 that 37% of households living under poverty in England were in “advantaged” stratum whilst 54% were in “disadvantaged” and 10% were in “ethnic” stratum [188].

Oversampling took place in the disadvantaged and ethnic minority strata [190]. A breakdown of the sample according to stratum is shown in Table 4-1. Approximately 47% of the sample were selected from a disadvantaged stratum, 13% from an ethnic stratum and the remaining 39% were selected from an advantaged stratum.



**Table 4-1.** Stratification Characteristics of the Millennium Cohort Study

<b>Stratum</b>	<b>N</b>	<b>%</b>
<i>England – Ethnic</i>	2,591	13.5
<i>England – Disadvantaged</i>	4,805	25.0
<i>England – Advantaged</i>	4,828	25.1
<i>Wales – Disadvantaged</i>	1,928	10.0
<i>Wales – Advantaged</i>	832	4.3
<i>Scotland – Disadvantaged</i>	1,191	6.2
<i>Scotland Advantaged</i>	1,145	6.0
<i>Northern Ireland – Disadvantaged</i>	1,200	3.8
<i>Northern Ireland – Advantaged</i>	723	6.2
<b>Total Ethnic</b>	<b>2,591</b>	<b>13.5</b>
<b>Total Disadvantaged</b>	<b>9,124</b>	<b>47.4</b>
<b>Total Advantaged</b>	<b>7,528</b>	<b>39.1</b>

**Table 4-1 Footnote:** Ethnic stratum are wards where 30% of the population are from an ethnic minority background. Disadvantaged Stratum are wards other than ethnic minority stratum, that are rated as the 25% poorest. Ethnic stratum was only included in England, due to small numbers of ethnic minorities in the other countries of the UK.

#### 4.1.2 Respondents

At sweeps 1, 2 and 3, the target responders were the main parent and their partner. From sweep 4 (age 7) onwards, the cohort member was interviewed as well as the parents. Typically, the main respondent was the natural mother, with this being the case for 98% of the main respondents at sweep 1 [190]. However, this was not always the case, such as where the partner was the primary caregiver, the mother was unwilling to participate but the partner was, the father was the only natural parent, or the cohort member did not live with natural parents [187]. Typically, the partner interview was the natural father in sweep 1, with 99.6% of the partner respondents identified as the natural father [190]. Although the main respondent was not strictly the same across sweeps, the same person, typically the natural mother, was usually selected as the main respondent across all sweeps, because the same selection process was adopted [187, 190]. The same was true of the partner interview who was typically the natural father across sweeps [190].

There were three different interviews that could be conducted with the parents, of which households received up to two. These were the main parental respondent interview, the partner interview and the proxy partner interview. The latter was used with the main respondent about the partner if they were unavailable to be interviewed themselves [187]. The partner interview was a subset of questions asked to the main respondent, and the proxy interview was a subset of the questions asked to the partner [187].

### 4.1.3 Response Rates

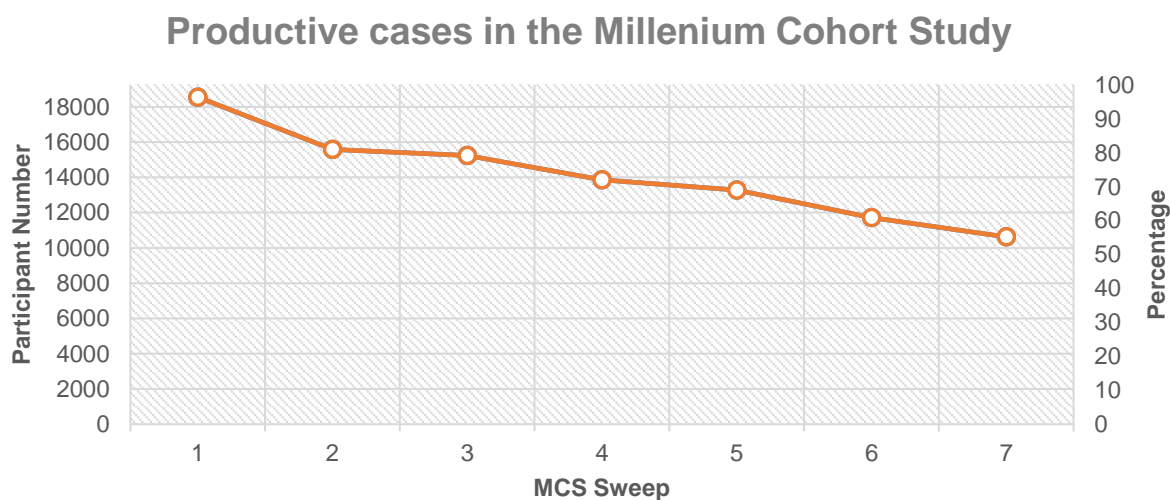
In total 9,130 families have responded to all sweeps of the MCS, whilst approximately 5,500 have missed one or more of the sweeps they were eligible for [191]. Reasons for unproductive participation were predominantly due to refusals to participate or due to the surveys not being issued, which occurred after two consecutive unproductive sweeps [192]. By sweep 7 the number of surveys no longer issued was 4,747, equivalent to 24.7% of the total sample [191, 193]. Other reasons for unproductive participation are because of participants moving and no longer being traceable, being ineligible either due to death or emigration, and because of non-contact, where it has not been possible to make contact with the family [191, 193, 194]. A full breakdown of reasons for unproductive participation across all seven sweeps of the MCS is shown in Table 4-2. In total, productive participation declined across sweeps (Figure 4-1), so that in the 7<sup>th</sup> sweep only 10,625 families were productive, equivalent to 55.2% of the sample. Of those that participated in the most recent sweep, 8,942 households were fully productive and the remaining 1,683 households were partially productive [191].

**Table 4-2.** Number of Productive and Unproductive Cases across Sweeps 1-7 in the Millennium Cohort Study, and Reasons for Non-Participation

	MCS1		MCS2		MCS3		MCS4		MCS5		MCS6		MCS7	
	N	%	N	%	N	%	N	%	N	%	N	%	N	%
<b>Productive Sample</b>	18,551	96.4	15,590	81.0	15,246	79.2	13,857	72.0	13,287	69.0	11,726	60.9	10,625	55.2
<b>Unproductive (Total)</b>			3,653	19.0	3,997	20.8	5,386	28.0	5,956	40.0	7,517	39.1	8,618	44.8
<i>Refusal</i>			1,739	9.0	2,315	12.0	1,811	9.4	2,195	11.4	3,029	15.7	2,898	15.1
<i>Ineligible</i>			167	0.9	300	1.6	126	0.65	78	0.4	45	0.2	29	0.2
<i>Moved (untraceable)</i>			686	3.6	546	2.8	706	3.7	388	2.0	428	2.2	348	1.8
<i>Non-Contact</i>			930	4.8	546	2.8	123	0.6	438	2.3	75	0.4	234	1.2
<i>Other</i>			131	0.7	290	1.5	408	2.1	6	0.0	112	0.6	362	1.9
<b>Not Issued</b>							2,212	11.5	2,851	14.8	3,828	19.9	4,747	24.7
<b>Not Entered</b>	692	3.6												
<b>Total</b>	19,243	100	19,243	100	19,243	100	19,243	100	19,243	100	19,243	100	19,243	100

**Table 4-2 Footnote:** MCS1 to MCS7 represent sweeps 1 through to 7, capturing ages 9 months to 17 years. Breakdown of response for MCS7 and reason for non-participation is excluding the online follow-up, where total productive was 10,799. Productive and unproductive cases are defined by families, as opposed to children (Total children N=19,517). Abbreviations: MCS – Millennium Cohort Study.

**Figure 4-1.** Attrition in the Millennium Cohort Study (MCS), Demonstrated by Total Number of Productive Cases and Productive Cases as Percentage of the Total Sample across MCS Sweeps 1-7.



**Figure 4-1. Footnote:** MCS Sweeps 1 to 7 correspond with ages 9 months, and ages 3, 5, 7, 11, 14 and 17 years respectively. Productive cases are number of families that participated in each sweep, as opposed to children. Abbreviations: MCS – Millennium Cohort Study.

#### 4.1.4 Ethical Considerations

Ethics approval was obtained from the National Health Service Research Ethics Committee (REC) up to age 14 [195], and the National Research Ethics Service (NRES) REC at age 17 [191]. Informed parental consent was obtained in advance of data collection up to age 14. Leaflets and letters detailing what participation would involve were sent to the parents ahead of data collection, and written consent was obtained. Where consent was obtained by parents for their child to participate, agreement by the child was also sought [195]. Written consent was also obtained for gathering further information from alternative sources, such as health, education and economic record and from teachers [195]. At age 17, verbal informed consent was provided by the cohort members, and interviewers did not need parental consent to approach cohort members as they were aged 16 years or older [191]. To ensure cohort members were fully informed on what their participation would entail, they were provided with a consent booklet before participating [191]. Across all sweeps participants were able to refuse to participate in any element of data collection or withdraw from the study at any time [191, 195].

## 4.2 Key Measures and Variables

### 4.2.1 Outcome Measures: Body Composition and Body Mass Index

Both height and weight have been measured since 3-years of age, whilst body composition was measured at ages 7, 11, 14 and 17 years through foot-to-foot BIA using Tanita Scales. BIA, is a non-invasive and low-cost device that estimates body composition by sending a low electrical current

through the body from one foot to the other, allowing total body water to be estimated [196]. The electrical signal moves faster through water, which is found at higher concentrations in FFM, and slower through FM. The resistance that occurs when moving through fat tissue, also known as impedance, can be used to estimate the proportion of fat.

Tanita scales output data as FM%, using population equations for a more accurate estimate. Equations used to calculate FM% are those used by the manufacturer (Tanita) [197], and are derived from large, multi-ethnic population studies. Readings from Tanita Scales have been found to have concordance with both DXA (which is used as the reference method) and hydrodensitometry, with results of BIA within +/-5% of the values obtained by these other methods [197, 198]. In a study that evaluated this in White and African-American children (aged 5 to 18 years) compared to values from DXA, the average error and absolute error was found to be smaller still (-1.0% and 3.9% respectively) [198].

Both weight (kg) and FM% were ascertained using Tanita Scales (Bf-522W), carried out by trained interviewers to standardised protocols. Scales were calibrated before use, and placed on a firm uncarpeted surface where possible, otherwise this was noted by the interviewers [191]. Cohort members were asked to wear light indoor clothing, to remove any bulky items such as watches and belts, and to remove socks and shoes. Cohort members that were pregnant or had an internal electric device were unable to have their body fat measured [191]. Age, height, and gender were entered into the scales to allow calculation of FM%. Weight could be measured in a "weight only" mode where it was not necessary for height to be entered into the scales. In the weight only mode, it was possible to take measurement for those individuals whose body fat measurements were not appropriate as no electrical current was used [191]. Cohort members were asked to step onto scales barefoot, to allow the electric current to move around their body through their feet. Weight was measured to the nearest 0.1kg and body fat to the nearest 0.1%.

Height was measured using a Leicester stadiometer, consisting of a baseplate, measuring rod and head-plate, and interviewers were trained in how to use the equipment before taking measurements [199]. Height was measured in standing position by the interviewer by lining up the Frankfort Plane (a line running through the ears and the bottom of the eyes) so that it was horizontal and parallel to the floor [199]. The child was gently stretched by the interviewer to the maximum height, ensuring that they did not lift their feet, and the head plate was lowered to the height of the child. Height was measured correctly if the child did not need to duck to leave the stadiometer after relaxing and breathing out [199]. Measurement was taken once the child stepped off the stadiometer, and was recorded in meters and centimetres, rounded down to the nearest full millimetre.

Equipment was set up on a firm surface, uncarpeted where possible, and ideally positioned against a wall to ensure the stadiometer did not change position during the measurement [191]. Cohort members were requested to remove socks, shoes, glasses, head accessories and to undo any hairstyles that could interfere with measurement, and a record was made by the interviewer of

anything that could have impacted the measurement e.g. presence of a top-knot, turban or the cohort members posture [191].

Using FM%, total body weight (BW) and Height (Ht) the measures of FM, FFM and FM:FFM ratio were calculated [200, 201]. FM in kg was calculated from FM% and body weight ((FM%/100) x weight) and FFM was calculated by subtracting FM from total weight. The ratio of FM to FFM was also calculated (FM/FFM).

#### *4.2.1.1 Indexing Body Composition Measures using the Benn Index*

Throughout this thesis FMI, FFMI and the ratio of FM:FFM are used as the primary outcome measures in body composition analysis. These measures were chosen as indexed measures are preferable to raw or percentage measures as they take into account height and are able to be interpreted independently of each other.

The rationale behind indexing weight to height is to remove the correlation between the two. This takes into account the fact that taller people are typically heavier and shorter people are typically lighter. This is usually done by using a Benn parameter (B) of 2 in the equation  $\text{kg}/\text{m}^B$ . However, a Benn parameter of two does not remove the correlation with height, as the parameter, B, that removes the correlation with height differs by age, sex, ethnicity and population and has also changed over time [202]. To calculate the correct value of B, log weight (kg) is regressed on log height (m), and the coefficient from linear regression is used. For analysis in this thesis, FMI, FFMI and BMI were calculated using the Benn index, with the parameters calculated separately at each age and by gender, shown in Table 4-3. The formulae used to calculate FMI, FFMI and BMI are:

$$FFMI = FFM \text{ (kg)} / (Ht \text{ (m)})^B$$

$$FMI = FM \text{ (kg)} / (Ht \text{ (m)})^B$$

$$BMI = BW \text{ (kg)} / (Ht \text{ (m)})^B$$

**Table 4-3.** Estimates of the Benn Parameter for FMI, FFMI and BMI in the Millennium Cohort Study

<b>Body Composition</b>	<b>Males</b>		<b>Females</b>	
	<b>Age (Years)</b>	<b>Benn Parameter</b>	<b>Age (Years)</b>	<b>Benn Parameter</b>
<i>FMI</i>	7	3.88	7	4.31
	11	4.76	11	4.55
	14	2.99	14	2.89
	17	1.97	17	3.35
<i>FFMI</i>	7	2.43	7	2.43
	11	2.63	11	2.57
	14	2.58	14	2.21
	17	2.07	17	1.41
<i>BMI</i>	7	2.79	7	2.89
	11	3.12	11	3.07
	14	2.61	14	2.37
	17	2.01	17	1.87

**Table 4-3 Footnote:** *Benn Parameter was calculated by using the regression coefficient from log weight (kg) regressed on log height (m). Abbreviations: FMI – Fat mass Index; FFMI – Fat-Free Mass Index; BMI – Body Mass Index.*

#### 4.2.2 Measures of Socioeconomic Position

The IMD is a measure of relative deprivation used in England, with similar measures used in the other countries of the UK. The measure splits England up into Lower Layer Super Output Areas (LSOA), small areas or neighbourhoods with an average population size of 1,500 individuals. In 2019 there were 32,844 LSOAs, and each of these was ranked from the most deprived to the least deprived [203]. The ranking of LSOAs occurs across 39 indicators, which are organised into 7 distinct domains: income; employment; health deprivation and disability; education, skills training; crime; barriers to housing and services; living environment [203]. The income and education domains have a higher weighting in the overall deprivation score than the other domains, which is based on both academic literature around the importance of different domains to overall deprivation, as well as the robustness of indicators used [203]. The IMD is available in all sweeps in the MCS.

Ten IMD groups were created using deciles based on rank. Data on IMD was linked to the address at interview and the LSOA. This was done individually by country, using IMD measures following the Office of the Deputy Prime Minister (ODPM) Indices of Deprivation 2004 in England; Welsh Assembly IMD 2005 in Wales; Scottish Assembly IMD 2004 in Scotland; Northern Ireland Statistics and Research Agency (NISRA) Multiple Deprivation Measure 2005 in Northern Ireland [204]. One IMD variable was created by merging the grouped IMD measures from each individual country, to help maintain sample size and ensure representation of the whole of the UK. This is a similar approach to that used previously [205], and those that have combined IMD across countries using standardised ranks [206]. In combining IMD in this way, assumptions were made that the relative position is similar

across the countries of the UK, and that the highest and lowest relative deprivation deciles are similar. This appears to be a reasonable assumption since work developing adjusted IMD scales across the UK found small changes in IMD ranking (2.6% to 10.3%) compared to the original country deprivation measures, and that rank correlation coefficients between adjusted and original IMD scales were high (0.97 to 0.94) [207]. For the sweep 1 measure, this was re-grouped into 5 categories ranging from the 20% most deprived to the 20% least deprived.

In this thesis, income is used as the primary measure of family-level SEP. Income was selected because it is a direct measure of material resources that is related to both education and occupational class and is ordered on a continuum allowing for investigation of how incremental changes in SEP are related to corresponding changes in body composition and BMI. Both parental occupation and education were used in descriptive statistics and as covariates in models. As was the case for the IMD, for sweep 1 measures of SEP, data from sweep 2 was used for new joiners.

Family income was measured using Organisation for Economic Co-operation and Development (OECD) equivalised quintiles of family income, derived by the MCS survey team at the Centre of Longitudinal studies (CLS) [190]. Income has been collected in a number of different ways across sweeps, with the main income question collecting a banded measure on income with additional information collected on a range of other income measures, e.g., benefit receipts. Banded income was collected by showing respondents cards with different weekly, monthly and yearly bands for total take-home income across all the potential income sources, after tax was deducted [204]. Different bands were used for lone parents and two parent households. Modified OECD scales were then used to equivalise banded income, so that it was relative to the needs of a childless couple, who's need was equal to 1 [204]. OECD relative income was then split into income fifths by the MCS survey teams, ranging from 1 (lowest income fifth) to 5 (highest income fifth).

Occupation was classified using the National Statistics Socio-Economic Classification (NS-SEC), which categorises occupation into five groups: 1) semi-routine and routine; 2) low supervisors and low technical occupations; 3) small employers and self-employed technical; 4) intermediate; and 5) managerial and professional. Both the main and partner respondents were asked about their occupation. The highest occupation of the household was selected, with managerial and professional occupations being taken as the highest, and semi-routine and routine as the lowest. If one parent was unemployed, the occupation of the other parent was selected for the household.

Education was obtained from both the main respondent and the partner. Respondents were asked about their highest academic or vocational qualification, and this was converted to a National Vocational Qualification (NVQ) scale ranging from "NVQ level 1" to "NVQ level 5", with an additional group for overseas qualifications and no qualifications. A breakdown of the equivalent qualifications according to the NVQ scale is shown in Table 4-4. The highest NVQ level of the household was taken, with NVQ 5 considered the highest level and NVQ 1 the lowest. Where one parent had no qualifications, their partner's was selected. When one parent had an overseas qualification, their

partner's qualification was selected, unless the partner had no qualifications, in which case the overseas qualification was used.

**Table 4-4.** National Vocation Qualification Levels and Equivalent Academic and Vocational Qualifications

<b>National Vocation Qualification</b>	<b>Equivalent Academic Qualification</b>	<b>Equivalent Vocational Qualification</b>
No Qualification	None of these qualifications	None of these qualifications
Overseas Qualification	Other academic qualification (including from overseas)	Other vocational qualification (including from overseas)
NVQ level 1	GCSE grades D-G	NVQ / SVQ / GSVQ level 1
NVQ level 2	O level / GCSE grades A-C	Trade apprenticeships or NVQ / SVQ / GSVQ level 2
NVQ level 3	A / AS / S Levels	NVQ / SVQ / GSVQ level 3
NVQ level 4	First Degree (i.e undergraduate) or Diploma in Higher Education	Professional qualification at degree level or nursing or other medical qualification
NVQ level 5	Higher degree (i.e PhD or Master's Degree)	N/A

**Table 4-4 Footnotes:** Description of different academic and vocational qualifications equivalent to NVQ 1-5. Abbreviations: NVQ – National Vocational Qualification; GCSE - General Certificate of Secondary Education; SVQ – Scottish Vocational Qualifications; GSVQ – General Scottish Vocational Qualifications; O Level – Ordinary Level; A / AS / S Level – Advanced / Advanced Subsidiary / Scholarship Levels; PhD – Doctor of Philosophy.

### 4.2.3 Strengths and Difficulties Questionnaire: Internalising and Externalising Symptoms

The SDQ is a behavioural screening questionnaire, designed for individuals aged 4 to 17, that assesses behaviour and mental wellbeing in children [208]. The SDQ is composed of 25 items assessing psychological attributes, which are rated on a 3 point scale of “not true”, “somewhat true”, “certainly true”. The 25 items are divided into 5 sub-scales, made up of five items each:

1. Emotional symptoms
2. Conduct Problems
3. Hyperactivity/ Inattention
4. Peer relationship problems
5. Pro-social behaviour

These five scales are combined to give a total difficulties score (scales 1 – 4), an internalising problems score (scales 1 and 4) and an externalising problems score (scales 2 and 3). The full list of items and how they group into each scale is shown in Table 4-5, including the abbreviated name (in brackets) adopted in this thesis for each item.

In the MCS, psychological wellbeing has been assessed at sweeps 2, 3, 4, 5 and 6 using parent-reported SDQ. At age 17, the young person themselves completed the SDQ. This thesis uses the



SDQ at sweep 5 (age 11). Positively worded items were reversed when combining scales, as indicated in Table 4-5.

**Table 4-5.** Variable Items in the Strengths and Difficulties Questionnaire in the Millennium Cohort Study

1. Emotional Symptom Scale	2. Conduct Problems	3. Hyperactivity Scale	4. Peer Problems	5. Pro-Social Scale**
1. Complains of headaches/stomach aches/sickness ( <i>Complains</i> )	1. Often has temper tantrums ( <i>Anger</i> )	1. Restless, overactive, cannot stay still for long ( <i>Restless</i> )	1. Tends to play alone ( <i>Alone</i> )	1. Considerate of others' feelings ( <i>Considerate</i> )
2. Often seems worried ( <i>Worried</i> )	2. Generally obedient* ( <i>Obedience</i> )	2. Constantly fidgeting ( <i>Fidget</i> )	2. Has at least one good friend* ( <i>Friend</i> )	2. Shares readily with others ( <i>Shares</i> )
3. Often unhappy ( <i>Unhappy</i> )	3. Fights with or bullies other children ( <i>Aggression</i> )	3. Easily distracted ( <i>Attention</i> )	3. Generally liked by other children* ( <i>Liked</i> )	3. Helpful if someone is hurt, upset or ill ( <i>Helpful</i> )
4. Nervous or clingy in new situation ( <i>Anxiety</i> )	4. Lies or Cheats ( <i>Lies</i> )	4. Can stop and think before acting* ( <i>Impulse</i> )	4. Picked on or bullied by other children ( <i>Bullied</i> )	4. Kind to younger children ( <i>Kind</i> )
5. Many fears, easily scared ( <i>Fear</i> )	5. Steals from home, school, elsewhere ( <i>Steals</i> )	5. Sees tasks through to the end* ( <i>Task</i> )	5. Gets on better with adults ( <i>Adults</i> )	5. Often volunteers to help others ( <i>Volunteers</i> )

**Table 4-5 Footnote:** \* Indicates positively worded items, that items were reversed when combining to create sub scales. \*\* All items on the Pro-Social Scale are positively worded, so no items in the pro-social scale were reversed. Scale 1 and 4 can be combined to create an internalising problem score. Scale 2 and 3 can be combined for an externalising problem score. Scales 1 to 4 are combined for total difficulties score. Names in brackets indicates abbreviated name used for each question.

#### 4.2.4 Environmental Characteristics

Measurements of the environment were taken from sweep 5, when cohort members were 11 years old. Measures of the environment selected were green spaces, domestic gardens, fast-food outlet density. Additionally, the IMD was also used as a proxy for environmental conditions (described above, Section 4.2.2). All environmental variables were grouped into three categories, from the most to least deprived or dense, to maintain sample size and statistical power in each group.

There are a number of challenges when measuring neighbourhood and area characteristics and linking these to individual outcomes. The ecological fallacy, whereby assumptions are made about the individual based on the area in which they live or by aggregate group data [209], leads to incorrect conclusions being drawn on individual experiences and associations. Selection bias may also occur,

whereby the selection of individuals or families into neighbourhoods is not independent from variables under study, such as individual-level SEP [210]. Careful consideration of individual-level characteristics that drive neighbourhood differences is therefore required. This is discussed in more detail in the selection of covariates below (Section 4.2.5, page 59). By adjusting for individual-level characteristics that may drive selection into neighbourhoods, the area-level effects can be correctly interpreted.

#### *4.2.4.1 Green Spaces and Domestic Gardens*

Green spaces and domestic garden data are available at LSOA level in England only, and was obtained from the Generalised Land Use Database (GLUD) [211]. The GLUD uses high geographical resolution in England to classify land use into nine categories, including green spaces and domestic gardens. GLUD provides an indicator of the percentage of total area represented by green space in each LSOA. Green space excluded domestic gardens and included both public and private spaces greater than 5m<sup>2</sup>. Because GLUD catches very small green spaces, it provides an overall indicator of “greenness” within a LSOA. In the MCS, both LSOA domestic gardens and green spaces (excluding domestic gardens) are categorised into 10 groups defined by deciles.

Also available in the MCS is ward level green space for the whole of the UK, which is estimated through a combination of GLUD data and from the Coordination of Information on the Environment (CORINE; EEA, 2000) [211]. CORINE uses remotely sensed satellite imagery for the whole of the UK to provide a land cover dataset from 2000. Unlike GLUD which is sensitive to smaller green spaces, CORINE only maps larger areas, with the smallest area mapped in the UK being 1 hectare. Therefore, CORINE is only sensitive to larger green spaces such as parks. In order to obtain a UK wide ward level indicator of green space, a regression model was developed to predict GLUD percentage of green spaces in England based on CORINE ward level data and population density from the 2001 census [211]. This model successfully predicted GLUD deciles in England and was therefore expanded to cover the whole of the UK to achieve UK-wide ward level green space deciles. To maintain sample size and ensure the sample is representative of the whole UK, ward level green spaces were selected in the analysis instead of LSOA-level.

For analysis, the percentage green space and domestic gardens measures were regrouped into high deprivation (those in the 30% most deprived areas), low deprivation (those in the 30% least deprived areas) and medium deprivation (the remaining 40%).

#### *4.2.4.2 Fast-food Density*

Fast-food outlet density was measured by the number of fast-food and takeaway outlets within 400, 800 and 1,600 meters of the cohort member’s address. Fast-food density was measured using the Ordnance Survey Point of Interest (PoI) data, whereby the MCS data were linked to points of interest based on their place of residence at the time of interview, and is available for sweep 4 to sweep 6 [212]. In the MCS, PoI data are only available for cohort members residing in Great Britain (countries of the UK excluding Northern Ireland).

Ordnance Survey Integrated Transport Network (ITN) data along with Geographic Information Systems (GIS) were used to create buffers (400, 800 and 1,600 meters) around cohort members houses, and counts of food outlets were created for each buffer. New categories were derived for specific food outlets, including fast-food outlets. The current analysis uses fast-food and takeaway outlet density within the 800m buffer at age 11 (sweep 5). Those with 20 or more fast-food chains within 800m were grouped as “20+” due to the low numbers of cohort members living near more than 20 fast-food outlets. The variable for fast-food density was regrouped into low density (0-2 fast-food outlets), medium density (3-7 outlets) and high density (8+) of fast-food outlets.

#### 4.2.5 Covariates

Ethnicity was included as a covariate in the analyses, as levels of obesity have previously been shown to differ by ethnicity [213], and individuals from ethnic minorities are more likely to live in disadvantaged circumstances [214]. Ethnicity was reported by the main respondent for the cohort member at sweep 1 with replacement for late joiners at sweep 2. Ethnicity was categorised according to the 2001 UK census categories. In these analyses, six groups are used: 1) White; 2) Mixed; 3) Indian; 4) Pakistani and Bangladeshi; 5) Black and Black British; 7) Other Ethnic Group.

Sex was included as a covariate in the analyses, as inequalities in adiposity have previously been shown to differ by sex. Although sex is not a confounder between SEP and obesity, as sex is unrelated to SEP, it is considered a competing exposure as it is related to body size. In the causal literature, adjustments can be made for confounders, but also competing exposure, with adjustment of the latter improving precision of the estimates [215]. Sex was reported by the main respondent at sweep 1 as either 1) male or 2) female, with replacement at sweep 2 for late joiners. Age was also included in analyses in this thesis to improve precision of estimates, as age is related to body size. Age was reported at the time of interview and was reported to the nearest 10<sup>th</sup> of a year.

For analyses with area-level SEP, individual level markers of SEP (family income, parental occupation and education) were controlled for, to address problems related to selective sorting. Individuals with more advantaged individual circumstances are likely to live in, or move to, more affluent areas, and therefore it is possible associations with area-level indicators are actually driven by individual circumstances and not neighbourhood effects. Therefore, adjustments were made for individual-level SEP to attempt to separate the effect of area-level from individual-level SEP.

Additionally, MCS stratification characteristics were included as a covariate in all models as recommended by CLS [204], to account for the fact that families were selected from electoral ward levels, that relied on aggregate data. Ethnicity was also controlled for in all models to address selective sorting, as ethnicity is also likely to influence selection into neighbourhoods and was also used to determine ward level selection of families into the MCS. Individual markers of SEP were included in models as predictors or covariates.

Few other covariates were considered in statistical models. There are a number of variables that are socially distributed, and therefore likely to be related to family SEP, that may also be predictors of childhood and adolescent body composition (e.g. birth weight, childhood growth and development,

maternal smoking and depression). However, they are unlikely to be confounders and more likely to be mediators of association between SEP and body composition, as they are not a determinant of SEP in infancy. As such, they were not included in statistical models aiming to test the total effect of SEP on body composition, or models that aimed to test the indirect effect of childhood internalising and externalising symptoms.

### 4.3 Descriptive Statistics

In this section, characteristics of the sample are described for the key variables.

#### 4.3.1 Body Composition and Anthropometric Measures

At age 7, 13,436 had complete data on body composition. At age 11 there were 12,723 observations, at age 14 there were 10,829 observations and at age 17 there were 9,169 observations. Table 4-6 shows the mean values for each body composition measure and anthropometric measure in girls and boys from ages 7 to 17. In general, average body composition and anthropometric measures increased with age. For FMI and FFMI using the Benn Index, there was a decline at age 11, and for FMI in girls again at age 17. In boys, the ratio of FM:FFM declined across sweeps. The Benn parameter was highest at age 11 for boys and ages 7 and 11 for girls for FM, due to the high variability between height and FM at these ages (Section 4.2.1.1, Table 4-3). Hence, large differences in FMI using the Benn parameter and FMI using height<sup>2</sup> are observed. The Benn index for FFM was closer to 2 at all ages.

**Table 4-6.** Mean and Standard Deviation for Body Composition and Anthropometric Measures by Sex at Ages 7, 11, 14 and 17

	<b>Boys</b>				<b>Girls</b>			
	Mean (SD)				Mean (SD)			
	<b>Age 7</b>	<b>Age 11</b>	<b>Age 14</b>	<b>Age 17</b>	<b>Age 7</b>	<b>Age 11</b>	<b>Age 14</b>	<b>Age 17</b>
<b>Body Composition (BIA)</b>								
<i>N</i>	6,791	6,428	5,493	4,587	6,645*	6,295	5,336	4,582
<i>Fat Mass (kg)</i>	5.32 (2.55)	8.78 (5.70)	10.79 (7.87)	12.54 (9.22)	5.86 (2.75)	11.03 (6.04)	16.48 (8.20)	19.08 (10.28)
<i>Fat Mass %</i>	20.02 (5.02)	20.08 (7.69)	16.91 (8.17)	15.97 (8.12)	22.15 (5.63)	24.64 (7.45)	27.28 (7.26)	28.25 (8.32)
<i>Fat Mass Index (kg/m<sup>2</sup>)</i>	3.41 (1.45)	4.05 (2.43)	3.85 (2.76)	4.00 (2.92)	3.81 (1.60)	5.04 (2.55)	6.31 (3.05)	7.06 (3.72)
<i>Fat Mass Index (Benn Index)</i>	2.27 (0.89)	1.42 (0.80)	2.33 (1.67)	4.07 (2.97)	2.34 (0.91)	1.89 (0.92)	4.13 (1.99)	3.62 (1.91)
<i>Fat-Free Mass (kg)</i>	20.33 (2.75)	32.02 (5.25)	48.09 (7.97)	59.44 (7.92)	19.6 (2.64)	31.17 (5.07)	41.06 (5.26)	44.75 (4.97)
<i>Fat-Free Mass %</i>	79.98 (5.02)	79.92 (7.69)	83.09 (8.17)	84.03 (8.12)	77.85 (5.63)	75.36 (7.45)	72.72 (7.26)	71.75 (8.32)
<i>Fat-Free Mass Index (kg/m<sup>2</sup>)</i>	13.15 (1.06)	14.97 (1.62)	17.19 (1.86)	18.96 (2.02)	12.86 (0.99)	14.40 (1.41)	15.77 (1.47)	16.63 (1.65)
<i>Fat-Free Mass Index (Benn Index)</i>	11.99 (0.94)	11.8 (1.23)	12.78 (1.33)	18.22 (1.94)	11.75 (0.87)	11.57 (1.09)	14.27 (1.33)	22.26 (2.14)
<i>FM: FFM Ratio</i>	0.26 (0.09)	0.27 (0.15)	0.22 (0.15)	0.20 (0.14)	0.29 (0.10)	0.34 (0.14)	0.39 (0.15)	0.41 (0.18)
<b>Anthropometrics**</b>								
<i>Weight (kg)</i>	25.63 (4.84)	40.81 (9.75)	58.85 (13.61)	72.26 (15.55)	25.46 (5.04)	42.21 (10.43)	57.56 (12.73)	63.84 (14.47)
<i>N</i>	6,894	6,512	5,572	4,660	6,741	6,361	5,396	4,670
<i>Height (m)</i>	1.24 (0.06)	1.46 (0.07)	1.67 (0.09)	1.77 (0.07)	1.23 (0.06)	1.47 (0.08)	1.61 (0.06)	1.64 (0.06)
<i>N</i>	6,930	6,594	5,642	4,723	6,758	6,489	5,622	4,850
<i>Body Mass Index (kg/m<sup>2</sup>)</i>	16.56 (2.26)	19.03 (3.54)	21.04 (4.05)	23.05 (4.61)	16.67 (2.41)	19.45 (3.77)	22.09 (4.39)	23.71 (5.07)
<i>N</i>	6,894	6,511	5,572	4,656	6,740	6,360	5,396	4,668
<i>Body Mass Index (Benn Index)</i>	13.97 (1.81)	12.46 (2.19)	15.41 (2.94)	22.92 (4.59)	13.84 (1.89)	12.9 (2.39)	18.52 (3.67)	25.28 (5.41)
<i>N</i>	6,894	6,511	5,572	4,656	6,740	6,360	5,396	4,668

**Table 4-6 Footnote:** \* Sample size is N=6,646 for fat mass percentage (FM%) in girls at age 7 because one observation had missing data for weight but not FM%. \*\*Samples size for weight, height, and body mass index are reported individually as they vary between measures at each age.

### 4.3.2 Area- and Family-level Socioeconomic Position

Table 4-7. shows the distribution of the sample belonging to each area-level deprivation group at sweep 1 (sweep 2 for new joiners). When sweep 2 was included alongside sweep 1, there were no missing data for the IMD. Almost 33% of the sample were living in the most deprived areas, whilst 14.5% were living in the least deprived.

Table 4-8 shows the distribution of the sample by family level SEP measures at sweep 1 and for those individuals who joined at sweep 2. Before accounting for new joiners, data was missing on roughly 5% of the sample for each SEP measure. Once new joiners were accounted for, between 1.5% and 3.3% of the sample had missing data. At sweep 1 after inclusion for those who enrolled at sweep 2, the majority of the sample were in the lowest fifth on the income distribution (25.06%), lived in a household with highest parental level of NVQ4 (31.1%) or NVQ2 (25.3%), and had highest household occupation of managerial and professional (36.6%) or semi-routine and routine (26.0%) occupations.

For income measured at sweep 4, a similar number of the sample were in each income fifth (Table 4-9), although with slightly higher number in the second income fifth (14.9%) and slightly lower in the highest income fifth (13.6%).

**Table 4-7.** Distribution of Sample by Index of Multiple Deprivation at Sweep 1 and for Sweep 2 New Joiners

<i>IMD</i>	<i>Sweep 1</i>		<i>Sweep 2 New Joiners</i>		<i>Sweep 1 with Replacement</i>	
	<b>N</b>	<b>(%)</b>	<b>N</b>	<b>(%)</b>	<b>N</b>	<b>(%)</b>
<i>Most Deprived 20%</i>	6,017	31.3	291	42.1	6,308	32.8
<i>40%</i>	4,218	21.9	151	21.8	4,369	22.7
<i>60%</i>	3,086	16.0	116	16.8	3,202	16.6
<i>80%</i>	2,508	13.0	74	10.7	2,582	13.4
<i>Least Deprived 20%</i>	2,722	14.2	60	8.7	2,782	14.5
<i>Missing</i>	692	3.6	-	-	-	-
<b>Total</b>	19,243	100	692	100	19,243	100

**Table 4-7 Footnote:** Sweep 1 with replacement includes observations for cohort members who joined at sweep 2 (new joiners).

**Table 4-8.** Distribution of Sample by Family Socioeconomic Position at Sweep 1 and for Sweep 2 New Joiners

	<b>Sweep 1</b>		<b>Sweep 2 New Joiners</b>		<b>Sweep 1 with Replacement</b>	
	<b>N</b>	<b>(%)</b>	<b>N</b>	<b>(%)</b>	<b>N</b>	<b>(%)</b>
<b>NVQ</b>						
<i>No Qualifications</i>	2,043	10.6	122	17.6	2,165	11.3
<i>Overseas Qualification</i>	412	2.1	43	6.2	455	2.4
<i>NVQ 1</i>	1,167	6.1	62	9.0	1,229	6.4
<i>NVQ 2</i>	4,679	24.3	197	28.5	4,876	25.3
<i>NVQ 3</i>	2,991	15.5	74	10.7	3,064	15.9
<i>NVQ 4</i>	5,828	30.3	148	21.4	5,976	31.1
<i>NVQ 5</i>	1,146	6.0	34	4.9	1,180	6.1
<i>Missing</i>	977	5.1	12	1.7	298	1.6
<b>NS-SEC</b>						
<i>Unemployed</i>	1,432	7.4	-	-	1,432	7.4
<i>Semi-routine and routine</i>	4,909	25.5	96	13.9	5,004	26.0
<i>Lower supervisory and technical</i>	1,552	8.1	36	5.2	1,588	8.3
<i>Small employers and self-employed</i>	1,078	5.6	54	7.8	1,132	5.9
<i>Intermediate</i>	2,374	12.3	43	6.2	2,417	12.6
<i>Managerial and Professional</i>	6,890	35.8	144	20.8	7,034	36.6
<i>Missing</i>	1,008	5.2	319	46.1	636	3.3
<b>Income</b>						
<i>Lowest Income Fifth</i>	4,580	23.8	242	35.0	4,822	25.01
<i>2<sup>nd</sup> Income Fifth</i>	4,103	21.3	182	26.3	4,284	22.3
<i>3<sup>rd</sup> Income Fifth</i>	3,450	17.9	89	12.9	3,539	18.4
<i>4<sup>th</sup> Income Fifth</i>	3,172	16.5	69	10.0	3,241	16.8
<i>Highest Income Fifth</i>	2,909	15.1	76	11.0	2,985	15.5
<i>Missing</i>	1,029	5.6	34	4.9	372	1.9
<b>Total</b>	<b>19,243</b>	<b>100</b>	<b>692</b>	<b>100</b>	<b>19,243</b>	<b>100</b>

**Table 4-8 Footnote:** NVQ – National Vocational Qualifications. NS-SEC – National Statistics Socioeconomic classification. Sweep 1 with replacement includes observations for cohort members who joined at sweep 2 (new joiners).

**Table 4-9.** Distribution of the Sample by Family Income at Sweep 4

<b>Income</b>	<b>Sweep 4 Income</b>	
	<b>N</b>	<b>(%)</b>
<i>Lowest Income Fifth</i>	2,854	14.8
<i>2<sup>nd</sup> Income Fifth</i>	2,862	14.9
<i>3<sup>rd</sup> Income Fifth</i>	2,797	14.5
<i>4<sup>th</sup> Income Fifth</i>	2,699	14.0
<i>Highest Income Fifth</i>	2,622	13.6
<i>Missing</i>	5,409	28.1
<b>Total:</b>	<b>19,243</b>	<b>100</b>

### 4.3.3 Strengths and Difficulties Questionnaire

Distribution for item response for the Strengths and Difficulties questionnaire is shown in Table 4-10. Items “liked” and “kind” had the highest number of respondents (N=12,778) and item “adults” had the lowest (N=12,436). For negatively worded items, the majority of respondents answered “not true”, and for positively worded answers “certainly true”, ranging from 60.02% for the item “restless” and 97.2% for the item “steals”. The exception to this was for the items “impulse” and “task” where the majority of respondents answered “somewhat true” (57.1% and 47.09%, respectively).

### 4.3.4 Environmental Characteristics

The distribution of the sample according to environmental characteristics at sweep 5 is shown in Table 4-11. For the IMD and domestic gardens there was a similar proportion of the sample in each group, with slightly greater numbers in the middle group (IMD: 36.4%, domestic gardens: 39.2%). For green spaces, around 40% of the sample lived in the most deprived or middle group, with 18.8% in the least deprived group. The distribution for fast-food density was skewed, with 72.7% of the sample in the low-density group.

### 4.3.5 Covariates

Distribution of the sample by age, sex and ethnicity is shown in Table 4-12. At sweep 4 age ranged from 6.33 years of age to 8.41 years of age, at sweep 5 from 10.08 to 12.33 years, at sweep 6 from 13.1 to 15.3 years, and at sweep 7 from 16.1 to 18.3 years. There was an even split of boys and girls in the sample, but with slightly more boys (51.4%) than girls (48.6%). The majority of the sample was of White ethnicity (81.3%) with the next largest ethnic group being Pakistani and Bangladeshi (6.9%). The other ethnic group made up the smallest proportion of the sample (1.6%).



**Table 4-10.** Distribution of the Sample by Strengths and Difficulties Questionnaire at Sweep 5

	Emotional Scale		Conduct Problems		Hyperactivity Scale		Peer Problems		Pro-Social Scale**	
	Frequency (N)	Percent (%)	Frequency (N)	Percent (%)	Frequency (N)	Percent (%)	Frequency (N)	Percent (%)	Frequency (N)	Percent (%)
	Complains		Anger		Restless		Alone		Considerate	
1. Not True	8,473	66.43	6,601	51.76	7,648	60.02	8,958	70.21	414	3.25
2. Somewhat True	3,450	27.05	4,353	34.13	3,536	27.75	3,066	24.03	2,666	20.96
3. Certainly True	832	6.52	1,800	14.11	1,558	12.23	735	5.76	9,639	75.78
<i>Total</i>	12,755	-	12,754	-	12,742	-	12,759	-	12,719	-
	Worried		Obedience*		Fidget		Friend*		Shares	
1. Not True	7,944	62.47	542	4.25	8,880	70.05	318	2.49	388	3.04
2. Somewhat True	3,941	30.99	4,392	34.41	2,768	21.84	1,091	8.55	2,673	20.92
3. Certainly True	831	6.54	7,829	61.34	1,028	8.11	11,357	88.96	9,715	76.04
<i>Total</i>	12,716	-	12,763	-	12,676	-	12,766	-	12,776	-
	Unhappy		Aggression		Attention		Liked*		Helpful	
1. Not True	10,588	83.14	11,812	92.73	5,729	45.04	144	1.13	247	1.93
2. Somewhat True	1,816	14.26	762	5.98	5,076	39.9	1,725	13.52	1,876	14.68
3. Certainly True	331	2.6	164	1.29	1,916	15.06	10,891	85.35	10,655	83.39
<i>Total</i>	12,735	-	12,738	-	12,721	-	12,760	-	12,778	-
	Anxiety		Lies		Impulse*		Bullied		Kind	
1. Not True	7,697	60.42	10,339	81.5	1,467	11.59	9,453	74.96	132	1.03
2. Somewhat True	4,017	31.53	2,065	16.28	7,223	57.06	2,558	20.29	1,328	10.38
3. Certainly True	1,025	8.05	282	2.22	3,969	31.35	599	4.75	11,328	88.58
<i>Total</i>	12,739	-	12,686	-	12,659	-	12,610	-	12,788	-
	Fear		Steals		Task*		Adults		Volunteers	
1. Not True	8,835	69.53	12,366	97.22	1,543	12.18	8,107	65.19	383	3.01
2. Somewhat True	3,210	25.26	263	2.07	5,968	47.09	3,376	27.15	3,885	30.5
3. Certainly True	662	5.21	91	0.72	5,162	40.73	953	7.66	8,471	66.5
<i>Total</i>	12,707	-	12,720	-	12,673	-	12,436	-	12,739	-

**Table 4-10 Footnote:** \* Indicates positively worded items, that items were reversed when combining to create sub scales. \*\* All items on the Pro-Social Scale are positively worded, so no items in the pro-social scale were reversed.

**Table 4-11.** Distribution of Sample by Environmental Characteristics at Sweep 5 (age 11) in the Millennium Cohort Study.

	<b>N</b>	<b>%</b>
<b>Index of Multiple Deprivation</b>		
Most deprived 30%	4,706	35.4
Middle 40%	4,837	36.4
Least deprived 30%	3,734	28.1
Missing	5,966	-
<b>Green Spaces</b>		
Most deprived 30%	5,338	40.2
Middle 40%	5,449	41.0
Least deprived 30%	2,492	18.8
Missing	5,963	-
<b>Domestic Garden</b>		
Most deprived 30%	2,646	30.5
Middle 40%	3,398	39.2
Least deprived 30%	2,627	30.3
Missing	10,572	-
<b>Fast-food Density</b>		
Low density (0-2)	8,346	72.7
Medium Density (3-7)	2,110	18.4
High Density (8+)	1,022	8.9
Missing	7,765	-

**Table 4-11 Footnote:** Data on domestic gardens was only collected in England, and data on fast-food density was only collected in Great Britain.

**Table 4-12.** Characteristics of Sample by Age, Sex and Ethnicity

	<b>Mean / N</b>	<b>SD / %</b>
<b>Age (years)</b>		
Age 7	7.23	0.25
Age 11	11.16	0.34
Age 14	14.27	0.35
Age 17	17.19	0.35
<b>Sex</b>		
Male	9,894	51.4
Female	9,349	48.6
<b>Ethnicity</b>		
White	15,638	81.3
Mixed	585	3.0
Indian	495	2.6
Pakistani and Bangladeshi	1,333	6.9
Black and Black British	720	3.7
Other Ethnic Group	299	1.6
Missing	173	0.9

**Table 4-12 Footnote:** Mean and SD presented for age. Count and percentage presented for sex and ethnicity. Age 7 N=13,844; age 11 N= 13,287; age 14 N= 11,690; age 17 N= 10,613. There was no missing data for sex. Abbreviations: SD – Standard Deviation.

## 4.4 Analytic Sample

The data used in this PhD are limited to one child in each family, to ensure independence of observations and to prevent clustering of variables by family. Therefore, in families with twins, triplets or multiple singletons (six families), data is only used from the child with the individual identifier marking them as the first child in the household. From here on, reference to variables from sweep 1 also refer to sweep 2 values for individuals that joined at this stage.

As body composition is the primary outcome in this PhD, the largest analytic sample used is constrained to those with at least one measure of body composition (Chapter 6, N=15,131, 79%). In further analysis, the largest sample sizes are limited to those with complete data on SDQ items at age 11 (Chapter 7, N=11,519) and BMI and body composition measured at age 17 (Chapter 8, N=9,324). Further break down of the analytic samples adopted are given in the individual chapters as the samples used differ between analysis.

## 4.5 Missing Data

This section describes the degree of missing data for key variables used in this thesis. Missingness for each variable is described for the total sample (N=19,243) and the analytic samples where appropriate.

### 4.5.1 Body Composition and Body Mass Index

Of the total sample (N=19,243), 38% (N= 7,242) had full data on body composition and BMI at ages 7 to 17, whilst 21% (N=4,068) had no data for any of the body composition or BMI measures (Table 4-12). All cohort members with complete data for the key body composition measures (FMI, FFMI and FM:FFM ratio) also had complete data for BMI. At all ages, those with complete data on one body composition measure (i.e FMI) had full data on the remaining body composition measures, with the exception of FM% in girls at age 7. At that age, one individual had FM% measured but not weight, preventing any further measures from being derived.

Missing data patterns for body composition and BMI are shown in further detail in Table 4-13. A total of 9% of the sample had missing data only at sweep 7, 8% of the sample had data missing at the final two sweeps, and 7% had data missing on all sweeps bar the first. A total of 79% (N=15,175) of the sample had body composition and BMI measures available for at least one sweep. Excluding BMI, the number of cohort members with at least one measure of body composition was 15,131 (79%).

Comparing the characteristics of the 15,175 with at least one measure of body composition or BMI with the group without any such measures, there was little difference in the proportion of missing data according to sex (Table 4-14). Those of white ethnicity had the lowest proportion of missing data (20.5%) and those of mixed ethnicity had the highest (28.4%), followed by Indian ethnicity (24.4%). For the IMD and income fifths, the highest proportion of missing data was found in those living in the most deprived areas (24.2%) and in the lowest income fifth (26.3%), whilst the lowest proportion of missing data was for those in the most advantaged areas (18.1%) and in the highest income fifth (15.0%). For the NS-SEC, those who were unemployed had the highest proportion of missing data

(28.7%), followed by those in semi-routine and routine occupations (26.7%). For the NVQ, the highest proportion of missing data was found among those with no qualifications (30.0%), overseas qualifications (27.2%) and NVQ level 1 (25.4%).

**Table 4-13.** Missing Data Patterns for Body Composition and Body Mass Index from age 7 to 17

Percentage Missing Data Pattern	Age 7		Age 11		Age 14		Age 17	
	BMI	BC*	BMI	BC*	BMI	BC*	BMI	BC*
38%								
21%								
9%								
8%								
7%								
3%								
3%								
2%								
2%								
1%								
1%								
5%	50 unique missing data patterns with missing data on one or more item. Each remaining missing data pattern is equivalent to <1% of the sample.							

**Table 4-13 Footnote:** \*BC indicates body compositions measures. Measures of body composition are fat mass index (FMI), fat-free mass index (FFMI) & fat mass to fat-free mass ratio (FM:FFM ratio). Missing Data for BMI and Body composition collected at sweeps 4-7 in the total sample (N=19,243). Squares shaded dark grey indicate missing data, whilst squares left blank indicate complete data.

**Table 4-14.** Characteristics of Missingness for Body Mass Index and Body Composition Measures in the Full Sample (N=19,243)

	<b>Some Complete Data* (N= 15,175)</b>		<b>Missing Data</b>	
	<b>N</b>	<b>%</b>	<b>N</b>	<b>%</b>
<b>Sex</b>				
Male	7,736	78.2	2,158	21.8
Female	7,439	79.6	1,910	20.4
<b>Ethnicity</b>				
White	12,427	79.5	3,211	20.5
Indian	442	75.6	143	24.4
Pakistani and Bangladeshi	378	76.4	117	23.6
Black and Black British	1,052	78.9	281	21.1
Other Ethnic Group	548	76.1	172	23.9
Mixed	214	71.6	85	28.4
<b>IMD</b>				
IMD Group 1 (Most deprived 20%)	4,561	75.8	1,456	24.2
IMD Group 2	3,278	77.7	940	22.3
IMD Group 3	2,486	80.6	600	19.4
IMD Group 4	2,048	81.7	460	18.3
IMD Group 5 (Most advantaged 20%)	2,230	81.9	492	18.1
<b>Income</b>				
Lowest Income Fifth	3,374	73.7	1,206	26.3
Second Fifth	3,144	76.6	959	23.4
Third Fifth	2,733	79.2	717	20.8
Fourth Fifth	2,644	83.4	528	16.7
Highest income fifth	2,473	85.0	436	15.0
<b>NS-SEC</b>				
Unemployed	1,021	71.3	411	28.7
Semi-routine and routine	3,597	73.3	1,312	26.7
Lower supervisory and technical	1,246	80.3	306	19.7
Small employers and self-employed	854	79.2	224	20.8
Intermediate	1,848	77.8	526	22.2
Managerial and Professional	5,801	84.2	1,089	15.8
<b>NVQ</b>				
No Qualifications	1,430	70.0	613	30.0
Other/overseas Qualifications	300	72.8	112	27.2
NVQ Level 1	871	74.6	296	25.4
NVQ Level 2	3,621	77.4	1,058	22.6
NVQ Level 3	2,366	79.1	625	20.9
NVQ Level 4	4,859	83.4	969	16.6
NVQ Level 5	955	83.3	191	16.7

**Table 4-14 Footnote:** \*Sample for complete data are those individuals with data available for body composition or BMI on at least one time point (N= 15,175). Sample for missing data are those with no body composition or BMI measure across any of the considered ages (age 7 to 17). Missing data N=4,068. Patterns of missingness are described using sex, ethnicity and SEP characteristics from sweep 1.

#### 4.5.2 Family and Area Socioeconomic Position

Once replacement of late joiners was accounted for, there was no missing IMD information at sweep 1. For income, NS-SEC and NVQ 96% of the total sample (N=19,243) had complete data. The most common missing data pattern after this was those with missing data on parental occupation (2%) followed by those with missing data on income, NS-SEC and NVQ. The remaining missing data patterns are shown in Table 4-15. Missing data patterns were the same when the sample was constrained to the analytic sample for Chapter 6 (N=15,131).

For income at sweep 4, a total of 5,406 observations were missing, equivalent to 28% of the total sample. When this was restricted to those with some data on either body composition or BMI at age 17 (analytic sample for Chapter 8, N=9,324), a total of 7% of the sample (N=684) had missing data on income.

For Income measured at sweep 1, there was little difference in the proportion with missing data between males and females (Table 4-16). Those of mixed ethnicity or other ethnicity had a higher proportion of missing data (12.0% and 9.7% respectively) and those of white ethnicity had the lowest proportion (3.9%). There was little difference in the proportion of missingness by the IMD, with percentages ranging from 1.6% in IMD group 2 and 2.0% in IMD group 5. There was the highest percentage of missing data in the English ethnic (9.5%) and disadvantaged strata (7.6%), and the lowest proportion in the Welsh disadvantaged stratum (1.4%). There was a greater proportion of missing data for those in England compared to the other countries of the UK. Characteristics of missingness for the NS-SEC (Appendix A4.1) and the NVQ (Appendix A4.2) at sweep 1 were similar to that for income.

For income measures at sweep 4, there was also little difference in the proportion with missingness between males and females (Table 4-17). Similar to measures at sweep 1, those of mixed and other ethnicity had the highest proportion of missingness for income measured at sweep 4 (39.5% and 37.6% respectively), and the lowest proportion was found among those of white ethnicity (26.3%). There was a higher proportion of missingness in the most deprived IMD group (34.4%), and missingness was lower in each subsequent group, and lowest in the most advantaged group (18.5%). Missingness for income at sweep 4 was also highest in those unemployed at sweep 1 (41.5%) and in semi-routine and routine occupations (35.8%). Missingness was lowest in managerial and professional occupations (19.8%). There was also a higher percentage of missing data among those with no qualifications (40.8%), overseas qualifications (38.6%) and NVQ level 1 (36.0%) whilst the lowest percentage was in NVQ levels 4 and 5 (both 20.7%).

**Table 4-15.** Missing Data Patterns for Family-Level Income at Sweep 1

<b>Percentage Missing Data Pattern</b>	<b>NVQ</b>	<b>Income</b>	<b>NS-SEC</b>
96%			
2%			
1%			
<1%			
<1%			
<1%			

**Table 4-15 Footnote:** Missing Data for family-level SEP collected at sweep 1 in the total sample (N=19,243). Squares shaded dark grey indicate missing data, whilst squares left blank indicate complete data.

**Table 4-16.** Characteristics of Missingness for Income at Sweep 1 in the Full Sample (N=19,243)

	<b>Complete Data (N= 18,214)</b>		<b>Missing Data</b>	
	<b>N</b>	<b>%</b>	<b>N</b>	<b>%</b>
<b>Sex</b>				
Male	9,372	94.7	522	5.3
Female	8,842	94.6	507	5.4
<b>Ethnicity</b>				
White	15,032	96.1	606	3.9
Indian	543	92.8	42	7.2
Pakistani and Bangladeshi	455	91.9	40	8.1
Black and Black British	1,245	93.4	88	6.6
Other Ethnic Group	650	90.3	70	9.7
Mixed	263	88.0	36	12.0
<b>IMD</b>				
IMD Group 1 (Most deprived 20%)	5,901	98.1	116	1.9
IMD Group 2	4,150	98.4	68	1.6
IMD Group 3	3,030	98.2	56	1.8
IMD Group 4	2,463	98.2	45	1.8
IMD Group 5 (Most advantaged 20%)	2,669	98.1	53	2.0
<b>Stratification Characteristics</b>				
England – Advantaged	4,541	94.1	287	5.9
England – Disadvantaged	4,438	92.4	367	7.6
England – Ethnic	2,346	90.5	245	9.5
Wales – Advantaged	819	98.4	13	1.6
Wales – Disadvantaged	1,901	98.6	27	1.4
Scotland – Advantaged	1,122	98.0	23	2.0
Scotland – Disadvantaged	1,173	98.5	18	1.5
Northern Ireland – Advantaged	706	97.7	17	2.4
Northern Ireland – Disadvantaged	1,168	97.3	32	2.7

**Table 4-16 Footnote:** Complete data N= 18,214. Missing data N= 1,029. Patterns of missingness are described using sex, ethnicity, IMD and stratification characteristics from sweep 1.

**Table 4-17.** Characteristics of Missingness for Income at Sweep 4 in the Full Sample (N=19,243)

		<b>Complete Data (N=13,837)</b>		<b>Missing Data</b>	
		<b>N</b>	<b>%</b>	<b>N</b>	<b>%</b>
<b>Sex</b>					
	<i>Male</i>	7,023	71.0	2,871	29.0
	<i>Female</i>	6,814	72.9	2,535	27.1
<b>Ethnicity</b>					
	<i>White</i>	11,519	73.7	4,119	26.3
	<i>Indian</i>	376	64.3	209	35.7
	<i>Pakistani and Bangladeshi</i>	342	69.1	153	30.9
	<i>Black and Black British</i>	872	65.4	461	34.6
	<i>Other Ethnic Group</i>	449	62.4	271	37.6
	<i>Mixed</i>	181	60.5	118	39.5
<b>IMD</b>					
	<i>IMD Group 1 (Most deprived 20%)</i>	3,945	65.6	2,072	34.4
	<i>IMD Group 2</i>	3,011	71.4	1,207	28.6
	<i>IMD Group 3</i>	2,322	75.2	764	24.8
	<i>IMD Group 4</i>	1,930	77.0	578	23.1
	<i>IMD Group 5 (Most advantaged 20%)</i>	2,137	78.5	585	21.5
<b>Income</b>					
	<i>Lowest Income Fifth</i>	2,880	62.9	1,700	37.1
	<i>Second Fifth</i>	2,813	68.6	1,290	31.4
	<i>Third Fifth</i>	2,558	74.1	892	25.9
	<i>Fourth Fifth</i>	2,522	79.5	650	20.5
	<i>Highest income fifth</i>	2,370	81.5	539	18.5
<b>NS-SEC</b>					
	<i>Unemployed</i>	838	58.5	594	41.5
	<i>Semi-routine and routine</i>	3,151	64.2	1,758	35.8
	<i>Lower supervisory and technical</i>	1,139	73.4	413	26.6
	<i>Small employers and self-employed</i>	780	72.4	298	27.6
	<i>Intermediate</i>	1,696	71.4	678	28.6
	<i>Managerial and Professional</i>	5,526	80.2	1,364	19.8
<b>NVQ</b>					
	<i>No Qualifications</i>	1,209	59.2	834	40.8
	<i>Other/overseas Qualifications</i>	253	61.4	159	38.6
	<i>NVQ Level 1</i>	747	64.0	420	36.0
	<i>NVQ Level 2</i>	3,229	69.0	1,450	31.0
	<i>NVQ Level 3</i>	2,200	73.6	791	26.5
	<i>NVQ Level 4</i>	4,622	79.3	1,206	20.7
	<i>NVQ Level 5</i>	909	79.3	237	20.7

**Table 4-17 Footnote:** Characteristics of missingness for household income collected at sweep 4. Complete data N= 13,837. Missing data N= 5,406. Patterns of missingness are described using sex, ethnicity and SEP characteristics from sweep 1.



### 4.5.3 Strengths and Difficulties Questionnaire

There was 60% (N = 11,519) of the total sample (N=19,243) with complete data on all of the SDQ items at age 11, and 33% with missing data for all SDQ items. For the remaining 7% of the sample, there were missing data on one or more SDQ item. There was partial or full missing data for 40% (n= 7,724) of the sample.

When limited to the analytic sample adopted in chapter 8 (body composition or BMI available at age 17, N=9,324), a total of 84% (N=7,794) of the sample had complete data for the SDQ items at age 11. A total of 8% had missing information on all SDQ items, and the remaining 8% had missing data on one or more parent-reported SDQ item. In total, of those with body composition and BMI available at age 17, 16% (N=1,530) had missing data for some or all of the SDQ items (Table 4-18).

Missing data were greatest for the item “adults” (gets on better with adults) in both the total sample (N=19,243) and analytic sample (chapter 8, N=9,324). Each individual item had roughly 8% of cases missing in the chapter 8 analytic sample, whilst for the item “adults” it was missing for roughly 10% of cases. As such, participants who were missing only for the item “adults” made up 1% of the missing data patterns. The remaining missing data patterns, bar those with complete and fully missing data, all made up less than 1% of the missing data patterns, with 349 and 233 unique patterns in the full and analytic sample respectively.

**Table 4-18.** Missing Data Patterns for Items of the Parent-Reported Strengths and Difficulties Questionnaire in the Full Sample and Restricted Sample

<b>% Missing Data Patterns</b>	<b>Description of Pattern</b>
<b>Full Sample (N=19,243)</b>	
60%	Data available on all SDQ items
33%	All SDQ items missing
1%	Missing data on item “adults” only**
6%	349 unique missing data patterns with missing data on one or more item. Each remaining missing data pattern is equivalent to <1% of the sample.
<b>Analytic Sample* (N=9,324)</b>	
84%	Data available on all SDQ items
8%	All SDQ items missing
2%	Missing data on item “adults” only**
6%	233 unique missing data patterns with missing data on one or more item. Each remaining missing data pattern is equivalent to <1% of the sample.

**Table 4-18 Footnote:** Description of missing data patterns for items in the Strengths and Difficulties Questionnaire (SDQ) measured at age 11. \* Analytic sample is limited to those individuals with no missing data on BMI or body composition at age 17 (analytic sample chapter 8, N=9,324). \*\* Item “adults” is the SDQ item with the greatest amount of missing data. As such, participants with missing data only on item “adults” is the only pattern to make up more than 1% of the missing data patterns, excluding patterns for fully complete and fully missing data. All other patterns for missing data make up less than 1% of the missing data patterns.

**Table 4-19.** Characteristics of Missingness for Strengths and Difficulties Questionnaire at Sweep 5 in the Full Sample (N=19,243)

	<b>Some Complete Data* (N=12,189)</b>		<b>Missing Data</b>	
	<b>N</b>	<b>%</b>	<b>N</b>	<b>%</b>
<b>Sex</b>				
Male	6,468	65.4	3,426	34.6
Female	6,351	67.9	2,998	32.1
<b>Ethnicity</b>				
White	10,711	68.5	4,927	31.5
Indian	370	63.3	215	36.8
Pakistani and Bangladeshi	291	58.8	204	41.2
Black and Black British	813	61.0	520	39.0
Other Ethnic Group	387	53.8	333	46.3
Mixed	161	53.9	138	46.2
<b>IMD</b>				
IMD Group 1 (Most deprived 20%)	3,820	60.6	2,488	39.4
IMD Group 2	2,816	64.5	1,553	35.6
IMD Group 3	2,261	70.6	941	29.4
IMD Group 4	1,887	73.1	695	26.9
IMD Group 5 (Most advantaged 20%)	2,035	73.2	747	26.9
<b>Income</b>				
Lowest Income Fifth	2,792	57.9	2,030	42.1
Second Fifth	2,673	62.4	1,611	37.6
Third Fifth	2,424	68.5	1,115	31.5
Fourth Fifth	2,428	74.9	813	25.1
Highest income fifth	2,289	76.7	696	23.3
<b>NS-SEC</b>				
Unemployed	750	52.4	682	47.6
Semi-routine and routine	2,929	58.5	2,075	41.5
Lower supervisory and technical	1,052	66.3	536	33.8
Small employers and self-employed	714	63.1	418	36.9
Intermediate	1,631	67.5	786	32.5
Managerial and Professional	5,342	76	1,692	24.1
<b>NVQ</b>				
No Qualifications	1,112	51.4	1,053	48.6
Other/overseas Qualifications	248	54.5	207	45.5
NVQ Level 1	740	60.2	489	39.8
NVQ Level 2	3,139	64.4	1,737	35.6
NVQ Level 3	2,057	67.1	1,007	32.9
NVQ Level 4	4,454	74.5	1,522	25.5
NVQ Level 5	894	75.8	286	24.2

**Table 4-19 Footnote:** \*Sample for complete data are those individuals with data available for at least one item of the Strengths and Difficulties Questionnaire (SDQ) at age 11 (N= 12,819). Sample for missing data are those with missing data for all items in the SDQ at age 11 (Missing data N=6,424). Patterns of missingness are described using sex, ethnicity and SEP characteristics from sweep 1.

Percentage of missingness for all SDQ items at sweep 5 was similar between boys and girls (Table 4-19). Similar to other key measures, percentage missing data for all SDQ items was greatest among mixed and other ethnic group (46.2 and 46.3% respectively) and lowest in those of white ethnicity (31.5%). Percentage of missing data was also highest in those living in the most deprived areas (39.4%) and in the lowest income fifth (42.1%), and lowest in the two most advantaged IMD groups (both 26.9%) and highest income fifth (23.3%). There was a greater percentage of missing data for SDQ items among those unemployed (47.6%) and in semi-routine and routine occupations (41.5%), and lowest in managerial and professional occupations (24.1%). There was also a higher percentage of missing data among those with no qualifications (48.6%), overseas qualifications (45.5%) and NVQ level 1 (39.8%) whilst the lowest percentage in NVQ level 5 (24.2%).

#### 4.5.4 Environmental Characteristics

A total of 44% of the total sample (N=19,243) had complete data on environmental characteristics at sweep 5, whilst 31% had missing data on all characteristics. The next most common missing data pattern was to have missing information on domestic gardens only, followed by missing data on both domestic gardens and fast-food density. These patterns were due to the fact that information on domestic gardens and fast-food density were only collected in England and Great Britain, respectively. The remaining missing data patterns are shown in Table 4-20.

When looking at the level of missingness for each variable in the analytic sample (N=9,324), both the IMD and green space had less than 5% missing data. Once the analytic samples were restricted to account for the characteristics collected only in England and Great Britain, missing data for domestic gardens was 0.1% and for fast-food density was 9.4% Table 4-21.

Percentage of missingness for the IMD (Table 4-22), green spaces (Appendix A4.3) and fast-food (Appendix A4.4) density were similar to that for other key variables, although the lowest percentage of missing data by ethnicity was typically found in Black and Black British then followed by white ethnicity. For domestic gardens (Appendix A4.5), a higher percentage of missing data was found among white ethnicity (58.9%), driven by the fact domestic gardens was only collected in England and the other countries of the UK had a predominantly white population. For domestic gardens, there was also the highest percentage of missing data in the IMD group 2 (58%) and semi-routine and routine (61%), whilst for the NS-SEC the lowest percentage of missing data was found among those with other/overseas category (46.8%).

However, patterns of missing data for domestic gardens are largely driven by those residing outside of England. When limited to those living in England, there were only 6 counts of missing data and differences between groups are negligible (not presented due to low count). Similarly, when fast-food density was limited to Great Britain only, the characteristics of missing data also differed slightly (Appendix A4.6). The lowest percentage of missing data was found in the Black and Black British ethnicity (30.0%) but then followed by Pakistani and Bangladeshi ethnic group (31.8%). The lowest percentage of missing data was also found in IMD group 3 (32.9%).

**Table 4-20.** Missing Data Patterns for Environmental Characteristics at Sweep 5 for the Full Sample (N=19,243)

<b>Percentage Missing Data Pattern</b>	<b>Green Spaces</b>	<b>IMD</b>	<b>Fast-Food Density</b>	<b>Domestic Gardens</b>
44%				
31%				
16%				
8%				
1%				
<1%				
<1%				

**Table 4-20 Footnote:** Missing Data for environmental characteristics collected at sweep 5 in the total sample (N=19,243). Squares shaded dark grey indicate missing data, whilst squares left blank indicate complete data.

**Table 4-21.** Missing Data in Environmental Characteristics in Analytic Sample in UK, England and Great Britain

<b>Analytic Sample *</b>	<b>UK Wide (N=9,324)</b>	<b>England Only (N=5,948)</b>	<b>Great Britain Only (N=8, 507)</b>	
<i>Environment Measure</i>	<i>IMD</i>	<i>Green Spaces</i>	<i>Domestic Gardens</i>	<i>Fat-Food Density</i>
<i>Complete Data</i>	95.4%	95.4%	99.9%	90.6%
<i>Missing</i>	4.6%%	4.6%%	0.1%	9.4%

**Table 4-21 Footnote:** \* Analytic sample is limited to those individuals with no missing data on BMI or body composition at age 17 (chapter 8 analytic sample). For Domestic Gardens and Fast-Food Density sample is also limited to those residing in England and Great Britain at sweep 5 respectively.

**Table 4-22.** Characteristics of Missingness for the Index of Multiple Deprivation at Sweep 5 in the Full Sample (N=19,243)

		<b>Complete Data (N=13,277)</b>		<b>Missing Data</b>	
		<b>N</b>	<b>%</b>	<b>N</b>	<b>%</b>
<b>Sex</b>					
	<i>Male</i>	6,708	67.8	3,186	32.2
	<i>Female</i>	6,569	70.3	2,780	29.7
<b>Ethnicity</b>					
	<i>White</i>	10,922	69.8	4,716	30.2
	<i>Indian</i>	383	65.5	202	34.5
	<i>Pakistani and Bangladeshi</i>	339	68.5	156	31.5
	<i>Black and Black British</i>	934	70.1	399	29.9
	<i>Other Ethnic Group</i>	426	59.2	294	40.8
	<i>Mixed</i>	183	61.2	116	38.8
<b>IMD</b>					
	<i>IMD Group 1 (Most deprived 20%)</i>	4,051	64.2	2,257	35.8
	<i>IMD Group 2</i>	2,932	67.1	1,437	32.9
	<i>IMD Group 3</i>	2,317	72.4	885	27.6
	<i>IMD Group 4</i>	1,912	74.1	670	26
	<i>IMD Group 5 (Most advantaged 20%)</i>	2,065	74.2	717	25.8
<b>Income</b>					
	<i>Lowest Income Fifth</i>	2,964	61.5	1,858	38.5
	<i>Second Fifth</i>	2,807	65.5	1,477	34.5
	<i>Third Fifth</i>	2,479	70.1	1,060	30
	<i>Fourth Fifth</i>	2,474	76.3	767	23.7
	<i>Highest income fifth</i>	2,325	77.9	660	22.1
<b>NS-SEC</b>					
	<i>Unemployed</i>	834	58.2	598	41.8
	<i>Semi-routine and routine</i>	3,087	61.7	1,917	38.3
	<i>Lower supervisory and technical</i>	1,080	68.0	508	32.0
	<i>Small employers and self-employed</i>	763	67.4	369	32.6
	<i>Intermediate</i>	1,666	68.9	751	31.1
	<i>Managerial and Professional</i>	5,427	77.2	1,607	22.9
<b>NVQ</b>					
	<i>No Qualifications</i>	1,241	57.3	924	42.7
	<i>Other/overseas Qualifications</i>	284	62.4	171	37.6
	<i>NVQ Level 1</i>	767	62.4	462	37.6
	<i>NVQ Level 2</i>	3,228	66.2	1,648	33.8
	<i>NVQ Level 3</i>	2,123	69.3	941	30.7
	<i>NVQ Level 4</i>	4,536	75.9	1,440	24.1
	<i>NVQ Level 5</i>	913	77.4	267	22.6

**Table 4-22 Footnote:** Characteristics of missingness for IMD at sweep 5. Complete data N= 13,277. Missing data N= 5,966. Patterns of missingness are described using sex, ethnicity and SEP characteristics from sweep 1.

## 4.6 Statistical Analysis

In this thesis, a number of different statistical approaches are adopted to address the questions and hypotheses set out in the aims and objectives. In Chapter 6, multilevel growth curve models are fitted to the data to model inequalities in trajectories in body composition. In Chapter 7, exploratory and confirmatory factor analyses were used to validate internalising and externalising symptoms, measured by the SDQ, as mediators between SEP and body composition. Finally, in Chapter 8, structural equation modelling is used to investigate the mediating role of internalising and externalising symptoms and the moderating role of environmental characteristics. Because of the varied methods used in this thesis, further details of each method are included in the relevant chapters. This section provides a brief overview of common statistical challenges relevant across the analyses, namely use of sampling weights and stratification characteristics, and handling of missing data.

### 4.6.1 Sampling Weights

Because of the clustered stratified design of the MCS, there are sampling weights to account for the unequal probability of selection [204]. These weights are either country specific or UK wide, dependent on the analysis being conducted. Additionally, because the MCS has experienced attrition at each sweep, attrition and non-response weights have been derived. These weights take into account the probability of non-response of participants and are determined individually for each sweep, and again are either UK wide or country specific. When using data from multiple sweeps, it has been advised to use the attrition weight from the most recent sweep that the outcome variable comes from [216]. This is because analysis that uses data from multiple sweeps will often, although not always, be restricted to the latest sweep sample.

When conducting analysis that estimates population means and variances, it is necessary to use weighted estimates [204]. Thus, descriptive analysis is conducted using STATA's survey command `svy`, and the baseline MCS stratification characteristics and attrition weights are used. When conducting analysis that estimates coefficients from regression techniques, it is advised to include the stratification characteristics as a covariate in the model [204].

How and when weights are used is dependent on the analysis, and thus further details of inclusion of weights is given in the methods section of each chapter.

### 4.6.2 Missing Data Approaches

The MCS has experienced attrition, which is commonplace in epidemiological studies, and in particular longitudinal studies where participants drop out over time. Attrition occurs when study members withdraw from participating and no longer respond to surveys over time. A common approach has been to use a complete case analysis (or list-wise deletion), where only those with complete data on all relevant variables are included. This may result in biased estimates, especially if participants who have missing data also have shared characteristics that are relevant to the analysis. The representativeness of the sample may also be reduced, if the remaining participants no longer

reflect the target population, therefore reducing the generalisability of results. Missing data also results in smaller sample sizes meaning analyses have less power.

Data can be missing in a number of different ways, relating to the probability of being missing [217]. Where the likelihood of being missing is the same across all cases, this is known as Missing Completely at Random (MCAR) [218]. In such cases the reasons for missingness do not introduce bias into the analysis, and therefore there is no need to adopt a missing data approach. However, it is uncommon for MCAR to occur in most datasets [218], and especially in longitudinal datasets.

The case when the likelihood of having a missing value can be explained by observed values in the dataset, and therefore there are systematic differences between the missing data and complete data, is known as Missing at Random (MAR) [218]. When data are MAR, bias is likely to be introduced into the analysis and missing data approaches should be considered. Compared to MCAR, MAR is a more likely to occur in epidemiological datasets and is often the starting assumption for missing data patterns [218]. A subset of MAR is Missing at Random in regards to X (MARX), where only the variable X has an influence on the missing data pattern [219].

The final missing data pattern is known as Missing Not at Random (MNAR), where even after accounting for missingness by observed variables, the missing data are not explained. In this case, the reasons for missingness are explained by unobserved variables. MNAR is the most complex of the missing data assumptions, and often requires further investigation to identify the unobserved variables that explain the missing data patterns [218].

In this thesis, missing data are present in the predictive variables and covariates across the analysis conducted. It has previously been suggested as a rule of thumb, that missing data approaches should be adopted if missing data are 5% or greater in the sample, and that loss to follow-up is determined by sample characteristics [220]. As has been demonstrated in Section 4.5 of this chapter, missing data in the analytic sample typically exceeds 5% and missing data in the outcome is related to observed variables under consideration. Additionally, the percentage of missingness for predictor variables and covariates differs by characteristics of the sample. Consequently, the missing data mechanism is not MCAR and instead is either MAR or MNAR. Therefore, missing data approaches should be considered to prevent introducing bias in the estimates. The approaches adopted to deal with missing data vary by analytical method and are thus described in the relevant chapters.

# Chapter 5 Systematic Review and Narrative Synthesis: Life Course Socioeconomic Position and Body Composition

## 5.1 Introduction

The first objective of this thesis is to explore the link between SEP and directly measured body composition. This is achieved through a systematic review of the literature in both children and adults from populations across the globe. Although the literature linking SEP and BMI has been extensively reviewed, the smaller number of studies linking SEP and body composition have not. Both adults and children, and individuals from high-, middle- and low- income countries were included in the review despite exceeding the scope of the remaining thesis. This was done as understanding of how associations differ between age groups has not previously been explored, nor is there understanding of how associations may differ according to country development. This is important to help frame and contextualise associations for children and adolescents in high-income countries. In particular, difference in inequalities between countries reinforces the focus on exploring environmental determinants of body composition in the remaining thesis, as they are likely to reflect differences in the environment, particularly the obesogenic environment and the role of the nutrition transition. Additionally, exploring generational differences helps frame associations in children and adolescents into a life course perspective, by understanding how inequalities are likely to differ or change as individuals age. Three hypotheses were tested:

**Hypothesis 1:** *There are social inequalities in fat mass (FM) and fat-free mass (FFM), with less advantaged SEP being related to higher FM and lower FFM in high-income countries, and associations in the opposite direction in middle- and low-income countries.*

**Hypothesis 2:** *Social inequalities in FM and FFM vary between generations, with greater evidence of inequalities in FM and FFM among those born more recently.*

**Hypothesis 3:** *Less advantaged SEP is related to higher FM and lower FFM more frequently in females compared to males in HICs.*

To test the hypotheses, associations were assessed between (a) SEP and body composition in childhood (up to and including 18 years), (b) SEP and body composition in adulthood and (c) childhood SEP and body composition in adulthood. Additional aims were to assess secular changes



in socioeconomic inequalities in body composition and explore heterogeneity by sex, SEP measures, body composition measure and income level of the country of study.

This chapter outlines the study protocol, methods adopted for the review, results and discusses the findings in the context of this thesis and wider literature. Work from this chapter has been reported on in three published papers (Bridger Staatz et al. *Systematic Reviews*. 2019: 8:26; Bridger Staatz et al. *International Journal of Obesity*. 2021: 45, 2300–2315; Bridger Staatz et al. *International Journal of Obesity*. 2021: 45, 2316–2334. Appendix A5.1).

## 5.2 Methods

A protocol for the systematic review was written and published prior to starting the review [221] (Appendix A5.1), and was registered with the PROSPERO database (CRD42019119937). The protocol was written in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses Protocols (PRISMA-P) checklist (Appendix A5.2), and the aims of the systematic review were outlined. Exploring heterogeneity by country income level was not initially included as an aim in the protocol due to anticipated small numbers of papers from middle- and low-income countries, despite hypothesised important effects of country level income in terms of direction of associations. As such, this was written into the protocol at a later point in the review process, and changes made to the published protocol were amended online via the PROSPERO database. The review was carried out according to Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist [222] (Appendix A5.3 and A5.4).

### 5.2.1 Definition of Key Terms

For this review, SEP was defined as any recognised indicator of social position in society, e.g. income, education, overcrowding, area-level deprivation [223]. The definition of “composite SEP” adopted in this review was any index combining two or more individual level SEP indicators, whilst area-level SEP was defined as any measure that captures the deprivation of an area. The same indicators of parental SEP were used as a marker for childhood SEP. In studies conducted in children, measures of SEP, such as occupation and education, relate to parents. Other markers such as overcrowding reflect the home in which the child lives, and some markers of education were based on the type of school attended by the child.

Body composition was defined as any measurement related to total FM and FFM (including other fat-free measures), location of FM and FFM or any proportion or ratio of measures of FM and FFM. Body composition was measured using any appropriate measure, excluding anthropometric indicators. Appropriate methods of measurement are listed below [63, 65]:

- i) Bioelectrical impedance analysis (BIA)
- ii) Dual-energy X-ray absorptiometry (DXA)
- iii) Computed tomography (CT)
- iv) Magnetic resonance imaging (MRI)

- v) Other less common methods: Total body water (TBW); bone density or densitometry; Total body counting and neutron activation; Air-displacement plethysmography.

### 5.2.2 Eligibility

Eligible papers were those that met the following criteria:

- Original studies published in peer-reviewed journals.
- Examined the association between at least one measure of SEP and a measure of body composition at the same or a later age.
- Used any recognised measure of SEP as described above.
- Used any recognised measure of body composition as described above.
- Were an observational study.
- Used samples selected from the general population.
- Were written in the English language.

Studies were excluded if they:

- Did not meet inclusion criteria.
- Were reviews.
- Studied specific groups e.g clinical or patient populations.
- Measured body composition through anthropometric measures, such as BMI, waist circumference (WC), waist-to-hip ratio (WHR) or waist-height ratio (WHtR).

### 5.2.3 Search Strategy

An electronic search was carried out to identify appropriate studies, with MEDLINE and Embase Classic + Embase being searched using OvidSP as the interface, and SPORTDiscus searched using EBSCO as the interface. Databases were searched from the earliest record entry until 30<sup>th</sup> of January 2019. The decision on which databases to include was reached following consultation with a librarian whilst writing the protocol. Medline was preferred to PubMed given it has 98% coverage of the papers included in PubMed and has an advanced search strategy that is better suited for systematic reviews.

The search terms used are shown in Table 5-1. Various search tools and techniques were adopted to ensure the search identified all relevant articles, as documented in Table 5-2. The results of the search were de-duplicated and stored in the reference manager, Endnote. Where full texts were difficult to access, authors were contacted directly or texts were accessed via UCLs Library Services resource sharing with other academic institutions. The database was exported to Rayyan Qatar Computing Research Institute (QCRI) [224] to conduct screening.

All titles and abstracts were screened for eligibility, followed by a subsequent full text screening of eligible papers for inclusion in the review. I reviewed all abstracts and texts, and two additional researchers split the role of second reviewer and independently carried out each stage of the

screening process. Additionally, the reference lists of eligible full texts were screened and searches of publications from key studies with relevant data were used to identify further papers. Disagreement regarding eligibility of the articles were resolved via discussion.

**Table 5-1.** Search Terms Used in the Systematic Review

<b>SEARCH TERMS</b>	
<b>Database</b>	<b>MeSH Terms</b>
<i>Medline</i>	<p><b>Body Composition</b> – exp Body Composition/; Adipose Tissue/; exp Body Fat Distribution/; Obesity/ or obesity, abdominal/.</p> <p><b>Body Composition Measures</b> - Electric Impedance/; Magnetic Resonance Imaging/; Tomography, X-Ray Computed/; Densitometry/; Whole-Body Counting/; Plethysmography/.</p> <p><b>Socioeconomic Position</b> - socioeconomic factors/ or poverty/ or poverty areas/ or social class/; Educational status/ or income/ or occupations/ or social conditions/.</p>
<i>Embase + Embase Classic</i>	<p><b>Body Composition</b> - Body composition/ or body distribution/ or body fat/ or body fat distribution/; Obesity/; lean body weight/; Fat mass/.</p> <p><b>Body Composition Measures</b> - Impedance/; nuclear magnetic resonance imaging/; computer assisted tomography/; densitometry/; whole body counting/; Total body water/; plethysmography/.</p> <p><b>Socioeconomic Position</b> - socioeconomic/ or educational status/ or income group/ or poverty/; income/ or occupation/ or household income/; social status/ or social background/ or social class/; education/;</p>
<i>SPORTDiscuss</i>	<p><b>Body Composition</b> - ((DE "BODY composition" OR DE "HUMAN body composition") OR (DE "OBESITY")) OR (DE "ADIPOSE tissues")</p> <p><b>Body Composition Measures</b> - (((DE "BIOELECTRIC impedance") OR (DE "COMPUTED tomography")) OR (DE "MAGNETIC resonance imaging")) OR (DE "BONE densitometry")) OR (DE "PLETHYSMOGRAPHY")</p> <p><b>Socioeconomic Position</b> - ((DE "EDUCATION") OR (DE "EDUCATIONAL attainment")) OR (DE "HEALTH &amp; income")</p>
<b>Search Concept</b>	<b>Free Text Search Terms</b>
<i>Body composition</i>	<ol style="list-style-type: none"> <li>1. Body Composition MeSH Terms</li> <li>2. (Body adj3 (composition or distribution))</li> <li>3. ((fat or adipos*) adj3 (composition or distribution or mass or index or kg or total))</li> <li>4. ((muscl* or lean) adj3 (composition or distribution or mass or index or kg or total))</li> <li>5. ((fat-free) adj3 (mass or kg or total))</li> <li>6. ((android or gynoid or visceral or appendicular or abdominal or intra-abdominal) adj3 (fat or lean or muscle or mass or adipos*))</li> <li>7. 1 OR 2 OR 3 OR 4 OR 5 OR 6</li> </ol>
<i>Body composition measurement technique</i>	<ol style="list-style-type: none"> <li>8. Body Composition Measures MeSH Terms</li> <li>9. ((impedance) adj3 (bioelectrical or foot-to-foot or hand-to-foot or analy?is))</li> <li>10. (bioimpedance or body fat analy?er or body composition analy?er or tanita)</li> <li>11. (dual x-ray absorptiometry or DEXA or DXA or dual-energy X-ray absorptiometry)</li> <li>12. (magnetic resonance imaging or MRI)</li> <li>13. (Computed tomography or CT or CAT scan)</li> <li>14. (densitometry)</li> <li>15. ((neuron activation or total body counting or whole body counting))</li> <li>16. (total body water)</li> <li>17. (air-displacement plethysmography)</li> <li>18. 8 OR 9 OR 10 OR 11 OR 12 OR 13 OR 14 OR 15 OR 16 OR 17</li> <li>19. 7 AND 18</li> </ol>

<i>Socioeconomic position</i>	20. Socioeconomic Position MeSH terms
	21. (social class or social status or social position or socio-economic or socioeconomic or social circumstance*)
	22. (sociodemo*)
	23. Occupation*
	24. Educat*
	25. (income* or manual or class)
	26. (depriv* or poverty or overcrowding)
	27. 20 OR 21 Or 22 OR 23 OR 24 OR 25 OR 26
	28. 19 AND 27
	29. Limit to English Language (and Human in OvidSP)

**Table 5-1 Footnote:** MeSH terms are main heading descriptor terms available in each database and are determined by the indexing method adopted by each database. Free text search terms were entered into all databases, along with the results of the database specific MeSH terms.

**Table 5-2.** Tools and Techniques for Searching Databases

Technique and description	Command	Example
All known synonyms of key words		<i>socioeconomic position</i> may include <i>socio-economic, education, occupation, income</i> etc.
Replace up to one character in the word – allows alternative spellings to be included.	?	<i>Analy?er</i> would include both <i>Analyser</i> and <i>Analyzer</i>
Truncation command – used to acknowledge and capture alternative endings to words.	“root word”*	<i>Adipos*</i> would additionally search for <i>adiposity</i> and <i>adipose</i>
Boolean logic operators - used to a) identify results with at least one of the search terms present; and, b) to combine results of different search terms.	a) “OR” b) “AND”	a) <i>Muscle OR Lean Mass Index</i> would retrieve articles that have either term. b) <i>Body Composition AND Socioeconomic Position</i> would only retrieve articles with both terms.
Proximity operators - used to identify words within a specified distance of each other.	Ovid: adj3 ESBCO: n3	<i>Occupation* adj3 father*</i> would identify articles whereby “occupation” and “father” are within three words of each other.

#### 5.2.4 Data Extraction and Quality Assessment

Relevant information was double extracted using a data extraction form (Appendix 5.5). I completed extraction of all papers and four other researchers (two new researchers in addition to the two that conducted screening) split the role of second reviewer and completed the independent second extraction. Data extracted included citation details (author, title, publication year, publication type), study details (cohort or sample description, study design, country, participant numbers), participant details (birth year or age of participants, sex of participants), exposure and outcome details (type of SEP and body composition variables presented, age at which variables were recorded, how the variables were ascertained and measured) and statistical methods and information on adjustment for potential confounders and mediators. All available statistics relating to the association under study were extracted, along with statements of direction in text where statistics were not presented.

Assessment of study quality was carried out, using an amended version of the Newcastle-Ottawa Quality Assessment scale [225] (Appendix 5.6). Quality assessment was not used to exclude papers from the review, but to inform on the variability of quality across the papers and potential bias arising. The quality assessment form was amended after the protocol was published to account for the variability in statistical reporting and the large number of cross-sectional studies identified (Appendix 5.6: questions 3bi, 3bii and 4). Google Forms was used to aid extraction and WebPlotDigitizer [226] was used to extract data only presented in graphs.

The same four researchers split the role of second reviewer and worked independently to complete the quality assessment. Any disagreements were resolved through discussion.

#### 5.2.5 Synthesis

A meta-analysis was not possible due to the considerable variability in analytic methods used and presentation of results. As such, it was not possible to assess the degree of publication bias across studies through use of a funnel plot. Instead, a narrative synthesis was conducted, guided by the Economic and Social Research Council Methods Programme guidelines [227], with a focus on identifying and exploring sources of heterogeneity. Synthesis was conducted according to the groupings of: a) childhood SEP and childhood body composition b) adult SEP and adult body composition; and, c) childhood SEP and adult body composition.

Multiple relevant associations were frequently presented in a single paper. The individual association, as opposed to the paper, were thus considered the unit of analysis, similar to methods adopted by McLaren (2007) [112], and Ball and Crawford (2005) [228]. This will, however, have resulted in greater contribution of results from a single paper where multiple associations were reported.

Each association reported was categorised as either a positive association (those reporting greater socioeconomic advantage associated with higher body composition measure), negative association (those reporting greater socioeconomic advantage associated with lower body composition measures), non-linear association (curvilinear or heterogenous) or no association. Associations were assigned to groups based on the effect estimates and 95% confidence intervals. Where estimates were not reported, assignment was based on trends identified in descriptive data or statements of

direction reported in text alongside p-values. Use of p-values on their own only occurred if they indicated a non-significant relationship in the absence of information on the direction of association.

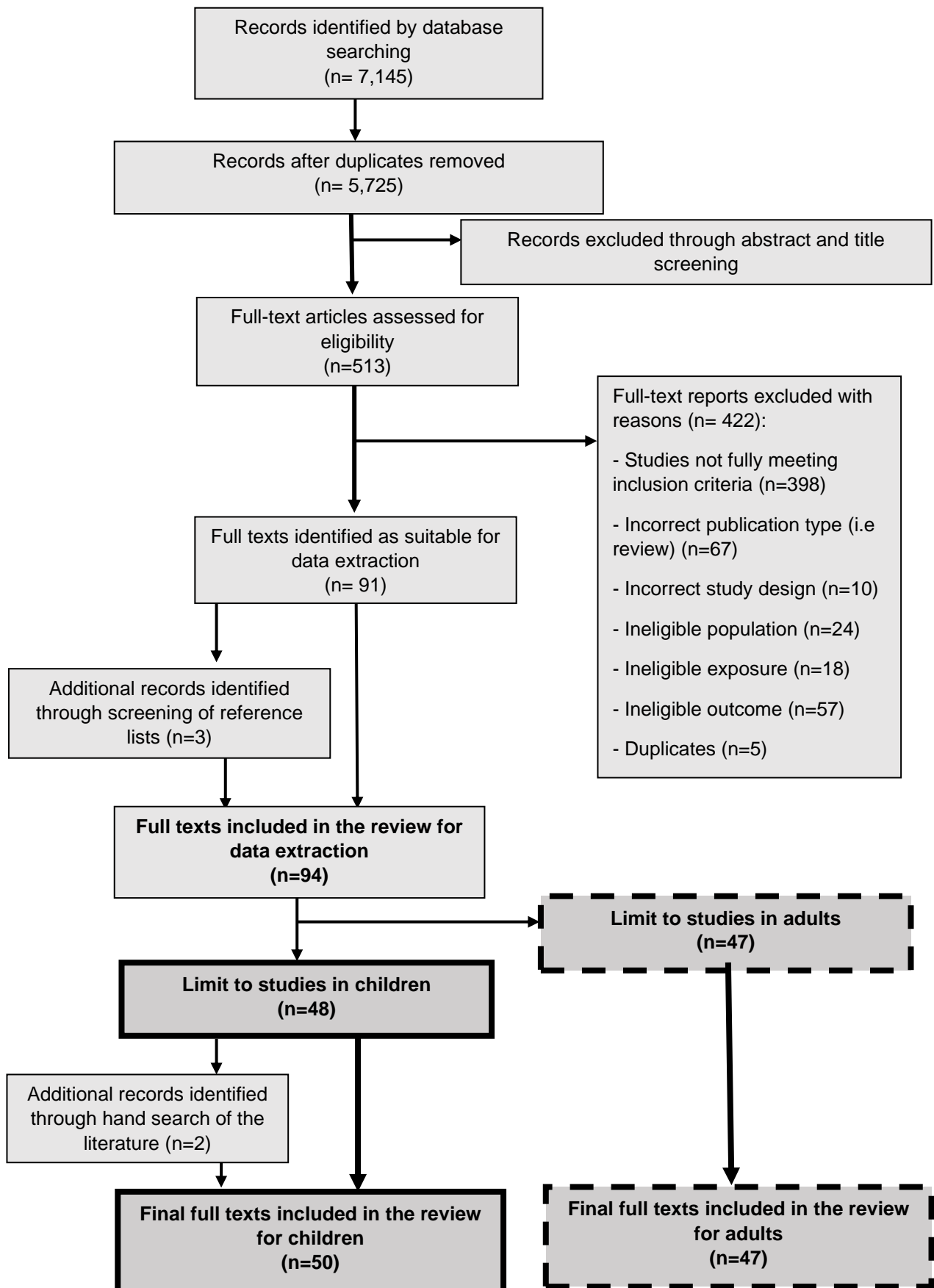
As outlined in the published protocol, heterogeneity in associations was explored according to body composition measure (FM, FFM, ratio and distribution), SEP measure and sex. Results from analyses using males and females combined were the primary results selected for summary. Where results were only presented for males and females separately, both associations were included in the summary results. It was not possible to investigate differences in body composition by birth year, as outlined in the protocol, due to lack of information provided on birth year across studies. On completion of screening, it became clear that country income level should also be considered a source of heterogeneity due to the large number of studies from middle-income countries and the hypothesised important influence on direction of associations. Thus, the protocol was updated to also investigate country income level as a source of heterogeneity. Studies were categorised into those in high-income countries (HIC), upper middle and lower middle-income countries, according to the World Bank classification in 2019 [229]. Those papers from upper middle and lower middle-income countries will all be referred to as “middle-income countries” (MIC).

### 5.3 Results

Figure 5-1 shows the study selection process, as outlined in the PRISMA flow chart. Searching the databases for potential papers returned 7,145 papers, with 5,725 once duplicates were removed. Title and abstract screening resulted in 513 papers being identified, with 91 papers remaining following full text screening. Searching the reference lists for additional papers returned 3, bringing the total included papers to 94. A further search of papers from key studies resulted in two further papers in children being identified, bringing the total number of included papers to 96. The final number of included papers in children was 50 [56, 141, 230-277], while 47 investigated either adult or childhood SEP and adult body composition [140, 260, 278-322]. One paper reported on body composition in both children and adults and was thus included in both groups.

Descriptive results and quality assessment for the included papers are shown in the Appendix (A5.7) separately for children and adults. The majority of papers across the review in both adults and children were rated as medium quality using the adapted Newcastle Ottawa assessment. In adults, 14 papers were rated as low quality ( $\leq 3^*$ ), and six papers were rated as high quality ( $\geq 7^*$ ), with the remaining 27 papers rated as medium. In children ten studies were rated as low quality and eight studies were rated as high quality. Those rated as high quality all presented full statistical results, including effect estimates and confidence intervals, whilst those rated as low quality typically had statistical reporting deemed inappropriate or incomplete. Only one paper presented p-values alone to report a non-significant result without provision of effect estimates, descriptive data or statement of direction in text.

Figure 5-1. PRISMA Flow Diagram





### 5.3.1 Characteristics of Included Studies in Children

There were 38 distinct samples studied across the 50 papers. ALSPAC and a sample from Merida, Mexico were the most commonly included studies, appearing in four and three separate papers, respectively. The majority of papers were conducted in population samples from HICs (N= 36, 72%) with the remaining papers from MICs (N=14). The UK and the US contributed the most papers (N=10 in the UK, N=8 in the US), with seven unique studies in both. Sample size across the papers ranged from 74 to 14,314, with a median sample size between 485 and 502.

There was substantial variation in body composition measures used, the definitions of which are outlined in Chapter 1, Table 1-1 (pg. 26). Fat measures were more frequently reported than FFM measures (in 46 papers compared to 22), with FM% being the most commonly analysed (29 papers). Among papers that investigated fat-free measures, LM was the most frequently used (8 papers). The majority of papers used either DXA (N=25), or BIA (N=22) to measure body composition, with two papers using both methods. Five studies used air-displacement plethysmograph with one of these also using BIA. One other paper used deuterium dilution in combination with DXA and plethysmography. The SEP variable most frequently reported was parental education (N=25).

A similar number of studies were conducted in children aged, or with a mean age, between four and ten (N=21) as were conducted in those children and adolescents over the age of ten (N=24). One study was conducted in new-borns and was the only study to be conducted in children under the age of four.

### 5.3.2 Characteristics of Included Studies in Adulthood

There were 40 distinct study samples across the 47 papers. The Study of Women's Health Across the Nation (SWAN), US was used in four papers. There were four other samples used in two papers each: Health 2000 Survey; Malmo Diet and Cancer study; The Korea National Health and Nutrition Examination Survey (KHANES); and the New England Family Study was used in addition to the Longitudinal Effects of Ageing on Perinatal (LEAP) project, which is a sub-set of the New England Family Study. The majority of papers were from Europe (N=18, 38%) or North America (N=14, 30%). There were 13 studies conducted in samples from the US and four each in Finland and the UK. There were 11 papers conducted in MICs. No studies from low-income countries (LICs) were identified. Sample sizes ranged from 86 to 162,691, with the median sample size between 629 and 637. One paper did not report sample size.

The majority of papers used DXA to measure body composition (N=22), with BIA analysis the next most common method (N=20). The remaining studies used underwater densitometry (N=1), CT scans (N=4), deuterium oxide dilution solution (total body water) (N=1), and abdominal ultrasonography (N=1). Two papers used more than one method.

Fat measures were considerably more frequently reported (in 30 papers) than fat-free measures (in 20 papers), with FM% the most frequently analysed (reported in 21 papers). LM was the most frequently used fat-free measure (in eight papers). The most frequently reported SEP variable was

education (in 32 papers). Composite SEP was used the same number of times as income (10 papers each). Occupational social class was used in seven papers, and area-level SEP in five papers.

The majority of papers contained analyses conducted in adults over 45 or in samples with a mean age over 45 (N=29). There were 13 studies where participants were either aged over 60 or had a mean age of 60+. Few cohort studies provided birth year (N=5), or information from which this could be calculated, preventing assessment of secular differences in body composition by birth cohort as specified in the protocol.

### 5.3.3 Childhood SEP and Childhood Body Composition

#### 5.3.3.1 Total Body Fat Mass

Table 5-3 provides a summary of the patterns of association reported for total FM measures. The non-linear group was removed from the summary tables for the childhood results, similar to the approach of McLaren [112], but only one association fell into this category. There were 124 associations tested across the 46 papers. Negative associations, where more advantaged SEP was associated with lower fat, were reported most often, in 42% (52 association across 31 papers) of the 124 associations and this was driven by predominantly negative associations in HICs. The remaining associations were split between positive associations (27%, 33 associations from seven papers), where more advantaged SEP was associated with greater fat, and no association (31%, 39 association from 19 papers).

#### Heterogeneity by Country Income Level

In HICs, associations were predominantly negative (66%, 46 associations from 28 papers) with greater socioeconomic advantage being associated with less fat. The remaining associations in HICs all showed no overall pattern of association (24 association from 13 papers). In MICs, the majority of associations were positive (61%) with greater socioeconomic advantage associated with higher levels of fat. Only 11% (six associations from three papers) reported negative associations, with the remaining associations (28%, 15 associations from six papers) reporting no overall pattern.

#### Heterogeneity by Body Composition Measures

The total body fat measure most frequently reported was FM%, being used 60 times (across 29 papers), followed by FM used 38 times (18 papers), and FMI used 26 times (13 papers). In HICs, using FM or FM% yielded a slightly greater number of negative associations, where greater advantage is related to lower levels of fat, (67% and 68% respectively) compared to FMI (60%). In MICs, FM and FMI presented almost exclusively positive associations, where greater advantage is related to higher levels of fat (79% and 82% respectively). FM% exhibited more mixed results with 45% finding positive associations, 17% finding negative and the remaining 38% finding no association.

#### Heterogeneity by Socioeconomic Position Measures

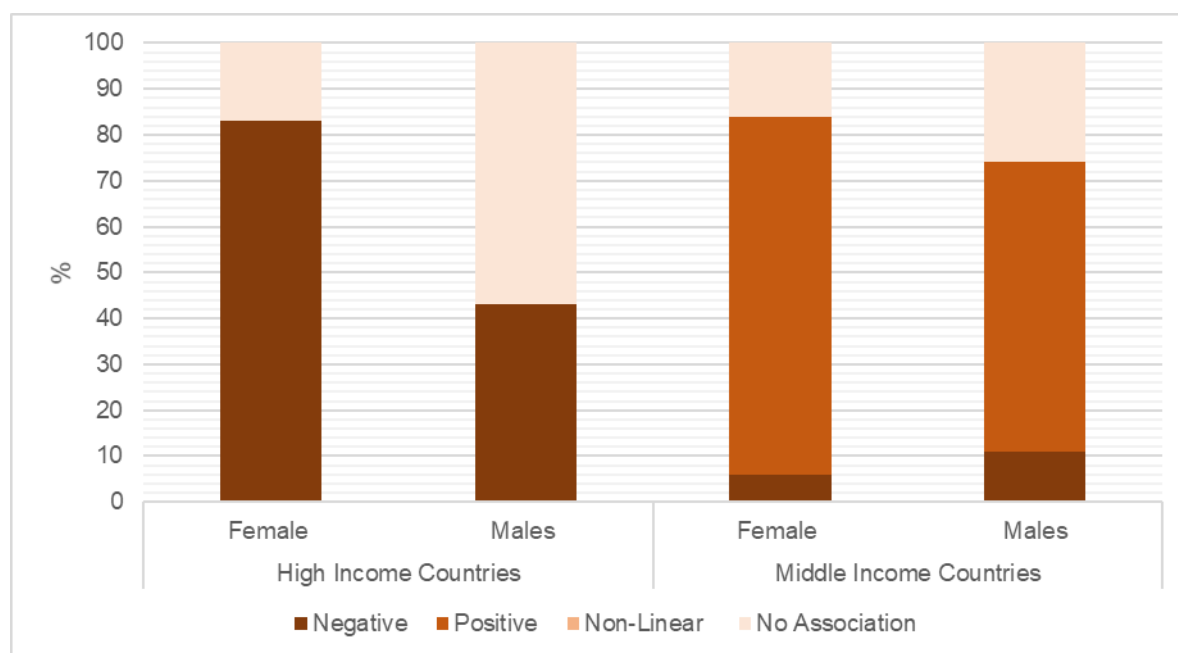
Parental education was the most commonly used SEP measure across the papers, used in 53 associations across 24 papers. In MICs, composite measures of SEP were the most frequently

recorded SEP measure, used in 22 associations across nine papers. Among HICs, negative associations were reported in the majority of associations ( $\geq 60\%$ ) for all SEP measures, with the exception of parental or household income, used in six papers, where no association was most frequently reported (6 association from four papers, out of 10 associations). In MICs, parental education, used in seven papers, yielded a higher number of positive associations (15 association from five papers, out of 18 associations, 83%) compared with composite SEP measures (14 associations from four papers, out of 22 associations, 64%).

### Heterogeneity by Sex

Sex-specific analysis was presented in 15 papers in HICs and eight papers in MICs, with 44 and 37 associations reported, respectively (Figure 5-2). Negative associations were more frequently reported among girls (83%) compared with boys (43%) in HICs. In MICs positive associations were somewhat more commonly reported in girls (78%) compared to boys (63%). Boys in both HICs and MICs were more likely to show no association between SEP and fat measures (HIC: 57%; MIC: 26%) compared to girls in either (HIC: 19%; MIC: 17%).

**Figure 5-2.** Distribution of Associations for Fat Measures by Gender and Income Level



**Figure 5-2. Footnotes**

Girls in HICs (N=23): 83% negative associations, 0% positive associations, 0% non-linear associations, 17% show no association; Boys in HICs (N=21): 43% negative associations, 0% positive associations, 0% non-linear associations, 57% show no association; Girls in MICs (N= 18) 6% negative associations, 78% positive associations, 0% non-linear associations, 17% show no association; Boys in MICs (N= 9) 11% negative associations, 63% positive associations, 0% non-linear associations, 26% show no association.

**Table 5-3.** Summary of Associations between Socioeconomic Position and Fat Measures in Children

SEP Indicator	Direction of SEP and Body Composition Association									Total	
	Positive Association			Negative Association			No clear/strong direction			N	%
	N	%	References	N	%	References	N	%	References		
<i>Body Fat Percentage</i>											
Parental Education	6	26%	[277] <sup>M\$</sup> [277] <sup>F\$</sup> [274] <sup>\$</sup> [261] <sup>\$</sup> [270] <sup>M\$</sup> [270] <sup>F\$</sup>	9	43%	[233] <sup>F</sup> [234] <sup>F</sup> [245] <sup>F</sup> [244] <sup>F\$</sup> [249] <sup>M</sup> [249] <sup>F</sup> [56, 248, 260]	8	30%	[233] <sup>M</sup> [234] <sup>M</sup> [245] <sup>M</sup> [244] <sup>M\$</sup> [262] <sup>\$</sup> [246] <sup>F</sup> [246] <sup>M</sup> [254]	23	19%
Composite SEP	4	33%	[270] <sup>M\$</sup> [270] <sup>F\$</sup> [277] <sup>M\$</sup> [277] <sup>F\$</sup>	3	25%	[243] <sup>M\$</sup> [259, 276]	5	41%	[244] <sup>\$</sup> [242] <sup>\$</sup> [238] <sup>F\$</sup> [238] <sup>M\$</sup> [273]	12	10%
Occupational Social Class	1	20%	[274] <sup>\$</sup>	3	60%	[233] <sup>F</sup> [244] <sup>\$</sup> [250]	1	20%	[233] <sup>M</sup>	5	4%
Income	1	16%	[274] <sup>\$</sup>	2	33%	[272] <sup>F</sup> [272] <sup>M</sup>	3	50%	[262] <sup>\$</sup> [234] <sup>F</sup> [234] <sup>M</sup>	6	5%
Area or school level SEP <sup>5</sup>	0	-	-	3	75%	[247, 264, 269]	1	25%	[261] <sup>\$</sup>	4	3%
PIR	0	-	-	3	100%	[266] <sup>F</sup> [266] <sup>M</sup> [267]	0	-	-	3	2%
School Type	0	-	-	2	66%	[244] <sup>F\$</sup> [262] <sup>\$</sup>	1	33%	[244] <sup>M\$</sup>	3	2%
Miscellaneous	1	25%	[274] <sup>\$</sup>	1	25%	[271] <sup>F</sup>	2	50%	[262] <sup>\$</sup> [242] <sup>\$</sup>	4	3%
<i>Body Fat (kg)</i>											
Parental Education	5	25%	[277] <sup>M\$</sup> [277] <sup>F\$</sup> [270] <sup>M\$</sup> [270] <sup>F\$</sup> [252] <sup>\$</sup>	11	55%	[249] <sup>M</sup> [249] <sup>F</sup> [234] <sup>F</sup> [239] <sup>F</sup> [233] <sup>F</sup> [255] <sup>F</sup> [255] <sup>M</sup> [256] <sup>F</sup> [248, 260, 268]	4	20%	[234] <sup>M</sup> [233] <sup>M</sup> [256] <sup>M</sup> [235]	20	16%
Composite SEP	5	83%	[277] <sup>M\$</sup> [277] <sup>F\$</sup> [270] <sup>M\$</sup> [270] <sup>F\$</sup> [258] <sup>F\$</sup>	1	17%	[243] <sup>M\$</sup>	0	-	-	6	5%
Income	0	-	-	2	40%	[230] <sup>M</sup> [230] <sup>F</sup>	3	60%	[234] <sup>M</sup> [234] <sup>F</sup> [235]	5	4%
Occupational Social Class	0	-	-	3	75%	[233] <sup>F</sup> [250, 265]	1	25%	[233] <sup>M</sup>	4	3%
Miscellaneous	1	33%	[252] <sup>\$</sup>	0	-	-	2	66%	[252] <sup>\$</sup> [252] <sup>\$</sup>	3	2%
<i>Fat Mass Index</i>											
Parental Education	4	40%	[277] <sup>M\$</sup> [277] <sup>F\$</sup> [270] <sup>M\$</sup> [270] <sup>F\$</sup>	4	40%	[246] <sup>F</sup> [56, 237, 257]	2	40%	[246] <sup>M</sup> [232]	10	8%
Composite SEP	5	63%	[277] <sup>M\$</sup> [277] <sup>F\$</sup> [270] <sup>M\$</sup> [270] <sup>F\$</sup> [253] <sup>\$</sup>	1	12.5%	[275]	2	25%	[231] <sup>F\$</sup> [231] <sup>M\$</sup>	8	7%

PIR	0	-	-	3	100%	[266] <sup>M</sup> [266] <sup>F</sup> [267]	0	-	-	3	2%
Occupational Social Class	0	-	-	1	33%	[240] <sup>M</sup>	2	66%	[240] <sup>F</sup> [232]	3	2%
Miscellaneous	0	-	-	0	-	-	1	50%	[237, 241]	2	2%
<b>Overall Distribution of Associations - Fat Measures Combined</b>											
<b>Combined SEP</b>	27%			42%			31%			124	100%
<b>HIC Combined SEP</b>	0%			66%			34%			70	56%
<b>MIC Combined SEP</b>	61%			11%			28%			54	44%

**Table 5-3 Footnote:** <sup>F</sup> Indicates results for girls only; <sup>M</sup> indicates results for boys only; <sup>S</sup> indicates study conducted in MIC. Positive associations indicate an increase in fat measure with an increase in socioeconomic advantage; negative associations indicate a decrease in fat measure with an increase in socioeconomic advantage. Miscellaneous SEP measures are where less than two papers reported on the measure. Total N represents the total number of reported associations between the given SEP measure and the body composition measure (i.e total number of associations reporting on education and FM). There may be two associations from one paper per SEP measure, if only sex-stratified data is presented. The N for the direction of relation groups (positive, negative, non-linear, no association), refers to the number of associations reporting each patterning within the given SEP measure and body composition measure combination (i.e number of positive associations reported between education and FM), and corresponds to the references included. The % for the direction of relation groups indicates the number of associations reporting a particular patterning (i.e positive) as a percentage of the total number of associations for the given SEP measure and body composition measure (i.e education and FM).

### 5.3.3.2 Total Body Fat-Free Mass Measures

Table 5-4 provides a summary of the patterns of association for total body FFM measure. There were 69 associations tested across 22 papers. Approximately half (33 associations in 13 papers) found positive associations (48%), with greater socioeconomic advantage being related to greater FFM. Only 12% demonstrated negative associations, with the remaining 41% reporting no association.

#### Heterogeneity by Country Income Level

Positive associations were reported more frequently in HICs (55%) compared to MICs (43%), whilst negative associations were only reported in MICs (20%).

#### Heterogeneity by Body Composition Measures

Raw fat-free measures, used in 14 papers, show positive associations in 59% of analyses (20 associations coming from nine papers, out of 34 associations). This was more frequently than both percentage measures (50%, 11 associations coming from six papers, out of 22 associations across eight papers) and considerably more often than indexed measures (15%, two associations coming from two papers, out of 13 associations across five papers). Measures that include bone in their assessment show positive associations slightly more often (54%) than those which exclude bone (42%).

#### Heterogeneity by Socioeconomic Position Measures

Parental education was the most frequently investigated SEP measure, used in 24 associations. Composite measures of SEP were also frequently used (N=18), with all except one such association tested in MICs. Parental occupational social class and measures of area-level SEP were used in eight and nine associations respectively, although for area-level SEP, eight were all tested in the same paper. Parental income was used three times in two papers. In MICs there was a slightly higher number of positive associations reported when using composite measures of SEP (53%) compared to education (40%). In HICs, approximately one-third of associations with both education and occupational social class were observed to be positive. In the small number of analyses including area-level SEP and income in HICs, only positive associations were seen.

#### Heterogeneity by Sex

In both HICs and MICs, only five papers presented sex-specific analysis, with 15 and 31 associations reported, respectively. Positive associations were reported 86% of the time in boys, and 88% of the time in girls (Figure 5-3). Results for sex-specific analysis in MICs was more similar to the pooled results, although there were slightly greater number of positive associations in boys (53%) compared with girls (46%).

**Table 5-4.** Summary of Associations between Socioeconomic Position and Fat-Free Measures in Children

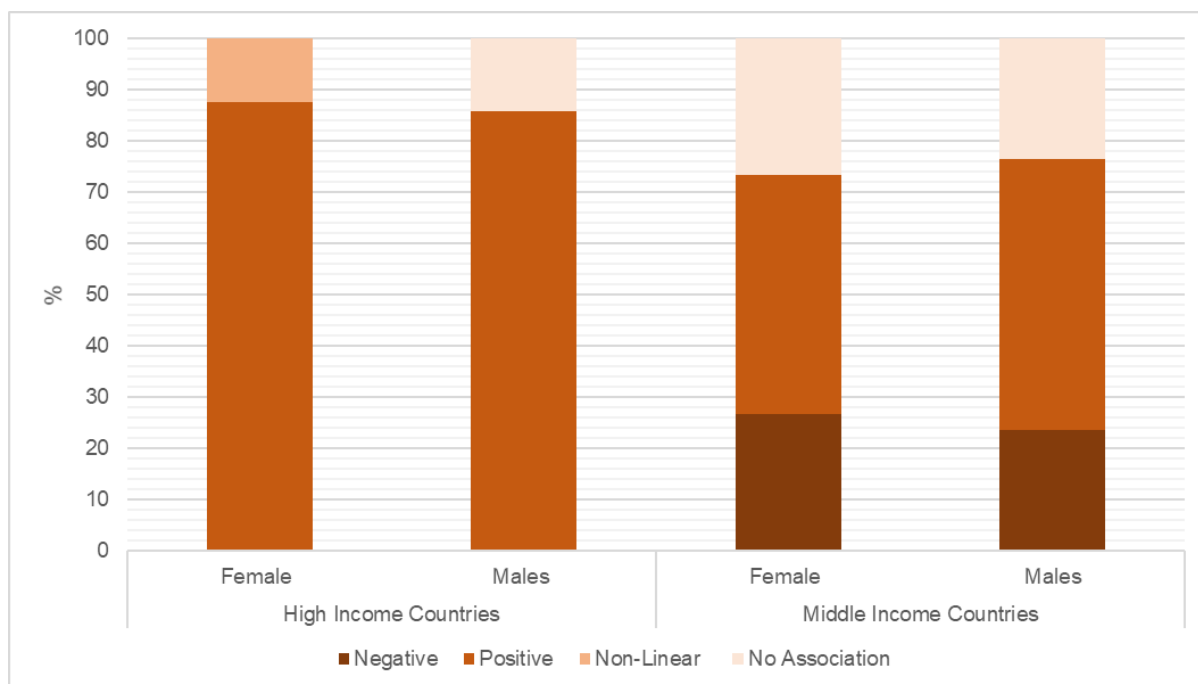
SEP Indicator	Direction of SEP and Body Composition Association									Total	
	Positive Association			Negative Association			No clear direction			N	%
	N	%	References	N	%	References	N	%	References		
<i>Fat-Free Mass</i>											
Miscellaneous	7	100%	[270] <sup>M</sup> [270] <sup>F</sup> [270] <sup>M</sup> [270] <sup>F</sup> [263] <sup>M</sup> [263] <sup>F</sup> [250]	0	-	-	0	-	-	7	10%
<i>Fat-Free Mass %</i>											
SEP	1	25%	[243] <sup>M</sup>	2	50%	[270] <sup>M</sup> [270] <sup>F</sup>	1	25%	[244] <sup>S</sup>	4	6%
Parental Education	1	25%	[244] <sup>F</sup>	2	50%	[270] <sup>M</sup> [270] <sup>F</sup>	1	25%	[244] <sup>M</sup>	4	6%
Miscellaneous	3	75%	[263] <sup>M</sup> [263] <sup>F</sup> [244] <sup>F</sup>	0	-	-	1	25%	[244] <sup>M</sup>	4	6%
<i>Fat-Free Mass Index</i>											
Occupational Social Class	1	33%	[240] <sup>F</sup>	0	-	-	2	66%	[240] <sup>M</sup> [232]	3	4%
Parental Education	0	-	-	0	-	-	3	100%	[56] <sup>L</sup> [232, 237]	3	4%
Miscellaneous	1	50%	[237]	0	-	-	1	50%	[275]	2	3%
<i>Dry Lean Mass</i>											
SEP	3	100%	[244] <sup>S</sup> [243] <sup>M</sup> [242] <sup>S</sup>	0	-	-	0	-	-	3	4%
Miscellaneous	2	40%	[244] <sup>M</sup> [244] <sup>M</sup>	0	-	-	3	60%	[244] <sup>F</sup> [244] <sup>F</sup> [242] <sup>S</sup>	5	7%
<i>Lean Mass</i>											
Parental Education <sup>1</sup>	3	43%	[277] <sup>F</sup> [277] <sup>M</sup> [260]	0	-	-	4	57%	[239] <sup>F</sup> [252] <sup>S</sup> [233, 251]	7	10%
SEP	3	100%	[277] <sup>F</sup> [277] <sup>M</sup> [258] <sup>F</sup>	0	-	-	0	-	-	3	4%
Occupational Social Class	0	-	-	0	-	-	3	100%	[233, 251, 265]	3	4%
Miscellaneous	0	-	-	0	-	-	4	100%	[252] <sup>S</sup> [252] <sup>S</sup> [252] <sup>S</sup> [251]	4	6%
<i>Lean Mass %</i>											
Parental Education	1	33%	[260]	2	66%	[277] <sup>F</sup> [277] <sup>M</sup>	0	-	-	3	4%
SEP	0	-	-	2	100%	[277] <sup>F</sup> [277] <sup>M</sup>	0	-	-	2	3%
<i>Lean Mass Index</i>											
SEP	0	-	-	0	-	-	3	100%	[253] <sup>S</sup> [277] <sup>F</sup> [277] <sup>M</sup>	3	4%
Parental Education	0	-	-	0	-	-	2	100%	[277] <sup>F</sup> [277] <sup>M</sup>	2	3%
<i>Appendicular Skeletal Muscle</i>											
School Level SEP	2	100%	[263] <sup>F</sup> [263] <sup>M</sup>	0	-	-	0	-	-	2	3%

<i>Appendicular Skeletal Muscle %</i>											
Miscellaneous	5	100%	[141] <sup>F</sup> [141] <sup>M</sup> [263] <sup>F</sup> [263] <sup>M</sup> [254]	0	-	-	0	-		5	7%
<b>Overall Distribution of Associations - Fat-Free Measures Combined</b>											
<b>Combined SEP</b>		48%			12%			41%		69	100%
<b>HIC Combined SEP</b>		55%			0%			45%		29	42%
<b>MIC Combined SEP</b>		43%			20%			38%		40	58%

**Table 5-4 Footnotes:** <sup>F</sup> Indicates results for girls only; <sup>M</sup> indicates results for boys only; <sup>S</sup> indicates study conducted in MICs. Positive associations indicate an increase in fat measure with an increase in socioeconomic advantage; negative associations indicate a decrease in fat measure with an increase in socioeconomic advantage. Miscellaneous SEP measures are where less than two papers reported on the measure. L indicates lean body mass instead of FFM. Total N represents the total number of reported associations between the given SEP measure and the body composition measure (i.e total number of associations reporting on education and FFM). There may be two associations from one paper per SEP measure, if only sex-stratified data is presented. The N for the direction of relation groups (positive, negative, non-linear, no association), refers to the number of associations reporting each patterning within the given SEP measure and body composition measure combination (i.e number of positive associations reported between education and FFM), and corresponds to the references included. The % for the direction of relation groups indicates the number of associations reporting a particular patterning (i.e positive) as a percentage of the total number of associations for the given SEP measure and body composition measure (i.e education and FFM).



**Figure 5-3.** Distribution of Associations for Fat-Free Measures by Sex and Income Level



**Figure 5-3. Footnote**

*Girls in HICs (N=8): 0% negative associations, 87.5% positive associations, 12.5% non-linear associations, 0% show no association; Boys in HICs (N=7): 0% negative associations, 86% positive associations, 0% non-linear associations, 14% show no association; Girls in MICs (N= 15) 27% negative associations, 47% positive associations, 0% non-linear associations, 27% show no association; Boys in MICs (N= 17) 24% negative associations, 53% positive associations, 0% non-linear associations, 24% show no association.*

**5.3.3.3 Ratio and Distribution Measures**

Five papers reported on the association between SEP and a ratio or distribution measure (Table 5-5). Two papers used the same sample from the US and the remaining papers reported results from the UK (N=2) and Brazil (N=1).

Four papers looked at the association between SEP and a measure of central fat. Two of these using the same sample found greater social advantage to be associated with decreases in trunk FM, total abdominal adipose tissue [236] and trunk FMI [275]. The other two papers found no association between any SEP variable considered and central fat [261, 265]. Only one paper looked at a ratio measure and found a lower mean muscle to fat ratio in lower parental income groups, except in girls aged 11-14 [263].

**Table 5-5.** Results of Associations between Socioeconomic Position and Childhood Ratio and Distribution Measures

<b>Paper</b>	<b>Country</b>	<b>N</b>	<b>Age</b>	<b>Study or Description of the Population</b>	<b>SEP Measure</b>	<b>Body Composition measure</b>	<b>Findings</b>
Willig (2011) [275]	USA	254	7-12	Children from ongoing cross-sectional study, whose parents classified them through self-report as either African American, European American, or Hispanic American.	SEP	Trunk FMI	SEP was negatively associated with trunk FMI (all at $p < 0.05$ ). Increase in advantage was associated with decreases trunk FMI.
Cardel (2012) [236]	USA	267	7-12	Children self-identifying as African American, European American, or Hispanic American from Birmingham, Alabama area.	SEP	Trunk FM, TAAT	Increase in social advantage associated with decreases in central adiposity.
Ness (2005) [265]	UK	5,917	10	ALSPAC	Social Occupational Class	Trunk FM	No Association
Magalhaes (2012) [261]	Brazil	183	4-7	Children aged 4-7 from a retrospective cohort who were monitored for the first months of life by a support program to breastfeeding (PROLAC) in the city of Vicoso, southeast Brazil.	Mothers education, Income per capita	%Android Fat	No Association
McCarthy (2015) [263]	UK	2,297	5-14	Caucasian children from inner city London and from more affluent surrounding counties.	School Level SEP	Muscle:Fat Ratio	Lower muscle to fat ratio in low-income groups in all age groups, except for females aged 11-14.

**Table 5-5 Footnotes:** SEP – Socioeconomic Position; FMI – Fat Mass Index; TAAT – Total abdominal adipose tissue; ALSPAC – Avon Longitudinal Study of Parents and Children.

### 5.3.4 Adult Socioeconomic Position and Adult Body Composition

There were 46 papers reporting on the association between SEP in adulthood and body composition in adulthood. Of these, 16 reported only in females, one only in males and the rest in both males and females.

#### 5.3.4.1 Total Body Fat Measures

Table 5-6 provides a summary of the patterns reported for each fat measure. There were 75 associations tested across 30 papers. Nearly half of associations (33 associations from 17 papers) between SEP and fat measures reported were negative (44%). Similar numbers (13 associations from seven papers) reported non-linear (17%), made up of heterogeneous and curvilinear associations, as reported positive (11%) patterning (eight associations from five papers), and almost a third of papers reported no association (28%, 21 associations from 14 papers).

#### Heterogeneity by Country Income Level

There were clear differences between findings in HICs and MICs. In HICs (54 associations from 20 papers), 32 associations from 16 papers were negative (59%) with no positive associations observed. In MICs (21 associations from ten papers) eight associations from five papers found positive associations (38%) and nine association from five papers found no association (43%), with only 5% reporting negative associations (three associations).

#### Heterogeneity by Body Composition Measure

FM% was used 44 times across 21 papers, FMI was used 19 times across five papers and FM 12 times across ten papers. Each fat measure showed predominantly negative associations, and this was driven by negative associations in HICs. In HICs negative associations were reported more frequently for FM (78%, seven association from five papers out of nine associations from seven papers) compared to FM% (58%, 18 association from 11 papers out of 31 association from 14 papers) and FMI (50%, seven out of 14 association from two papers). Non-linear associations (including both heterogeneous and curvilinear) were more frequently reported for FMI (43%, six associations from two papers) than for other measures. In MICs, FM% and FMI predominantly showed positive associations (38% and 40% respectively) and no association (38% and 60% respectively). FM was used only three times from three different papers in MICs with all patterns of association being different.

#### Heterogeneity by Socioeconomic Position Measure

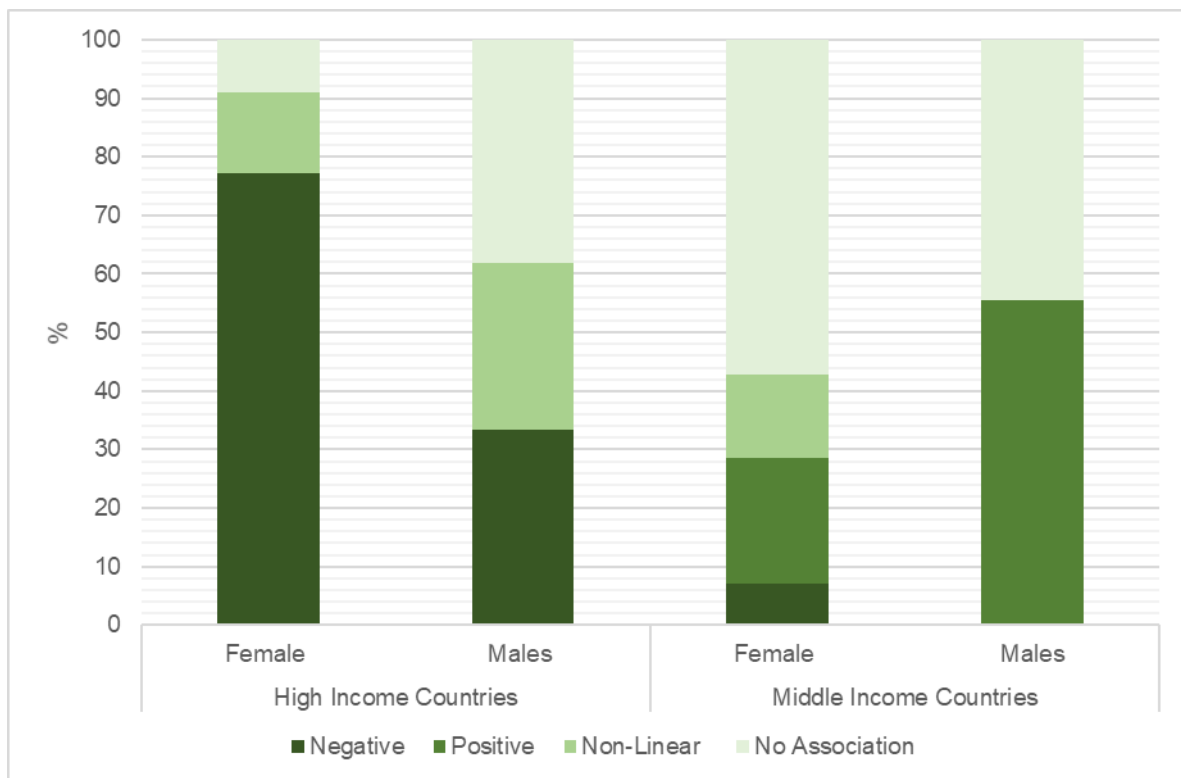
For education, the most commonly studied SEP measure, 12 of 29 associations showed a negative association with a measure of fat (41%), again driven by findings in HICs. In MICs, education showed predominantly no association (six out of nine associations, from seven papers). Negative associations were predominantly observed for occupational social class (six out of ten), but the measure was used almost exclusively in HICs (in eight associations from five papers). For area-level SEP, which was used only in HICs, predominantly negative associations (six out of eight, from five papers) were reported. In contrast, four out of nine associations with composite SEP showed positive associations. All of those reporting positive associations were conducted in MICs. Four of the remaining five studies

that showed either negative or no-association were conducted in HICs. Non-linear associations were most frequently reported in studies investigating income in both HICs and MICs (in two out of three associations in both).

### Heterogeneity by Sex

In studies that presented sex-stratified or sex-specific analysis (66 associations across 22 papers), negative associations were reported over twice as frequently in females (77%) compared to males (33%) in HICs. There was a higher proportion of non-linear associations in males (29%) compared to females (14%) (Figure 5-4). In studies from MICs, no association was more commonly reported in women (57%) compared with men (44%). Positive associations were more commonly reported in men (55%) than women (21%).

**Figure 5-4.** Distribution of Associations for Fat Measures by Sex and Income Level



**Figure 5-4. Footnotes**

*Females in HICs (N=22): 77% negative associations, 0% positive associations, 14% non-linear associations, 9% show no association; Males in HICs (N=21): 33% negative associations, 0% positive associations, 29% non-linear associations, 38% show no association; Females in MICs (N= 14) 7% negative associations, 21% positive associations, 14% non-linear associations, 57% show no association; Males in MICs (N= 9) 0% negative associations, 56% positive associations, 0% non-linear associations, 44% show no association.*

**Table 5-6.** Summary of Associations between Socioeconomic Position and Fat Measures in Adults in High-Income Countries and Middle-Income Countries.

SEP Indicator	Direction of Relation Between SEP and Fat Measure												Total	
	Positive			Negative			Non-Linear			No Relation				
	N	%	References	N	%	References	N	%	References	N	%	References	N	%
<i>Fat Mass (kg)</i>														
Education	0	-	-	2	40%	[283] <sup>F</sup> [280]	1	20%	[317]	2	40%	[286] <sup>F</sup> [299]	5	7%
SEP	1	50%	[310] <sup>M</sup> §	1	50%	[280]	0	-	-	0	-	-	2	3%
Area-Level SEP	0	-	-	3	100%	[294] <sup>F</sup> [288] <sup>F</sup> [280]	0	-	-	0	-	-	3	4%
Miscellaneous	0	-	-	1	50%	[260]	1	50%	[301] <sup>SF</sup>	0	-	-	2	3%
<i>Fat Mass %</i>														
SEP	2	40%	[307] <sup>F</sup> § [310] <sup>M</sup> §	1	20%	[314] <sup>F</sup>	0	-	-	2	40%	[314] <sup>M</sup> [321]	5	7%
Education	1	6%	[290] <sup>S</sup>	8	44%	[320] <sup>F</sup> [304] <sup>F</sup> [289] <sup>F</sup> [297] <sup>F</sup> § [317] <sup>F</sup> [303, 311, 315]	2	11%	[320] <sup>M</sup> [304] <sup>M</sup>	7	38%	[289] <sup>M</sup> [318] <sup>M</sup> [317] <sup>M</sup> [296] <sup>M</sup> § [296] <sup>F</sup> § [319] <sup>F</sup> § [281] <sup>F</sup> §	18	24%
Occupational Social Class	0	-	-	4	57%	[302] <sup>F</sup> [303] <sup>F</sup> [322] <sup>M</sup> [322] <sup>F</sup>	0	-	-	3	43%	[296] <sup>F</sup> § [302] <sup>M</sup> [318] <sup>M</sup>	7	9%
Income	0	-	-	1	33%	[315]	2	33%	[290] <sup>S</sup> [319] <sup>F</sup> §	0	-	-	3	4%
Area-Level SEP	0	-	-	2	66%	[304] <sup>F</sup> [304] <sup>M</sup>	0	-	-	1	33%	[293]	3	4%
Miscellaneous	2	25%	[296] <sup>M</sup> § [296] <sup>F</sup> §	3	38%	[304] <sup>F</sup> [304] <sup>M</sup> [260]	1	13%	[304] <sup>M</sup>	2	25%	[304] <sup>F</sup> [318] <sup>M</sup>	8	11%
<i>Fat Mass Index</i>														
Education	1	17%	[297] <sup>F</sup> §	2	33%	[285] <sup>F</sup> [285] <sup>M</sup>	2	33%	[304] <sup>F</sup> [304] <sup>M</sup>	1	17%	[313] <sup>S</sup>	6	8%
Income	0	-	-	0	-	-	2	66%	[285] <sup>F</sup> [285] <sup>M</sup>	1	33%	[313] <sup>S</sup>	3	4%
Miscellaneous	1	10%	[310] <sup>M</sup> §	5	50%	[304] <sup>F</sup> [304] <sup>M</sup> [304] <sup>M</sup> [285] <sup>F</sup> [285] <sup>M</sup>	2	20%	[304] <sup>F</sup> [304] <sup>M</sup>	2	20%	[304] <sup>F</sup> [313] <sup>S</sup>	10	13%
<b>Overall Distribution of Associations – Fat Measures Combined</b>														
<b>Combined SEP</b>	11%			44%			17%			28%			75	100%
<b>HIC Combined SEP</b>	0%			59%			19%			22%			54	72%
<b>MIC Combined SEP</b>	38%			5%			14%			43%			21	28%

**Table 5-6 Footnote:** *F* Indicates results for females only; *M* indicates results for males only; Positive associations indicate an increase in fat measure with an increase in socioeconomic advantage; negative associations indicate a decrease in fat measure with an increase in socioeconomic advantage; non-linear indicate the association between SEP and fat measures is either curvilinear or heterogeneous. Miscellaneous SEP measures are where less than two papers reported on the measure. Total N represents the total number of reported associations between the given SEP measure and the body composition measure (i.e total number of associations reporting on education and FM). Total N represents the total number of reported associations between the given SEP measure and the body composition measure (i.e total number of associations reporting on education and FM). There may be two associations from one paper per SEP measure, if only sex-stratified data is presented. The N for the direction of relation groups (positive, negative, non-linear, no association), refers to the number of associations reporting each patterning within the given SEP measure and body composition measure combination (i.e number of positive associations reported between education and FM), and corresponds to the references included. The % for the direction of relation groups indicates the number of associations reporting a particular patterning (i.e positive) as a percentage of the total number of associations for the given SEP measure and body composition measure (i.e education and FM).

#### 5.3.4.2 Total Body Fat-Free Measures

Table 5-7 provides a summary of the patterns identified for each of the different fat-free measures. There were 44 associations tested across 19 papers. The majority found no association between SEP and fat-free measures (55%, 24 associations from 14 papers), whilst 32% (14 associations from eight papers) found evidence of positive associations, 7% found negative and 7% found non-linear associations.

#### Heterogeneity by Country Income Level

More associations were tested in HICs (30 association across 14 papers) compared to MICs (14 associations across five papers), and patterns of association were similar in both settings.

#### Heterogeneity by Body Composition Measure

LM was used ten times across eight papers while FFM, Appendicular skeletal muscle (ASM) and ASM index (ASMI) were used nine to 11 times each across three to six papers. LM%, lean mass index (LMI), and FFMI were used in one paper each. FFM showed a greater frequency of positive associations (50%) while for all other measures no association was most commonly observed. ASMI reported a higher number of positive associations (36%) compared to LM and ASM. LM and ASM found positive associations in 10-30% of associations. Negative and non-linear patterns were reported in approximately 0-20% of the associations for LM, FFM, ASM and ASMI.

For measures that include bone (FFM and LBM) (N=11), there was a higher proportion of positive associations reported (45%) compared to measures that did not include bone i.e. LM (N=33, 27%). There was little difference in the distribution of associations between those that used index or percentage measures compared to those that used raw measures. Two papers made additional adjustments [285, 316], for either FM or body size which resulted in associations reversing in direction to become positive or increasing in strength.

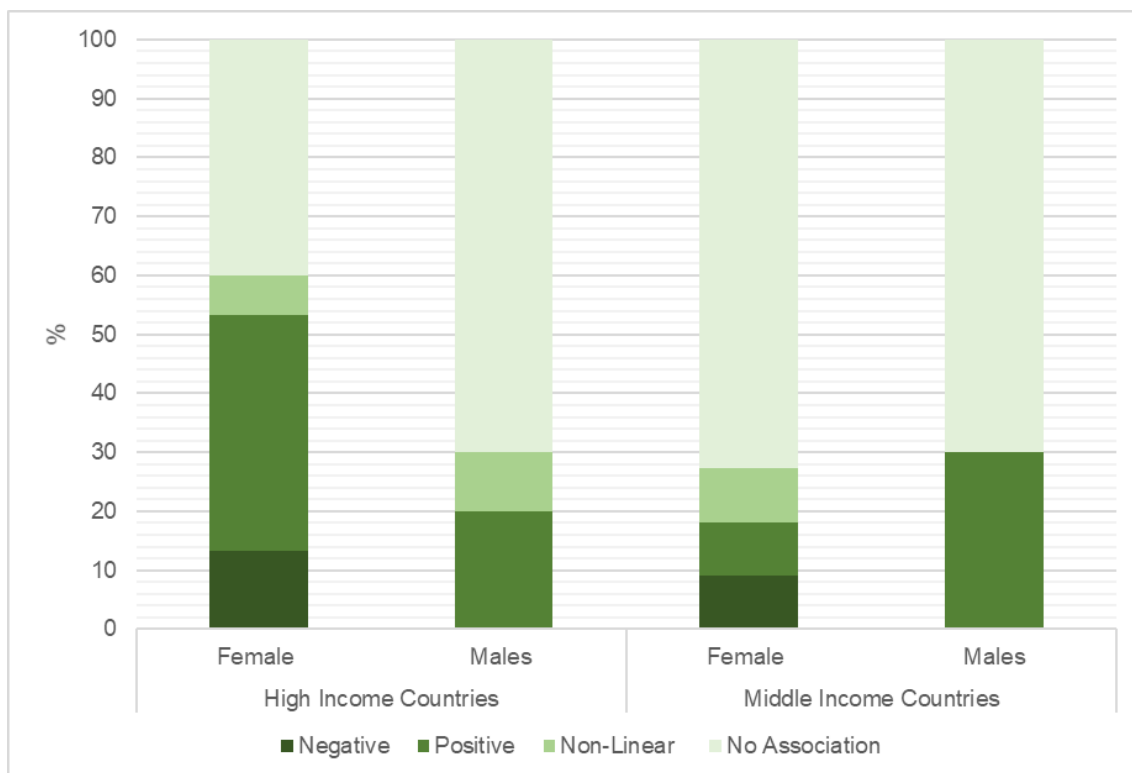
## Heterogeneity by Socioeconomic Position Measure

For all measures of SEP, most studies reported no association with fat-free measures. Positive associations were more frequently reported for education (35%, 8 associations of 23) and income (29%, two associations out of seven) compared with social class (17%, one association out of six). Income showed a higher percentage of non-linear associations (29%) and a lower number of no associations (43%) compared to all other SEP measures. Composite measures of SEP were used twice, finding a positive association in one paper and no association in the other. Area-level SEP was used once, finding no association.

## Heterogeneity by Sex

Among studies that presented sex-specific or sex-stratified analysis (46 associations across 16 papers), there was greater variability in the patterns of associations observed among women in HICs compared with men in both HICs and MICs and women in MICs (Figure 5-5). There were a higher percentage of positive (40%) and negative (16%) relationships, and a lower percentage reporting no association (40%). Among men and women in MICs and men in HICs, there was more consistent evidence of no association across the studies (70-73%).

**Figure 5-5.** Distribution of Associations for Fat-Free Measures by Sex and Income Level



### Figure 5-5. Footnotes

Females in HICs (N=15): 13% negative associations, 40% positive associations, 7% non-linear associations, 40% show no association; Males in HICs (N=10): 0% negative associations, 20% positive associations, 10% non-linear associations, 70% show no association; Females in MICs (N= 11) 9% negative associations, 9% positive associations, 9% non-linear associations, 73% show no association; Males in MICs (N= 10) 0% negative associations, 30% positive associations, 0% non-linear associations, 70% show no association.

**Table 5-7.** Summary of Associations between Socioeconomic Position and Fat-Free Measures in Adults in High-Income Countries and Middle-Income Countries.

SEP Indicator	Direction of Relation Between SEP and Lean Measure												Total	
	Positive			Negative			Non-linear			No Relation				
	N	%	References	N	%	References	N	%	References	N	%	References	N	%
<i>Fat-Free Mass</i>														
Education	4	50%	[284] <sup>M\$</sup> [286] <sup>F\$</sup> [316] <sup>F</sup> [316] <sup>M</sup>	1	13%	[284] <sup>F\$</sup>	0	0%	-	3	38%	[317] <sup>F</sup> [317] <sup>M</sup>	7	16%
Miscellaneous	1	33%	[310] <sup>M\$</sup>	1	33%	[279] <sup>F</sup>	0	0%	-	1	33%	[279] <sup>M</sup>	3	7%
<i>Fat-Free Mass Index</i>														
SEP	0	-	-	0	-	-	0	-	-	1	100%	[310] <sup>M\$</sup>	1	2%
<i>Lean Mass</i>														
Education	2	33%	[320] <sup>F</sup> [283] <sup>F</sup>	0	0%	-	0	0%	-	4	66%	[320] <sup>M</sup> [282] <sup>F</sup> [299] [311]	6	14%
Miscellaneous	1	25%	[260]	0	0%	-	2	50%	[282] <sup>F</sup> [301] <sup>F\$</sup>	1	25%	[288] <sup>F</sup>	4	9%
<i>Lean Mass %</i>														
Fathers Education	1	100%	[260]	0	-	-	0	-	-	0	-	-	1	2%
<i>Lean Mass Index</i>														
Occupational Social Class	0	0%	-	1	50%	[279] <sup>F</sup>	0	0%	-	1	50%	[279] <sup>M</sup>	2	5%
<i>Appendicular Skeletal Muscle</i>														
Education	1	20%	[284] <sup>F\$</sup>	0	0%	-	0	0%	-	4	80%	[284] <sup>M\$</sup> [313] <sup>\$</sup> [298] <sup>M</sup> [298] <sup>F</sup>	5	11%
Income	0	0%	-	0	0%	-	1	33%	[298] <sup>M</sup>	2	66%	[298] <sup>F</sup> [313] <sup>\$</sup>	3	7%
Employment	0	0%	-	0	0%	-	0	0%	-	1	100%	[313] <sup>\$</sup>	1	2%
<i>Appendicular Skeletal Muscle Index</i>														
Education	1	20%	[285] <sup>F</sup>	0	0%	-	0	0%	-	4	80%	[313] <sup>\$</sup> [292] <sup>F</sup> [300] <sup>F</sup> [285] <sup>M</sup>	5	11%
Income	2	66%	[285] <sup>F</sup> [285] <sup>M</sup>	0	0%	-	0	0%	-	1	33%	[313] <sup>\$</sup>	3	7%
Miscellaneous	1	33%	[285] <sup>F</sup>	0	0%	-	0	0%	-	2	66%	[313] <sup>\$</sup> [285] <sup>M</sup>	3	7%
<b>Overall Distribution of Associations – Fat-Free Measures Combined</b>														
<b>Combined SEP</b>	32%			7%			7%			55%			44	100%



<b>HIC Combined SEP</b>	33%	7%	7%	53%	30	68%
<b>MIC Combined SEP</b>	29%	7%	7%	57%	14	32%

**Table 5-7 Footnotes.** *F* Indicates results for females only; *M* indicates results for males only; *\$* indicates study conducted in MIC. Positive associations indicate an increase in fat-free measure with an increase in socioeconomic advantage; negative associations indicate a decrease in fat-free measure with an increase in socioeconomic advantage; non-linear indicate the association between SEP and fat-free measures is either curvilinear or heterogeneous. Miscellaneous SEP measures are where less than two papers reported on the measure. Results for Pirila are reported as LBM but have been included here with FFM due to similarity of measure. Total N represents the total number of reported associations between the given SEP measure and the body composition measure (i.e total number of associations reporting on education and FFM). There may be two associations from one paper per SEP measure, if only sex-stratified data is presented. The N for the direction of relation groups (positive, negative, non-linear, no association), refers to the number of associations reporting each patterning within the given SEP measure and body composition measure combination (i.e number of positive associations reported between education and FFM), and corresponds to the references included. The % for the direction of relation groups indicates the number of associations reporting a particular patterning (i.e positive) as a percentage of the total number of associations for the given SEP measure and body composition measure (i.e education and FFM).

#### *5.3.4.3 Ratio and Distribution Measures*

Thirteen papers reported on the association between SEP and ratio or distribution measures (Table 5-8). Only two were from MICs preventing comparison of differences by income level of the country.

There were 14 associations across ten papers that investigated the association between SEP and a measure of central fat. Four papers used the same all-female sample (SWAN) [291, 295, 305, 308], and one additional paper also tested the association in women only [301]. The majority of associations (nine out of 14, 64%) reported that greater socioeconomic advantage was associated with lower central adiposity. For education, five out of the seven associations found evidence that lower education level was associated with greater central fat, whilst two out of the three papers using income found some evidence of higher central fat among those with lower income.

Two papers used a distribution measure other than central fat. One found women who engaged in more labour-intensive occupations had lower leg FM [301]. The other found no association between education and the distribution of either upper or lower skeletal muscle mass [284].

Six associations across three papers reported on ratios, one conducted in an all-female population. One paper, that tested the association using education, occupational social class and income, found greater socioeconomic advantage was related to a lower android-to-gynoid ratio [285]. Men showed a stronger association for education compared to other SEP measures, and also a stronger association than that observed in women when measured by education, whilst a heterogeneous association was observed for occupational social class [285]. Another paper found those with higher education level were more likely to have a normal compared to high ratio of fat to lean mass, whilst employment was related to the ratio in a non-linear fashion [312]. In the third study no association was found between income and the ratios of central fat types in females [295].

### **5.3.5 Childhood Socioeconomic Position and Adult Body Composition**

There were 7 papers investigating the associations between childhood SEP and adult body composition. It was not possible to assess heterogeneity due to the small number of papers, all of which were conducted in HICs. Results are summarised in Table 5-9.

#### *5.3.5.1 Total Body Fat Measures*

Five papers reported on SEP in childhood and measures of fat in adulthood, testing eight different associations. In four papers, testing seven associations, greater socioeconomic advantage in childhood was associated with lower FMI or FM% in adulthood [285, 303, 304, 322]. One study reported that the association was somewhat explained by own SEP in adulthood [285], with the others not assessing this. The fifth paper found father's education not to be directly related to a standardised FM% score (z-FM %) at age 32 but reported an indirect relation through own education and current physical activity levels [311].

#### *5.3.5.2 Total Body Fat-Free Measures*

Three papers reported on SEP in childhood and fat-free measures in adulthood, testing five associations. Two papers found evidence of positive associations, with greater socioeconomic

advantage in childhood being related to higher LM [311] and higher ASMI [285] in adulthood. One of these presented only sex-stratified analyses and found the association with ASMI to be stronger in males before adjusting for FMI, whilst stronger in females after the adjustment for FMI [285]. The final paper reported no association with LM [322].

#### *5.3.5.3 Ratio and Distribution Measures*

Three associations across three papers investigated central fat. One found a negative association in females only [278], one a curvilinear relation, where those in the medium third of android fat had the most advantaged SEP in childhood [306]. The final paper found no association [311].

Two papers reported on android-to-gynoid ratio, presenting four associations. Both papers found greater socioeconomic advantage in childhood to be associated with lower android-to-gynoid ratio. In one paper this was only the case in females [278], whilst in the other, the association was stronger in females than males [285]. The latter paper found the association remained with adjustment for SEP in adulthood [285].

**Table 5-8.** Results of Associations between Socioeconomic Position and Adult Ratio and Distribution Measures in Adults

Paper	Country	N	Age	Study or Description of the Population	SEP Measure	Body Composition measure	Findings
Kazlauskaitė (2012) [295]	USA	257	52	SWAN (females only)	Income	Log IAT, IAT:SAT Ratio	No association between income and Log IAT or with IAT:SAT ratio in all three models. Model 1 adjusted for risk groups (ethnicity, menopausal status), income, age and percent body fat. Model 2 = Model 1 plus low physical activity (Kaiser Physical Activity Survey score) and smoking status Model 3 = Model 2 plus energy intake ethnicity interaction
Loucks (2015) [306]	USA	394	46-48	New England Family Study	Education	Android FM	Highest percentage of individuals with low education (% with less than high school education) in highest android fat tertile.
Beydoun (2009) [140]	USA	1,227	30-64	HANDLS	SEP	Trunk Fat, Trunk fat as % of total body fat	Association differed by sex and ethnicity. SEP was inversely associated to measures of central adiposity in white American women, and positively associated in African American women. There was no association between SEP and central adiposity in white American or African American men.
Pirila (2012) [311]	Finland	158	32	Sample taken from birth cohort of full-term babies born in Helsinki in 1975.	Education	% Trunk fat	Decrease in % Trunk fat with an increase in education level ( $r=-0.147$ , $p=0.07$ ).
Bhupathiraju (2011) [287]	USA	F: 465 M: 164	54-75	Boston Puerto Rican Osteoporosis Study	Education, Income	Abdominal Fat	Significant difference between fat tertiles for those with the least and most education in women. Lowest percentage with low education (<9th grade) and highest percentage with high education (at least some college) in lowest fat tertile. No significant difference between fat groups for percentage with medium education. Income inversely related to abdominal fat in women, with increase in central adiposity with increase in income ( $p=0.02$ ). No association between education or income with abdominal fat in men.
Dugan (2010) [291]	USA	369	50.7	SWAN (females only)	Education	IAT	Little evidence of an association between education and IAF (Beta: 7.1, SE= 6.5, $p=0.28$ )

Mongraw-Chaffin (2017) [309]	USA	1,910	45-85	MESA	Education, Income	Visceral Fat	Greater percentage of those with most advantaged education or income level in the lowest visceral fat tertile ( $p=0.001$ and $p=0.055$ respectively. Cuzick non-parametric test).
McClure (2011) [308]	USA	301	46-58	SWAN (females only)	Education, Financial Strain	Visceral Fat	No association between education or financial strain and visceral fat.
Lewis (2009) [305]	USA	418	42-61	SWAN (females only)	Education	Visceral Fat	Weak inverse association between years in education and visceral fat ( $B = -1.31$ , $Beta = -0.05$ , $SE = 0.99$ , $p = 0.19$ )
Kulkarni (2010) [301]	India	278	41	Adult women who were not pregnant or lactating, residing in a large urban slum (Addagutta) in Hyderabad.	Occupation Type	Trunk FM, Leg FM	Trunk FM and leg FM were lower among sweepers ( $Beta = -554$ , $p = 0.009$ and $Beta = -587$ , $p = 0.002$ , respectively) and construction workers ( $Beta = -506$ , $p = 0.013$ and $Beta = -548$ , $p = 0.003$ , respectively) compared to home makers. Leg FM was also lower in servant maids ( $Beta = -354$ , $p = 0.036$ ). Trunk FM and leg FM did not significantly differ in beedi makers compared to home makers.
Powell (2016) [312]	Italy	3,441	18-81	Participants selected from ongoing cohort in Milan	Education, Employment	VAT:FFMI, FM:FFM,	Higher percentage with university degree and lower percentage with elementary school in normal compared to high VAT:FFMI and FM:FFM groups (both $p < 0.001$ ). Curvilinear relation for employment, with differences in VAT:FFMI and FM:FFM across employment groups (both $p < 0.001$ ).
Bann (2014) [285]	UK	M: 746 F: 812	60-64	NSHD	Education (age 26), Occupational Class (age 53), Household Income (age 60-64)	Android to Gynoid Ratio	Inverse associations between all SEP measures and android to gynoid ratio in both males and females (SII ranges from 3.26 to 8.42 and p-values range from $< 0.01$ to 0.04), with exception for occupational class in males, where deviation from linearity indicates a heterogeneous association (SII: 3.81; 95% CI: $-0.33$ to 7.96; $p = 0.07$ ). Significant sex interaction for association between education and android to gynoid ratio ( $p = 0.05$ ).
Bai (2016) [284]	China	212	60-99	Men and women recruited through printed advertisement from the health survey	Education	Upper limb skeletal muscle mass; lower limb skeletal muscle mass	Education a non-significant predictor of upper and lower limb skeletal muscle mass.

**Table 5-8 Footnotes.** *F- Female; M – Male; SEP – Socioeconomic Position; SII – Slope of Inequality Index; FM - Fat Mass; FFM - Fat-Free Mass; FFMI – Fat-Free Mass Index; IAT – intra-abdominal adipose tissue; SAT – subcutaneous abdominal adipose tissue; VAT – visceral adipose tissue. SWAN- The Study of Women's Health Across the Nation; HANDLS- Healthy Aging in Neighbourhoods of Diversity across the Life Span; NSHD- National Survey of Health and Development.*

**Table 5-9.** Direction of Association Reported between Socioeconomic Position in Childhood and Body Composition in Adulthood, Presented in Narrative Form

Paper	Country	N	SEP Measure and time of measurement	Body Composition measure and age	Findings
Agha (2013) [278]	USA	F: 228 M: 172	Prenatal SEI	Android FM, Android to Gynoid Ratio, Trunk to Limb FM Ratio Age: 48	Inverse associations between prenatal SEI and android FM, android to gynoid ratio and trunk to limb FM ratio in females with adjustment for age, race and maternal variables. No association in males.
Bann (2014) [285]	UK	M: 746 F: 812	Occupational Social Class (age 4) Mothers and Fathers Education (age 6)	FMI, ASMI, Android to Gynoid Ratio Age: 60-64	All three childhood SEP measures were significantly ( $p < 0.05$ ) related to FMI and Android to Gynoid Ratio in men and women, and significantly associated with appendicular lean mass index in men before adjustment for fat mass, but not in women. Following adjustment for fat mass, the association with appendicular lean mass index was significant in women but not men. Own education and adult SEP may explain part of the association between paternal education age 6 and appendicular lean mass index in women, and FMI in men.
Lahmann (2000a) [303]	Sweden	5145	Parental Occupation (recalled)	FM% Age: 45-73	Only tested in women - Inverse association with parental occupational class and FM%.
Lewin (2014) [304]	France	4,079	Parental Education (recalled)	FMI and FM% Age: 30-79	Significant slightly inverse association between parental education and FM% in males. Association between parental education and FM% not reported for females as insignificant. Significant associations in both males and females between parental education and FMI, with lowest FMI in highest parental education group. Stronger effect in women.
Loucks (2016) [306]	USA	394	Childhood SEI (age 7)	Android Fat Age: 47	Curvilinear association between childhood SEI and android fat – those in middle tertile of android fat tended to be from the most advantaged position.
Pirila (2012) [311]	Finland	158	Fathers Education (recalled)	FM% z score, LM, % Trunk Fat Age: 32	No direct association between father's education and FM% z-score. Indirect pathways through own education and physical activity levels. Similar for % Trunk Fat. Evidence of direct association between father's education and LM.
Ylihärsilä (2007) [322]	Finland	M: 928 F: 1,075	Childhood Social Class (derived from multiple points in childhood, highest record taken )	FM%, LM Age: 61.5	Lower FM% was associated with higher social class (Males $p < 0.001$ , Females $p = 0.031$ ) No results reported for social class and lean mass – assumed to be insignificant.

**Table 5-9 Footnotes.** Results are presented in narrative form due to the small number of papers looking at socioeconomic position (SEP) in childhood and body composition in adulthood, and because of heterogeneity in SEP measures and body composition outcomes, preventing direct comparisons between studies. M – Male; F- Female; SEP - Socioeconomic Position; FM - Fat Mass; FMI - Fat Mass Index; ASMI - Appendicular Skeletal Muscle Index; LM - Lean Mass.

## 5.4 Discussion

### 5.4.1 Summary

This chapter aimed to test three hypotheses through a systematic review of the literature exploring associations between SEP and body composition. A summary of the findings in relation to the hypotheses are given below.

***Hypothesis 1:*** *There are social inequalities in fat mass (FM) and fat-free mass (FFM), with less advantaged SEP being related to higher FM and lower FFM in high-income countries, and associations in the opposite direction in middle- and low-income countries.*

In line with hypothesis 1, this systematic review finds evidence of socioeconomic inequalities in body composition in both childhood and adulthood. However, the direction and strength of these inequalities varies by economic development of the country of study, life stage, measure of body composition, measure of SEP and sex.

Among children there was clear evidence of negative associations for all measures of fat. Negative associations were more frequently observed for fat in samples from HICs compared with MICs, with positive associations only observed in studies from MICs. The review generally finds evidence of associations between advantaged SEP in both childhood and adulthood and lower levels of total body FM in adulthood in HICs, irrespective of which SEP or fat measure is used. In MICs, the majority of studies showed positive or no association for fat.

Somewhat consistent with hypothesis 1, greater socioeconomic advantage was associated with greater FFM in approximately half of the associations studied in children, but such associations were less common with outcome measures indexed to body size in both HICs and MICs. For fat-free measures in adulthood, findings were more mixed, with the majority of papers reporting no association or greater socioeconomic advantage being associated with greater FFM. There is some evidence of positive associations between childhood SEP and adult fat-free measures in HICs, although the small number of studies means caution is required in interpretation of these findings. Contrary to hypothesis 1, there was little evidence of differences in associations for fat-free measures by level of economic development, especially in adults.

No studies reported associations for childhood SEP and adult body composition in MICs, and no studies in children or adults conducted in LICs were identified. There was a lack of research using area-level measures of SEP, parental income and using more detailed measures of body composition, such as ratio and distribution measures.

***Hypothesis 2:*** *Social inequalities in FM and FFM vary between generations, with greater evidence of inequalities in FM and FFM among those born more recently.*

In line with hypothesis 2, studies in children reported negative associations for FM in HICs and positive associations for FFM in HICs and MICs more frequently than studies conducted in adults. For FM in adults, there was slightly less evidence of positive associations in MICs compared to children,



and greater evidence of non-linear associations in both HICs and MICs, and no association in MICs. Too few studies reported birth year to investigate secular differences in the inequalities.

**Hypothesis 3:** *Less advantaged SEP is related to higher FM and lower FFM more frequently in females compared to males in HICs.*

Consistent with hypothesis 3, negative associations for FM were found more frequently in girls compared with boys in HICs, whilst in MICs girls more often showed positive associations. In adults, associations between advantaged SEP and lower levels of total body FM were more frequently observed among females than males. For fat-free measures, there was little evidence of sex differences in children or for adults in MICs, whilst for adults in HICs females were more likely to report positive associations compared to males.

#### 5.4.2 Comparison to Previous Literature

The findings for FM in children and adults are broadly consistent with those from reviews using BMI as the measure of adiposity. Shrewsbury and Wardle (2008) [323] and Barriuso et al. (2015) [324], found associations between greater socioeconomic disadvantage and higher levels of adiposity among children and adolescents from HICs, identifying almost no associations in the opposite direction. Sobal and Stunkard (1989) [111] and Dinsa et al. (2012) [113] observed consistent evidence of disadvantaged SEP being related to lower levels of obesity among children in MICs. However, where Sobal and Stunkard (1989) [111] found the association between SEP and obesity, measured largely by BMI, to be inconsistent among children in HICs, the results of the current review in body composition found more consistent evidence that greater socioeconomic disadvantage was associated with higher FM.

The findings for adult SEP and fat measures are also consistent with previous systematic reviews, showing predominantly negative associations for anthropometric measures of adiposity such as BMI and WC in HICs, especially among women [111, 112]. Also similar to reviews based on BMI and other anthropometric measures [111-113], this review finds positive associations to only be observed in MICs. The results also show SEP to be negatively related to measures of central fat, whilst evidence for other measures of fat distribution is too sparse to draw conclusions [112].

The findings in relation to childhood SEP and adult body fat measures in this review are similar to those for BMI that find mostly negative associations [116, 117]. However, the small number of studies in this review mean that conclusions regarding differences between males and females were not possible. No studies reported on the association in MICs, and it was therefore not possible to compare findings between MICs and HICs.

The literature review in Chapter 2 identified no previous systematic reviews that had considered social inequalities in measures of FFM. One review did find more consistent evidence than this review that advantaged SEP in childhood is related to better physical capability in adulthood [325], with such measures of physical capability (i.e. grip strength) being correlated with muscle mass and strength [326, 327].

Previous research using anthropometric measures of obesity have reported that, among women and children in HICs, studies using education or parental education report negative associations most frequently [112, 323, 324] consistent with the current findings. However, this was largely explained by differences in measures adopted between HICs and MICs. In both adults and children, this review found less consistent associations for income, whilst McLaren (2007) found greater evidence of positive associations for income in MICS only [112]. However, fewer studies in this review adopted income as a measure of SEP compared to other SEP measures. Area-level measures of SEP have previously been shown to be particularly strong predictors of obesity in systematic reviews [112]. However, very few studies used area-level SEP in relation to body composition, especially in childhood.

### 5.4.3 Sources of Heterogeneity

This section discusses each of the sources of heterogeneity investigated in this review, considers them in the context of previous research and explores possible explanations for the patterns observed between SEP and body composition.

#### 5.4.3.1 High-Income and Middle-Income Countries

Differences in findings between HICs and MICs, particularly for FM, may be, at least in part, explained by the nutrition transition. Consumption of energy-dense food that are high in fats and sugars is related to higher adiposity, whilst protein and micronutrients are required for muscle tissue development as well as height [328-332]. As countries develop, food becomes more abundant and accessible and, in particular, more frequently characterised by high energy-dense and calorific foods [333]. In MICs, those with greater socioeconomic advantage have greater food security and access to the high energy-dense foods, and more calorific diets [334]. In HICs, high energy-dense and calorific foods tend to be cheaper and consumed more frequently among individuals of disadvantaged SEP [335, 336]. The existence of predominantly positive associations between SEP and fat measures in adults and children, and also positive associations in fat-free measures in children and to a lesser extent adults in MICs, may reflect that those in disadvantaged circumstances are more likely to be food insecure and lack essential macronutrients and micronutrients [337]. Therefore, those in disadvantaged circumstances are likely be shorter and have lower levels of both FM and FFM [338].

Physical activity is an important determinant of muscle development and maintenance, as well as being important for maintenance of healthy adiposity levels. Levels of physical activity between MICs and HICs may be affected by different timings in the onset of the obesogenic environment and nutrition transition [333], which is in part characterised by a shift from more labour intensive lifestyles to more sedentary lifestyles [339]. Those in disadvantaged socioeconomic circumstances are more likely to work in manual jobs, and therefore have higher occupational physical activity. However, those in positions of socioeconomic advantage in HICs tend to participate in more leisure time physical activity compared to those in disadvantaged positions [340, 341]. In particular, there is evidence of greater vigorous activity from leisure time activities among those in more advantaged positions in HICs [341]. Research conducted in the UK Biobank study demonstrated that physical activity conducted through manual occupations does not contribute to reduced all-cause mortality risk,

indicating leisure activity outside of work is more important to overall health [342]. Among children and adolescents, those in more advantaged socioeconomic circumstances in HICs are more likely to engage in physical activity [343, 344]. This, combined with differences in nutrition, may explain the existence of negative associations for fat measures and positive associations for lean measures in HICs in both children and adults, where those with low fat are also leaner.

The nutrition transition can also help explain differences in results between children and adults. As most studies in this review were conducted in children born post 1984, that means in HICs they were all born into an obesogenic environment, the onset of which is generally agreed to be in the 1980s [150, 151]. As outlined in Chapter 2, disadvantaged SEP, after the onset of the obesogenic environment, has been associated with increased proximity to fast-food outlets [152, 154], larger advertising of fast-food [156], and worse access to sports facilities [159] and green space [157] in HICs. Children are particularly influenced by advertising [345] and the food environment [346], and are less likely than adults to have a beneficial relationship between the built environment and levels of physical activity [347].

In contrast to HICs and similar to adults in MICs, disadvantaged SEP was associated with lower levels of body fat for children in MICs. Children of advantaged SEP in these countries have greater exposure to a western lifestyle compared to those of disadvantaged SEP, and in particular greater access to more expensive and energy-dense foods [113]. Transnational food companies that have expanded to MICs often target children with their adverts, therefore making children particularly vulnerable to their efforts to increase purchase and consumption in MICs [348]. In addition, differences in physical activity may play a role. A study from India found higher rates of obesity in private schools compared with government schools [349], explained in part by a greater reliance on cars or buses to get to private schools, whilst children who attended government schools were more likely to walk or cycle [348]. There may also be cultural differences in perception of obesity between HICs and MICs [350], with overweight children in MICs being considered healthier by parents [349] and poorer understanding of the health consequences of obesity among mothers in MICs [351].

#### *5.4.3.2 Body Composition Measures*

The majority of papers used a raw measure of FM or FFM, rather than indexed measures that aim to, at least partially, remove the correlation with height. The review found stronger evidence of negative association for fat when using the raw measure in adults and to a lesser extent in children, rather than indexed measures. This indicated less evidence of inequalities in fat when appropriately accounting for height. Similarly, among children there were fewer observed positive associations for FFM when an indexed measure was used, although this was not the case for adults. Associations of greater disadvantage and lower FFM may therefore be explained, at least partially by height. There is evidence that disadvantaged SEP is associated with shorter height across childhood and adulthood in most populations [352], although in HICs there is evidence this inequality has narrowed [28]. Studies which appropriately adjust FFM measures for height are required to assess this.

There was variation in associations according to what composition of FFM was captured. Studies that used measures which included bone mass showed a higher proportion of positive associations in both children and adults compared to those which did not, indicating that bone may be contributing to observed inequalities in FFM. Additionally, there were inconsistencies in the adjustment of LM for FM. Previous research has shown adaptive increases in LM occur with increases in FM, highlighting a need to consider adjustment for FM [85, 353, 354]. In this review, adjusting FFM measures for FM or body size resulted in the direction of association reversing, becoming positive [285] or existing positive associations increasing in strength [316].

#### *5.4.3.3 Measure of Socioeconomic Position*

Differences observed in associations according to SEP measures in both adults and children could largely be explained by differences between HICs and MICs. Both occupational social class and area-level measures of SEP were predominantly used in HICs and also reported the highest proportion of negative relations with total FM. Composite measures of SEP were more frequently used in MICs and reported a higher proportion of positive associations with total FM. Income was the only measure used in HICs that showed a low number of negative associations in both adults and children, and a high number of non-linear associations in adults and high number of no associations in children for FM measures.

Income is considered a direct measure of material resources, and is most prone to short-term change [110], which may explain the greater observed heterogeneity. Education is a more stable measure of SEP that captures early life conditions whilst also a determinant of later life SEP, and reflects knowledge assets and health literacy as well as health behaviours [110]. For children, higher parental education and thus greater health literacy in parents may translate into differences in up-bringing in early life, including activity related care [355], and feeding practices that may influence appetite [355, 356]. Similarly, occupational social class is a good overall measure of SEP as it captures aspects of an individual's education, income, social standing in addition to their occupation [110]. For children, parental occupational social class may capture important elements of the home environment, such as parental characteristics, social norms, resource access and household income, that are important determinants of obesity in childhood [357]. It is possible that area-level SEP may be related to body composition in both adults and children in HICs due to the close link with obesogenic elements in the environment [161, 162], but also because of social and cultural elements of the environment that influence social norms and behaviours [148].

#### *5.4.3.4 Sex*

The observation that women and girls in HICs were most likely to experience inequalities in fat measures, and women in HICs for fat-free measures, compared to other groups may be explained by increases in gross national product shifting the burden of obesity to those of a less advantaged position within society, affecting women first [114]. As highlighted in Chapter 2, this may be a result of the greater pressures of weight related ideals faced by women, which are easier to maintain in a position of advantage [115]. The fact that inequalities are observed among children for fat measures

where previously there had been less evidence of them, could indicate that gendered and weight-related pressures are impacting girls at a younger age than previously.

#### *5.4.3.5 Differences between Children and Adults*

There were a greater percentage of negative associations between SEP and fat measures among children compared to adults in HICs. This difference may indicate life course differences in the association between SEP and adiposity, or secular changes in inequalities given that the studies conducted in children typically include individuals born more recently than those conducted in adults. A comparison of the British Birth Cohorts demonstrated increasing inequalities in BMI with age within the cohorts, and inequalities in childhood and adolescence were only observed in the most recently born cohorts [28]. Research using the Fels Longitudinal Study demonstrated a secular increase in FM% in children and adolescents from 1960 to 1999 [119]. The results for directly measured adiposity in children compared to the results in adults broadly match the trends seen in studies demonstrating secular increases in the inequalities in BMI [28, 29, 117]. Follow-up of childhood cohorts into adulthood will be needed to distinguish a secular trend from an age effect.

In adults, there was predominantly no association between SEP and fat-free measures, although with slight evidence of positive associations among women in HICs. In contrast, among children there is greater evidence for inequalities in fat-free measures, especially in HICs, and with few differences in associations between boys and girls. Greater inequalities in FFM in childhood compared to adulthood may reflect a secular decline in levels of FFM, which are likely to be accompanied by growing inequalities, in the opposite direction to inequalities seen for adiposity. The Fels Longitudinal Study has shown mean FFMI to be lower in boys born in the 1990s compared with boys of the same age born decades before [119]. Serial data more recently has shown a secular decline in muscle strength, measured by handgrip, sit-ups, bent-arm hang and standing broad-jump tests, among children in the UK [121]. It is likely that secular changes in body composition would coincide with secular change in the inequalities, as has been observed with BMI [28]. Peak muscle function is determined across childhood and early adulthood and then maintained through midlife [358], and early development has been shown to be an important determinant of LM in later adult life [359]. It is therefore probable that inequalities in FFM observed in more recent generations in children are likely to persist into adulthood and old age.

Compared to studies conducted solely in childhood or adulthood, fewer studies looked at the relation between childhood SEP and adult body composition, and all of them were conducted in HICs. Results were in general similar to those in adults for FM and the small number of studies looking at FFM showed predominantly positive associations, indicating a possible life course effect of SEP on body composition. Few of these studies tested if associations with childhood SEP were independent of adult SEP, which has implications for the lasting role of early development in nutrition and physical activity patterns [360, 361]. Consideration of both child and adult SEP would allow investigation of latency, trajectory or accumulation effects of SEP across the life course and help to identify the best time to intervene to prevent inequalities in body composition.

#### 5.4.4 Research Implications

Few studies investigated the association between childhood SEP and adulthood body composition, and there were no such studies in MICs. Childhood SEP has previously been shown to be a predictor of BMI in adulthood, and further research is needed to understand how disadvantage may accumulate over the life course and influence body composition in adulthood. No data was available from LICs across the whole review, an area that warrants further research.

Only a small number of studies provided birth years of participants, preventing investigation of secular differences in inequalities in body composition in the context of persisting inequalities in BMI. The majority of studies in adults were conducted in those aged 40 and above, with fewer studies looking at young to mid-adult life, preventing full assessment of difference in body composition across the adult life course.

#### 5.4.5 Policy Implications

The results of this review indicate that the established evidence on inequalities in BMI likely capture inequalities in FM in adults. In monitoring inequalities, BMI may accurately capture FM at a population level in childhood and adulthood. However, the review suggests contrasting findings on inequalities in FFM in HICs in children compared with BMI. This may, assuming such associations are not fully explained by inequalities in height, mean that BMI underestimates the inequalities in the health risks related to body composition among children. As this review has shown greater evidence of inequalities in body composition in children compared with adults, tracking of body composition through the life course in more recent generations could have important implications for inequalities in physical capability in later life. Follow up of these childhood cohorts is needed to confirm whether these are secular rather than age-related changes in inequalities, or the existence of both simultaneously and how this might influence social inequalities.

The differences in associations between SEP and FM between HICs and MICs indicate emerging, and in some cases, reversing inequalities in body composition as countries go through the nutrition transition and with the onset of the obesogenic environment. These findings suggest that action is required in MICs to mitigate the negative effects of this transition. Mitigating action is likely needed in LICs also, which are expected to be further behind in the nutrition transition than MICs, and so information in LICs is needed. In particular, efforts should continue to focus on reducing the abundance of cheap energy-dense food in poorer communities and ensure access to healthy and nutritious food across SEP groups, as a way to combat inequalities in FM. Additionally, there should be a reduction of targeted advertising of fast-food to children. Attention should also be paid to promotion of physical activity and access to sport facilities should be prioritised. Especially in poorer communities to ensure healthy levels of FFM across all SEP groups, and into older age where muscle mass may be more important for metabolic and functional outcomes.

#### 5.4.6 Strengths and Limitations

This review was registered with PROSPERO and was carried out according to the published protocol [221]. The review has a generous inclusion criterion, capturing a broad range of evidence, thereby

reducing selection bias. Bias was also reduced by having two independent reviewers conducting each stage of the review, including selection of studies into the review and extraction of data, as well as completion of a quality assessment which was used to inform of the variability in study quality.

Three large biomedical databases were selected to conduct the literature search, with Medline being selected as the primary database instead of alternative large databases such as PubMed. A PubMed search that is limited to MeSH terms will be similar in scope to an Ovid MEDLINE search. Given both MeSH and free text search terms were included in the Medline search, the results would be comparable. Together with the inclusion of the additional databases Embase + Embase Classic, which has a larger coverage than either Medline or PubMed and additional searching of reference lists, the additional texts covered in PubMed would have been unlikely to capture papers suitable for the review. For example, older texts (pre 1879 – earliest entry of Medline) were unlikely to have direct measures of body composition.

The generous inclusion criteria resulted in considerable heterogeneity in samples, study design and measures used. This variation, together with heterogeneity in the analytical approaches and reporting of results, prevented a meta-analysis from being conducted. Additionally, the associations, not papers, was used as the unit of analysis since most papers reported more than one association, meaning that in some cases a single paper may have contributed more weight to the overall summary of findings. The same data sets were also used by multiple authors in multiple papers.

In reporting the results, an effort was made to balance granularity in the presentation of results against the overall key messages from the studies. To do this, results were first summarised as a whole, and then sources of heterogeneity that were pre-specified in the protocol were investigated, including the use of broad summaries for SEP and body composition to clarify the key patterns. The large heterogeneity between individual studies meant it was difficult to present results for every specific SEP and body composition measure, while exploring other sources of heterogeneity such as sex and level of country development.

As it was not possible to conduct a meta-analysis, it was not possible to assess publication bias. It is, however, possible that publication bias exists. Papers based on small sample sizes showing positive results are more likely to be published than those showing null findings. However, studies that tested the association of interest as part of wider set of analyses were included in addition to those with a specific hypothesis on SEP differences. This may have reduced the impact of publication bias. Papers were, therefore, included that did not report estimates where associations were found to be non-significant in preliminary analysis.

This review prioritised assigning patterns to associations using the effect estimates and confidence intervals to overcome problems related to a reliance on p-values [362, 363]. Effect estimates and 95% confidence intervals convey more about the direction and strength of effect, and the accuracy of these estimates [363, 364]. However, many of the papers included reported p-values alongside only descriptive data or description of the association in the text. As p-values are influenced by the sample size of the study, lack of associations observed in such studies is likely due to a lack of statistical

power. The studies included in this review ranged in size, with sample sizes as low as 74 participants. Studies in children were generally smaller than adults, with a median sample size between 485 and 502 compared to 629 and 637. Additionally, because of the heterogeneity in SEP measures, outcomes, statistical approaches and the reporting of results it was not possible to make comparisons of effect size across papers, even among those studies that did use appropriate statistical methods. There may also have been overadjustment as studies have adjusted for factors which may actually be mediators rather than confounders, such as diet and physical activity.

There is inconsistency in the literature relating to the terminology used to describe FFM [68], with a small number of papers using incorrect terminology based on the description of their body composition measure. Despite best efforts to ensure comparability by applying standard definitions of fat-free measures across the review (outlined in Table 1-1, pg. 26), some papers did not provide enough clarity on the measures used to do this confidently i.e. such as clearly stating if bone was included or excluded in estimates of FFM. This highlights the need for consistent definitions to be applied and used across the body composition literature, and for authors to provide clarity on the measures used. Specifically, ensuring use of the correct term if bone is included (FFM or LBM) or excluded (LM) and using appropriate and consistent terminology throughout when measures have been indexed or converted to percentages. It was not possible to assess the differences in association by birth year due to lack of information. Nor was it possible to assess differences between childhood SEP and adulthood body composition by age, sex and income level of country due to the small number of papers reporting such associations.

The protocol was amended slightly, due to the need to analyse heterogeneity by country income level, as more papers were identified from MICs than expected and it was clear that this was an unignorable source of heterogeneity. There were no papers included from LICs, limiting the ability to explore SEP and body composition associations in countries at an earlier stage of the nutrition transition, which would have been valuable for understanding the changing relationship of SEP and obesity with economic development.



# Chapter 6 Area- and Family-Level Socioeconomic Position and Body Composition Trajectories in the UK Millennium Cohort Study

## 6.1 Introduction

The second objective of this thesis is to understand associations between SEP in early life and trajectories in body composition across childhood and adolescence in the UK. The systematic review provided the context for inequalities in adolescents in HICs and found evidence that disadvantaged SEP was associated with greater FM and somewhat lower FFM among children in HICs, but few studies indexed measures to height. This thesis prioritised looking at associations among adolescents in HICs as opposed to MICs or LICs, so as to provide comparison with work done previously in the British Birth Cohorts looking at BMI. There is a need to understand how and when inequalities in body composition emerge in a contemporary UK population, and how these inequalities change with age. This is particularly important for inequalities in FFM, as levels of FFM in older age are dependent on peak FFM and age-related decline, with peak FFM being reached in earlier adulthood, unlike FM which increases into older age [97, 365]. Therefore, the early peak means that inequalities evident in childhood may persist into adulthood where they might be more difficult to address in older age.

Few studies in the systematic review used area-level measures of SEP. It is important to distinguish the influence of area- and individual-level measures of SEP, to understand the role of neighbourhood environments on adiposity beyond individual circumstances. This is to ensure policies are effective in reducing obesity, since social inequalities in adiposity emerged with the onset of a more obesogenic environment [29].

Women typically have lower FFM and higher FM than men [365]. Sex differences in inequalities in BMI have been well described in children [112, 118], but whether the same sex differences in inequalities of body composition in children are observed, especially in relation to FFM, remains unclear. Understanding whether there are sex differences, is important to fully understand the later life public health implications of inequalities in body composition.

This chapter explores life course inequalities in trajectories of body composition, by investigating the associations between social circumstances in early life and body composition across childhood and adolescence. By using SEP in infancy, a life course approach is adopted that aims to better understand the consequences of early life social circumstances on life course body composition

trajectories, and thus highlights potential times for early intervention. Work relating to this chapter has previously been published (Bridger Staatz et al. (2021) Lancet Public Health. 6: e598–607. Appendix A6.1). To address the gaps highlighted, three hypotheses are tested in this chapter using data from the MCS:

**Hypothesis 4:** *Disadvantaged SEP is related to higher FM, lower FFM and a higher FM:FFM ratio among children and adolescents in the UK at age 7, and these inequalities widen from age 7 through adolescence to age 17.*

**Hypothesis 5:** *Inequalities in FM, FFM and FM:FFM are greater in females compared to males.*

**Hypothesis 6:** *Area-level inequalities in body composition trajectories are observed, and are not explained by inequalities in family-level SEP.*

## 6.2 Methods

### 6.2.1 Multilevel Modelling for Growth Curves

Multilevel modelling is a statistical technique that is used where data is organised at different levels or is nested. A common example used to demonstrate this is students (level 1) nested into classrooms (level 2), and classrooms nested into schools (level 3), where there is a lack of independence of observations between individuals in the same class, or classes within the same school, as the observations come from the same unit. Multilevel modelling can also be used for repeated measures (level 1) in a single individual (level2). For example, repeated measures of test scores from a single student, which will not be independent, as a student who does well in one test will likely also do well in others. Multilevel modelling therefore provides the advantage of separating estimation of differences between individuals from within individual changes [366]. Standard regression methods do not take the non-independence of observations into account, and therefore may result in biased estimates as the assumption of independent observations is violated. Multi-level models therefore account for the correlations between observations, or between repeated measures in the same individual, and contain both a fixed and random part in the model [367].

The basic variance component multilevel model is expressed in the equation:

$$y_{ij} = \beta_0 + u_j + e_{ij}$$

where  $y_{ij}$  is the outcome measurement for occasion  $j$  in person  $i$ . The fixed part of the equation is represented by the intercept  $\beta_0$  which is the overall mean of  $y$  [368]. The random part of the model has two terms, the first is at the second level (i.e between person) and is a person-level residual that is time constant ( $u_j$ ). The second term is at the first level of the model (i.e within person) and is a person and time specific residual ( $e_{ij}$ ). The time-specific residuals are assumed to be normally distributed with a mean of zero [368].

Multilevel models can be expanded to model growth curves that fit different intercepts and/or slopes for each individual, which allows for variation between and within individuals. This allows estimation of individual trajectories [368].

Multilevel growth curve models specify linear change over time, which is modelled by including time as an independent variable in the fixed part of the model [368]. Multilevel growth curve models can also be used to explore non-linear change in  $y$ . However, at a minimum, four repeated measures are needed for a quadratic curve, but more repeated measures are recommended [369]. The minimum number of repeated measures also increases for other non-linear models, such as cubic and quartic models [369].

Time can be specified in a number of different ways. For example, time can be modelled as the time between waves of assessment, chronological age, the length of time from entry into the study, or the time before and after an event. Most commonly time is taken as the wave of assessment or as age, centred to zero so that the intercept is interpreted as the mean value of  $y$  at the first observation or a particular age [368].

The basic model for growth curves is expressed in the equation:

$$y_{ij} = \beta_{li} + \beta_{si} \text{time}_{ij} + \varepsilon_{ij}$$

where  $y_{ij}$  is the outcome measurement for occasion  $j$  in person  $i$  and  $\beta_{li}$  is the mean intercept plus the random effect, and  $\beta_{si}$  is the mean slope plus random effect.  $\beta_{li}$  and  $\beta_{si}$  are expressed below where the random effect for the intercept is  $u_{li}$  and for the slope is denoted by  $u_{si}$ .

*Intercept Equation:*

$$\beta_{li} = \beta_{l0} + u_{li}$$

*Slope Equation:*

$$\beta_{si} = \beta_{s0} + u_{si}$$

In addition to modelling trajectories of change over time, growth curve models can be used to investigate factors that influence differences in the trajectories. That is, both differences in the initial levels of  $y$  and differences in the rate of change of  $y$  [366]. These factors can either be time-varying, which change with time, or time-invariant, which do not change.

Compared with other methods for analysing longitudinal data, growth curves hold a number of advantages, such as being able to handle non-evenly spaced repeated measures and the ability to include partially missing data [366]. For growth curves to be estimated, adequate sample size is required ( $n > 100$ ) and there typically needs to be three or more repeated measures [366].

### 6.2.2 Analytic Approach

All analyses were conducted using STATA 15.1 [370]. Mean FMI, FFMI and FM:FFM Ratio at ages 7, 11, 14, and 17 were compared between boys and girls, and by ethnicity, income and IMD. The

adjusted Wald test was used to compare equality of means across groups. Differences in the IMD and income by other sociodemographic variables (sex, ethnicity, NVQ and NSSEC) were tested using Pearson's Chi-squared test.

For each of FMI, FFMI, and FM:FFM ratio, multilevel growth curve models, with random intercepts and slopes, were used to estimate change in body composition measure from age 7 to age 17 years. Change with age was modelled as linear, as there were too few data points to model non-linear growth curves. Age was centred at 7 so that the intercept is interpreted as the mean value of the body composition measure at age 7. For each model, observations were nested within the MCS participant identification variable.

A number of models were fitted, all of which included stratification characteristics (advantaged, disadvantaged or ethnic minority stratum within country) as a covariate, as recommended by the Centre of Longitudinal Studies [204]. Firstly, the IMD and income recorded at 9 months (or 3 years for late joiners) were added, separately, as time invariant predictors and an age by SEP indicator interaction was tested to assess whether social inequalities changed with age (Model 1). Model 2 added sex to model 1, and Model 3 additionally included a sex by age interaction to test how changes in the outcome varied by sex, and a sex by SEP interaction to test whether inequalities differed according to sex. Ethnicity was added in another model (model 4). An additional model was fitted where IMD was the exposure, which included family income, highest parental occupation and education level recorded when study members were 9 months (model 5) to test the independence of IMD from family measures.

Two additional models were fitted where FFMI was the outcome of interest. FMI (time varying) was added to model 4 (model 6) because adaptive changes in lean tissue are seen with increases in fat, so it is possible that fat drives observations for FFM and therefore mediates associations. A final model (model 7) made adjustment for FMI and family income, highest parental occupation and education level, where IMD was the exposure variable and FFMI was the outcome variable of interest.

Models 1 and 4 to 7 were re-run separately in girls and boys.

### 6.2.3 Multiple Imputation

Multiple imputation is a methods proposed by Rubin that addresses missing data by imputing a number of plausible values for those missing [217]. The plausible missing values are predicted by observed values in the dataset and created over a number of different imputed datasets ( $m$  datasets). Across the multiple datasets created the values for the observed values stay the same, whilst those for the missing values are imputed. It is common to include auxiliary variables into the imputation models. Auxiliary variables are not included in the analytical model but help improve predictions of the plausible values in the imputation model as they are predictors of values being imputed [371].

Once multiple data sets have been created with each missing value assigned a plausible value, the analytic method can be applied across each of the datasets. The estimates will differ across the datasets, as the plausible values in each dataset differ. The degree to which they differ is the level of

uncertainty about the imputed value. The final step in the imputation is to combine the estimates from the  $m$  datasets into a single estimate, by recalculating the model parameters using Rubin's rule [372], which takes into account the uncertainty of the missing values [373].

#### *6.2.3.1 Analytic Approach*

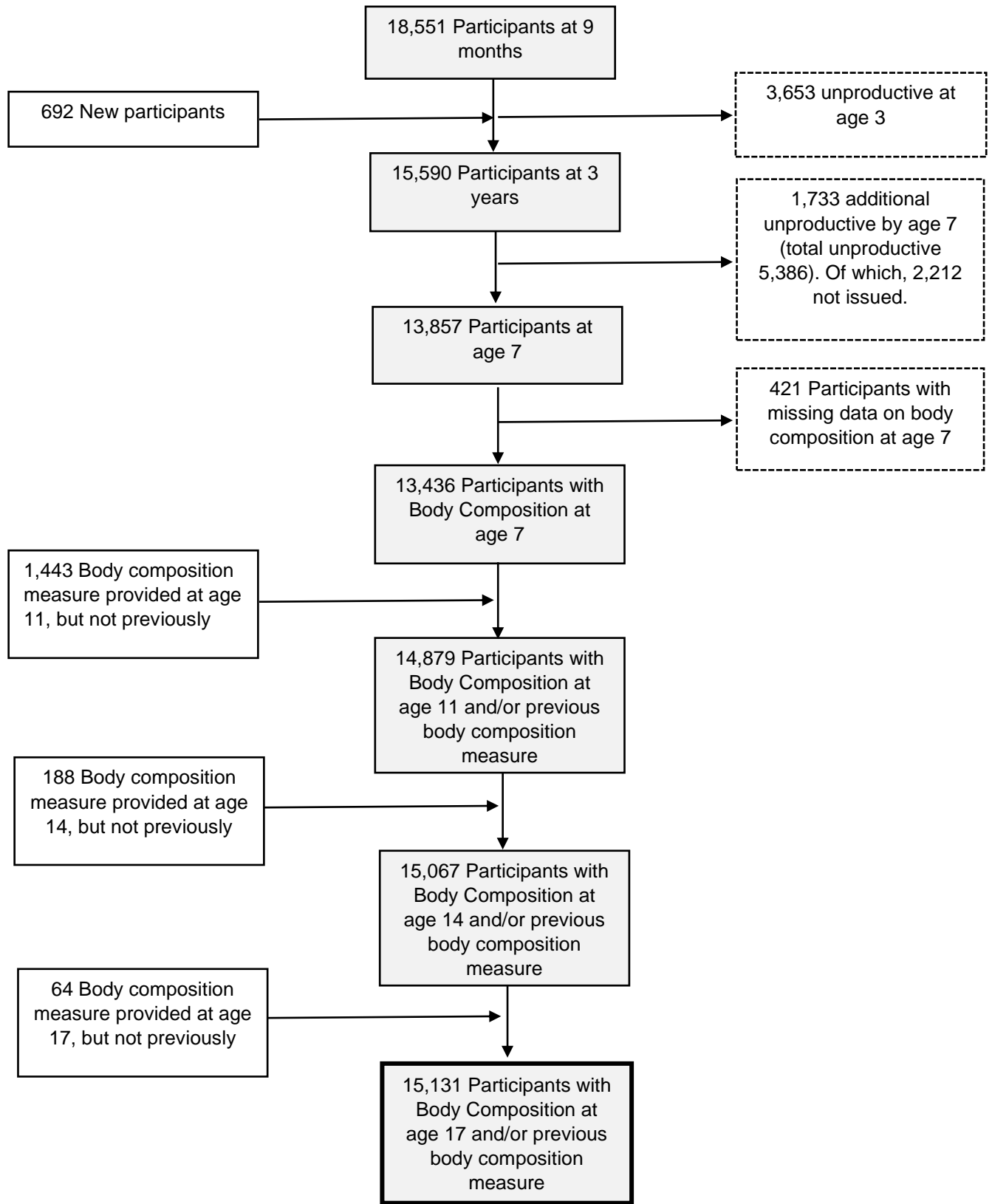
A total of 30 imputed data sets were obtained. Variables for the mothers and fathers NVQ and NS-SEC were both added to the imputation model separately, and a passive variable was created following imputation, combining mothers and fathers' values to take the highest of the household for the NVQ and NS-SEC respectively.

Auxiliary variables were added to the imputation model to improve predictions. These auxiliary variables were: housing tenure, partners BMI, mothers BMI, birthweight, combined labour status, ever breast fed, longstanding illness, self-rated financial difficulty, and main respondent's experience of depression, smoking status and alcohol consumption. Further description of auxiliary variables is provided in the Appendix (A6.2). All auxiliary variables were predictors of one or more variables being imputed. Stratification characteristics and sampling weights were included as a covariates in imputation models to improve predictions, as recommended by the Centre for Longitudinal Studies [190]. Once stratification characteristics have been accounted for in the imputation model, there is no need to use sample weights in the statistical analysis as study design is already accounted for [374]. Stratification characteristics were still included in regression analysis as recommended [188].

#### **6.2.4 Analytic Sample**

As imputation was used to maintain sample size in the predictor variables and covariates, the analytic sample was determined by those with body composition data available. Participants remained in the sample if they had complete data on body composition at one or more time point. The analytic sample therefore contained 15,131 individuals as outlined in Figure 6-1, with a total of 46,157 observations (13,436 participants at age 7, 12,723 at 11, 10,829 at 14 and 9,169 at 17). A total of 7,245 participants had observations for body composition at all four time points, 3,423 at three time points, 2,445 at two time points and 2,018 at only one time point.

**Figure 6-1.** Analytic Sample for Growth Curve Models



## 6.3 Results

### 6.3.1 Descriptive Results

For all measures of body composition and at all ages, mean values of body composition differed between boys and girls. Typically, across all ages girls had higher measures of fat and higher FM:FFM ratio, whilst boys had higher measures of FFM, with the exception of FFMI measured using the Benn Index at ages 14 and 17 (Appendix A6.3). Weight and height were typically higher in boys, whilst BMI was typically greater in girls (Appendix A6.4).

Differences in body composition measures and anthropometric measures (FMI, FFMI, FM:FFM ratio and BMI) by key sociodemographic characteristics were explored. For FMI (Table 6-1) there were differences in mean FMI across ethnic groups, with those of Black and Black British ethnicity followed by Pakistani and Bangladeshi ethnicity typically having the highest FMI across sweeps. Those of white ethnicity had the lowest FMI at all ages, although at age 17 mean FMI was the same for those of White and Indian ethnicity. For income and the IMD, those in the lowest income fifth and those in the 20% most deprived areas had the highest FMI. This was lower across groups, so that with each improvement in deprivation or increase in income bracket, FMI was lower.

Table 6-2 shows the mean FFMI by ethnicity, IMD and income. Mean FFMI differed across ethnic groups at each sweep. Those of White, Black and Black British and of other ethnicity typically had higher mean FFMI, and those of Indian ethnicity typically had the lowest. Those in the lowest income bracket typically had the highest FFMI across sweeps, and those in the highest income bracket had the lowest FFMI. For the IMD there was little difference in FFMI across groups at age 7, but at age 11 to 17 this was more notable. Those in the most deprived areas had the highest FFMI and those in the least deprived areas had the lowest.

Table 6-3 shows the mean FM:FFM ratio according to ethnicity, IMD and income. Similar to FMI, those of Black and Black British ethnicity and those of Pakistani and Bangladeshi typically had the highest FM:FFM ratio, whilst those of white ethnicity typically had the lowest. Those in the most deprived areas and in the lowest income fifth typically had a higher FM:FFM ratio, and those in the most advantaged and highest income fifth had the lowest FM:FFM ratio.

There was no difference between boys and girls in terms of IMD (Appendix 6.5). However, there was a difference in IMD across ethnic groups. Those who were of white ethnicity had the lowest percentage in the most deprived areas (26.5%) and those of Pakistani and Bangladeshi ethnicity had the highest, where 79.7% of the sample lived in the most deprived areas. The white ethnic group had the highest percentage living in the most advantaged areas (17.0%) whilst Black and Black British had the lowest proportion living in the most advantaged areas (0.8%).

**Table 6-1.** Mean Fat Mass Index according to Ethnicity, Index of Multiple Deprivation and Income

	<b>Fat Mass Index</b>											
	<b>Age 7</b>			<b>Age 11</b>			<b>Age 14</b>			<b>Age 17</b>		
	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>
<b>Ethnicity</b>			13,339			12,635			10,762			9,114
White	2.28	0.85	11,191	1.62	0.86	10,477	3.11	1.96	8,841	3.77	2.37	7,372
Mixed	2.37	1.01	366	1.70	0.91	359	3.42	2.10	301	4.26	2.73	265
Indian	2.35	1.15	334	1.68	0.89	320	3.24	1.97	296	3.77	3.60	272
Pakistani and Bangladeshi	2.45	1.36	841	1.89	1.17	896	3.68	2.85	811	4.31	3.95	725
Black and Black British	2.59	1.21	433	1.82	1.07	405	3.50	2.08	344	4.95	3.13	322
Other Ethnic Group	2.30	0.94	174	1.73	0.92	178	3.46	1.88	169	4.19	3.50	158
<i>p value</i>	<0.001			<0.001			<0.001			<0.001		
<b>IMD</b>			13,436			12,273			10,829			9,169
Most Deprived 20%	2.43	1.12	3,988	1.76	1.04	3,861	3.46	2.26	3,165	4.31	3.29	2,605
40%	2.34	0.98	3,025	1.71	0.94	2,808	3.24	2.08	2,394	4.08	2.87	2,018
60%	2.29	0.82	2,347	1.63	0.84	2,222	3.18	1.97	1,899	3.76	2.18	1,602
80%	2.21	0.75	1,932	1.56	0.77	1,840	2.99	1.83	1,603	3.55	2.08	1,401
Least Deprived 20%	2.18	0.73	2,144	1.48	0.73	1,992	2.77	1.70	1,768	3.46	1.96	1,543
<i>p value</i>	<0.001			<0.001			<0.001			<0.001		
<b>Income</b>			13,210			12,502			10,654			9,033
Lowest Fifth	2.40	1.00	2,926	1.76	0.93	2,822	3.49	2.01	2,242	4.38	2.98	1,783
2 <sup>nd</sup> Fifth	2.38	0.97	2,836	1.72	0.93	2,679	3.40	2.13	2,251	4.22	2.95	1,869
3 <sup>rd</sup> Fifth	2.31	0.89	2,557	1.65	0.88	2,372	3.09	1.96	2,026	3.77	2.31	1,714
4 <sup>th</sup> Fifth	2.27	0.86	2,521	1.60	0.85	2,389	3.00	1.97	2,098	3.70	2.45	1,819
Highest Fifth	2.12	0.74	2,370	1.43	0.77	2,240	2.67	1.77	2,037	3.28	1.81	1,848
<i>p value</i>	<0.001			<0.001			<0.001			<0.001		

**Table 6-1 Footnote:** *P value for Adjusted Wald Test. IMD – Index of multiple deprivation; SD – Standard Deviation.*



**Table 6-2.** Differences in Fat-Free Mass Index according to Ethnicity, Index of Multiple Deprivation and Income

<i>Fat-Free Mass Index</i>												
	<b>Age 7</b>			<b>Age 11</b>			<b>Age 14</b>			<b>Age 17</b>		
	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>
<b>Ethnicity</b>			13,339			12,635			10,762			9,114
White	11.91	0.87	11,191	11.72	1.14	10,477	13.51	1.47	8,841	20.13	2.74	7,372
Mixed	11.89	0.87	366	11.72	1.28	359	13.45	1.61	301	20.88	3.48	265
Indian	11.40	1.01	334	11.21	1.15	320	12.89	1.65	296	19.69	2.83	272
Pakistani and Bangladeshi	11.42	1.37	841	11.43	1.40	896	13.24	2.17	811	19.78	3.95	725
Black and Black British	11.97	1.05	433	11.78	1.29	405	13.52	1.49	344	21.07	3.30	322
Other Ethnic Group	11.54	1.04	174	11.20	1.21	178	13.50	1.44	169	20.22	3.02	158
<i>p value</i>		<0.001			<0.001			<0.001			0.001	
<b>IMD</b>			13,436			12,723			10,829			9,169
Most Deprived 20%	11.91	1.14	3,988	11.78	1.32	3,861	13.57	1.65	3,165	20.26	3.34	2,605
40%	11.89	0.99	3,025	11.75	1.22	2,808	13.55	1.63	2,394	20.45	3.09	2,018
60%	11.87	0.79	2,347	11.69	1.25	2,222	13.51	1.41	1,899	20.00	2.70	1,602
80%	11.84	0.74	1,932	11.63	0.94	1,840	13.42	1.40	1,603	19.97	2.52	1,401
Least Deprived 20%	11.84	0.83	2,144	11.55	0.94	1,992	13.27	1.39	1,768	20.17	2.52	1,543
<i>p value</i>		0.16			<0.001			<0.001			0.028	
<b>Income</b>			13,210			12,502			10,654			9,033
Lowest Quintile	11.90	1.03	2,926	11.78	1.12	2,822	13.64	1.51	2,242	20.37	2.93	1,783
2 <sup>nd</sup> Quintile	11.91	0.98	2,836	11.74	1.12	2,679	13.55	1.53	2,251	20.30	3.24	1,869
3 <sup>rd</sup> Quintile	11.90	0.85	2,557	11.75	1.47	2,372	13.47	1.44	2,026	20.12	2.80	1,714
4 <sup>th</sup> Quintile	11.88	0.85	2,521	11.67	1.08	2,389	13.44	1.52	2,098	20.11	2.84	1,819
Highest Quintile	11.77	0.83	2,370	11.47	0.98	2,240	13.20	1.49	2,037	19.95	2.55	1,848
<i>p value</i>		<0.001			<0.001			<0.001			0.13	

**Table 6-2 Footnote:** *P value for Adjusted Wald Test. IMD – Index of multiple deprivation; SD – Standard Deviation.*

**Table 6-3.** Differences in Fat Mass to Fat-Free Mass Ratio according to Ethnicity, Index of Multiple Deprivation and Income

	<b>Fat Mass:Fat-Free Mass Ratio</b>											
	<b>Age 7</b>			<b>Age 11</b>			<b>Age 14</b>			<b>Age 17</b>		
	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>
<b>Ethnicity</b>			13,339			12,635			10,762			9,114
White	0.27	0.09	11,191	0.29	0.14	10,477	0.29	0.17	8,841	0.30	0.18	7,372
Mixed	0.28	0.10	366	0.32	0.16	359	0.32	0.18	301	0.35	0.22	265
Indian	0.29	0.13	334	0.33	0.16	320	0.32	0.17	296	0.30	0.20	272
Pakistani and Bangladeshi	0.30	0.15	841	0.35	0.20	896	0.35	0.24	811	0.33	0.27	725
Black and Black British	0.32	0.14	433	0.35	0.20	405	0.33	0.17	344	0.41	0.23	322
Other Ethnic Group	0.28	0.10	174	0.32	0.15	178	0.32	0.15	169	0.31	0.21	158
<i>p value</i>	<0.001			<0.001			<0.001			<0.001		
<b>IMD</b>			13,436			12,723			10,829			9,169
Most Deprived 20%	0.29	0.12	3,988	0.32	0.17	3,861	0.32	0.19	3,165	0.33	0.24	2,605
40%	0.28	0.11	3,025	0.31	0.17	2,808	0.30	0.17	2,394	0.33	0.21	2,018
60%	0.27	0.10	2,347	0.30	0.14	2,222	0.30	0.16	1,899	0.29	0.17	1,602
80%	0.26	0.08	1,932	0.29	0.12	1,840	0.28	0.16	1,603	0.29	0.16	1,401
Least Deprived 20%	0.26	0.08	2,144	0.28	0.12	1,992	0.27	0.15	1,768	0.28	0.16	1,543
<i>p value</i>	<0.001			<0.001			<0.001			<0.001		
<b>Income</b>			13,210			12,502			10,654			9,033
Lowest Quintile	0.28	0.11	2,926	0.32	0.15	2,822	0.32	0.17	2,242	0.34	0.22	1,783
2 <sup>nd</sup> Quintile	0.28	0.11	2,836	0.31	0.17	2,679	0.32	0.18	2,251	0.33	0.22	1,869
3 <sup>rd</sup> Quintile	0.27	0.10	2,557	0.30	0.15	2,372	0.29	0.17	2,026	0.29	0.17	1,714
4 <sup>th</sup> Quintile	0.27	0.10	2,521	0.30	0.14	2,389	0.29	0.17	2,098	0.30	0.18	1,819
Highest Quintile	0.26	0.08	2,370	0.27	0.13	2,240	0.26	0.15	2,037	0.27	0.15	1,848
<i>p value</i>	<0.001			<0.001			<0.001			<0.001		

**Table 6-3 Footnote:** *P* value for Adjusted Wald Test. *IMD* – Index of multiple deprivation; *SD* – Standard Deviation.

Of those in the lowest income bracket, the majority lived in the most deprived neighbourhoods according to IMD (60.0%), whilst only 2.6% were living in the most advantaged areas. For those in the highest income bracket, the majority lived in the least deprived areas by IMD (35.7%) whilst only 7.4% of households on the highest income were living in the most deprived areas. Those who were unemployed were most likely to live in the most deprived areas (70.1%) with only 1.9% living in the most advantaged areas. For those in managerial professions, the largest proportion lived in the most advantaged areas (27.0%), and this was a greater proportion than any other occupational groups. A similar pattern was observed for parental education where 64.4% of those with no qualifications lived in the most deprived areas, whilst among those with the highest qualification, 31.8% were living in the most advantaged areas.

Similar to the IMD, there was no differences in income fifths between boys and girls, whereas differences were observed for ethnicity, parental occupation and parental education (Appendix 6.6). Those of Black and Black British ethnicity and Pakistani and Bangladeshi had a higher proportion of the sample in the lowest income fifth compared to other ethnic groups, whilst there was a higher proportion of those of white ethnicity in the highest income bracket. Those of Indian ethnicity had the lowest proportion in the lowest income fifth (17.5%) whilst those of Pakistani and Bangladeshi had the lowest proportion in the highest income fifth (2.3%).

Households with no qualifications had the highest proportion in the lowest income bracket (66.2%), whilst those with and NVQ level 5 had the highest proportion in the highest income bracket (61.5%). For all NVQ levels lower than NVQ level 3, less than 9% of the sample were in the highest income bracket. Looking at income distribution according to parental occupation, households that were unemployed had the highest proportion in the lowest income bracket (74.7%), whilst those in managerial and professional jobs had the highest proportion in the top fifth of the income distribution (36%) compared to households with other occupations.

### 6.3.2 Inequalities in FMI, FFMI and FM:FFM Ratio

In unadjusted models (model 1), sex-adjusted models (models 2 and 3) and models adjusted for ethnicity (model 4), FMI and FM:FFM ratio were greater in those from more disadvantaged SEP at age 7 and associations widened with age. For models 1 to 3, the addition of sex (model 2) and an age by sex and SEP by sex interaction (model 3) resulted in slight reductions in the estimate for inequality at age 7, but not for the change in inequality with age. Additional adjustment for ethnicity (model 4), resulted in the inequality at age 7 being similar to model 3 for FMI and FM:FFM ratio.

Model 4 showed that inequalities increased over five-fold for FMI and three-fold for FM:FFM ratio between ages 7 and 17 (Table 6-4, Figure 6-2a and Figure 6-5a). At 7 years, mean FMI in the most deprived IMD group was 0.12 kg/m<sup>B</sup> (95% CI: 0.05 to 0.20, p=0.001) higher than in the most advantaged IMD group. By age 17, the difference in FMI was 0.63 kg/m<sup>B</sup> (95% CI: 0.50 to 0.76, p<0.001) (Model 4, Figure 6-2a). The ratio of FM:FFM was 0.018 (95% CI: 0.009 to 0.027, p=0.01) higher in the most deprived IMD group compared to the least deprived at age 7, and 0.045 (95%CI: 0.035 to 0.055, p<0.001) higher by 17 years of age (Model 4, Figure 6-5a).

**Table 6-4.** Socioeconomic Inequalities by Index of Multiple Deprivation in Fat Mass Index (FMI), Fat-Free Mass Index (FFMI) and Fat Mass to Fat-Free Mass (FM:FFM) Ratio at Age 7, and Changes in Inequalities across Childhood and Adolescence.

<i>Model</i>	<i>Difference in body composition at age 7 by IMD Group (1: high deprivation to 5: low deprivation)</i>		<i>IMD x Age (Years) Interaction</i>		<i>Random Effects: Intercept</i>	<i>Random Effects: Slope</i>	<i>Age x Sex</i>	<i>SEP x Sex</i>
	Coefficient (95% CI)	p value	Coefficient (95% CI)	p value	SD (95% CI)	SD (95% CI)	p value	p value
<b>Fat Mass Index</b>								
1: Age	-0.048 (-0.064, -0.032)	<0.001	-0.013 (-0.016, -0.01)	<0.001	0.24 (0.19, 0.30)	0.21 (0.21, 0.22)	N/A	N/A
2: Model 1 + Sex	-0.043 (-0.059, -0.028)	<0.001	-0.013 (-0.016, -0.01)	<0.001	0.13 (0.07, 0.26)	0.21 (0.21, 0.22)	N/A	N/A
3: Model 2 + Age*Sex + SEP*Sex	-0.033 (-0.052, -0.014)	0.001	-0.013 (-0.016, -0.009)	<0.001	0.13 (0.07, 0.26)	0.21 (0.21, 0.22)	<0.001	0.034
4: Model 3 + Ethnicity	-0.031 (-0.049, -0.012)	0.001	-0.013 (-0.016, -0.009)	<0.001	0.12 (0.058, 0.27)	0.21 (0.21, 0.22)	<0.001	0.031
5: Model 4 + education + occupational social class + income	-0.001 (-0.021, 0.019)	0.913	-0.013 (-0.016, -0.009)	<0.001	0.093 (0.023, 0.37)	0.21 (0.21, 0.22)	<0.001	0.032
<b>Fat-Free Mass Index</b>								
1: Age	-0.075 (-0.104, -0.046)	<0.001	0.001 (-0.003, 0.006)	0.56	8.79E-09 (8.79E-09, 8.79E-09)	0.17 (0.17, 0.18)	N/A	N/A
2: Model 1 + Sex	-0.064(-0.093, -0.035)	<0.001	0.000 (-0.004, 0.005)	0.91	6.86E-08 (6.86E-08, 6.86E-08)	0.13 (0.12, 0.13)	N/A	N/A
3: Model 2 + Age*Sex + SEP*Sex	-0.066 (-0.098, -0.034)	<0.001	0.003 (-0.002, 0.007)	0.23	3.99E-07 (3.99E-07, 3.99E-07)	0.083 (0.075, 0.091)	<0.001	0.17
4: Model 3 + Ethnicity	-0.064 (-0.096, -0.032)	<0.001	0.002 (-0.002, 0.006)	0.27	4.26E-07 (4.26E-07, 4.26E-07)	0.082 (0.074, 0.090)	<0.001	0.17
5: Model 4 + education + occupational social class + income	-0.046 (-0.079, -0.013)	0.007	0.002 (-0.002, 0.006)	0.27	4.29E-07 (4.29E-07, 4.29E-07)	0.081 (0.073, 0.091)	<0.001	0.17
6: Model 4 + FMI	-0.041 (-0.069, -0.013)	0.004	0.012 (0.008, 0.016)	<0.001	8.61E-11 (8.61E-11, 8.61E-11)	1.24E-10 (1.24E-10, 1.24E-10)	<0.001	0.089
7: Model 5 + FMI	-0.055 (-0.084, -0.26)	<0.001	0.012 (0.008, 0.016)	<0.001	6.24E-11 (6.24E-11, 6.24E-11)	8.55E-11 (8.55E-11, 8.55E-11)	<0.001	0.088
<b>Fat Mass to Fat-Free Mass Ratio</b>								
1: Age	-0.006 (-0.007, -0.004)	<0.001	-0.001 (-0.001, -0.001)	<0.001	0.10 (0.099, 0.10)	0.014 (0.014, 0.015)	N/A	N/A
2: Model 1 + Sex	-0.005 (-0.007, -0.003)	<0.001	-0.001 (-0.001, -0.001)	<0.001	0.097 (0.095, 0.098)	0.013 (0.013, 0.014)	N/A	N/A
3: Model 2 + Age*Sex + SEP*Sex	-0.005 (-0.007, -0.003)	<0.001	-0.001 (-0.001, 0.00)	<0.001	0.10 (0.099, 0.10)	0.010 (0.010, 0.010)	<0.001	0.45
4: Model 3 + Ethnicity	-0.004 (-0.007, -0.002)	<0.001	-0.001 (-0.001, 0.00)	<0.001	0.10 (0.099, 0.10)	0.010 (0.010, 0.010)	<0.001	0.41
5: Model 4 + education + occupational social class + income	-0.002 (-0.004, 0.001)	0.162	-0.001 (-0.001, 0.00)	<0.001	0.10 (0.098, 0.10)	0.010 (0.010, 0.010)	<0.001	0.42

**Table 6-4 Footnote:** Index of Multiple Deprivation (IMD) groups are ranked from 1 (most deprived) to 5 (least deprived). Coefficients for difference in body composition at age 7 show the change in body composition for one unit increase in IMD group at age 7. The coefficients for IMD x Age Interaction show the change in body composition per one unit increase in IMD per one year increase in age. Columns “Age x Sex” represents the interaction for age and sex, whilst “SEP x Sex” represents the interaction with SEP (IMD) and sex. Results presented are for models that include the IMD\*Sex interaction (Shown as SEP\*sex in table), with the exception of models 1 and 2. Abbreviations: FMI – Fat Mass Index; FFMI - fat-free mass index; FM:FFM - fat mass to fat-free mass ratio; SEP – Socioeconomic Position; 95% CI – 95% Confidence Intervals; N/A - not applicable.

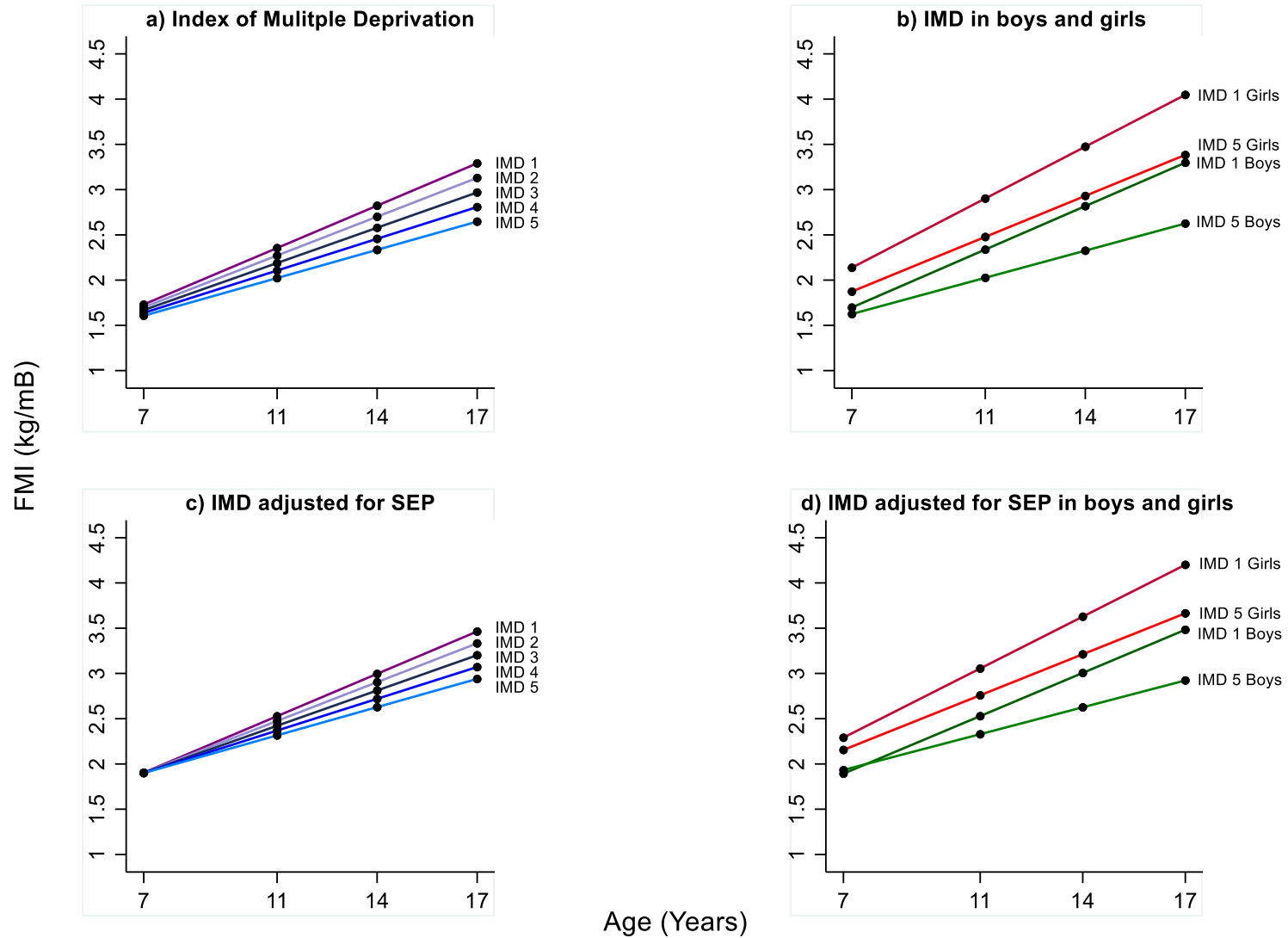
**Table 6-5.** Socioeconomic Inequalities by Income in Fat Mass Index (FMI), Fat-Free Mass Index (FFMI) and Fat Mass to Fat-Free Mass (FM:FFM) Ratio at Age 7, and Changes in Inequalities across Childhood and Adolescence

<b>Model</b>	<b>Difference in body composition at age 7 by Income (1: lowest income fifth, to 5: highest income fifth)</b>		<b>Income x Age (Years) Interaction</b>		<b>Random Effects: Intercept</b>	<b>Random Effects: Slope</b>	<b>Age x Sex</b>	<b>SEP x Sex</b>
	Coefficient (95% CI)	p value	Coefficient (95% CI)	p value	SD (95% CI)	SD (95% CI)	p value	p value
<b>Fat Mass Index</b>								
1: Age	-0.044 (-0.057, -0.031)	<0.001	-0.013 (-0.016, -0.009)	<0.001	0.23 (0.19, 0.29)	0.21 (0.21, 0.22)	N/A	N/A
2: Model 1 + Sex	-0.043 (-0.056, -0.030)	<0.001	-0.013 (-0.016, -0.009)	<0.001	0.12 (0.055, 0.27)	0.21 (0.21, 0.22)	N/A	N/A
3: Model 2 + Age*Sex + SEP*Sex	-0.036 (-0.053, -0.019)	<0.001	-0.013 (-0.016, -0.009)	<0.001	0.12 (0.055, 0.27)	0.21 (0.21, 0.22)	<0.001	0.20
4: Model 3 + Ethnicity	-0.034 (-0.051, -0.017)	0.001	-0.013 (-0.016, -0.009)	<0.001	0.12 (0.047, 0.28)	0.21 (0.21, 0.22)	<0.001	0.19
<b>Fat-Free Mass Index</b>								
1: Age	-0.08 (-0.11, -0.055)	<0.001	0.008 (0.003, 0.013)	0.001	7.76E-09 (7.76E-09, 7.76E-09)	0.18 (0.17, 0.18)	N/A	N/A
2: Model 1 + Sex	-0.074 (-0.10, -0.048)	<0.001	0.006 (0.002, 0.011)	0.005	7.17E-09 (7.17E-09, 7.17E-09)	0.13 (0.12, 0.13)	N/A	N/A
3: Model 2 + Age*Sex + SEP*Sex	-0.072 (-0.102, -0.042)	<0.001	0.007 (0.003, 0.011)	0.001	4.58E-07 (4.58E-07, 4.58E-07)	0.083 (0.076, 0.092)	<0.001	0.58
4: Model 3 + Ethnicity	-0.074 (-0.104, -0.045)	<0.001	0.007 (0.002, 0.011)	0.002	3.77E-07 (3.77E-07, 3.77E-07)	0.082 (0.074, 0.091)	<0.001	0.59
6: Model 4 + FMI	-0.056 (-0.082, -0.03)	<0.001	0.017 (0.013, 0.02)	<0.001	9.52E-11 (9.52E-11, 9.52E-11)	1.22E-10 (1.22E-10, 1.22E-10)	<0.001	0.66
<b>Fat Mass to Fat-Free Mass Ratio</b>								
1: Age	-0.005 (-0.006, -0.003)	<0.001	-0.001 (-0.001, -0.001)	<0.001	0.10 (0.099, 0.10)	0.014 (0.014, 0.015)	N/A	N/A
2: Model 1 + Sex	-0.004 (-0.006, -0.003)	<0.001	-0.001 (-0.001, -0.001)	<0.001	0.096 (0.095, 0.098)	0.013 (0.013, 0.014)	N/A	N/A
3: Model 2 + Age*Sex + SEP*Sex	-0.004 (-0.006, -0.002)	<0.001	-0.001 (-0.001, -0.001)	<0.001	0.10 (0.099, 0.10)	0.010 (0.010, 0.010)	<0.001	0.22
4: Model 3 + Ethnicity	-0.003 (-0.005, -0.001)	0.002	-0.001 (-0.001, -0.001)	<0.001	0.10 (0.099, 0.10)	0.010 (0.010, 0.010)	<0.001	0.20

**Table 6-5 Footnote:** Income quintiles are ranked from the lowest to the highest fifth of income. Coefficients for difference in body composition at age 7 show the change in body composition for one unit increase in income fifth at age 7. The coefficients for Income x Age Interaction show the change in body composition per one unit increase in income fifth per one year increase in age. Columns “Age x Sex” represents the interaction for age and sex, whilst “SEP x Sex” represents the interaction with SEP (income) and sex. Results presented are for models that include the income\*Sex interaction (Shown as SEP\*Sex in table), with the exception of models 1 and 2. Abbreviations: FMI – Fat Mass Index; FFMI - fat-free mass index; FM:FFM - fat mass to fat-free mass ratio; SEP – Socioeconomic Position; 95% CI – 95% Confidence Intervals; N/A - not applicable.

**Figure 6-2.** Estimated Mean Trajectories of Fat Mass Index (FMI) by Index of Multiple Deprivation (IMD)

## Trajectories of Fat Mass Index (FMI) by Index of Multiple Deprivation (IMD)

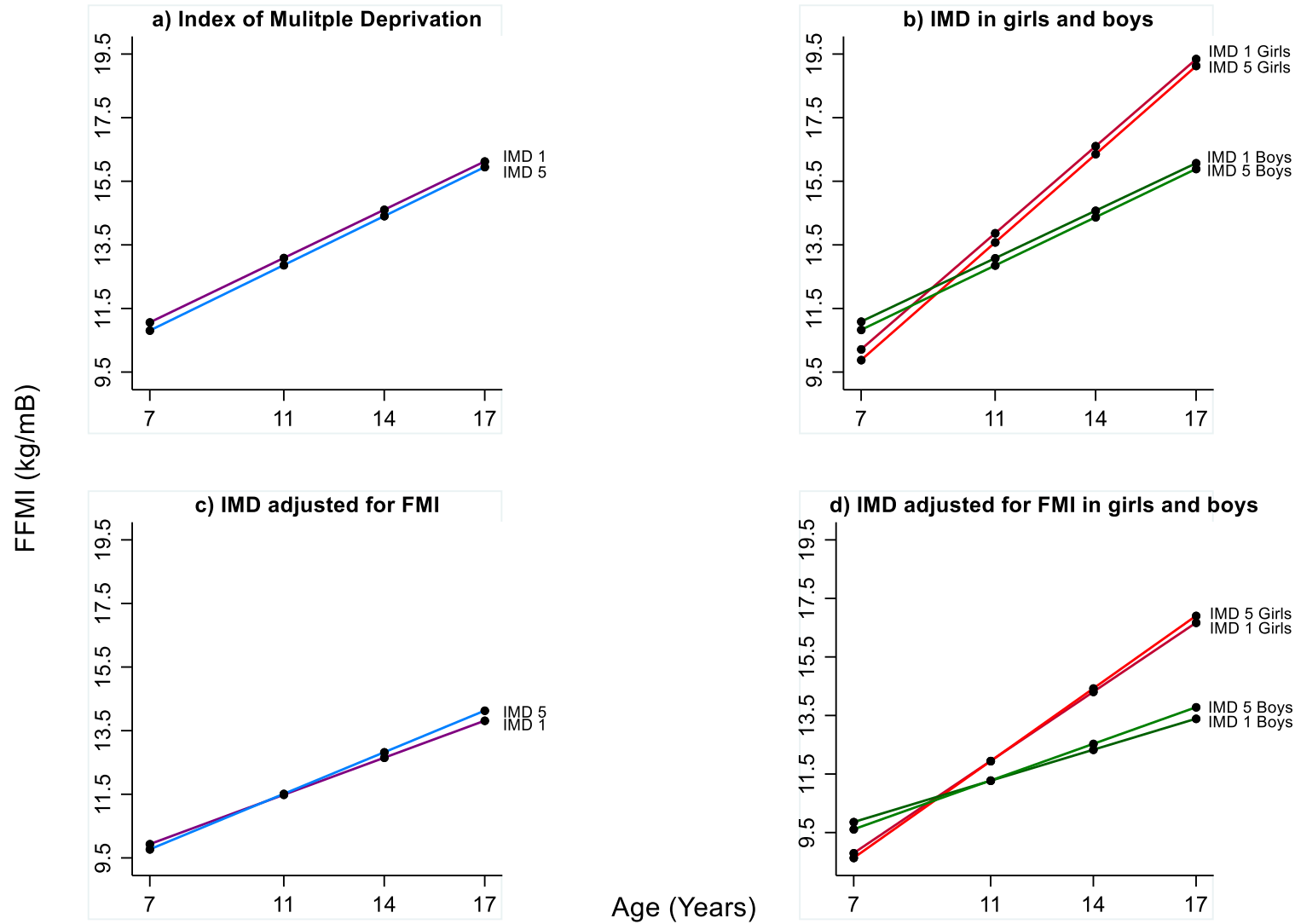


**Figure 6-2. Footnote**

Difference in FMI ( $\text{kg}/\text{m}^2$ ) at ages 7, 11, 14 and 17 by index of multiple deprivation group. Graph a) adjusted only for sex and ethnicity (model 4); b) Model 4 in boys and girls separately; c) adjusted for sex, ethnicity, parental education, family income, NS-SEC (model 5); d) Model 5 in boys and girls separately.

**Figure 6-3.** Trajectories of Fat-Free Mass Index (FFMI) by Index of Multiple Deprivation (IMD), with Adjustment for Fat Mass Index (FMI)

### Trajectories of Fat-Free Mass Index (FFMI) by Index of Multiple Deprivation (IMD)

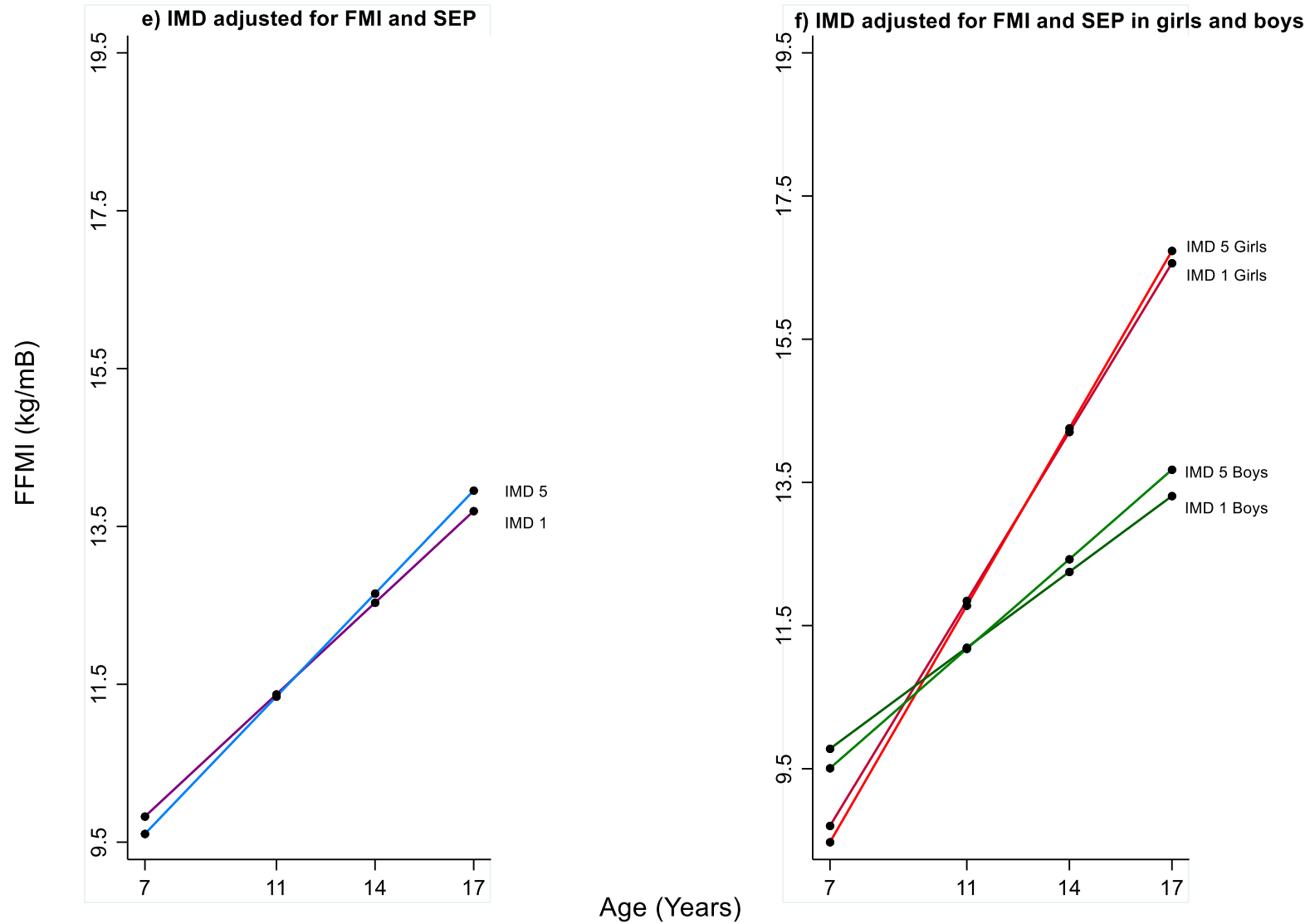


**Figure 6-3. Footnote**  
 Difference in FFMI ( $\text{kg/m}^2$ ) at ages 7, 11, 14 and 17 by index of multiple deprivation group. Graph a) adjusted only for sex and ethnicity (model 4); b) Model 4 in boys and girls separately; c) Adjusted for sex, ethnicity and FMI (model 6); d) Model 6 in boys and girls separately.



**Figure 6-4.** Trajectories of Fat Free Mass Index (FFMI) by Index of Multiple Deprivation (IMD) with Adjustment for Fat Mass Index (FMI) and Family Socioeconomic Position

## Trajectories of Fat-Free Mass Index (FFMI) by Index of Multiple Deprivation (IMD)

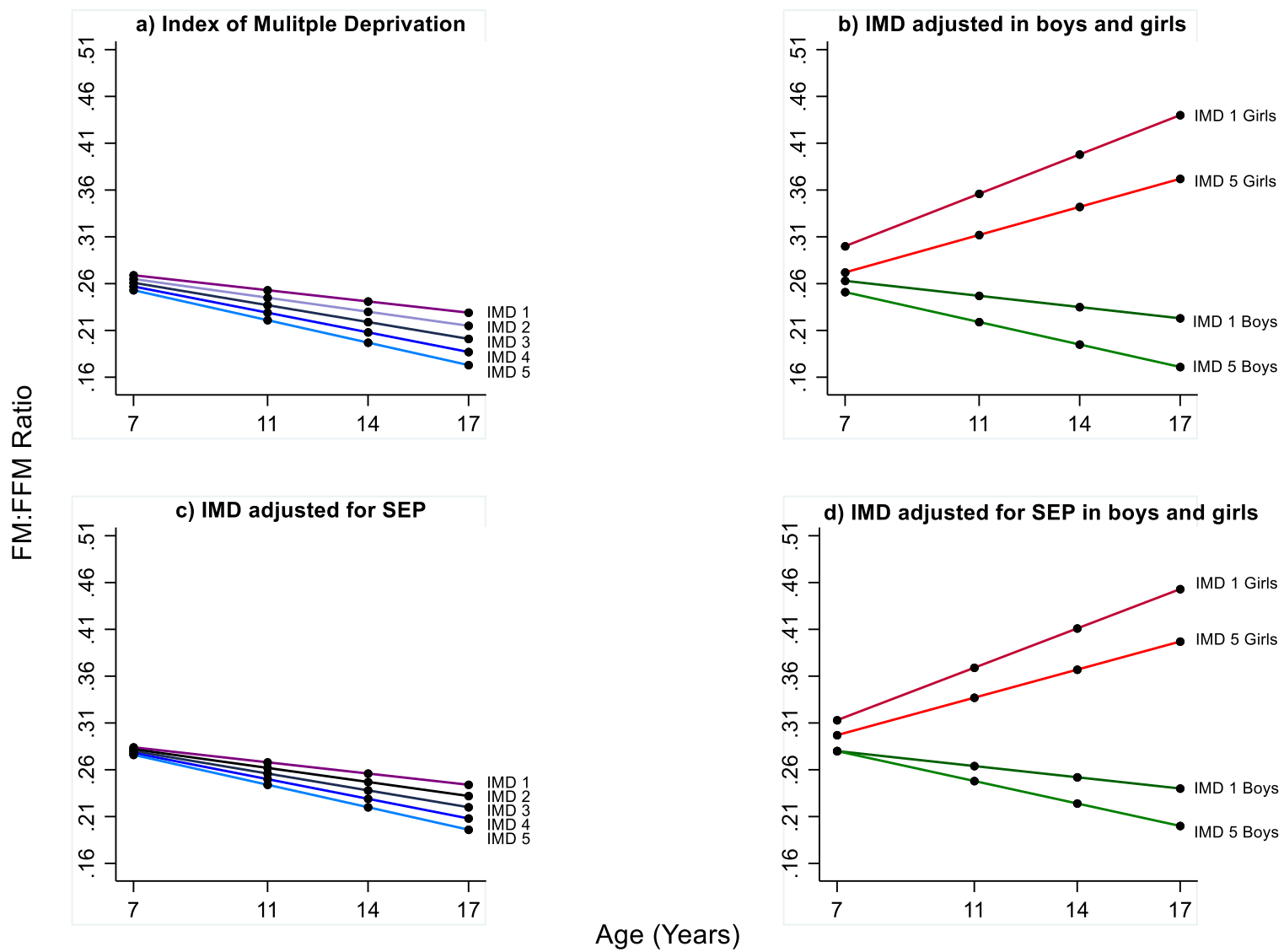


**Figure 6-4. Footnote**

Difference in FFMI (kg/m<sup>2</sup>) at ages 7, 11, 14 and 17 by index of multiple deprivation group. Graph e) Adjusted for sex, ethnicity, FMI, parental education, family income, NS-SEC (model 7); f) Model 7 in boys and girls separately.

**Figure 6-5.** Trajectories of FM:FFM Ratio by Index of Multiple Deprivation (IMD)

### Trajectories of FM:FFM Ratio by Index of Multiple Deprivation (IMD)



**Figure 6-5. Footnote**

Difference in FM:FFM ratio at ages 7, 11, 14 and 17 by index of multiple deprivation group. Graph a) adjusted only for sex and ethnicity (model 4); b) Model 4 in boys and girls separately; c) adjusted for sex, ethnicity, parental education, family income, NS-SEC (model 5); d) Model 5 in boys and girls separately

Inequalities in trajectories in FMI and FM:FFM according to income were similar to those observed for the IMD (Table 6-5). At age 7 the difference in FMI between those in the highest and lowest income groups was 0.14kg/m<sup>B</sup> (95% CI: 0.067 to 0.20, p<0.001) and by the age of 17 this difference had increased by almost five-fold to 0.64kg/m<sup>2</sup> (95% CI: 0.51 to 0.77, p<0.001. Appendix A6.7). For FM:FFM ratio the difference at age 7 was 0.013 (95% CI: 0.005 to 0.021, p=0.002) increasing to 0.049 (95% CI: 0.039 to 0.059, p<0.001) at age 17 (Appendix A6.9).

At age 7, FFMI was 0.26 kg/m<sup>B</sup> (95% CI: 0.13 to 0.38, p<0.001) higher in the most deprived IMD group, and the difference slightly declined to 0.16 kg/m<sup>B</sup> (95% CI: -0.30 to -0.022, p=0.023) by age 17 (Model 4, Figure 6-3a). Similar differences in FFMI were observed at age 7 according to income, but by age 17 there was no longer a difference in FFMI between groups (0.034 kg/m<sup>B</sup>, 95% CI: -0.10 to 0.17, p=0.62). After adjusting for FMI (model 6), those in the most advantaged IMD groups had the fastest increase in FFMI with age. By age 17 the association between IMD and FFMI had reversed, so that those in the most deprived IMD group had a FFMI -0.32 kg/m<sup>B</sup> (95% CI: -0.43 to -0.20, p<0.001) lower than the most advantaged IMD group (Figure 6-3c). A similar pattern was observed for income, but a larger difference between income groups was observed at 17 years when adjusting for FMI (-0.45 kg/m<sup>B</sup>, 95% CI: -0.56 to -0.343, p<0.001. Appendix A6.8).

For FMI, there was a large standard deviation in FMI at age 7 between individuals in model 1 (0.24 SD, 95% CI: 0.19, 0.30), which got smaller with additional adjustments (Model 4, 0.12 SD, 95% CI: 0.058, 0.27). However, the standard deviation in rate of change remained consistent across models (SD 0.21, 95% CI 0.21 to 0.22). Random effects were small (near zero) for FFMI intercepts in all models, indicating very little variation between individuals. However, there was variation in rate of change over time in models 1 to 4 (0.17 to 0.081 SD, respectively), whilst adjusting for FMI resulted in the SD in rate of change reducing to near zero. For FM:FFM ratio, there was approximately a 0.1SD variation between individuals at age 7, whilst the standard deviation in rate of change reduced from 0.014SD (Model 1) to 0.01SD (Model 4) with additional adjustments. Similar patterns in the random effects were observed for models with income.

### 6.3.3 Sex Differences in Inequalities

Differences were observed between boys and girls in the mean trajectories for all body composition measures as indicated by sex by age interactions. However, evidence of a sex by SEP interaction was only observed for the association between IMD and FMI (Table 6-4).

In sex-stratified models 4 and 5, inequalities by IMD in FMI were evident in girls but not in boys at age 7, but inequalities widened faster in boys (Table 6-6, Figure 6-2b and 6-2d). In models adjusted for ethnicity (model 4) girls in the most deprived IMD group had a mean FMI 0.26 kg/m<sup>B</sup> (95% CI: 0.17 to 0.36, p<0.001) higher than those in the most advantaged IMD group at 7 years of age, whilst the difference was 0.07kg/m<sup>B</sup> (95% CI: -0.024 to 0.17, p=0.14) in boys. By 17 years, the difference in FMI between groups had almost tripled in girls (0.67 kg/m<sup>B</sup>, 95% CI: 0.45 to 0.76, p<0.001), whilst there was almost a ten-fold increase in the difference among boys (0.68kg/m<sup>B</sup>, 95% CI: 0.50 to 0.87, p<0.001, Figure 6-2b). The standard deviation for the intercept for FMI at age 7 was larger in girls (SD

0.58, 95% CI 0.55 to 0.61) compared to in boys (near zero). However, there was a larger standard deviation in the rate of change in boys (SD 0.23, 95% CI: 0.23 to 0.24) compared to girls (SD 0.16, 95% CI 0.15 to 0.16). There was no sex by income interaction observed for FMI, and inequalities were observed in both boys and girls at age 7 and widening of inequalities was similar (Table 6-7).

**Table 6-6.** Socioeconomic Inequalities and Changes in Inequalities in Fat Mass Index (FMI), Fat-Free Mass Index (FFMI) and Fat Mass to Fat-Free Mass (FM:FFM) Ratio by the Index of Multiple Deprivation (IMD) in Boys and Girls

<i>Model</i>	<i>Difference in body composition at age 7 by Index of Multiple Deprivation Group (1: high deprivation to 5: low deprivation)</i>		<i>Index of Multiple Deprivation x Age (Years) Interaction</i>		<i>Random Effects: Intercept</i>	<i>Random Effects: Slope</i>
	Coefficient (95% CI)	p value	Coefficient (95% CI)	p value	SD (95% CI)	SD (95% CI)
<b>BOYS</b>						
<b>Fat Mass Index</b>						
1: Age	-0.02 (-0.043, 0.004)	0.10	-0.015 (-0.02, -0.01)	<0.001	1.57E-10 (1.57E-10, 1.57E-10)	0.23 (0.23, 0.24)
4: Model 1 + Ethnicity	-0.018 (-0.041, 0.006)	0.14	-0.015 (-0.02, -0.01)	<0.001	1.07E-09 (1.07E-09, 1.07E-09)	0.23 (0.23, 0.24)
5: Model 4 + education + occupational social class + income	0.01 (-0.015, 0.035)	0.43	-0.015 (-0.02, -0.01)	<0.001	1.72E-09 (1.72E-09, 1.72E-09)	0.23 (0.23, 0.24)
<b>Fat-Free Mass Index</b>						
1: Age	-0.066 (-0.1, -0.032)	<0.001	0.002 (-0.003, 0.007)	0.46	8.62E-07 (8.62E-07, 8.62E-07)	0.13 (0.12, 0.13)
4: Model 1 + Ethnicity	-0.065 (-0.099, -0.031)	<0.001	0.002 (-0.003, 0.007)	0.50	1.06E-06 (1.06E-06, 1.06E-06)	0.13 (0.12, 0.14)
5: Model 4 + education + occupational social class + income	-0.045 (-0.081, -0.01)	0.013	0.002 (-0.003, 0.007)	0.50	1.18E-06 (1.18E-06, 1.18E-06)	0.13 (0.12, 0.13)
6: Model 4 + FMI	-0.062 (-0.089, -0.036)	<0.001	0.016 (0.012, 0.02)	<0.001	2.37E-09 (2.37E-09, 2.37E-09)	0.12 (0.12, 0.13)
7: Model 5 + FMI	-0.068 (-0.096, -0.04)	<0.001	0.016 (0.012, 0.02)	<0.001	1.24E-09 (1.24E-09, 1.24E-09)	0.12 (0.11, 0.13)
<b>Fat Mass to Fat-Free Mass Ratio</b>						
1: Age	-0.003 (-0.006, -0.001)	0.09	-0.001 (-0.001, -0.001)	<0.001	0.097 (0.094, 0.099)	0.009 (0.008, 0.009)
4: Model 1 + Ethnicity	-0.003 (-0.006, 0.00)	0.023	-0.001 (-0.001, -0.001)	<0.001	0.096 (0.093, 0.098)	0.009 (0.008, 0.009)
5: Model 4 + education + occupational social class + income	0.00 (-0.003, 0.002)	0.74	-0.001 (-0.001, -0.001)	<0.001	0.095 (0.093, 0.098)	0.009 (0.008, 0.009)
<b>GIRLS</b>						
<b>Fat Mass Index</b>						
1: Age	-0.069 (-0.093, -0.046)	<0.001	-0.01 (-0.014, -0.006)	<0.001	0.58 (0.55, 0.61)	0.16 (0.15, 0.16)
4: Model 1 + Ethnicity	-0.066 (-0.09, -0.042)	<0.001	-0.01 (-0.014, -0.006)	<0.001	0.58 (0.55, 0.61)	0.16 (0.15, 0.16)
5: Model 4 + education + occupational social class + income	-0.034 (-0.059, -0.008)	0.01	-0.01 (-0.014, -0.006)	<0.001	0.57 (0.54, 0.61)	0.16 (0.15, 0.16)

<b>Fat-Free Mass Index</b>							
<i>1: Age</i>	-0.087 (-0.132, -0.042)	<0.001	0.003 (-0.003 to 0.01)	0.33	5.50E-09 (5.50E-09, 5.50E-09)	1.63E-08 (1.63E-08, 1.63E-08)	
<i>4: Model 1 + Ethnicity</i>	-0.084 (-0.129, -0.038)	<0.001	0.003(-0.003, 0.009)	0.37	8.55E-10 (8.55E-10, 8.55E-10)	2.08E-09 (2.08E-09, 2.08E-09)	
<i>5: Model 4 + education + occupational social class + income</i>	-0.066 (-0.113, -0.019)	0.006	0.003(-0.003, 0.009)	0.36	7.54E-10 (7.54E-10, 7.54E-10)	2.24E-09 (2.24E-09, 2.24E-09)	
<i>6: Model 4 + FMI</i>	-0.041 (-0.083, 0.001)	0.053	0.01 (0.004, 0.016)	0.001	2.89E-10 (2.89E-10, 2.89E-10)	1.59E-10 (1.59E-10, 1.59E-10)	
<i>7: Model 5 + FMI</i>	-0.057 (-0.1, -0.013)	0.01	0.01 (0.004, 0.016)	0.001	9.74E-10 (9.74E-10, 9.74E-10)	5.59E-10 (5.59E-10, 5.59E-10)	
<b>Fat Mass to Fat-Free Mass Ratio</b>							
<i>1: Age</i>	-0.008 (-0.01, -0.005)	<0.001	-0.001 (-0.001, 0.00)	<0.001	0.11 (0.10, 0.11)	0.011 (0.011, 0.012)	
<i>4: Model 1 + Ethnicity</i>	-0.007 (-0.01, -0.004)	<0.001	-0.001 (-0.001, 0.00)	<0.001	0.11 (0.10, 0.11)	0.011 (0.011, 0.012)	
<i>5: Model 4 + education + occupational social class + income</i>	-0.004 (-0.007, -0.001)	0.008	-0.001 (-0.001, 0.00)	<0.001	0.10 (0.10, 0.11)	0.011 (0.011, 0.012)	

**Table 6-6 Footnotes:** Coefficients for difference in body composition at age 7 show the change in body composition for one unit increase in index of multiple deprivation (IMD) at age 7. The coefficients for IMD x Age Interaction show the change in body composition per one unit increase in IMD group per one year increase in age. Abbreviations: FMI – Fat Mass Index; 95% CI – 95% Confidence Intervals.

**Table 6-7.** Socioeconomic Inequalities in Fat Mass Index (FMI), Fat-Free Mass Index (FFMI) and Fat Mass to Fat-Free Mass (FM:FFM) Ratio at age 7 according to Income in Boys and Girls, and Changes in Inequalities across Childhood and Adolescence.

<i>Model</i>	<i>Difference in body composition at age 7 by Income (1: lowest income fifth, to 5: highest income fifth)</i>		<i>Income x Age (Years) Interaction</i>		<i>Random Effects: Intercept</i>	<i>Random Effects: Slope</i>
	Coefficient (95% CI)	p value	Coefficient (95% CI)	p value	SD (95% CI)	SD (95% CI)
<b>BOYS</b>						
<b>Fat Mass Index</b>						
<i>1: Age</i>	-0.037 (-0.056, -0.017)	<0.001	-0.012 (-0.017, -0.007)	<0.001	1.05E-09 (1.05E-09, 1.05E-09)	0.23 (0.23, 0.24)
<i>4: Model 1 + Ethnicity</i>	-0.034 (-0.054, -0.014)	0.001	-0.012 (-0.017, -0.007)	<0.001	1.41E-09 (1.41E-09, 1.41E-09)	0.23 (0.23, 0.24)
<b>Fat-free Mass Index</b>						
<i>1: Age</i>	-0.082 (-0.11, -0.051)	<0.001	0.009 (0.003, 0.014)	0.001	6.69E-07 (6.69E-07, 6.69E-07)	0.13 (0.12, 0.13)
<i>4: Model 1 + Ethnicity</i>	-0.086 (-0.116, -0.055)	<0.001	0.009 (0.003, 0.014)	0.002	1.32E-06 (1.32E-06, 1.32E-06)	0.13 (0.12, 0.13)
<i>6: Model 4 + FMI</i>	-0.076 (-0.099, -0.053)	<0.001	0.022 (0.018, 0.026)	<0.001	2.33E-09 (2.33E-09, 2.33E-09)	0.12 (0.11, 0.13)
<b>Fat Mass to Fat-free Mass Ratio</b>						
<i>1: Age</i>	-0.003 (-0.005, -0.001)	0.003	-0.001 (-0.001, -0.001)	<0.001	0.096 (0.094, 0.099)	0.009 (0.008, 0.009)
<i>4: Model 1 + Ethnicity</i>	-0.003 (-0.005, 0.00)	0.02	-0.001 (-0.001, -0.001)	<0.001	0.096 (0.093, 0.098)	0.009 (0.008, 0.009)
<b>GIRLS</b>						
<b>Fat Mass Index</b>						
<i>1: Age</i>	-0.048 (-0.067, -0.029)	<0.001	-0.014 (-0.018, -0.01)	<0.001	0.58 (0.55, 0.62)	0.16 (0.15, 0.16)
<i>4: Model 1 + Ethnicity</i>	-0.045 (-0.065, -0.026)	<0.001	-0.014 (-0.017, -0.01)	<0.001	0.58 (0.55, 0.61)	0.16 (0.15, 0.16)
<b>Fat-free Mass Index</b>						
<i>1: Age</i>	-0.072 (-0.11, -0.031)	<0.001	0.005 (-0.001, 0.012)	0.13	6.30E-09 (6.30E-09, 6.30E-09)	2.47E-08 (2.47E-08, 2.47E-08)
<i>4: Model 1 + Ethnicity</i>	-0.073 (-0.11, -0.032)	<0.001	0.005 (-0.002, 0.011)	0.14	9.84E-10 (9.84E-10, 9.84E-10)	2.49E-09 (2.49E-09, 2.49E-09)
<i>6: Model 4 + FMI</i>	-0.043 (-0.08, -0.006)	0.023	0.015 (0.009, 0.021)	<0.001	3.02E-10 (3.02E-10, 3.02E-10)	1.12E-10 (1.12E-10, 1.12E-10)
<b>Fat Mass to Fat-free Mass Ratio</b>						
<i>1: Age</i>	-0.006 (-0.008, -0.004)	<0.001	-0.001 (-0.001, -0.001)	<0.001	0.11 (0.10, 0.11)	0.011 (0.011, 0.011)
<i>4: Model 1 + Ethnicity</i>	-0.005 (-0.008, -0.003)	<0.001	-0.001 (-0.001, -0.001)	<0.001	0.10 (0.10, 0.11)	0.011 (0.011, 0.011)

**Table 6-7 Footnotes:** Coefficients for difference in body composition at age 7 show the change in body composition for one unit increase in income fifths at age 7. The coefficients for Income x Age Interaction show the change in body composition per one unit increase in income fifths per one year increase in age. Abbreviations: FMI – Fat Mass Index; 95% CI – 95% Confidence Intervals.



### 6.3.4 IMD Adjusted for Family-Level SEP

After adjusting for family-level SEP (model 5), inequalities in FMI and FM:FFM ratio at age 7 were considerably reduced compared to the model without this adjustment (model 4) (Table 6-4). However, the rate of change stayed the same, such that by age 17 years FMI was 0.51 kg/m<sup>B</sup> (95% CI: 0.37 to 0.64, p<0.001) and FM:FFM ratio was 0.034 (95% CI: 0.023 to 0.044, p<0.001) higher in the most deprived compared to the most advantaged IMD group.

When looking at boys and girls separately, there was evidence of inequalities at age 7 in girls but not boys for both FMI and FM:FFM ratio (Table 6-6, Figure 6-2d and Figure 6-5d). By age 17, the difference in FMI between the most and least advantaged groups in boys (0.57 kg/m<sup>B</sup>, 95% CI: 0.38 to 0.76, p<0.001) and girls (0.54 kg/m<sup>B</sup>, 95% CI: 0.39 to 0.70, p<0.001) was similar, representing a four-times increase in the inequality in girls and 14-times increase in boys.

Inequalities in FFMI at age 7, when adjusted for family-level SEP, were slightly smaller compared to model 4, although those in the most deprived areas still had higher FFMI. Because the rate of change remained similar to model 4, with slightly faster increases in FFMI among the most advantaged areas, there was little evidence of a difference in FFMI (-0.09 kg/m<sup>B</sup>, 95% CI: -0.235 to 0.054, p=0.22) between IMD groups by age 17.

When FMI was added to the model along with family-level SEP (model 7), the rate of change was larger than model 5 (adjusted for family-level SEP and ethnicity), so that there were faster increases among those in the most advantaged areas. Association between IMD and FFMI reversed by age 17 so that there was a 0.26 kg/m<sup>B</sup> (95% CI 0.14 to 0.38, p<0.001) higher FFMI in the most compared to least advantaged IMD group. Slightly larger inequalities were observed in boys (0.38 kg/m<sup>B</sup>, 95% CI: 0.23 to 0.52, p<0.001) compared to girls at age 17 (0.18 kg/m<sup>B</sup>, 95% CI: -0.007 to 0.36, p=0.06. Model 7, Figure 6-4e-f).

## 6.4 Discussion

### 6.4.1 Summary

Three hypotheses were tested in this chapter. A summary of the findings in relation to these hypotheses is provided below.

**Hypothesis 4:** *Disadvantaged SEP is related to higher FM, lower FFM and a higher FM:FFM ratio among children and adolescents in the UK at age 7, and these inequalities widen from age 7 through adolescence to age 17.*

In line with hypothesis 4, those aged 7 through 17 years growing up in disadvantaged circumstances had higher FMI and FM:FFM ratio compared with their more advantaged counterparts when SEP was measured by both income and the IMD. These differences increased with age for FMI and FM:FFM ratio as hypothesised.

Contrary to the hypothesis, FFMI was higher for children living in disadvantaged circumstances although the differences declined with age. However, with adjustment for FMI, associations for FFMI reversed in adolescence so they were in the direction hypothesised, with those in the most advantaged position having higher FFMI conditional on FMI.

Without adjustment for FMI, if the observed rates of change continue into adulthood, inequalities in FFMI would eventually reverse given the faster increase in FFMI with age among those from the most advantaged circumstances. Continued follow up of the cohort is needed to confirm this.

**Hypothesis 5:** *Inequalities in FM, FFM and FM:FFM are greater in females compared to males.*

Consistent with hypothesis 5, there was greater evidence of inequalities in FMI in girls when measured by the IMD at age 7, with the inequality over three and half times greater in girls compared to boys. However, inequalities widened at a faster rate in boys so that by age 17 there had been roughly a ten-fold increase in the difference in FMI between the most advantaged and most deprived IMD groups in boys. For girls there was a three-times increase, resulting in similar overall differences in the inequality between boys and girls by age 17.

For FFMI and FM:FFM ratio, and all body composition measures by income fifths, there was little difference in the inequality and rate of change in the inequality between boys and girls.

**Hypothesis 6:** *Area-level inequalities in body composition trajectories are observed, and are not explained by inequalities in family-level SEP.*

The results of this study were in line with hypothesis 6 in girls for FMI and FM:FFM ratio, with inequalities by area-level SEP apparent at age 7 after adjusting for family-level SEP. For girls, these inequalities continued to widen to age 17, whilst for boys inequalities emerged around age 11 when adjusted for family-level SEP and widened to age 17.

For FFMI, inequalities at age 7 were apparent for boys and girls when adjusted for family-SEP, but in the opposite direction hypothesised with those in the most advantaged IMD group having the lowest FFMI. Inequalities in FFMI were no longer apparent at age 17, as they declined with age due to the faster increase in FFMI among those living in the most advantaged areas. However, when adjustment was made for FMI in addition to family-SEP, inequalities at age 17 were apparent with those in the most advantaged IMD group having greater FFMI.

#### 6.4.2 Comparison with Previous Literature

Consistent with the findings in this chapter, previous research using the MCS at ages 7 and 11 identified inequalities in FM% that increased between the two ages [230]. The current analysis demonstrates, using FMI, that this increase continues through to age 17. Research conducted in ALSPAC demonstrated a widening of inequalities in FM for girls from age 9 to 15, but unlike the findings in this chapter, not for boys [256]. In a study among Swedish adolescents followed up at 15, 17 and 20.5, fathers' education was found to be negatively related with fat percentage and FM at each age, whilst positively associated with LM% and LM [260]. The results from the present analysis are similar to research that has looked at inequalities in trajectories of BMI in the UK that demonstrate emerging inequalities in childhood and widening with age [375], including research conducted in the MCS [118].

Whilst there were greater inequalities in FM among girls in childhood, boys from more disadvantaged circumstances exhibited faster rises in FM such that inequalities are similar by adolescence. This differs to previous research conducted in ALSPAC where widening of inequalities for FM was observed for girls but not boys [256] and greater inequalities existed in female BMI based on maternal education levels [375]. The results also differ to those conducted into older generations where females typically demonstrate greater evidence of social inequalities in obesity [112] and body composition [376].

This research demonstrates associations of area-level SEP with body composition trajectories independent of family-level SEP. Unlike these findings, previous research conducted in Australia found inequalities in BMI trajectories, measured using area-level SEP, to be fully attenuated once accounting for family SEP [377]. Research using area-level measures of SEP in the UK have found mixed results. A study of approximately 400 children from greater London and Cambridgeshire found no association was observed between area-level SEP and FMI [241]. Among 2,300 children from East London, Hertfordshire and Cambridgeshire, greater advantage was associated with lower FM% [269]. Similar to these findings, research using the MCS has previously demonstrated social gradients in FM% among the most and least affluent neighbourhoods [378]. Of the research reviewed no previous study has looked at how area-level deprivation relates to changes in body composition over time, considering FFMI and FM:FFM ratio as well as FMI, whilst accounting for individual SEP.

### 6.4.3 Interpretation of Findings

#### *6.4.3.1 Fat Mass Index and Fat-Free Mass Index*

The results for FMI were in line with those observed for fat measures in the systematic review, with those in the most disadvantaged positions typically having higher FMI than their more advantaged counterparts. As such, the interpretation for findings for FMI are likely similar to those discussed in Chapter 5 for inequalities in HICs among children (section 5.4.3.1, pg. 111 and section 5.4.3.5, pg. 114).

The results for FFMI differ slightly from the systematic review conducted in Chapter 5, where there was some evidence that advantaged SEP was associated with greater fat-free measures in children, and little evidence of associations in the opposite direction [379]. The systematic review found studies had predominantly used raw or percentage measures of FFM, which do not account for the contribution of height [71] and as those from more advantaged SEP are generally taller, this may explain the contrary findings. Among previous studies that used indexed measures of FFM in children, fewer found evidence of association with SEP than those that used raw or percentage measures. The results from this chapter provides support for the observation from the systematic review, that inequalities in height, which have reduced over time in the UK [28], may be important in understanding inequalities in FFM. In the current analysis the correlation with height was addressed in the measures used by indexing FFM using the Benn parameter, and negative associations were observed unless adjusted for FMI. However, inequalities in height in the MCS are small compared to older cohorts [28], so there may also be other reasons for the difference in findings.

There may be differences in inequalities by cohort that explain the results for FFMI, as levels of obesity in the MCS are greater compared to previous generations, and inequalities in BMI are larger [28]. Greater disadvantage was associated with higher FFMI, particularly in childhood, mirroring the direction of association observed for FMI. This may be a result of the adaptive increases in muscle as a result of the higher FMI [354] in disadvantaged children. However, the difference narrowed with age as FFMI increased faster in those from more advantaged SEP, resulting in increasing inequalities in the FM:FFM ratio. This could be due to increasing inequalities in health-related behaviours that build lean mass, such as physical activity. If the more rapid increase in FMI but slower increase in FFMI observed in the more disadvantaged groups in MCS continues past adolescence this will result in continuing widening of the FM:FFM ratio. Given that FFM peaks in mid-adulthood [365], it will be more difficult to intervene to address inequalities in FFM after the peak is reached, and to improve FM:FFM ratio. Inequalities in FFMI also add to the likely impact of body composition on inequalities in health, given the importance of FFM for later cardio-metabolic disease and physical capability in older age [89, 380].

#### *6.4.3.2 Sex Differences*

As the MCS is one of the most recent nationally representative cohorts in the UK, the observation that boys in disadvantaged circumstances have a faster increase in FM compared to girls, may represent a secular change in inequalities in adiposity. The inequality in males may be increasing to match that

observed in females, resulting in a narrowing of the gap between males and females in more recent generations. As previously mentioned in Chapter 2 and 5, it is theorised that inequalities for obesity are greater in females compared to males because of societal pressures for thinness placed on women and girls, that are easier to maintain in positions of advantage [115]. It is possible that as the obesity epidemic has progressed, the same societal pressures also face young males, resulting in a narrowing of the gap. Continued follow up of childhood cohorts is needed to monitor if the observed faster widening of inequalities among males continues into adulthood.

#### *6.4.3.3 Area-level Socioeconomic Position*

Area-level measures of SEP may be related to levels of obesity due to their correlation with aspects of the obesogenic environment. The results somewhat support this by indicating that the IMD captures elements of the environment beyond family SEP in adolescence for both boys and girls, but less so in early childhood for boys. The aspects of the environment that area-level SEP capture is unclear. It is possible that the cultural and social environment are represented by area-level measures, and that through this social norms, networks and peer behaviours influence diet and physical activity in adolescence [148]. Additionally, in the UK more disadvantaged areas, as indicated by IMD, have greater fast-food density [160] highlighting a link with the built environment. Previous conceptual models have highlighted how area-level SEP relates to obesity through both the built and social environment [148], and it is likely that both factors are relevant for understanding the results.

#### *6.4.3.4 Life course effects*

The results demonstrate the long-term impacts of disadvantage measured at 9 months old on body composition in childhood and adolescence. Disadvantage at 9 months is likely to reflect disadvantage over subsequent years, and disadvantage is related to development of health behaviours associated with obesity. For example, family-level SEP in early life influences the development of nutrition and physical activity patterns and these behaviours track across childhood [381]. There may also be clustered risks associated with disadvantage in early life, such as social vulnerabilities [382], and social vulnerabilities at birth have previously been shown to be related to obesity from age six in a Spanish Cohort [383]. The literature review in Chapter 2 highlighted the potential role of emotional and behavioural problems as a mediator in associations, which is explored in Chapter 8, and this may be another factor that clusters with disadvantage and social vulnerabilities.

The systematic review demonstrated that early life SEP is related to adult FM, but with limited and less consistent results for FFM, and no research conducted for FM:FFM ratio. Further follow up of childhood cohorts will be needed to investigate whether these early life effects persist as with these previous cohorts, and if inequalities in adult body composition are a result of accumulation of disadvantage in childhood and adulthood. However, SEP does change over the life course, and for area-level measures this can be both because people move home and as areas can change around people over time. Hence in further consideration of the life course impacts of SEP, research into the effects of social mobility, individually and according to area, is also required.

Understanding inequalities in trajectories of body composition is important, especially in adolescence as rates of change over this period has been related to later life health. Research in ALSPAC demonstrated that faster rate of development of FFM in adolescent boys has been shown to be protective of cardiovascular disease risk, whilst gains in FM in both girls and boys from age 10 to 25 increased cardiovascular disease risk [86]. Additionally, levels of FFM in older age are dependent on peak FFM and age-related decline, with peak FFM being reached in earlier adulthood, unlike FM which increases into older age [97, 365]. It has been demonstrated that body composition tracks across the life course [239, 384], which may result in the maintenance of inequalities that develop in adolescence.

#### 6.4.4 Implications

If widening of inequalities persists into adulthood or if inequalities are maintained, this will result in large differences in body composition, with subsequent consequences for health inequalities. The findings of this chapter support the need for effective policies to tackle inequalities in obesity in childhood that consider differences in the environment. There is also a need to consider promotion of physical activity in disadvantaged settings from an early age to improve quality of FFM, especially muscle, and therefore the FM:FFM ratio. Steps taken earlier will avert the need for public health interventions at later ages, where such measures may be less effective at reducing inequalities than action earlier in the life course.

#### 6.4.5 Strengths and Limitations

There are several strengths and limitations relevant to this analysis. Multilevel growth curve models were selected to measure the change in body composition with age. This is a better approach than alternative techniques such as repeated measures analysis of variance, as multilevel models have less strict assumptions, and are better able to handle hierarchical structures and a degree of missing data in the outcome [367]. Multilevel growth curve models allow the data to be fitted using maximum likelihood estimation, meaning those with data at a single time point still contributed to the estimates based on MAR assumptions. This would not have been possible with other approaches that require complete data on all time points. In this analysis missing values were imputed for predictor variables and covariates therefore reducing bias in the estimates.

This analysis models income and IMD at 9 months (or at 3 years for late joiners), and therefore takes advantage of the longitudinal nature of the MCS by investigating how early life circumstance relate to body composition trajectories. However, this means that the impact of changes in SEP over the life course are not considered. It is likely that SEP in later childhood and adolescence also influence trajectories in body composition. The current analysis does not allow separation of the effects of early life SEP from SEP later in the life course, and therefore ignores the possible role of social mobility.

Body composition was only collected at four time points meaning it was not possible to test higher order terms, and therefore linear growth with age was assumed. Increases in BMI are typically linear after the adiposity rebound at age of 7 up until later adolescence [385]. In growth curves developed for body composition, FM does not show a curvilinear association with age from 5-20 years old, and

whilst lean mass (LM) shows tendency to be curvilinear, the curvilinear association is reduced when lean mass is indexed to height [386]. It is therefore possible that the effect estimates may be biased if the assumption of linearity does not hold.

The IMD is used to capture social and economic elements of the obesogenic environment, an area that has previously been understudied. By looking at the areas in which people live as well as their individual circumstances, the study starts to untangle the role of neighbourhood environments. However, the IMD is a broad measure of the social and economic environment that does not directly measure or distinguish aspects of the physical environment, although it has been shown to be correlated with fast-food density [160]. These aspects of the obesogenic environment may be better captured by other measures of the physical environment such as fast-food density and green spaces. These measures are available in the MCS, although fast-food density was not collected at sweep 1 or 2. Further, this chapter only developed hypothesis pertaining to role of the social and economic environment in infancy, and how area-level measures of SEP differ in their effect to family-level measures. The role of the physical environment as a moderator of associations is explored in further detail in Chapter 8.

In this analysis, the IMD was combined across countries to maintain sample size and ensure representation of the four countries of the UK in analysis. However, in doing so assumptions were made about the similarity and comparability of deprivation measures across the countries. These assumptions may not hold, as relative ranks were combined, and varying components were weighted differently. However, recent work that has developed adjusted deprivation scores for the IMD across countries found only a small change in rank when applied [207]. It is therefore likely that combining across countries would not have substantially altered estimates although, there may have been some benefit in examining how associations differ between the four countries of the UK. Alternatively, using the income and education components of the IMD, which are comparable across countries, may have been an alternative approach as has been developed in adjusted IMD models [207, 387].

FMI is treated as a confounder in associations with FFMI. This is because of the strong correlation between FM and FFM and evidence that indicates there are adaptive increases in muscle with FM, whilst FM is also associated with SEP. However, it is possible this was an overadjustment in this analysis, for two reasons: Firstly, the analysis uses FFMI instead of LMI that excludes bone, yet it is assumed that it is muscle increasing alongside fat, as opposed to other components of FFM (i.e bone). However, it has been demonstrated that FFM, and not just LM, increases alongside FM in obese children [354, 388] and that in the general adult population increases in FM are associated with accompanying increases in FFM in the same direction [354]. Secondly, as the reason the adjustment is made is because with increasing fat there is increasing muscle, FMI may therefore sit on the pathway from SEP to FFMI and represent a mediator instead of a confounder. Therefore, models with and without adjustment for FMI were presented to understand how the inclusion of FMI as a covariate impacts estimates.

Only a limited number of additional covariates, selected *a priori*, were included in models. Only variables considered to be confounders (ethnicity) or that acted as competing exposures (sex) were included as these are the only ones that should be controlled for when estimating total effects of SEP on body composition [215]. However, it remains possible that there were actual confounders that were not included in models which could have resulted in biased estimates and an overestimation of the effect of IMD. For example, it may be that rural/urban location should have been adjusted for in models that included the IMD as the exposure as patterning of deprivation differs between rural and urban areas in the UK [389]. However, it is unclear whether rural/urban location would confound associations, as urbanicity in and of itself is unlikely to be causal of deprivation in an area. Instead, it may be that it moderates associations with deprivation. A study in France found associations between deprivation and overweight to be moderated by urbanicity, with associations stronger in suburbs and peri-urban areas, whilst weaker or null in small towns and rural areas [390]. Further research could therefore look at the potential moderating role of urbanicity, although there may not be enough granularity in the binary rural/urban variable available in the MCS to detect important moderating effects.

There are a number of other variables which are socially patterned and predictors of adolescent body composition, such as maternal smoking and depression. Prevalence of both maternal smoking and maternal depression are higher among those living in disadvantaged circumstances [128, 391, 392], although they are unlikely to determine SEP. Smoking during pregnancy has been found to be associated with BMI [393] and body composition [394], with foetal programming and changes to endocrine control of body weight a likely explanation for associations [394, 395]. Similarly, maternal depression has been found to be associated with offspring BMI, explained through parenting style [396], influencing mother and child eating behaviours [397], and offspring engagement in physical activity and screen time [398]. It is possible that maternal depression could causally influence family SEP, however, the effect is likely to be small, whilst maternal smoking is unlikely to have any causal relation with family SEP. Therefore, it is probable that both variables sit on the pathway between SEP in infancy and body composition and represent mediators of associations [399], especially as the mechanisms that relate them to body composition are related to early life development and establishment of health behaviours. As the aim of this chapter was to assess the total effect of SEP on body composition, they were not included in analysis.



# Chapter 7 Factor Analysis of the Parent-Reported Strengths and Difficulties Questionnaire in the Millennium Cohort Study

## 7.1 Background

In this thesis internalising and externalising symptoms in early adolescence are hypothesised to be individual factors that are in part a consequence of social circumstances earlier in life, and act as a mediator between SEP and body composition in later adolescence. In this sense, a life course approach is adopted with a chain of risk model that sees emotional and behavioural problems as on the pathway between socioeconomic disadvantage and adverse body composition. By looking at psychological symptoms, this thesis also explores psychosocial pathways from disadvantage to health, relating to a social determinants approach. In addition to treating internalising and externalising symptoms as mediators, it is also hypothesised that they are moderated by environmental characteristics, and therefore the mediating effect differs between individuals experiencing different environmental conditions.

In order to test these hypotheses, it is first necessary to validate the scale used to measure internalising and externalising symptoms, ensuring that items group onto constructs as hypothesised and that the constructs measure what is intended to be measured. As outlined in Chapter 4, in the MCS internalising and externalising symptoms are measured through the Strengths and Difficulties Questionnaire (SDQ), by combining two of the five subscales for each set of symptoms: emotional symptoms and peer problems for internalising symptoms; hyperactivity and conduct problems for externalising symptoms.

It is also necessary to demonstrate that both sets of symptoms are measured in the same way according to the levels of the moderating variables, so that differences between groups reflect the moderating effect as opposed to differences in the way the symptoms are measured. Therefore, it needs to be demonstrated that internalising and externalising symptoms were measured in the same way between those living in high, medium and low areas of deprivation or other environmental characteristics.

The five SDQ subscales were originally developed through theory and further refined through exploratory factor analysis (EFA) [208]. Recent work has used confirmatory factor analysis (CFA) to test the underlying factor structure of the SDQ in different groups and populations, finding mixed

support for the five-factor model [400]. In work that has tested the underlying structure, a number found poor model fit using a five-factor model as hypothesised [401, 402], whilst others report adequate fit indices but low factor loadings (<0.4) [403]. As a result, there has been exploration of alternative factorial structures.

One factorial structure that is supported by theory and empirical work, is a structure with two distinct factors for internalising and externalising symptoms that are measured by their respective sub-scales [400]. This can be achieved through a first-order structure with three-factors, or a second-order structure with two-factors and one first-order factor for the pro-social scale. Both have previously been tested in different populations finding mixed support for both structures [400, 404-407]. In the UK, a structure with two second-order factors for internalising and externalising symptoms was found to be most appropriate for the general population, whilst a five-factor model was more appropriate among children when screening for disorders [400]. A study using the MCS found support for a five-factor model from ages 3 to 7 when conducting CFA, assessed through adequate model fit and factor loadings, internal reliability and external validity, and ability to predict clinical outcomes [408]. However, the study did not investigate the structure at age 11, nor did it test second-order internalising and externalising structures [408]. Additionally, invariance between groups of environmental conditions, which is needed to conduct further sub-group analysis, has not previously been demonstrated in the MCS.

Few researchers report the validity of psychometric scales when they use them, despite recommendations to provide evidence on the internal structure of the scales [409]. This chapter therefore aims to investigate the appropriateness of using the SDQ as a measure of internalising and externalising symptoms in the MCS, through conducting an EFA followed by CFA testing competing factorial structures. Additionally, this chapter aims to demonstrate the appropriateness of using internalising and externalising latent traits in multi-group analysis, by demonstrating the similarity by which constructs are measured between groups. The following two hypotheses are therefore tested:

***Hypothesis 7:*** *The Strength and Difficulties Questionnaire captures internalising and externalising latent traits in a UK population of adolescents and the latent factors are appropriate to use as mediators in further analysis.*

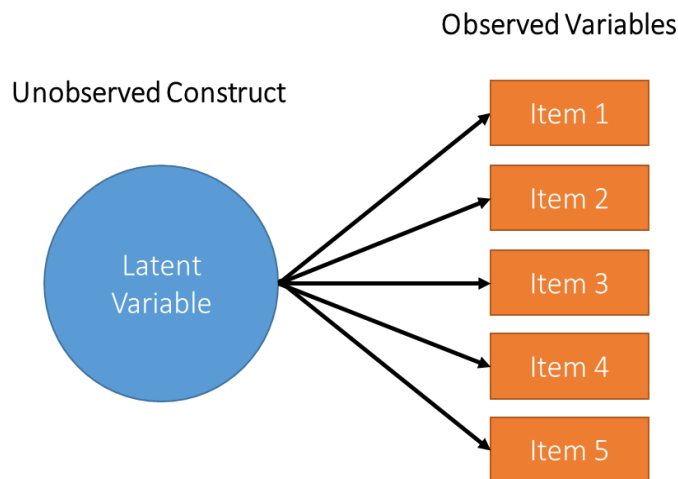
***Hypothesis 8:*** *The latent constructs of internalising and externalising symptoms are measured in the same way in boys and girls, and across different environmental conditions (i.e high and low deprivation, high and low green space, domestic garden and fast-food outlet density).*

## 7.2 Methods

### 7.2.1 Factor Analysis

Factor analysis is a method of data reduction. Unlike other approaches to data reduction, such as with principal component analysis (PCA) that creates a new “component”, factor analysis assumes the existence of a “latent factor”. A latent factor is an unobserved construct, that can be measured by observed variables. As such, the paths in a factor analysis go from the latent factor to the observed variables, as is shown in Figure 7-1.

**Figure 7-1.** Diagram of Latent Variable Measured by Observed Variables



**Figure 7-1 Footnote:** Diagram showing relationship between observed variables and a latent variable in factor analysis. Measured variables (items) are considered the outcome in factor analysis, as the latent factor is an unobserved construct that is measured by the items, and therefore the observed variables are a consequence of the unobserved construct. Hence, arrows in factors analysis are directed at the measured items.

Factor analysis is often applied in psychology where a set of questions is used to assess a psychological construct, such as internalising and externalising symptoms, that cannot be directly measured. Instead, the psychological construct is assessed through questionnaires that are thought to “tap in” to the unobserved constructs. Factor analysis is favoured above adopting sum scores, which create a mean score across item responses and assume items are measured in a similar way. Sum scores are criticised as they make two key assumptions that are often violated [410]:

1. The question items are “pure” and contain no error.
2. All items are assumed to be equally important in measuring the latent factor.

Factor analysis addresses these assumptions: firstly, factor analysis “partials out” the error; Secondly, factor analysis provides weights, also known as factor loadings, for each item. This allows for the determination of which items are stronger manifestations of the underlying trait. Factor loadings are correlation coefficients or standardised regression coefficients that express the degree of relatedness of the item to the factor, with a higher factor loading indicating that the item is well measured by the factor [411]. Factor analysis uses correlation matrices, usually Pearson’s correlations, to identify

factor loadings. However, when latent factors are psychological constructs, the items measuring them often use a Likert Scale and are therefore ordinal. When this is the case, polychoric correlations should be used to account for the ordinal nature of the measures [412]. Polychoric correlations are based on maximum likelihood and assume that there is an underlying continuum on which the ordinal variables are based, meaning the items in factor analysis do not need to be normally distributed, nor do they need to be continuous.

There are two main types of factor analysis: EFA and CFA. It is recommended when conducting factor analysis, that the dataset should randomly be split in half, with one half used for development (EFA) and the other half for testing (CFA). This allows cross-validation by testing a model in a different dataset to the one in which it was constructed, which therefore avoid problems of overfitting the data and overestimating model fit [413].

EFA is data driven, and used for exploring patterns and potential factor structures, and explores the way items load on to non-hypothesised factor structures [414]. Eigenvalues are used to select how many factors should be adopted during EFA, with factor models that have an eigenvalue greater than one often being selected [413]. Such a value is used as it indicates that the factor explains more variance than a single observed item. Rotated factor loadings are then used to explore how different items load onto the non-hypothesised structures. Traditionally, in EFA a rotated factor loading greater than 0.4 is used to indicate a strong loading onto a factor [411], although other thresholds may be taken.

Hypotheses related to the number of factors, and the factorial structures, are then tested using CFA. As CFA is used to test hypotheses it requires strong theory underlying the model, as well as often being preceded by EFA that supports the hypothesised factorial structure [414]. CFA can also be data driven as well as hypothesis driven, in cases where there is a poor fit in initial CFA models [413].

It is common in CFA to allow correlations between errors as it increases the proportion of variance explained, therefore improving model fit without changing the conclusion of underlying hypothesised structure [400]. However, not all errors should be allowed to correlate because of issues raised by doing so [415]. For example, the inclusion of an excessive number of correlations between items violates the assumption in CFA of uncorrelated errors, and therefore may result in a mis-specified model not being identified as misfitting. Additionally, the inclusion of all correlated errors will result in the model being fully saturated.

In order to assess the fit of factorial structures during EFA and CFA, different fit indices are used, these being the chi-squared ( $\chi^2$ ) statistic, Comparative Fit Index (CFI), Tucker-Lewis Index (TLI), Root Mean Square Error of Approximation (RMSEA) and Standardized Root Mean Squared Residual (SRMR). Thresholds for good fit are TLI/CFI >0.95; RMSEA <0.06; SRMR <0.08 and those considered acceptable are TLI/CFI >0.9; RMSEA <0.08 [416, 417]. For the chi-squared statistic, a good fit is indicated by a non-significant value ( $P > 0.05$ ). However, when there is a large sample size it is highly unlikely a model would return a non-significant chi-squared value. Therefore, models with lower chi-squared values are sought.

### 7.2.1.1 Measurement Invariance

Measurement invariance is important to demonstrate that the scale used to measure latent factors is interpreted the same way between respondents. Configural invariance is the simplest form of invariance and indicates that the items and factors arrange in the same way across groups [50]. Metric invariance indicates that a set of items are measuring the factor in the same way, by fixing factor loadings equal across the groups [418]. Scalar invariance means that the items are measuring the factors in the same way, but also that the latent factor captures the mean differences in the shared variance of the items, by constraining either the intercepts or thresholds to be equal across groups as well as the factor loadings [418].

It is standard to test invariance using the chi-squared difference test, despite the fact that the chi-squared difference test is liable to the same constraints as using the absolute chi-squared test to assess model fit [419]. Because the chi-squared test is sensitive to both large sample and non-normality assumptions being violated, it is common for nested models to be rejected because of significant chi-squared difference tests, even when invariance is achieved. Therefore, as proposed by Chen (2007), a better alternative is to use CFI/TLI, RMSEA and SRMR difference tests, where a difference between nested models greater than 0.010 for CFI/TLI, 0.015 for RMSEA and 0.030 for SRMR indicates non-invariance [419]. This is considered a “bottom-up” approach to measurement invariance and validates the functioning of the scale.

For second-order factors, it is necessary to adopt a “top-down” approach whereby scalar invariance is tested by manually constraining the intercepts and variances to be equal in the second-order models and fixing the intercepts of the first-order factors to zero [420]. Strong factorial invariance is demonstrated if the model has a good fit, based on the CFI, TLI, RMSEA and SRMR fit indices. A top-down approach demonstrates the strictest level of invariance, and therefore shows it is appropriate to proceed with use of the factors in further models, such as regression analysis or further SEMs.

### 7.2.1.2 Ordinal Alpha

Ordinal alpha is a measure of internal scale validity and reliability that should be assessed after the factor analysis, with its purpose being to measure how similar a set of related items are [421]. Ordinal alpha is used instead of Cronbach’s Alpha when the items in the scale are ordinal. To calculate ordinal alpha, the average correlation of a set of items is estimated using polychoric correlations ( $r$ ). This along with the number of items in the scale ( $k$ ) is used to estimate the ordinal alpha, using the equation below [422]:

$$\text{Ordinal alpha} = (k*r)/(1+(k-1)*r)$$

Where:

$r$  = average correlation

$k$  = number of items in scale

An ordinal alpha greater than 0.7 is deemed to demonstrate good internal validity. However, caution is required in the interpretation of the ordinal alpha, as it should be considered a hypothetical tool and its use as a reliability measure has come under question [423].

### 7.2.1.3 Scale Validation

Scale validation, which assesses that the scale measures what it is intended to measure, can be demonstrated in a number of ways. Internal convergent validity tests the internal strength of the factor, and tests that the items measuring the same construct all load strongly onto the same single factor. External discriminant validity assesses whether different constructs measured in similar ways, are not strongly correlated and therefore are distinct factors.

Both external discriminant validity and internal convergent validity can be assessed using average variance extracted (AVE) scores, which represent the average variance explained by the factor in the items that it is measured by. AVE scores are simply the average  $R^2$  score, and thus are calculated using the following equation:

$$AVE = (R^2_1 + R^2_2 + R^2_3 \dots + R^2_n) / N$$

$$N = \text{number of items}$$

In order for a factor to demonstrate internal convergent validity, AVE scores should be greater than 0.5 [424]. For external discriminant validity to be demonstrated, the AVE score of a factor, which represents its internal strength, should be greater than its squared correlation with other factors, or in other words the strength of external associations [424].

Scale validity can also be assessed through its predictive properties. This is known as criterion validity and is the ability of factors to predict outcomes that are known to be related to the construct. This is done using SEM to obtain a regression coefficient for the association between the factors and relevant outcomes.

## 7.2.2 Analytic Approach

All data cleaning and descriptive analysis were conducted in STATA 15.1 [370], whilst EFA and CFA were conducted in Mplus Version 8.5 [425]. All analyses were weighted using non-response and sampling weights from the age 11 sweep for the whole UK sample, stratification characteristics and the cluster variable. To conduct EFA and CFA the data set was split randomly in half, with one half for developing models and the other for testing models. EFA was conducted in the development half of the dataset and eigenvalues, model fit indices and geomin rotated factor loadings were obtained for up to an eight-factor structure. Rotated factor loadings of  $> 0.4$  were deemed good, whilst a lower threshold of  $> 0.35$  was used to identify weaker but still potentially important loadings. CFA was conducted in the testing half to evaluate and compare different factorial structures.

In the CFA, six different models were compared: Model 1) a first-order model with five-factors; Model 2) a first-order five-factor model with correlated errors; Model 3) a first-order three-factor model; Model 4) a three-factor model with correlated errors; Model 5) a second-order model with five first-order

factors and two second-order factors (internalising and externalising symptoms); and finally, Model 6) A second-order model with correlated errors. The competing models without correlated errors (models 1,3,5) are shown in Figure 7-2. The same models were tested again in a sensitivity analysis (Appendix Models 7-12), but with removal of cross-loadings highlighted by the EFA.

These factorial structures were selected based on previous theory and the original model that indicated the existence of five constructs measured by the SDQ: emotional problems, peer problems, behavioural problems, hyperactivity and pro-social behaviour [208]. A three-factor (models 3 and 4), and second-order factor model (models 5 and 6) were considered alongside the five-factor model (models 1 and 2), as previous theory supports the existence of internalising and externalising symptoms [400]. Inclusion of both a three-factor and second-order model meant it was possible to test if these constructs existed independently of the first-order factors, or if they were better measured by the first-order factors. These were similar to the competing models tested in Goodman et al.'s (2010) investigation of the SDQ in the British Child and Adolescent Mental Health Surveys [400].

The errors that were allowed to correlate were identified by the modindices function in Mplus, and by the similarity of construct measured supported by harmonisation work on mental health items that identified questions measuring similar constructs [426]. Correlations were only allowed between unique variances of items that were measuring the same factor. The same correlated errors were selected for models 2, 4 and 6. The number of correlated errors was limited to prevent the model from becoming saturated.

As observed variables were ordinal, the CFA was estimated using the Weighted Least Squares, Mean and Variance Adjusted (WLSMV) estimator. As the default in Mplus when the WLSMV is the estimator for CFA, a polychoric correlation matrix with probit regression is adopted. In CFA, factor loadings greater than 0.5 were deemed acceptable and >0.7 deemed strong loadings [427].

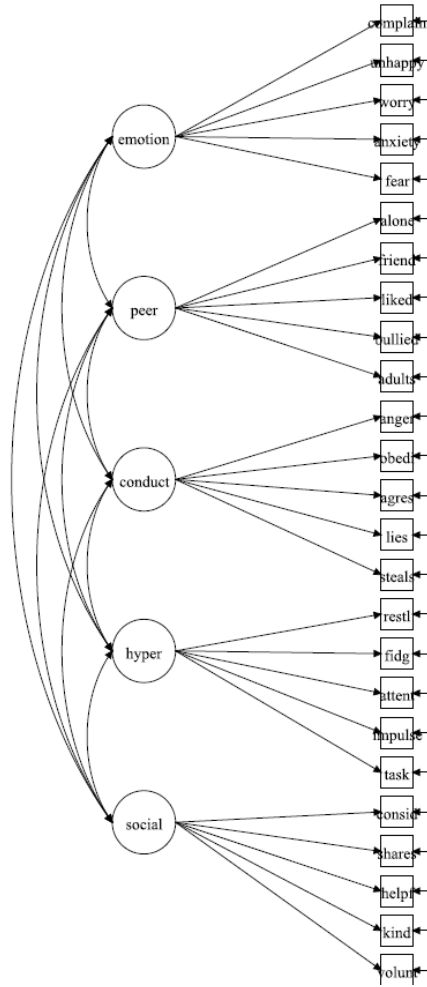
Ordinal alpha was calculated for each of the factors to demonstrate internal consistency. AVE scores were calculated for each factor in the model to assess internal convergent validity and compared to their respective squared correlations to assess external discriminant validity.

#### *7.2.2.1 Measurement Invariance*

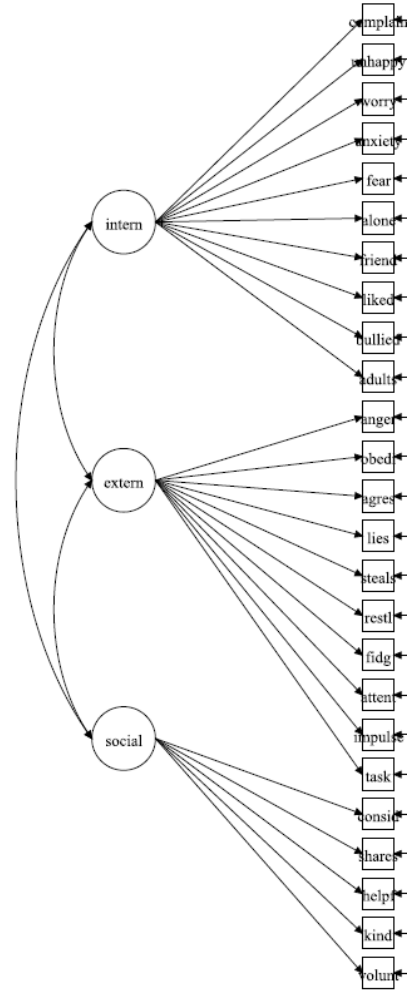
Configural, metric and scalar invariance was tested between boys and girls and between those living in areas with different levels (i.e high, medium and low deprivation) of environmental characteristics (IMD, green spaces, domestic gardens and fast-food Density). Invariance was tested between the metric and configural models, and the scalar and metric models, using the inbuilt function in Mplus for first-order models. Differences between nested models less than 0.010 for CFI/TLI, 0.015 for RMSEA and 0.030 for SRMR were sought to demonstrate invariance. For the second-order factor, a “top down” approach was adopted where scalar invariance was achieved if good fit was demonstrated when intercepts and variances were constrained to be equal in the second-order model, and the intercepts of the first-order factors were fixed to zero.

**Figure 7-2** Competing Factorial Structures.

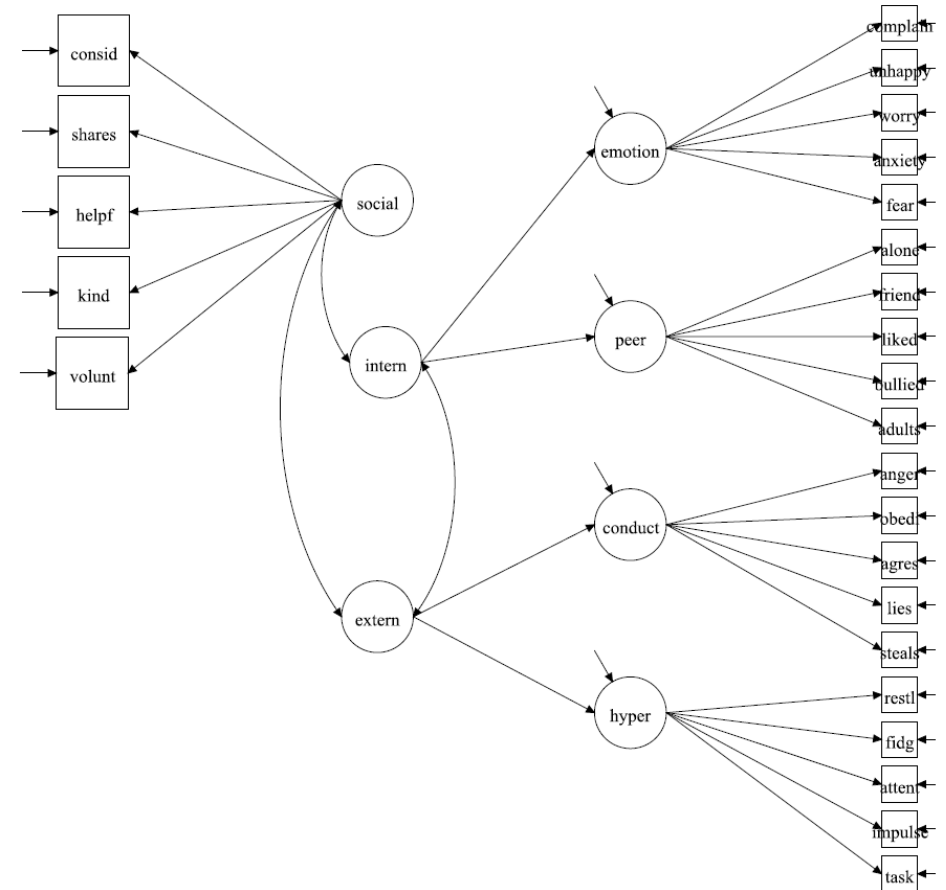
**a) Model 1 – First-order five-factor model**



**b) Model 3 – First-order three-factor model**



**c) Model 5 – Second-order two-factor model, with one first order factor**



**Figure 7-2. Footnote.** Figure showing competing models tested. Image a) shows model 1, a first-order model with five-factors for emotional symptoms, conduct problems, hyperactivity, peer relationship problems and pro-social behaviour. Image b) shows model 3, a first-order model with three-factors for internalising and externalising symptoms and pro-social behaviour. Image c) shows model 5, a second-order model with two-factors from internalising and externalising symptoms, indicated by the first-order factors of emotional symptoms and peer problems for internalising symptoms, and conduct problems and hyperactivity for externalising symptoms, with a separate first-order factor for pro-social behaviour. Models 2, 4 and 6 are the same as 1,3 and 5, but with correlated errors between some items.



### 7.2.2.2 Predictive Validity

Predictive validity was tested using depression diagnosis at age 17, and autism/Asperger's syndrome and ADHD diagnosis at age 14. As outlined in Chapter 2, internalising symptoms are often considered a precursor to depression, and externalising symptoms are associated with conditions such as ADHD.

At age 17, cohort members were asked if they had ever received a diagnosis of depression and the age at which they were diagnosed. These two questions were combined retaining those who received a diagnosis aged 13 and older, and depression diagnosis was categorised as either "yes" or "no". At age 14, parents were asked if the cohort member has a diagnosis of ADHD and autism/Asperger's, and the respondent either answered "yes" or "no".

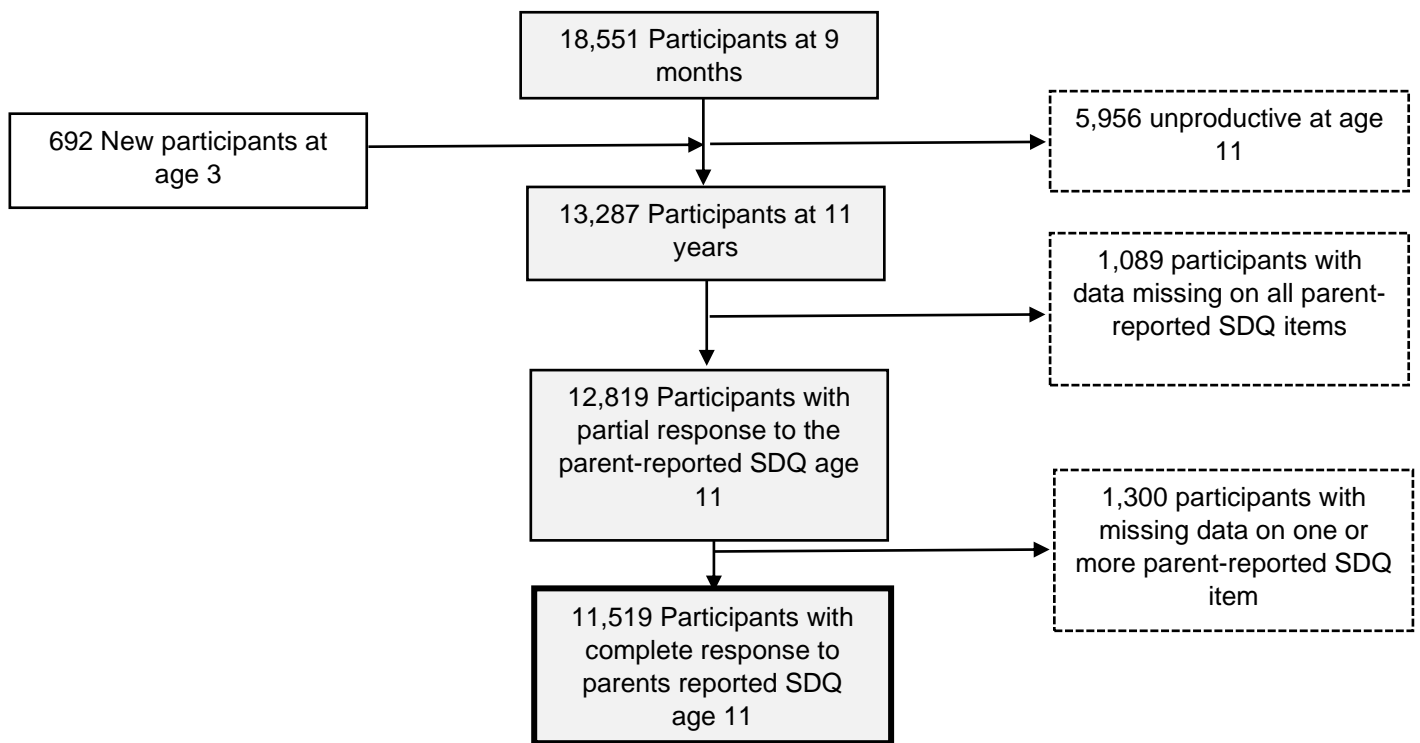
Probit regression was applied to assess associations between the factors and each of the diagnostic criteria, with associations between factors and clinical outcomes indicating criterion validity. Probit regression coefficients range from -1 to 1, and the coefficient should be interpreted as the change in the predicted probability given a 1 unit increase in the predictor, with positive values indicating an increased predicted probability and negative values a decreased predicted probability [428]. A minimally adjusted model with covariates sex, ethnicity and stratification characteristics included was also tested. Listwise deletion was used, and weighted estimates were obtained using the non-response and sampling weights at age 14 for autism and ADHD, and non-response weights at age 17 for depression.

### 7.2.3 Analytic Sample and Missing Data

In the EFA and CFA analysis, the analytic sample was determined by those with complete parent-report SDQ. A total of 12,819 participants had complete or partially complete data for items in the SDQ. Of those, the majority of variables had less than 1% missing data, with only six items exceeding this. The item "adult" had the highest missing data at 2.99% and was the only item to exceed 2% missing data. It has previously been suggested that missing data on a single variable less than 5% does not present a problem as it is unlikely to introduce bias [429], and therefore the analysis was limited to complete cases through list-wise deletion. Thus, 1,300 participants were removed due to incomplete data (10%), with a total of 11,519 participants included (Figure 7-3). The sample size in the development dataset was 5819 (50.5%), whilst the sample size in the testing set was 5700 (49.5%). For sensitivity analysis for models 7-12, sample size (N=11,575) differed slightly from the main analysis due to the removal of cross-loading variables (Appendix A7.1).

For the predictive validity analysis, the sample was restricted to those with complete data on depression diagnosis (N= 8,108) at age 17, autism/Asperger's (N=9,438) and ADHD diagnosis (N=9,492) at age 14 and complete data on selected covariates (Appendix A7.2).

**Figure 7-3.** Analytic Sample for Exploratory and Confirmatory Factor Analysis



**Figure 7-3 Footnote:** sample size in the development dataset was 5,819, whilst the sample size in the testing set was 5,700.

## 7.3 Results

### 7.3.1 Descriptive Results

Distribution for item response by sex for the SDQ is shown in Appendix A7.3 for internalising symptoms, externalising symptoms, and for the pro-social scale.

For items that measure internalising symptoms, all items are skewed so that the majority of respondents answers were “not true”, or “certainty true” when items are positively worded. For emotional problems, respondents for boys were typically more likely to respond “not true” compared to respondents for girls. For the peer problems scale, differences in the responses between boys and girls varied across items.

For items that measure externalising symptoms, the majority of responses were skewed, with “Not True” (or “Certainly True” when positively worded) the most common response. However, there were three items in boys and two items in girls from the hyperactivity scale, that were not skewed. For items “Attention”, “Impulse” and “Task” the most common response among boys was “Somewhat True”, followed by “Certainly True” in the positively worded items and “Not True” in negatively worded items. Amongst girls, a similar pattern was observed for the items “Impulse” and “Task”.

For the pro-social scale, all items were skewed so that the majority of respondents answered “Certainly True”. Respondents typically indicated more pro-social behaviour among girls, as responses were more often “certainly true” and less frequently “not true” than for boys.

### 7.3.2 Exploratory Factor Analysis and Confirmatory Factor Analysis

Results from the EFA indicated that a five-factor structure has the best model fit (CFI=0.977 TLI = 0.963; RMSEA = 0.025; SRMR =0.034), whilst also retaining an eigenvalue greater than one (Table 7-1). Geomin rotated loadings indicated a unique factor for the emotional symptoms, but with a weaker loading (<0.4) for the item “complain” (0.36) (Table 7-2). The peer problem scale had cross-loading for the item “liked” with the pro-social scale. For the hyperactivity and conduct factors, there was cross-loading of items “restless” and “fidget”. There was also weak cross-loadings for the items “impulse” (-0.37) between the pro-social scale and the hyperactivity scale, and the item “considerate” (-0.36) between the pro-social and conduct problem scale.

Because of cross-loadings, in addition to models 1-6 outlined in the methods, a five-factor (models 7 and 8 with correlated errors), three-factor (models 9 and 10 with correlated errors) and second-order factor model (models 11 and 12 with correlated errors) were also tested using CFA with the items “impulse” and “liked” removed. These items were selected for removal because of cross-loadings between conceptually distinct factors (i.e pro-social and peer, pro-social and hyperactivity), whilst the cross-loading between “restless” and “fidget” were allowed to remain due to similarity of the latent factors (hyperactivity and conduct). The item “considerate” was also not removed due to the lower factor loading (0.36) on the conduct problem factor, whilst having higher loading onto the pro-social scale factor (0.52). The geomin rotated factor loadings for the EFA five-factor model with cross-loadings removed is shown in the Appendix (A7.4).

**Table 7-1.** Eigenvalues and Model Fit for Exploratory Factor Analysis (EFA) Models

<b>Model</b>	<b>Eigenvalues</b>	<b>Degrees of freedom</b>	<b><math>\chi^2</math></b>	<b>CFI</b>	<b>TLI</b>	<b>RMSEA</b>	<b>SRMR</b>
1- Factor Model	9.20	275	6070.33	0.799	0.781	0.060	0.110
2- Factor Model	2.57	251	3348.39	0.892	0.871	0.046	0.076
3- Factor Model	1.71	228	1910.61	0.942	0.923	0.036	0.055
4- Factor Model	1.28	206	1325.04	0.961	0.943	0.031	0.044
5- Factor Model	1.17	185	844.40	0.977	0.963	0.025	0.034
6- Factor Model	0.86	165	534.34	0.987	0.977	0.020	0.027
7- Factor Model	0.84	146	353.74	0.993	0.985	0.016	0.020
8- Factor Model	0.69	128	245.02	0.996	0.990	0.013	0.016

**Table 7-1. Footnote:** Abbreviations:  $\chi^2$  – chi-squared; CFI – Comparative Fit Index; TLI – Tucker-Lewis Index; RMSEA – Root Mean Square Error of Approximation; SRMR – standardized root mean squared residual.

**Table 7-2.** Geomin Rotated Loadings for Five-Factor Model

	<b>Factor 1</b>	<b>Factor 2</b>	<b>Factor 3</b>	<b>Factor 4</b>	<b>Factor 5</b>
<b>Emotional Symptom Scale</b>					
<i>Complains</i>	0.360*	0.273*	0.153*	-0.014	0.034
<i>Unhappy</i>	0.623*	0.311*	0.011	-0.064*	0.128*
<i>Worried</i>	0.767*	0.056*	0.056*	-0.005	0.035
<i>Anxiety</i>	0.625*	-0.045	-0.079*	0.108*	0
<i>Fear</i>	0.798*	-0.009	0.004	0.055*	-0.011
<b>Peer Problems</b>					
<i>Alone</i>	0.242*	-0.169*	-0.177*	-0.009	0.516*
<i>Friend</i>	0.017	-0.03	-0.290*	0.032	0.545*
<i>Liked</i>	0.112*	0.022	-0.433*	0.034	0.526*
<i>Bullied</i>	0.339*	0.149*	-0.015	0.042	0.398*
<i>Adults</i>	0.078	0.056	0.005	-0.007	0.611*
<b>Hyperactivity Scale</b>					
<i>Restless</i>	-0.124*	0.461*	0.047*	0.486*	0.257*
<i>Fidget</i>	-0.040*	0.419*	0.051*	0.477*	0.279*
<i>Attention</i>	0.085*	0.201*	-0.128*	0.665*	0.038
<i>Impulse</i>	0.021	0.206*	-0.365*	0.396*	-0.045
<i>Task</i>	0.062*	-0.066	-0.415*	0.718*	-0.018
<b>Conduct Problems</b>					
<i>Anger</i>	0.170*	0.596*	-0.060*	0.100*	-0.025
<i>Obedience</i>	-0.007	0.504*	-0.348*	0.097*	-0.085*
<i>Aggression</i>	0.099*	0.552*	-0.198*	0.015	0.194*
<i>Lies</i>	0.140*	0.641*	-0.085*	0.054	-0.080*
<i>Steals</i>	0.05	0.778*	-0.115	-0.091	-0.003
<b>Pro-Social Scale</b>					
<i>Considerate</i>	0.025	-0.355*	0.521*	0.055*	-0.045
<i>Shares</i>	0.011	-0.206*	0.566*	0.076*	-0.097*
<i>Helpful</i>	0.055*	-0.041	0.750*	0.091*	-0.042
<i>Kind</i>	0.055	-0.110*	0.665*	0.002	-0.044
<i>Volunteers</i>	-0.009	0.02	0.707*	-0.096*	0.208*

**Table 7-2. Footnote:** \* indicates  $p < 0.05$ . Loadings greater or equal to 0.4 are highlighted in dark grey. Greater or equal to 0.35 are highlighted in light grey.

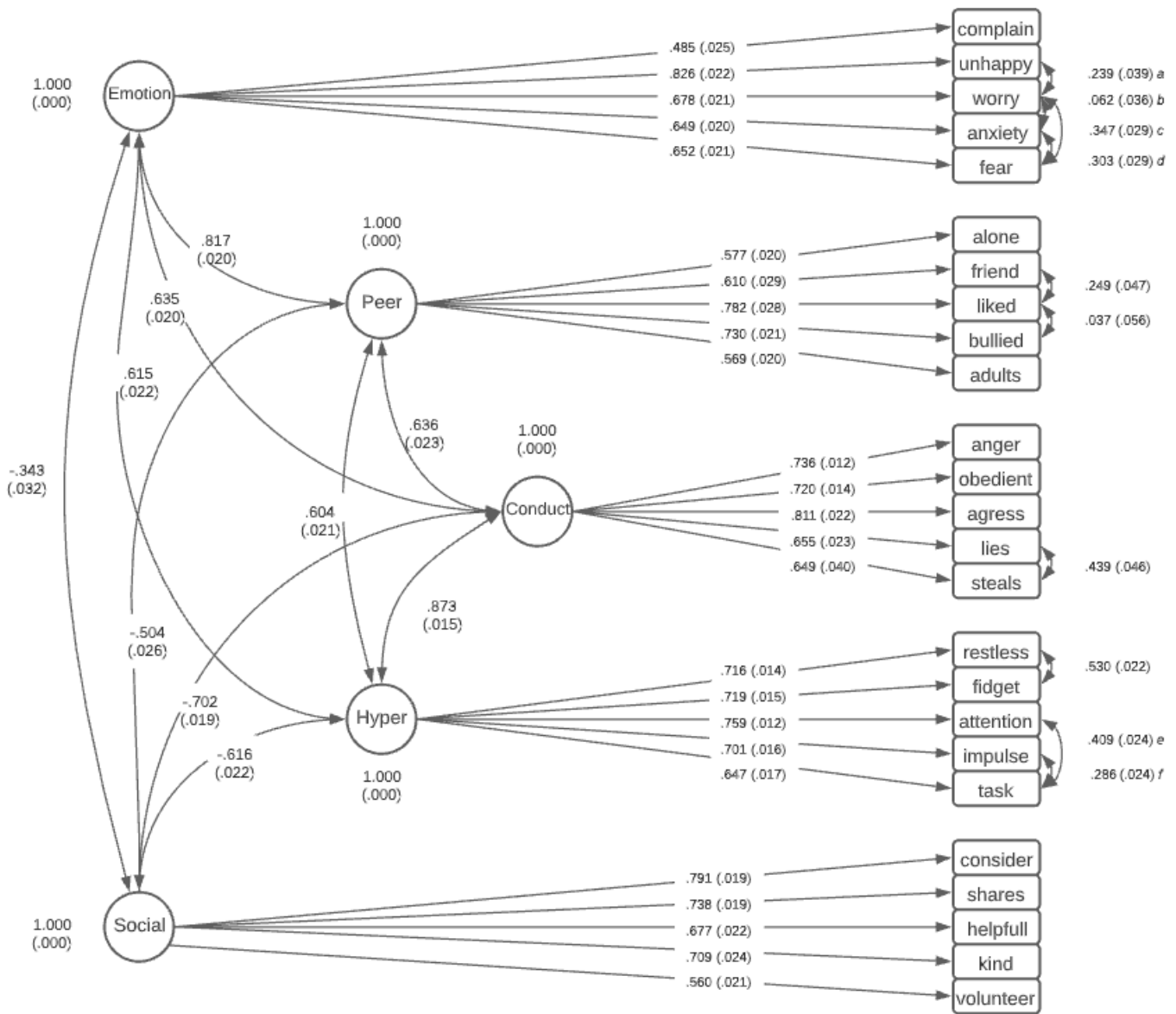
For models 1, 3 and 5, CFA fit statistics for the five-factor model (model 1, CFI =0.921, TLI= 0.911, RMSEA = 0.036, SRMR = 0.062) and the second-order model (model 5, CFI = 0.921, TLI= 0.911, RMSEA = 0.036, SRMR =0.064) were comparable, and indicated better fit than the three-factor model (model 3, CFI =0.893, TLI= 0.882, RMSEA = 0.042, SRMR = 0.075) (Table 7-3). In models 2, 4 and 6, the correlated errors adopted were “fear” with “anxiety” and “worry”; “anxiety” with “worry”; “worry” with “unhappy”; “bullied” with “liked”; “liked” with “friend”; “task” with “attention” and “impulse”; “fidget” with “restless”; “steals” with “lies”. Inclusion of correlated errors improved model fit, although for CFI/TLI the fit indices were still considered only acceptable rather than good. Model 2 (first-order five-factor model with correlated errors) and model 6 (second-order factor model with correlated errors) had a comparable fit, that was better than the other models. Therefore, models 2 and 6 were selected for subsequent analyses.

In the models with cross-loading items removed, the five-factor model with correlated errors (model 8) and the second-order model with correlated errors (model 12) had the best fit (Appendix A7.5). For model 8 fit indices were CFI = 0.956, TLI =0.948, RMSEA = 0.028, SRMR = 0.051 and for model 12 were CFI = 0.953, TLI =0.945, RMSEA = 0.029, SRMR = 0.053. Due to the good model fit, models 8 and 12 with cross-loading items “impulse” and “liked” removed were selected for further sensitivity analysis (Appendix A7.5), alongside the main analysis models 2 and 6.

The majority of the standardised factor loadings (Tables 7.4 and 7.5) for the five-factor model and second-order model with correlated errors (models 2 and 6, respectively) demonstrated acceptable loading (>0.5), with only one item having a loading marginally smaller than 0.5 (for item “complains”, loading of 0.49 and 0.48 respectively). For both models, over half of the first-order item loadings (N=13) exceeded 0.7. For the five-factor model, loadings for the emotional symptom factor ranged from 0.49 to 0.83, for conduct problems from 0.65 to 0.81, for hyperactive scale from 0.65 to 0.76, for the peer problem scale from 0.58 to 0.78, and for the pro-social scale from 0.56 to 0.79 (Table 7-4, Figure 7-4). In the second-order model (model 6), first-order loadings were similar to the five-factor model (model 2), whilst loadings onto the second-order factor were 0.88 and 0.93 for internalising symptoms, and 0.95 and 0.88 for externalising symptoms (Table 7-5, Figure 7-5). Factor loadings were strong for the items “liked” (0.78) and “impulse” (0.7) which were removed in the sensitivity analysis.

For all items bar one (“complains”), the underlying factor explained >30% of the variance for the items in both the first-order and second-order models. The internal consistency of the subscales and the second-order factors, as indicated by the ordinal alpha, were all good (>0.7). The ordinal alphas ranged from 0.8 for the peer problems scale to 0.86 for the hyperactivity scale for first-order factors, and the second-order internalising and externalising factors were 0.89 and 0.92 respectively (Table 7-6).

**Figure 7-4** First-Order Five-Factor Structures with Standardised Factor Loadings and Standard Errors



**Figure 7-4 Footnote:** Values on straight paths with single-headed arrows, between factor and observed variables, indicate standardised estimates for factor loadings. Values on curved paths with double-headed arrows, between either two-factors or two observed variables, are correlation estimates. Values in brackets are standard errors (SE). Correlated errors a-f represent a) unhappy with worry; b) worry with anxiety; c) worry with fear; d) anxiety with fear; e) attention with task; f) impulse with task.

**Table 7-3.** Goodness of Fit Indices for Competing Models in Confirmatory Factor Analysis.

<i>Model</i>	$\chi^2$	<i>df</i>	<i>CFI</i>	<i>TLI</i>	<i>RMSEA</i>	<i>SRMR</i>
1) <i>Baseline five-factor Model</i>	2227.962	265	0.921	0.911	0.036	0.062
2) <i>Five-factor model with correlated errors</i>	1710.710	255	0.942	0.931	0.032	0.057
3) <i>Baseline three-factor Model</i>	2947.547	272	0.893	0.882	0.042	0.075
4) <i>Three-factor model with correlated errors</i>	1921.125	262	0.934	0.924	0.033	0.062
5) <i>Second-order two-factor model</i>	2247.816	268	0.921	0.911	0.036	0.064
6) <i>Second-order two-factor model with correlated errors</i>	1741.387	258	0.941	0.931	0.032	0.058

**Table 7-3. Footnote:** Abbreviations:  $\chi^2$  – chi-squared; *df* – degrees of freedom; *CFI* – Comparative Fit Index; *TLI* – Tucker-Lewis Index; *RMSEA* – Root Mean Square Error of Approximation; *SRMR* – Standardized Root Mean Squared Residual.

**Table 7-4.** Standardised Factor Loadings for the First-Order Five-Factor Model, with Correlated Errors (Model 2).

<b>Items</b>	<b>Loadings</b>	<b>S/E</b>	<b>R<sup>2</sup></b>	<b>P Values</b>
<b>Emotional Symptom Scale</b>				
<i>Complains</i>	0.49	0.03	0.24	<0.001
<i>Unhappy</i>	0.83	0.02	0.68	<0.001
<i>Worried</i>	0.68	0.02	0.46	<0.001
<i>Anxiety</i>	0.65	0.02	0.42	<0.001
<i>Fear</i>	0.65	0.02	0.43	<0.001
<b>Conduct Problems</b>				
<i>Anger</i>	0.74	0.01	0.51	<0.001
<i>Obedience</i>	0.72	0.01	0.52	<0.001
<i>Aggression</i>	0.81	0.02	0.58	<0.001
<i>Lies</i>	0.66	0.02	0.49	<0.001
<i>Steals</i>	0.65	0.04	0.42	<0.001
<b>Hyperactivity Scale</b>				
<i>Restless</i>	0.72	0.01	0.33	<0.001
<i>Fidget</i>	0.72	0.02	0.37	<0.001
<i>Attention</i>	0.76	0.01	0.61	<0.001
<i>Impulse</i>	0.70	0.02	0.53	<0.001
<i>Task</i>	0.65	0.02	0.32	<0.001
<b>Peer Problems</b>				
<i>Alone</i>	0.58	0.02	0.54	<0.001
<i>Friend</i>	0.61	0.03	0.52	<0.001
<i>Liked</i>	0.78	0.03	0.66	<0.001
<i>Bullied</i>	0.73	0.02	0.43	<0.001
<i>Adults</i>	0.57	0.02	0.42	<0.001
<b>Pro-Social Scale</b>				
<i>Considerate</i>	0.79	0.02	0.63	<0.001
<i>Shares</i>	0.74	0.02	0.55	<0.001
<i>Helpful</i>	0.68	0.02	0.46	<0.001
<i>Kind</i>	0.71	0.02	0.5	<0.001
<i>Volunteers</i>	0.56	0.02	0.31	<0.001

**Table 7-4 Footnote:** Shorthand names for items are presented. Full breakdown of item questions is reported in Chapter 4.

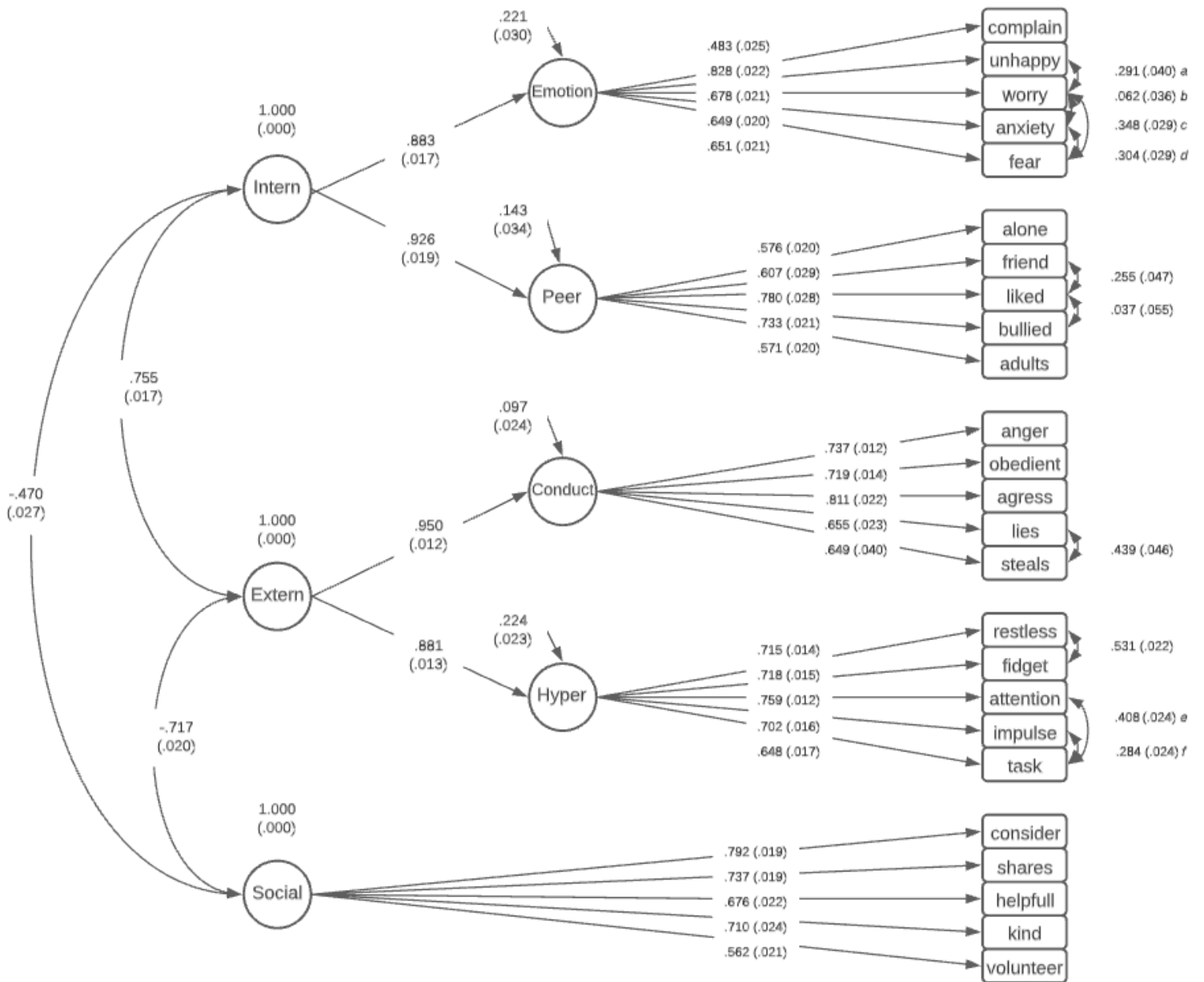
**Table 7-5.** Standardised Factor Loadings for the Second-Order Two-Factor Model, with Correlated Errors (Model 6).

Items	Loadings	S/E	R <sup>2</sup>	P Values
<b>First-order factors</b>				
<b>Emotional Symptom Scale</b>				
<i>Complains</i>	0.48	0.03	0.23	<0.001
<i>Unhappy</i>	0.83	0.02	0.69	<0.001
<i>Worried</i>	0.68	0.02	0.46	<0.001
<i>Anxiety</i>	0.65	0.02	0.42	<0.001
<i>Fear</i>	0.65	0.02	0.42	<0.001
<b>Conduct Problems</b>				
<i>Anger</i>	0.74	0.01	0.54	<0.001
<i>Obedience</i>	0.72	0.01	0.52	<0.001
<i>Aggression</i>	0.81	0.02	0.66	<0.001
<i>Lies</i>	0.66	0.02	0.43	<0.001
<i>Steals</i>	0.65	0.04	0.42	<0.001
<b>Hyperactivity Scale</b>				
<i>Restless</i>	0.72	0.01	0.51	<0.001
<i>Fidget</i>	0.72	0.02	0.52	<0.001
<i>Attention</i>	0.76	0.01	0.58	<0.001
<i>Impulse</i>	0.70	0.02	0.49	<0.001
<i>Task</i>	0.65	0.02	0.42	<0.001
<b>Peer Problems</b>				
<i>Alone</i>	0.58	0.02	0.33	<0.001
<i>Friend</i>	0.61	0.03	0.37	<0.001
<i>Liked</i>	0.78	0.03	0.61	<0.001
<i>Bullied</i>	0.73	0.02	0.54	<0.001
<i>Adults</i>	0.57	0.02	0.33	<0.001
<b>Pro-Social Scale</b>				
<i>Considerate</i>	0.79	0.02	0.63	<0.001
<i>Shares</i>	0.74	0.02	0.54	<0.001
<i>Helpful</i>	0.68	0.02	0.46	<0.001
<i>Kind</i>	0.71	0.02	0.50	<0.001
<i>Volunteers</i>	0.56	0.02	0.32	<0.001
<b>Second-order factors</b>				
<b>Internalising</b>				
<i>Emotional Symptom Scale</i>	0.88	0.02	0.78	<0.001
<i>Peer Problems</i>	0.93	0.02	0.86	<0.001
<b>Externalising</b>				
<i>Conduct Problems</i>	0.95	0.01	0.90	<0.001
<i>Hyperactivity Scale</i>	0.88	0.01	0.78	<0.001

**Table 7-5 Footnote:** Shorthand names for items are presented. A full breakdown of item questions is reported in Chapter 4.



**Figure 7-5** Second-Order Factor Structure with Standardised Factor Loadings and Standard Errors.



**Figure 7-5 Footnote:** Values on straight paths with single-headed arrows, between factor and observed variables, indicate standardised estimates for factor loadings. Values on curved paths with double-ended arrows, between either two-factors or two observed variables, are correlation estimates. Values at end of a straight single-headed arrow directed at a first-order factor are residual variances. Values in brackets are standard errors (SE). Correlated errors a-f represent a) unhappy with worry; b) worry with anxiety; c) worry with fear; d) anxiety with fear; e) attention with task; f) impulse with task.

**Table 7-6.** Ordinal Alpha for Strengths and Difficulties Sub-Scales

	<b>Emotional Symptom Scale</b>	<b>Conduct Problems</b>	<b>Hyperactivity Scale</b>	<b>Peer Problems</b>	<b>Pro-Social Scale</b>	<b>Internalising Symptoms</b>	<b>Externalising Symptoms</b>
Average correlation ( <i>r</i> ):	0.48	0.52	0.55	0.44	0.48	0.46	0.54
Items in scale ( <i>k</i> ):	5	5	5	5	5	10	10
Ordinal Alpha:	0.82	0.84	0.86	0.8	0.82	0.89	0.92

**Table 7-6. Footnote:** Ordinal alpha for items in measurement scales. Ordinal alpha represented by the equation  $(k*r)/(1+(k-1)*r)$

### 7.3.3 Measurement Invariance

Structural, factorial and strong factorial invariance were demonstrated for the five-factor first-order model across all groups (Model 2, Table 7-7). The differences between the configural and metric models for sex were 0.002 (CFI), 0.004 (TLI), 0.001 (RMSEA) and 0.00 (SRMR). The differences between scalar and metric models for sex were 0.001 (CFI), 0.004 (TLI), 0.001 (RMSEA) and 0.001 (SRMR). Similar differences were also observed for the environmental categories for RMSEA and SRMR, whilst slightly larger differences were observed for CFI/TLI, but none that exceeded the cut-offs.

For the second-order factor model (model 6), scalar invariance was demonstrated as the model fit was good or acceptable for all indices across all groups tested (Table 7-8). For sex, the fit indices were 0.946 (CFI), 0.943 (TLI), 0.029 (RMSEA) and 0.065 (SRMR). A similar or better fit was identified for the IMD, green spaces, domestic gardens and fast-food density. Therefore, the second-order factor had strong factorial invariance. Because structural and factorial invariance was demonstrated in the first-order part of the model, and strong factorial invariance was demonstrated in the second-order model, structural and factorial invariance could also be assumed for the second-order model.

**Table 7-7.** Fit Indices Difference Tests to Confirm Configural, Metric and Scalar Measurement Invariance in the Five-Factor First-Order Model

<b>Model</b>	<b>ΔCFI</b>	<b>ΔTLI</b>	<b>ΔRMSEA</b>	<b>ΔSRMR</b>
<b>Sex</b>				
<i>Metric Vs Configural</i>	0.002	0.004	0.001	0.00
<i>Scalar Vs Metric</i>	0.001	0.004	0.001	0.001
<b>Index of Multiple Deprivation</b>				
<i>Metric Vs Configural</i>	0.004	0.008	0.001	0.00
<i>Scalar Vs Metric</i>	0.002	0.006	0.002	0.001
<b>Green Spaces</b>				
<i>Metric Vs Configural</i>	0.003	0.006	0.002	0.00
<i>Scalar Vs Metric</i>	0.002	0.004	0.001	0.00
<b>Domestic Gardens</b>				
<i>Metric Vs Configural</i>	0.004	0.007	0.002	0.001
<i>Scalar Vs Metric</i>	0.004	0.007	0.002	0.00
<b>Fast-food Density</b>				
<i>Metric Vs Configural</i>	0.005	0.007	0.001	0.00
<i>Scalar Vs Metric</i>	0.003	0.006	0.002	0.001

**Table 7-3. Footnote:** Difference between nested models greater than 0.010 for CFI/TLI, 0.015 for RMSEA and 0.030 for SRMR indicates non-invariance. Abbreviations: CFI – Comparative Fit Index; TLI – Tucker-Lewis Index; RMSEA – Root Mean Square Error of Approximation; SRMR – Standardized Root Mean Squared Residual.

**Table 7-8.** Fit Indices for the Scalar Model for the Second-Order Factor

<b>Model</b>	<b>CFI</b>	<b>TLI</b>	<b>RMSEA</b>	<b>SRMR</b>
<i>Sex</i>	0.946	0.943	0.029	0.065
<i>Index of multiple deprivation</i>	0.946	0.944	0.027	0.067
<i>Green spaces</i>	0.957	0.955	0.026	0.065
<i>Domestic gardens</i>	0.954	0.952	0.029	0.069
<i>Fast-food density</i>	0.958	0.956	0.024	0.066

**Table 7-3. Footnote:** Abbreviations: CFI – Comparative Fit Index; TLI – Tucker-Lewis Index; RMSEA – Root Mean Square Error of Approximation; SRMR – Standardized Root Mean Squared Residual.

### 7.3.4 Average Variance Extracted

For the five-factor model (model 2), AVE scores were above the 0.5 threshold for the emotional problems and conduct problem factors, and borderline for the pro-social factor (AVE 0.49), whilst the peer problem and hyperactivity factors had AVE scores lower than 0.5 (Table 7-10). For the second-order factor (model 6), AVE scores for the internalising and externalising symptoms were 0.81 and 0.84 respectively (Table 7-9).

In the five-factor model (model 2, Table 7-10), the AVE scores for peer problem (0.44) and emotional problems (0.51) latent variables were smaller than their squared correlation (0.72), but larger than any other squared correlation. This indicating that peer and emotional factors may be measuring a similar construct, but are distinct from other constructs. Similarly, the squared correlation for conduct and hyperactivity factors (0.62) were greater than the AVE scores for either factor (0.5 and 0.43, respectively). When a second-order model is adopted, the AVE scores of internalising (0.81) and externalising (0.84) symptoms were greater than their squared correlation (0.57), indicating they were measuring separate constructs (Table 7-9).

In the five-factor model (model 2) the AVE score for the pro-social factor (0.49) was equal to the squared correlation with the conduct factor (0.49). In the second-order (model 6), the pro-social AVE score was lower than the squared correlation with the externalising symptoms factor (0.51), indicating there may be some cross-over in the traits measured.

In the sensitivity analysis, AVE scores in the five-factor structure (model 8) were similar to the main analysis, with only the emotion (0.52) and conduct (0.54) factors meeting the 0.5 threshold (Appendix A7.6). The AVE scores for the second-order structure (model 12) compared to the main analysis were the same for the pro-social scale (0.49), slightly higher for the internalising symptoms (0.85) and marginally lower for externalising symptoms (AVE 0.79). The pattern for squared correlations was similar to the main analysis for the five-factor structure (model 8, Appendix A7.6), with the pro-social factor having an equal AVE score to the squared correlations with the conduct factor. When the second-order structure (model 12) was adopted (Appendix A7.6), unlike the main analysis, the AVE score for the pro-social factor (0.49) slightly exceeded the squared correlation with the externalising symptom factor (0.47).

**Table 7-9.** Average Variance Extracted and Squared Correlations for Second-Order Factor Model

		<b>Correlation</b>			<b>Squared Correlation</b>		
	<b>AVE</b>	<b>Intern</b>	<b>Extern</b>	<b>Social</b>	<b>Intern</b>	<b>Extern</b>	<b>Social</b>
<b>Intern</b>	0.81						
<b>Extern</b>	0.84	0.76			0.57		
<b>Social</b>	0.49	-0.47	-0.72		0.22	0.51	

**Table 7-9 Footnote:** AVE scores are the average  $R^2$  score, and represent the average variance explained by the factor in the items that it is measured by. Shorthand name “intern” refers to internalising symptoms, “extern” to externalising symptoms, “social” to pro-social scale. Abbreviation: AVE – Average Variance Explained.

**Table 7-10.** Average Variance Extracted and Squared Correlations for Five-Factor Model

	AVE	Correlation					Squared Correlation				
		Peer	Emotion	Conduct	Hyper	Social	Peer	Emotion	Conduct	Hyper	Social
<b>Peer</b>	0.44										
<b>Emotion</b>	0.51	0.85					0.72				
<b>Conduct</b>	0.50	0.64	0.60				0.40	0.36			
<b>Hyper</b>	0.43	0.59	0.63	0.79			0.35	0.40	0.62		
<b>Social</b>	0.49	-0.41	-0.34	-0.70	-0.51		0.17	0.12	0.49	0.26	

**Table 7-10 Footnote:** AVE scores are the average  $R^2$  score, and represent the average variance explained by the factor in the items that it is measured by. Shorthand name “Peer” refers to Peer Problems, “Emotion” to Emotional Symptoms, “Conduct” to Conduct Problems, “Hyper” to Hyperactivity and “Social” to Pro-Social. Abbreviation: AVE – Average Variance Explained.

### 7.3.5 Predictive Validity

Prevalence of ADHD and autism/Asperger’s at age 14 were 1.4% and 2.3%, respectively. Age 17 reported incidence of depression diagnosis since age 13 was 9.4%. The distribution of the sample according to diagnostic criteria are presented in the Appendix (A7.7). Standardised probit regression coefficients for associations between the second-order factor and clinical outcomes are shown in Table 7-11, and for the first-order factor in Table 7-12.

For the second-order factors (model 6) a higher level of internalising symptoms were associated with increased likelihood of depression in both the unadjusted and adjusted models, and a higher level of externalising symptoms were associated with ADHD in both models (Table 7-11). For ADHD, pro-social symptoms were also associated with a formal diagnosis. For autism, internalising symptoms were positively related to autism diagnosis whilst pro-social symptoms were negatively related. No association is observed with externalising symptoms (Table 7-11).

For the five-factor model (model 2) emotional problems and conduct problems were positively associated with depression, and hyperactivity negatively associated with this outcome in unadjusted models. Only emotional and conduct problems were associated in the minimally adjusted model (Table 7-12). Peer problems, emotional problems and hyperactivity predicted ADHD diagnosis, with hyperactivity having the strongest effect and emotional problems being negatively associated. For autism, all factors bar emotional problems were associated with diagnosis, with peer problems being the strongest. Hyperactivity was positively related to diagnosis, whilst conduct problems were negatively related.

Results for the sensitivity analysis with cross-loadings removed (model 8 and 12, Appendix A7.8) were comparable to the results in the main analysis. For the second-order factor, there was additional evidence that internalising symptoms were negatively associated with ADHD at age 14 (-0.15, SE 0.09,  $p=0.09$ ). In the five-factor model, hyperactivity also negatively predicted depression (-0.16, SE

0.08,  $p=0.044$ ), and emotional symptoms were negatively associated with autism (-0.41, SE 0.17,  $p=0.016$ ).

**Table 7-11.** Predictive Validity of Second-Order Factors Structure

	<b>Mutually Adjusted</b>			<b>Minimally Adjusted</b>		
	Estimate	SE	P Value	Estimate	SE	P Value
<b>Depression</b>						
<i>Internalising Symptoms</i>	0.37	0.09	<0.001	0.32	0.08	<0.001
<i>Externalising Symptoms</i>	0.05	0.1	0.66	0.07	0.09	0.42
<i>Pro-social</i>	0.1	0.07	0.13	0.05	0.06	0.47
<b>ADHD</b>						
<i>Internalising Symptoms</i>	-0.16	0.09	0.071	-0.09	0.08	0.29
<i>Externalising Symptoms</i>	0.92	0.12	<0.001	0.8	0.11	<0.001
<i>Pro-social</i>	0.14	0.08	0.083	0.15	0.08	0.04
<b>Autism/ Asperger's</b>						
<i>Internalising Symptoms</i>	0.54	0.07	<0.001	0.58	0.15	<0.001
<i>Externalising Symptoms</i>	0.11	0.1	0.25	0.05	0.09	0.60
<i>Pro-social</i>	-0.17	0.07	0.018	-0.14	0.08	0.07

**Table 7-11 Footnote:** Standardised probit regression coefficients and standard errors (SE). In mutually adjusted models, all factors are included in the model at the same time (*internalising symptoms, externalising symptoms, pro-social*). In minimally adjusted models, additional adjustments are made for sex, ethnicity and stratification characteristics. ADHD – Attention Deficit and Hyperactivity Disorder.

**Table 7-12.** Predictive Validity of Five-factor Model

	<b>Mutually Adjusted</b>			<b>Minimally Adjusted</b>		
	Estimate	SE	P Value	Estimate	SE	P Value
<b>Depression</b>						
<i>Emotional Symptoms</i>	0.31	0.1	0.002	0.29	0.09	0.001
<i>Peer Problems</i>	-0.03	0.08	0.71	-0.02	0.07	0.75
<i>Conduct Problems</i>	0.36	0.16	0.026	0.25	0.13	0.055
<i>Hyperactivity</i>	-0.19	0.1	0.063	-0.10	0.09	0.23
<i>Pro-social</i>	0.14	0.09	0.12	0.06	0.08	0.42
<b>ADHD</b>						
<i>Emotional Symptoms</i>	-0.44	0.14	0.002	-0.39	0.13	0.004
<i>Peer Problems</i>	0.43	0.14	0.002	0.42	0.13	0.001
<i>Conduct Problems</i>	0.09	0.15	0.574	0.13	0.14	0.36
<i>Hyperactivity</i>	0.67	0.12	<0.001	0.55	0.1	<0.001
<i>Pro-social</i>	0.09	0.09	0.36	0.11	0.09	0.20
<b>Autism/Asperger's</b>						
<i>Emotional Symptoms</i>	-0.17	0.13	0.19	-0.17	0.12	0.18
<i>Peer Problems</i>	0.69	0.11	<0.001	0.70	0.19	<0.001
<i>Conduct Problems</i>	-0.55	0.19	0.003	-0.51	0.24	0.035
<i>Hyperactivity</i>	0.66	0.13	<0.001	0.59	0.21	0.005
<i>Pro-social</i>	-0.22	0.09	0.014	-0.2	0.11	0.078

**Table 7-12 Footnote:** Standardised probit regression coefficients and standard errors (SE). In mutually adjusted models, all factors are included in the model at the same time (*Emotional Symptoms, Peer Problems, Conduct Problems, Hyperactivity, Pro-Social*). In minimally adjusted models, additional adjustments are made for sex, ethnicity and stratification characteristics. ADHD – Attention Deficit and Hyperactivity Disorder.

## 7.4 Discussion

### 7.4.1 Summary

This chapter aimed to assess the validity of the SDQ for use in subsequent mediation analysis, and two hypotheses were tested to achieve this. The findings of this chapter are summarised in relation to these hypotheses.

***Hypothesis 7: The Strength and Difficulties Questionnaire captures internalising and externalising latent traits in a UK population of adolescents and the latent factors are appropriate to use as mediators in further analysis.***

The results indicate that either a first-order five-factor or a second-order two-factor model is appropriate for use in further analysis in the UK MCS. Structural validity was demonstrated through EFA and CFA, and internal consistency was demonstrated by ordinal alpha for all sub-scales and factors. There was greater evidence of internal convergent validity and external discriminant validity for the second-order factor, with larger AVE scores and lower squared correlations between constructs. Criterion validity was demonstrated for both models, but the second-order factor had associations more consistent with those hypothesised for ADHD and depression diagnosis.

There was some evidence that items “impulse” and “liked” should be removed due to cross-loading in EFA, and good model fit in the CFA. However, removing the items did not substantially improve internal convergent validity, external discriminant validity or predictive validity.

***Hypothesis 8: The latent constructs of internalising and externalising symptoms are measured in the same way in boys and girls, and across different environmental conditions (i.e high and low deprivation, high and low green space, domestic garden and fast-food outlet density).***

Invariance for both models was achieved across groups, demonstrating that the latent constructs were measured in the same way in males and females, and across levels of environmental conditions. Therefore, the use of internalising and externalising factors are appropriate for sub-group analysis in SEM.

### 7.4.2 Comparison to Previous Literature

The results were consistent with work conducted previously using the MCS from ages 3 to 7, which demonstrated a five-factor model fitted the data better when compared with a three-factor and one-factor model [408]. Similar to the work by Croft et al. (2015), there was mixed support for internal convergent validity in the five-factor model, as demonstrated by low AVE scores for factors [408], although the present work has a greater number of factors meeting the 0.5 threshold. Unlike Croft et al. (2015), who did not test a second-order factor, the current analysis finds better convergent validity as indicated by AVE scores in the second-order model. Compared to Croft et al. (2015), who demonstrated adequate external discriminant validity for all factors in the five-factor model [408], this study finds poor discriminant validity between emotional symptoms and peer problems, and between hyperactivity and conduct problems indicating they may be measuring the same construct. The



relation between AVE scores and squared correlations improved in the second-order model where discriminant validity was achieved for the internalising and externalising symptoms. The worse model fit of the three-factor model, provides additional justification for adopting a second-order model.

Croft et al. (2015) demonstrated poor predictive validity for personal, social, and emotional development (PSE) at age 5 by the peer problems and emotional subscales in the five-factor model at age 3, as they did not independently predict PSE at age 5. However, hyperactivity and conduct problems positively predicted ADHD at age 5, and hyperactivity negatively predicted PSE at the same age [408]. Croft et al. (2015) note that the scale may better predict future internalising problems, such as depression, which was not collected in their analysis. The current analysis was able to test associations for depression and found the incidence of depression from 13-17 was positively predicted by emotional problems and conduct problems in the five-factor model. ADHD prevalence at 14 was negatively predicted by emotional symptoms, and positively predicted by hyperactivity and peer problems. This analysis finds predictive validity consistent with that hypothesised for the second-order factors for ADHD and depression, but less so for autism/Asperger's which was predicted by internalising symptoms and the pro-social scale, but not externalising symptoms.

The study was also consistent with work conducted in other populations, which had demonstrated a three-factor model with internalising, externalising and a pro-social factor fit the data worse when compared to a five-factor or second-order model [400, 404, 407, 408, 430]. In the UK, Goodman et al. (2010) found the best fit for a second-order factor model in the general population aged 5-16, whilst recommended a five-factor model in clinical populations when screening for disorders. Support for the second-order structure has also been found in an Italian population of children aged 3-15 [406] and in a Danish population of children aged 5-7 and 10-12 which, similar to this study, also found it comparable to a five-factor model [405]. However, in Spain, data from a population of children aged 4-14 provided evidence that a five-factor model was a better fit than a second-order model [407]. Similarly, in a Spanish population of adolescents aged 11-19, a five-factor model, or a bi-factorial model was shown to be the most appropriate whilst the second-order factor showed worse fit [404]. In a cross-country comparison in Europe including a sample from England of adolescents aged 12-17, the five-factor model had the best fit across all countries included, with better fit than the second-order model, whilst the three-factor model had the worst fit [430].

The current work did not test discriminant and convergent validity of the SDQ for internalising and externalising symptoms across informants (i.e parents or teachers). This analysis only adopts parent-reported SDQ, but it is important to consider how measures may differ by respondent, as this may impact the validity of results when expanded to further analysis. Goodman et al. (2010) previously demonstrated that the second-order internalising and externalising model that showed the clearest and most consistent validity across informants in a UK sample, whereas the five-factor first-order model had poor cross-informant discriminant validity [400]. This was the case for the emotional and peer problems subscales, and was particularly poor between the behavioural, hyperactivity and pro-social subscales when used in low-risk populations. This indicated that when using the five-factor model, each of the factors may not measure the same psychological constructs when measured by

different reporters [400]. However, in low-risk populations internalising and externalising symptoms traits are measured similarly across informants. This supports the use of parent-reported SDQ in the MCS as it is unlikely the measured constructs would differ significantly if teacher-reported measures were used.

### 7.4.3 Methodological Considerations

In adopting a second-order factorial structure the two internalising and externalising factors were measured by only two first-order factors. This has typically been advised against, with recommendations that at least three items measure a single factor [431]. This is because factors measured by two items may have low reliability and therefore be harder to replicate across samples and may also result in models being unidentified [427]. However, in the current analysis, we demonstrated good internal reliability as measured by the ordinal alpha, and all models were identified. Additionally, a second-order factor with two variables can be considered when the first-order factors are highly correlated with each other ( $>0.7$ ) but not with other factors [432]. In the present analysis, correlations between the first-order items that measures the same second-order factor were higher than 0.7, with other correlations lower (with the exception of pro-social and conduct which is equal to 0.7).

A further exception to the rule is when there are strong theoretical reasons to have only two items loading on a factor [433]. In the present analysis, the decision to allow two items was based on previous theory and validation work that has demonstrated this approach is appropriate for the SDQ [405, 406], and in particular among general populations in the UK [400]. Finally, a factor measured by two items should only be retained if it can be interpreted in a meaningful way [434], which was demonstrated through the predictive validity of internalising and externalising symptoms in relation to depression and ADHD diagnosis, respectively.

The study finds low AVE scores for some of the first-order factors, with some not reaching the 0.5 threshold. Some of the AVE scores were only marginally lower than the indicated 0.5 cut-off, and therefore may not be problematic. It has been suggested that it is possible to still use the factors with low AVE scores if the model fit is deemed good, factor loading is strong and there is predictive validity [435].

The AVE scores for the second-order internalising and externalising factors were higher than 0.5, and were also higher than their respective squared correlation, demonstrating external discriminant validity. However, this was not the case for the pro-social factor which had a lower AVE score than its squared correlation with the externalising factor. Consideration should be taken as to whether the pro-social scale should be combined with the externalising symptoms factor, cross-loadings removed, or alternate factor structures considered.

Removal of cross-loadings was explored in sensitivity analysis as the EFA indicated cross-loading of items “impulse” and “liked” that may be problematic. However, removal of cross-loading factors did not improve the AVE scores substantially in the first-order factor model, although there was some evidence that the pro-social scale was a distinct factor in the second-order model.

Similar model fit and predictive validity were demonstrated with and without inclusion of items “impulse” and “liked”, and factor loadings in the main analysis for both items are strong. As this thesis only proposed hypotheses relating to the mediating role of externalising and internalising symptoms, the poor external discriminant validity for the pro-social factor will likely have little consequence in further analysis.

CFA should be hypothesis and theory driven, as opposed to data driven. Therefore, the decision was made to retain the “impulse” and “liked” items in line with the objectives of CFA to identify both an empirically and conceptually supported factor structure, and sensitivity analysis indicated the factorial structures were comparable in their model fit and predictive ability. However, future analysis that intends to adopt the pro-social scale may wish to explore alternative factorial structures in line with theoretical models.

It was not possible to assess measurement invariance using a “bottom up” approach. However, it was possible to demonstrate this for the first-order five-factor structure, which is a pre-condition to demonstrating invariance for second-order factors [436], and it was possible to demonstrate scalar invariance for the second-order structure using a “top-down” approach. This has been deemed an acceptable approach for second-order factors [437]. Therefore, there is reasonable certainty that metric and configural invariance was also achieved for the second-order model, as the scalar model is the strictest form of invariance.

#### 7.4.4 Strengths and Limitations

There are a number of strengths and limitations of the current work. Firstly, by adopting EFA and CFA in further analysis, the common problems associated with using sum scores will be avoided when using internalising and externalising symptoms as mediators. In addition, measurement error will be reduced. Furthermore, the validity of the scale has been demonstrated for future analysis, in particular, structural validity, internal convergent validity, criterion validity and partial external discriminant validity has been demonstrated for the second-order factor.

A further strength of this work is that it was possible to test predictive validity as there was data on clinical diagnosis from later sweeps than when the SDQ was collected. However, these were measured in different ways (incidence vs prevalence) and at different time points. For ADHD and autism/Asperger’s which measured the prevalence of conditions at age 14, there may be a temporality issue whereby cohort members may have received the diagnosis at an earlier time point and before the SDQ measures were taken. Additionally, because of differences in the way that timing of diagnosis was measured for each condition, the predictive ability of factors for ADHD and autism/Asperger’s may not be directly comparable to the predictive ability for depression.

A limitation of this work is that external convergent validity was not demonstrated as there was no comparison between parent-reported SDQ with other reporters such as teachers or cohort members. Cohort member SDQ would have been the preferred informant given research that has demonstrated the utility in self-reported mental health compared to parent-reported [438, 439]. However, in the MCS self-completed SDQ’s were not available at age 11 meaning this was not possible. Although similarity

of measures has been demonstrated across informants previously in a UK sample for the SDQ [400], the validity of the current work would be improved with comparison to alternative informants, and caution would be necessary if adopting SDQ reported by other informants.

A weakness of this analysis was that listwise deletion was used instead of pairwise deletion, meaning there may have been a loss of statistical power. There is the possibility that there is bias attributable to missing data. In Mplus, WLSMV estimation is used for ordinal categorical items, which allows missing data to be a function of the observed covariates but not the observed outcomes [440], and WLSMV has been found to produce consistent estimates under different missing data assumptions [219]. Given missing data for the majority of items was <1% for those that at least partially completed the SDQ, this is unlikely to impact estimates [429].

#### 7.4.5 Conclusion

Previous theory and empirical findings provide strong conceptual reasoning to adopt internalising and externalising constructs in the analysis of child psychopathology. Additionally, previous scale validation literature recommended the use of internalising and externalising scales in general population samples [400, 405]. The current CFA demonstrated an acceptable fit for a second-order model, along with better internal convergent, external discriminant and predictive validity than the five-factor model. Overall, the results indicate that the parent-report SDQ appropriately measures internalising and externalising symptoms in the MCS. Therefore, the internalising and externalising second-order factor model were selected as the primary measures to use when testing mediating pathways between SEP and body composition.

# Chapter 8 Investigating Area Deprivation and Individual Characteristic Interactions to Understand Inequalities in Body Mass Index and Body Composition

## 8.1 Background

This chapter uses the results of the CFA in the previous chapter, to explore potential mediation pathways between SEP and body composition. Internalising and externalising symptoms are firstly treated as mediators between family-level SEP and body composition and BMI. It is hypothesised that disadvantaged individual SEP is associated with greater levels of internalising and externalising symptoms, and this in turn is related to less healthy body composition. It is then hypothesised that the mediating pathway via internalising and externalising symptoms is moderated, and that mediation by symptoms is stronger among individuals living in more obesogenic environments. This thesis adopts a broad understanding of the obesogenic environment that includes the tangible and built environment, such as green spaces and fast-food outlet density, alongside area-level measures of deprivation, which may better capture the social, cultural and economic environment. Additionally, a less commonly studied measure of the environment - the density of domestic gardens - is adopted as gardens may better reflect environments used by cohort members, especially in urban settings [441].

The theory of affective states is applied to understand how environmental conditions may moderate associations between emotional and behavioural problems and body composition. It is hypothesised that the environment influences the coping strategies of individuals to manage their psychological symptoms. For example, individuals experiencing internalising symptoms in an area with high density of fast-food outlets may have greater stimulus and opportunity to regulate these emotions with consumption of unhealthy foods.

This chapter therefore has two aims: 1) To test whether internalising and externalising symptoms mediate the relationship between individual SEP and body composition and BMI; 2) To identify if the environment influences the degree to which internalising and externalising symptoms mediate associations between SEP and body composition and BMI by testing conditional indirect effects. Two hypotheses are tested to achieve these aims:

**Hypothesis 9:** *Social inequalities in body composition (and BMI) among children and adolescents in the UK are mediated by internalising and externalising symptoms, so that those in disadvantaged circumstances exhibit higher levels of these symptoms, which in turn are related to less healthy body composition (higher FM and lower FFM).*

**Hypothesis 10:** *Environmental characteristics modify the mediating effect of internalising and externalising symptoms among children and adolescents in the UK, such that those experiencing a more obesogenic environment show greater mediation by internalising and externalising symptoms, such that the association between symptoms and less healthy body composition is strongest in the most obesogenic areas.*

## 8.2 Methods

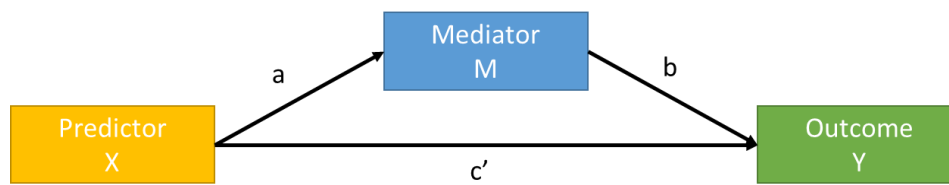
### 8.2.1 Structural Equation Modelling

SEMs are an expansion of general linear models that allow several regression paths to be tested simultaneously and which may include both observed and unobserved variables [442]. Such unobserved variables are latent factors, as determined through CFA. SEMs can be thought of as a type of path analysis [442], but has the advantage of being able to test models that have many complexities, such as those including mediators and moderators, as well as allowing for sub-group analysis. By including latent variables in SEMs, reliability of measures is improved as the measurement error associated with observed variables is reduced. Therefore, SEM provides more appropriate tests of relationships between variables, as constructs are better measured.

### 8.2.2 Mediation Models

Mediation is a type of statistical modelling that aims to explain why a relationship exists between a predictor (X) and outcome (Y) and hypothesises a third mediating variable (M) that is on the pathway between X and Y (Figure 8-1). Traditionally, the method proposed by Baron and Kenny used a four step model that individually tested each of the regression paths, and deduced mediation by the change in strength of relationship when the mediator is included in the model compared to when it is omitted [443]. However, this approach has a number of flaws. Firstly, the Baron and Kenny method relies on multiple hypotheses to test mediation which is more likely to result in type 1 errors in the decisions made in building the mediation models, and, secondly, it does not offer a single test for the indirect effect via the mediator [444]. Further, mediation can still exist even if paths a and b (Figure 8-1), or the total effect of X on Y, are not statistically significant. However, the Baron and Kenny Method requires a statistically significant association between X and Y [444]. In cases of “inconsistent mediation” the sign of the mediating path may be in the opposite to the direct effect, in which case no total effect is observed. Additionally, the Baron and Kenny Method struggles to identify small mediating effects, especially with smaller sample sizes [444]. The method is also not easily extendable to more complex analysis, such as sub-group analysis.

**Figure 8-1.** Diagram of Basic Mediation



**Figure 8-1 Footnote:** Diagram showing mediation. Regression path *a* represents the path from the predictor (*X*) to mediator (*M*), regression path *b* represents the path from mediator (*M*) to outcome (*Y*), and path *c'* represents the direct effect between *X* and *Y*. The indirect path is represented  $a*b$ , and therefore the total effect (*c*) is represented by the equation  $c = a*b + c'$ .

To avoid these limitations, in SEMs a single test for the indirect effect can be used. The indirect effect is the multiplied effect of the regression path from the predictor to the mediator (*a*) and from the mediator to the outcome (*b*) (Figure 8-1). The total effect (*c*) is the direct effect from the predictor to the outcome (*c'*) plus the indirect effect through the mediator ( $a*b$ ). The indirect effect of *X* on *Y* can be derived from the regression equation for the outcome (*Y*) and the regression equation for the Mediator (*M*), as shown in the equation below. The indirect effect ( $a*b$ ) is the part of the change in *Y* that is caused by a one unit increase in *X* that is caused by the effect of *X* on *M*, and the resulting effect of *M* on *Y* [445].

Equation 1 - Regression Equation for *Y*:

$$Y = i_1 + bM + c'X$$

Where  $i_1$  is the intercept for *Y*, and  $bM$  is the slope for the mediator, and  $c'X$  is the direct slope for *X*.

Equation 2 - Regression Equation for *M*:

$$M = i_2 + aX$$

Where  $i_2$  is the intercept for the mediator, and  $aX$  is the slope for *X*.

Substituting equation 2 (*M*) in equation 1 (*Y*) gives the total effect:

$$Y = i_1 + b(i_2 + aX) + c'X$$

Multiply out the brackets, this is equivalent to:

$$Y = i_1 + bi_2 + (a*b)X + c'X$$

Re-arrange to standard linear regression form, predicting *Y* from *X*:

$$Y = (i_1 + bi_2) + (a*b+c')X$$

Therefore, the total effect of *X* on *Y* is:

$$a*b+c'$$

The total effect (*c*) of *X* on *Y* is made up of the indirect effect and direct effect (*c'*):

$$C = \text{indirect} + c'$$

Therefore:

$$C = a*b + c'$$

$$\text{Indirect effect} = a*b$$

There are a number of causal assumptions, with three key assumptions that need to be met for the causal model to be valid. The first assumption is that the mediator causes the outcome, instead of there being reverse causality [446]. This can be addressed either in the design, if the mediator is measured temporally before the outcome, or by using cross-lagged models to test the hypothesised mediating path and also the reverse. The second assumption known as sequential ignorability stipulates that there must be no unmeasured confounders that cause both the mediator and the outcome and therefore need to be controlled for in the model [447]. The final assumption is that of no measurement error in the mediator, which can be addressed through use of a latent variable measured by multiple observed variables [446].

#### *8.2.2.1 Analytic Approach*

Three mediation models were tested for each of the body composition variables and for BMI at age 17, using the second-order factors with correlated errors at age 11 as mediating latent variables (based on the results of Chapter 7), and household income at age 7 as a marker of family-level SEP (Figure 8-2):

**Model 1** – Inequalities in body composition/BMI mediated by internalising symptoms.

**Model 2** - Inequalities in body composition/BMI mediated by externalising symptoms.

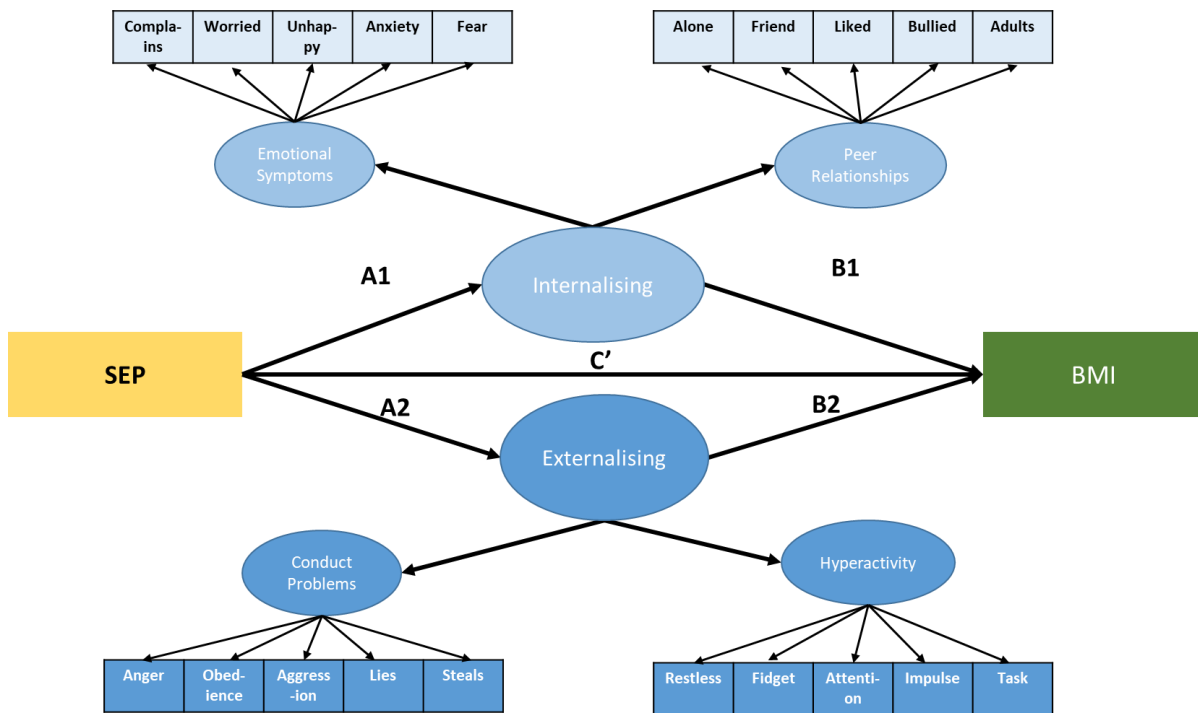
**Model 3** - Inequalities in body composition/BMI mediated by externalising and internalising symptoms.

In each model, linear regression was used to calculate estimates for the direct and indirect paths. Mediation was tested using the model indirect function in Mplus version 8.5. The Weighted Least Squares, Mean and Variance adjusted (WLSMV) estimator was used, to allow the first-order factors to be estimated. Using WLSMV was necessary as the observed indicator variables were ordinal variables. In all models, age of cohort members at sweep 7, sex of the cohort member, stratification characteristics (stratum) and ethnicity were included as covariates. Analysis was weighted using the non-response and sampling weights from the age 17 sweep, therefore accounting for attrition.

For models using FFMI as the outcome of interest, models were also run adjusting for FMI to account for the strong correlation between FM and FFM as a result of adaptive increases in FFM with greater FM. Because there is possibility that FM may actually be a mediator, not a confounder in associations, a sensitivity analysis was conducted treating FMI as an additional mediator.



**Figure 8-2.** Conceptual Model of Mediation Between SEP and Body Size.



**Figure 8-2 Footnote.** Figure shows the hypothesised mediation Model 3 from SEP to BMI, mediated by the second-order latent variables of internalising and externalising symptoms. Path  $A1*B1$  represents the specific indirect effect for internalising symptoms. Path  $A2*B2$  represents the specific indirect effect for externalising symptoms.  $C'$  represent the direct effect from SEP to body size. The total effect represented by  $C$  (not shown in diagram) is equal to  $A1*B1 + A2*B2 + C'$ . Not shown in the diagram, but included in the statistical model, are covariates (age, sex, ethnicity, stratification characteristics) and correlation between internalising and externalising symptoms.

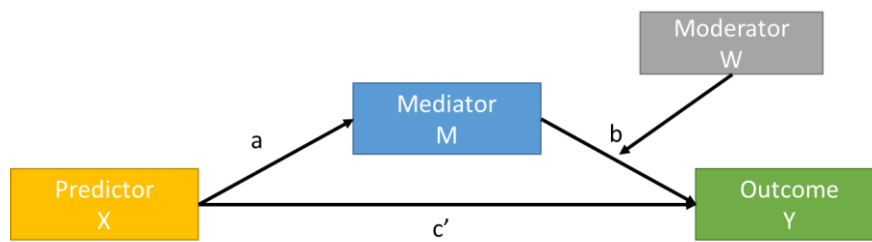
### 8.2.3 Moderated Mediation Models

Moderated mediation is where both mediation and moderation occur [444]. A moderating variable changes the relationship between  $X$  and  $Y$  but is not on the pathway between  $X$  and  $Y$ . For example, the relationship between  $X$  and  $Y$  may be different between males and females, in which case sex is a moderating variable.

In moderated mediation, the path between the predictor and the mediator or between the mediator and outcome is moderated by an additional variable (Figure 8-3). However, it is not possible to represent this conceptual model statistically or computationally. In statistical models this is represented by a path from the moderator to the outcome, and a path from a new variable that is a multiplication of the moderator with mediator, that is also directed to the outcome (Figure 8-4). To calculate the moderated mediating effect, also known as the conditional indirect effect, because the indirect effect is conditional on the level of an additional variable, the equation below is applied.

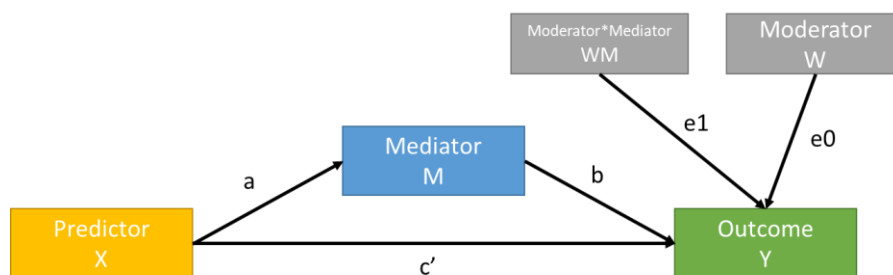
$$\text{Conditional Indirect Effect} = a1*(b+e1*W)$$

**Figure 8-3.** Conceptual Diagram of Moderated Mediation



**Figure 8-3 footnote:** Conceptual diagram demonstrating moderated mediation. Moderator variable ( $W$ ) moderates regression path  $b$  from mediator ( $M$ ) to outcome ( $Y$ ).

**Figure 8-4.** Statistical Diagram of Moderated Mediation



**Figure 8-4 Footnote:** Statistical diagram demonstrating moderated mediation. Moderator variable ( $W$ ) moderates regression path  $b$  from mediator ( $M$ ) to outcome ( $Y$ ). To represent moderation of a path, which is not statistically or computationally plausible, an interaction term is included between the moderator and mediator, and the regression path with the outcome is estimated ( $e1$ ). A regression path from the moderator to outcome is also estimated ( $e0$ ).

### 8.2.3.1 Analytic Approach

In this thesis, interest was in assessing whether a moderating effect of the environment on the mediating pathway from SEP to body composition and BMI, via internalising and externalising symptoms, could be observed. A number of problems arise in estimating the moderated effect of the environment. Using the WLSMV estimator, it is not possible to test interactions with latent variables in Mplus. The maximum likelihood with robust standard errors (MLR) estimator with random slopes and numerical integration for estimation of parameters, can be used to test interactions with latent and observed variables. However, the MLR estimator is not compatible with correlations between errors when the items are categorical, as they are for the SDQ. Additionally, numerical integration is very computationally demanding when MLR is used with categorical items, and where there is a large sample size and number of factors [440]. It is therefore not possible to run latent interactions using the factor structure identified in Chapter 7.

There are two alternative approaches that allow for moderated mediation to be investigated through a triangulation of evidence. The first is to test mediation in each sub-group and compare the mediation between groups. The second is to treat the SDQ items as continuous and fit an interaction term for environmental conditions and psychological symptoms. By using both approaches, moderated

mediation can first be inferred from observed differences between groups, and then interaction terms when items are treated as continuous can be used as a proxy for testing differences in conditional indirect paths between groups.

### Sub-Group Analysis

The indirect effect of SEP on body composition in each sub-group of IMD, green spaces, domestic gardens and fast-food density was estimated, using the categorical variables of each area-level variable (high, medium, low) to maintain sample size and power. Because information about domestic gardens was only collected in England, and fast-food density was only collected in Great Britain, analysis in these sub-groups was restricted to those residing in England and Great Britain at the time of data collection, respectively.

Mediation within each group was tested using the model indirect function in Mplus, adopting the WLSMV estimator with multiple imputation. The same mediation equations used previously (Section 8.2.2) were applied. Similar to the mediation model, analysis was weighted using the non-response and sampling weights at age 17 and for each sub-group covariates age, sex, ethnicity and stratification characteristics (stratum) were included. For some environmental sub-groups, one or more of the dummy variable stratum were removed from the models due to their being 0 or 1 observations within them, meaning the variable had no variance.

### Interaction Models

To maintain the use of the second-order factor structure identified in Chapter 7, an interaction model was run using the MLR estimator where the SDQ ordinal items were treated as continuous. A sensitivity analysis was conducted where mediation model 3 from Section 8.2.2.1, above, was run with continuous items, in order to demonstrate the similarity of estimates when treating items as continuous.

A latent interaction was created between internalising and externalising symptoms and dummy variables for the categorical environmental characteristics with low deprivation or density as the reference category. The latent factors were standardised by setting the variances of both second-order factors equal to one and freeing up the first-order factor loadings. Model fit was assessed for the standardised factors, using the same fit indices outlined in Chapter 7 (Section 7.2.1, pg. 147).

In this model the conditional indirect effect for internalising and externalising symptoms are represented by the equations below, where “e” represents the path from the interaction to the outcome, and “environment” indicates the level of the moderator (i.e high, medium, low) (Figure 8-5):

$$\text{Internalising} = a1*(b1+e1*\text{environment})$$

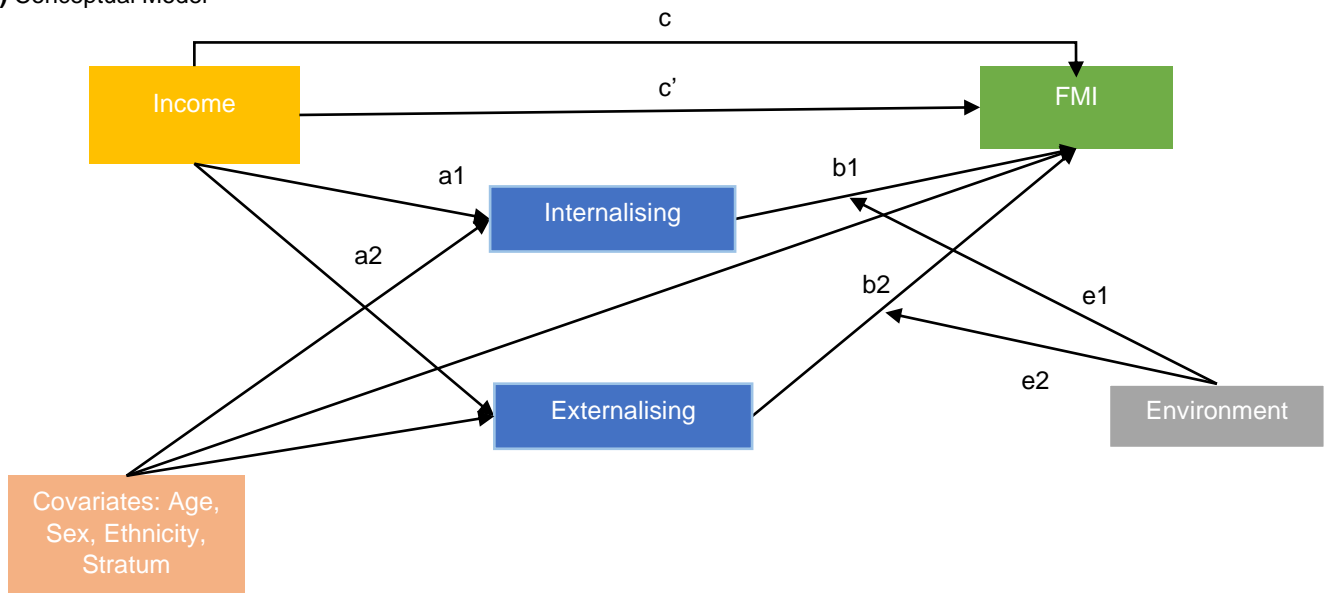
$$\text{externalising} = a2*(b2+e2*\text{environment})$$

Therefore, three mediating paths were estimated from X to Y that are conditional on the levels of the moderator. Differences between these paths can be calculated, and moderated mediation is demonstrated by a significant interaction term and significant difference between paths. The p-value

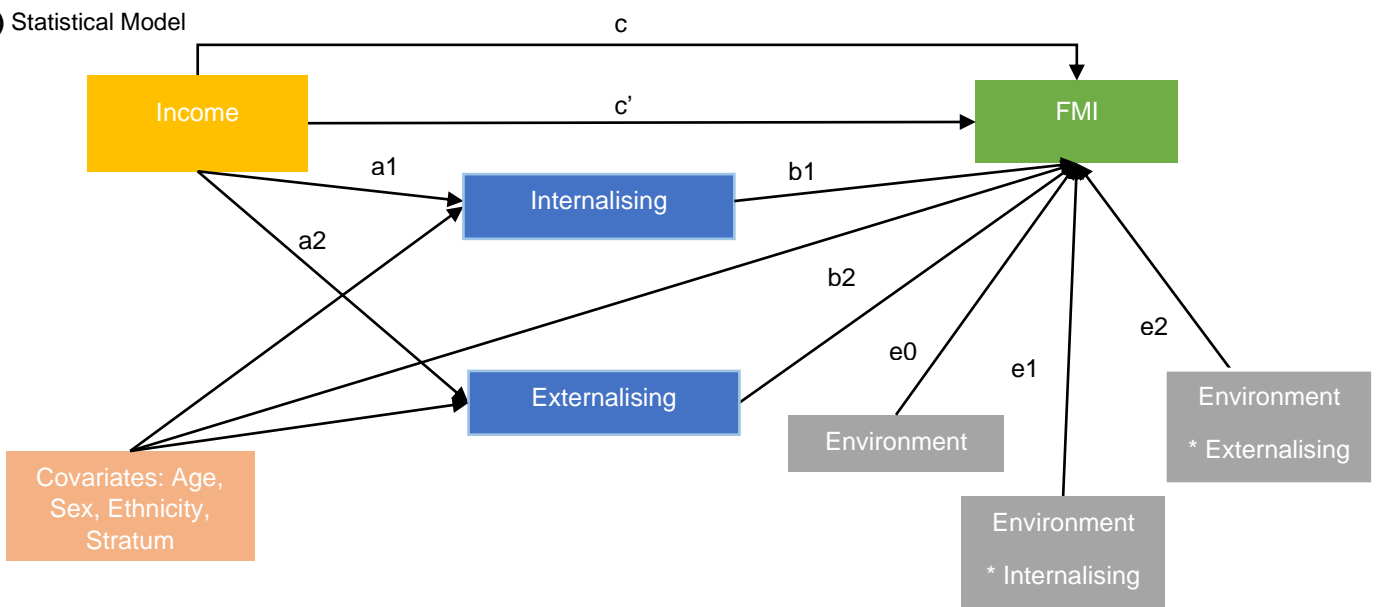
for the interaction term would be the same, or similar, to the p-value for the difference between conditional indirect paths. Similar to previous analysis, models were adjusted for age, sex, ethnicity and stratum and weighted estimates were calculated.

**Figure 8-5.** Conceptual and Statistical Models for Testing Conditional Indirect Effects

**a) Conceptual Model**



**b) Statistical Model**



**Figure 8-5 Footnote:** Conceptual (figure a) and statistical (figure b) models showing moderated mediation for fat mass index (FMI). Regression path c represents the total effect, regression path a represents the path from the predictor to mediator, regression path b represents the path from mediator to outcome, and path c' represents the direct effect. In both figures, internalising and externalising symptoms are mediating associations between income and FMI. In figure a) the environment moderates regression paths b1 and b2. However, this is not possible to represent statistically or computationally, so in figure b), an interaction term is included between the environment and both mediators. These are included as predictors in regression equations for outcome FMI alongside the environment variable. Moderated mediation is therefore represented by the equations:  $Internalising = a1*(b1+e1*environment)$ ; and  $externalising = a2*(b2+e2*environment)$ .

## 8.2.4 Missing Data Approaches

### *8.2.4.1 Weighted Least Squares Estimation and Multiple Imputation*

WLSMV is adopted when observed variables are categorical and non-normally distributed, and it allows missing data to be a function of the observed covariates but not the observed outcomes [440]. WLSMV has been found to produce consistent estimates under different missing data assumptions, in particular MARX [219]. However, WLSMV does not hold under all missing data assumptions, including the more general MAR. It is possible to combine the WLSMV estimator with other missing data methods, including multiple imputation, to provide reliable estimates under MAR assumptions [219].

As the percentage of missing data exceeded 5% on income (7%) and SDQ items (16%) among those with data on body composition and BMI at age 17, multiple imputation was adopted for these variables to maintain sample size under the assumption of MAR. A total of 15 imputed data sets were obtained, and Rubin's rule was used to combine the point estimates from regression models from each of the 15 datasets. Auxiliary variables (NS-SEC sweep 4, NVQ sweep 4, IMD sweep 4, partners BMI, ever breast fed, longstanding illness, self-rated financial difficulty, and main respondent's experience of depression, smoking status and alcohol consumption) were added to the model to improve predictions.

### *8.2.4.2 Full-Information Maximum Likelihood*

Full Information maximum likelihood (FIML) is another method of parameter estimation used in SEM in the presence of missing data, that is adopted when variables are continuous and is robust to non-normally distributed observed variables. Under the assumptions of MAR, MCAR and MNAR, FIML is able to produce consistent estimates that are comparable to those calculated using multiple imputation. FIML estimates are also asymptotically efficient [448]. For FIML, missingness is not allowed for exogenous covariates, and as such, no distributional assumptions are made about these covariates in the models [440]. Missingness can be addressed in covariates if they are brought into the model by including their variances, in which case distributional assumptions are made about them [440]. FIML is able to estimate parameters even within the presence of missing data by constructing a likelihood function. In FIML this is done using the likelihood products of both complete and incomplete cases, making full use of available data. The overall likelihood is optimised and used to calculate the model estimates. For models including the interaction tests, FIML estimation was adopted in this thesis using the MLR estimator.

## 8.2.5 Analytic Sample

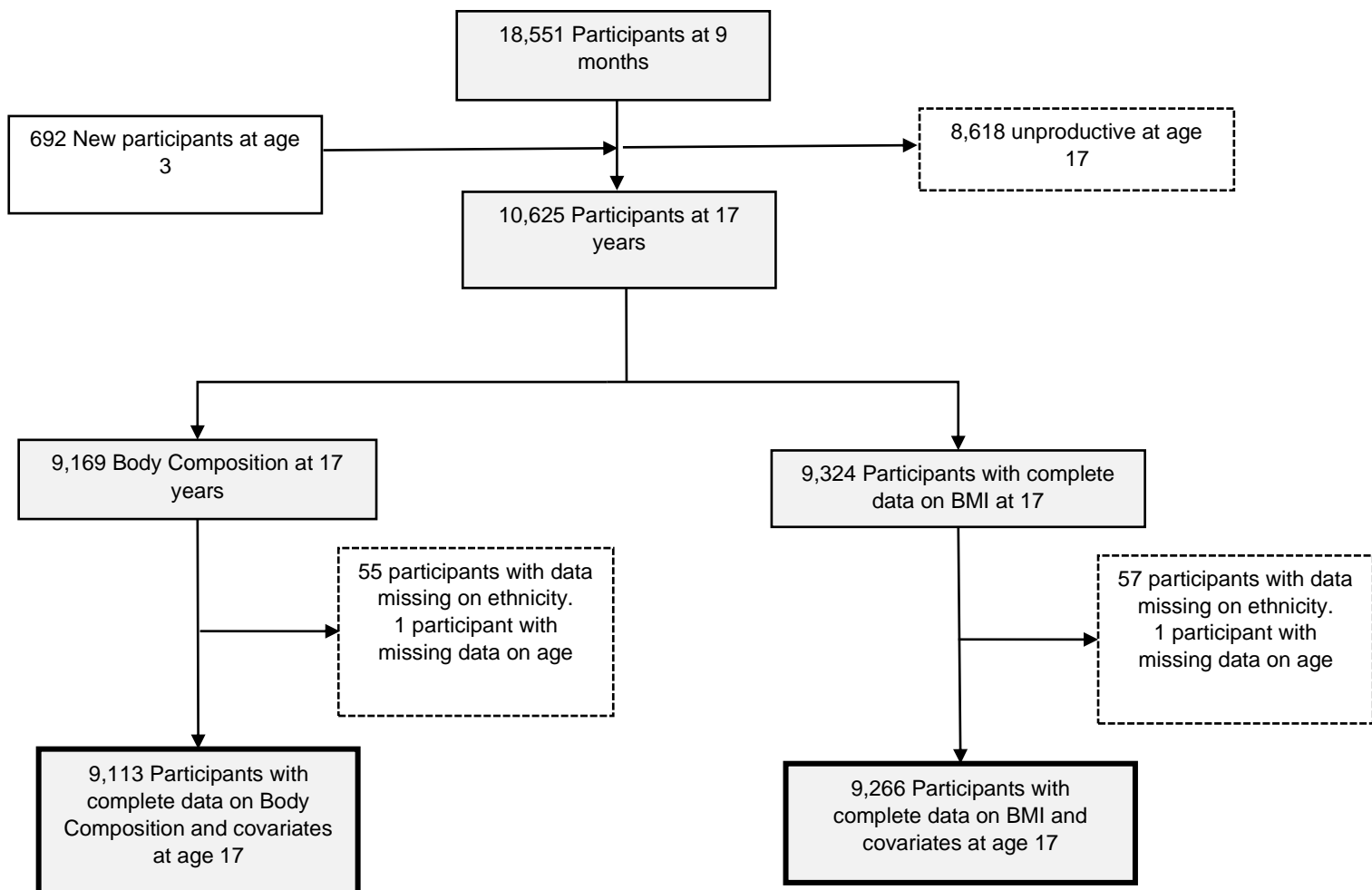
For mediation models, the analysis was limited to those with complete data on BMI or body composition and covariates, with imputation for SDQ items and income (Figure 8-6). Therefore, sample size for mediation analysis with body composition as the outcome was 9,113 and for BMI it was 9,266.

For sub-group analysis, samples were further limited to those with complete data on each environmental characteristic, and for the case of domestic gardens and fast-food density analysis

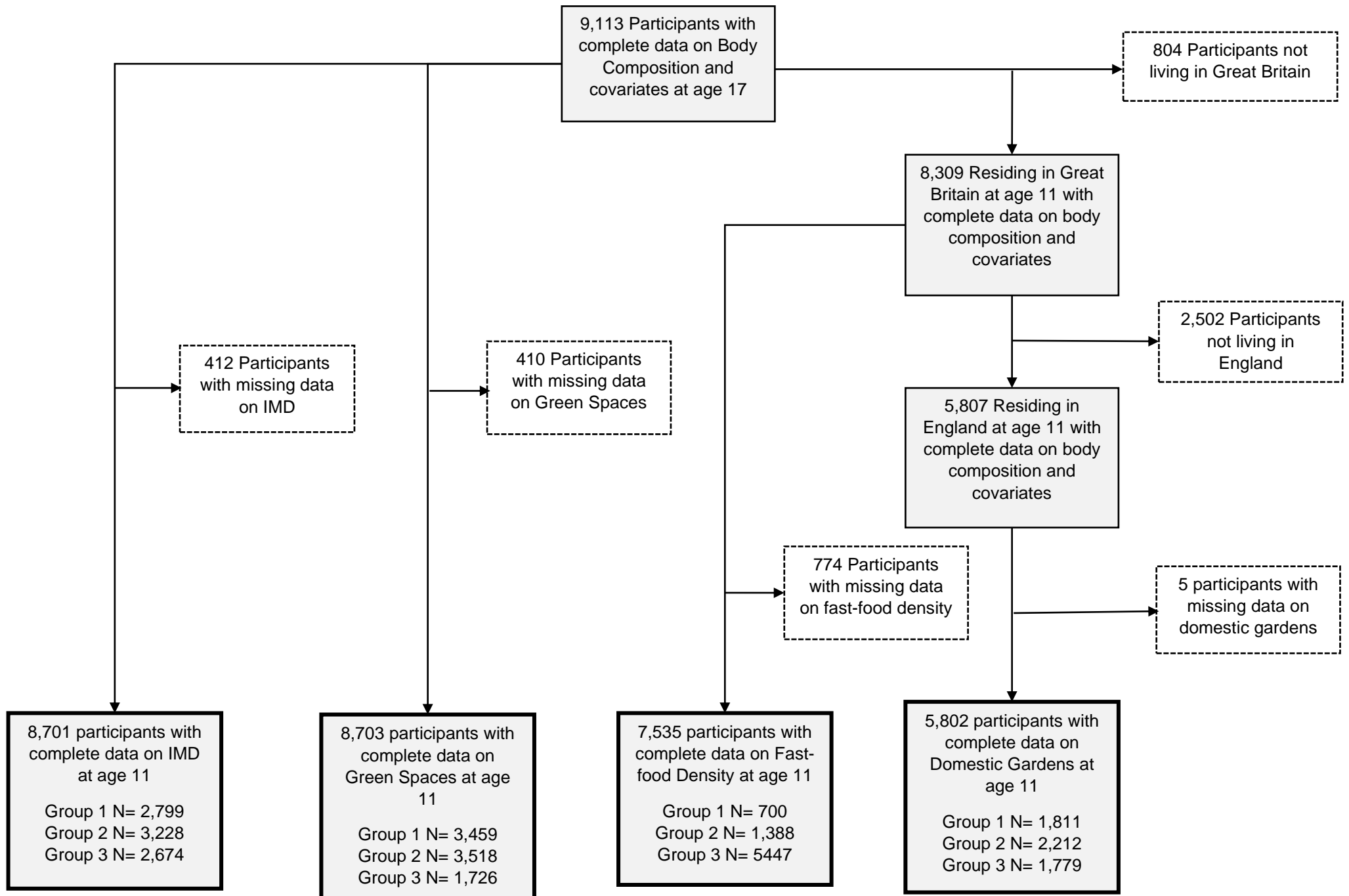
were also limited to England and Great Britain respectively (Figure 8-7, Appendix A8.1). As such, sample sizes were: IMD N=8,701 (BMI N=8,840); green spaces N=8,703 (BMI N=8,842); domestic gardens N=5,802 (BMI N=5,894); fast-food density N=7,535 (BMI N=7,657).

For interaction models, FIML estimation was applied using the MLR estimator. Because missingness is conditional on observed covariates, sample sizes were limited to those with complete data on all independent variables (income) and covariates (sex, age and ethnicity), and those with data available on dependent variables (body composition and SDQ items). For these models sample sizes for body composition were 9,228 (BMI N= 9,230) for IMD and green spaces, 6,100 for domestic gardens (BMI N= 6,102), 7,957 for fast-food density (BMI N= 7,959). Because FMI is included as a covariate in adjusted models for FFMI, the variance of FMI was also included in the model to maintain sample size and comparability of results.

**Figure 8-6.** Analytic Sample for Mediation Analysis



**Figure 8-7.** Analytic Sample for Body Composition Sub-Group Analysis by Environmental Characteristics



## 8.3 Results

### 8.3.1 Descriptive Results

Differences in mean body composition by key demographic variables, including the IMD, were provided in Chapter 6 (Section 6.3.1, pg. 124). Table 8-1 shows mean BMI by sociodemographic variables. Differences by ethnicity for BMI were similar to that of FMI and FM:FFM ratio with those of Black and Black British ethnicity typically having higher mean BMI, but those of Indian ethnicity typically had the lowest BMI. The difference in mean BMI by income and IMD was also similar to those seen for FMI and FM:FFM ratio, where those in the most deprived areas had a higher mean BMI which declined with increasing advantage.

Differences in mean body composition and BMI by environmental characteristics are shown in Table 8-2. For the IMD measured at sweep 5, mean FMI, FFMI, FM:FFM ratio and BMI was highest in the most deprived group and lowest in the least deprived group. For green spaces, mean FMI was higher in areas with less green space and lower in areas with more green space. For FFMI, the opposite patterns were seen, where higher levels of FFMI were observed in the greenest areas. Mean FM:FFM ratio and BMI were similar across differing levels of green spaces. Mean FMI and BMI were greater in the medium group for domestic gardens, whilst mean FFMI and FM:FFM ratio were similar across domestic garden groups. There was little difference in means for FMI, FFMI, FM:FFM ratio and BMI across groups for fast-food density.

Pairwise correlations were calculated using the continuous measure of fast-food density and using deciles of green spaces, IMD and domestic gardens and are shown in Table 8-3. All measures were correlated with all other measures. However, the size of the correlations was negligible (absolute values of  $\rho$ :  $0 < \rho \leq 0.1$ ) between domestic gardens and the IMD and domestic gardens and fast-food density, whilst the remaining correlations were weak to moderate (weak: absolute values  $0.1 < \rho < 0.4$ , moderate:  $0.4 \leq \rho < 0.7$  [449]). Green spaces and higher IMD values indicating less deprivation were weakly positively correlated. Higher values of the IMD indicating less deprivation and fast-food density were negatively correlated, meaning that lower area deprivation was associated with lower fast-food density. Domestic gardens and green spaces were also negatively correlated, where areas with higher density of domestic gardens had lower density of green spaces.



**Table 8-1.** Mean Body Mass Index according to Ethnicity, Index of Multiple Deprivation and Income

	<b>Body Mass Index (BMI)</b>											
	<b>Age 7</b>			<b>Age 11</b>			<b>Age 14</b>			<b>Age 17</b>		
	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>
<b>Ethnicity</b>			13,536			12,783			10,900			9,267
<i>White</i>	13.91	1.75	11362	12.65	2.22	10590	16.82	3.52	8945	23.95	4.84	7497
<i>Mixed</i>	13.97	2.03	370	12.83	2.41	365	17.19	3.86	304	25.34	6.59	273
<i>Indian</i>	13.54	2.25	337	12.34	2.39	329	16.41	3.60	301	22.92	5.66	278
<i>Pakistani and Bangladeshi</i>	13.67	2.73	849	12.90	3.04	906	17.22	5.14	828	24.13	7.37	735
<i>Black and Black British</i>	14.38	2.39	442	13.09	2.55	413	17.34	3.64	351	26.34	5.89	325
<i>Other Ethnic Group</i>	13.54	1.99	176	12.32	2.24	180	17.21	3.35	171	24.32	5.83	159
<i>P value</i>			<0.001			0.001			0.03			0.002
<b>IMD</b>			13,634			12,871			10,968			9,324
<i>Most Deprived 20%</i>	14.12	2.32	4050	12.97	2.64	3913	17.32	4.00	3216	24.80	6.47	2672
<i>40%</i>	13.97	1.99	3063	12.82	2.42	2845	17.01	3.79	2433	24.53	5.62	2046
<i>60%</i>	13.88	1.64	2384	12.65	2.21	2248	16.89	3.47	1918	23.86	4.61	1631
<i>80%</i>	13.76	1.52	1969	12.45	1.96	1859	16.58	3.29	1620	23.49	4.31	1415
<i>Least Deprived 20%</i>	13.70	1.55	2168	12.26	1.90	2006	16.17	3.12	1781	23.46	4.06	1560
<i>P value</i>			<0.001			<0.001			<0.001			<0.001
<b>Income</b>			13,407			12,648			10,790			9,186
<i>Lowest Quintile</i>	14.07	2.06	2970	12.95	2.34	2864	17.40	3.60	2276	25.16	5.81	1827
<i>2<sup>nd</sup> Quintile</i>	14.03	1.96	2884	12.87	2.36	2711	17.21	3.79	2292	24.70	6.08	1902
<i>3<sup>rd</sup> Quintile</i>	13.95	1.79	2597	12.72	2.42	2402	16.75	3.46	2056	23.89	4.66	1749
<i>4<sup>th</sup> Quintile</i>	13.86	1.72	2557	12.57	2.17	2404	16.62	3.54	2115	23.75	4.87	1842
<i>Highest Quintile</i>	13.57	1.59	2399	12.10	1.95	2267	15.99	3.28	2051	22.97	3.88	1866
<i>P value</i>			<0.001			<0.001			<0.001			<0.001

**Table 8-1 Footnote:** *p-value for Adjusted Wald Test.*

**Table 8-2.** Mean Body Composition and Body Mass Index by Environmental Characteristics

	<b>FMI</b>		<b>FFMI</b>		<b>FM: FFM</b>		<b>BMI</b>	
	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>
<b>Index of Multiple Deprivation</b>								
<i>High Deprivation</i>	4.32	3.38	20.32	3.30	0.34	0.23	24.75	6.40
<i>Medium</i>	3.83	2.40	20.12	2.87	0.31	0.18	23.95	4.93
<i>Low Deprivation</i>	3.43	1.95	20.04	2.43	0.28	0.15	23.24	3.90
<i>P Value</i>	<0.001		0.05		<0.001		<0.001	
<b>Green Spaces</b>								
<i>Low Green Spaces</i>	3.89	2.78	20.07	2.91	0.31	0.19	23.91	5.26
<i>Medium</i>	3.82	2.43	20.11	2.80	0.30	0.18	23.91	4.87
<i>High Green Spaces</i>	3.68	2.26	20.34	2.72	0.30	0.18	23.88	4.73
<i>P Value</i>	0.24		0.11		0.87		0.99	
<b>Domestic Gardens</b>								
<i>Low Domestic Gardens</i>	3.68	2.16	20.18	2.63	0.30	0.17	23.76	4.37
<i>Medium</i>	3.93	2.75	20.15	2.98	0.31	0.20	24.09	5.37
<i>High Domestic Gardens</i>	3.75	2.52	19.95	2.87	0.30	0.18	23.59	4.98
<i>P Value</i>	0.07		0.25		0.19		0.03	
<b>Fast-Food Density</b>								
<i>Low Fast-Food Density</i>	3.78	2.44	20.17	2.79	0.30	0.18	23.90	4.90
<i>Medium</i>	3.86	2.56	20.07	2.96	0.31	0.19	23.89	5.20
<i>High Fast-Food Density</i>	4.09	3.41	20.08	3.20	0.31	0.22	24.13	6.00
<i>P Value</i>	0.41		0.64		0.41		0.80	

**Table 8-2 Footnote:** Mean body composition and BMI by environmental characteristics at sweep 5. P-values for Adjusted Wald Test.

**Table 8-3.** Correlation Matrix of Environmental Characteristics

	<b>IMD</b>	<b>Green Spaces</b>	<b>Domestic Gardens</b>	<b>Fast-food Density</b>
<b>IMD</b>	1.00			
<i>P Value</i>	-			
<b>Green Spaces</b>	0.37	1.00		
<i>P Value</i>	<0.001	-		
<b>Domestic Gardens</b>	0.02	-0.43	1.00	
<i>P Value</i>	0.06	<0.001	-	
<b>Fast-food Density</b>	-0.34	-0.37	0.09	1.00
<i>P Value</i>	<0.001	<0.001	<0.001	-

**Table 8-3 Footnote:** Pairwise correlation between environmental variables. P-values for persons correlation ( $\rho$ ). Abbreviations: IMD – Index of Multiple Deprivation.

## 8.3.2 Mediation

### 8.3.2.1 Models 1 and 2: Separate Mediation

When including internalising and externalising symptoms in separate mediation models (models 1 & 2, Table 8-4), a negative indirect effect between income and all of FMI, FFMI, FM:FFM ratio and BMI was observed in both models. In other words, higher income was related to lower level of internalising and externalising symptoms, and lower level of symptoms were associated with a lower levels of FMI, FFMI, FM:FFM and BMI. Conversely, lower income was associated with higher levels of internalising and externalising symptoms, higher levels of which were associated with higher levels of FMI, FFMI, FM:FFM and BMI.

When additionally adjusted for FMI, there was no indirect effect via internalising symptoms between income and FFMI, but a negative indirect effect was observed for externalising symptoms in the same direction as that for the other measures of body composition.

### 8.3.2.2 Model 3: Combined Mediation

When both internalising and externalising symptoms were included together, only internalising symptoms mediated the associations between income and FMI and FM:FFM ratio (model 3, Table 8-5). The standardised direct effect between income and FMI suggests that a one SD increase in income was associated with a -0.035 (95% CI: -0.084 to 0.013) SD decline in FMI. The indirect effect is  $\beta$  -0.047 (95% CI: -0.09 to -0.003), suggesting partial mediation by internalising symptoms. The total effect is equivalent to a -0.079 SD (95% CI: -0.133 to -0.026) change in FMI per one SD increase in income. Similar levels of mediation were observed for FM:FFM ratio, where the indirect effect accounts for over half of the total effect (Table 8-5). For BMI, the combined indirect effect of both internalising and externalising symptoms ( $\beta$  -0.049, 95% CI: -0.077 to -0.021) was roughly half of the total effect ( $\beta$  -0.098, 95% CI: -0.147 to -0.048), but neither symptom demonstrated an individual mediating pathway, as indicated by statistically non-significant paths.

There was no evidence of a direct effect between income and FFMI ( $\beta$  -0.006, 95% CI: -0.036 to 0.025), whilst there was an observed indirect effect for internalising symptoms and externalising symptoms combined ( $\beta$  -0.035, 95% CI: -0.055 to -0.015) but neither symptom demonstrated an individual indirect effect. Lower income was related to higher symptoms, which in turn were related to higher FFMI. This resulted in a total effect between income and FFMI where a one SD increase in income was associated with -0.04 SD change in FFMI (95% CI: -0.076 to -0.005).

When adjusted for FMI, there was no longer a direct effect between income and FFMI ( $\beta$  0.011, 95% CI: -0.01 to 0.032), but there was slight evidence of an indirect effect for externalising symptoms ( $\beta$  -0.024, 95% CI: -0.051 to 0.004) and evidence of a total indirect effect for internalising and externalising symptoms combined ( $\beta$  -0.013, 95% CI: -0.026 to 0.00). However, there was no overall association between income and FFMI as the signs of the direct effect and total indirect effect were in the opposite directions, resulting in inconsistent mediation.

### 8.3.2.3 Sensitivity Analysis

In the sensitivity analysis treating FMI as a mediator not a confounder (Appendix A8.2), there was a positive direct effect in models 1-3 (Model 3:  $\beta$  0.032, 95% CI: 0.01 to 0.055), where lower income was related to lower FFMI. This is contrary to the main model reported in this chapter. In the sensitivity analysis, the mediating paths for internalising and externalising symptoms were almost identical to FFMI models that made no adjustment for FMI. However, there was a negative indirect path through FMI in all models (Model 3:  $\beta$  -0.038, 95% CI: -0.064 to -0.012) where income was related to higher FMI which in turn was related to higher FFMI. This resulted in a negative total effect (Model 3:  $\beta$  -0.04, 95% CI: -0.076 to -0.005) in the opposite direction to the direct effect.

**Table 8-4.** Mediation of Socioeconomic Position on Body Composition and Body Size by Internalising Symptoms (Models 1 & 2).

Model	Estimate	Lower CI	Upper CI	P Value	Fit Statistics:					
					RMSEA	CFI	TFI	SRMR	$\chi^2$	df
<b>MODEL 1 - INTERNALISING SYMPTOMS</b>										
<b>FMI</b>										
Total	-0.079	-0.13	-0.026	0.004	0.01	0.953	0.94	0.085	350.76	180
Indirect	-0.042	-0.064	-0.022	<0.001	-	-	-	-	-	-
Direct	-0.037	-0.089	0.017	0.16	-	-	-	-	-	-
<b>FFMI</b>										
Total	-0.04	-0.076	-0.005	0.027	0.01	0.961	0.95	0.084	355.63	180
Indirect	-0.025	-0.041	-0.009	0.002	-	-	-	-	-	-
Direct	-0.015	-0.049	0.019	0.39	-	-	-	-	-	-
<b>FFMI adjusted for FMI</b>										
Total	-0.002	-0.024	0.020	0.84	0.01	0.98	0.974	0.08	365.73	189
Indirect	-0.004	-0.014	0.005	0.38	-	-	-	-	-	-
Direct	0.002	-0.019	0.024	0.85	-	-	-	-	-	-
<b>FM: FFM</b>										
Total	-0.074	-0.12	-0.031	0.001	0.01	0.957	0.945	0.082	353.87	180
Indirect	-0.033	-0.051	-0.014	0.001	-	-	-	-	-	-
Direct	-0.041	-0.083	0.001	0.056	-	-	-	-	-	-
<b>BMI</b>										
Total	-0.098	-0.15	-0.048	<0.001	0.01	0.952	0.938	0.124	362.52	180
Indirect	-0.043	-0.067	-0.019	<0.001	-	-	-	-	-	-
Direct	-0.055	-0.11	-0.004	0.033	-	-	-	-	-	-
<b>MODEL 2 - EXTERNALISING SYMPTOMS</b>										
<b>FMI</b>										
Total	-0.079	-0.133	-0.026	0.004	0.01	0.977	0.971	0.13	332.95	182
Indirect	-0.040	-0.066	-0.014	0.002	-	-	-	-	-	-
Direct	-0.039	-0.088	0.010	0.12	-	-	-	-	-	-
<b>FFMI</b>										
Total	-0.04	-0.076	-0.005	0.027	0.01	0.98	0.974	0.13	331.91	182
Indirect	-0.033	-0.053	-0.013	0.001	-	-	-	-	-	-
Direct	-0.007	-0.038	0.024	0.65	-	-	-	-	-	-
<b>FFMI adjusted for FMI</b>										
Total	-0.002	-0.024	0.020	0.84	0.009	0.986	0.982	0.123	345.83	191
Indirect	-0.013	-0.025	-0.001	0.036	-	-	-	-	-	-
Direct	0.011	-0.009	0.031	0.30	-	-	-	-	-	-
<b>FM: FFM</b>										
Total	-0.073	-0.12	-0.031	0.001	0.009	0.979	0.973	0.124	331.98	182
Indirect	-0.027	-0.049	-0.005	0.017	-	-	-	-	-	-
Direct	-0.046	-0.086	-0.007	0.022	-	-	-	-	-	-
<b>BMI</b>										
Total	-0.098	-0.15	-0.048	<0.001	0.009	0.978	0.972	0.146	333.72	182
Indirect	-0.045	-0.072	-0.019	0.001	-	-	-	-	-	-
Direct	-0.052	-0.098	-0.007	0.025	-	-	-	-	-	-

**Table 8-4 Footnote:** All models are adjusted for sex, age at sweep 7, ethnicity and stratification characteristics. Abbreviations:  $\chi^2$  – chi-squared; df – degrees of freedom; CFI – Comparative Fit Index; TLI – Tucker-Lewis Index; RMSEA – Root Mean Square Error of Approximation; SRMR – Standardized Root Mean Squared Residual.

**Table 8-5.** Mediation of Socioeconomic Position on Body Composition and Body Size by Internalising and Externalising Symptoms (Model 3).

Model	Estimate	Lower CI	Upper CI	P Value	Fit Statistics:					
					RMSEA	CFI	TFI	SRMR	$\chi^2$	df
<b>FMI</b>										
Total	-0.079	-0.13	-0.026	0.004	0.01	0.954	0.946	0.105	857.11	461
Total Indirect	-0.044	-0.072	-0.016	0.002	-	-	-	-	-	-
Specific Indirect - Internalising	-0.047	-0.09	-0.003	0.035	-	-	-	-	-	-
Specific Indirect - Externalising	0.003	-0.05	0.055	0.92	-	-	-	-	-	-
Direct	-0.035	-0.084	0.013	0.16	-	-	-	-	-	-
<b>FFMI</b>										
Total	-0.04	-0.076	-0.005	0.027	0.01	0.957	0.949	0.105	861.59	461
Total Indirect	-0.035	-0.055	-0.015	0.001	-	-	-	-	-	-
Specific Indirect - Internalising	-0.013	-0.049	0.024	0.49	-	-	-	-	-	-
Specific Indirect - Externalising	-0.022	-0.065	0.022	0.32	-	-	-	-	-	-
Direct	-0.006	-0.036	0.025	0.72	-	-	-	-	-	-
<b>FFMI adjusted for FMI</b>										
Total	-0.002	-0.024	0.02	0.84	0.01	0.965	0.959	0.101	885.39	479
Total Indirect	-0.013	-0.026	0.00	0.048	-	-	-	-	-	-
Specific Indirect - Internalising	0.01	-0.013	0.034	0.39	-	-	-	-	-	-
Specific Indirect - Externalising	-0.024	-0.051	0.004	0.097	-	-	-	-	-	-
Direct	0.011	-0.01	0.032	0.31	-	-	-	-	-	-
<b>FM: FFM</b>										
Total	-0.073	-0.115	-0.031	0.001	0.01	0.955	0.947	0.102	861.25	461
Total Indirect	-0.03	-0.054	-0.006	0.013	-	-	-	-	-	-
Specific Indirect - Internalising	-0.042	-0.082	-0.002	0.038	-	-	-	-	-	-
Specific Indirect - Externalising	0.012	-0.034	0.059	0.60	-	-	-	-	-	-
Direct	-0.043	-0.082	-0.004	0.032	-	-	-	-	-	-
<b>BMI</b>										
Total	-0.098	-0.15	-0.048	<0.001	0.01	0.955	0.947	0.113	871.29	461
Total Indirect	-0.049	-0.077	-0.021	0.001	-	-	-	-	-	-
Specific Indirect - Internalising	-0.038	-0.091	0.015	0.16	-	-	-	-	-	-
Specific Indirect - Externalising	-0.011	-0.071	0.049	0.71	-	-	-	-	-	-
Direct	-0.048	-0.094	-0.003	0.035	-	-	-	-	-	-

**Table 8-5 Footnote:** All models are adjusted for sex, age at sweep 7, ethnicity and stratification characteristics. Abbreviations:  $\chi^2$  – chi-squared; df – degrees of freedom; CFI – Comparative Fit Index; TLI – Tucker-Lewis Index; RMSEA – Root Mean Square Error of Approximation; SRMR – Standardized Root Mean Squared Residual.

### 8.3.3 Moderated Mediation

The sensitivity analysis demonstrated similar results for mediation model 3 when SDQ items were treated as continuous (as opposed to categorical ordinal), with the exception of FFMI models adjusted for FMI (Appendix A8.3). Model fit was better in these models, as measured by the SRMR fit indices. Additionally, the standardised factor had good model fit (Appendix A8.4) demonstrating it was appropriate to use in interaction terms for further analysis. Only the p-value for differences are reported in results below, as they will be the same or similar to the p-value for interaction, which is reported in the tables.

#### 8.3.3.1 Index of Multiple Deprivation

For FMI, BMI and FM:FFM ratio, a negative total effect and direct effect between income and the body composition measures were only observed in the least and most deprived areas, with a stronger gradient in the most deprived areas (Table 8-6). For FFMI a total and direct effect was not observed in any groups, with the exception of a negative total effect in the most deprived IMD group without adjustment for FMI (Table 8-6).

#### Mediation by Internalising Symptoms

For FMI, mediation through internalising symptoms was strongest in the most deprived IMD group ( $\beta$  -0.076, 95% CI: -0.14 to -0.015), whilst no mediation was observed in the least deprived group. In all deprivation groups, low income was related to increased levels of internalising symptoms, but only in the most deprived and medium deprivation group were higher levels of internalising symptoms related to higher FMI (Table 8-6). These results were supported by evidence of differences between the conditional indirect path in the most deprived and least deprived areas ( $p=0.05$ ) and the medium deprivation and least deprived ( $p=0.04$ ) areas (Table 8-7).

A similar pattern was observed for BMI and FM:FFM ratio, where the strongest negative indirect effect was observed in the most deprived group, with no mediating path observed in the most advantaged groups. Like the results for FMI, income was negatively related to internalising symptoms in all groups, but higher levels of internalising symptoms were only related to increases in BMI and FM:FFM ratio in the most deprived and medium deprivation group, with the association strongest in the most deprived group (Table 8-6). For both BMI and FM:FFM ratio the results in the sub-group analysis were consistent with the conditional indirect paths and differences were observed between the most deprived and least deprived groups (BMI  $p=0.01$ ; FM:FFM  $p=0.03$ ) and the medium deprivation and least deprived (BMI  $p=0.05$ ; FM:FFM  $p=0.02$ ) groups (Table 8-7). No difference was observed between conditional indirect effects in the medium deprivation group and high deprivation group for FM:FFM, whilst there is slight evidence of a difference for BMI ( $p=0.08$ ).

For FFMI there was a negative indirect path in the most deprived group ( $\beta$  -0.042, 95% CI: -0.078 to -0.005), but not in the other IMD groups. This was due to a positive association between internalising symptoms and FFMI only in the most deprived group (Table 8-6). However, the interaction model only indicated slight evidence of a difference between conditional indirect effects between the high and low deprivation group ( $p=0.08$ ) but not between the other groups (high vs medium  $p=0.21$ ; medium vs low

$p=0.33$ , Table 8-7). When adjusted for FMI, there was no evidence of mediating paths in any of the groups, and no differences in the conditional indirect effects are observed.

### Mediation by Externalising Symptoms

For FMI, a positive indirect effect was observed in the most deprived group ( $\beta$  0.068, 95% CI: -0.004 to 0.14) where those with lower income exhibited greater levels of externalising symptoms ( $\beta$  -0.26, 95% CI: -0.34 to -0.17) which in turn was related to lower FMI ( $\beta$  -0.27, 95% CI: -0.51 to -0.022) (Table 8-6). For the middle deprivation group, no mediating path was observed for externalising symptoms, whilst in the least deprived group, a negative indirect path was observed ( $\beta$  -0.039, 95% CI: -0.08 to 0.002). For this group, income was still negatively associated with externalising, but unlike the association in the most deprived group, externalising symptoms were related to an increase in FMI. A similar pattern was observed for conditional indirect paths in the interaction model (Table 8-7), and evidence of differences between the conditional indirect paths was observed between the most deprived group and the middle ( $p=0.07$ ) and low deprivation group ( $p=0.06$ ).

A similar pattern was observed for BMI, where a positive indirect path existed for externalising symptoms in the most deprived group (Table 8-6). There was a negative indirect path in the medium deprivation group, and as such differences in conditional indirect paths were observed between the high deprivation and medium deprivation groups ( $p=0.02$ ), high deprivation and low deprivation groups ( $p=0.01$ ), but not between the medium and low deprivation groups ( $p=0.5$ , Table 8-7).

For FM:FFM ratio, a similar positive indirect path between income and FM:FFM ratio through externalising symptoms was observed in the most deprived group, but there was no mediating path observed in the other two groups (Table 8-6). Differences in conditional indirect effects were observed between the high and low deprivation groups ( $p=0.03$ ) and between the high and medium deprivation groups ( $p=0.07$ ), but not between the medium and low deprivation groups (Table 8-7).

For FFMI, no mediating path was observed in the most deprived group whilst a negative indirect effect was observed for both the medium deprivation group and the low deprivation group (Table 8-6). However, there was little evidence of differences between the conditional indirect effects (high vs low  $p=0.11$ ; high vs medium  $p=0.14$ ; medium vs low  $p=0.77$ , Table 8-7). When adjusted for FMI, only the medium deprivation group demonstrated mediation, with those in the lowest income households exhibiting greater externalising symptoms, which in turn was related to higher FFMI. However, similar to the unadjusted model, no difference between conditional paths was observed (Table 8-7).



**Table 8-6.** Standardised Coefficients for Mediating Paths for Body Composition and Body Mass Index across Index of Multiple Deprivation Groups

<i>Model</i>	<i>Group 1: High Deprivation</i>				<i>Group 2: Middle</i>				<i>Group 3: Low Deprivation</i>			
	$\beta$ Coefficient	Lower CI	Upper CI	P Value	$\beta$ Coefficient	Lower CI	Upper CI	P Value	$\beta$ Coefficient	Lower CI	Upper CI	P Value
<b><i>Fat Mass Index (FMI)</i></b>												
<i>Total Effect</i>	-0.10	-0.18	-0.028	0.008	-0.018	-0.072	0.037	0.53	-0.098	-0.18	-0.02	0.014
<i>Total Indirect</i>	-0.009	-0.06	0.043	0.75	-0.049	-0.071	-0.027	<0.001	-0.027	-0.05	-0.004	0.022
<i>Internalising Indirect</i>	-0.076	-0.14	-0.015	0.014	-0.037	-0.066	-0.009	0.01	0.012	-0.014	0.038	0.36
<i>Externalising Indirect</i>	0.068	-0.004	0.14	0.064	-0.011	-0.032	0.01	0.29	-0.039	-0.08	0.002	0.061
<i>Direct Effect</i>	-0.096	-0.17	-0.021	0.012	0.031	-0.019	0.081	0.22	-0.071	-0.15	0.003	0.061
Path a1: Income to Internalising	-0.20	-0.29	-0.1	<0.001	-0.23	-0.32	-0.14	<0.001	-0.19	-0.27	-0.12	<0.001
Path a2: Income to Externalising	-0.26	-0.34	-0.17	<0.001	-0.20	-0.29	-0.11	<0.001	-0.23	-0.32	-0.14	<0.001
Path b1: Internalising to FMI	0.39	0.17	0.61	0.001	0.16	0.056	0.27	0.003	-0.063	-0.20	0.07	0.35
Path b2: Externalising to FMI	-0.27	-0.51	-0.022	0.033	0.057	-0.045	0.16	0.28	0.17	0.005	0.33	0.043
<b><i>Fat-Free Mass Index (FFMI)</i></b>												
<i>Total Effect</i>	-0.043	-0.094	0.008	0.096	-0.003	-0.044	0.038	0.88	-0.027	-0.07	0.015	0.211
<i>Total Indirect</i>	-0.01	-0.039	0.019	0.50	-0.026	-0.04	-0.011	0.001	-0.017	-0.034	0.00	0.046
<i>Internalising Indirect</i>	-0.042	-0.078	-0.005	0.027	-0.005	-0.022	0.012	0.60	0.011	-0.005	0.027	0.17
<i>Externalising Indirect</i>	0.032	-0.012	0.075	0.16	-0.021	-0.039	-0.003	0.024	-0.028	-0.056	-0.001	0.041
<i>Direct Effect</i>	-0.033	-0.082	0.015	0.18	0.023	-0.019	0.064	0.28	-0.01	-0.05	0.029	0.62
Path a1: Income to Internalising	-0.20	-0.29	-0.10	<0.001	-0.23	-0.32	-0.14	<0.001	-0.19	-0.27	-0.12	<0.001
Path a2: Income to Externalising	-0.26	-0.34	-0.17	<0.001	-0.2	-0.29	-0.11	<0.001	-0.23	-0.33	-0.14	<0.001
Path b1: Internalising to FFMI	0.21	0.058	0.37	0.007	0.02	-0.053	0.093	0.59	-0.059	-0.14	0.023	0.16
Path b2: Externalising to FFMI	-0.12	-0.29	0.039	0.14	0.11	0.03	0.18	0.006	0.12	0.019	0.23	0.053
<b><i>Fat-Free Mass Index (Adjusted for FMI)</i></b>												
<i>Total Effect</i>	0.01	-0.023	0.043	0.56	0.005	-0.026	0.037	0.75	0.018	-0.008	0.044	0.17
<i>Total Indirect</i>	-0.005	-0.017	0.007	0.40	-0.003	-0.012	0.006	0.47	-0.004	-0.012	0.004	0.38
<i>Internalising Indirect</i>	0.00	-0.022	0.022	0.998	0.011	-0.004	0.025	0.14	0.005	-0.005	0.014	0.34
<i>Externalising Indirect</i>	-0.005	-0.036	0.026	0.75	-0.014	-0.028	0.00	0.045	-0.008	-0.021	0.005	0.21
<i>Direct Effect</i>	0.015	-0.021	0.051	0.41	0.009	-0.024	0.041	0.60	0.022	-0.005	0.048	0.11
Path a1: Income to Internalising	-0.18	-0.26	-0.091	<0.001	-0.22	-0.31	-0.14	<0.001	-0.19	-0.26	-0.12	<0.001
Path a2: Income to Externalising	-0.25	-0.34	-0.16	<0.001	-0.20	-0.28	-0.11	<0.001	-0.22	-0.31	-0.13	<0.001

Path b1: Internalising to FFMI	0.00	-0.12	0.12	0.996	-0.049	-0.11	0.014	0.13	-0.025	-0.074	0.024	0.32
Path b2: Externalising to FFMI	0.02	-0.10	0.14	0.75	0.072	0.012	0.13	0.02	0.038	-0.016	0.092	0.17
<b>Fat Mass: Fat-Free Mass Ratio (FM:FFM)</b>												
<i>Total Effect</i>	-0.087	-0.15	-0.022	0.008	-0.021	-0.064	0.023	0.36	-0.07	-0.12	-0.018	0.009
<i>Total Indirect</i>	-0.005	-0.047	0.038	0.83	-0.038	-0.057	-0.02	<0.001	-0.014	-0.028	0	0.051
<i>Internalising Indirect</i>	-0.064	-0.116	-0.011	0.017	-0.031	-0.055	-0.006	0.013	0.004	-0.014	0.022	0.65
<i>Externalising Indirect</i>	0.059	-0.002	0.12	0.06	-0.007	-0.024	0.009	0.37	-0.018	-0.045	0.008	0.17
<i>Direct Effect</i>	-0.082	-0.14	-0.02	0.009	0.017	-0.023	0.058	0.40	-0.056	-0.11	-0.005	0.033
Path a1: Income to Internalising	-0.20	-0.29	-0.10	<0.001	-0.23	-0.32	-0.14	<0.001	-0.19	-0.27	-0.12	<0.001
Path a2: Income to Externalising	-0.26	-0.34	-0.17	<0.001	-0.2	-0.29	-0.11	<0.001	-0.23	-0.33	-0.14	<0.001
Path b1: Internalising to FM:FFM	0.32	0.13	0.52	0.001	0.13	0.048	0.22	0.002	-0.022	-0.12	0.072	0.64
Path b2: Externalising to FM:FFM	-0.23	-0.44	-0.019	0.033	0.037	-0.043	0.12	0.36	0.08	-0.03	0.19	0.15
<b>Body Mass Index (BMI)</b>												
<i>Total Effect</i>	-0.095	-0.16	-0.027	0.006	-0.023	-0.075	0.03	0.39	-0.069	-0.14	-0.003	0.041
<i>Total Indirect</i>	-0.012	-0.058	0.035	0.62	-0.047	-0.069	-0.026	<0.001	-0.025	-0.048	-0.003	0.03
<i>Internalising Indirect</i>	-0.075	-0.13	-0.017	0.011	-0.027	-0.055	-0.002	0.041	0.011	-0.013	0.034	0.37
<i>Externalising Indirect</i>	0.063	-0.002	0.13	0.056	-0.02	-0.041	0.002	0.071	-0.036	-0.074	0.003	0.072
<i>Direct Effect</i>	-0.084	-0.15	-0.018	0.013	0.024	-0.025	0.074	0.34	-0.044	-0.11	0.019	0.17
Path a1: Income to Internalising	-0.20	-0.29	-0.11	<0.001	-0.24	-0.33	-0.15	<0.001	-0.19	-0.26	-0.12	<0.001
Path a2: Income to Externalising	-0.26	-0.34	-0.17	<0.001	-0.21	-0.29	-0.12	<0.001	-0.23	-0.33	-0.14	<0.001
Path b1: Internalising to BMI	0.38	0.16	0.60	0.001	0.12	0.015	0.22	0.024	-0.056	-0.18	0.066	0.37
Path b2: Externalising to BMI	-0.25	-0.48	-0.02	0.033	0.097	0.001	0.19	0.048	0.15	0.001	0.31	0.049

**Table 8-6 Footnote:** Table showing total, indirect and direct effects for BMI and each body composition outcome in each deprivation (IMD) group. Path A is the regression path from income to internalising/externalising symptoms, where the symptoms are the outcome. Path B is the regression path for internalising/externalising symptoms to FMI where FMI is the outcome.

**Table 8-7.** Unstandardised Regression Coefficients for Conditional Indirect Effects and Interaction Terms for Index of Multiple Deprivation

Model	Group 1: High Deprivation				Group 2: Middle				Group 3: Low Deprivation				P Value Difference**		
	Coefficient	SE	P Value	P Value Interaction*	Coefficient	SE	P Value	P Value Interaction*	Coefficient	SE	P Value	P Value Interaction*	G1 Vs G3	G1 Vs G2	G2 Vs G3
<b>FMI</b>															
Internalising	-0.22	0.11	0.039	0.043	-0.086	0.033	0.01	0.046	-0.016	0.014	0.266	n/a	0.054	0.182	0.044
Externalising	0.20	0.12	0.085	0.062	0.004	0.034	0.90	0.62	-0.013	0.019	0.47	n/a	0.063	0.069	0.62
<b>FFMI</b>															
Internalising	-0.078	0.045	0.085	0.078	-0.018	0.022	0.40	0.33	0.006	0.014	0.66	n/a	0.079	0.21	0.33
Externalising	0.054	0.053	0.31	0.10	-0.026	0.024	0.26	0.77	-0.035	0.019	0.059	n/a	0.11	0.14	0.77
<b>FFMI (Adjusted for FMI)</b>															
Internalising	-0.01	0.029	0.73	0.46	0.023	0.014	0.096	0.60	0.014	0.011	0.23	n/a	0.46	0.28	0.60
Externalising	0.006	0.035	0.87	0.40	-0.023	0.015	0.12	0.90	-0.025	0.013	0.059	n/a	0.40	0.43	0.90
<b>FM:FFM Ratio</b>															
Internalising	-0.01	0.004	0.019	0.025	-0.005	0.002	0.004	0.024	-0.001	0.001	0.46	n/a	0.032	0.25	0.022
Externalising	0.01	0.005	0.047	0.031	0.001	0.002	0.54	0.34	-0.001	0.001	0.42	n/a	0.033	0.065	0.34
<b>BMI</b>															
Internalising	-0.37	0.13	0.006	0.006	-0.14	0.061	0.021	0.056	-0.009	0.031	0.78	n/a	0.009	0.082	0.053
Externalising	0.32	0.15	0.03	0.011	-0.003	0.064	0.96	0.45	-0.06	0.04	0.14	n/a	0.012	0.023	0.45

**Table 8-7 Footnote:** Estimates presented are unstandardised coefficients. \* Indicates p-value for path between the interaction of environmental dummy variables with internalising/externalising symptom to the outcome. \*\* G1 = Group 1, G2 = Group 2, G3 = Group 3. The p-values for the interaction term for group 1 will be the similar or the same as the p-value for the difference between group 1 and 3, and the p-value for group 2 will be similar to the p value for the difference between group 2 and 3. No p-value for the interaction term is presented for Group 3, as group 3 was the reference category in models. Abbreviations: FMI – fat mass index; FFMI – fat-free mass index; FM:FFM – fat mass to fat-free mass; BMI – body mass index; SE – standard error; n/a – not applicable.

### 8.3.3.2 Green Spaces

For FMI, FM:FFM Ratio and BMI, negative total effects between income and the body composition variables were observed in all green space groups, although larger effects were seen in the greenest areas followed by the least green areas (Table 8-8). The same patterns were observed for direct effects but with typically no direct path observed in the middle green space group. For FFMI, no direct paths were observed, but a negative total effect between income and FFMI were seen in the most and least green areas. No direct or total effects were observed in any group when FFMI was adjusted for FMI.

#### Mediation by Internalising Symptoms

For FMI, there was a negative mediating path in the least green areas ( $\beta$  -0.13, 95% CI: -0.20 to -0.063) but in no other green space groups. Similar to the results for IMD, this negative indirect effect was because lower income was related to greater levels of internalising symptoms, and higher levels of internalising symptoms were related to higher FMI (Table 8-8). In the greener areas, internalising symptoms were not related to FMI. The interaction model was consistent with the sub-group analysis and demonstrated differences in the conditional indirect paths between the least green areas and both the medium ( $p=0.04$ ) and most green areas ( $p=0.009$ ), but no difference between the medium and the greenest areas (Table 8-9). The same pattern was observed for both BMI and FM:FFM ratio where a negative indirect effect was only observed for those living in the areas with the least green spaces (Table 8-8). Differences were observed in the paths between the least green area and the other groups for BMI (medium  $p=0.01$ ; most green  $p=0.07$ ) and between the least green areas and the middle group for FM:FFM ratio ( $p=0.02$ , Table 8-9).

For FFMI, there was a negative indirect effect in the least green areas but not in the other groups, because higher internalising symptoms were only related to higher FFMI among individuals living in areas with low levels of green space (Table 8-8). A Difference in mediating effect was only observed between the low green space group and the middle group ( $p=0.07$ , Table 8-9). When adjusted for FMI, there was no longer a mediating path in the areas with less green space, and no difference between groups is observed.

#### Mediation by Externalising Symptoms

Similar to the results for IMD, a positive indirect path was observed in the least green areas for FMI, BMI and FM:FFM ratio, where lower income was related to higher levels of externalising symptoms, which in turn were related to lower FMI, BMI and FM:FFM ratio (Table 8-8). For all three body composition outcomes, there was a negative mediating path in the middle group for green spaces, where externalising symptoms were related to higher FMI, BMI and FM:FFM ratio, whilst less evidence of a mediating path was found in areas with the highest levels of green spaces. As such, differences between groups were observed between the areas with the lowest levels of green spaces and the highest levels (FMI  $p=0.02$ ; BMI  $p=0.05$ ; FM:FFM  $p=0.05$ ), and between the least green areas and middle green space group (FMI  $p=0.003$ ; BMI  $p=0.01$ ; FM:FFM  $p=0.01$ ), but not between the middle group and the areas with most green spaces (Table 8-9).

**Table 8-8.** Standardised Coefficients for Mediating Paths for Body Composition and Body Mass Index across Green Space Groups

<i>Model</i>	<i>Group 1: Low Green Spaces</i>				<i>Group 2: Middle</i>				<i>Group 3: High Green Space</i>			
	$\beta$ Coefficient	Lower CI	Upper CI	P Value	$\beta$ Coefficient	Lower CI	Upper CI	P Value	$\beta$ Coefficient	Lower CI	Upper CI	P Value
<b><i>Fat Mass Index (FMI)</i></b>												
<i>Total Effect</i>	-0.12	-0.20	-0.045	0.002	-0.057	-0.114	0.001	0.054	-0.16	-0.25	-0.072	<0.001
<i>Total Indirect</i>	-0.054	-0.10	-0.008	0.022	-0.032	-0.046	-0.018	<0.001	-0.058	-0.098	-0.018	0.004
<i>Internalising Indirect</i>	-0.13	-0.20	-0.063	<0.001	-0.003	-0.025	0.019	0.80	-0.005	-0.048	0.039	0.83
<i>Externalising Indirect</i>	0.079	0.015	0.14	0.015	-0.029	-0.053	-0.005	0.017	-0.054	-0.13	0.019	0.15
<i>Direct Effect</i>	-0.066	-0.14	0.008	0.08	-0.024	-0.084	0.035	0.42	-0.10	-0.18	-0.027	0.007
Path a1: Income to Internalising	-0.31	-0.40	-0.22	<0.001	-0.21	-0.29	-0.13	<0.001	-0.24	-0.33	-0.145	<0.001
Path a2: Income to Externalising	-0.30	-0.39	-0.20	<0.001	-0.25	-0.33	-0.18	<0.001	-0.29	-0.39	-0.193	<0.001
Path b1: Internalising to FMI	0.43	0.26	0.59	<0.001	0.014	-0.088	0.12	0.79	0.02	-0.16	0.21	0.83
Path b2: Externalising to FMI	-0.27	-0.44	-0.087	0.004	0.12	0.023	0.21	0.015	0.18	-0.039	0.41	0.11
<b><i>Fat-Free Mass Index (FFMI)</i></b>												
<i>Total Effect</i>	-0.038	-0.081	0.006	0.087	-0.015	-0.055	0.026	0.48	-0.065	-0.12	-0.009	0.024
<i>Total Indirect</i>	-0.031	-0.052	-0.01	0.004	-0.018	-0.029	-0.007	0.001	-0.049	-0.079	-0.02	0.001
<i>Internalising Indirect</i>	-0.047	-0.084	-0.009	0.017	0.005	-0.011	0.021	0.51	-0.001	-0.028	0.027	0.97
<i>Externalising Indirect</i>	0.016	-0.018	0.048	0.37	-0.023	-0.043	-0.004	0.019	-0.049	-0.095	-0.001	0.042
<i>Direct Effect</i>	-0.007	-0.05	0.037	0.76	0.003	-0.04	0.047	0.88	-0.016	-0.062	0.03	0.51
Path a1: Income to Internalising	-0.31	-0.40	-0.22	<0.001	-0.21	-0.29	-0.13	<0.001	-0.24	-0.33	-0.15	<0.001
Path a2: Income to Externalising	-0.30	-0.39	-0.20	<0.001	-0.25	-0.33	-0.18	<0.001	-0.29	-0.39	-0.19	<0.001
Path b1: Internalising to FFMI	0.15	0.039	0.26	0.008	-0.025	-0.1	0.049	0.51	0.003	-0.11	0.12	0.96
Path b2: Externalising to FFMI	-0.053	-0.17	0.06	0.36	0.093	0.019	0.17	0.17	0.17	0.033	0.30	0.015
<b><i>Fat-Free Mass Index (Adjusted for FMI)</i></b>												
<i>Total Effect</i>	0.022	-0.004	0.049	0.102	0.011	-0.019	0.041	0.47	0.014	-0.019	0.047	0.412
<i>Total Indirect</i>	-0.002	-0.014	0.01	0.76	-0.003	-0.011	0.005	0.49	-0.018	-0.033	-0.003	0.02
<i>Internalising Indirect</i>	0.014	-0.008	0.037	0.22	0.006	-0.005	0.017	0.32	0.001	-0.015	0.017	0.88
<i>Externalising Indirect</i>	-0.016	-0.039	0.008	0.18	-0.008	-0.023	0.006	0.27	-0.019	-0.043	0.004	0.11
<i>Direct Effect</i>	0.024	-0.003	0.052	0.086	0.014	-0.018	0.045	0.39	0.032	0.001	0.062	0.04
Path a1: Income to Internalising	-0.28	-0.36	-0.20	<0.001	-0.21	-0.29	-0.13	<0.001	-0.21	-0.30	-0.11	<0.001
Path a2: Income to Externalising	-0.28	-0.38	-0.19	<0.001	-0.25	-0.32	-0.17	<0.001	-0.27	-0.36	-0.17	<0.001

Path b1: Internalising to FFMI	-0.05	-0.13	0.028	0.21	-0.027	-0.08	0.025	0.31	-0.006	-0.083	0.071	0.88
Path b2: Externalising to FFMI	0.056	-0.022	0.14	0.16	0.034	-0.025	0.094	0.26	0.072	-0.008	0.15	0.077
<b>Fat Mass: Fat-Free Mass Ratio (FM:FFM)</b>												
<i>Total Effect</i>	-0.096	-0.15	-0.037	0.001	-0.055	-0.10	-0.007	0.025	-0.12	-0.18	-0.054	<0.001
<i>Total Indirect</i>	-0.046	-0.082	-0.01	0.012	-0.021	-0.031	-0.01	<0.001	-0.038	-0.066	-0.01	0.008
<i>Internalising Indirect</i>	-0.104	-0.16	-0.047	<0.001	-0.004	-0.023	0.014	0.66	-0.013	-0.041	0.016	0.39
<i>Externalising Indirect</i>	0.058	0.007	0.11	0.024	-0.016	-0.035	0.003	0.091	-0.025	-0.073	0.022	0.29
<i>Direct Effect</i>	-0.05	-0.11	0.008	0.091	-0.035	-0.085	0.015	0.17	-0.079	-0.14	-0.022	0.007
Path a1: Income to Internalising	-0.31	-0.40	-0.22	<0.001	-0.21	-0.29	-0.13	<0.001	-0.24	-0.33	-0.15	<0.001
Path a2: Income to Externalising	-0.30	-0.39	-0.20	<0.001	-0.25	-0.33	-0.18	<0.001	-0.29	-0.39	-0.19	<0.001
Path b1: Internalising to FM:FFM	0.33	0.20	0.47	<0.001	0.019	-0.067	0.11	0.66	0.055	-0.069	0.18	0.18
Path b2: Externalising to FM:FFM	-0.19	-0.34	-0.046	0.01	0.065	-0.011	0.14	0.096	0.087	-0.065	0.24	0.24
<b>Body Mass Index (BMI)</b>												
<i>Total Effect</i>	-0.11	-0.18	-0.04	0.002	-0.052	-0.11	0.004	0.069	-0.13	-0.21	-0.055	0.001
<i>Total Indirect</i>	-0.056	-0.094	-0.017	0.005	-0.027	-0.041	-0.014	<0.001	-0.066	-0.104	-0.028	0.001
<i>Internalising Indirect</i>	-0.11	-0.17	-0.049	0.001	0.001	-0.022	0.023	0.96	-0.015	-0.052	0.022	0.44
<i>Externalising Indirect</i>	0.056	0.00	0.11	0.048	-0.028	-0.051	-0.004	0.02	-0.052	-0.11	0.012	0.11
<i>Direct Effect</i>	-0.053	-0.12	0.016	0.14	-0.024	-0.083	0.034	0.41	-0.065	-0.13	-0.002	0.045
Path a1: Income to Internalising	-0.32	-0.40	-0.23	<0.001	-0.22	-0.30	-0.14	<0.001	-0.23	-0.32	-0.15	<0.001
Path a2: Income to Externalising	-0.3	-0.40	-0.21	<0.001	-0.25	-0.33	-0.18	<0.001	-0.29	-0.39	-0.20	<0.001
Path b1: Internalising to BMI	0.36	0.20	0.51	<0.001	-0.002	-0.104	0.099	0.97	0.063	-0.096	0.22	0.44
Path b2: Externalising to BMI	-0.19	-0.36	-0.017	0.031	0.11	0.018	0.20	0.019	0.18	-0.016	0.37	0.072

**Table 8-8 Footnote:** Table showing total, indirect and direct effects for BMI and each body composition outcome in each green space group. Path A is the regression path from income to internalising/externalising symptoms, where the symptoms are the outcome. Path B is the regression path for internalising/externalising symptoms to FMI where FMI is the outcome.

**Table 8-9.** Unstandardised Regression Coefficients for Conditional Indirect Effects and Interaction Terms for Green Spaces

Model	Group 1: Low Green Spaces				Group 2: Middle				Group 3: High Green Spaces				P Value Difference**		
	Coefficient	SE	P Value	P Value Interaction*	Coefficient	SE	P Value	P Value Interaction*	Coefficient	SE	P Value	P Value Interaction*	G1 Vs G3	G1 Vs G2	G2 Vs G3
<b>FMI</b>															
Internalising	-0.11	0.03	<0.001	0.028	-0.017	0.023	0.47	0.83	-0.024	0.027	0.37	n/a	0.035	0.009	0.83
Externalising	0.061	0.03	0.039	0.014	-0.045	0.027	0.10	0.94	-0.041	0.031	0.19	n/a	0.018	0.003	0.94
<b>FFMI</b>															
Internalising	-0.049	0.027	0.064	0.26	0.009	0.018	0.63	0.58	-0.008	0.026	0.74	n/a	0.27	0.066	0.58
Externalising	0.018	0.029	0.53	0.19	-0.049	0.024	0.04	0.62	-0.032	0.026	0.21	n/a	0.20	0.062	0.62
<b>FFMI (Adjusted for FMI)</b>															
Internalising	0.009	0.013	0.49	0.84	0.02	0.015	0.19	0.48	0.005	0.016	0.75	n/a	0.84	0.60	0.48
Externalising	-0.014	0.015	0.38	0.83	-0.026	0.018	0.15	0.46	-0.009	0.017	0.61	n/a	0.83	0.60	0.46
<b>FM:FFM Ratio</b>															
Internalising	-0.007	0.002	0.001	0.09	-0.001	0.001	0.33	0.82	-0.002	0.002	0.34	n/a	0.101	0.024	0.82
Externalising	0.004	0.002	0.047	0.044	-0.002	0.001	0.25	0.99	-0.002	0.002	0.41	n/a	0.05	0.014	0.99
<b>BMI</b>															
Internalising	-0.223	0.071	0.002	0.058	-0.017	0.043	0.69	0.62	-0.054	0.06	0.36	n/a	0.069	0.011	0.62
Externalising	0.122	0.073	0.095	0.04	-0.09	0.052	0.081	0.78	-0.068	0.059	0.25	n/a	0.045	0.013	0.78

**Table 8-9 Footnote:** Estimates presented are unstandardised coefficients. \* Indicates p-value for path between the interaction of environmental dummy variables with internalising/externalising symptom to the outcome. \*\* G1 = Group 1, G2 = Group 2, G3 = Group 3. The p-values for the interaction term for group 1 will be the similar or the same as the p-value for the difference between group 1 and 3, and the p-value for group 2 will be similar to the p value for the difference between group 2 and 3. No p-value for the interaction term is presented for Group 3, as group 3 was the reference category in models. Abbreviations: FMI – fat mass index; FFMI – fat-free mass index; FM:FFM – fat mass to fat-free mass; BMI – body mass index; SE – standard error; n/a – not applicable.

For FFMI, no mediating path was observed in the area with the lowest levels of green spaces, whilst negative indirect paths were observed in the area with medium and high levels of green spaces (Table 8-8). Differences between paths were only observed between the medium and low level green space groups ( $p=0.062$ , Table 8-9). When adjusting FFMI for FMI, mediating effects were observed in none of the green space groups, and as such no differences between groups was observed.

### *8.3.3.3 Domestic Gardens*

For the medium domestic garden group, the dummy variable for the advantaged stratum in Northern Ireland at baseline was removed from models due to 0 or 1 observation in this stratum. The remaining Scottish, Welsh and Northern Ireland strata were not removed, as there were some individuals who lived in the other countries of the UK at baseline but moved between sweeps, so were still included when analysis was restricted to those residing in England at sweep 5. For the low domestic garden areas, the dummy variables for both advantaged and disadvantaged strata in Northern Ireland were also removed from models for the same reason.

Similar to the IMD and green spaces results, negative total and direct effects between income and FMI, FM:FFM ratio and BMI were typically observed in the least and most deprived areas in terms of domestic gardens, with less evidence of association or weaker effects in the middle group (Table 8-10). No direct or total effects were observed between income and FFMI.

### *Mediation by Internalising Symptoms*

For FMI and FM:FFM ratio, no mediating path was observed in the middle group, whilst some evidence of a negative indirect effect was observed in the areas with the lowest level of domestic gardens and areas with highest levels of domestic gardens (Table 8-10). For BMI, a slight negative indirect effect was only observed in the areas with the lowest levels of domestic gardens. No differences between conditional indirect paths were observed for FMI, FM:FFM ratio or BMI (Table 8-11).

For FFMI, no mediating path was observed across groups with or without adjustment for FMI, and no difference between conditional indirect effects was observed.

### *Mediation by Externalising Symptoms*

No mediating path through externalising symptoms was observed for BMI, FMI and FM:FFM ratio in any of the groups based on domestic garden density, and no differences in conditional indirect paths were observed. For FFMI, a negative indirect path was observed in the group with the lowest deprivation for domestic gardens, including when adjusted for FMI. However, no difference in mediating paths between domestic garden areas were observed (Table 8-11).



**Table 8-10.** Standardised Coefficients for Mediating Paths for Body Composition and Body Mass Index across Domestic Garden Groups

<i>Model</i>	<i>Group 1: Low Domestic Gardens</i>				<i>Group 2: Middle</i>				<i>Group 3: High Domestic Gardens</i>			
	$\beta$ Coefficient	Lower CI	Upper CI	P Value	$\beta$ Coefficient	Lower CI	Upper CI	P Value	$\beta$ Coefficient	Lower CI	Upper CI	P Value
<b><i>Fat Mass Index (FMI)</i></b>												
<i>Total Effect</i>	-0.105	-0.168	-0.042	0.001	-0.059	-0.142	0.023	0.16	-0.14	-0.23	-0.059	0.001
<i>Total Indirect</i>	-0.015	-0.035	0.004	0.122	-0.039	-0.061	-0.018	<0.001	-0.093	-0.15	-0.041	<0.001
<i>Internalising Indirect</i>	-0.027	-0.054	0.00	0.05	-0.013	-0.05	0.025	0.51	-0.058	-0.12	0.009	0.089
<i>Externalising Indirect</i>	0.012	-0.022	0.046	0.5	-0.027	-0.072	0.019	0.26	-0.036	-0.097	0.025	0.25
<i>Direct Effect</i>	-0.09	-0.149	-0.03	0.003	-0.02	-0.101	0.061	0.63	-0.05	-0.12	0.021	0.17
Path a1: Income to Internalising	-0.17	-2.88	2.55	0.90	-0.23	-6.62	6.16	0.94	-0.37	-0.49	-0.25	<0.001
Path a2: Income to Externalising	-0.23	-1.03	0.57	0.58	-0.27	-5.31	4.78	0.92	-0.34	-0.43	-0.25	<0.001
Path b1: Internalising to FMI	0.16	-2.43	2.75	0.90	0.054	-1.46	1.57	0.94	0.16	-0.016	0.33	0.076
Path b2: Externalising to FMI	-0.051	-0.29	0.18	0.67	0.099	-1.75	1.95	0.92	0.11	-0.068	0.28	0.23
<b><i>Fat-Free Mass Index (FFMI)</i></b>												
<i>Total Effect</i>	-0.02	-0.065	0.025	0.38	-0.026	-0.082	0.031	0.38	-0.042	-0.098	0.013	0.14
<i>Total Indirect</i>	-0.012	-0.023	-0.001	0.027	-0.027	-0.043	-0.011	0.001	-0.055	-0.087	-0.024	0.001
<i>Internalising Indirect</i>	-0.004	-0.02	0.012	0.62	-0.004	-0.033	0.024	0.77	-0.012	-0.055	0.032	0.60
<i>Externalising Indirect</i>	-0.008	-0.03	0.014	0.46	-0.022	-0.057	0.012	0.20	-0.044	-0.087	0	0.05
<i>Direct Effect</i>	-0.008	-0.054	0.039	0.75	0.001	-0.056	0.058	0.98	0.013	-0.04	0.066	0.63
Path a1: Income to Internalising	-0.17	-3.22	2.88	0.91	-0.23	-6.60	6.13	0.94	-0.37	-0.49	-0.24	<0.001
Path a2: Income to Externalising	-0.23	-1.14	0.68	0.63	-0.27	-5.07	4.54	0.91	-0.34	-0.43	-0.25	<0.001
Path b1: Internalising to FFMI	0.024	-0.41	0.46	0.91	0.019	-0.58	0.62	0.95	0.032	-0.085	0.15	0.59
Path b2: Externalising to FFMI	0.037	-0.13	0.20	0.67	0.083	-1.42	1.59	0.91	0.13	0.008	0.25	0.037
<b><i>Fat-Free Mass Index (Adjusted for FMI)</i></b>												
<i>Total Effect</i>	0.026	-0.012	0.064	0.17	0.004	-0.034	0.041	0.85	0.031	-0.008	0.07	0.12
<i>Total Indirect</i>	-0.006	-0.016	0.004	0.22	-0.007	-0.015	0.002	0.13	-0.005	-0.023	0.012	0.55
<i>Internalising Indirect</i>	0.008	-0.005	0.022	0.23	0.002	-0.013	0.017	0.83	0.021	-0.01	0.052	0.19
<i>Externalising Indirect</i>	-0.014	-0.033	0.004	0.14	-0.008	-0.025	0.009	0.36	-0.026	-0.055	0.003	0.075
<i>Direct Effect</i>	0.032	-0.008	0.073	0.12	0.01	-0.028	0.048	0.60	0.037	-0.002	0.075	0.065
Path a1: Income to Internalising	-0.15	-3.15	2.84	0.92	-0.22	-6.44	5.99	0.94	-0.33	-0.47	-0.19	<0.001
Path a2: Income to Externalising	-0.22	-1.45	1.01	0.73	-0.26	-4.67	4.15	0.91	-0.31	-0.39	-0.23	<0.001

Path b1: Internalising to FFMI	-0.054	-1.09	0.99	0.92	-0.007	-0.24	0.22	0.95	-0.064	-0.16	0.031	0.18
Path b2: Externalising to FFMI	0.065	-0.32	0.45	0.74	0.032	-0.50	0.56	0.91	0.085	-0.005	0.18	0.063
<b>Fat Mass: Fat-Free Mass Ratio (FM:FFM)</b>												
<i>Total Effect</i>	-0.085	-0.133	-0.037	<0.001	-0.035	-0.10	0.031	0.30	-0.12	-0.19	-0.045	0.001
<i>Total Indirect</i>	-0.008	-0.024	0.007	0.29	-0.027	-0.044	-0.011	0.001	-0.076	-0.12	-0.033	0.001
<i>Internalising Indirect</i>	-0.019	-0.041	0.003	0.095	-0.018	-0.052	0.015	0.28	-0.067	-0.13	-0.008	0.026
<i>Externalising Indirect</i>	0.011	-0.018	0.039	0.47	-0.009	-0.046	0.028	0.63	-0.008	-0.058	0.042	0.74
<i>Direct Effect</i>	-0.077	-0.124	-0.03	0.001	-0.008	-0.074	0.059	0.82	-0.041	-0.10	0.02	0.19
Path a1: Income to Internalising	-0.17	-3.17	2.83	0.91	-0.23	-6.55	6.09	0.94	-0.37	-0.49	-0.24	<0.001
Path a2: Income to Externalising	-0.23	-1.19	0.74	0.64	-0.27	-5.39	4.86	0.92	-0.34	-0.43	-0.25	<0.001
Path b1: Internalising to FM:FFM	0.11	-1.93	2.15	0.91	0.079	-2.03	2.19	0.94	0.19	0.032	0.34	0.018
Path b2: Externalising to FM:FFM	-0.047	-0.28	0.19	0.69	0.034	-0.64	0.70	0.92	0.025	-0.12	0.17	0.74
<b>Body Mass Index (BMI)</b>												
<i>Total Effect</i>	-0.079	-0.095	-0.015	0.007	-0.053	-0.131	0.026	0.19	-0.116	-0.20	-0.035	0.005
<i>Total Indirect</i>	-0.016	-0.023	0.001	0.076	-0.038	-0.059	-0.017	<0.001	-0.094	-0.14	-0.048	<0.001
<i>Internalising Indirect</i>	-0.022	-0.032	0.002	0.087	-0.017	-0.058	0.024	0.43	-0.052	-0.12	0.015	0.13
<i>Externalising Indirect</i>	0.006	-0.017	0.026	0.7	-0.021	-0.069	0.026	0.38	-0.042	-0.11	0.02	0.19
<i>Direct Effect</i>	-0.064	-0.083	-0.005	0.027	-0.015	-0.093	0.063	0.71	-0.022	-0.094	0.051	0.56
Path a1: Income to Internalising	-0.17	-2.58	2.24	0.89	-0.23	-5.64	5.17	0.93	-0.37	-0.49	-0.25	<0.001
Path a2: Income to Externalising	-0.23	-0.95	0.50	0.54	-0.27	-4.32	3.78	0.90	-0.34	-0.43	-0.25	<0.001
Path b1: Internalising to BMI	0.13	-1.68	1.94	0.89	0.071	-1.62	1.77	0.93	0.14	-0.031	0.31	0.11
Path b2: Externalising to BMI	-0.027	-0.20	0.15	0.76	0.079	-1.16	1.32	0.90	0.12	-0.054	0.30	0.17

**Table 8-10 Footnote:** Table showing total, indirect and direct effects for BMI and each body composition outcome in each domestic garden density group. Path A is the regression path from income to internalising/externalising symptoms, where the symptoms are the outcome. Path B is the regression path for internalising/externalising symptoms to FMI where FMI is the outcome.

**Table 8-11.** Unstandardised Regression Coefficients for Conditional Indirect Effects and Interaction Terms for Domestic Gardens

Model	Group 1: Low Domestic Gardens				Group 2: Middle				Group 3: High Domestic Gardens				P Value Difference**		
	Coefficient	SE	P Value	P Value Interaction*	Coefficient	SE	P Value	P Value Interaction*	Coefficient	SE	P Value	P Value Interaction*	G1 Vs G3	G1 Vs G2	G2 Vs G3
<b>FMI</b>															
Internalising	-0.059	0.028	0.035	0.415	-0.057	0.037	0.118	0.52	-0.026	0.033	0.44	n/a	0.41	0.98	0.52
Externalising	-0.005	0.027	0.84	0.36	0.016	0.041	0.71	0.26	-0.042	0.035	0.23	n/a	0.36	0.65	0.26
<b>FFMI</b>															
Internalising	-0.016	0.024	0.50	0.65	-0.011	0.031	0.72	0.81	-0.002	0.024	0.94	n/a	0.65	0.89	0.81
Externalising	-0.019	0.026	0.45	0.40	-0.017	0.033	0.62	0.46	-0.047	0.027	0.085	n/a	0.41	0.94	0.46
<b>FFMI (Adjusted for FMI)</b>															
Internalising	0.018	0.018	0.32	0.72	0.021	0.019	0.27	0.64	0.01	0.014	0.47	n/a	0.72	0.93	0.64
Externalising	-0.018	0.019	0.35	0.88	-0.025	0.021	0.23	0.90	-0.022	0.017	0.19	n/a	0.88	0.81	0.90
<b>FM:FFM Ratio</b>															
Internalising	-0.003	0.002	0.066	0.73	-0.004	0.002	0.073	0.61	-0.003	0.002	0.22	n/a	0.73	0.82	0.61
Externalising	0.00	0.002	0.96	0.80	0.002	0.002	0.36	0.38	-0.001	0.002	0.78	n/a	0.80	0.47	0.38
<b>BMI</b>															
Internalising	-0.11	0.058	0.054	0.40	-0.11	0.075	0.14	0.52	-0.045	0.062	0.46	n/a	0.40	0.98	0.51
Externalising	-0.004	0.059	0.95	0.28	0.043	0.08	0.59	0.19	-0.09	0.067	0.18	n/a	0.28	0.62	0.19

**Table 8-11 Footnote:** Estimates presented are unstandardised coefficients. \* Indicates p-value for path between the interaction of environmental dummy variables with internalising/externalising symptom to the outcome. \*\* G1 = Group 1, G2 = Group 2, G3 = Group 3. The p-values for the interaction term for group 1 will be the similar or the same as the p-value for the difference between group 1 and 3, and the p-value for group 2 will be similar to the p value for the difference between group 2 and 3. No p-value for the interaction term is presented for Group 3, as group 3 was the reference category in models. Abbreviations: FMI – fat mass index; FFMI – fat-free mass index; FM:FFM – fat mass to fat-free mass; BMI – body mass index; SE – standard error; n/a – not applicable.

#### 8.3.3.4 Fast-Food Density

For the highest density fast-food group and the medium density fast-food group, the two Northern Ireland strata dummy variables were removed from models due to lack of observations.

A negative total effect between income and FMI was observed in the areas with the highest fast-food density ( $\beta$  -0.15, 95% CI: -0.28 to -0.013) and the lowest ( $\beta$  -0.12, 95% CI: -0.17 to -0.061) showing that lower income was related to higher FMI. A direct effect was only observed in the lowest fast-food density group (Table 8-12). For FM:FFM ratio negative total and direct effects were only observed in the areas with the lowest fast-food density. For FFMI, only a negative total effect was observed in the areas with the lowest fast-food density, but when adjusted for FMI a positive direct effect was observed in the areas with the highest fast-food density and medium fast-food density, reflecting that low income was related to lower FFMI.

#### Mediation by Internalising Symptoms

For FMI, BMI and FM:FFM ratio, no mediating path was observed for internalising symptoms in the areas with the highest density and medium density of fast-food outlets, whilst a negative indirect effect was observed in areas with the lowest density of fast-food outlets (Table 8-12). However, no differences between conditional indirect effects were observed for FMI, BMI and FM:FFM ratio (Table 8-13).

For FFMI, no mediating paths were observed across fast-food density groups, and no differences between conditional indirect effects were observed in models both before and after adjustment for FMI.

#### Mediation by Externalising Symptoms

No mediating paths from fast-food density through externalising symptoms were observed for any body composition measures. As such, no differences were observed between conditional indirect effects (Table 8-13).

**Table 8-12.** Standardised Coefficients for Mediating Paths for Body Composition and Body Mass Index across Fast-Food Density Groups

<i>Model</i>	<i>Group 1: High Fast-Food Density</i>				<i>Group 2: Middle</i>				<i>Group 3: Low Fast-Food Density</i>			
	$\beta$ Coefficient	Lower CI	Upper CI	P Value	$\beta$ Coefficient	Lower CI	Upper CI	P Value	$\beta$ Coefficient	Lower CI	Upper CI	P Value
<b><i>Fat Mass Index (FMI)</i></b>												
<i>Total Effect</i>	-0.15	-0.28	-0.013	0.032	-0.058	-0.14	0.024	0.16	-0.12	-0.17	-0.061	<0.001
<i>Total Indirect</i>	-0.031	-0.097	0.036	0.37	-0.01	-0.043	0.024	0.57	-0.055	-0.079	-0.031	<0.001
<i>Internalising Indirect</i>	-0.027	-0.52	0.46	0.91	-0.029	-0.073	0.015	0.20	-0.038	-0.063	-0.012	0.004
<i>Externalising Indirect</i>	-0.003	-0.47	0.46	0.99	0.019	-0.041	0.079	0.53	-0.017	-0.045	0.011	0.24
<i>Direct Effect</i>	-0.11	-0.27	0.037	0.14	-0.049	-0.13	0.035	0.26	-0.063	-0.11	-0.014	0.012
Path a1: Income to Internalising	-0.34	-0.51	-0.16	<0.001	-0.17	-0.31	-0.029	0.018	-0.27	-0.64	0.098	0.15
Path a2: Income to Externalising	-0.31	-0.46	-0.15	<0.001	-0.25	-0.37	-0.13	<0.001	-0.29	-0.40	-0.18	<0.001
Path b1: Internalising to FMI	0.088	-1.43	1.61	0.91	0.18	-0.047	0.40	0.12	0.14	-0.063	0.34	0.18
Path b2: Externalising to FMI	0.002	-1.55	1.55	1.00	-0.077	-0.31	0.15	0.51	0.06	-0.038	0.16	0.23
<b><i>Fat-Free Mass Index (FFMI)</i></b>												
<i>Total Effect</i>	-0.014	-0.11	0.081	0.77	0.017	-0.042	0.076	0.57	-0.052	-0.089	-0.014	0.007
<i>Total Indirect</i>	-0.018	-0.27	0.23	0.89	-0.012	-0.033	0.009	0.26	-0.03	-0.044	-0.017	<0.001
<i>Internalising Indirect</i>	0.43	-1.44	2.30	0.65	-0.004	-0.033	0.025	0.79	-0.013	-0.031	0.005	0.16
<i>Externalising Indirect</i>	-0.45	-2.22	1.32	0.62	-0.008	-0.053	0.037	0.72	-0.017	-0.038	0.004	0.11
<i>Direct Effect</i>	0.004	-0.25	0.25	0.97	0.029	-0.034	0.092	0.37	-0.021	-0.056	0.013	0.23
Path a1: Income to Internalising	-0.34	-0.51	-0.16	<0.001	-0.054	-0.305	-0.029	0.017	-0.27	-0.094	-0.056	0.16
Path a2: Income to Externalising	-0.31	-0.46	-0.15	<0.001	-0.12	-0.37	-0.13	<0.001	-0.29	-0.19	-0.11	<0.001
Path b1: Internalising to FFMI	-1.31	-7.31	4.68	0.67	0.15	-0.15	0.20	0.78	0.047	-0.13	0.81	0.31
Path b2: Externalising to FFMI	1.49	-4.60	7.57	0.63	0.14	-0.15	0.21	0.72	0.061	-0.03	0.49	0.096
<b><i>Fat-Free Mass Index (Adjusted for FMI)</i></b>												
<i>Total Effect</i>	0.066	-0.009	0.14	0.083	0.045	-0.003	0.094	0.068	0.005	-0.018	0.027	0.69
<i>Total Indirect</i>	-0.017	-0.22	0.19	0.87	-0.008	-0.025	0.008	0.33	-0.003	-0.01	0.004	0.42
<i>Internalising Indirect</i>	0.37	-1.03	1.76	0.61	0.011	-0.009	0.031	0.29	0.004	-0.007	0.016	0.44
<i>Externalising Indirect</i>	-0.38	-1.74	0.97	0.58	-0.019	-0.051	0.013	0.24	-0.007	-0.02	0.005	0.23
<i>Direct Effect</i>	0.082	-0.10	0.27	0.38	0.054	0.003	0.10	0.038	0.008	-0.015	0.03	0.51
Path a1: Income to Internalising	-0.31	-0.49	-0.14	<0.001	-0.16	-0.30	-0.021	0.024	-0.25	-0.54	0.051	0.11

Path a2: Income to Externalising	-0.29	-0.44	-0.14	<0.001	-0.25	-0.37	-0.12	0	-0.27	-0.37	-0.17	<0.001
Path b1: Internalising to FFMI	-1.2	-5.90	3.51	0.62	-0.068	-0.18	0.046	0.24	-0.018	-0.068	0.032	0.48
Path b2: Externalising to FFMI	1.33	-3.48	6.13	0.59	0.078	-0.045	0.2	0.21	0.028	-0.018	0.073	0.23
<b>Fat Mass: Fat-Free Mass Ratio (FM:FFM)</b>												
Total Effect	-0.098	-0.22	0.023	0.11	-0.04	-0.11	0.028	0.25	-0.097	-0.14	-0.054	<0.001
Total Indirect	-0.024	-0.09	0.043	0.48	-0.002	-0.033	0.029	0.92	-0.039	-0.057	-0.021	<0.001
Internalising Indirect	-0.031	-0.46	0.40	0.89	-0.03	-0.072	0.013	0.17	-0.034	-0.056	-0.012	0.002
Externalising Indirect	0.007	-0.40	0.42	0.97	0.028	-0.029	0.085	0.33	-0.005	-0.027	0.016	0.64
Direct Effect	-0.074	-0.22	0.066	0.30	-0.039	-0.11	0.035	0.31	-0.058	-0.098	-0.019	0.004
Path a1: Income to Internalising	-0.33	-0.51	-0.16	<0.001	-0.17	-0.31	-0.029	0.018	-0.27	-0.093	-0.055	0.15
Path a2: Income to Externalising	-0.3	-0.46	-0.15	<0.001	-0.25	-0.37	-0.13	<0.001	-0.29	-0.19	-0.11	<0.001
Path b1: Internalising to FM:FFM	0.10	-1.25	1.46	0.88	0.18	-0.029	0.39	0.092	0.13	0.022	0.097	0.18
Path b2: Externalising to FM:FFM	-0.03	-1.41	1.35	0.97	-0.11	-0.33	0.10	0.3	0.018	-0.014	0.023	0.63
<b>Body Mass Index (BMI)</b>												
Total Effect	-0.099	-0.24	0.041	0.16	-0.021	-0.097	0.055	0.59	-0.11	-0.16	-0.057	<0.001
Total Indirect	-0.024	-0.16	0.11	0.73	-0.011	-0.045	0.022	0.51	-0.05	-0.071	-0.029	<0.001
Internalising Indirect	0.17	-0.61	0.95	0.67	-0.031	-0.077	0.015	0.18	-0.035	-0.061	-0.009	0.009
Externalising Indirect	-0.19	-0.90	0.51	0.59	0.02	-0.041	0.081	0.53	-0.016	-0.044	0.013	0.29
Direct Effect	-0.075	-0.27	0.12	0.44	-0.01	-0.092	0.072	0.82	-0.06	-0.11	-0.012	0.015
Path a1: Income to Internalising	-0.34	-0.5	-0.17	<0.001	-0.17	-0.31	-0.035	0.014	-0.27	-0.57	0.029	0.076
Path a2: Income to Externalising	-0.29	-0.44	-0.14	<0.001	-0.25	-0.37	-0.13	<0.001	-0.29	-0.37	-0.20	<0.001
Path b1: Internalising to BMI	-0.51	-2.78	1.76	0.66	0.19	-0.042	0.41	0.11	0.13	-0.037	0.29	0.13
Path b2: Externalising to BMI	0.66	-1.61	2.94	0.57	-0.079	-0.31	0.16	0.51	0.054	-0.042	0.15	0.27

**Table 8-12 Footnote:** Table showing total, indirect and direct effects for BMI and each body composition outcome in each fast-food density group. Path A is the regression path from income to internalising/externalising symptoms, where the symptoms are the outcome. Path B is the regression path for internalising/externalising symptoms to FMI where FMI is the outcome.

**Table 8-13.** Unstandardised Regression Coefficients for Conditional Indirect Effects and Interaction Terms for Fast-Food Density

Model	Group 1: High Fast-Food Density				Group 2: Middle				Group 3: Low Fast-Food Density				P Value Difference**		
	Coefficient	SE	P Value	P Value Interaction*	Coefficient	SE	P Value	P Value Interaction*	Coefficient	SE	P Value	P Value Interaction*	G1 Vs G3	G1 Vs G2	G2 Vs G3
<b>FMI</b>															
Internalising	-0.044	0.055	0.43	0.93	-0.08	0.069	0.24	0.64	-0.049	0.017	0.005	n/a	0.93	0.67	0.64
Externalising	0.018	0.063	0.77	0.69	0.007	0.078	0.93	0.86	-0.007	0.019	0.73	n/a	0.69	0.91	0.86
<b>FFMI</b>															
Internalising	0.009	0.044	0.85	0.66	-0.022	0.044	0.62	0.82	-0.011	0.016	0.47	n/a	0.66	0.62	0.82
Externalising	-0.059	0.057	0.30	0.54	-0.012	0.052	0.82	0.81	-0.025	0.018	0.17	n/a	0.54	0.51	0.81
<b>FFMI (Adjusted for FMI)</b>															
Internalising	0.029	0.03	0.33	0.67	0.014	0.023	0.54	0.93	0.016	0.011	0.14	n/a	0.67	0.69	0.93
Externalising	-0.065	0.041	0.11	0.28	-0.008	0.026	0.77	0.64	-0.021	0.013	0.092	n/a	0.28	0.23	0.64
<b>FM:FFM Ratio</b>															
Internalising	-0.003	0.004	0.44	0.93	-0.006	0.004	0.13	0.43	-0.003	0.001	0.006	n/a	0.93	0.50	0.43
Externalising	0.001	0.004	0.84	0.87	0.003	0.005	0.47	0.49	0.00	0.001	0.88	n/a	0.87	0.67	0.49
<b>BMI</b>															
Internalising	-0.068	0.13	0.59	0.93	-0.20	0.13	0.12	0.36	-0.08	0.036	0.027	n/a	0.93	0.43	0.35
Externalising	-0.02	0.15	0.89	0.99	0.084	0.15	0.58	0.49	-0.023	0.04	0.56	n/a	0.99	0.59	0.49

**Table 8-13 Footnote:** Estimates presented are unstandardised coefficients. \* Indicates p-value for path between the interaction of environmental dummy variables with internalising/externalising symptom to the outcome. \*\* G1 = Group 1, G2 = Group 2, G3 = Group 3. The p-values for the interaction term for group 1 will be the similar or the same as the p-value for the difference between group 1 and 3, and the p-value for group 2 will be similar to the p value for the difference between group 2 and 3. No p-value for the interaction term is presented for Group 3, as group 3 was the reference category in models. Abbreviations: FMI – fat mass index; FFMI – fat-free mass index; FM:FFM – fat mass to fat-free mass; BMI – body mass index; SE – standard error; n/a – not applicable.

## 8.4 Discussion

### 8.4.1 Summary

This chapter aimed to investigate the mediating role of internalising and externalising symptoms, and the moderating role of environmental conditions on these mediating pathways. Two hypotheses were tested, and a summary of the findings in relation to the hypotheses is provided below.

**Hypothesis 9:** *Social inequalities in body composition (and BMI) among children and adolescents in the UK are mediated by internalising and externalising symptoms, so that those in disadvantaged circumstances exhibit higher levels of these symptoms, which in turn are related to less healthy body composition (higher FM and lower FFM).*

In line with hypothesis 9, the results indicated that internalising symptoms mediated the association between household income and FMI and FM:FFM ratio. However, there was no evidence that externalising symptoms likewise mediated the relationship. Those in disadvantaged socioeconomic circumstances exhibited higher levels of internalising and externalising symptoms, but only internalising symptoms were associated with increased FMI and FM:FFM ratio. For BMI, the combined effect of internalising and externalising symptoms together mediated associations, but not each on their own.

Similarly, internalising and externalising symptoms combined mediated associations for FFMI such that those with lower household income exhibit higher levels of both symptoms, and these in turn were related to higher levels of FFMI. When adjusted for FMI, the opposing directions of the positive direct effect, and negative indirect effect for externalising symptoms, resulted in inconsistent mediation and therefore no overall association between income and FFMI. In models where FMI was treated as a mediator and not a confounder, the indirect and total effects did not substantially differ from the unadjusted analysis, whilst there was evidence of a positive direct effect in which higher income was related to higher FFMI.

**Hypothesis 10:** *Environmental characteristics modify the mediating effect of internalising and externalising symptoms among children and adolescents in the UK, such that those experiencing a more obesogenic environment show greater mediation by internalising and externalising symptoms, such that the association between symptoms and less healthy body composition is strongest in the most obesogenic areas.*

The results were consistent with hypothesis 10 for green spaces and area deprivation measured by the IMD, which both moderated the mediating effects of internalising symptoms. Although income was similarly related to internalising symptoms across deprivation groups, in areas of higher deprivation and fewer green spaces the association between internalising symptoms and FMI, BMI and FM:FFM ratio was typically stronger.

Both the IMD and green spaces also moderated the mediating path for externalising symptoms, although in the opposite direction to that hypothesised. Whilst lower income was still related to higher levels of externalising symptoms, higher externalising symptoms were related to lower FMI, BMI and



FM:FFM ratio in the most deprived areas, and associated with higher FMI, BMI and FM:FFM ratio in the most advantaged areas. Neither green space nor the IMD moderated mediating paths for FFMI, indicating moderating effects largely impact the adiposity-based components of body composition.

Contrary to hypothesis 10, no moderating effect for fast-food density or domestic gardens were observed for any of the body composition outcomes.

#### 8.4.2 Comparison to Previous Literature

Few studies have investigated the mediating role of psychological symptoms in the relationship between SEP and body composition and BMI in childhood and adolescence, especially in relation to body composition, although each of the separate pathways has previously been tested. The results of the current study are consistent with work carried out previously in the MCS. Higher levels of internalising and externalising symptoms have been related to later obesity measured by BMI, at ages 3, 5 and 11 [41, 42] whilst externalising symptoms have been found to be related to obesity at ages 3 and 5 [41]. Other research using the MCS found internalising symptoms and obesity were more likely to co-occur as children get older [450]. Additionally, both the prevalence of overweight and obesity and mental health problems were highest in the most deprived IMD deciles [451]. Research conducted in adults in the USA has directly tested mediating pathways via depression. Findings from the USA study were consistent with the findings in MCS demonstrating that depressive scores mediated the inverse relationship between SEP and WC and WHR, but not trunk fat, in white women [140]. However, the USA paper did not test associations in children. This thesis adds to the existing literature by explicitly demonstrating that internalising symptoms mediate associations between parental income in childhood and adiposity in adolescents.

Previous work in the British Birth Cohorts suggests a changing relationship between psychological symptoms and life course BMI. In the 1946 cohort, those who exhibited internalising symptoms in childhood had lower BMI in adolescence, but faster increases in BMI across adulthood [39]. For girls in the 1958 cohort, stress at age 7 was associated with increased risk of obesity at 16 years [452]. In the 1970 cohort, individuals with conduct and hyperactivity problems at ages 5 and 10 had increased risk of obesity in later adult life [453]. Differences in associations between the cohorts hint at the possible role of the environment, with the earlier cohorts demonstrating less evidence of associations in early life before the onset of obesogenic environments in the UK, whilst there was greater evidence of associations in later adult life after the onset.

Limited work has looked directly at how the mediating role of internalising and externalising symptoms may differ across environmental conditions, especially when investigating mediation for body composition. Research conducted previously in the MCS found stronger associations between household income and FM% in the areas with highest and lowest deprivation [378]. This is consistent with the current analysis which finds some evidence of stronger total effects between household income and body composition in the least and most deprived areas by IMD and green spaces. The current study extends this by demonstrating that internalising and externalising mediating paths differ

in the most and least deprived areas, which somewhat explains the overall difference in inequality gradients between area deprivation.

Relating to the results for green spaces, research conducted in the MCS has found access to gardens, parks and playgrounds reduced peer, conduct and hyperactivity problems among children aged 3-5, whilst neighbourhood green spaces were associated with lower levels of emotional problems among poorer children [176]. The results of the analysis presented in this chapter are consistent with these previous findings, as there was typically a weaker association between income and emotional problems in the greener areas. The current results expanded the previous work by exploring how internalising symptoms mediate relations between income and body composition and finds that in general among adolescents living in less green areas, stronger mediating pathways exist. This was largely driven by weaker or no association between internalising symptoms and adiposity in greener areas.

### 8.4.3 Interpretation of findings

#### *8.4.3.1 Index of Multiple Deprivation*

##### **Internalising Symptoms**

The findings in this chapter lend support to the hypothesis that in more deprived neighbourhoods, which tend to be more obesogenic, there is an increase in maladaptive health behaviours or coping strategies that increase risk of adiposity among those who exhibit greater internalising symptoms. These maladaptive coping strategies are likely to be related to health behaviours that results in increases in adiposity, such as overeating or increased sedentary behaviours. Such pathways may be explained by the affect regulation model [106], whereby eating, or in particular bingeing, is used as a tool to regulate negative emotions [143]. Studies have supported this by finding: associations between depression and emotional eating among 8-18 years olds both seeking and not seeking treatment for obesity [142]; associations between stress-related eating and obesity among adolescents in Finland, especially among girls [454]; and highest BMI among those who are stress-driven eaters in a population based study of adult men and women in Finland [455]. A systematic review of 13 studies in children also supports the association between stress and unhealthy eating behaviours [144].

A model by Hemmingsson et al. (2014) [456] emphasised the role of maladaptive coping strategies such as over-eating in response to stress accumulated over the life course (e.g. stress from living in disadvantaged circumstances). Psychological and emotional distress is expected to be higher among children and adolescents growing up in disharmonious environments, driven by experiencing disadvantage, resulting in maladaptive coping strategies and weight gain [456]. Evidence supports the pathways between psychological symptoms and obesity through eating as a regulatory mechanism for emotions [106, 142-144]. However, the current findings do not find that living in areas of high fast-food density increases the strength of the mediating pathway, indicating that it may be the social, psychological and cultural aspects of area deprivation [148] that promote maladaptive coping strategies as opposed to increased access to fast-food. It may also be that the impact of increased

fast-food density on intake is homogenous across the population and therefore does not have a moderating effect on internalising and externalising symptoms.

More generally, as adolescence is a period of rapid development socially, psychologically and physically [15], behaviours that develop as a consequence of internalising symptoms in childhood and adolescence are likely to be maintained and may become harder to alter as adolescents age. The maladaptive health behaviours, such as overeating or binge eating, that develop over this period may be maintained over a longer period and thus have consequences for inequalities in body composition over the life course. Increasing adiposity also subsequently results in reductions in physical activity [457, 458] which subsequently may also impact body composition, including the ratio of FM to FFM.

### Externalising Symptoms

The results observed for externalising symptoms were contrary to the hypothesis which set out that externalising symptoms would negatively mediate the associations, as it was hypothesised externalising symptoms are related to increased impulsivity and a tendency to overeat. However, the results find externalising symptoms in more deprived settings are related to lower levels of adiposity. This may be explained by the fact that externalising symptoms, which are associated with conditions such as ADHD, are also typically characterised with hyperactivity and therefore may co-present with higher levels of physical activity [143]. Previous research using the MCS found that less sedentary behaviour at age 7 was predictive of ADHD symptoms and diagnosis at age 14 [459]. This is supported by systematic reviews that have also identified a link between activity levels and ADHD symptoms [460].

The study conducted in the MCS identified lower sedentary activity, rather than higher physical activity time, as a key predictor of both SDQ hyperactivity/inattention scales and ADHD diagnosis. This may support a pathway opposite to the one initially hypothesised, where externalising symptoms are associated with reduced sedentary time, and as a result there would be inconsistent mediation where the indirect effect opposes the direct effect. We only see this direction of mediation in the most deprived areas suggesting that externalising symptoms, in particular hyperactivity symptoms, may be exacerbated in more deprived areas and in areas with fewer green spaces. Previous research supports this hypothesis by demonstrating higher cases of ADHD among the most deprived areas according to the IMD in England [461]. The current work generally found slightly stronger associations between income and externalising symptoms in the most deprived setting.

Mediation by externalising symptoms in the direction hypothesised was only observed in the middle and low IMD deprivation groups and middle and high green space density groups. For the IMD, it is possible that relative individual-level inequality gaps could help explain the observed indirect effect, which are more apparent in more advantaged areas. Previous research has identified the role of income rank on mental wellbeing and highlighted that relative rank or perceived rank has high impact on psychological wellbeing [462]. It is possible that those from lower income households in more affluent areas have lower wellbeing as a result of relative position.

A study in Korea highlighted that income inequality of an area, but not absolute poverty, was related to externalising symptoms in children [463]. Research elsewhere has demonstrated that adolescents who are relatively poorer compared to their neighbourhood exhibit greater problem behaviours, including depression, social phobia, aggression and parental conflict [464]. A Canadian study demonstrated that relative deprivation contributed to risk factors for obesity independent of absolute affluence, with those experiencing greater relative deprivation adopting less healthy behaviours [465]. It is possible that this effect is exacerbated among individuals with externalising symptoms where these healthy decisions become harder, and impulsivity is a more dominant trait than hyperactivity. As such, it is likely that less healthy behaviours, such as overeating and binge eating are adopted in response to symptoms experienced.

#### *8.4.3.2 Green Spaces*

The positive impact of green spaces on mental health has been widely documented [43-45] including among adolescents [46]. It is possible that in areas with higher levels of green space there are increases in positive affect – the positive ways in which people regulate emotions and interactions – and this alters the coping strategies adopted among individuals exhibiting internalising symptoms. Again, this is likely explained by differences in health behaviours, in particular differences in physical activity levels and amount of sedentary time. As adolescence is an important period of development, healthy behaviours, such as regular physical activity, that develop as coping strategies in areas with better green space access, are more likely to be maintained over the life course. Previous research has demonstrated that green spaces improve mental wellbeing and reduce mental distress [173, 174]. Among individuals who move to greener areas, mental health as measured by the GHQ improves, whilst among those who move to less green areas it worsens [175]. Other research has demonstrated that positive affect is associated with increased physical activity, and that this mediates associations between affect and heart disease and all-cause mortality [466]. Additionally, green spaces are associated with increased physical activity [47, 48], with those living in closer proximity to urban green spaces more likely to meet recommended guidelines for physical activity [170]. This may increase positive wellbeing among adolescents exhibiting internalising symptoms and may increase the likelihood that positive coping strategies, such as physical activity are adopted, as opposed to negative strategies such as overeating.

#### *8.4.3.3 Domestic Gardens*

Domestic gardens were included as a moderator as it was hypothesised that they may better reflect the environmental areas that people use, and previous research in the MCS has found access to gardens to be related to reduced emotional and behavioural problems between the ages of 3 and 5 [176]. However, there was little evidence of differences in mediation of associations between income and body composition by domestic garden density. This is possibly driven by the fact access to domestic gardens may be inconsistent even in areas with a moderate number of gardens (i.e in cities), and that garden quality may vary. Research into green spaces has previously indicated that quality of green spaces is more important for use than quantity and access [467], and this may be true of gardens also. Furthermore, research has demonstrated people are willing to travel for quality green

space use [468]. Therefore, domestic gardens may not be related to health behaviours such as physical activity if this is more likely to be done in alternative settings. Additionally, density of domestic gardens was typically negatively and poorly correlated with other environmental measures, indicating that it may capture a different aspect, if any, of deprivation compared to other environmental measures.

#### *8.4.3.4 Fast-Food Density*

Fast-food density was also included as a moderator yet showed little impact on the internalising and externalising symptoms mediating paths. Fast-food density has previously been linked to increased levels of obesity, including in the MCS where closer proximity to fast-food outlets was related to increased levels of FM% and BMI, although the effect sizes were small [469]. Maternal education was found to be a potential mechanism explaining these associations, with fast-food proximity having little association with increased obesity among those children with highly educated mothers. Another study conducted in the MCS demonstrated that associations between fast-food density and obesity are confounded by the IMD [470]. Whilst research in the UK of children aged 4-5 and 10-11 found fast-food density was related to weight status, it had only a small mediating role between deprivation and obesity in older children [152]. Additionally, positive associations between fast-food density and weight status only remained for older children, but not younger children, once other environmental characteristics were adjusted for [152].

#### **8.4.4 Strengths and Limitations**

One of the key strengths of these analyses is the use of latent factors based on observed indicators, as opposed to a sum score of those indicators, to model internalising and externalising symptoms, which reduced the measurement error in the mediator. Additionally, the assumption of temporality was met, in that the measurement of the independent variables preceded the mediator, and the mediator preceded the dependent variable. However, as traits such as internalising and externalising symptoms and body composition track over time, it is not possible to fully rule out reverse causality between symptoms and body composition, which may be better explored by more complex models such as a cross-lagged model. Finally, confounding variables were considered and included in the model, which reduces the risk of bias and reduces likelihood that causal assumptions were violated. However, it is still possible that there were unmeasured or unknown confounders which were not able to be accounted for. Another strength is that causal mediation methods were adopted as opposed to the more traditional Barron and Kenny method, which have a number of flaws as outlined in section 8.2.2.

Because of the limitations of the software, in the interaction models the ordinal SDQ items were treated as continuous to allow the latent factor structure identified in Chapter 7 to be adopted. Although the items of the SDQ are Likert scales and therefore could be treated as continuous data, there is no evidence that the categories can be treated as interval scaled. Additionally, ordinal variables with three categories have limited information justifying their use as continuous variables. However, the estimates from the mediation model when using the MLR estimator and treating the SDQ items as continuous were largely similar to those from the same models using the WLSMV

estimator and treating the same items in the factor structure as categorical. Therefore, estimates of the interaction model using the MLR estimator are likely to be consistent with those if it was possible to use the WLSMV estimator.

The exception to this was for FFMI models adjusted for FMI, where estimates differed in the sensitivity analysis compared to the main mediation analysis. Therefore, caution should be taken in interpreting the interaction effects for this outcome. However, by considering the two approaches to assess moderated mediation, a triangulation of evidence was used, and the results of the WLSMV sub-group analysis were supported by those from the interaction models.

A broad range of environmental factors were used that allowed for investigation of different elements of the environment. This was in line with recommendations to expand academic understanding of what constitutes an obesogenic environment [109]. However, the analysis was only able to consider the environments in which the cohort members lived. It is likely that cohort members interact with environments outside of their immediate vicinity and this was not taken into account. For example, previous research has found people often travel to access green spaces, and that fast-food outlets near school or work may be more important than those near where people live [469]. Additionally, fast-food density is only investigated within an 800m radius of the cohort member's house. It is possible that outlets at distances further than this may be important [469].

Multiple imputation was adopted for SDQ items and household income in mediation models and sub-group analyses alongside estimation using WLSMV. This has previously been found to be a good approach to handling missing data [219], both in term of producing unbiased estimates and in maintaining sample sizes. However, it was not possible to impute environmental characteristics as the plausible values imputed varied across imputations, preventing convergence of models in sub-group analyses. Similarly, this was the case for ethnicity, which was entered into models as a dummy variable. However, for green spaces, domestic gardens, the IMD and ethnicity, missing data was less than 5% in the possible sample. Missing data of 5% or less has previously been used as a cut off for negligible rates of missing data and therefore exclusion of these variables from the imputation models is unlikely to have biased the estimates. However, for fast-food density missing data was 9.4% in the feasible sample (cohort members residing in Great Britain with BMI and body composition at age 17). It is therefore possible that estimates may be biased for fast-food density because of missing data.

The environmental characteristics were re-categorised into three groups to maintain sample size and power in sub-group analyses. However, in doing so, information was lost about the distribution of these variables within groups. Additionally, because data on fast-food density and domestic gardens were only collected in Great Britain and England, respectively, there was loss of sample size and results in these analyses may not be representative of the rest of the UK. Further, the fast-food density measure was highly skewed, meaning that analysis in the high-density fast-food group may have been under powered due to a lower sample size. The analysis did see similar effect sizes for mediating paths across fast-food density groups, but in areas with higher density of fast-food outlets where sample sizes were smaller, confidence intervals were wider.

This analysis tested a moderating effect between the mediator and body composition and BMI outcomes. However, it is also possible that moderation occurs on the path from income to the mediator. In the stratified sub-group analysis, which was non-specific in the path being moderated, there was a tendency for the association between the mediator and body composition and BMI to drive the differences in the indirect effect between groups, consistent with the hypothesis tested. The association between income and internalising or externalising symptoms was generally consistent between groups, with the exception of fast-food density and domestic gardens. Therefore, future analysis could explore if moderation occurs for the path between SEP and internalising and externalising symptoms.

Similar to analysis conducted in Chapter 6, only a small number of covariates that were considered to confound associations of interest were included in the models. However, like Chapter 6, there are a number of other covariates which could have been considered for inclusion, such as maternal smoking and depression, and for models of moderation by environmental characteristics, rural or urban location. This latter adjustment may have been important for green spaces, where the type and quality of green spaces is likely to differ by rural/urban characteristics [471]. Likewise, both maternal smoking and depression are socially patterned [128, 391, 392], but are unlikely to be a causal determinant of family SEP, and therefore represent mediators of associations. In addition to the plausible pathways with body composition discussed in Chapter 6, maternal depression [472] and maternal smoking [473] are associated with internalising/externalising symptoms in children. For maternal depression this is explained through negative maternal behaviours, parenting styles (i.e. excessive criticism) and interactions between mother and child [473, 474]. For maternal smoking, associations with internalising and externalising symptoms are explained through impacts on cognitive development are thought to arise from smoking in utero [475], but may also be confounded by other psychosocial factors [476]. Therefore, there are multiple mediating pathways by which maternal smoking and depression might explain some of the SEP gradient in body composition, and these could be explored in further research. The analysis presented in this chapter only intended to isolate the indirect effect of internalising and externalising symptoms in children and adolescents and did not explore other potential pathways.

# Chapter 9 Discussion

This thesis aimed to describe inequalities in body composition and investigate the reasons for inequalities, as understood through the environment and emotional and behavioural problems in late childhood. The novel analyses presented in this thesis, using both a systematic review and analysis conducted in the MCS, have addressed the gaps identified in the literature review (Chapter 2) and tested the hypotheses set out in Chapter 3. This chapter summarises the findings of these analytic chapters, discusses them in the context of theoretical underpinnings and other relevant literature, reviews policy implications, and discusses methodological challenges. Finally, potential avenues for future research are considered.

## 9.1 Summary of Key Findings

The thesis hypothesised that disadvantage is related to higher adiposity and lower fat-free mass in childhood and adolescence, and that these inequalities can be partly explained by psychological characteristics and moderating effects of the environment. The objectives of the thesis were to:

1. Systematically review the literature on socioeconomic inequalities in body composition.
2. Assess the degree of socioeconomic inequality in directly measured body composition in childhood and adolescence using a contemporary UK cohort.
3. Assess the appropriateness of using the Strengths and Difficulties Questionnaire to measure internalising and externalising symptoms as mediators, in a contemporary cohort of children in the UK
4. Assess the mediating role of internalising and externalising symptoms in understanding inequalities in body composition, and to explore moderating effects by the environment.

The first objective was achieved through a systematic review that had global reach and explored the association between SEP and body composition in both adults and children (Chapter 5). The systematic review demonstrated that inequalities in adiposity as measured by FM are similar to those observed for BMI, with socioeconomic advantage typically being related to lower fat in HICs and higher fat in MICs. For FFM, the associations were less clear, with some evidence that socioeconomic advantage was associated with greater FFM in both HICs and MICs. The review demonstrated that inequalities for both FM and FFM were more apparent in children than adults, and among females and girls compared to males and boys, with the exception of FFM which found no differences between boys and girls.

The second objective was achieved by demonstrating that early life SEP, measured both by family circumstances and area deprivation, was related to childhood and adolescent body composition in the MCS. Those living in disadvantaged circumstances had higher levels of FMI and a higher FM:FFM ratio (Chapter 6). This work also demonstrated the life course impact of inequalities, by showing that socioeconomic circumstances at birth were related to inequalities in early childhood and that these inequalities continued into adolescence and widened with age.



The third objective was addressed in Chapter 7 which demonstrated the appropriateness of using the SDQ to capture internalising and externalising symptoms in early adolescence. These analyses revealed that a second-order factor structure was most appropriate in early adolescence, with separate domains for internalising and externalising. Importantly, Chapter 7 demonstrated that the SDQ captures internalising and externalising symptoms in similar ways between boys and girls, but also between individuals living in different environmental conditions (i.e., high and low levels of deprivation), therefore permitting sub-group analysis by these characteristics.

The final objective of this thesis was addressed in Chapter 8, by exploring the mediating role of internalising and externalising symptoms and the effect of the environment in which individuals live, in explaining the observed inequalities in body composition. Chapter 8 demonstrated that internalising symptoms mediated associations between SEP in childhood and both FMI and FM:FFM ratio. Moreover, both internalising and externalising symptoms mediated associations between SEP in childhood and FFMI and BMI. Additionally, Chapter 8 demonstrated that these mediating paths were moderated by the environment. Of the environmental measures considered (IMD, green spaces, domestic gardens and fast-food density), the measure of area deprivation (IMD) and green spaces had moderating effects on mediating pathways. In particular, internalising symptoms more strongly mediated the relationship between childhood SEP and adiposity-related outcomes (FMI, FM:FFM and BMI) in the most deprived and least green areas.

## 9.2 Interpretation of Findings

Understanding the results of this thesis requires recognition of the socio-political and historical context in which the findings are derived. In particular, the nutrition transition and secular changes to the environment that have occurred over the last century in HICs, are important in placing the results of this thesis in context. Differences in inequalities in body composition between HICs and MICs identified in the systematic review likely reflect different stages of the nutrition transition [333], with energy-dense food being more accessible to those in the most advantaged positions in MICs [334], whilst cheaper and therefore more accessible for those in disadvantaged positions in HICs [336]. The greater number of studies that find disadvantaged SEP to be associated with higher FM in children compared to adults in HICs, likely reflects secular changes to the environment, which have become increasingly obesogenic over time, and impacts disadvantaged groups in society the most.

These two processes are also important for understanding the inequalities in body composition observed in the analysis of the MCS. The UK has gone through the nutrition transition and the environment has become increasingly obesogenic, and children in the MCS were born into a more obesogenic environment than prior generations. As such, the inequalities in body composition observed in the MCS are predominantly in the same direction as those observed in the systematic review for children in HICs. Given what is known about changing inequalities in BMI across the British Birth Cohorts [28], the inequalities observed in the MCS are likely to reflect secular changes to the inequalities. However, a life course effect cannot be ruled out in conjunction with these secular changes. Indeed, research conducted in BMI also typically shows inequalities increase with age within the same generation [28, 29]. It is probable that as MCS cohort members age, FM will continue to

increase most rapidly in the disadvantaged groups, representing a life course effect alongside a secular change. As such inequalities in FM in adulthood will likely be greater than in childhood, but also greater than previous generations at the same age.

There are important implications of these findings for population health, with children born today likely to spend more of their lives overweight and with a less healthy body composition. Research has highlighted that the earlier age at which individuals first become overweight, measured by BMI, is related to increased risk of health outcomes in later life, such as chronic kidney diseases [477] and cardiovascular disease [478]. This is likely the case for unhealthy body composition too. Both BMI and body composition track across the life course [9, 239, 384], and life course changes in BMI are typically increases rather than decreases, most often driven by increasing FM as opposed to muscle. The findings of this thesis, along with previous research using BMI, indicate lifetime exposure to unhealthy body composition is likely to affect the disadvantaged in society most. If the inequalities observed in the MCS continue into adulthood, or widen as expected, the excess adiposity and the resulting extended state of inflammation, are likely to have negative consequences for subsequent population health, especially in the most disadvantaged groups. This in turn will likely increase health inequalities across the adult life course for other key health indicators.

FFMI, as well as the ratio of FM to FFM, were considered in this thesis. Less research has considered inequalities in FFM or ratio measures. This is despite them having important consequences for health in addition to FM, that may differ to those for FM. For example, skeletal muscle is a site of glucose uptake [479], and increases in relative muscle mass are associated with decreased insulin resistance and diabetes risk [90], and also CVD mortality [79], whilst low FFM is related to poorer functional capability in older age [87]. Although this thesis found modest evidence of inequalities in FFMI, there is some evidence that disadvantaged SEP is related to lower FFMI when adjusted for FMI. In addition, there is an association between greater disadvantage and higher FM to FFM ratio which gets wider with age as the proportion of FM increased more rapidly than the proportion of FFM in the most disadvantaged groups. This is an important contribution to the field and encourages consideration of which components of body compositions are driving inequalities in body size more generally (i.e BMI), and how this may relate more broadly to health inequalities.

Throughout this thesis a life course approach to inequalities was adopted, by investigating how disadvantage in early life relates to subsequent body composition outcomes and their trajectories. Although the systematic review identified only a small number of studies, there was indication that childhood disadvantage was related to greater adiposity in adulthood. In the life course, adolescence is an important time of development [15], and Chapter 6 demonstrated how inequalities in body composition emerge over this period of life. The analysis in the MCS showed that disadvantage, as indicated by family income and area deprivation in infancy, was associated with adiposity trajectories across childhood and adolescence with inequalities widening. Additionally, inequalities in FFMI change direction over adolescence (after adjustment for FMI), so that at the age of 17 those in the most advantaged position have higher FFMI.

The findings of Chapter 6 may be explained through an accumulation of risk model, where different exposures that promote adiposity and are related to early life SEP, cluster and accumulate over the life course [91]. The analysis was also consistent with a social determinants approach that examines the “causes of the causes” and makes assumptions about the ways SEP is related to body composition through socially patterned behavioural risk factors [98, 101, 102]. The clustered and socially patterned risk factors are likely related to physical activity and dietary behaviours [381], family [480] and neighbourhood environments [148], social vulnerabilities [382] and mental health, the latter of which was explored in Chapter 8.

The results of Chapter 8 can be understood through a life course chain of risk model that explores the mechanisms linking disadvantage to body composition [91, 92]. In Chapter 8, emotional and behavioural problems were shown to act as mediators between social circumstances in early life and body composition in adolescence, and therefore explores the psychosocial mechanisms that link disadvantage with body compositions. As such, the findings of the analytic chapters are also consistent with the conceptual framework of the social determinants of health, which highlights the role SEP plays on health outcomes through psychosocial pathways [98]. In particular, that health behaviours such as eating unhealthy foods, and lack of exercise, may be adopted as coping mechanisms in response to stressful circumstances [98]. Consistent with a social determinants approach, Chapter 8 demonstrated that disadvantage is related to mental health in children, which has subsequent consequences for physical health. Associations between internalising and externalising symptoms and body composition are explained through the affect regulation model that sees different coping strategies adopted in response to psychological symptoms [104]. It is hypothesised that those experiencing internalising symptoms are more likely to regulate their psychological state through negative coping strategies, such as comfort eating or bingeing [143].

The different mediating role of internalising and externalising symptoms according to differences in the environment can also be further conceptualised through the affect regulation model [106, 107]. The environment influences the varying affective states, cues and stimuli, and accordingly influences the coping strategies adopted. Previous research has demonstrated that among individuals who are clinically at risk, negative affective states can further increase the risk of engaging in negative coping strategies, whilst cues and stimuli in the environment increase engagement in hedonic risk behaviours among individuals experiencing positive affective states [107]. Relating to the findings in Chapter 8, in more deprived areas, it is likely those experiencing internalising symptoms adopt negative coping strategies whilst those in less deprived areas and greener areas may be more likely to adopt positive strategies to regulate their emotions. The findings in Chapter 8 are consistent with previous work that has highlighted the impact of green spaces [46] and deprivation [52] on mental health and extends this by demonstrating how it may be consequently related to body composition. However, the moderating effect of the environment on behavioural and emotional problems is not straightforward, with externalising symptoms showing counterintuitive associations in the most deprived areas in the analyses in Chapter 8. As discussed in Chapter 8, this is likely explained through higher levels of hyperactivity and lower levels of sedentary time [143, 459].

The finding that the neighbourhood deprivation (as measured by IMD) had a significant impact on body composition trajectories (Chapter 6), as well as having a moderating effect on mediating paths via internalising and externalising symptoms (Chapter 8), can also be understood through the lens of a social determinants of disease approach. The social determinants of disease framework emphasises the impact of social structures and environments on population health [100]. In previous research using the MCS, area deprivation measured by the IMD explained much of the association between fast-food density and adiposity [470], indicating that fast-food density may not be the prime reason for variation in body size according to area. Other research into the life course effects of neighbourhood deprivation on self-rated health found the impact of deprivation accumulates with age, and that tackling inequalities in neighbourhood deprivation may be important in improving population health [481]. A key takeaway message from this thesis is that there are elements of the social and cultural environment related to deprivation that may be important for adiposity outcomes above and beyond the physical environment or individual socioeconomic circumstances [148]. These may include social networks, capital and cultural norms, as well as crime levels and local service provision, related to neighbourhood deprivation [148].

The need for researchers to consider these broader definitions of the environment that incorporate deprivation and social environments is consistent with a social ecological framework [108, 109]. In the UK 2007 Foresight report the diversity of environmental variables related to the obesogenic environment was highlighted, with the environment being grouped into one of five categories: deprivation, availability and access, urban form, aesthetics and quality, and supportiveness [109]. The report called for the research community to identify new and relevant definitions of the environment and to incorporate these into research [109], which has been done in this thesis through adoption of both physical and socioeconomic measures.

### 9.3 Policy Implications

Since 1992 there have been multiple policies introduced in the UK aimed at tackling childhood obesity, with three chapters in the childhood obesity strategy published in 2016, 2018 and 2019 [14]. So far, the policies introduced have been largely unsuccessful, especially in reducing socioeconomic disparities in obesity, while these disparities may increase if interventions are not designed with inequalities in mind [27]. In addition to inequalities in BMI, this thesis demonstrates that inequalities in body composition also exist for adiposity-based components, and to a lesser extent for fat-free components. The social determinants approach to health suggests that policy action should be taken by the government to change environments to improve health [98]. This is repeated in a 2021 Parliamentary Office of Science and Technology (POST) POSTnote that highlighted the need to address the environment in tackling obesity, and the shortcomings of historic policies that have often relied on individual agency to alter behaviours [14]. This thesis supports the need for environmental interventions, by demonstrating the impact of area-deprivation and green spaces on inequalities in adiposity. However, this thesis identified emotional and behavioural problems as a mediator in associations, also highlighting the need for interventions that target the individual to improve mental health or coping strategies, especially in deprived areas where mediation was more apparent.

### 9.3.1 Tackling Individual Level Disadvantage

A key theme that runs across this thesis is the infliction of inequality on society, and the impact this has for mental and physical health. To fully address the disparity in body composition observed, the causes of structural inequality should be tackled, and this requires tackling the social determinants of obesity. Disadvantage should be addressed, especially in early life, to alleviate both mental health and physical health burdens in society [94]. This may be an ambitious goal for policy makers or public health advisors, but many of the patterns observed in this thesis are rooted in structural inequalities that start early in life.

Increasing targeted fundings of schemes which are aimed at improving early life circumstances could therefore be effective, given this thesis demonstrates that SEP in infancy has impacts on body composition across childhood and adolescence. An example is the Sure Start scheme, set up in recognition of the importance of the first few years after birth for health and development. Between 2011 and 2018, funding for Sure Start by local authorities declined by 53%, with larger cuts in more deprived settings [482]. These cuts are thought to have resulted in an increase in childhood obesity compared to levels had the schemes continued at the original funding level [482].

Other schemes that may reduce health inequalities are the introduction of a universal basic income or widespread adoption of the living wage, given this thesis demonstrated inequalities in income are related to differences in body composition. Low pay has previously been linked to higher rates of obesity [483], with the suggestion that increasing pay could reduce levels of obesity. A study in Alaska, where there has been a universal income programme for over 35 years demonstrated that cases of childhood obesity at three-years of age are lower than expected without the programme, equivalent to 500 fewer cases a year, or a 22.4% reduction, in obesity among three-year olds [484]. In 2014, Public Health England reported based on observational studies conducted in London, UK and the USA, that adoption of the living wage could improve psychological health and wellbeing and increase life expectancy [485]. This thesis demonstrated that higher levels of emotional problems in adolescents mediated the association between lower household income and less healthy body composition. Therefore, increasing wages for those on the lowest income may reduce the incidence of obesity mediated through psychological symptoms.

### 9.3.2 Addressing the Environment and Local Communities

Policy approaches that include investment into local communities and deprived neighbourhoods, such as improving access to, and provision of, community centres and sports facilities, may also alleviate inequalities in body composition. The findings in this thesis demonstrated that in more deprived environments, there is greater mediation of inequalities in body composition by psychological symptoms, driven by a stronger association between symptoms and body composition. Lower engagement in physical activity of adolescents living in deprived areas, particularly those with higher levels of internalising symptoms, is a mechanism that could explain the observed pattern of mediating paths. A systematic review showed that environmental accessibility, in particular provision and quality of sports facilities available and perceived safety in accessing them, is a barrier to physical activity engagement among adolescents from disadvantaged socioeconomic circumstances [486]. Among

more advantaged groups, the environmental barriers to physical activity are less pertinent and instead adolescents reported positive experience of accessing sports facilities in their neighbourhoods [486]. Improving the quality, access and provision of sports facilities, which tend to be worse in deprived areas, may remove the barriers that stop poorer individuals engaging in physical activity. This may also weaken the link between psychological symptoms and body composition, by altering the regulatory mechanisms adopted in more disadvantaged areas.

In addition to demonstrating the negative impact of disadvantage and deprivation on body composition, this thesis also demonstrated the positive impacts that green spaces can have on mental health, and consequently adiposity. The benefits of green space for mental and physical health have been demonstrated previously [46], in particular urban green spaces are thought to have benefit by providing areas for physical activity, social interactions and for psychological restoration [487]. Improving the greenness of areas may break the link between internalising symptoms in childhood and less healthy body composition in adolescence. This in turn could help to reduce the inequalities in body composition observed in this thesis. In 2020, Public Health England released a report on the benefits of green spaces, which are felt most by those in disadvantaged groups, and reported that socioeconomic related health inequalities are smaller in greener areas [488]. The report included a call for local authorities to increase collaboration between public health teams and urban planners: to consider green spaces in urban planning; to create new green spaces, especially in areas of deprivation; and, to improve and maintain the green spaces already available [488].

### 9.3.3 High-Agency vs Structural Interventions

The policy approaches discussed so far require a large amount of investment by local and central governments to address inequalities. They largely rely on structural changes and any resulting improvement in body composition inequalities may take time to observe. Among governments and the general public, there is a tendency for high-agency public health interventions, such as educational campaigns, to be favoured [489]. This is because they are often seen as less invasive, tend to be cheaper for governments to implement and it is argued they don't restrict people's freedoms or choices. However, policies relying on individual agency or educational messages to change behaviour are typically less effective and may also be less equitable [489]. Often it is those in advantaged positions most likely to change their behaviour in response to public health messaging [490, 491]. Community-based interventions, those that result in structural changes to the environment, and price-based interventions have been found to be more effective in less advantaged groups and more likely to reduce inequalities in obesity [490, 491]. Examples of community-based and environmental interventions have already been discussed, whilst an example of a price-based intervention is the 2018 Soft Drink Industry Levy taxes introduced in the UK, otherwise known as the "sugar tax". Other taxes and levies could also be considered for other unhealthy and ultra-processed foods, alongside subsidies for healthier foods, which are typically more expensive [336, 492].

Despite high-agency interventions typically being less effective, there is still value in using them alongside interventions that target the environment. In a study that investigated the relationship between individual level psychological interventions and food environments, less healthy food

environments in shops (indicated by availability, pricing and promotion of unhealthy food) were associated with lower psychological resources to select healthy food [493]. This relates to the finding in this thesis, that environments moderate associations between psychological symptoms and body composition. Addressing cues and stimuli in the environment, that promote increased food intake, combined with behavioural change strategies that increase psychological resources to make healthier decisions, could be an effective approach to address obesity [493] and unhealthy body composition.

### 9.3.4 Improving Fat-Free Mass

Many of the policies and interventions discussed relate to tackling adiposity-based components of body composition. This thesis found more evidence of inequalities in FM than FFM, potentially highlighting the need for a greater focus on reducing adiposity than increasing muscle mass. Research conducted in ALSPAC found higher lean mass to be associated with lower atherogenic traits, but not as strongly as the positive association between FM and atherogenic traits, indicating that fat may be more important for cardiovascular health [86]. However, the research in ALSPAC did find that gains in lean mass in adolescents (13-18 years) increased protection from CVD in later life [86], indicating this may be an important time in the life course to intervene to increase muscle. Additionally, peak muscle mass is reached at an earlier point in the life course, in mid-adulthood, than peak fat, which increases into older age and declines thereafter [97, 365], and inequalities in FM:FFM ratio were demonstrated in this thesis.

If children from more disadvantaged groups have lower levels of muscle mass, as well as higher levels of fat, as demonstrated in the systematic review and analysis in the MCS, and these differences track across the life course [239], there may be important implications for inequalities in outcomes in later life which also require good muscle function. For example, cardio-metabolic disease and physical capability in older age are related to FFM [89, 380]. Previous research has demonstrated that there are certain points in the life course, including adolescence and early adulthood, where muscle strengthening activities may be particularly important for later health [86, 87]. Therefore, promotion of physical activity in early life to increase muscle mass as well as to decrease adiposity should be an important consideration in interventions to improve body composition. In particular, a focus is needed to ensure access to sports facilities in disadvantaged settings, which as stated previously has been found to be a barrier for children in poorer families [486].

## 9.4 Methodological Considerations

This section considers the methodological approaches adopted in this thesis and their strengths and weaknesses. As strengths and weaknesses were discussed in the individual chapters, only those with relevance across the thesis are discussed here.

### 9.4.1 The Millennium Cohort Study

One of the key strengths of methodology adopted in this thesis is the use of data from a large, prospectively collected, nationally representative cohort of children in the UK. The MCS has a rich source of longitudinal data, with information on social and economic circumstances and health outcomes from a young age. Repeated measures of body composition allowed for the longitudinal

nature of the MCS to be effectively utilised to address the research questions in this thesis. Further to this, mediation models could be tested because of the repeated collection and temporality of key variables, such as emotional and behavioural difficulties (measured using the SDQ), at different ages. By using the MCS, it was also possible to investigate the influence that different aspects of the environment (e.g. area deprivation and green spaces) had on mediating pathways. Detailed information was available on the areas in which cohort members lived, and was linked to characteristics of the environment, which is not available in other cohorts at young ages.

#### 9.4.2 Measuring Body Composition

A strength of this thesis is the ways in which adiposity was measured. The MCS measured FM% through BIA, which is a non-evasive and low-cost method that can accurately measure FM%. The BIA machines were calibrated before use, used prediction equations derived from large multi-ethnic populations, and have been validated against DXA [198]. However, even though all participants were asked to remove bulky items and wear light indoor clothing, no further controls were adopted, such as voiding or not drinking beforehand, or taking measures from participants at the same time of day. As BIA uses water content to estimate body composition, the MCS measurement protocol may therefore have adversely impacted the accuracy of adiposity measures.

From FM% measured through BIA, it was possible to derive FM and FFM, and consequently index measures by height. FMI is a better measure of adiposity than BMI or percentage measures of fat, as FMI is a direct measure of fat that accounts for height. In this thesis, population, age and sex-specific Benn parameters were calculated when indexing FM and FFM. By doing this, the correlation of mass with height was removed [202].

It has been usual for researchers to adopt percentage measures of FM and FFM, yet FFM% will always be 100 minus FM%. Additionally, FM will be in both the numerator and denominator when calculating FM% (and similarly for FFM%), as both FM and FFM make up total body weight. Therefore FM and FFM cannot be interpreted independently of each other [72]. In the systematic review, studies that used raw or percentage measures of FFM more frequently demonstrated evidence of positive associations with socioeconomic circumstances than those seen for studies that used indexed measures, suggesting inequalities in height may explain some of the associations, and both raw and percentage measures do not account for height.

DXA is considered the gold standard method in body composition research due to the more detailed analysis, including location of both fat and lean tissue. However, DXA is relatively expensive and cannot be assessed at home, making it less practical for large cohort studies such as the MCS. Research has demonstrated that BIA and DXA methods take similar measures at the population level (BIA values within +/-5% of measures from other methods [197, 198]), especially for FM, but there is low concordance between methods within individuals [66]. Therefore, it is possible the BIA measures in the MCS may be inaccurate for individual cohort members within the study, but this is unlikely to have substantially impacted population level estimates of inequalities.



Because body composition was collected through BIA, rather than DXA, it was not possible to distinguish the contribution of bone mass and LM [494]. Prediction equations do exist to calculate LM from BIA, but these are very population specific and therefore are not generalisable to dissimilar populations to the ones they were developed in [494]. Development of prediction equations for a specific population also needs a reference method (such as DXA) and the raw impedance measures, which were not available in the MCS. Therefore, more detailed analysis of FFM was not possible in the MCS. It has previously been demonstrated that associations with FFM could partially represent associations with bone density [256]. As the interest in this thesis was to estimate inequalities in LM, if inequalities in bone differ substantially to those for LM, inequalities in FFM could either over or underestimate associations for LM.

### 9.4.3 Statistical Challenges

Ethnicity was treated as a confounder in analysis, because it is known to be associated with both disadvantage and body composition. Based on previous evidence using anthropometric measures, it is possible that there are differences in inequalities in body composition according to ethnicity. However, it was not possible to assess ethnic difference in the systematic review due to the small number of papers stratifying results by ethnicity. Similarly, for growth curve analysis there were small numbers in some of the ethnic groups, making analysis by ethnicity difficult without collapsing into a single crude “non-white” or “Black, Asian, and minority ethnic” group. This would not have been an appropriate approach, as information on the diversity of ethnic categories would have been lost. Additionally, strong (and unrealistic) assumptions would have to be made about the similarity of experience among those ethnicities defined as “non-white”, yet the relation between ethnicity and social inequalities is known to vary considerably.

Only the second-order internalising and externalising factors were considered in mediation models, as this thesis had hypothesis relating to the role of these higher order constructs. However, there would be value in exploring if and how each of the first-order factors (e.g. the conduct problem and hyperactivity factors that make up externalising symptoms, and the peer problem and emotional scale factors that make up internalising symptoms) individually mediate associations, and how these are moderated by the environment. In Chapter 7, it was demonstrated that the first-order factors had somewhat different predictive properties for depression, ADHD and autism/Asperger’s, and it is plausible that this is also the case for the ways in which they mediate associations with body composition. It is possible that for externalising symptoms, the mediating role of the first-order hyperactivity and conduct problem factors differ based on levels of green spaces and deprivation. Differences in mediating role by the first-order factors may explain the counterintuitive result for externalising symptoms in the most deprived and least green settings.

Although there was temporal ordering in the measurement of the independent, mediating and dependent variables in the analysis, more complex models could also be adopted to strengthen claims to causality. Adoption of a cross-lagged model that includes SEP, internalising and externalising symptoms, and body composition at multiple time points would allow investigation of possible reverse causality. Associations between internalising and externalising symptoms with

obesity measured by BMI have been demonstrated in both directions previously [450]. It would strengthen understanding of the associations between SEP, symptoms and body composition, to examine whether reverse causality exists between internalising and externalising symptoms, and how this affects the mediating paths.

Another expansion of the analysis conducted in this thesis would be to test whether the path from SEP to internalising and externalising symptoms is moderated by the environment. This thesis only investigated moderation of the mediating path from symptoms to body composition, as it was hypothesised that the environment provided cues and stimuli that influenced the coping strategies adopted in response to symptoms. However, it is possible that the environment also influences the association between SEP and symptoms, with SEP being more strongly associated with symptoms in more obesogenic or deprived areas. There was some evidence of this in Chapter 8 for domestic gardens and fast-food density. Therefore, extensions of this analysis could explore whether the environment moderates alternative mediating paths that represent different underlying mechanisms to those explored in this thesis.

Attrition in a longitudinal study such as the MCS is inevitable, and approaches to deal with missing data were used. For SEMs, this was addressed by including the non-response and sampling weight at age 17, which is the recommended approach. For growth curve models this was not done because body composition was used at multiple sweeps, and a non-response weight from one sweep would not accurately account for attrition at another. However, maximum likelihood estimation was used, which uses all information available to provide robust estimates when there are missing data in the outcome. Additionally, stratification characteristics and sample weights were included in imputation modes, and when this is done, analysis with inclusion of non-response weights has been shown to have little impact on estimates [188]. However, Chapter 4 demonstrated those in disadvantaged positions were more likely to have missing data for body composition and BMI. Therefore, it is still possible that in growth curve models, estimates are biased because of attrition in the outcome, and that they may underestimate the association between SEP and body composition if those who dropped out also had less healthy body composition.

## 9.5 Direction for Future Research

This thesis addressed a number of questions that help to understand life course inequalities in body composition. However, the research also highlighted a number of further questions that could be addressed, following on from the work presented.

### 9.5.1 Muscle Mass

A key theme that came out of this thesis is the way in which FFM is measured, and in particular the need to isolate muscle mass. Being able to track muscle across life is needed to understand how life course trajectories in muscle relate to social inequalities and disease in later life. What this thesis demonstrates is the need for greater understanding on the differences in inequalities in muscle between generations and how this develops across the life course, as has been done previously for

BMI. By having this understanding, it would be possible to make predictions about what might happen in older age as muscle starts to weaken, for young people growing up today.

In particular, future research should investigate inequalities for LM, as opposed to the more general FFM that also includes bone. Using precise measures of LM may be particularly important for research on ageing, as muscle becomes increasingly important for functional outcomes. Conditions such as sarcopenia are characterised by loss of skeletal muscle and muscle strength, whilst lower levels of LM increases risk of osteoporosis as LM is positively associated with bone mineral density [495].

Investigating generational and life course differences in LM is possible in other longitudinal or cohort studies, such as ALSPAC, which has five repeated measures of body composition using DXA. However, ALSPAC is a regional cohort study and therefore would not be generalisable to the rest of the UK. Similarly, investigation of trajectories in body composition at older ages would be beneficial, but at current none of the other British Birth Cohort studies have repeated measures over substantial periods of the life course, and only the 1946 NSHD uses DXA. Other studies such as UK Biobank do have more extensive collection of body composition through DXA, but at present the number of repeated collections is small and only over midlife [496]. Additionally, Biobank has low generalisability as the sample is a more advantaged population comparative to the rest of the UK.

### 9.5.2 Ethnic Differences in Inequalities

A further avenue of research that comes out of this thesis is the need to explore other dimensions of social inequalities and the factors that influence and moderate inequalities. The systematic review identified a small number of studies that also investigated ethnicity, but the numbers were too small to explore this as a source of heterogeneity. Ethnic differences in inequalities are likely to be complex, influenced by cultural factors, migration status and structural racism. Previous research has demonstrated that in HICs, people from minority ethnic groups tend to have higher prevalence of obesity compared to the majority [497], and are more likely to be living in disadvantaged circumstances [214]. In the US, whilst lower income was associated with lower levels of obesity among African American and Caribbean Black men, the opposite association is observed for women [498]. In the UK, there is evidence that increased acculturation results in convergence in obesity among minority ethnic groups, except for Black Caribbean groups [499].

Further research is needed to understand the complexity of ethnic differences in body composition inequalities in both the UK and globally. In particular, there is a need to understand how ethnic differences contribute to the socioeconomic inequalities observed, and vice versa, if social inequalities can help explain observed ethnic differences.

Given the likely ethnic differences in social inequalities, it is also important to understand if the mediating paths are the same across ethnic groups, or if the pathways that result in the development of social inequalities in body composition differ by ethnicity. There is some evidence that levels of internalising and externalising symptoms as well as more general mental health problems differ according to ethnicity in the US [500] and UK [501], and by migrant status - which often reflects non-

native ethnicity - in Europe [502]. There may therefore also be differences in the mediating path between SEP and body composition, which is unlikely to be homogenous given known differences in inequalities in obesity by ethnicity.

Previous research has found use of green spaces to differ by deprivation and ethnicity [44]. Research conducted in the UK has demonstrated access to and quality of urban green spaces is an important predictor of general health in minority ethnic groups but not in the white population where social aspects of the environment (e.g., levels of belonging and neighbourhood trust) are a greater determinant of general health [503]. Research using the Born in Bradford study in the UK found that ethnicity moderates the association between green space and wellbeing in children aged 4 years [504]. It is likely these observed ethnic differences in use of green spaces and consequences for health also have implications for the moderating role that green places have for body composition. Understanding these differences is important, so that interventions or policies that are aimed at reducing inequalities, are sensitive to nuances and the different impacts they may have on different segments of the population. This ensures that unintended adverse impacts of interventions for specific groups can be effectively mitigated, while targeting appropriate interventions for groups that will benefit the most.

### 9.5.3 Investigating Coping Mechanisms

This thesis demonstrated that internalising and externalising symptoms relate to body composition, and that the association is moderated by environmental conditions. It is hypothesised that diet and physical activity are on the pathways from symptoms to body composition as they relate to the potential coping mechanisms adopted. The MCS has collected physical activity data, including information on leisure time, sedentary time and active travel from ages 3 to 17. At ages 7 and 14 activity data were collected through the use of accelerometers, with additional time use diaries at age 14.

Using this detailed information, it would be possible to test the hypothesis that higher levels of physical activity among those exhibiting internalising symptoms are observed in areas with greater levels of green spaces, thereby moderating associations between internalising symptoms and body composition through effects on physical activity. Accelerometry and sedentary time data could also be used to investigate if reduced sedentary time explains the finding that in the most deprived and least green areas, externalising symptoms are related to lower adiposity, possibly because of reduced time in a sedentary state.

The MCS has also collected some dietary data. It would be possible to test if those from disadvantaged backgrounds exhibiting externalising symptoms in less deprived settings consume more unhealthy food, reflecting higher impulsivity. It would also be possible to explore if both diet and physical activity explain the mediating paths for internalising symptoms, with higher intake of unhealthy foods and lower levels of physical activity expected in more obesogenic areas, as a coping mechanism for emotional symptoms.

Both physical activity and dietary data are notoriously difficult to collect, with self-report measures often over estimating activity and underestimating calorie intake. Unlike physical activity, objective measures for diet are typically more expensive, and not feasible or routinely used in large cohort studies like the MCS [505]. Furthermore, the MCS has not used food frequency questionnaires (FFQs) or other established dietary assessment tools. The dietary data available in the MCS is therefore limited to a handful of questions that explore consumption of a few specific foods (e.g., milk, bread, sweetened drinks, fruit and vegetables), regularity of meals, and frequency of eating fast-food products. The ability to investigate coping mechanisms in the MCS would be limited to these elements of diet. As such, it would be beneficial to explore the role of diet in more detail in other cohorts, such as ALSPAC, which have routinely used FFQs.

There would be additional value in exploring these coping mechanisms in different cohorts more generally, to identify if they have changed between generations. As accessibility to energy-dense foods has increased over time, it is possible the coping mechanisms adopted in response to psychological symptoms have also changed. For example, the 1946 cohort grew up with post-war food rationing, and this resulted in fewer SEP differences in diet. Therefore, the ability to overconsume as a regulatory mechanism would be limited across all social groups. Additionally, there would be value in exploring differences in coping strategies adopted at different ages, as strategies observed in childhood and adolescence in the MCS may not be applicable to adults and older individuals, who may regulate psychological symptoms in alternative ways.

#### 9.5.4 Investigating the Role of Personality Traits

Other elements of mental health or personality traits, such as, conscientiousness may be important because of the possibility that they buffer the influence of environmental conditions.

Conscientiousness has been found to relate to lower levels of obesity over the life course [506, 507] as it is related to higher levels of self-discipline and is a measure of the ability to regulate short-term urges and desires in favour of longer-term outcomes [507]. Conscientiousness is one of five personality traits, along with agreeableness, openness, neuroticism and extroversion. Each of these traits has previously been found to have different predictive properties in relation to obesity [506-508], with traits such as neuroticism and extroversion and traits characterised by higher levels of impulsivity being related to increased BMI [506, 507].

It has been demonstrated that personality traits explain some of the social gradient for all-cause mortality [509], and health behaviours such as smoking [510]. Furthermore, conscientiousness and neuroticism have been found to interact with SEP to predict inflammation, with smaller SEP differences observed with higher levels of conscientiousness, and larger SEP differences with higher neuroticism [511]. The differences in the interaction were partly explained through differences in adiposity. It is possible that these personality traits partly explain associations between SEP and body composition and are additionally moderated by environmental conditions.

This may be particularly pertinent for conscientiousness where obesogenic cues in the environment may impact the ability to exert self-discipline in relation to food choices, and for impulse related traits

where the environment may encourage more impulsive decisions surrounding food choices. In the MCS, conscientiousness and other personality traits were only collected in cohort members at age 17. Therefore, it would not have been possible to explore it as a mediator in associations whilst also ensuring temporality between the predictor, mediator and outcome. With future data collections, this would be possible and a valuable contribution to understanding how the environment moderates individual characteristics in the development of obesity and body composition.

### 9.5.5 Cross-Cohort Comparisons and Longitudinal Follow-Up

Another approach to investigating the different impacts of the mediating path for emotional and behavioural problems in response to differing environments is to look across generations. By looking across the British Birth Cohort studies, it would be possible to assess the changing role of the environment. As previously mentioned, the 1946 cohort was characterised by post-war rationing in childhood compared to the MCS where cohort members have experienced an obesogenic environment from birth. This would create a “natural” experiment where the ordering of the birth cohorts would allow observation of how the different environments over time influences the existence and strength of mediating paths. Due to the lack of body composition measures in early data collection in the other cohorts, BMI would have to be used instead of FMI.

It is expected that there would be greater evidence of mediation, resulting from associations between internalising and externalising symptoms and BMI, in the more recent born cohorts. It is likely that in the earlier born cohorts, SEP would still be related to emotional and behavioural problems in childhood and adolescence. However, only with the changing environment that has become more obesogenic, is it expected that these symptoms would result in increases in BMI. In an obesogenic environment, there are greater opportunities to adopt coping strategies that increase risk of obesity, such as bingeing on unhealthy foods and engaging in sedentary behaviours. In the more recent born cohorts that have experienced obesogenic environments from a young age, it would be expected that higher symptoms are associated with increases in BMI in adolescence. Whilst this would not be the case for the earlier born cohorts.

A recurring theme in this thesis is the need for continued follow up of childhood cohorts so comparisons can be made across generations. This will help to confirm whether there are secular rather than age-related changes in inequalities, or the existence of both simultaneously. Continued collection of body composition in the MCS will allow more detailed modelling of inequalities in trajectories in the future, including non-linear growth curves. This will allow investigation of the impact of SEP in early life on body composition over the life course, which in turn can be used to understand the consequences for health in adulthood and later life.

### 9.5.6 Country Comparisons

In analysis using the IMD, deprivation scales across the four countries of the UK were combined. However, as each of the deprivation indices uses a different weighting, they are not fully comparable. This has implications for the analysis in this thesis to compare social inequalities across countries of the UK. The impact on findings of alternative approaches to establishing comparative indices, for

example using an adjusted IMD scale that utilises the income and education components of the IMD [207, 387], needs to be established. Once the most comparable approach has been established, investigating differences in inequalities between countries could be useful in understanding the most effective policies, given differences across the UK. Policies that support active lifestyle and healthy eating differ across regions, and it is necessary to identify the effective policies to promote health lifestyles and improve body composition in children and adolescents. Moreover, there is a need to understand the likely drivers of differences in the environment, such as green space and fast-food density, as well as cultural and policy differences between the four countries of the UK [512].

Differences in levels of obesity have previously been observed between each of the countries of the UK, with children from Northern Ireland and Wales more likely to be obese than children in England [512]. These differences were observed after considering individual-level social circumstances [512], but it is likely disparities in area-level deprivation between the countries may further explain some of the observed variation in obesity, and also in body composition.

## 9.6 Concluding Remarks

The determinants of adiposity are both distal and proximal, they are social, environmental, behavioural and biological and they are shaped by policy. This thesis demonstrates that the inequalities observed in BMI extend to FM. Although inequalities in BMI will be largely driven by disparities in fat, work here on FFM suggest that there are smaller, but still potentially important inequalities in FFM, that results in inequalities in their ratio. This thesis suggests that the inequalities in body composition seen, may be affecting younger generations earlier than previous generations, and that there are notable inequalities in FM in children and adolescents growing up today. Continued follow-up of current generations of young people and comparison with earlier born cohorts will allow us to better understand the potential future impact of these inequalities in body composition on health and ageing.

Younger generations are spending more of their life overweight or obese and with higher cumulative levels of FM than generations before them, as a result of increasingly obesogenic environments. This results in an extended state of inflammation and will undoubtedly have large consequences for later life health, and as such consequences for future health inequalities. Steps taken earlier to address the inequalities highlighted in this thesis will avert the need for public health interventions at later ages, where such measures may be less effective at reducing inequalities than action earlier in the life course.

This thesis highlighted the important role of area-level deprivation in these inequalities, both as a determinant of inequalities and as a moderating factor. Addressing the environment may be an effective way to break the association between emotional and behavioural problems and adiposity in childhood. Improving the environments in which people live, by improving resources and infrastructure, investing in local communities and increasing high quality green spaces, especially in deprived areas, should therefore be considered as an effective set of solutions to tackle inequalities in body composition.

# Appendices

This section presents the appendices for the thesis, starting with the appendix for Chapter 4.

## A4. Chapter 4 Appendices

### A4.1 Characteristics of Missingness for NS-SEC at Sweep 1

	<b>Complete Data (N=18,235)</b>		<b>Missing Data</b>	
	<b>N</b>	<b>%</b>	<b>N</b>	<b>%</b>
<b>Sex</b>				
<i>Male</i>	9,377	94.8	517	5.2
<i>Female</i>	8,858	94.8	491	5.3
<b>Ethnicity</b>				
<i>White</i>	15,015	96	623	4.0
<i>Indian</i>	542	92.7	43	7.4
<i>Pakistani and Bangladeshi</i>	457	92.3	38	7.7
<i>Black and Black British</i>	1,258	94.4	75	5.6
<i>Other Ethnic Group</i>	649	90.1	71	9.9
<i>Mixed</i>	266	89	33	11.0
<b>IMD</b>				
<i>Most deprived 20%</i>	5,929	98.5	88	1.5
<i>20-40%</i>	4,159	98.6	59	1.4
<i>40-60%</i>	3,026	98.1	60	1.9
<i>60-80%</i>	2,455	97.9	53	2.1
<i>Most advantaged 20%</i>	2,665	97.9	57	2.1
<b>Stratification Characteristics</b>				
<i>England - Advantaged</i>	4,530	93.8	298	6.2
<i>England - Disadvantaged</i>	4,442	92.5	363	7.6
<i>England - Ethnic</i>	2,368	91.4	223	8.6
<i>Wales - Advantaged</i>	812	97.6	20	2.4
<i>Wales - Disadvantaged</i>	1,900	98.6	28	1.5
<i>Scotland - Advantaged</i>	1,126	98.3	19	1.7
<i>Scotland - Disadvantaged</i>	1,170	98.2	21	1.8
<i>Northern Ireland - Advantaged</i>	712	98.5	11	1.5
<i>Northern Ireland - Disadvantaged</i>	1,175	97.9	25	2.1

**Table A4.1 Footnote:** Missingness presented for the NS-SEC by key variables in the full sample (N=19,243).  
Abbreviations: NS-SEC - National Statistics Socioeconomic classification; IMD - Index of Multiple Deprivation.



## A4.2 Characteristics of Missingness for NVQ at Sweep 1

	<b>Complete Data (N=18,266)</b>		<b>Missing Data</b>	
	<b>N</b>	<b>%</b>	<b>N</b>	<b>%</b>
<b>Sex</b>				
Male	9,401	95	493	5
Female	8,865	94.8	484	5.2
<b>Ethnicity</b>				
White	15,048	96.2	590	3.8
Indian	543	92.8	42	7.2
Pakistani and Bangladeshi	459	92.7	36	7.3
Black and Black British	1,254	94.1	79	5.9
Other Ethnic Group	651	90.4	69	9.6
Mixed	265	88.6	34	11.4
<b>IMD</b>				
Most deprived 20%	5,927	98.5	90	1.5
20-40%	4,164	98.7	54	1.3
40-60%	3,034	98.3	52	1.7
60-80%	2,467	98.4	41	1.6
Most advantaged 20%	2,673	98.2	49	1.8
<b>Stratification Characteristics</b>				
England - Advantaged	4,545	94.1	283	5.9
England - Disadvantaged	4,448	92.6	357	7.4
England - Ethnic	2,365	91.3	226	8.7
Wales - Advantaged	820	98.6	12	1.4
Wales - Disadvantaged	1,901	98.6	27	1.4
Scotland - Advantaged	1,126	98.3	19	1.7
Scotland - Disadvantaged	1,177	98.8	14	1.2
Northern Ireland - Advantaged	709	98.1	14	1.9
Northern Ireland - Disadvantaged	1,175	97.9	25	2.1

**Table A4.2 Footnote:** Missingness presented for the NVQ by key variables in the full sample (N=19,243).

Abbreviations: NVQ - National Vocational Qualifications; IMD - Index of Multiple Deprivation.

### A4.3 Characteristics of missingness for Green Spaces at Sweep 5

	<b>Complete Data</b> <b>(N=13,280)</b>		<b>Missing Data</b>	
	<b>N</b>	<b>%</b>	<b>N</b>	<b>%</b>
<b>Sex</b>				
Male	6,710	67.8	3,184	32.2
Female	6,570	70.3	2,779	29.7
<b>Ethnicity</b>				
White	10,923	69.9	4,715	30.2
Indian	383	65.5	202	34.5
Pakistani and Bangladeshi	339	68.5	156	31.5
Black and Black British	935	70.1	398	29.9
Other Ethnic Group	426	59.2	294	40.8
Mixed	183	61.2	116	38.8
<b>IMD</b>				
IMD Group 1 (Most deprived 20%)	4,053	64.3	2,255	35.8
IMD Group 2	2,932	67.1	1,437	32.9
IMD Group 3	2,318	72.4	884	27.6
IMD Group 4	1,912	74.1	670	26
IMD Group 5 (Most advantaged 20%)	2,065	74.2	717	25.8
<b>Income</b>				
Lowest Income Fifth	2,965	61.5	1,857	38.5
Second Fifth	2,807	65.5	1,477	34.5
Third Fifth	2,479	70.1	1,060	30.9
Fourth Fifth	2,475	76.4	766	23.6
Highest income fifth	2,325	77.9	660	22.1
<b>NS-SEC</b>				
Unemployed	834	58.2	598	41.8
Semi-routine and routine	3,088	61.7	1,916	38.3
Lower supervisory and technical	1,080	68	508	32
Small employers and self-employed	763	67.4	369	32.6
Intermediate	1,666	68.9	751	31.1
Managerial and Professional	5,428	77.2	1,606	22.8
<b>NVQ</b>				
No Qualifications	1,241	57.3	924	42.7
Other/overseas Qualifications	284	62.4	171	37.6
NVQ Level 1	767	62.4	462	37.6
NVQ Level 2	3,228	66.2	1,648	33.8
NVQ Level 3	2,124	69.3	940	30.7
NVQ Level 4	4,537	75.9	1,439	24.1
NVQ Level 5	914	77.5	266	22.5

**Table A4.3 Footnote:** Missingness presented for the Green Spaces at sweep 5 by key variables in the full sample (N=19,243). Abbreviations: NS-SEC - National Statistics Socioeconomic classification; NVQ - National Vocational Qualifications; IMD - Index of Multiple Deprivation.

#### A4.4 Characteristics of missingness for Fast-Food Density at Sweep 5

	<b>Complete Data (N=11,478)</b>		<b>Missing Data</b>	
	<b>N</b>	<b>%</b>	<b>N</b>	<b>%</b>
<b>Sex</b>				
Male	5,809	58.7	4,085	41.3
Female	5,669	60.6	3,680	39.4
<b>Ethnicity</b>				
White	9,151	58.5	6,487	41.5
Indian	375	64.1	210	35.9
Pakistani and Bangladeshi	337	68.1	158	31.9
Black and Black British	933	70	400	30
Other Ethnic Group	422	58.6	298	41.4
Mixed	177	59.2	122	40.8
<b>IMD</b>				
IMD Group 1 (Most deprived 20%)	3,618	57.4	2,690	42.6
IMD Group 2	2,544	58.2	1,825	41.8
IMD Group 3	2,004	62.6	1,198	37.4
IMD Group 4	1,600	62.0	982	38.0
IMD Group 5 (Most advantaged 20%)	1,712	61.5	1,070	38.5
<b>Income</b>				
Lowest Income Fifth	2,599	53.9	2,223	46.1
Second Fifth	2,458	57.4	1,826	42.6
Third Fifth	2,129	60.2	1,410	39.8
Fourth Fifth	2,103	64.9	1,138	35.1
Highest income fifth	1,997	66.9	988	33.1
<b>NS-SEC</b>				
Unemployed	752	52.5	680	47.5
Semi-routine and routine	2,680	53.6	2,324	46.4
Lower supervisory and technical	963	60.6	625	39.4
Small employers and self-employed	652	57.6	480	42.4
Intermediate	1,417	58.6	1,000	41.4
Managerial and Professional	4,631	65.8	2,403	34.2
<b>NVQ</b>				
No Qualifications	1,095	50.6	1,070	49.4
Other/overseas Qualifications	265	58.2	190	41.8
NVQ Level 1	681	55.4	548	44.6
NVQ Level 2	2,789	57.2	2,087	42.8
NVQ Level 3	1,858	60.6	1,206	39.4
NVQ Level 4	3,866	64.7	2,110	35.3
NVQ Level 5	769	65.2	411	34.8

**Table A4.4 Footnote:** Missingness presented for the Fast-Food Density at sweep 5 by key variables in the full sample (N=19,243). Patterns of missingness are described using sex, ethnicity and SEP characteristics from sweep 1. Abbreviations: NS-SEC - National Statistics Socioeconomic classification; NVQ - National Vocational Qualifications; IMD - Index of Multiple Deprivation.

#### A4.5 Characteristics of missingness for Domestic Gardens at Sweep 5

	<b>Complete Data (N=8,671)</b>		<b>Missing Data</b>	
	<b>N</b>	<b>%</b>	<b>N</b>	<b>%</b>
<b>Sex</b>				
Male	4,385	44.3	5,509	55.7
Female	4,286	45.8	5,063	54.2
<b>Ethnicity</b>				
White	6,430	41.1	9,208	58.9
Indian	344	58.8	241	41.2
Pakistani and Bangladeshi	331	66.9	164	33.1
Black and Black British	901	67.6	432	32.4
Other Ethnic Group	411	57.1	309	42.9
Mixed	174	58.2	125	41.8
<b>IMD</b>				
IMD Group 1 (Most deprived 20%)	2,882	45.7	3,426	54.3
IMD Group 2	1,837	42.1	2,532	58.0
IMD Group 3	1,561	48.8	1,641	51.3
IMD Group 4	1,252	48.5	1,330	51.5
IMD Group 5 (Most advantaged 20%)	1,139	40.9	1,643	59.1
<b>Income</b>				
Lowest Income Fifth	1,943	40.3	2,879	59.7
Second Fifth	1,862	43.5	2,422	56.5
Third Fifth	1,583	44.7	1,956	55.3
Fourth Fifth	1,551	47.9	1,690	52.1
Highest income fifth	1,578	52.9	1,407	47.1
<b>NS-SEC</b>				
Unemployed	613	42.8	819	57.2
Semi-routine and routine	1,953	39.0	3,051	61.0
Lower supervisory and technical	693	43.6	895	56.4
Small employers and self-employed	540	47.7	592	52.3
Intermediate	1,032	42.7	1,385	57.3
Managerial and Professional	3,499	49.7	3,535	50.3
<b>NVQ</b>				
No Qualifications	876	40.5	1,289	59.5
Other/overseas Qualifications	242	53.2	213	46.8
NVQ Level 1	531	43.2	698	56.8
NVQ Level 2	2,116	43.4	2,760	56.6
NVQ Level 3	1,279	41.7	1,785	58.3
NVQ Level 4	2,924	48.9	3,052	51.1
NVQ Level 5	584	49.5	596	50.5

**Table A4.5 Footnote:** Missingness presented for the Domestic Gardens at sweep 5 by key variables in the full sample (N=19,243). Patterns of missingness are described using sex, ethnicity and SEP characteristics from sweep 1. Abbreviations: NS-SEC - National Statistics Socioeconomic classification; NVQ - National Vocational Qualifications; IMD - Index of Multiple Deprivation.

#### A4.6 Characteristics of missingness for Fast-Food Density at Sweep 5 restricted to Great Britain

	<b>Complete Data (N=11,478)</b>		<b>Missing Data</b>	
	<b>N</b>	<b>%</b>	<b>N</b>	<b>%</b>
<b>Sex</b>				
Male	5,809	62.9	3,434	37.2
Female	5,669	65.2	3,026	34.8
<b>Ethnicity</b>				
White	9,151	63.8	5,198	36.2
Indian	375	64.8	204	35.2
Pakistani and Bangladeshi	337	68.2	157	31.8
Black and Black British	933	70.1	399	30.0
Other Ethnic Group	422	58.6	298	41.4
Mixed	177	59.6	120	40.4
<b>IMD</b>				
IMD Group 1 (Most deprived 20%)	3,618	61.2	2,297	38.8
IMD Group 2	2,544	62.9	1,503	37.1
IMD Group 3	2,004	67.1	982	32.9
IMD Group 4	1,600	65.9	829	34.1
IMD Group 5 (Most advantaged 20%)	1,712	66.9	849	33.2
<b>Income</b>				
Lowest Income Fifth	2,599	57.7	1,904	42.3
Second Fifth	2,458	61.3	1,550	38.7
Third Fifth	2,129	64.8	1,156	35.2
Fourth Fifth	2,103	70.3	887	29.7
Highest income fifth	1,997	71.1	813	28.9
<b>NS-SEC</b>				
Unemployed	752	55.5	604	44.5
Semi-routine and routine	2,680	57.7	1,968	42.3
Lower supervisory and technical	963	64.5	531	35.5
Small employers and self-employed	652	61.3	412	38.7
Intermediate	1,417	63.5	813	36.5
Managerial and Professional	4,631	70.9	1,904	29.1
<b>NVQ</b>				
No Qualifications	1,095	54.1	931	46.0
Other/overseas Qualifications	265	60.4	174	39.6
NVQ Level 1	681	58.8	478	41.2
NVQ Level 2	2,789	61.5	1,746	38.5
NVQ Level 3	1,858	64.9	1,006	35.1
NVQ Level 4	3,866	69.6	1,687	30.4
NVQ Level 5	769	70.8	318	29.3

**Table A4.6 Footnote:** Missingness presented for the Fast-Food Density at sweep 5 by key variables in the sample limited to those residing in Great Britain (n=17,938). Patterns of missingness are described using sex, ethnicity and SEP characteristics from sweep 1. NS-SEC: National Statistics Socioeconomic classification. NVQ: National Vocational Qualifications. IMD: Index of Multiple Deprivation.

## A5. Chapter 5 Appendices

### A5.1 Publications from This Work

#### *A5.1.1 Systematic Review Protocol*

Bridger Staatz C, Kelly Y, Lacey R, Hardy R (2019) “Socioeconomic position and body composition across the life course: a systematic review protocol” *Systematic Reviews*. 8:26.

<https://doi.org/10.1186/s13643-019-1197-z>

#### *A5.1.2 Systematic Review in Children*

Bridger Staatz, C., Kelly, Y., Lacey, R.E. et al. Socioeconomic position and body composition in childhood in high- and middle-income countries: a systematic review and narrative synthesis. *Int J Obes* (2021). 45, 2316–2334. <https://doi.org/10.1038/s41366-021-00899-y>

#### *A5.1.3 Systematic Review in Adults*

Bridger Staatz, C., Kelly, Y., Lacey, R.E. et al. Life course socioeconomic position and body composition in adulthood: a systematic review and narrative synthesis. *Int J Obes* (2021). 45, 2300–2315. <https://doi.org/10.1038/s41366-021-00898-z>

## A5.2 PRISMA-P 2015 Checklist

**Note:** This checklist was submitted with the published protocol, and therefore the line numbers match up to the protocol and not the line numbers in this thesis.

**This checklist has been adapted for use with systematic review protocol submissions to BioMed Central journals from Table 3 in Moher D et al:** Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015 statement. *Systematic Reviews* 2015 4:1

An Editorial from the Editors-in-Chief of *Systematic Reviews* details why this checklist was adapted – **Moher D, Stewart L & Shekelle P:** Implementing PRISMA-P: recommendations for prospective authors. *Systematic Reviews* 2016 5:15

Section/topic	#	Checklist item	Information reported		Line number(s)
			Yes	No	
<b>ADMINISTRATIVE INFORMATION</b>					
<b>Title</b>					
Identification	1a	Identify the report as a protocol of a systematic review	<input checked="" type="checkbox"/>	<input type="checkbox"/>	1-3
Update	1b	If the protocol is for an update of a previous systematic review, identify as such	<input checked="" type="checkbox"/>	<input type="checkbox"/>	n/a
<b>Registration</b>	2	If registered, provide the name of the registry (e.g., PROSPERO) and registration number in the Abstract	<input checked="" type="checkbox"/>	<input type="checkbox"/>	43
<b>Authors</b>					
Contact	3a	Provide name, institutional affiliation, and e-mail address of all protocol authors; provide physical mailing address of corresponding author	<input checked="" type="checkbox"/>	<input type="checkbox"/>	7-19
Contributions	3b	Describe contributions of protocol authors and identify the guarantor of the review	<input checked="" type="checkbox"/>	<input type="checkbox"/>	194-197
<b>Amendments</b>	4	If the protocol represents an amendment of a previously completed or published protocol, identify as such and list changes; otherwise, state plan for documenting important protocol amendments	<input checked="" type="checkbox"/>	<input type="checkbox"/>	87-88
<b>Support</b>					
Sources	5a	Indicate sources of financial or other support for the review	<input checked="" type="checkbox"/>	<input type="checkbox"/>	188
Sponsor	5b	Provide name for the review funder and/or sponsor	<input checked="" type="checkbox"/>	<input type="checkbox"/>	190-192

Section/topic	#	Checklist item	Information reported		Line number(s)
			Yes	No	
Role of sponsor/funder	5c	Describe roles of funder(s), sponsor(s), and/or institution(s), if any, in developing the protocol	<input checked="" type="checkbox"/>	<input type="checkbox"/>	194-197
<b>INTRODUCTION</b>					
<b>Rationale</b>	6	Describe the rationale for the review in the context of what is already known	<input checked="" type="checkbox"/>	<input type="checkbox"/>	47-83
<b>Objectives</b>	7	Provide an explicit statement of the question(s) the review will address with reference to participants, interventions, comparators, and outcomes (PICO)	<input checked="" type="checkbox"/>	<input type="checkbox"/>	77-83 Intervention N/A – see cover letter.
<b>METHODS</b>					
<b>Eligibility criteria</b>	8	Specify the study characteristics (e.g., PICO, study design, setting, time frame) and report characteristics (e.g., years considered, language, publication status) to be used as criteria for eligibility for the review	<input checked="" type="checkbox"/>	<input type="checkbox"/>	106-120
<b>Information sources</b>	9	Describe all intended information sources (e.g., electronic databases, contact with study authors, trial registers, or other grey literature sources) with planned dates of coverage	<input checked="" type="checkbox"/>	<input type="checkbox"/>	122-127
<b>Search strategy</b>	10	Present draft of search strategy to be used for at least one electronic database, including planned limits, such that it could be repeated	<input checked="" type="checkbox"/>	<input type="checkbox"/>	Table.1
<b>STUDY RECORDS</b>					
Data management	11a	Describe the mechanism(s) that will be used to manage records and data throughout the review	<input checked="" type="checkbox"/>	<input type="checkbox"/>	131-134
Selection process	11b	State the process that will be used for selecting studies (e.g., two independent reviewers) through each phase of the review (i.e., screening, eligibility, and inclusion in meta-analysis)	<input checked="" type="checkbox"/>	<input type="checkbox"/>	134-146
Data collection process	11c	Describe planned method of extracting data from reports (e.g., piloting forms, done independently, in duplicate), any processes for obtaining and confirming data from investigators	<input checked="" type="checkbox"/>	<input type="checkbox"/>	139-144
<b>Data items</b>	12	List and define all variables for which data will be sought (e.g., PICO items, funding sources), any pre-planned data assumptions and simplifications	<input checked="" type="checkbox"/>	<input type="checkbox"/>	139-143 Additional File 2



Section/topic	#	Checklist item	Information reported		Line number(s)
			Yes	No	
<b>Outcomes and prioritization</b>	13	List and define all outcomes for which data will be sought, including prioritization of main and additional outcomes, with rationale	<input checked="" type="checkbox"/>	<input type="checkbox"/>	139-143 Additional File 2
<b>Risk of bias in individual studies</b>	14	Describe anticipated methods for assessing risk of bias of individual studies, including whether this will be done at the outcome or study level, or both; state how this information will be used in data synthesis	<input checked="" type="checkbox"/>	<input type="checkbox"/>	145-146 171-173 Additional File 3
<b>DATA</b>					
<b>Synthesis</b>	15a	Describe criteria under which study data will be quantitatively synthesized	<input checked="" type="checkbox"/>	<input type="checkbox"/>	158-163
	15b	If data are appropriate for quantitative synthesis, describe planned summary measures, methods of handling data, and methods of combining data from studies, including any planned exploration of consistency (e.g., $I^2$ , Kendall's tau)	<input checked="" type="checkbox"/>	<input type="checkbox"/>	158-163
	15c	Describe any proposed additional analyses (e.g., sensitivity or subgroup analyses, meta-regression)	<input checked="" type="checkbox"/>	<input type="checkbox"/>	158-163
	15d	If quantitative synthesis is not appropriate, describe the type of summary planned	<input checked="" type="checkbox"/>	<input type="checkbox"/>	152-156
<b>Meta-bias(es)</b>	16	Specify any planned assessment of meta-bias(es) (e.g., publication bias across studies, selective reporting within studies)	<input type="checkbox"/>	<input checked="" type="checkbox"/>	
<b>Confidence in cumulative evidence</b>	17	Describe how the strength of the body of evidence will be assessed (e.g., GRADE)	<input checked="" type="checkbox"/>	<input type="checkbox"/>	145-146 Additional File 3

### A5.3 PRISMA Checklist - Children

**Note:** This checklist was submitted with the published paper reviewing findings in children, and therefore the page numbers match up to the manuscript and not the line numbers in this thesis.

Section/topic	#	Checklist item	Reported on page #
<b>TITLE</b>			
Title	1	Identify the report as a systematic review, meta-analysis, or both.	1
<b>ABSTRACT</b>			
Structured summary	2	Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number.	2
<b>INTRODUCTION</b>			
Rationale	3	Describe the rationale for the review in the context of what is already known.	3
Objectives	4	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	4
<b>METHODS</b>			
Protocol and registration	5	Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.	4
Eligibility criteria	6	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	4+5
Information sources	7	Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched.	4+5
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	Table 1

Study selection	9	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	4+5 Figure 1
Data collection process	10	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	5
Data items	11	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	5+ Sup file 2
Risk of bias in individual studies	12	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	5+6
Summary measures	13	State the principal summary measures (e.g., risk ratio, difference in means).	6
Synthesis of results	14	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I <sup>2</sup> ) for each meta-analysis.	6 (Narrative Synthesis)

Page 1 of 2

Section/topic	#	Checklist item	Reported on page #
Risk of bias across studies	15	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	6
Additional analyses	16	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	6+7
<b>RESULTS</b>			
Study selection	17	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	7 +Figure 1
Study characteristics	18	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	Table 2
Risk of bias within studies	19	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	7 + Table 2
Results of individual studies	20	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	N/A – Summary

			Tables provided instead. (Tables 3,4+5)
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	N/A – narrative synthesis of results pages 8 to 10
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see Item 15).	N/A
Additional analysis	23	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	N/A – narrative synthesis of results pages 8 to 10
<b>DISCUSSION</b>			
Summary of evidence	24	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).	11-13
Limitations	25	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	14-15
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	15
<b>FUNDING</b>			
Funding	27	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	16

From: Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group (2009). Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. PLoS Med 6(7): e1000097. doi:10.1371/journal.pmed1000097

For more information, visit: [www.prisma-statement.org](http://www.prisma-statement.org).

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## A5.4 PRISMA Checklist - Adults

**Note:** This checklist was submitted with the published paper reviewing findings in adults, and therefore the page numbers match up to the manuscript and not the line numbers in this thesis.

Section/topic	#	Checklist item	Reported on page #
<b>TITLE</b>			
Title	1	Identify the report as a systematic review, meta-analysis, or both.	1
<b>ABSTRACT</b>			
Structured summary	2	Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number.	2
<b>INTRODUCTION</b>			
Rationale	3	Describe the rationale for the review in the context of what is already known.	3
Objectives	4	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	4
<b>METHODS</b>			
Protocol and registration	5	Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.	4
Eligibility criteria	6	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	4
Information sources	7	Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched.	4
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	Table 1.

Study selection	9	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	4+5
Data collection process	10	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	5
Data items	11	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	5
Risk of bias in individual studies	12	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	5 + Sup file 2
Summary measures	13	State the principal summary measures (e.g., risk ratio, difference in means).	5
Synthesis of results	14	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I <sup>2</sup> ) for each meta-analysis.	6 (Narrative Synthesis)

Page 1 of 2

Section/topic	#	Checklist item	Reported on page #
Risk of bias across studies	15	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	6
Additional analyses	16	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	6
<b>RESULTS</b>			
Study selection	17	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	7 + Figure 1
Study characteristics	18	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	Table 2.
Risk of bias within studies	19	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	7 and Table 2
Results of individual studies	20	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	N/A – Summary Tables provided

			instead. (Tables 3+4)
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	N/A
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see Item 15).	N/A
Additional analysis	23	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	N/A
<b>DISCUSSION</b>			
Summary of evidence	24	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).	13
Limitations	25	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	17
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	13-19
<b>FUNDING</b>			
Funding	27	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	19

From: Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group (2009). Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. PLoS Med 6(7): e1000097. doi:10.1371/journal.pmed1000097

For more information, visit: [www.prisma-statement.org](http://www.prisma-statement.org).



## A5.5 Data Extraction Form

### Inclusion/Exclusion form: Socioeconomic position and body composition

Reference details					
A1. Ref ID					
A2. 1 <sup>st</sup> Author					
A3. Title of paper					
A4. Journal					
A5. Volume					
A6. Year of publication					
A7. Publication type	Paper <sup>1</sup>		Abstract <sup>2</sup>		
A8. Assessor's name	CBS <sup>1</sup>		AG <sup>2</sup>		JB <sup>3</sup>
A9. Date					

B. Study included in systematic review:

Yes <sup>1</sup>		No <sup>2</sup>	
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Reason(s) for exclusion (if excluded):	Yes <sup>1</sup>	No <sup>2</sup>
C1. Ineligible exposure		
C2. Ineligible outcome		
C3. Ineligible population (i.e clinical population)		
C4. Review article		
C5. Duplicate (Insert Ref ID of other study)		
	Ref ID:	
C6. Not in English language		
C7. Other please specify:		

### Data extraction form: Socioeconomic position and body composition

Reference details					
A1. Ref ID					
A2. 1 <sup>st</sup> Author					
A3. Title of paper					
A4. Journal					
A5. Volume					
A6. Year of publication					
A7. Publication type	Paper <sup>1</sup>		Abstract <sup>2</sup>		Other <sup>3</sup>
A8. Assessor's name	CBS <sup>1</sup>		AG <sup>2</sup>		JB <sup>3</sup>
A9. Date					

Study details	
B1. Name of study/cohort	

B2. Design	Cross-sectional <sup>1</sup>	Prospective cohort <sup>2</sup>	Retrospective cohort <sup>3</sup>	Case-control <sup>4</sup>	Other <sup>5</sup>
B2A. If other:					
B2B. Participants birth year, if given					
B3. Country	US <sup>1</sup>	UK <sup>2</sup>	AUS <sup>3</sup>	Other <sup>4</sup>	
B3A. If other:					

SEP exposures used					
C1. Own education	Yes <sup>1</sup>			No <sup>2</sup>	
C2. Own Occupation	Yes <sup>1</sup>			No <sup>2</sup>	
C3. Own Income	Yes <sup>1</sup>			No <sup>2</sup>	
C.4 Own Social Class	Yes <sup>1</sup>			No <sup>2</sup>	
C.5 Own SEP	Yes <sup>1</sup>			No <sup>2</sup>	
C6. Father's education	Yes <sup>1</sup>			No <sup>2</sup>	
C7. Mother's education	Yes <sup>1</sup>			No <sup>2</sup>	
C8. Father's occupation	Yes <sup>1</sup>			No <sup>2</sup>	
C9. Mother's occupation	Yes <sup>1</sup>			No <sup>2</sup>	
C10. Father's Social Class	Yes <sup>1</sup>			No <sup>2</sup>	
C11. Mother's Social Class	Yes <sup>1</sup>			No <sup>2</sup>	
C12. Family Income	Yes <sup>1</sup>			No <sup>2</sup>	
C13. Family SEP	Yes <sup>1</sup>			No <sup>2</sup>	
C14. Area-level deprivation index	Yes <sup>1</sup>			No <sup>2</sup>	
C15. Other please list:					
C16. How ascertained	Prospectively <sup>1</sup>			Retrospectively <sup>2</sup>	
C17. Age recorded (yrs.)					
C18. Age Recorded (Grouped)	Childhood <sup>1</sup>		Adolescence <sup>2</sup>		Adulthood <sup>3</sup>

Body Composition Outcomes										
D1. Body Composition Measure	Fat Mass Index <sup>1</sup>		Lean Mass index <sup>2</sup>		Proportion Fat-to-Lean <sup>3</sup>		Location of fat or fat free mass <sup>4</sup>		Other <sup>5</sup>	
D1A. If location please list										
D1B. If other please describe										
D2. How ascertained	BIA		MRI		DXA		CT		Other	
D2B. If Other, please describe										
D4. Age(s) ascertained (yrs.)										
D5. Comments										

Available participant numbers	
E1. Baseline	Yes <sup>1</sup> No <sup>2</sup> If yes, number
E2. Excluded	Yes <sup>1</sup> No <sup>2</sup> If yes, number
E3. Refused	Yes <sup>1</sup> No <sup>2</sup> If yes, number
E4. Lost to follow-up	Yes <sup>1</sup> No <sup>2</sup> If yes, number
E5. Other losses	Yes <sup>1</sup> No <sup>2</sup> If yes, number
E6. Included in analysis	Yes <sup>1</sup> No <sup>2</sup> If yes, number
E7. All accounted for	Yes <sup>1</sup> No <sup>2</sup>

Variable details	
F1. SEP Measure	Continuous <sup>1</sup> Categorical <sup>2</sup> Quantiles <sup>3</sup>
F2. Body Composition	Categories <sup>1</sup> Continuous (original scale) <sup>2</sup> Other <sup>3</sup>
F2A. If other please describe	
F3. Comments	

Analysis					
G1. How results analysed	Linear regression <sup>1</sup>		Logistic regression <sup>2</sup>		Other <sup>3</sup>
G1A. If other:					
G2. Included in analysis	Males and females <sup>1</sup>		Males only <sup>2</sup>		Females only <sup>3</sup>
G3. List models presented incl. unadjusted					

Effect estimates					
Association tested and which group e.g. M/F/All	No. analysed	Type of effect estimate and category comparison/value of unit change	Effect estimate	95% CI; SE; p	Avai

## A5.6 Quality Assessment Form

### NEWCASTLE - OTTAWA QUALITY ASSESSMENT SCALE (amended)

Note: A study can be awarded a maximum of one star for each numbered item within the Selection and Outcome categories. A maximum of two stars can be given for Comparability

#### Selection

- 1) Representativeness of the exposed cohort/study
  - a) Truly representative of the source population \*
  - b) Somewhat representative of the source population \*
  - c) Selected group of users e.g. nurses, volunteers
  - d) No description of the derivation of the cohort
- 2) Ascertainment of SEP
  - a) Prospectively from parents/family/own (or linking to area-level indicators) \*
  - b) Structured interview (recall) \*
  - c) Written self-report
  - d) No description

#### Comparability

- 1) Comparability of cohorts on the basis of the design or analysis
  - a) Study controls for birth weight \*
  - b) Study controls for any additional relevant factors (e.g. age, sex, ethnicity) \*
  - c) Only unadjusted model presented

#### Outcome

- 1) Assessment of body composition
  - a) Measure indexed to body size or ratio (i.e fat mass index or fat:lean ratio) \*
  - b) No indexation or ratio
  - c) No description
- 2) Was follow-up long enough for outcomes to occur (if a) go to 3a, if b) go to 3bi and 3bii)
  - a) Longitudinal \*
  - b) Cross-sectional
- 3a) For longitudinal -Adequacy of follow up of cohorts
  - a) Complete follow up - all subjects accounted for \*
  - b) Subjects lost to follow up unlikely to introduce bias - small number lost - > 75% follow up, or description provided of those lost) \*
  - c) Follow up rate < 75% and no description of those lost
  - d) No statement
- 3bi) For Cross sectional - Sample size:
  - a) Justified and satisfactory. \*
  - b) Not justified.
- 3bii) For Cross sectional - Non-respondents:
  - a) Comparability between respondents and non-respondents characteristics is established, and the response rate is satisfactory. \*

- b) The response rate is unsatisfactory, or the comparability between respondents and non-respondents is unsatisfactory.
- c) No description of the response rate or the characteristics of the responders and the non-responders.

4) Statistical test:

- a) The statistical test used to analyze the data is clearly described and appropriate, and the measurement of the association is presented, including confidence intervals and the probability level (p value). \*
- b) The statistical test is not appropriate, not described or incomplete.

$\geq 7$  \* = high quality

$< 7$  \* = low quality

## A5.7 Descriptive Characteristics of Included Studies

<i>First author</i>	<i>Year published</i>	<i>Country</i>	<i>Data set used (if named)/ description of sample</i>	<i>N</i>	<i>Sep measures</i>	<i>Body composition measures</i>	<i>Technique</i>	<i>Age</i>	<i>Quality score</i>
<b>CHILDHOOD PAPERS</b>									
<b>APOUEY [230]</b>	2016	UK	MCS	14,314	Family Income	FM	BIA	6-8 and 10-12	6*
<b>AZCORRA [231]</b>	2016	Mexico	Mother–child dyads taken from two cross-sectional studies from Merida, Mexico. Recruited through public and private schools with diverse socioeconomic backgrounds.	197	SEI (composite score using mother’s education, father’s occupation and household crowding)	FMI	BIA	6-10 (mean: 8.53)	6*
<b>BAIRD [232]</b>	2016	UK	Southampton Women's Survey	587	Mothers Education; Parental Education	FMI; FFMI	BIA	Mean: 4.1 (0.1) SEP measured before age of 3	7*
<b>BOOT [233]</b>	1997	Netherlands	Caucasian children and adolescents from three primary and one secondary school in Rotterdam.	403	Parental Occupation; Fathers Education; Mothers Education	FM; %FM; LM	Both BIA and DXA	4-20	3*
<b>BROWN [234]</b>	2011	USA	Children in either Kindergarten or third grade in eight elementary schools in the Hilo, Hawaii were invited to participate, with oversampling of native Hawaiian children.	125	Household Income; Mothers Education; Fathers Education	%FM; FM	BIA and plethysmograph ('Bod Pod')	Two age groups: Kindergarten mean age 5.6, Third grade mean age 8.7	4*
<b>BURDETTE [235]</b>	2006	USA	Preschool aged children part of a prospective cohort, born full term or after, without chronic health problems affecting growth and development, and with parents either both black or both white.	313	Maternal Education; Household Income	FM	DXA	4.8-5.2 (mean 5.0) SEP measured age 3.3 (0.3)	4*
<b>CARDEL [236]</b>	2012	USA	Children self-identifying as African American, European American, or Hispanic American from Birmingham, Alabama area.	267	SEP (Hollingshead 4-factor index of social class)	Trunk FM; TAAT	DXA	7-12 (mean: 9.4-9.7)	2*

<b>CARTER [237]</b>	2011	New Zealand	FLAME	244	Mothers Education; Income	FMI; FFMI	Both BIA and DXA	Mean 7 SEP measures age 3	8*
<b>CASTRO [238]</b>	2017	Brazil	Women randomly selected from large maternity hospital in São Paulo city, Brazil.	210	SES (measured by maternal education and housing conditions)	%FM	Air displacement plethysmography	39.42 (weeks) Gestational age	4*
<b>CHENG [239]</b>	2009	Finland	Girls contacted through class teachers in 61 schools in Jyväskylä and its surroundings in Central Finland.	236	Parents Education	FM; LM	DXA	Baseline age 9-13, followed up at multiple points over 7.5 years	6*
<b>CHOMTHO [240]</b>	2008	UK	Healthy children born term from Greater London and Cambridgeshire, recruited through advertisements in schools and sport clubs, the intranet, local newspapers, and word of mouth.	391	Social Class (using the standard occupational classification)	FMI; FFMI	4C Model (combination of Air-displacement plethysmography, Deuterium dilution, DXA)	4-20 (mean: 11.7)	2*
<b>COLLINGS [241]</b>	2015	UK	The ROOTS prospective cohort study	728	Area-Level SES (based on post code)	FMI	BIA	Baseline mean 15, follow up at 17.5	4*
<b>DATTA BANIK [242]</b>	2014a	Mexico	Purposive non-probability sample of adolescents from public and private schools in Merida, Yucatan.	321	SES (based on school type, parents' education, fathers occupation, monthly household food expenditure, crowding index); Mothers Occupation	FM%; DLM	BIA	15-17 (mean: 16.41)	4*
<b>DATTA BANIK<sup>A</sup> [243]</b>	2011	Mexico	Cross sectional study of 13-14 boys from schools in Merida, Mexico.	74	SES (based on school type, parents' education, fathers' occupation and per capita monthly household food expenditure)	DLM; %FFM; %FM; FM	BIA	13-14	2*
<b>DATTA BANIK [244]</b>	2014b	Mexico	Purposive, non-probability sample of adolescents selected from public and private schools in Merida, Yucatan.	270	School Type; Mothers Education; SES (based on mothers' education, fathers occupation, type of school	%FM; %FFM; DLM	BIA	12-16 (mean boys: 14.01; mean girls: 13.90)	3*



					(e.g. private), type of medical care (e.g. private))				
<b>DE VRIENDT [245]</b>	2011	Multiple <sup>c</sup>	HELENA-CSS	1,121	Parents Education	%FM	BIA	12.5-17.5 (mean boys: 14.7; mean girls: 14.8)	6*
<b>DOWDA [246]</b>	2017	USA	TRACK	658	Parental Education	FMI; %FM	BIA	Baseline mean 10.6, followed for two school years	6*
<b>DUNCAN [247]</b>	2008	New Zealand	Children randomly selected from 27 primary schools in Auckland, /New Zealand.	1,229	School level SES (estimated using Ministry of Education decile classification system)	%FM <sup>B</sup>	BIA	5 to 11 (mean 8.4)	6*
<b>EBENEGGER [248]</b>	2011	Switzerland	Randomly selected kindergarten children from 40 classes with high migrant prevalence in two Swiss Cantons.	542	Parents Education; Mothers Education; Fathers Education	FM; %FM	BIA	Mean: 5.1	4*
<b>EKELUND [249]</b>	2005	Sweden	SWEDES	445	Maternal Education	FM; %FM;	Air-displacement plethysmograph	Mean boys: 16.9 Mean girls: 16.8	6*
<b>EKELUND [250]</b>	2006	Sweden	SWEDES	248	Mothers Occupation	%FM; FM; FFM	Air-displacement plethysmograph	Mean boys: 16.9 Mean girls: 16.8	8*
<b>GRACIA-MARCO [251]</b>	2012	Spain	HELENA	322	Affluence Scale; Mothers education; Fathers Education; Mothers Occupation; Fathers Occupation	LM	DXA	12.5-17.5 (mean 14.8)	3*
<b>GRIFFITHS [252]</b>	2013	South Africa	Bt20	346	Maternal Education; Home Ownership; Index of School Environment; Neighbourhood Economic Index	FM, LM	DXA	Mean: 16 SEP measures in infancy and age 16	7*
<b>GRIFFITHS [253]</b>	2008	South Africa	Bt20	281	SES index (created using PCA of multiple measures)	FMI; LMI	DXA	9-10 (mean 9.72)	7*

								SEP measures at birth and 9-10	
<b>HOU [254]</b>	2014	Hong Kong	Hong Kong's 'Children of 1997' birth cohort	502	Parental Education	%ASM; %FM	DXA	15.3	8*
								SEP assumed to be measured at birth (exact age not given)	
<b>HOWE [255]</b>	2010	UK	ALSPAC	7,772	SII for Mother's Education	FM	DXA	Mean 9.9	6*
								SEP collected at 32 weeks gestation	
<b>HOWE [256]</b>	2013	UK	ALSPAC	6,702	Mothers Education	FM	DXA	Mean 9	6*
<b>JOHNSON [257]</b>	2008	UK	ALSPAC	509	Mothers Education	FMI	DXA	Mean 9	4*
								SEP collected at 32 weeks gestation	
<b>KHADILKAR [258]</b>	2012	India	Randomly selected girls from higher and lower socioeconomic stratum schools and colleges in Pune, India.	390	SES (kuppuswamy socioeconomic scale)	LM; FM	DXA	8-17 (mean 12.6)	4*
<b>LAGOA<sup>A</sup> [259]</b>	2014	Portugal	Children from 6 schools in the Porto district, Portugal.	566	Fathers SES; Mothers SES	%FM	BIA	Children	3*
<b>LANTZ<sup>E</sup> [260]</b>	2008	Sweden	Random selection of adolescents from population register from industrial town Trollhättan, Sweden.	203 and 149 at respective ages	Fathers Education	FM; LM; %FM; %LM	DXA	Two age groups: 15 and 17	6*
<b>MAGALHAES [261]</b>	2012	Brazil	Children aged 4-7 from a retrospective cohort who were monitored for the first months of life by a support program to breastfeeding (PROLAC) in the city of Vicosá, southeast Brazil.	185	Mothers Education; Income per capita	%FM <sup>B</sup> ; %Android Fat;	DXA	4-7 (mean 6)	5*
<b>MATSUDO [262]</b>	2016	Brazil	ISCOLE	485	School Type; Income; Maternal Education; Fathers Education	%FM <sup>B</sup>	BIA	9-11	6*

<b>MCCARTHY<sup>A</sup> [263]</b>	2015	UK	Caucasian children from inner city London and from more affluent surrounding counties.	2,297	School Level SEP (based on percentage of children eligible for free school meals)	FFM; ASM; %FFM; ASM as % of Body Weight; Muscle:Fat Ratio; ASM as % of FFM	BIA	5-14	4*
<b>MOLINA-GARCIA [264]</b>	2017	Spain	IPEN	265	Area -level SES (measured by educational level of census blocks)	%FM	BIA	14-18 (mean 16.4)	5*
<b>MOON [141]</b>	2018	Korea	KHANES	1,233	Household Income	%ASM	DXA	12-18 (mean from 13.68 to 15.63)	3*
<b>NESS [265]</b>	2005	UK	ALSPAC	5,917	Social Class (using the 1991 UK Office of Population Censuses and Surveys classification based on occupation)	FM; LM; Trunk FM	DXA	9.9, SEP measured 32 weeks gestation	6*
<b>NGUYEN [266]</b>	2012	USA	NHANES	5,436	PIR	FM; %FM;	DXA	8-19	7*
<b>NGUYEN<sup>A</sup> [267]</b>	2011	USA	NHANES	7,479	PIR	%FM <sup>B</sup> ; FMI	DXA	8-19	4*
<b>PLACHTA-DANIELZIK<sup>A</sup> [268]</b>	2015	Germany	Kiel Obesity Prevention Study	5,352	Parental Education	FM	BIA	5-16	2*
<b>SAMANI-RADIA [269]</b>	2011	UK	Subjects taken from two previous data sets, the first from children in East London and the second from children living in Hertfordshire, Cambridgeshire and West London.	2,298	School Level SEP (based on school location and percentage of children eligible for free school meals)	%FM <sup>B</sup> ; z-%FM	BIA	5-14 (mean 8.86)	5*
<b>SANTOS [270]</b>	2014	Brazil	Pelotas Birth Cohort	3,350	SES; Maternal Education	FM; %FM; FMI; FFM; %FFM	Air-displacement plethysmography	Mean: 6.8	5*
<b>SCHAEFER [271]</b>	2009	USA	ACT	144	Free or reduced meal program (FRMP)	%FM	DXA	Mean: 11.6	3*
<b>SHAKIR [272]</b>	2018	AUS	Data from large multi-centre case-control study of obese and healthy weight adolescents from three Australian states.	234	Household Income	%FM	DXA	10-13 (mean: 11.9)	4*
<b>ULBRICHT [273]</b>	2018	Brazil	Adolescents meeting inclusion criteria (parents authorised, not taking	675	SEP (based on purchasing power of families)	%FM <sup>B</sup>	DXA	11-18 (mean: 14.7)	4*

			medicine containing calcium, haven't undergone radiography/computed tomography a week prior, and were not suspecting pregnancy) from the city of Curitiba-PR, Brazil, and 29 other municipalities.						
<b>VAN DEN BERG [56]</b>	2012	Netherlands	ABCD	1,965	Mothers Education	FMI; LMI; %FM;	BIA	5-6 (mean: 5.7)	7*
<b>VEENA [274]</b>	2014	India	Mysore Parthenon Birth Cohort Study	540	Standard of living Index; Mothers Education; Fathers Education; Occupation; Income (head of the family)	%FM	BIA	9-10 (mean: 9.7)	6*
<b>WILLIG [275]</b>	2011	USA	Children from ongoing cross-sectional study, whose parents classified them through self-report as either African American, European American, or Hispanic American.	254	SEP (Hollingshead 4-factor index of social class)	FMI; FFMI Trunk FMI	DXA	7-12 (means range from 9.4-9.6)	5*
<b>WOHLFAHRT-VEJE [276]</b>	2014	Denmark	Danish Population-Based Mother-Child Cohort	950	SEP (based on parental education and occupation)	%FM	DXA	6-15	5*
<b>ZANINI [277]</b>	2014	Brazil	Pelotas Birth Cohort	3,373	SEI (Constructed through PCA based on consumer goods and education of head of family); Mothers Education	FM;LM; %FM; %LM; FMI; LMI	DXA	6-7 (mean 6.7) SEP measures from perinatal study	6*
<b>ADULTHOOD PAPERS</b>									
<b>AGHA<sup>A</sup> [278]</b>	2013	USA	LEAP	400	Prenatal SEI (a composite score using a weighted percentile of both parents educational attainment, occupation and income relative to the US population).	Android Fat Mass; Android-Gynoid Ratio; Trunk-limb Ratio	DXA	Mean: 48 SEP measured prenatally	6*
<b>AL-QAOUUD [279]</b>	2011	UK	Whitehall II	5,533	Occupation	FFM; LMI	BIA	55-79 (mean: 66) <sup>D</sup>	5*

<b>AMADOR [280]</b>	2017	Scotland	Scottish Family Health Study	11,118	Socioeconomic Covariates (SIMD, years of education, household size, vehicle ratio and job status); SIMD; Education	FM	BIA	18-98	2*
<b>AMANI [281]</b>	2007	Iran	Healthy married women who'd been to one of the 14 main city health centres for a periodic child check-up.	637	Education	%FM	BIA	18 - 40 (mean: 27)	2*
<b>AZARBAL [282]</b>	2016	USA	Women's Health Initiative Clinical Trial and Observational Study	8,832	Income; Education	LM	DXA	50 - 79 (mean: 80)	4*
<b>BAE<sup>A</sup> [283]</b>	2018	Korea	KHANES	3,837	Education	LM, FBF	DXA	50+	2*
<b>BAI [284]</b>	2016	China	Men and women recruited through printed advertisement from the health survey centre of Shanghai Huadong Hospital.	415	Education	ASM; FFM	BIA	60-100 (mean: 72)	3*
<b>BANN [285]</b>	2014	UK	NSHD	1,558	Paternal Occupational Class; Maternal Education; Paternal Education; Own Education; Own Occupational class; Household Income	FMI; Android-Gynoid Ratio; ASMI;	DXA	60-64 SEP collected multiple times from age 4	8*
<b>BARRERA [286]</b>	2017	Chile	Independently living older women in metropolitan Santiago belonging to community centres for older people.	86	Education	FM; FFM	DXA	Mean: 73	1*
<b>BEYDOUN [140]</b>	2009	USA	HANDLS	1,227	SES (a single measures on a standardised z score scale obtained through PCA of education and PIR).	Trunk FM; Trunk FM as % of Body Fat; Total body FM	DXA	30-64	7*
<b>BHUPATHIRAJU [287]</b>	2011	USA	Boston Puerto Rican Osteoporosis Study	629	Education; Income	Abdominal Fat	DXA	Mean: ~60	7*
<b>BRENNAN [288]</b>	2009	Australia	Geelong Osteoporosis Study (GOS)	1,043	Area based SES (Socioeconomic Indexes For Areas (SEIFA) value generated based on the 2006 Census for each subject).	FM; LM	DXA	20-92 (mean: 49)	6*

<b>BUERMANN [289]</b>	1995	Canada	Quebec Family Study	726	Education	%FM	Underwater Densitometry	Means: 42-46	4*
<b>DE MARCHI [290]</b>	2012	Brazil	Random sample of South Brazilians.	471	Income; Education	%FM <sup>B</sup>	BIA	60-80+	7*
<b>DUGAN [291]</b>	2010	USA	SWAN	369	Education	Intra-Abdominal Fat	CT	Mean: 51	5*
<b>DUPUY [292]</b>	2013	France	EPIDOS	1,989	Education	ASM <sup>B</sup>	DXA	Mean: 80	6*
<b>FEDEWA [293]</b>	2014	USA	First year college students recruited through email and print advertising.	177	Area-level SES (Area-level deprivation index)	%FM	DXA	18-20 (mean 18)	3*
<b>GUO [294]</b>	2018	UK	Biobank	162,691	Area-level SES (Townsend deprivation index)	FM	DXA	40-70 (mean 59)	3*
<b>KAZLAUSKAITE [295]</b>	2012	USA	SWAN	257	Income	IAT; IAT-SAT ratio	CT and DXA	Mean: 52	6*
<b>KEIGHLEY [296]</b>	2006	Samoa & American Samoa (USA)	Samoa Family Study of Overweight and Diabetes	1,711	Education; Material Lifestyle; Occupation	%FM	BIA	2 age groups: 18-44, >45 (max age 90)	4*
<b>KEINO<sup>A</sup> [297]</b>	2017	Kenya	Random selection of women age 15-45 in Kenya.	Not Reported	Education	%FM; FMI	Deuterium oxide dilution solution (total body water)	15-45	2*
<b>KIM [298]</b>	2015	Republic of Korea	KHANES	3,285	Education; Income	ASM <sup>B</sup>	DXA	65+	4*
<b>KRUEGER<sup>A</sup> [299]</b>	2010	USA	MIDUS	211	Education	LM; FM	DXA	38-86 (mean 54)	1*
<b>KRUGER [300]</b>	2016	South Africa	PURE	247	Education	ASMI <sup>B</sup>	DXA	45+ (mean 57)	4*
<b>KULKARNI [301]</b>	2010	India	Adult women who were not pregnant or lactating, residing in a large urban slum (Addagutta) in Hyderabad.	278	Type of Employment	LM; FM; Leg FM; Trunk FM	DXA	Mean: 41	5*
<b>LAHMANN [302]</b>	2000b	Sweden	Malmö Diet and Cancer study	27,808	Occupation	%FM	BIA	45-73 (Mean: 57-59)	7*
<b>LAHMANN [303]</b>	2000a	Sweden	Malmö Diet and Cancer study	5,464	Education; Employment; Occupation; Parental Occupation	%FM	BIA	45-73 Parental occupation recalled	6*

<b>LANTZ<sup>E</sup> [260]</b>	2008	Sweden	Random selection of adolescents from population register from industrial town Trollhättan, Sweden.	106	Fathers Education	FM; LM; %FM; %LM	DXA	20.5	6*
<b>LEWIN [304]</b>	2014	France	RECORD	4,078	Own Education, Own Employment, Parental Education; Financial Strain; Neighbourhood Education Level	FMI; %FM	BIA	30-79 Childhood SEP variables recalled	7*
<b>LEWIS [305]</b>	2009	USA	SWAN	418	Education	Visceral Fat	DXA	42-62 Those in original SWAN cohort has SEP measured at baseline (31 - 56)	6*
<b>LOUCKS [306]</b>	2015	USA	New England Family Study	394	Childhood SEI (weighted percentile of both parents' educational attainment, occupation, and income relative to the US population); Education	Android Fat	DXA	46-48 (median: 47) Childhood SEP from age 7	4*
<b>LOURENCO [307]</b>	2008	Brazil (Amazonia)	Adults from Surui' population, an indigenous society from the Brazilian Amazon.	188	SES (based on: materials used in house building; number of sleeping rooms; presence of modern household appliances; and, presence of western style furniture)	%FM	BIA	20 – 85 Grouped into those 20 - 49.9 and 50+	4*
<b>MCCLURE [308]</b>	2011	USA	SWAN	301	Financial Strain; Education	Visceral Fat	CT	46-58 (means: 50-51) SEP measured at base line interview (42–52)	6*
<b>MONGRAW-CHAFFIN [309]</b>	2017	USA	MESA	1,910	Education; Income	Visceral Fat	CT	45-84 (mean 65)	4*

<b>ÖZENER [310]</b>	2007	Turkey	Sample of males made up of labourers from low SEP, non-labourers of low SEP and non-labourers of high SEP living in Ankara, Turkey.	309	SEP (determined by occupation (labourer or student), type of schooling (i.e private schooling or vocational training) and area of city abide in for school and work).	FM; %FM; FFM; FMI; FFMI	BIA	17-20 (mean 18)	1*
<b>PIRILA [311]</b>	2012	Finland	Sample taken from birth cohort of full-term infants with a birth weight over 3,000 g, born at the Helsinki University Central Hospital between January and March 1975.	158	Education; Fathers Education	z-%FM <sup>B</sup> ; LM; %Trunk Fat	DXA	32, childhood SEP variables recalled	6*
<b>POWELL [312]</b>	2016	Italy	Participants selected from ongoing cohort (Milan), followed at the International Centre for the Assessment of Nutritional Status (ICANS, University of Milan).	3,341	Education; Occupation	VAT:FFMI <sup>B</sup> ; FM:FFM <sup>B</sup>	Abdominal ultrasonography and BIA	18-81 (mean 46)	3*
<b>RANGEL PENICHE [313]</b>	2018	Mexico	Non-random sample of health adults from two regions of Mexico.	430	Income; Employment Status; Education	ASM; ASMI; FM; FMI; %FM	DXA	60-83 (means: 69-72)	2*
<b>REBATO [314]</b>	2001	Spain	Caucasian adults living in marginal districts of Bilbao, Spain.	446	SES (low SES determined from degree of poverty and marginality (i.e homelessness, receiving state assistance)).	%FM	BIA	18-65	3*
<b>SALLINEN [315]</b>	2011	Finland	Health 2000 Survey	2,139	Education; Income	%FM <sup>B</sup>	BIA	55+ (mean 67)	5*
<b>SEPPANEN-NUIJTEN [316]</b>	2009	Finland	Health 2000 Survey	5,789	Education	FFM <sup>B</sup>	BIA	30+ Split into those aged 30-64 and 65+	6*
<b>SOTILLO [317]</b>	2007	Spain	Probabilistic and stratified sample of adults in the region of Andalusia.	394	Education	FFM; %FM; FM	BIA	20-60 (means: 42-44)	6*
<b>SUDER [318]</b>	2009	Poland	Young working males employed by the Sendzimir Metallurgical Plant in Cracow, and other companies on its premises.	259	SES (based on birthplace, place of residence in childhood, social class, education level, type of work)	%FM	BIA	20-30 (mean 27)	3*



<b>VELASQUEZ-MELEN [319]</b>	2005	Brazil	Women of good health with no chronic or acute metabolic or infectious complaints, recruited from municipal health centre in the city of Belo Horizonte.	410	Education; Income	%FM	BIA	20-55 (mean 33)	4*
<b>VISSER [320]</b>	1998	USA	Framingham Heart Study	753	Education	LM <sup>B</sup> ; %FM <sup>B</sup>	DXA	72-95 (mean 76-79) Baseline measures taken 30-62	6*
<b>WU [321]</b>	2003	Taiwan	Tainan Diabetes and Related Chronic Disease Survey	1,103	SES (Hollingshead index).	%FM	BIA	20+ (mean 48)	5*
<b>YLIHA'RSILA'' [322]</b>	2007	Finland	Helsinki Birth Cohort	2,003	Childhood Social Class (based on fathers occupation); Social Class (based on own occupation)	%FM	BIA	Mean: 62 SEP derived from census data from 23 years earlier and multiple points in childhood	6*

**Table A5.7 Footnote:** Where papers have reported either body fat or fat mass, the variable is listed as just fat mass. Mean ages are rounded to one full year. Range of mean age is given when only the means of sub-groups, and not the full samples, are presented (i.e. mean ages presented separately for males/females).

<sup>A</sup> Indicates abstract only.

<sup>B</sup> Papers have created a categorical or dichotomous variable (i.e. underfat/normal fat/excess fat) based on the indicated continuous measure.

<sup>C</sup> Sweden, Austria, Hungary, Greece, Spain, Belgium.

<sup>D</sup> Al-Qaoud for those individuals that had retired at phase 9, an earlier measure of SEP was taken.

<sup>E</sup> Lantz is included in both adults and children.

\* Indicates star rating. i.e. 6\* is equivalent to "6 stars" on quality assessment.

**Abbreviations:** SEI – Socioeconomic Index; SIMD – Scottish Index of Multiple Deprivation; SEP – Socioeconomic Position; SES – Socioeconomic Status; SII – Slope of Inequality Index; PIR – Poverty Income Ratio; FRMP - Free or Reduced Meal Program; PCA – Principle Component Analysis; FM - Fat Mass; FFM - Fat-Free Mass; FMI - Fat Mass Index; FFMI – Fat-Free Mass Index; ASM – Appendicular Skeletal Muscle; ASMI – Appendicular Skeletal Muscle Index; LM – Lean Mass; DLM – Dry Lean Mass; LMI – Lean Mass Index; TAAT – Total Abdominal Adipose Tissue; IAT – intra-abdominal adipose tissue; SAT – subcutaneous abdominal adipose tissue; VAT – visceral adipose tissue; BIA – Bioelectrical Impedance Analysis; DXA – Dual X-ray Absorptiometry;

*CT - Computed Tomography; PCA – Principle Component Analysis.*

**Abbreviations Study Names Children:** *ABCD- Amsterdam Born Children and their Development; ACT - the Adequate Calcium Today project, ; ALSPAC - Avon Longitudinal Study of Parents and Children; Bt20 - Birth to Twenty; FLAME - The Family Lifestyle, Activity, Movement and Eating study ; HELENA - the Healthy Lifestyle in Europe by Nutrition in Adolescence; HELENA-CSS - the Healthy Lifestyle in Europe by Nutrition in Adolescence cross-sectional study ; IPEN - the International Physical Activity and the Environment Network; ISCOLE - the International Study of Childhood Obesity, Lifestyle and Environment; KHANES - The Korea National Health and Nutrition Examination Survey; MCS – Millennium Cohort Study; NHANES- National Health and Nutrition Examination Survey; SWEDES - The Stockholm Weight Development Study; TRACK - the Transitions and Activity Changes in Kids study.*

**Abbreviations Study Names Adults:** *TAPS – Tsimane’ Amazonian Panel Study; MESA- The Multi-Ethnic Study of Atherosclerosis; SWAN- The Study of Women’s Health Across the Nation; RECORD- Residential Environment and Coronary Heart Disease Cohort Study; PURE- Prospective Urban and Rural Epidemiology; MIDUS- The Midlife in the United States; KNHANES- The Korea National Health and Nutrition Examination Survey; EPIDOS- EPIDémiologie de l’OStéoporose; HANDLS- Healthy Aging in Neighbourhoods of Diversity across the Life Span; NSHD- National Survey of Health and Development; LEAP - Longitudinal Effects on Aging Perinatal Project.*

## **A6. Chapter 6 Appendix**

### **A6.1 Publications from This Work**

Bridger Staatz C, Kelly Y, Lacey R, Hardy R (2021) “Area- and family-level socioeconomic position and body composition trajectories in the UK Millennium Cohort Study” *Lancet Public Health* 2021;6: e598–607

## A6.2 Description of Auxiliary Variables Included in Imputation Model

<b>Auxiliary Variable</b>	<b>Question Wording</b>	<b>Question Responses</b>
<i>Housing Tenure</i>	Current housing tenure at time of interview	Own outright / Own - mortgage/loan / Part rent/part mortgage (shared equity) /Rent from local authority / Rent from Housing Association / Rent privately / Living with parents / Live rent free / Squatting or Other
<i>Partners BMI</i>	Partner BMI at interview	BMI Value (continuous)
<i>Mothers BMI</i>	Natural Mothers BMI at Interview	BMI Value (continuous)
<i>Birthweight</i>	Birthweight	Value in Kg
<i>Combined Labour Status</i>	Combined labour market status of Main and Partner	Both in work / One Parent in work / Both not in work / Only one parent - in work / Only one parent - not in work
<i>Ever Breast Fed</i>	Have you ever tried to breast feed?	Yes/No
<i>Longstanding Illness</i>	Do you have a longstanding illness? Main respondent	Yes/No
<i>Self-Rated Financial Difficulty</i>	Self-rated financial status from main respondent	living comfortably / doing alright / just about getting by / finding it quite difficult / finding it very difficult
<i>Main Depression</i>	Experience of depression in main respondent	Yes/No
<i>Main Smoking Status</i>	Current Smoking Status in main respondent	Yes/No
<i>Main Alcohol Consumption</i>	Frequency of current alcohol consumption in main respondent.	Every Day / 5-6 times a week /3-4 times per week / 1-2 times per week / 1-2 times per month / Less than once a month / Never

**Table A6.2 Footnote:** All variables were taken from sweep 1 or from both sweep 1 and 2 (*Mothers BMI and Birthweight*). Squatting and other groups were combined for housing tenure, as low count ( $\leq 10$ ) of respondents listed squatting as their housing circumstances.

### A6.3 Mean Body Composition Measures by Sex and Age with Adjusted Wald Test for Differences Between Sex

	<b>Age 7</b>		<b>Age 11</b>		<b>Age 14</b>		<b>Age 17</b>	
	<b>Mean (SD)</b>		<b>Mean (SD)</b>		<b>Mean (SD)</b>		<b>Mean (SD)</b>	
	<b>Boys</b>	<b>Girls</b>	<b>Boys</b>	<b>Girls</b>	<b>Boys</b>	<b>Girls</b>	<b>Boys</b>	<b>Girls</b>
<b>N</b>	6,791	6,645*	6,428	6,295	5,493	5,336	4,587	4,582
<b>Fat Mass (kg)</b>	5.32 (2.55)	5.86 (2.75)	8.78 (5.70)	11.03 (6.04)	10.79 (7.87)	16.48 (8.20)	12.54 (9.22)	19.08 (10.28)
<i>P value</i>	<0.001		<0.001		<0.001		<0.001	
<b>Fat Mass %</b>	20.02 (5.02)	22.15 (5.63)	20.08 (7.69)	24.64 (7.45)	16.91 (8.17)	27.28 (7.26)	15.97 (8.12)	28.25 (8.32)
<i>P value</i>	<0.001		<0.001		<0.001		<0.001	
<b>Fat Mass Index (kg/m<sup>2</sup>)</b>	3.41 (1.45)	3.81 (1.60)	4.05 (2.43)	5.04 (2.55)	3.85 (2.76)	6.31 (3.05)	4.00 (2.92)	7.06 (3.72)
<i>P value</i>	<0.001		<0.001		<0.001		<0.001	
<b>Fat Mass Index (Benn Index)</b>	2.27 (0.89)	2.34 (0.91)	1.42 (0.80)	1.89 (0.92)	2.33 (1.67)	4.13 (1.99)	4.07 (2.97)	3.62 (1.91)
<i>P value</i>	<0.001		<0.001		<0.001		<0.001	
<b>Fat-Free Mass (kg)</b>	20.33 (2.75)	19.6 (2.64)	32.02 (5.25)	31.17 (5.07)	48.09 (7.97)	41.06 (5.26)	59.44 (7.92)	44.75 (4.97)
<i>P value</i>	<0.001		<0.001		<0.001		<0.001	
<b>Fat-Free Mass %</b>	79.98 (5.02)	77.85 (5.63)	79.92 (7.69)	75.36 (7.45)	83.09 (8.17)	72.72 (7.26)	84.03 (8.12)	71.75 (8.32)
<i>P value</i>	<0.001		<0.001		<0.001		<0.001	
<b>Fat-Free Mass Index (kg/m<sup>2</sup>)</b>	13.15 (1.06)	12.86 (0.99)	14.97 (1.62)	14.40 (1.41)	17.19 (1.86)	15.77 (1.47)	18.96 (2.02)	16.63 (1.65)
<i>P value</i>	<0.001		<0.001		<0.001		<0.001	
<b>Fat-Free Mass Index (Benn Index)</b>	11.99 (0.94)	11.75 (0.87)	11.8 (1.23)	11.57 (1.09)	12.78 (1.33)	14.27 (1.33)	18.22 (1.94)	22.26 (2.14)
<i>P value</i>	<0.001		<0.001		<0.001		<0.001	
<b>FM: FFM Ratio</b>	0.26 (0.09)	0.29 (0.10)	0.27 (0.15)	0.34 (0.14)	0.22 (0.15)	0.39 (0.15)	0.20 (0.14)	0.41 (0.18)
<i>P value</i>	<0.001		<0.001		<0.001		<0.001	

**Table A6.3 Footnote:** \* Sample size is N=6,646 for fat mass percentage (FM%) in girls at age 7 because one observation had missing data for weight but not FM%. P-value for adjusted Wald test. Abbreviations: FM:FFM – fat mass to fat-free mass ratio; SD – standard deviation.

#### A6.4 Mean Anthropometric Measures by Sex and Age with Adjusted Wald Test for Differences Between Sex

	<b>Age 7</b>		<b>Age 11</b>		<b>Age 14</b>		<b>Age 17</b>	
	<b>Mean (SD)</b>		<b>Mean (SD)</b>		<b>Mean (SD)</b>		<b>Mean (SD)</b>	
	<b>Boys</b>	<b>Girls</b>	<b>Boys</b>	<b>Girls</b>	<b>Boys</b>	<b>Girls</b>	<b>Boys</b>	<b>Girls</b>
<b>Weight (kg)</b>	25.63 (4.84)	25.46 (5.04)	40.81 (9.75)	42.21 (10.43)	58.85 (13.61)	57.56 (12.73)	72.26 (15.55)	63.84 (14.47)
<i>N</i>	6,894	6,741	6,512	6,361	5,572	5,396	4,660	4,670
<i>P value</i>	0.092		<0.001		<0.001		<0.001	
<b>Height (m)</b>	1.24 (0.06)	1.23 (0.06)	1.46 (0.07)	1.47 (0.08)	1.67 (0.09)	1.61 (0.06)	1.77 (0.07)	1.64 (0.06)
<i>N</i>	6,930	6,758	6,594	6,489	5,642	5,622	4,723	4,850
<i>P value</i>	<0.001		<0.001		<0.001		<0.001	
<b>Body Mass Index (kg/m<sup>2</sup>)</b>	16.56 (2.26)	16.67 (2.41)	19.03 (3.54)	19.45 (3.77)	21.04 (4.05)	22.09 (4.39)	23.05 (4.61)	23.71 (5.07)
<i>N</i>	6,894	6,740	6,511	6,360	5,572	5,396	4,656	4,668
<i>P value</i>	0.028		<0.001		<0.001		0.002	
<b>Body Mass Index (Benn Index)</b>	13.97 (1.81)	13.84 (1.89)	12.46 (2.19)	12.9 (2.39)	15.41 (2.94)	18.52 (3.67)	22.92 (4.59)	25.28 (5.41)
<i>N</i>	6,894	6,740	6,511	6,360	5,572	5,396	4,656	4,668
<i>P value</i>	0.001		<0.001		<0.001		<0.001	

**Table A6.4 Footnote:** Samples size for weight, height, and body mass index are reported individually as they vary between measures at each age. *P*-value for adjusted Wald test.

Abbreviations: *SD* – standard deviation.

A6.5 Distribution of Sample by the Index of Multiple Deprivation at Sweep 1 according to Sociodemographic Variables at Sweep 1

	<i>Most Deprived 20%</i>	<i>40%</i>	<i>60%</i>	<i>80%</i>	<i>Least Deprived 20%</i>	<i>P Value</i>
<b>Sex</b>						
Boys	3,232 (32.7%)	2,211 (22.4%)	1,653 (16.7%)	1,344 (13.6%)	1,454 (14.7%)	0.41
Girls	3,076 (32.9%)	2,158 (23.1%)	1,549 (16.6%)	1,238 (13.2%)	1,328 (14.2%)	
<b>Ethnicity</b>						
White	4,145 (26.5%)	3,646 (23.3%)	2,818 (18.0%)	2,371 (15.2%)	2,658 (17.0%)	<0.001
Mixed	256 (43.8%)	145 (24.8%)	83 (14.2%)	66 (11.3%)	35 (6.0%)	
Indian	185 (37.4%)	164 (33.1%)	74 (15.0%)	41 (8.3%)	31 (6.3%)	
Pakistani and Bangladeshi	1,062 (79.7%)	148 (11.1%)	73 (5.5%)	24 (1.8%)	26 (2.0%)	
Black and Black British	472 (65.6%)	153 (21.3%)	66 (9.2%)	23 (3.2%)	6 (0.8%)	
Other Ethnic Group	120 (40.1%)	74 (24.8%)	58 (19.4%)	33 (11.0%)	14 (4.7%)	
<b>Income</b>						
Lowest Quintile	2,892 (60.0%)	1,099 (22.8%)	490 (10.2%)	214 (4.4%)	127 (2.6%)	<0.001
2nd Quintile	1,920 (44.8%)	1,084 (25.3%)	636 (14.9%)	370 (8.6%)	274 (6.4%)	
3rd Quintile	780 (22.0%)	1,002 (28.3%)	745 (21.1%)	537 (15.2%)	475 (13.4%)	
4th Quintile	364 (11.2%)	681 (21.0%)	701 (21.6%)	708 (21.9%)	787 (24.3%)	
Highest Quintile	220 (7.4%)	427 (14.3%)	568 (19.0%)	705 (23.6%)	1,065 (35.7%)	
<b>National Vocational Qualification</b>						
No Qualifications	1,395 (64.4%)	449 (20.7%)	224 (10.3%)	64 (3.0%)	33 (1.5%)	<0.001
Overseas Qualification	279 (61.3%)	99 (21.8%)	41 (9.0%)	23 (5.1%)	13 (2.9%)	
NVQ 1	654 (53.2%)	303 (24.7%)	146 (11.9%)	84 (6.8%)	42 (3.4%)	
NVQ 2	1,854 (38%)	1,262 (25.9%)	828 (17.0%)	524 (10.7%)	408 (8.4%)	
NVQ 3	903 (29.5%)	790 (25.8%)	571 (18.6%)	415 (13.5%)	385 (12.6%)	
NVQ 4	970 (16.2%)	1,214 (20.3%)	1,131 (18.9%)	1,184 (19.8%)	1,477 (24.7%)	
NVQ 5	155 (13.1%)	195 (16.5%)	208 (17.6%)	247 (20.9%)	375 (31.8%)	
<b>NS-SEC</b>						
Unemployed	1,004 (70.1%)	259 (18.1%)	114 (8.0%)	28 (2.0%)	27 (1.9%)	<0.001
Semi-routine and routine	2501 (50.0%)	1329 (26.6%)	643 (12.8%)	335 (6.7%)	196 (3.9%)	
Lower supervisory and technical	594 (37.4%)	425 (26.8%)	288 (18.1%)	155 (9.8%)	126 (7.9%)	
Small employers and self-employed	335 (29.6%)	299 (26.4%)	226 (20.0%)	156 (13.8%)	116 (10.2%)	
Intermediate	693 (28.7%)	597 (24.7%)	455 (18.8%)	325 (13.4%)	347 (14.4%)	
Managerial and Professional	907 (12.9%)	1,342 (19.1%)	1,374 (19.5%)	1,511 (21.5%)	1,900 (27.0%)	

**Table A6.5 Footnote:** Distribution of the sample by index of multiple deprivation (IMD) according to sex, ethnicity, parental education, household income, and parental occupation at sweep 1. P-values for persons chi-squared statistic.

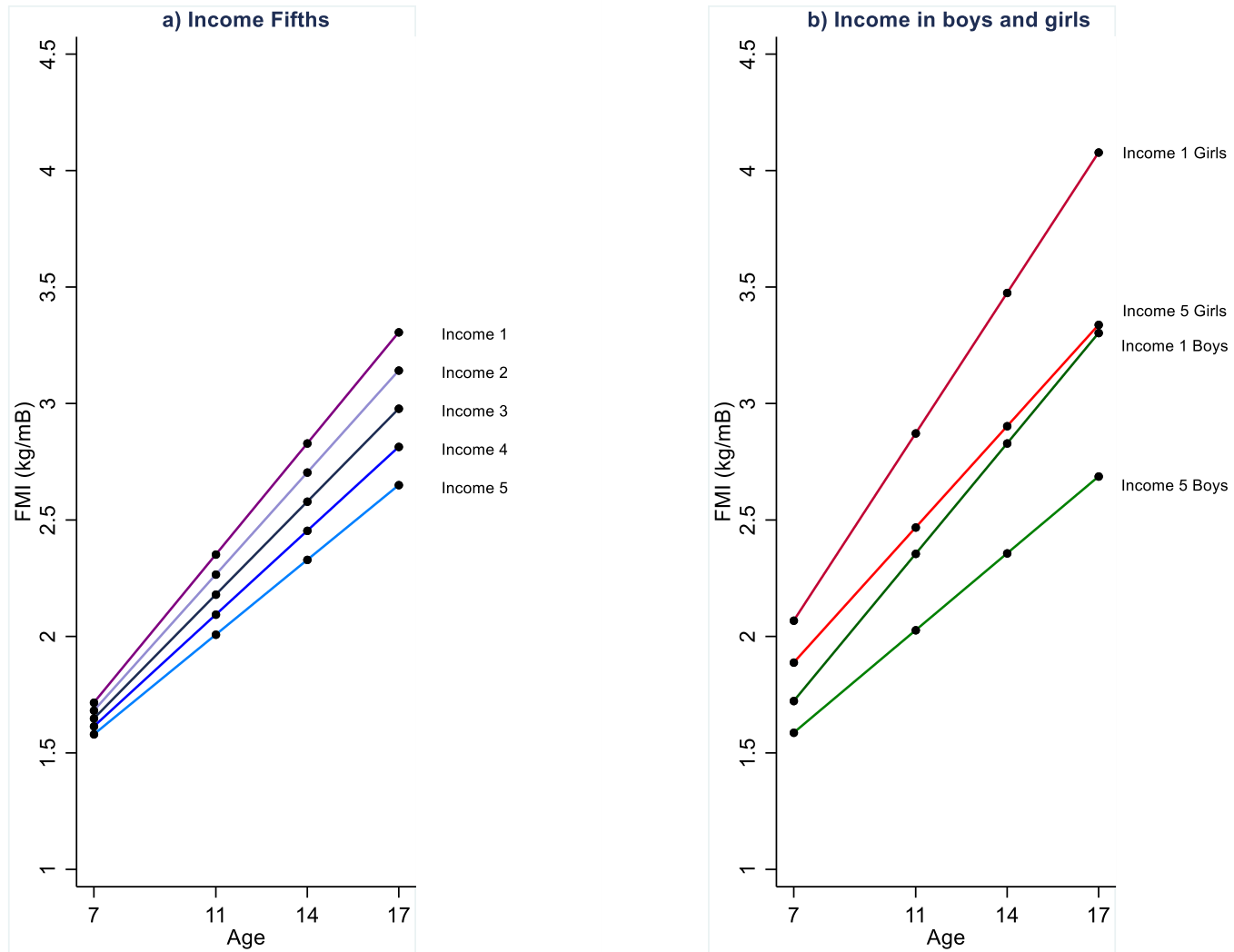
A6.6 Distribution of the Sample by Income Fifths at Sweep 1 according to Sociodemographic Variables at Sweep 1

	<b>Lowest Income Fifth</b>	<b>Second Income Fifth</b>	<b>Third Income Fifth</b>	<b>Fourth Income Fifth</b>	<b>Highest Income Fifth</b>	<b>P Value</b>
<b>Sex</b>						
Boys	2,471 (25.4%)	2,222 (22.9%)	1,833 (18.9%)	1,675 (17.2%)	1,515 (15.6%)	0.99
Girls	2,351 (25.7%)	2,062 (22.5%)	1,706 (18.6%)	1,566 (17.1%)	1,470 (16.1%)	
<b>Ethnicity</b>						
White	3,438 (22.3%)	3,228 (20.9%)	3,132 (20.3%)	2,945 (19.1%)	2,683 (17.4%)	<0.001
Mixed	216 (37.3%)	129 (22.3%)	68 (11.7%)	70 (12.1%)	96 (16.6%)	
Indian	85 (17.5%)	147 (30.2%)	106 (21.8%)	67 (13.8%)	81 (16.7%)	
Pakistani and Bangladeshi	639 (48.7%)	510 (38.8%)	88 (6.7%)	46 (3.5%)	30 (2.3%)	
Black and Black British	347 (49.0%)	160 (22.6%)	84 (11.9%)	65 (9.2%)	52 (7.3%)	
Other Ethnic Group	82 (27.7%)	95 (32.1%)	48 (16.2%)	39 (13.2%)	32 (10.8%)	
<b>National Vocational Qualification</b>						
No Qualifications	1,426 (66.2%)	546 (25.3%)	129 (6.0%)	38 (1.8%)	15 (0.7%)	<0.001
Overseas Qualification	222 (49.3%)	161 (35.8%)	44 (9.8%)	13 (2.9%)	10 (2.2%)	
NVQ 1	656 (53.9%)	359 (29.5%)	138 (11.3%)	53 (4.4%)	12 (1.0%)	
NVQ 2	1474 (30.4%)	1,489 (30.7%)	1,124 (23.1%)	528 (10.9%)	241 (5.0%)	
NVQ 3	580 (19.0%)	855 (28.0%)	774 (25.3%)	581 (19.0%)	267 (8.7%)	
NVQ 4	418 (7.0%)	810 (13.6%)	1,234 (20.7%)	1,780 (29.9%)	1,716 (28.8%)	
NVQ 5	46 (3.9%)	64 (5.4%)	96 (8.1%)	248 (21.1%)	724 (61.5%)	
<b>NS-SEC</b>						
Unemployed	1,039 (74.7%)	266 (19.1%)	47 (3.4%)	20 (1.4%)	19 (1.4%)	<0.001
Semi-routine and routine	2,365 (47.5%)	1,684 (33.8%)	649 (13.0%)	213 (4.3%)	72 (1.4%)	
Lower supervisory and technical	339 (21.4%)	593 (37.4%)	423 (26.7%)	172 (10.9%)	58 (3.7%)	
Small employers and self-employed	189 (16.9%)	328 (29.3%)	309 (27.6%)	199 (17.8%)	95 (8.5%)	
Intermediate	391 (16.2%)	611 (25.3%)	727 (30.1%)	493 (20.4%)	190 (7.9%)	
Managerial and Professional	293 (4.2%)	719 (10.2%)	1,356 (19.3%)	2,125 (30.3%)	2,527 (36.0%)	

**Table A6.6 Footnote:** Distribution of the sample by income fifths according to sex, ethnicity, parental education, and parental occupation at sweep 1. P-values for persons chi-squared statistic.



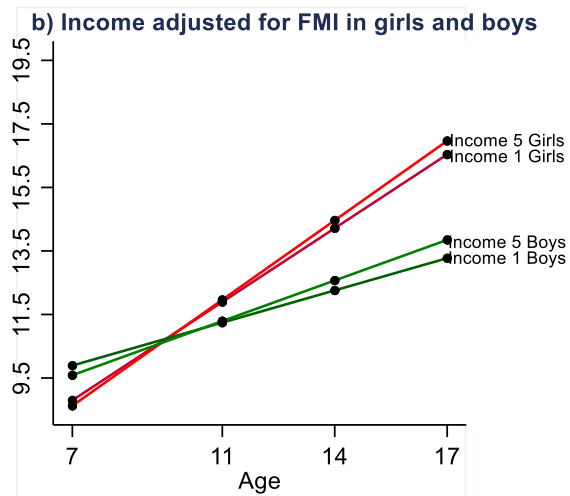
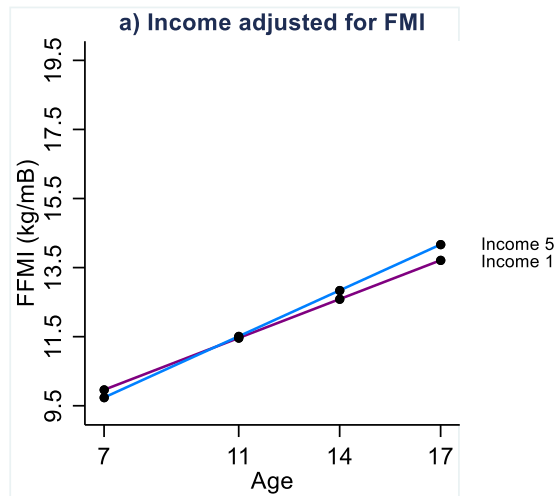
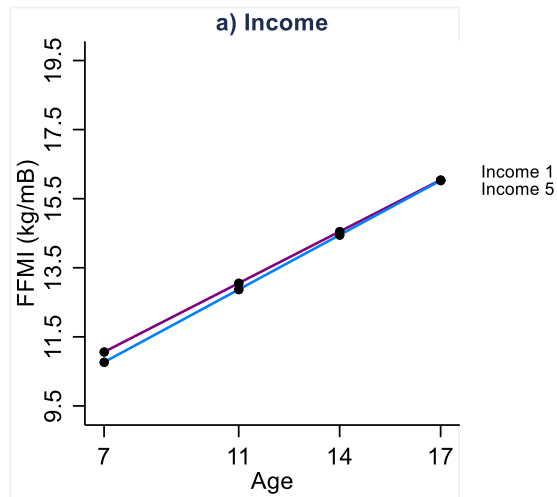
## A6.7 Trajectories of Fat Mass Index (FMI) by Income Fifths



**Figure A6.7. Footnote**

Estimated mean trajectories of Fat Mass Index (FMI) by income fifths. Income 1 = lowest income fifth. Income 5 = Highest income fifth. Difference in FMI ( $\text{kg}/\text{m}^2$ ) at ages 7, 11, 14 and 17. Graph a) adjusted only for sex and ethnicity (model 4); b) Model 4 in boys and girls separately.

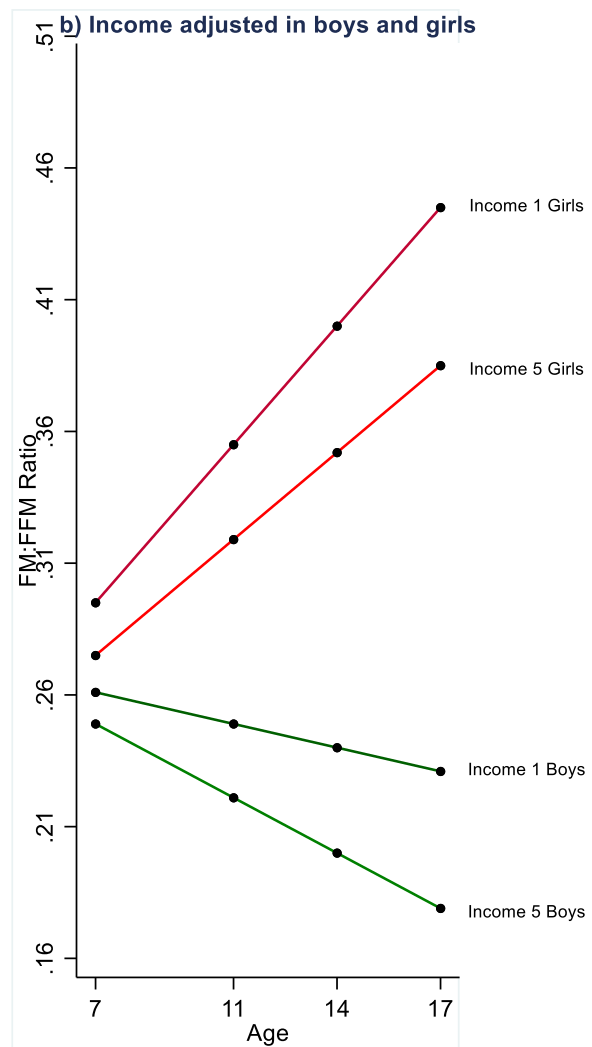
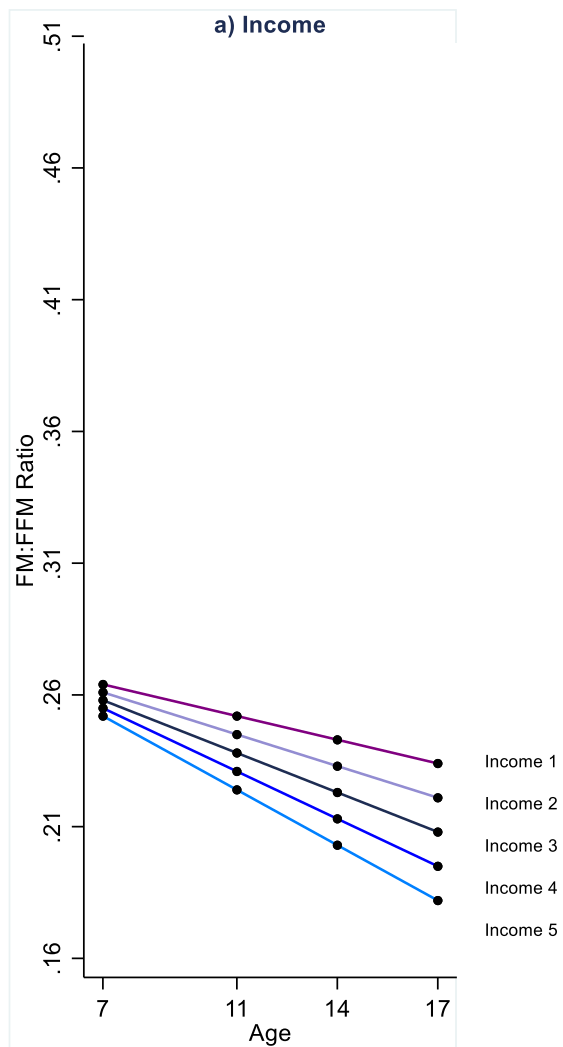
## A6.8 Trajectories of Fat-Free Mass Index (FFMI) by Income Fifths



**Figure A6.8. Footnote**

Estimated mean trajectories of fat-free mass index (FFMI) by income fifths. Income 1 = lowest income fifth. Income 5 = Highest income fifth. Difference in FFMI ( $\text{kg}/\text{m}^2$ ) at ages 7, 11, 14 and 17. Graph a) adjusted only for sex and ethnicity (model 4); b) Model 4 in boys and girls separately; c) Adjusted for sex, ethnicity and FMI (model 6); d) Model 6 in boys and girls separately.

## A6.9 Trajectories of Fat Mass: Fat-Free Mass by Income Fifths



**Figure A6.9. Footnote**

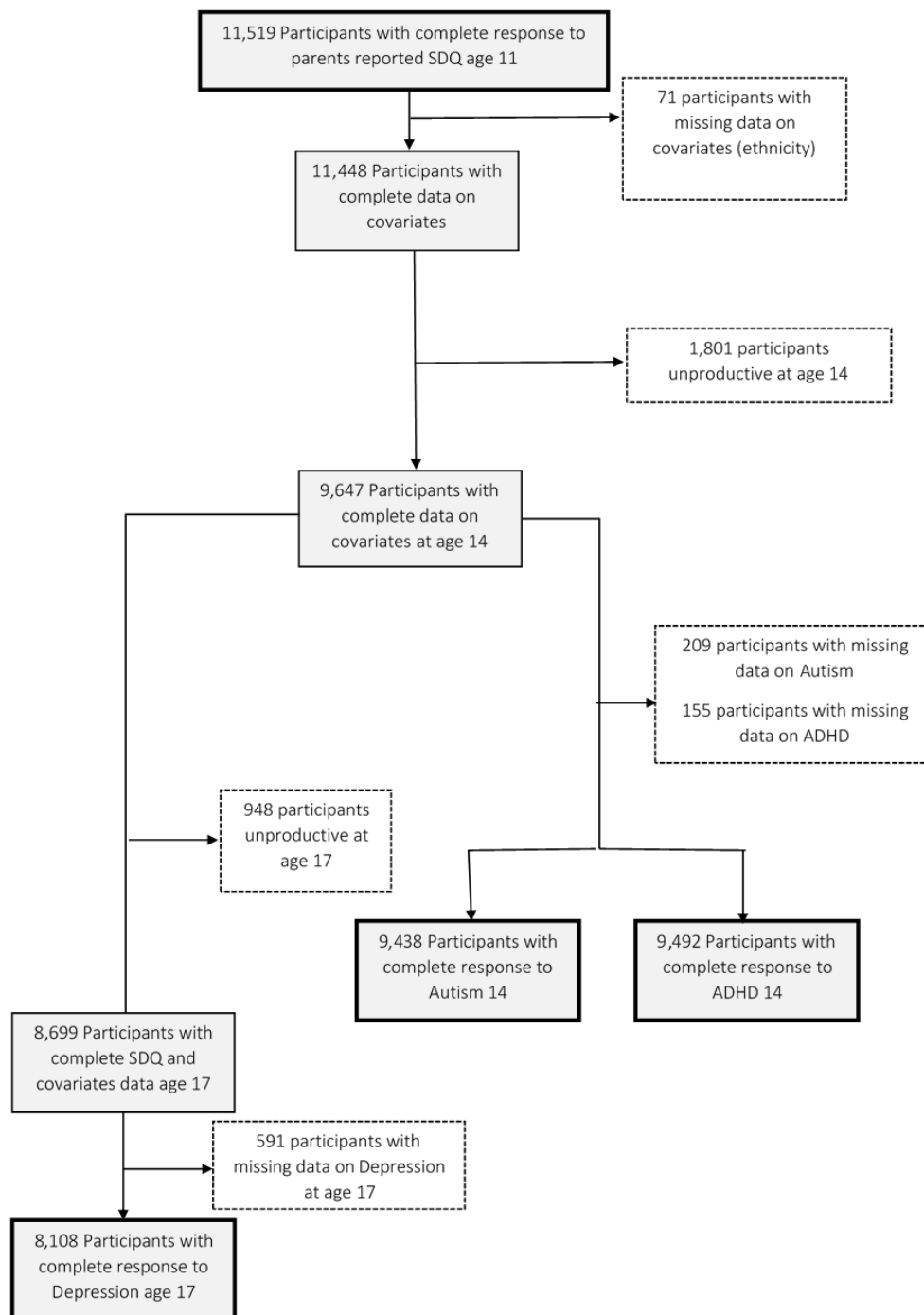
Estimated mean trajectories of fat mass: fat-free mass (FM:FFM) ratio by income fifths. Income 1 = lowest income fifth. Income 5 = Highest income fifth. Difference in FM:FFM ratio at ages 7, 11, 14 and 17. Graph a) adjusted only for sex and ethnicity (model 4); b) Model 4 in boys and girls separately.

## A7. Chapter 7 Appendix

### A7.1 Description of Analytic Sample for Factor Model with Cross Loadings Removed

At age 11, a total of 11,575 participants had complete data on the SDQ for all items apart from “impulse” and “liked” which were removed due to cross-loadings. In EFA and CFA, the sample size for the development side was 5,846 whilst for testing was 5,729. When testing predictive validity, the sample size for depression analysis was 8,032, ADHD analysis was 9,530 and Autism/Asperger’s was 9,474.

### A7.2 Analytic Sample for Predictive Validity Analysis



## A7.3 Distribution of Sample by SDQ Subscales

### A7.3.1 Internalising Symptoms Sub-Scales

	Emotional Scale (Proportion)			Peer Problems (Proportion)		
	Boys	Girls	Total	Boys	Girls	Total
	<b>COMPLAINS</b>			<b>ALONE</b>		
<b>1. Not True</b>	0.36	0.30	0.66	0.34	0.35	0.69
<b>2. Somewhat True</b>	0.12	0.15	0.27	0.13	0.11	0.25
<b>3. Certainly True</b>	0.03	0.04	0.07	0.04	0.02	0.06
<b>Total</b>	0.51	0.49	1.00	0.51	0.49	1.00
<i>P Value</i>	<0.001			<0.001		
	<b>WORRIED</b>			<b>FRIEND*</b>		
<b>1. Not True</b>	0.31	0.30	0.61	0.02	0.01	0.03
<b>2. Somewhat True</b>	0.16	0.16	0.32	0.05	0.04	0.09
<b>3. Certainly True</b>	0.04	0.03	0.07	0.45	0.43	0.88
<b>Total</b>	0.51	0.49	1.00	0.51	0.49	1.00
<i>P Value</i>	0.015			0.074		
	<b>UNHAPPY</b>			<b>LIKED*</b>		
<b>1. Not True</b>	0.42	0.41	0.82	0.01	0.00	0.01
<b>2. Somewhat True</b>	0.08	0.07	0.15	0.08	0.06	0.14
<b>3. Certainly True</b>	0.01	0.01	0.03	0.42	0.42	0.84
<b>Total</b>	0.51	0.49	1.00	0.51	0.49	1.00
<i>P Value</i>	0.031			<0.001		
	<b>ANXIETY</b>			<b>BULLIED</b>		
<b>1. Not True</b>	0.31	0.29	0.60	0.37	0.37	0.73
<b>2. Somewhat True</b>	0.15	0.16	0.31	0.12	0.10	0.21
<b>3. Certainly True</b>	0.05	0.04	0.08	0.03	0.02	0.05
<b>Total</b>	0.51	0.49	1.00	0.51	0.49	1.00
<i>P Value</i>	0.015			0.005		
	<b>FEAR</b>			<b>ADULTS</b>		
<b>1. Not True</b>	0.36	0.33	0.69	0.32	0.32	0.64
<b>2. Somewhat True</b>	0.12	0.13	0.25	0.15	0.13	0.28
<b>3. Certainly True</b>	0.03	0.03	0.06	0.04	0.03	0.08
<b>Total</b>	0.51	0.49	1.00	0.51	0.49	1.00
<i>P Value</i>	0.17			0.012		

**Table A.7.3.1 Footnote:** Table showing proportions for responses to SDQ items that make up internalising symptoms sub-scales, by sex. *P*-Values are for Pearson's Chi Squared statistics.

### A7.3.2 Externalising Symptoms Sub-Scales

	Conduct Problems (Proportion)			Hyperactivity Scale (Proportion)		
	Boys	Girls	Total	Boys	Girls	Total
	<b>ANGER</b>			<b>RESTLESS</b>		
<b>1. Not True</b>	0.25	0.25	0.50	0.27	0.32	0.59
<b>2. Somewhat True</b>	0.18	0.17	0.35	0.16	0.12	0.28
<b>3. Certainly True</b>	0.09	0.07	0.16	0.08	0.05	0.13
<b>Total</b>	0.51	0.49	1.00	0.51	0.49	1.00
<i>P Value</i>	<0.001			<0.001		
	<b>OBEDIENCE*</b>			<b>FIDGET</b>		
<b>1. Not True</b>	0.03	0.02	0.05	0.32	0.36	0.69
<b>2. Somewhat True</b>	0.19	0.16	0.35	0.14	0.09	0.23
<b>3. Certainly True</b>	0.29	0.31	0.60	0.06	0.03	0.09
<b>Total</b>	0.51	0.49	1.00	0.52	0.48	1.00
<i>P Value</i>	<0.001			<0.001		
	<b>AGRESSION</b>			<b>ATTENTION</b>		
<b>1. Not True</b>	0.46	0.46	0.91	0.19	0.25	0.44
<b>2. Somewhat True</b>	0.04	0.02	0.07	0.22	0.18	0.40
<b>3. Certainly True</b>	0.01	0.00	0.02	0.10	0.06	0.16
<b>Total</b>	0.51	0.49	1.00	0.51	0.49	1.00
<i>P Value</i>	<0.001			<0.001		
	<b>LIES</b>			<b>IMPULSE*</b>		
<b>1. Not True</b>	0.39	0.41	0.80	0.08	0.05	0.13
<b>2. Somewhat True</b>	0.11	0.07	0.18	0.30	0.27	0.57
<b>3. Certainly True</b>	0.02	0.01	0.03	0.13	0.16	0.30
<b>Total</b>	0.51	0.49	1.00	0.51	0.49	1.00
<i>P Value</i>	<0.001			<0.001		
	<b>STEALS</b>			<b>TASK*</b>		
<b>1. Not True</b>	0.49	0.47	0.97	0.09	0.05	0.13
<b>2. Somewhat True</b>	0.02	0.01	0.02	0.27	0.22	0.48
<b>3. Certainly True</b>	0.01	0.00	0.01	0.16	0.23	0.39
<b>Total</b>	0.51	0.49	1.00	0.51	0.49	1.00
<i>P Value</i>	<0.001			<0.001		

**Table A.7.3.2 Footnote:** Table showing proportions for responses to SDQ items that make up externalising symptoms sub-scales, by sex. *P*-Values are for Pearson's Chi Squared statistics.

### A7.3.3 Pro-social Sub-Scales

	Pro-Social Scale (Proportion)		
	Boys	Girls	Total
<b>CONSIDERATE</b>			
<b>1. Not True</b>	0.02	0.01	0.03
<b>2. Somewhat True</b>	0.13	0.10	0.23
<b>3. Certainly True</b>	0.36	0.38	0.74
<b>Total</b>	0.51	0.49	1.00
<i>P Value</i>	<0.001		
<b>SHARES</b>			
<b>1. Not True</b>	0.02	0.01	0.03
<b>2. Somewhat True</b>	0.13	0.09	0.22
<b>3. Certainly True</b>	0.37	0.38	0.75
<b>Total</b>	0.52	0.48	1.00
<i>P Value</i>	<0.001		
<b>HELPFUL</b>			
<b>1. Not True</b>	0.01	0.01	0.02
<b>2. Somewhat True</b>	0.10	0.06	0.16
<b>3. Certainly True</b>	0.40	0.42	0.82
<b>Total</b>	0.51	0.49	1.00
<i>P Value</i>	<0.001		
<b>KIND</b>			
<b>1. Not True</b>	0.01	0.00	0.01
<b>2. Somewhat True</b>	0.08	0.04	0.11
<b>3. Certainly True</b>	0.43	0.44	0.88
<b>Total</b>	0.51	0.49	1.00
<i>P Value</i>	<0.001		
<b>VOLUNTEERS</b>			
<b>1. Not True</b>	0.02	0.01	0.03
<b>2. Somewhat True</b>	0.21	0.11	0.32
<b>3. Certainly True</b>	0.29	0.37	0.65
<b>Total</b>	0.51	0.49	1.00
<i>P Value</i>	<0.001		

**Table A7.3.3 Footnote:** Table showing proportions for responses to SDQ items that make up pro-social sub-scale, by sex. P-Values are for Pearson's Chi Squared statistics.

## A7.4 Geomin Rotation Loadings Factor Model Without Cross-Loadings

	<b>Factor 1</b>	<b>Factor 2</b>	<b>Factor 3</b>	<b>Factor 4</b>	<b>Factor 5</b>
<b>Emotional Symptom Scale</b>					
<i>Complains</i>	0.347*	0.274*	0.002	0.147*	0.034
<i>Unhappy</i>	0.640*	0.359*	-0.067*	0.013	0.084*
<i>Worried</i>	0.777*	0.102*	-0.016	0.064*	-0.014
<i>Anxiety</i>	0.625*	-0.061*	0.123*	-0.100*	-0.002
<i>Fear</i>	0.794*	0.01	0.054*	-0.003	-0.038
<b>Conduct Problems</b>					
<i>Anger</i>	0.142*	0.584*	0.134*	-0.039	-0.006
<i>Obedience</i>	-0.021	0.531*	0.125*	-0.296*	-0.106*
<i>Aggression</i>	0.115*	0.541*	0.04	-0.190*	0.207*
<i>Lies</i>	0.101*	0.648*	0.086*	-0.057	-0.059
<i>Steals</i>	0.013	0.801*	-0.04	-0.091	0.005
<b>Hyperactivity Scale</b>					
<i>Restless</i>	-0.113*	0.302*	0.563*	0.040*	0.309*
<i>Fidget</i>	-0.021	0.260*	0.552*	0.038*	0.327*
<i>Attention</i>	0.093*	0.077	0.737*	-0.102*	0.046*
<i>Task</i>	0.097*	-0.088*	0.708*	-0.327*	-0.051*
<b>Peer Problems</b>					
<i>Alone</i>	0.348*	-0.216*	0.002	-0.228*	0.484*
<i>Friend</i>	0.164*	0.022	0.025	-0.247*	0.398*
<i>Bullied</i>	0.427*	0.188*	0.035	0.017	0.301*
<i>Adults</i>	0.187*	0.015	0.007	-0.028	0.573*
<b>Pro-Social Scale</b>					
<i>Considerate</i>	0.017	-0.378*	0.033	0.511*	-0.036
<i>Shares</i>	-0.015	-0.228*	0.053*	0.569*	-0.074*
<i>Helpful</i>	0.033	-0.048	0.053*	0.771*	-0.02
<i>Kind</i>	0.034	-0.120*	-0.021	0.653*	-0.034
<i>Volunteers</i>	-0.002	-0.005	-0.114*	0.677*	0.223*

**Table**

**A7.4 Footnote:** Geomin Rotated Loadings for Five-Factor Model with items "impulse" and "liked" removed. \* Indicates  $p < 0.05$ . Loadings greater or equal to 0.4 are highlighted in dark grey. Greater or equal to 0.35 are highlighted in light grey



## A7.5 Model Fit Indices for Factorial Structure with Cross-Loadings Removed

<b>Model</b>	<b><math>\chi^2</math></b>	<b>df</b>	<b>CFI</b>	<b>TLI</b>	<b>RMSEA</b>	<b>SRMR</b>
7) Baseline five-factor Model with cross-loadings removed	1567.287	220	0.939	0.930	0.033	0.056
8) Five-factor model with correlations between unique variances and cross-loadings removed	1191.873	213	0.956	0.948	0.028	0.051
9) Baseline three-factor Model with cross-loadings removed	2337.829	227	0.905	0.894	0.040	0.070
10) Three-factor model with correlations between unique variances and cross-loadings removed	1497.401	220	0.942	0.934	0.032	0.057
11) Second-order two-factor model with cross-loadings removed	1624.597	223	0.937	0.928	0.033	0.058
12) Second-order two-factor model with correlations between unique variances and cross-loadings removed	1260.741	216	0.953	0.945	0.029	0.053

**Table A7.5 Footnote:** Sensitivity analysis showing model fit for competing models with cross-loading items removed (“impulse” and “liked”). Abbreviations:  $\chi^2$  – chi-squared; df – degrees of freedom; CFI – Comparative Fit Index; TLI – Tucker-Lewis Index; RMSEA – Root Mean Square Error of Approximation; SRMR – Standardized Root Mean Squared Residual.

## A7.6 Average Variance Explained for Factorial Structure with Cross-Loadings Removed

### A7.6.1 Sensitivity analysis: Second-order factor model

	<b>Ave</b>	<b>Correlation</b>			<b>Squared Correlation</b>		
		<b>Intern</b>	<b>Extern</b>	<b>Social</b>	<b>Intern</b>	<b>Extern</b>	<b>Social</b>
<b>Intern</b>	0.85						
<b>Extern</b>	0.79	0.76			0.57		
<b>Social</b>	0.49	-0.40	-0.68		0.16	0.47	

**Table A7.6.1 Footnote:** Sensitivity analysis showing AVE scores for second-order factors with cross-loading items removed (“impulse” and “liked”). AVE – Average Variance Explained. Shorthand name “intern” refers to internalising symptoms, “extern” to externalising symptoms, “social” to pro-social scale.

### A7.6.2 Sensitivity analysis: First-order five-factor model

	Correlation						Squared Correlation				
	Ave	Peer	Emotion	Conduct	Hyper	Social	Peer	Emotion	Conduct	Hyper	Social
<b>Peer</b>	0.45										
<b>Emotion</b>	0.52	0.85					0.72				
<b>Conduct</b>	0.54	0.60	0.64				0.36	0.40			
<b>Hyper</b>	0.41	0.59	0.63	0.79			0.35	0.40	0.62		
<b>Social</b>	0.49	-0.41	-0.34	-0.70	-0.51		0.17	0.12	0.49	0.26	

**Table A7.6.2 Footnote:** Sensitivity analysis showing AVE scores for first-order factors with cross-loading items removed (“impulse” and “liked”). AVE – Average Variance Explained. Shorthand name “Peer” refers to Peer Problems, “Emotion” to Emotional Symptoms, “Conduct” to Conduct Problems and “Hyper” to Hyperactivity.

### A7.7 Distribution of Sample by Diagnostic Criteria Age 14 and 17

Diagnosis	Depression diagnosis from age 13 to age 17				ADHD diagnosis age 14				Autism/Asperger’s diagnosis age 14			
	Full		Analytic*		Full		Analytic*		Full		Analytic *	
Sample	Freq.	Percent	Freq.	Percent	Freq.	Percent	Freq.	Percent	Freq.	Percent	Freq.	Percent
<b>No</b>	8,950	90.87	7,350	90.65	11,281	98.4	9,355	98.56	11,141	97.58	9,222	97.71
<b>Yes</b>	899	9.13	758	9.35	184	1.6	137	1.44	276	2.42	216	2.29
<b>Total</b>	9,849	100	8,108	100	11,465	100	9,492	100	11,417	100	9,438	100

**Table A7.7 Footnote:** \* Analytic sample are those with complete data on SDQ items at age 11, clinical diagnosis at age 14 for ADHD and autism/Asperger’s or 13 to 17 for depression, and selected covariates (sex, ethnicity and stratification characteristics). Full sample N=19,243.

## A7.8 Sensitivity Analysis of Predictive Validity for Factorial Structure with Cross-Loadings Removed

### A7.8.1 Sensitivity Analysis: Second-Order Factor Model

	<i>Mutually Adjusted</i>			<i>Minimally Adjusted</i>		
	Estimate	SE	P Value	Estimate	SE	P Value
<b>Depression</b>						
<i>Internalising</i>	0.37	0.09	<0.001	0.31	0.08	<0.001
<i>Externalising</i>	0.01	0.11	0.93	0.06	0.1	0.56
<i>Pro-social</i>	0.06	0.07	0.4	0.01	0.06	0.88
<b>ADHD</b>						
<i>Internalising</i>	-0.23	0.1	0.02	-0.15	0.09	0.09
<i>Externalising</i>	0.96	0.12	<0.001	0.84	0.11	<0.001
<i>Pro-social</i>	0.14	0.08	0.09	0.14	0.08	0.06
<b>Autism</b>						
<i>Internalising</i>	0.55	0.07	<0.001	0.58	0.07	<0.001
<i>Externalising</i>	0.04	0.11	0.68	-0.01	0.1	0.95
<i>Pro-social</i>	-0.25	0.08	0.001	-0.22	0.07	<0.001

**Table A7.8.1 Footnote:** Sensitivity analysis showing regression coefficients for probit regression between second-order factors with cross-loading items removed (“impulse” and “liked”) and clinical outcomes at age 14 (ADHD and Autism) and 13 to 17 (Depression).

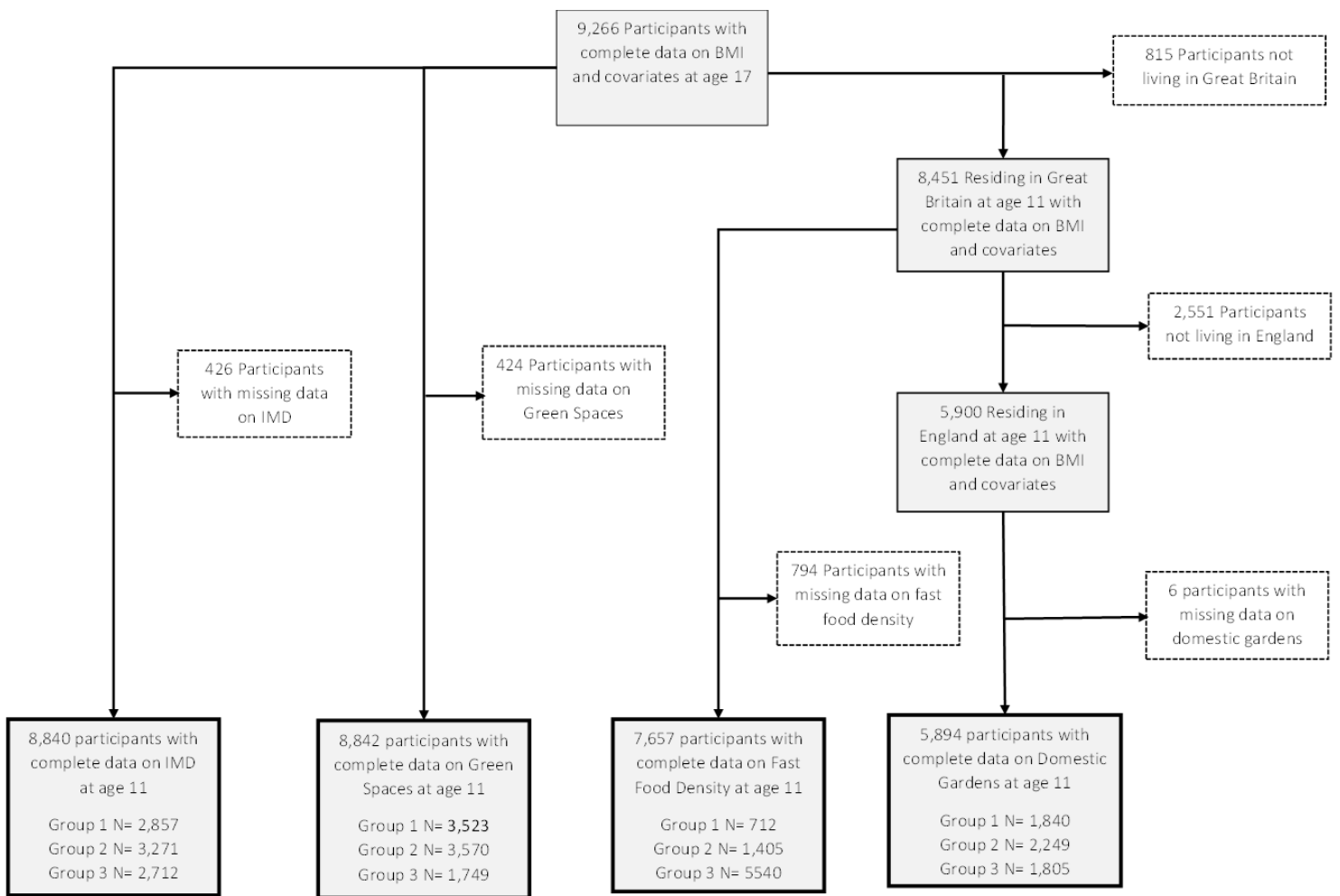
A7.8.2 Sensitivity analysis: First-order five-factor model

		<i>Mutually Adjusted</i>			<i>Minimally Adjusted</i>		
		Estimate	SE	P Value	Estimate	SE	P Value
<b>Depression</b>							
	<i>Emotion</i>	0.41	0.11	<0.001	0.35	0.09	<0.001
	<i>Peer</i>	-0.11	0.08	0.142	-0.09	0.07	0.225
	<i>Conduct</i>	0.38	0.16	0.016	0.31	0.13	0.019
	<i>Hyper</i>	-0.24	0.1	0.012	-0.16	0.08	0.044
	<i>Social</i>	0.14	0.09	0.124	0.08	0.08	0.303
<b>ADHD</b>							
	<i>Emotion</i>	-0.57	0.18	0.001	-1.08	0.36	0.003
	<i>Peer</i>	0.53	0.18	0.003	0.89	0.28	0.002
	<i>Conduct</i>	0.17	0.14	0.242	0.27	0.2	0.181
	<i>Hyper</i>	0.59	0.1	<0.001	0.76	0.14	<0.001
	<i>Social</i>	0.01	0.09	0.907	0.05	0.12	0.708
<b>Autism</b>							
	<i>Emotion</i>	-0.42	0.16	0.009	-0.41	0.17	0.016
	<i>Peer</i>	0.94	0.14	<0.001	0.91	0.22	<0.001
	<i>Conduct</i>	-0.45	0.18	0.011	-0.43	0.2	0.03
	<i>Hyper</i>	0.53	0.12	<0.001	0.5	0.16	0.001
	<i>Social</i>	-0.32	0.09	<0.001	-0.3	0.11	0.005

**Table A7.8.2 Footnote:** Sensitivity analysis showing regression coefficients for probit regression between first-order factors with cross-loading items removed (“impulse” and “liked”) and clinical outcomes at age 14 (ADHD and Autism) and 13 to 17 (Depression).

## A8. Chapter 8 Appendix

### A8.1 Analytic Sample for Moderated Body Mass Index Analysis



## A8.2 Sensitivity Analysis: Mediation of Socioeconomic Position and Fat-Free Mass Index with Fat Mas Index Treated as a Mediator

<i>Model</i>	<i>Estimate</i>	<i>Lower CI</i>	<i>Upper CI</i>	<i>P Value</i>
<b>MODEL 1 – INTERNALISING SYMPTOMS</b>				
<i>Total</i>	-0.04	-0.076	-0.005	0.027
<i>Total Indirect</i>	-0.063	-0.097	-0.029	<0.001
<i>FMI Indirect</i>	-0.038	-0.064	-0.012	0.004
<i>Internalising Indirect</i>	-0.025	-0.041	-0.009	0.002
<i>Direct</i>	0.023	-0.001	0.047	0.063
<b>MODEL 2 – EXTERNALISING SYMPTOMS</b>				
<i>Total</i>	-0.04	-0.076	-0.005	0.027
<i>Total Indirect</i>	-0.071	-0.108	-0.034	<0.001
<i>FMI Indirect</i>	-0.038	-0.064	-0.012	0.004
<i>Externalising Indirect</i>	-0.033	-0.053	-0.013	0.001
<i>Direct</i>	0.031	0.008	0.053	0.007
<b>MODEL 3 – INTERNALISING &amp; EXTERNALISING SYMPTOMS</b>				
<i>Total</i>	-0.04	-0.076	-0.005	0.027
<i>Total Indirect</i>	-0.073	-0.111	-0.034	<0.001
<i>FMI Indirect</i>	-0.038	-0.064	-0.012	0.004
<i>Internalising Indirect</i>	-0.013	-0.049	0.024	0.494
<i>Externalising Indirect</i>	-0.022	-0.065	0.022	0.323
<i>Direct</i>	0.032	0.01	0.055	0.005

**Table A8.3 Footnotes:** Sensitivity analysis is for mediation models 1, 2 and 3 with fat-free mass index as the outcome, and with additional inclusion of fat mass index as mediator. Total, indirect and direct effect estimates for mediation models 1, 2 and 3. All models are adjusted for sex, age at sweep 7, ethnicity and stratification characteristics.

### A8.3 Sensitivity Analysis: Mediation Results with Robust Maximum Likelihood Estimation

Model	Estimate	Lower CI	Upper CI	p value	Fit Statistics:					
					RMSEA	CFI	TFI	SRMR	X <sup>2</sup>	df
<b>FMI</b>										
Total	-0.079	-0.116	-0.043	<0.001	0.024	0.921	0.907	0.025	2965.63	461
Total Indirect	-0.034	-0.047	-0.022	<0.001	-	-	-	-	-	-
Specific Indirect										
- Internalising	-0.03	-0.048	-0.011	0.002						
Specific Indirect										
- Externalising	-0.004	-0.025	0.017	0.681						
Direct	-0.045	-0.083	-0.007	0.019						
<b>FFMI</b>										
Total	-0.022	-0.046	0.001	0.056	0.024	0.93	0.918	0.026	2967.10	461
Total Indirect	-0.018	-0.026	-0.011	<0.001	-	-	-	-	-	-
Specific Indirect										
- Internalising	-0.007	-0.021	0.006	0.277						
Specific Indirect										
- Externalising	-0.011	-0.027	0.005	0.176						
Direct	-0.004	-0.028	0.02	0.732						
<b>FFMI adjusted for FMI</b>										
Total	0.014	-0.002	0.031	0.082	0.024	0.934	0.924	0.028	3108.88	495
Total Indirect	-0.002	-0.007	0.003	0.345	-	-	-	-	-	-
Specific Indirect										
- Internalising	0.006	-0.003	0.014	0.178						
Specific Indirect										
- Externalising	-0.008	-0.018	0.002	0.102						
Direct	0.017	0.001	0.033	0.042						
<b>FM: FFM</b>										
Total	-0.069	-0.099	-0.039	<0.001	0.024	0.926	0.912	0.026	2983.90	461
Total Indirect	-0.023	-0.033	-0.014	<0.001	-	-	-	-	-	-
Specific Indirect										
- Internalising	-0.025	-0.041	-0.009	0.002						
Specific Indirect										
- Externalising	0.002	-0.015	0.018	0.815						
Direct	-0.046	-0.077	-0.015	0.004						
<b>BMI</b>										
Total	-0.072	-0.106	-0.037	<0.001	0.024	0.921	0.907	0.025	2985.28	461
Total Indirect	-0.031	-0.043	-0.02	<0.001	-	-	-	-	-	-
Specific Indirect										
- Internalising	-0.027	-0.046	-0.008	0.005						
Specific Indirect										
- Externalising	-0.004	-0.026	0.017	0.69						
Direct	-0.04	-0.075	-0.005	0.025						

**Footnote:** Mediation model 3 (internalising and externalising included together) for factors estimated using maximum likelihood robust estimation. Sample size for body composition analysis is 9,526 and for BMI is 9,535.

#### A8.4 Model Fit for Standardised Factors

Chi Squared	DF	RMSEA	CFI	TLI	SRMR
1944.289	155	0.030	0.939	0.925	0.033

**Table A8.2 Footnote:** Fit indices for standardised factors loading, by setting the variances of both second-order factors equal to one and freeing up the first-order factor loadings. Abbreviations: DF – Degrees of Freedom, CFI – Comparative Fit Index, TLI – Tucker-Lewis Index, RMSEA – Root Mean Square Error of Approximation, SRMR – standardized root mean squared residual.



# Bibliography

1. Whitehead, M., *A typology of actions to tackle social inequalities in health*. J Epidemiol Community Health, 2007. **61**(6): p. 473-8.
2. James, P.T., et al., *The Worldwide Obesity Epidemic*. Obesity Research, 2001. **9**(S11): p. 228S-233S.
3. Hruby, A. and F.B. Hu, *The Epidemiology of Obesity: A Big Picture*. Pharmacoeconomics, 2015. **33**(7): p. 673-689.
4. Abdelaal, M., C.W. le Roux, and N.G. Docherty, *Morbidity and mortality associated with obesity*. Annals of Translational Medicine, 2017. **5**(7).
5. Houston, D.K., et al., *Overweight and obesity over the adult life course and incident mobility limitation in older adults: the health, aging and body composition study*. Am J Epidemiol, 2009. **169**(8): p. 927-36.
6. de Wit, L., et al., *Depression and obesity: a meta-analysis of community-based studies*. Psychiatry Res, 2010. **178**(2): p. 230-5.
7. Carey, M., et al., *Prevalence of comorbid depression and obesity in general practice: a cross-sectional survey*. British Journal of General Practice, 2014. **64**(620): p. E122-E127.
8. Allison, D.B., et al., *Obesity Among Those with Mental Disorders A National Institute of Mental Health Meeting Report*. American Journal of Preventive Medicine, 2009. **36**(4): p. 341-350.
9. Simmonds, M., et al., *Predicting adult obesity from childhood obesity: a systematic review and meta-analysis*. Obes Rev, 2016. **17**(2): p. 95-107.
10. NHS. *Statistics on Obesity, Physical Activity and Diet, England, 2019*. Lifestyles Team: NHS Digital 2019; Available from: <https://digital.nhs.uk/data-and-information/publications/statistical/statistics-on-obesity-physical-activity-and-diet/statistics-on-obesity-physical-activity-and-diet-england-2019>.
11. Public Health England. *Health matters: obesity and the food environment*. Public Health England 2017; Available from: <https://www.gov.uk/government/publications/health-matters-obesity-and-the-food-environment/health-matters-obesity-and-the-food-environment--2>.
12. Butland, B., et al., *Tackling Obesities: Future Choices – Project Report*, G.O.f. Science, Editor. 2007: gov.uk.
13. HM Government, *Childhood Obesity: A Plan for Action* P.M.s.O. Department of Health and Social Care, 10 Downing Street, HM Treasury, and Cabinet Office, Editor. 2016: gov.uk.
14. POST, *Childhood Obesity*, POST, Editor. 2021, Parliamentary Office of Science and Technology, UK Parliament: Westminster, London.
15. Viner, R.M., et al., *Life course epidemiology: recognising the importance of adolescence*. J Epidemiol Community Health, 2015. **69**(8): p. 719-20.
16. Finucane, M.M., et al., *National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants*. Lancet, 2011. **377**(9765): p. 557-567.
17. Di Cesare, M., et al., *Trends in adult body-mass index in 200 countries from 1975 to 2014: a pooled analysis of 1698 population-based measurement studies with 19.2 million participants*. Lancet, 2016. **387**(10026): p. 1377-1396.
18. Ezzati, M., et al., *Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults*. Lancet, 2017. **390**(10113): p. 2627-2642.
19. Zaninotto, P. and C. Lassale, *Socioeconomic trajectories of body mass index and waist circumference: results from the English Longitudinal Study of Ageing*. BMJ Open, 2019. **9**(4): p. e025309.

20. Low, S., M.C. Chin, and M. Deurenberg-Yap, *Review on Epidemic of Obesity*. Annals Academy of Medicine Singapore, 2009. **38**(1): p. 57-65.
21. Baker, C., *Briefing Paper: Obesity Statistics* H.o.C. Library, Editor. 2021: commonslibrary.parliament.uk.
22. NHS, *Health Survey for England 2017: Adult and child overweight and obesity*, in *NHS Digital 2018*: NHS Digital
23. Stamatakis, E., et al., *Time trends in childhood and adolescent obesity in England from 1995 to 2007 and projections of prevalence to 2015*. J Epidemiol Community Health, 2010. **64**(2): p. 167-74.
24. Johnson, W., et al., *How Has the Age-Related Process of Overweight or Obesity Development Changed over Time? Co-ordinated Analyses of Individual Participant Data from Five United Kingdom Birth Cohorts*. PLoS Medicine, 2015. **12**(5): p. e1001828.
25. El-Sayed, A.M., P. Scarborough, and S. Galea, *Unevenly distributed: a systematic review of the health literature about socioeconomic inequalities in adult obesity in the United Kingdom*. BMC Public Health, 2012. **12**: p. 18.
26. El-Sayed, A.M., P. Scarborough, and S. Galea, *Socioeconomic inequalities in childhood obesity in the United Kingdom: a systematic review of the literature*. Obes Facts, 2012. **5**(5): p. 671-92.
27. O'Dowd, A., *Fight to tackle unhealthy lifestyles has widened gap in health inequalities*. British Medical Journal, 2012. **345**.
28. Bann, D., et al., *Socioeconomic inequalities in childhood and adolescent body-mass index, weight, and height from 1953 to 2015: an analysis of four longitudinal, observational, British birth cohort studies*. Lancet Public Health, 2018. **3**(4): p. E194-E203.
29. Bann, D., et al., *Socioeconomic Inequalities in Body Mass Index across Adulthood: Coordinated Analyses of Individual Participant Data from Three British Birth Cohort Studies Initiated in 1946, 1958 and 1970*. PLoS Med, 2017. **14**(1): p. e1002214.
30. Egger, G. and B. Swinburn, *An "ecological" approach to the obesity pandemic*. British Medical Journal, 1997. **315**(7106): p. 477-480.
31. Smith, D.M.a.C., S., *Obese Cities: How Our Environment Shapes Overweight*. Geography Compass 2009. **3**(1): p. 518-535.
32. Ulijaszek, S.J. and H. Lofink, *Obesity in biocultural perspective*. Annual Review of Anthropology, 2006. **35**: p. 337-360.
33. Swinburn, B., G. Egger, and F. Raza, *Dissecting obesogenic environments: the development and application of a framework for identifying and prioritizing environmental interventions for obesity*. Prev Med, 1999. **29**(6 Pt 1): p. 563-70.
34. Swinburn, B. and G. Egger, *Preventive strategies against weight gain and obesity*. Obes Rev, 2002. **3**(4): p. 289-301.
35. Collishaw, S., *Annual research review: Secular trends in child and adolescent mental health*. J Child Psychol Psychiatry, 2015. **56**(3): p. 370-93.
36. Bor, W., et al., *Are child and adolescent mental health problems increasing in the 21st century? A systematic review*. Aust N Z J Psychiatry, 2014. **48**(7): p. 606-16.
37. Collishaw, S., et al., *Trends in adolescent emotional problems in England: a comparison of two national cohorts twenty years apart*. J Child Psychol Psychiatry, 2010. **51**(8): p. 885-94.
38. Collishaw, S., et al., *Time trends in adolescent mental health*. J Child Psychol Psychiatry, 2004. **45**(8): p. 1350-62.
39. Gaysina, D., et al., *Symptoms of depression and anxiety, and change in body mass index from adolescence to adulthood: results from a British birth cohort*. Psychol Med, 2011. **41**(1): p. 175-84.
40. Platt, L., *Millennium Cohort Study Age 11 Survey Initial Findings*. 2014: London: Centre for Longitudinal Studies.

41. Griffiths, L.J., C. Dezateux, and A. Hill, *Is obesity associated with emotional and behavioural problems in children? Findings from the Millennium Cohort Study*. *International Journal of Pediatric Obesity*, 2011. **6**(2-2): p. E423-E432.
42. Anderson, S., et al., *Self-regulation and household routines at age three and obesity at age eleven: longitudinal analysis of the UK Millennium Cohort Study*. *International Journal of Obesity*, 2017. **41**: p. 1459–1466.
43. van den Berg, M., et al., *Health benefits of green spaces in the living environment: A systematic review of epidemiological studies*. *Urban Forestry & Urban Greening*, 2015. **14**(4): p. 806-816.
44. Lee, A.C. and R. Maheswaran, *The health benefits of urban green spaces: a review of the evidence*. *J Public Health (Oxf)*, 2011. **33**(2): p. 212-22.
45. Callaghan, A., et al., *The impact of green spaces on mental health in urban settings: a scoping review*. *Journal of Mental Health*, 2021. **30**(2): p. 179-193.
46. Zhang, Y.J., et al., *The Association between Green Space and Adolescents' Mental Well-Being: A Systematic Review*. *International Journal of Environmental Research and Public Health*, 2020. **17**(18).
47. Dadvand, P., et al., *Green spaces and General Health: Roles of mental health status, social support, and physical activity*. *Environment International*, 2016. **91**: p. 161-167.
48. Akpinar, A., *How is quality of urban green spaces associated with physical activity and health?* *Urban Forestry & Urban Greening*, 2016. **16**: p. 76-83.
49. Burgoine, T., et al., *Associations between exposure to takeaway food outlets, takeaway food consumption, and body weight in Cambridgeshire, UK: population based, cross sectional study*. *Bmj-British Medical Journal*, 2014. **348**.
50. Bremner, J.D., et al., *Diet, Stress and Mental Health*. *Nutrients*, 2020. **12**(8).
51. Reiss, F., *Socioeconomic inequalities and mental health problems in children and adolescents: a systematic review*. *Soc Sci Med*, 2013. **90**: p. 24-31.
52. Noonan, R.J., *The effect of childhood deprivation on weight status and mental health in childhood and adolescence: longitudinal findings from the Millennium Cohort Study*. *J Public Health (Oxf)*, 2019. **41**(3): p. 456-461.
53. Green, M.A., *Do we need to think beyond BMI for estimating population-level health risks?* *Journal of Public Health*, 2016. **38**(1): p. 192-193.
54. Reilly, J.J., et al., *Determining the worldwide prevalence of obesity*. *Lancet*, 2018. **391**(10132): p. 1773-1774.
55. Okorodudu, D.O., et al., *Diagnostic performance of body mass index to identify obesity as defined by body adiposity: a systematic review and meta-analysis*. *Int J Obes (Lond)*, 2010. **34**(5): p. 791-9.
56. van den Berg, G., et al., *BMI may underestimate the socioeconomic gradient in true obesity*. *Pediatric Obesity*, 2012. **8**(3): p. e37-40.
57. Caleyachetty, R., et al., *Ethnicity-specific BMI cutoffs for obesity based on type 2 diabetes risk in England: a population-based cohort study*. *Lancet Diabetes Endocrinol*, 2021. **9**(7): p. 419-426.
58. Buckinx, F., et al., *Pitfalls in the measurement of muscle mass: a need for a reference standard*. *J Cachexia Sarcopenia Muscle*, 2018. **9**(2): p. 269-278.
59. Muller, M.J., et al., *Beyond BMI: Conceptual Issues Related to Overweight and Obese Patients*. *Obes Facts*, 2016. **9**(3): p. 193-205.
60. Prado, C.M., et al., *A population-based approach to define body-composition phenotypes*. *Am J Clin Nutr*, 2014. **99**(6): p. 1369-77.
61. Franco, L.P., C.C. Morais, and C. Cominetti, *Normal-weight obesity syndrome: diagnosis, prevalence, and clinical implications*. *Nutr Rev*, 2016. **74**(9): p. 558-70.
62. Vecchie, A., et al., *Obesity phenotypes and their paradoxical association with cardiovascular diseases*. *European Journal of Internal Medicine*, 2018. **48**: p. 6-17.

63. Wells, J.C. and M.S. Fewtrell, *Measuring body composition*. Arch Dis Child, 2006. **91**(7): p. 612-7.
64. Andreoli, A., et al., *Body composition assessment by dual-energy X-ray absorptiometry (DXA)*. Radiol Med, 2009. **114**(2): p. 286-300.
65. Duren, D.L., et al., *Body composition methods: comparisons and interpretation*. J Diabetes Sci Technol, 2008. **2**(6): p. 1139-46.
66. Achamrah, N., et al., *Comparison of body composition assessment by DXA and BIA according to the body mass index: A retrospective study on 3655 measures*. PLoS One, 2018. **13**(7): p. e0200465.
67. Beaudart, C., et al., *Equation models developed with bioelectric impedance analysis tools to assess muscle mass: A systematic review*. Clin Nutr ESPEN, 2020. **35**: p. 47-62.
68. Scafoglieri, A. and J.P. Clarys, *Dual energy X-ray absorptiometry: gold standard for muscle mass?* J Cachexia Sarcopenia Muscle, 2018. **9**(4): p. 786-787.
69. Janmahasatian, S., et al., *Quantification of lean bodyweight*. Clin Pharmacokinet, 2005. **44**(10): p. 1051-65.
70. Hansen, R.D., et al., *Determination of skeletal muscle and fat-free mass by nuclear and dual-energy x-ray absorptiometry methods in men and women aged 51-84 y (1-3)*. Am J Clin Nutr, 1999. **70**(2): p. 228-33.
71. Liu, P., et al., *The utility of fat mass index vs. body mass index and percentage of body fat in the screening of metabolic syndrome*. BMC Public Health, 2013. **13**: p. 629.
72. Wells, J.C., *Toward body composition reference data for infants, children, and adolescents*. Adv Nutr, 2014. **5**(3): p. 320S-9S.
73. Kelly, T.L., K.E. Wilson, and S.B. Heymsfield, *Dual Energy X-Ray Absorptiometry Body Composition Reference Values from NHANES*. Plos One, 2009. **4**(9).
74. Eto, C., et al., *Validity of the body mass index and fat mass index as an indicator of obesity in children aged 3-5 year*. J Physiol Anthropol Appl Human Sci, 2004. **23**(1): p. 25-30.
75. Cole, T.J. and T. Lobstein, *Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity*. Pediatr Obes, 2012. **7**(4): p. 284-94.
76. Weber, D.R., et al., *Fat and lean BMI reference curves in children and adolescents and their utility in identifying excess adiposity compared with BMI and percentage body fat*. American Journal of Clinical Nutrition, 2013. **98**(1): p. 49-56.
77. Dangardt, F., et al., *Association between fat mass through adolescence and arterial stiffness: a population-based study from The Avon Longitudinal Study of Parents and Children*. Lancet Child & Adolescent Health, 2019. **3**(7): p. 474-481.
78. Alpizar, M., et al., *Fat Mass Index (FMI) as a Trustworthy Overweight and Obesity Marker in Mexican Pediatric Population*. Children-Basel, 2020. **7**(3).
79. Srikanthan, P., T.B. Horwich, and C.H. Tseng, *Relation of Muscle Mass and Fat Mass to Cardiovascular Disease Mortality*. American Journal of Cardiology, 2016. **117**(8): p. 1355-1360.
80. Okosun, I.S., J.P. Seale, and R. Lyn, *Commingling effect of gynoid and android fat patterns on cardiometabolic dysregulation in normal weight American adults*. Nutrition & Diabetes, 2015. **5**.
81. Kang, S.M., et al., *Android Fat Depot Is More Closely Associated with Metabolic Syndrome than Abdominal Visceral Fat in Elderly People*. Plos One, 2011. **6**(11).
82. Vasan, S.K., et al., *Comparison of regional fat measurements by dual-energy X-ray absorptiometry and conventional anthropometry and their association with markers of diabetes and cardiovascular disease risk*. International Journal of Obesity, 2018. **42**(4): p. 850-857.
83. Samsell, L., et al., *Importance of android/gynoid fat ratio in predicting metabolic and cardiovascular disease risk in normal weight as well as overweight and obese children*. J Obes, 2014. **2014**: p. 846578.

84. Coutinho, T., et al., *Central obesity and survival in subjects with coronary artery disease: a systematic review of the literature and collaborative analysis with individual subject data*. J Am Coll Cardiol, 2011. **57**(19): p. 1877-86.
85. Cooper, R., et al., *Body Mass Index From Age 15 Years Onwards and Muscle Mass, Strength, and Quality in Early Old Age: Findings From the MRC National Survey of Health and Development*. Journals of Gerontology Series a-Biological Sciences and Medical Sciences, 2014. **69**(10): p. 1253-1259.
86. Bell, J.A., et al., *Body muscle gain and markers of cardiovascular disease susceptibility in young adulthood: A cohort study*. PLoS Med, 2021. **18**(9): p. e1003751.
87. Skelton, D.A. and A. Mavroei, *How do muscle and bone strengthening and balance activities (MBSBA) vary across the life course, and are there particular ages where MBSBA are most important?* J Frailty Sarcopenia Falls, 2018. **3**(2): p. 74-84.
88. Peeters, G., et al., *A life-course perspective on physical functioning in women*. Bull World Health Organ, 2013. **91**(9): p. 661-70.
89. Takamura, T., et al., *Weight-adjusted lean body mass and calf circumference are protective against obesity-associated insulin resistance and metabolic abnormalities*. Heliyon, 2017. **3**(7): p. e00347.
90. Srikanthan, P. and A.S. Karlamangla, *Relative Muscle Mass Is Inversely Associated with Insulin Resistance and Prediabetes. Findings from The Third National Health and Nutrition Examination Survey*. Journal of Clinical Endocrinology & Metabolism, 2011. **96**(9): p. 2898-2903.
91. Kuh, D., et al., *Life course epidemiology*. J Epidemiol Community Health, 2003. **57**(10): p. 778-83.
92. Kuh, D. and Y.B. Shlomo, *A Life Course Approach to Chronic Disease Epidemiology*. 2004, Oxford: OUP Oxford.
93. Jacob, C.M., et al., *The importance of a life-course approach to health: Chronic disease risk from preconception through adolescence and adulthood: White paper*. . 2017
94. Schmidt, K.L., et al., *Society to cell: How child poverty gets "Under the Skin" to influence child development and lifelong health*. Developmental Review, 2021. **61**.
95. Riley, J.C., *Sickness, recovery and death: a history and forecast of ill-health*. 1989, Basingstoke: Palgrave, Macmillan.
96. Ben-Shlomo, Y. and D. Kuh, *A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives*. Int J Epidemiol, 2002. **31**(2): p. 285-93.
97. Kuh, D., et al., *A Life Course Approach to Healthy Ageing*. 2013, Oxford: Oxford University Press.
98. Marmot, M. and R. Wilkinson, *Social Determinants of Health*. 2005, OUP Oxford: Oxford University Press.
99. Wilkinson, R. and M. Marmot, *Social Determinants of Health: The Solid Facts*, ed. W.H.C. Project. 2003: WHO.
100. Braveman, P. and L. Gottlieb, *The Social Determinants of Health: It's Time to Consider the Causes of the Causes*. Public Health Reports, 2014. **129**: p. 19-31.
101. WHO, *A Conceptual Framework for Action on the Social Determinants of Health*, in *Discussion Paper Series on Social Determinants of Health*, W.H. Organisation, Editor. 2010: Geneva.
102. Gauvin, L., et al., *Association between neighborhood active living potential and walking*. American Journal of Epidemiology, 2008. **167**(8): p. 944-953.
103. Crowley, S.L. and K.W. Merrell, *Convergent and discriminant validity of the internalizing symptoms scale for children*. Journal of Psychoeducational Assessment, 2000. **18**(1): p. 4-16.
104. Van Cappellen, P., et al., *Positive affective processes underlie positive health behaviour change*. Psychology & Health, 2018. **33**(1): p. 77-97.

105. Stice, E., et al., *Psychological and behavioral risk factors for obesity onset in adolescent girls: a prospective study*. J Consult Clin Psychol, 2005. **73**(2): p. 195-202.
106. Puder, J.J. and S. Munsch, *Psychological correlates of childhood obesity*. International Journal of Obesity, 2010. **34**: p. S37-S43.
107. Ferrer, R.A., et al., *The role of incidental affective states in appetitive risk behavior: A meta-analysis*. Health Psychol, 2020. **39**(12): p. 1109-1124.
108. Kirk, S.F., T.L. Penney, and T.L. McHugh, *Characterizing the obesogenic environment: the state of the evidence with directions for future research*. Obes Rev, 2010. **11**(2): p. 109-17.
109. Jones, A., et al., *Tackling Obesities: Future Choices Obesogenic Environments—Evidence Review*. 2007, Office of Science and Innovation:: London, UK.
110. Galobardes, B., et al., *Indicators of socioeconomic position (part 1)*. J Epidemiol Community Health, 2006. **60**(1): p. 7-12.
111. Sobal, J. and A.J. Stunkard, *Socioeconomic status and obesity: a review of the literature*. Psychol Bull, 1989. **105**(2): p. 260-75.
112. McLaren, L., *Socioeconomic status and obesity*. Epidemiol Rev, 2007. **29**: p. 29-48.
113. Dinsa, G.D., et al., *Obesity and socioeconomic status in developing countries: a systematic review*. Obes Rev, 2012. **13**(11): p. 1067-79.
114. Monteiro, C.A., et al., *Socioeconomic status and obesity in adult populations of developing countries: a review*. Bull World Health Organ, 2004. **82**(12): p. 940-6.
115. Pudrovska, T., et al., *Gender and Reinforcing Associations between Socioeconomic Disadvantage and Body Mass over the Life Course*. Journal of Health and Social Behavior, 2014. **55**(3): p. 283-301.
116. Parsons, T.J., et al., *Childhood predictors of adult obesity: a systematic review*. International Journal of Obesity, 1999. **23**: p. S1-S107.
117. Senese, L.C., et al., *Associations Between Childhood Socioeconomic Position and Adulthood Obesity*. Epidemiologic Reviews, 2009. **31**(1): p. 21-51.
118. Goisis, A., A. Sacker, and Y. Kelly, *Why are poorer children at higher risk of obesity and overweight? A UK cohort study*. European Journal of Public Health, 2016. **26**(1): p. 7-13.
119. Sun, S.S., et al., *Secular trends in body composition for children and young adults: the Fels Longitudinal Study*. Am J Hum Biol, 2012. **24**(4): p. 506-14.
120. Johnson, W., et al., *Secular trends in the fat and fat-free components of body mass index in children aged 8-18 years born 1958-1995*. Annals of Human Biology, 2013. **40**(1): p. 107-110.
121. Sandercock, G.R.H. and D.D. Cohen, *Temporal trends in muscular fitness of English 10-year-olds 1998-2014: An allometric approach*. J Sci Med Sport, 2018.
122. Bann, D., et al., *Birth weight and growth from infancy to late adolescence in relation to fat and lean mass in early old age: findings from the MRC National Survey of Health and Development*. International Journal of Obesity, 2014. **38**(1): p. 69-75.
123. Yliharsila, H., et al., *Body mass index during childhood and adult body composition in men and women aged 56-70 y*. Am J Clin Nutr, 2008. **87**(6): p. 1769-75.
124. Suglia, S.F., et al., *Social and behavioral risk factors for obesity in early childhood*. J Dev Behav Pediatr, 2013. **34**(8): p. 549-56.
125. Ogundele, M.O., *Behavioural and emotional disorders in childhood: A brief overview for paediatricians*. World J Clin Pediatr, 2018. **7**(1): p. 9-26.
126. Lorant, V., et al., *Socioeconomic inequalities in depression: a meta-analysis*. Am J Epidemiol, 2003. **157**(2): p. 98-112.
127. Lemstra, M., et al., *A systematic review of depressed mood and anxiety by SES in youth aged 10-15 years*. Can J Public Health, 2008. **99**(2): p. 125-9.
128. Freeman, A., et al., *The role of socio-economic status in depression: results from the COURAGE (aging survey in Europe)*. BMC Public Health, 2016. **16**(1): p. 1098.
129. Lorant, V., et al., *Depression and socio-economic risk factors: 7-year longitudinal population study*. Br J Psychiatry, 2007. **190**: p. 293-8.

130. Flouri, E., E. Midouhas, and H. Joshi, *Family and neighbourhood risk and children's problem behaviour: The moderating role of intelligence*. *Intelligence*, 2015. **53**: p. 33-42.
131. Flouri, E. and E. Midouhas, *School composition, family poverty and child behaviour*. *Social Psychiatry and Psychiatric Epidemiology*, 2016. **51**(6): p. 817-826.
132. Midouhas, E., *School poverty effects on trajectories of child behaviour: Do they depend on gender and ethnicity?* *Health & Place*, 2017. **46**: p. 281-292.
133. Gundersen, C., et al., *Linking psychosocial stressors and childhood obesity*. *Obes Rev*, 2011. **12**(5): p. e54-63.
134. Incedon, E., M. Wake, and M. Hay, *Psychological predictors of adiposity: systematic review of longitudinal studies*. *Int J Pediatr Obes*, 2011. **6**(2-2): p. e1-11.
135. Graziano, P.A., S.D. Calkins, and S.P. Keane, *Toddler self-regulation skills predict risk for pediatric obesity*. *Int J Obes (Lond)*, 2010. **34**(4): p. 633-41.
136. Tiffin, P.A., et al., *Modelling the relationship between obesity and mental health in children and adolescents: findings from the Health Survey for England 2007*. *Child Adolesc Psychiatry Ment Health*, 2011. **5**: p. 31.
137. Pine, D.S., et al., *The association between childhood depression and adulthood body mass index*. *Pediatrics*, 2001. **107**(5): p. 1049-1056.
138. Hasler, G., et al., *Depressive symptoms during childhood and adult obesity: the Zurich Cohort Study*. *Mol Psychiatry*, 2005. **10**(9): p. 842-50.
139. Anderson, S.E., et al., *Externalizing behavior in early childhood and body mass index from age 2 to 12 years: longitudinal analyses of a prospective cohort study*. *BMC Pediatr*, 2010. **10**: p. 49.
140. Beydoun, M.A., et al., *Role of depressive symptoms in explaining socioeconomic status disparities in dietary quality and central adiposity among US adults: A structural equation modeling approach*. *American Journal of Clinical Nutrition*, 2009. **90**(4): p. 1084-1095.
141. Moon, J.H., M.H. Kong, and H.J. Kim, *Low Muscle Mass and Depressed Mood in Korean Adolescents: a Cross-Sectional Analysis of the Fourth and Fifth Korea National Health and Nutrition Examination Surveys*. *Journal of Korean medical science*, 2018. **33**(50): p. e320.
142. Goossens, L., et al., *Loss of control over eating in overweight youngsters: the role of anxiety, depression and emotional eating*. *Eur Eat Disord Rev*, 2009. **17**(1): p. 68-78.
143. Haedt-Matt, A.A. and P.K. Keel, *Revisiting the affect regulation model of binge eating: a meta-analysis of studies using ecological momentary assessment*. *Psychol Bull*, 2011. **137**(4): p. 660-681.
144. Hill, D.C., et al., *Stress and eating behaviors in children and adolescents: Systematic review and meta-analysis*. *Appetite*, 2018. **123**: p. 14-22.
145. Bratanova, B., et al., *Poverty, inequality, and increased consumption of high calorie food: Experimental evidence for a causal link*. *Appetite*, 2016. **100**: p. 162-71.
146. Spinosa, J., et al., *From Socioeconomic Disadvantage to Obesity: The Mediating Role of Psychological Distress and Emotional Eating*. *Obesity (Silver Spring)*, 2019. **27**(4): p. 559-564.
147. Moore, C.J. and S.A. Cunningham, *Social position, psychological stress, and obesity: a systematic review*. *J Acad Nutr Diet*, 2012. **112**(4): p. 518-26.
148. Suglia, S.F., et al., *Why the Neighborhood Social Environment Is Critical in Obesity Prevention*. *J Urban Health*, 2016. **93**(1): p. 206-12.
149. Inagami, S., D.A. Cohen, and B.K. Finch, *Non-residential neighborhood exposures suppress neighborhood effects on self-rated health*. *Social Science & Medicine*, 2007. **65**(8): p. 1779-1791.
150. Cohen, D.A., *Obesity and the built environment: changes in environmental cues cause energy imbalances*. *Int J Obes (Lond)*, 2008. **32 Suppl 7**: p. S137-42.
151. Maguire, E.R., T. Burgoine, and P. Monsivais, *Area deprivation and the food environment over time: A repeated cross-sectional study on takeaway outlet density and supermarket presence in Norfolk, UK, 1990-2008*. *Health & Place*, 2015. **33**: p. 142-147.

152. Cetateanu, A. and A. Jones, *Understanding the relationship between food environments, deprivation and childhood overweight and obesity: Evidence from a cross sectional England-wide study*. Health & Place, 2014. **27**: p. 68-76.
153. Cummins, S.C., L. McKay, and S. MacIntyre, *McDonald's restaurants and neighborhood deprivation in Scotland and England*. Am J Prev Med, 2005. **29**(4): p. 308-10.
154. Hurvitz, P.M., et al., *Arterial roads and area socioeconomic status are predictors of fast food restaurant density in King County, WA*. International Journal of Behavioral Nutrition and Physical Activity, 2009. **6**.
155. Thornton, L.E., K.E. Lamb, and K. Ball, *Fast food restaurant locations according to socioeconomic disadvantage, urban-regional locality, and schools within Victoria, Australia*. SSM Popul Health, 2016. **2**: p. 1-9.
156. Adams, J., E. Ganiti, and M. White, *Socio-economic differences in outdoor food advertising in a city in Northern England*. Public Health Nutr, 2011. **14**(6): p. 945-50.
157. Hoffmann, E., H. Barros, and A.I. Ribeiro, *Socioeconomic Inequalities in Green Space Quality and Accessibility-Evidence from a Southern European City*. Int J Environ Res Public Health, 2017. **14**(8).
158. Wang, Q. and Z.L. Lan, *Park green green spaces, public health and social inequalities: Understanding the interrelationships for policy implications*. Land Use Policy, 2019. **83**: p. 66-74.
159. Gordon-Larsen, P., et al., *Inequality in the built environment underlies key health disparities in physical activity and obesity*. Pediatrics, 2006. **117**(2): p. 417-24.
160. Public Health England, *Obesity and the environment: Density of fast food outlets*. 2018: Public Health England publication gateway reference 2018064.
161. Navalpotro, L., et al., *Area-based socioeconomic environment, obesity risk behaviours, area facilities and childhood overweight and obesity: socioeconomic environment and childhood overweight*. Prev Med, 2012. **55**(2): p. 102-7.
162. Tyrrell, J., et al., *Gene-obesogenic environment interactions in the UK Biobank study*. International Journal of Epidemiology, 2017. **46**(2): p. 559-575.
163. Spence, J.C., et al., *Relation between local food environments and obesity among adults*. Bmc Public Health, 2009. **9**.
164. Barrett, M., et al., *Greater access to healthy food outlets in the home and school environment is associated with better dietary quality in young children*. Public Health Nutr, 2017. **20**(18): p. 3316-3325.
165. Lachowycz, K. and A.P. Jones, *Greenspace and obesity: a systematic review of the evidence*. Obes Rev, 2011. **12**(5): p. e183-9.
166. Rigolon, A., et al., *Green Space and Health Equity: A Systematic Review on the Potential of Green Space to Reduce Health Disparities*. Int J Environ Res Public Health, 2021. **18**(5).
167. Papas, M.A., et al., *The built environment and obesity*. Epidemiol Rev, 2007. **29**: p. 129-43.
168. Dunton, G.F., et al., *Physical environmental correlates of childhood obesity: a systematic review*. Obesity Reviews, 2009. **10**(4): p. 393-402.
169. Lovasi, G.S., et al., *Built environments and obesity in disadvantaged populations*. Epidemiol Rev, 2009. **31**: p. 7-20.
170. Garrett, J.K., et al., *Urban nature and physical activity: Investigating associations using self-reported and accelerometer data and the role of household income*. Environmental Research, 2020. **190**.
171. Claassen, M.A., et al., *A systematic review of psychosocial explanations for the relationship between socioeconomic status and body mass index*. Appetite, 2019. **132**: p. 208-221.
172. Barnes, R., et al., *Does Neighbourhood Walkability Moderate the Effects of Mass Media Communication Strategies to Promote Regular Physical Activity?* Annals of Behavioral Medicine, 2013. **45**: p. S86-S94.



173. White, M.P., et al., *Spending at least 120 minutes a week in nature is associated with good health and wellbeing*. Scientific Reports, 2019. **9**.
174. White, M.P., et al., *Would You Be Happier Living in a Greener Urban Area? A Fixed-Effects Analysis of Panel Data*. Psychological Science, 2013. **24**(6): p. 920-928.
175. Alcock, I., et al., *Longitudinal Effects on Mental Health of Moving to Greener and Less Green Urban Areas*. Environmental Science & Technology, 2014. **48**(2): p. 1247-1255.
176. Flouri, E., E. Midouhas, and H. Joshi, *The role of urban neighbourhood green space in children's emotional and behavioural resilience*. Journal of Environmental Psychology, 2014. **40**: p. 179-186.
177. Ohly, H., et al., *Attention Restoration Theory: A systematic review of the attention restoration potential of exposure to natural environments*. J Toxicol Environ Health B Crit Rev, 2016. **19**(7): p. 305-343.
178. Ulrich, R.S., *Natural Versus Urban Scenes - Some Psychophysiological Effects*. Environment and Behavior, 1981. **13**(5): p. 523-556.
179. Ulrich, R.S., et al., *Stress Recovery during Exposure to Natural and Urban Environments*. Journal of Environmental Psychology, 1991. **11**(3): p. 201-230.
180. Kinnafick, F.-E. and C. Thøgersen-Ntoumani, *The effect of the physical environment and levels of activity on affective states*. Journal of Environmental Psychology, 2014. **38**: p. 241-251.
181. Mitchell, R.J., et al., *Neighborhood Environments and Socioeconomic Inequalities in Mental Well-Being*. Am J Prev Med, 2015. **49**(1): p. 80-4.
182. Flouri, E., E. Midouhas, and M. Francesconi, *Neighbourhood deprivation and child behaviour across childhood and adolescence*. . Longitudinal and Life Course Studies. , 2019. **11**
183. Li, F., et al., *Obesity and the built environment: does the density of neighborhood fast-food outlets matter?* Am J Health Promot, 2009. **23**(3): p. 203-9.
184. Papadakis, T., S.G. Ferguson, and B. Schuz, *Within-Day Variability in Negative Affect Moderates Cue Responsiveness in High-Calorie Snacking*. Front Psychol, 2020. **11**: p. 590497.
185. Emery, C.F., et al., *Home environment and psychosocial predictors of obesity status among community-residing men and women*. Int J Obes (Lond), 2015. **39**(9): p. 1401-7.
186. Connelly, R. and L. Platt, *Cohort profile: UK Millennium Cohort Study (MCS)*. Int J Epidemiol, 2014. **43**(6): p. 1719-25.
187. Fitzsimons, E., *MCS Sixth Survey 2015-2016 User Guide*. 2020, Institute of Education: Centre for Longitudinal Studies.
188. Plewis, I., et al., *The Millennium Cohort Study: Technical Report on Sampling. 4th Edition I*. Plewis, Editor. 2007, Centre for Longitudinal Studies University of London.
189. Fitzsimons, E., *Millennium Cohort Study Sixth Sweep (MCS6): User Guide (First Edition)*. 2017: Centre for Longitudinal Studies, UCL Institute of Education.
190. Hansen, K., et al., *Millennium Cohort Study: A Guide to the Datasets (Eighth Edition). First, Second, Third, Fourth and Fifth Surveys*, K. Hansen, Editor. 2014, Centre for Longitudinal Studies
191. CLS, *Millennium Cohort Study Seventh Sweep (MCS7) - Technical Report*. 2019, Centre for Longitudinal Studies, UCL Institute of Education Ipsos MORI.
192. Mostafa, T. and G. Ploubidis, *Millennium Cohort Study Technical report on response (Age 14): Sixth Survey 2015-2016*. 2017: London: Centre for Longitudinal Studies.
193. Fitzsimons, E., et al., *Millennium Cohort Study Age 17 Sweep (MCS7): User Guide*. 2020: London: UCL Centre for Longitudinal Studies.
194. CLS, *Millennium Cohort Study Sixth Sweep (MCS6) - Technical Report*. 2017, Centre for Longitudinal Studies, UCL Institute of Education: Ipsos MORI.
195. Shepherd, P. and E. Gilbert, *Millennium Cohort Study: Ethical Review and Consent*. 2019: Centre for Longitudinal Studies: UCL Institute of Education.

196. Lee, S.Y. and D. Gallagher, *Assessment methods in human body composition*. Curr Opin Clin Nutr Metab Care, 2008. **11**(5): p. 566-72.
197. TANITA. *Tanita: Frequently Asked Questions* 2021 [12/05/2021]; Available from: <https://tanita.eu/help-guides/f-a-q/>.
198. Barreira, T.V., A.E. Staiano, and P.T. Katzmarzyk, *Validity assessment of a portable bioimpedance scale to estimate body fat percentage in white and African-American children and adolescents*. *Pediatr Obes*, 2013. **8**(2): p. e29-32.
199. Joanna Chaplin Gray, R.G., Nadine Simmonds and Yachien Huang, *Millennium Cohort Study: Sweep 4. Technical Report*. 2010: NatCen.
200. Griffiths, L.J., et al., *Objectively measured physical activity and sedentary time: cross-sectional and prospective associations with adiposity in the Millennium Cohort Study*. *Bmj Open*, 2016. **6**(4).
201. Sera, F., et al., *Effects of physical activity on reduction of adiposity across the entire obesity distribution in primary school-aged children: findings from the Millennium Cohort Study*. *Lancet*, 2013. **382**: p. 89-89.
202. Johnson, W., et al., *Differences in the relationship of weight to height, and thus the meaning of BMI, according to age, sex, and birth year cohort*. *Ann Hum Biol*, 2020. **47**(2): p. 199-207.
203. National Statistics, *The English Indices of Deprivation 2019 (IoD2019) in National Statistics: Statistical Release*. 2019: Ministry of Housing, Communities & Local Government
204. CLS, *Millennium Cohort Study: User Guide (Surveys 1 – 5). 9th Edition*. 2020: Centre for Longitudinal Studies, UCL Institute of Education. London.
205. Emerson, E., *Overweight and obesity in 3- and 5-year-old children with and without developmental delay*. *Public Health*, 2009. **123**(2): p. 130-3.
206. Flouri, E., N. Tzavidis, and C. Kallis, *Area and family effects on the psychopathology of the Millennium Cohort Study children and their older siblings*. *J Child Psychol Psychiatry*, 2010. **51**(2): p. 152-61.
207. Payne, R.A. and G.A. Abel, *UK indices of multiple deprivation – a way to make comparisons across constituent countries easier*, in *Health Statistics Quarterly*. 2012, Office for National Statistics: Office for National Statistics.
208. Goodman, R., *The strengths and difficulties questionnaire: A research note*. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 1997. **38**(5): p. 581-586.
209. Piantadosi, S., D.P. Byar, and S.B. Green, *The Ecological Fallacy*. *American Journal of Epidemiology*, 1988. **127**(5): p. 893-904.
210. Bergström, L., M. van Ham, and M. van Ham, *Understanding Neighbourhood Effects: Selection Bias and Residential Mobility*. IZA Discussion Paper, 2010(No. 5193).
211. Ioakeimidi, S., E. Midouhas, and D. Church, *Data Note: Green space data at Lower layer Super Output LSOA-level (England) and ward-level (UK), linked to MCS1-MCS5, in CLS Data Note / User guide to the data (First Edition)*, U.I.o.E. Centre for Longitudinal Studies, Editor. 2017: UCL Institute of Education.
212. Libuy, N., D. Church, and E. Fitzsimons, *Millennium Cohort Study: Linkage with the Point of Interest Data*, U.I.o.E. centre for Longitudinal Studies, Editor. 2021: UCL Institute of Education.
213. Gatineau, M. and S. Mathrani, *Ethnicity and obesity in the UK*. *Perspect Public Health*, 2011. **131**(4): p. 159-60.
214. El-Sayed, A.M., P. Scarborough, and S. Galea, *Ethnic inequalities in obesity among children and adults in the UK: a systematic review of the literature*. *Obes Rev*, 2011. **12**(5): p. e516-34.
215. Tennant, P.W.G., et al., *Use of directed acyclic graphs (DAGs) to identify confounders in applied health research: review and recommendations*. *International Journal of Epidemiology*, 2021. **50**(2): p. 620-632.

216. Ketende, S.C. and E.M. Jones, *The Millenium Cohort Study: User Guide to Analysing MCS Data Using STATA*. 2011: The Centre for Longitudinal Studies, Institute of Education, University of London.
217. Rubin, D.B., *Inference and Missing Data*. Biometrika, 1976. **63**(3): p. 581-590.
218. Buuren, S.v., *Flexible Imputation of Missing Data*. Second Edition ed. 2012: Chapman and Hall/CRC.
219. Asparouhov, T. and B. Muthen, *Weighted Least Squares Estimation with Missing Data*. 2010.
220. Jakobsen, J.C., et al., *When and how should multiple imputation be used for handling missing data in randomised clinical trials - a practical guide with flowcharts*. BMC Medical Research Methodology, 2017. **17**.
221. Bridger Staatz, C., et al., *Socioeconomic position and body composition across the life course: a systematic review protocol*. Syst Rev, 2019. **8**(1): p. 263.
222. Moher, D., et al., *Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015 statement*. Syst Rev, 2015. **4**: p. 1.
223. Krieger, N., D.R. Williams, and N.E. Moss, *Measuring social class in US public health research: concepts, methodologies, and guidelines*. Annu Rev Public Health, 1997. **18**: p. 341-78.
224. Ouzzani, M., et al., *Rayyan-a web and mobile app for systematic reviews*. Syst Rev, 2016. **5**(1): p. 210.
225. Wells G, S.B., O'connell D, Peterson J, Welch V, Losos M, Tugwell P., *The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses*. 2000.
226. Rohatgi, A. *WebPlotDigitizer*. 2020 02/12/2020]; Available from: <https://automeris.io/WebPlotDigitizer>.
227. Popay, J.R., H. Sowden, A. Petticrew, M. Arai, L. Rodgers, M. Britten, N. Roen, K. Duffy, S., *Guidance on the Conduct of Narrative Synthesis in Systematic Reviews* 2006, Lancaster University: Lancaster.
228. Ball, K. and D. Crawford, *Socioeconomic status and weight change in adults: a review*. Soc Sci Med, 2005. **60**(9): p. 1987-2010.
229. The World Bank. *World Bank Country and Lending Groups*. 2019 [cited 2019 13/11/19]; Available from: <https://datahelpdesk.worldbank.org/knowledgebase/articles/906519-world-bank-country-and-lending-groups>.
230. Apouey, B.H., *Child physical development in the UK: the imprint of time and socioeconomic status*. Public Health, 2016. **141**: p. 255-263.
231. Azcorra, H., F. Dickinson, and S. Datta Banik, *Maternal height and its relationship to offspring birth weight and adiposity in 6- to 10-year-old Maya children from poor neighborhoods in Merida, Yucatan*. American Journal of Physical Anthropology, 2016. **161**(4): p. 571-579.
232. Baird, J., et al., *Duration of sleep at 3 years of age is associated with fat and fat-free mass at 4 years of age: the Southampton Women's Survey*. Journal of Sleep Research, 2016. **25**(4): p. 412-8.
233. Boot, A.M., et al., *Determinants of body composition measured by dual-energy X-ray absorptiometry in Dutch children and adolescents*. American Journal of Clinical Nutrition, 1997. **66**(2): p. 232-8.
234. Brown, D.E., et al., *Measures of adiposity in two cohorts of Hawaiian school children*. Annals of Human Biology, 2011. **38**(4): p. 492-9.
235. Burdette, H.L., et al., *Breastfeeding, introduction of complementary foods, and adiposity at 5 y of age*. American Journal of Clinical Nutrition, 2006. **83**(3): p. 550-8.
236. Cardel, M., et al., *Parental feeding practices and socioeconomic status are associated with child adiposity in a multi-ethnic sample of children*. Appetite, 2012. **58**(1): p. 347-353.
237. Carter, P.J., et al., *Longitudinal analysis of sleep in relation to BMI and body fat in children: the FLAME study*. BMJ, 2011. **342**: p. d2712.
238. Castro, N.P., et al., *The Relationship between Maternal Plasma Leptin and Adiponectin Concentrations and Newborn Adiposity*. Nutrients, 2017. **9**(3): p. 23.

239. Cheng, S., et al., *Trait-specific tracking and determinants of body composition: a 7-year follow-up study of pubertal growth in girls*. BMC Medicine, 2009. **7**: p. 5.
240. Chomtho, S., et al., *Associations between birth weight and later body composition: evidence from the 4-component model*. American Journal of Clinical Nutrition, 2008. **88**(4): p. 1040-8.
241. Collings, P.J., et al., *Objectively measured physical activity and longitudinal changes in adolescent body fatness: an observational cohort study*. Pediatric Obesity, 2015. **11**(2): p. 107-14.
242. Datta Banik, S., et al., *Body fatness in relation to physical activity and selected socioeconomic parameters of adolescents aged 15-17 years in Merida, Yucatan*. Ann Hum Biol, 2014. **41**(6): p. 497-505.
243. Datta Banik, S., A. Andrade, and F. Dickinson, *Adiposity and body composition of 13-and 14-year-old boys in Merida, Mexico: A study in relation with macronutrient consumption rates and socioeconomic status*. Obesity, 2011. **1**: p. S214-S215.
244. Datta Banik, S., et al., *The effect of socioeconomic indicators and macronutrient intake rate on body composition in adolescents 12 to 16 years old in Merida, Yucatan*. Anthropologischer Anzeiger, 2014. **71**(4): p. 347-68.
245. De Vriendt, T., et al., *European adolescents' level of perceived stress and its relationship with body adiposity--the HELENA Study*. European Journal of Public Health, 2011. **22**(4): p. 519-24.
246. Dowda, M., et al., *Physical Activity and Changes in Adiposity in the Transition from Elementary to Middle School*. Childhood Obesity, 2017. **13**(1): p. 53-62.
247. Duncan, J.S., et al., *Risk factors for excess body fatness in New Zealand children*. Asia Pacific Journal of Clinical Nutrition, 2008. **17**(1): p. 138-147.
248. Ebenegger, V., et al., *Independent contribution of parental migrant status and educational level to adiposity and eating habits in preschool children*. European Journal of Clinical Nutrition, 2011. **65**(2): p. 210-8.
249. Ekelund, U., et al., *Associations between physical activity and fat mass in adolescents: the Stockholm Weight Development Study*. American Journal of Clinical Nutrition, 2005. **81**(2): p. 355-60.
250. Ekelund, U., et al., *Upward weight percentile crossing in infancy and early childhood independently predicts fat mass in young adults: the Stockholm Weight Development Study (SWEDES)*. American Journal of Clinical Nutrition, 2006. **83**(2): p. 324-30.
251. Gracia-Marco, L., et al., *Socioeconomic status and bone mass in Spanish adolescents. The HELENA Study*. Journal of Adolescent Health, 2012. **50**(5): p. 484-90.
252. Griffiths, P.L., et al., *In urban South Africa, 16 year old adolescents experience greater health equality than children*. Economics & Human Biology, 2013. **11**(4): p. 502-14.
253. Griffiths, P.L., et al., *Socio-economic status and body composition outcomes in urban South African children*. Archives of Disease in Childhood, 2008. **93**(10): p. 862-7.
254. Hou, W.W., et al., *Adolescent testosterone, muscle mass and glucose metabolism: Evidence from "children of 1997" in hong kong*. American Journal of Epidemiology, 2014. **11**: p. S93.
255. Howe, L.D., et al., *Are there socioeconomic inequalities in cardiovascular risk factors in childhood, and are they mediated by adiposity? Findings from a prospective cohort study*. International Journal of Obesity, 2010. **34**(7): p. 1149-59.
256. Howe, L.D., D.A. Lawlor, and C. Propper, *Trajectories of socioeconomic inequalities in health, behaviours and academic achievement across childhood and adolescence*. J Epidemiol Community Health, 2013. **67**(4): p. 358-64.
257. Johnson, L., et al., *A prospective analysis of dietary energy density at age 5 and 7 years and fatness at 9 years among UK children*. International Journal of Obesity, 2008. **32**(4): p. 586-93.
258. Khadilkar, A.V., et al., *Poor bone health in underprivileged Indian girls: an effect of low bone mass accrual during puberty*. Bone, 2012. **50**(5): p. 1048-53.

259. Lagoa, M.J., et al., *Parent-children resemblance and associations of family behaviour on children body fat*. *Obesity Facts*, 2015. **1**: p. 120.
260. Lantz, H., et al., *Body composition in a cohort of Swedish adolescents aged 15, 17 and 20.5 years*. *Acta Paediatrica*, 2008. **97**(12): p. 1691-7.
261. Magalhaes, T.C., et al., *Exclusive breastfeeding and other foods in the first six months of life: effects on nutritional status and body composition of Brazilian children*. *TheScientificWorldJournal*, 2012. **2012**: p. 468581.
262. Matsudo, V.K.R., et al., *Socioeconomic status indicators, physical activity, and overweight/obesity in Brazilian children*. *Revista Paulista de Pediatria*, 2016. **34**(2): p. 162-170.
263. McCarthy, H.D., C. Van Eeden, and D. Samani-Radia, *Influence of socioeconomic status on measures of skeletal muscle mass in children*. *Proceedings of the Nutrition Society. Conference: Winter Meeting Nutrition and Age Related Muscle Loss, Sarcopenia and Cachexia*. United Kingdom, 2015. **74**.
264. Molina-Garcia, J., et al., *Neighborhood built environment and socio-economic status in relation to multiple health outcomes in adolescents*. *Preventive Medicine*, 2017. **105**: p. 88-94.
265. Ness, A.R., et al., *The social patterning of fat and lean mass in a contemporary cohort of children*. *International Journal of Pediatric Obesity*, 2006. **1**(1): p. 59-61.
266. Nguyen, T.T., N.F. Butte, and Y. Wang, *Demographic and socioeconomic correlates of adiposity assessed with dual-energy X-ray absorptiometry in US children and adolescents*. *American Journal of Clinical Nutrition*, 2012. **96**(5): p. 1104-12.
267. Nguyen, T.T., N.F. Butte, and Y. Wang, *Demographic and socioeconomic correlates of body fat assessed using DXA in US children and adolescents*. *FASEB Journal. Conference: Experimental Biology*, 2011. **25**.
268. Plachta-Danielzik, S., et al., *Family and lifestyle factors mediate the association between socio-economic status and fat mass in children and adolescents*. *Obesity Facts*, 2015. **1**: p. 145.
269. Samani-Radia, D. and H.D. McCarthy, *Comparison of children's body fatness between two contrasting income groups: contribution of height difference*. *International Journal of Obesity*, 2011. **35**(1): p. 128-33.
270. Santos, I.S., et al., *Cohort profile update: 2004 pelotas (Brazil) birth cohort study. Body composition, mental health and genetic assessment at the 6 years follow-up*. *International Journal of Epidemiology*, 2014. **43**(5): p. 1437-1437f.
271. Schaefer, S.E., et al., *Influence of race, acculturation, and socioeconomic status on tendency toward overweight in Asian-American and Mexican-American early adolescent females*. *Journal of Immigrant & Minority Health*, 2009. **11**(3): p. 188-97.
272. Shakir, R.N., et al., *Not all sedentary behaviour is equal: Children's adiposity and sedentary behaviour volumes, patterns and types*. *Obesity Research and Clinical Practice*, 2018. **12**(6): p. 506-512.
273. Ulbricht, L., et al., *Prevalence of excessive body fat among adolescents of a south Brazilian metropolitan region and State capital, associated risk factors, and consequences*. *BMC Public Health*, 2018. **18**(1): p. 312.
274. Veena, S.R., et al., *Relationship between adiposity and cognitive performance in 9-10-year-old children in South India*. *Archives of Disease in Childhood*, 2014. **99**(2): p. 126-34.
275. Willig, A.L., et al., *Birth weight is associated with body composition in a multiethnic pediatric cohort*. *Open Obesity Journal*, 2011. **3**: p. 4-8.
276. Wohlfahrt-Veje, C., et al., *Body fat throughout childhood in 2647 healthy Danish children: agreement of BMI, waist circumference, skinfolds with dual X-ray absorptiometry*. *European Journal of Clinical Nutrition*, 2014. **68**(6): p. 664-70.

277. Zanini, R.V., et al., *Body composition assessment using DXA in six-year-old children: the 2004 Pelotas Birth Cohort, Rio Grande do Sul State, Brazil*. *Cadernos de Saude Publica*, 2014. **30**(10): p. 2123-33.
278. Agha, G., et al., *Prenatal socioeconomic index in relation to adulthood fat mass and fat distribution*. *American Journal of Epidemiology*, 2013. **11**: p. S10.
279. Al-Qaoud, T.M., et al., *Socioeconomic status and reduced kidney function in the Whitehall II Study: role of obesity and metabolic syndrome*. *American Journal of Kidney Diseases*, 2011. **58**(3): p. 389-97.
280. Amador, C., et al., *Regional variation in health is predominantly driven by lifestyle rather than genetics*. *Nature communications*, 2017. **8**(1): p. 801.
281. Amani, R., *Comparison between bioelectrical impedance analysis and body mass index methods in determination of obesity prevalence in Ahvazi women*. *European Journal of Clinical Nutrition*, 2007. **61**(4): p. 478-82.
282. Azarbal, F., et al., *Lean body mass and risk of incident atrial fibrillation in post-menopausal women*. *European Heart Journal*, 2016. **37**(20): p. 1606-1613.
283. Bae, Y.J., *Association between nutrient intake and osteosarcopenic obesity: Korea national health and nutrition examination survey 2008-2010*. *Osteoporosis International*, 2018. **29**: p. S421.
284. Bai, H.J., et al., *Age-related decline in skeletal muscle mass and function among elderly men and women in Shanghai, China: a cross sectional study*. *Asia Pacific Journal of Clinical Nutrition*, 2016. **25**(2): p. 326-32.
285. Bann, D., et al., *Socioeconomic position across life and body composition in early old age: findings from a British birth cohort study*. *J Epidemiol Community Health*, 2014. **68**(6): p. 516-23.
286. Barrera, G., et al., *Associations between socioeconomic status, aging and functionality among older women*. *Geriatric Nursing*, 2017. **38**(4): p. 347-351.
287. Bhupathiraju, S.N., et al., *Centrally located body fat is associated with lower bone mineral density in older Puerto Ricans*. *American Journal of Epidemiology*, 2011. **11**: p. S116.
288. Brennan, S.L., et al., *Socioeconomic status and risk factors for obesity and metabolic disorders in a population-based sample of adult females*. *Prev Med*, 2009. **49**(2-3): p. 165-71.
289. Buemann, B., A. Tremblay, and C. Bouchard, *Social-Class Interacts with the Association between Macronutrient Intake and Subcutaneous Fat*. *International Journal of Obesity*, 1995. **19**(11): p. 770-775.
290. De Marchi, R.J., et al., *Association between number of teeth, edentulism and use of dentures with percentage body fat in south Brazilian community-dwelling older people*. *Gerodontology*, 2010. **29**(2): p. e69-76.
291. Dugan, S.A., et al., *Physical Activity and Reduced Intra-abdominal Fat in Midlife African-American and White Women*. *Obesity (19307381)*, 2010. **18**(6): p. 1260-1265.
292. Dupuy, C., et al., *Dietary vitamin D intake and muscle mass in older women. Results from a cross-sectional analysis of the EPIDOS study*. *Journal of Nutrition, Health & Aging*, 2013. **17**(2): p. 119-24.
293. Fedewa, M.V., et al., *Area-level socioeconomic status, adiposity, physical activity, and inflammation in young adults, 2013*. *Preventing Chronic Disease*, 2014. **11**: p. E130.
294. Guo, W., T.J. Key, and G.K. Reeves, *Adiposity and breast cancer risk in postmenopausal women: Results from the UK Biobank prospective cohort*. *International Journal of Cancer*, 2018. **143**(5): p. 1037-1046.
295. Kazlauskaitė, R., et al., *The association between self-reported energy intake and intra-abdominal adipose tissue in perimenopausal women*. *Journal of Obesity*, 2012. **2012**(567320).
296. Keighley, E.D., et al., *Farming and adiposity in Samoan adults*. *American Journal of Human Biology*, 2006. **18**(1): p. 112-22.

297. Keino, S., G. Plasqui, and B. Van Den Borne, *Measuring adiposity among women in Narok county, Kenya: Comparison between body mass index, waist-hip ratio, waist circumference, fat mass index and percentage fat mass*. *Annals of Nutrition and Metabolism*, 2017. **71**: p. 1044.
298. Kim, J., et al., *Association between healthy diet and exercise and greater muscle mass in older adults*. *Journal of the American Geriatrics Society*, 2015. **63**(5): p. 886-92.
299. Krueger, D., et al., *Factors affecting vitamin D status: Evaluation of a midus cohort*. *Journal of Clinical Densitometry*, 2010. **13**: p. 125.
300. Kruger, H.S., et al., *Physical Activity Energy Expenditure and Sarcopenia in Black South African Urban Women*. *Journal of Physical Activity & Health*, 2016. **13**(3): p. 296-302.
301. Kulkarni, B., et al., *Regional body composition of Indian women from a low-income group and its association with anthropometric indices and reproductive events*. *Annals of Nutrition & Metabolism*, 2010. **56**(3): p. 182-9.
302. Lahmann, P.H., et al., *Differences in body fat and central adiposity between Swedes and European immigrants: the Malmo Diet and Cancer Study*. *Obesity Research*, 2000. **8**(9): p. 620-31.
303. Lahmann, P.H., et al., *Sociodemographic factors associated with long-term weight gain, current body fatness and central adiposity in Swedish women*. *International Journal of Obesity & Related Metabolic Disorders: Journal of the International Association for the Study of Obesity*, 2000. **24**(6): p. 685-94.
304. Lewin, A., et al., *Residential neighborhood, geographic work environment, and work economic sector: associations with body fat measured by bioelectrical impedance in the RECORD Study*. *Annals of Epidemiology*, 2014. **24**(3): p. 180-6.
305. Lewis, T.T., et al., *Hostility is associated with visceral, but not subcutaneous, fat in middle-aged african American and white women*. *Psychosomatic Medicine*, 2009. **71**(7): p. 733-740.
306. Loucks, E.B., et al., *Associations of Dispositional Mindfulness with Obesity and Central Adiposity: the New England Family Study*. *International Journal of Behavioral Medicine*, 2015. **23**(2): p. 224-33.
307. Lourenco, A.E.P., et al., *Nutrition transition in Amazonia: Obesity and socioeconomic change in the Surui Indians from Brazil*. *American Journal of Human Biology*, 2008. **20**(5): p. 564-571.
308. McClure, C., et al., *Breastfeeding and Subsequent Maternal Visceral Adiposity*. *Obesity (19307381)*, 2011. **19**(11): p. 2205-2213.
309. Mongraw-Chaffin, M., et al., *CT-Derived Body Fat Distribution and Incident Cardiovascular Disease: The Multi-Ethnic Study of Atherosclerosis*. *Journal of Clinical Endocrinology & Metabolism*, 2017. **102**(11): p. 4173-4183.
310. Ozener, B., I. Duyar, and D. Atamturk, *Body composition of young laborers: the results of a bioelectrical impedance analysis*. *Collegium Antropologicum*, 2007. **31**(4): p. 949-54.
311. Pirila, S., et al., *Breastfeeding and determinants of adult body composition: A prospective study from birth to young adulthood*. *Hormone Research in Paediatrics*, 2012. **77**(5): p. 281-290.
312. Powell, M., et al., *Association between ratio indexes of body composition phenotypes and metabolic risk in Italian adults*. *Clinical Obesity*, 2016. **6**(6): p. 365-375.
313. Rangel Peniche, D.B., et al., *Differences in Body Composition in Older People from Two Regions of Mexico: Implications for Diagnoses of Sarcopenia and Sarcopenic Obesity*. *BioMed Research International*, 2018. **2018**: p. 7538625.
314. Rebato, E., et al., *Nutritional status by socioeconomic level in an urban sample from Bilbao (Basque Country)*. *American Journal of Human Biology*, 2001. **13**(5): p. 668-78.
315. Sallinen, J., et al., *Effect of age on the association between body fat percentage and maximal walking speed*. *Journal of Nutrition, Health and Aging*, 2011. **15**(6): p. 427-432.
316. Seppanen-Nuijten, E., et al., *Fat free mass and obesity in relation to educational level*. *BMC Public Health*, 2009. **9**: p. 448.

317. Sotillo, C., et al., *Body composition in an adult population in southern Spain: influence of lifestyle factors*. International Journal for Vitamin & Nutrition Research, 2007. **77**(6): p. 406-14.
318. Suder, A., *Body fatness and its social and lifestyle determinants in young working males from Cracow, Poland*. Journal of Biosocial Science, 2009. **41**(1): p. 139-54.
319. Velasquez-Melendez, G., et al., *Relationship between sitting-height-to-stature ratio and adiposity in Brazilian women*. American Journal of Human Biology, 2005. **17**(5): p. 646-53.
320. Visser, M., et al., *Body fat and skeletal muscle mass in relation to physical disability in very old men and women of the Framingham Heart Study*. Journals of Gerontology Series A-Biological Sciences & Medical Sciences, 1998. **53**(3): p. M214-21.
321. Wu, C.H., et al., *Relationship among habitual tea consumption, percent body fat, and body fat distribution*. Obesity Research, 2002. **11**(9): p. 1088-95.
322. Yliharsila, H., et al., *Birth size, adult body composition and muscle strength in later life*. International Journal of Obesity, 2007. **31**(9): p. 1392-1399.
323. Shrewsbury, V. and J. Wardle, *Socioeconomic status and adiposity in childhood: A systematic review of cross-sectional studies 1990-2005*. Obesity, 2008. **16**(2): p. 275-284.
324. Barriuso, L., et al., *Socioeconomic position and childhood-adolescent weight status in rich countries: a systematic review, 1990-2013*. BMC Pediatr, 2015. **15**: p. 129.
325. Birnie, K., et al., *Childhood socioeconomic position and objectively measured physical capability levels in adulthood: a systematic review and meta-analysis*. PLoS One, 2011. **6**(1): p. e15564.
326. Wind, A.E., et al., *Is grip strength a predictor for total muscle strength in healthy children, adolescents, and young adults?* Eur J Pediatr, 2010. **169**(3): p. 281-7.
327. Bouchard, D.R., M. Heroux, and I. Janssen, *Association between muscle mass, leg strength, and fat mass with physical function in older adults: influence of age and sex*. J Aging Health, 2011. **23**(2): p. 313-28.
328. Churchward-Venne, T.A., N.A. Burd, and S.M. Phillips, *Nutritional regulation of muscle protein synthesis with resistance exercise: strategies to enhance anabolism*. Nutr Metab (Lond), 2012. **9**(1): p. 40.
329. Phillips, S.M., *A brief review of critical processes in exercise-induced muscular hypertrophy*. Sports Med, 2014. **44 Suppl 1**: p. S71-7.
330. Kulkarni, B., *Addressing the Double Burden of Malnutrition in Developing Countries: Need for Strategies to Improve the Lean Body Mass*. Food Nutr Bull, 2018. **39**(2\_suppl): p. S69-S76.
331. Braun, K.V., et al., *Dietary Intake of Protein in Early Childhood Is Associated with Growth Trajectories between 1 and 9 Years of Age*. J Nutr, 2016. **146**(11): p. 2361-2367.
332. Perkins, J.M., et al., *Adult height, nutrition, and population health*. Nutr Rev, 2016. **74**(3): p. 149-65.
333. Popkin, B.M., *Nutrition Transition and the Global Diabetes Epidemic*. Curr Diab Rep, 2015. **15**(9): p. 64.
334. Mayen, A.L., et al., *Socioeconomic determinants of dietary patterns in low- and middle-income countries: a systematic review*. American Journal of Clinical Nutrition, 2014. **100**(6): p. 1520-1531.
335. Darmon, N. and A. Drewnowski, *Does social class predict diet quality?* American Journal of Clinical Nutrition, 2008. **87**(5): p. 1107-1117.
336. Darmon, N. and A. Drewnowski, *Contribution of food prices and diet cost to socioeconomic disparities in diet quality and health: a systematic review and analysis*. Nutrition Reviews, 2015. **73**(10): p. 643-660.
337. Muller, O. and M. Krawinkel, *Malnutrition and health in developing countries*. CMAJ, 2005. **173**(3): p. 279-86.
338. Wells, J.C.K., *Body composition of children with moderate and severe undernutrition and after treatment: a narrative review*. BMC Med, 2019. **17**(1): p. 215.



339. Hallal, P.C., et al., *Global physical activity levels: surveillance progress, pitfalls, and prospects*. Lancet, 2012. **380**(9838): p. 247-57.
340. Gidlow, C., et al., *A systematic review of the relationship between socio-economic position and physical activity*. Health Education Journal, 2006. **65**(4): p. 338–367.
341. Beenackers, M.A., et al., *Socioeconomic inequalities in occupational, leisure-time, and transport related physical activity among European adults: a systematic review*. Int J Behav Nutr Phys Act, 2012. **9**: p. 116.
342. Pearce, M., et al., *Is occupational physical activity associated with mortality in UK Biobank?* Int J Behav Nutr Phys Act, 2021. **18**(1): p. 102.
343. Love, R., et al., *Socioeconomic and ethnic differences in children's vigorous intensity physical activity: a cross-sectional analysis of the UK Millennium Cohort Study*. BMJ Open, 2019. **9**(5): p. e027627.
344. Stalsberg, R. and A.V. Pedersen, *Effects of socioeconomic status on the physical activity in adolescents: a systematic review of the evidence*. Scand J Med Sci Sports, 2010. **20**(3): p. 368-83.
345. Russell, S.J., H. Croker, and R.M. Viner, *The effect of screen advertising on children's dietary intake: A systematic review and meta-analysis*. Obesity Reviews, 2019. **20**(4): p. 554-568.
346. Osei-Assibey, G., et al., *The influence of the food environment on overweight and obesity in young children: a systematic review*. BMJ Open, 2012. **2**(6).
347. Ferdinand, A.O., et al., *The Relationship Between Built Environments and Physical Activity: A Systematic Review*. American Journal of Public Health, 2012. **102**(10): p. E7-E13.
348. Gupta, N., et al., *Childhood obesity in developing countries: epidemiology, determinants, and prevention*. Endocr Rev, 2012. **33**(1): p. 48-70.
349. Bhardwaj, S., et al., *Childhood obesity in Asian Indians: a burgeoning cause of insulin resistance, diabetes and sub-clinical inflammation*. Asia Pac J Clin Nutr, 2008. **17 Suppl 1**: p. 172-5.
350. Rguibi, M. and R. Belahsen, *Body size preferences and sociocultural influences on attitudes towards obesity among Moroccan Sahraoui women*. Body Image, 2006. **3**(4): p. 395-400.
351. Hossain, M.S., et al., *Is Childhood Overweight/Obesity Perceived as a Health Problem by Mothers of Preschool Aged Children in Bangladesh? A Community Level Cross-Sectional Study*. International Journal of Environmental Research and Public Health, 2019. **16**(2).
352. Penuelas, J., et al., *Increasing gap in human height between rich and poor countries associated to their different intakes of N and P*. Sci Rep, 2017. **7**(1): p. 17671.
353. Chaston, T.B., J.B. Dixon, and P.E. O'Brien, *Changes in fat-free mass during significant weight loss: a systematic review*. Int J Obes (Lond), 2007. **31**(5): p. 743-50.
354. Forbes, G.B., *Some adventures in body composition, with special reference to nutrition*. Acta Diabetologica, 2003. **40**: p. S238-S241.
355. Yin, H.S., et al., *Parent health literacy and "obesogenic" feeding and physical activity-related infant care behaviors*. J Pediatr, 2014. **164**(3): p. 577-83 e1.
356. Costarelli, V., et al., *Parental health literacy and nutrition literacy affect child feeding practices: A cross-sectional study*. Nutr Health, 2021: p. 2601060211001489.
357. Strauss, R.S. and J. Knight, *Influence of the home environment on the development of obesity in children*. Pediatrics, 1999. **103**(6): p. e85.
358. Dodds, R.M., et al., *Grip strength across the life course: normative data from twelve British studies*. PLoS One, 2014. **9**(12): p. e113637.
359. Kuh, D., et al., *Birth weight, childhood size, and muscle strength in adult life: evidence from a birth cohort study*. Am J Epidemiol, 2002. **156**(7): p. 627-33.
360. Cruz, F., et al., *Tracking of food and nutrient intake from adolescence into early adulthood*. Nutrition, 2018. **55-56**: p. 84-90.
361. Elhakeem, A., et al., *Childhood socioeconomic position and adult leisure-time physical activity: a systematic review*. Int J Behav Nutr Phys Act, 2015. **12**: p. 92.

362. Sterne, J.A. and G. Davey Smith, *Sifting the evidence-what's wrong with significance tests?* BMJ, 2001. **322**(7280): p. 226-31.
363. Halsey, L.G., *The reign of the p-value is over: what alternative analyses could we employ to fill the power vacuum?* Biology Letters, 2019. **15**(5).
364. Wasserstein, R.L., A.L. Schirm, and N.A. Lazar, *Moving to a World Beyond "p < 0.05"*. American Statistician, 2019. **73**: p. 1-19.
365. Strugnell, C., et al., *Influence of age and gender on fat mass, fat-free mass and skeletal muscle mass among Australian adults: the Australian diabetes, obesity and lifestyle study (AusDiab)*. J Nutr Health Aging, 2014. **18**(5): p. 540-6.
366. Curran, P.J., K. Obeidat, and D. Losardo, *Twelve Frequently Asked Questions About Growth Curve Modeling*. Journal of Cognition and Development, 2010. **11**(2): p. 121-136.
367. Brown, V.A., *An Introduction to Linear Mixed-Effects Modeling in R*. Advances in Methods and Practices in Psychological Science, 2021. **4**: p. 1-19.
368. Lu, W. and A. Sacker, *A Case Study of the Application of a Multilevel Growth Curve Model and the Prediction of Health Trajectories*, in *Research Methods Cases: Medicine and Health*. 2020: SAGE Publications Ltd.
369. Singer, J.D. and J.B. Willett, *Modeling Discontinuous and Nonlinear Change*, in *Applied Longitudinal Data Analysis: Modeling Change and Event Occurrence*. 2003: Oxford Scholarship Online.
370. StataCorp, *Stata Statistical Software: Release 15*. 2017, StataCorp LLC.: College Station, TX.
371. Cohen, M.P., *Auxiliary variable*, in *Encyclopedia of survey research methods*, P.J. Lavrakas, Editor. 2011, SAGE. p. 46.
372. Rubin, D., *Multiple Imputation for Nonresponse in Surveys*. 2004, New York: John Wiley and Sons.
373. Sterne, J.A., et al., *Multiple imputation for missing data in epidemiological and clinical research: potential and pitfalls*. BMJ, 2009. **338**: p. b2393.
374. Skinner, C.J.a.W., *Jon Introduction to the design and analysis of complex survey data*. . Statistical Science, 2017. **32**(2): p. 165-175.
375. Howe, L.D., et al., *Socioeconomic disparities in trajectories of adiposity across childhood*. Int J Pediatr Obes, 2011. **6**(2-2): p. e144-53.
376. Bridger Staatz, C., et al., *Life course socioeconomic position and body composition in adulthood: a systematic review and narrative synthesis*. International Journal of Obesity, 2021.
377. Jansen, P.W., et al., *Family and neighbourhood socioeconomic inequalities in childhood trajectories of BMI and overweight: longitudinal study of Australian children*. PLoS One, 2013. **8**(7): p. e69676.
378. Mireku, M.O. and A. Rodriguez, *Family Income Gradients in Adolescent Obesity, Overweight and Adiposity Persist in Extremely Deprived and Extremely Affluent Neighbourhoods but Not in Middle-Class Neighbourhoods: Evidence from the UK Millennium Cohort Study*. Int J Environ Res Public Health, 2020. **17**(2).
379. Bridger Staatz, C., et al., *Socioeconomic position and body composition in childhood in high- and middle-income countries: a systematic review and narrative synthesis*. International Journal of Obesity, 2021.
380. Janssen, I., S.B. Heymsfield, and R. Ross, *Low relative skeletal muscle mass (sarcopenia) in older persons is associated with functional impairment and physical disability*. Journal of the American Geriatrics Society, 2002. **50**(5): p. 889-896.
381. Mollborn, S., E. Lawrence, and P.M. Krueger, *Developing Health Lifestyle Pathways and Social Inequalities Across Early Childhood*. Popul Res Policy Rev, 2020.
382. Iguacel, I., et al., *Social vulnerabilities as risk factor of childhood obesity development and their role in prevention programs*. International Journal of Obesity, 2021. **45**(1): p. 1-11.

383. Iguacel, I., et al., *Social vulnerabilities as determinants of overweight in 2-, 4-and 6-year-old Spanish children*. European Journal of Public Health, 2018. **28**(2): p. 289-295.
384. Guo, S.S., et al., *Age- and maturity-related changes in body composition during adolescence into adulthood: the Fels longitudinal study*. Applied Radiation and Isotopes, 1998. **49**(5-6): p. 581-585.
385. Cole, T.J., J.V. Freeman, and M.A. Preece, *Body mass index reference curves for the UK, 1990*. Arch Dis Child, 1995. **73**(1): p. 25-9.
386. Wells, J.C., et al., *Body-composition reference data for simple and reference techniques and a 4-component model: a new UK reference child*. Am J Clin Nutr, 2012. **96**(6): p. 1316-26.
387. Abel, G.A., M.E. Barclay, and R.A. Payne, *Adjusted indices of multiple deprivation to enable comparisons within and between constituent countries of the UK including an illustration using mortality rates*. BMJ Open, 2016. **6**(11): p. e012750.
388. Griffiths, M., et al., *Metabolic-Rate and Physical Development in Children at Risk of Obesity*. Lancet, 1990. **336**(8707): p. 76-78.
389. Bertin, M., et al., *Can a deprivation index be used legitimately over both urban and rural areas?* International Journal of Health Geographics, 2014. **13**.
390. Feuillet, T., et al., *Influence of the urban context on the relationship between neighbourhood deprivation and obesity*. Social Science & Medicine, 2020. **265**.
391. Hiscock, R., et al., *Socioeconomic status and smoking: a review*. Addiction Reviews, 2012. **1248**: p. 107-123.
392. Ren, J. and Y. Huang, *Socioeconomic Status and Women's Smoking Behavior: A Literature Review*. Healthmed, 2011. **5**(2): p. 343-349.
393. Magalhaes, E.I.D., et al., *Maternal smoking during pregnancy and offspring body mass index and overweight: a systematic review and meta-analysis*. Cadernos De Saude Publica, 2019. **35**(12).
394. Magalhaes, E.I.D., et al., *Maternal smoking during pregnancy and offspring body composition in adulthood: Results from two birth cohort studies*. Bmj Open, 2019. **9**(6).
395. Sayer, A.A. and C. Cooper, *Fetal programming of body composition and musculoskeletal development*. Early Human Development, 2005. **81**(9): p. 735-744.
396. Topham, G.L., et al., *Maternal depression and socio-economic status moderate the parenting style/child obesity association*. Public Health Nutrition, 2010. **13**(8): p. 1237-1244.
397. El-Behadli, A.F., et al., *Maternal depression, stress and feeding styles: towards a framework for theory and research in child obesity*. British Journal of Nutrition, 2015. **113**: p. S55-S71.
398. Duarte, C.S., et al., *Maternal depression and child BMI: longitudinal findings from a US sample*. Pediatric Obesity, 2012. **7**(2): p. 124-133.
399. Mortensen, L.H., et al., *The social gradient in birthweight at term: quantification of the mediating role of maternal smoking and body mass index*. Human Reproduction, 2009. **24**(10): p. 2629-2635.
400. Goodman, A., D.L. Lamping, and G.B. Ploubidis, *When to Use Broader Internalising and Externalising Subscales Instead of the Hypothesised Five Subscales on the Strengths and Difficulties Questionnaire (SDQ): Data from British Parents, Teachers and Children*. Journal of Abnormal Child Psychology, 2010. **38**(8): p. 1179-1191.
401. Mellor, D. and M. Stokes, *The factor structure of the Strengths and Difficulties Questionnaire*. European Journal of Psychological Assessment, 2007. **23**(2): p. 105-112.
402. Ronning, J.A., et al., *The Strengths and Difficulties Self-Report Questionnaire as a screening instrument in Norwegian community samples*. European Child & Adolescent Psychiatry, 2004. **13**(2): p. 73-82.
403. Van Leeuwen, K., et al., *The Strengths and Difficulties Questionnaire in a community sample of young children in Flanders*. European Journal of Psychological Assessment, 2006. **22**(3): p. 189-197.

404. Ortuno-Sierra, J., et al., *The assessment of emotional and Behavioural problems: Internal structure of The Strengths and Difficulties Questionnaire*. International Journal of Clinical and Health Psychology, 2015. **15**(3): p. 265-273.
405. Niclasen, J., et al., *A confirmatory approach to examining the factor structure of the Strengths and Difficulties Questionnaire (SDQ): a large scale cohort study*. J Abnorm Child Psychol, 2013. **41**(3): p. 355-65.
406. Tobia, V., M.A. Gabriele, and G.M. Marzocchi, *The Italian Version of the Strengths and Difficulties Questionnaire (SDQ)Teacher: Psychometric Properties*. Journal of Psychoeducational Assessment, 2013. **31**(5): p. 493-505.
407. Ortuno-Sierra, J., R. Aritio-Solana, and E. Fonseca-Pedrero, *Mental health difficulties in children and adolescents: The study of the SDQ in the Spanish National Health Survey 2011-2012*. Psychiatry Research, 2018. **259**: p. 236-242.
408. Croft, S., et al., *Validity of the Strengths and Difficulties Questionnaire in Preschool-Aged Children*. Pediatrics, 2015. **135**(5): p. E1210-E1219.
409. McNeish, D. and M.G. Wolf, *Thinking twice about sum scores*. Behav Res Methods, 2020. **52**(6): p. 2287-2305.
410. DiStefano, C., M. Zhu, and D. Mindrilă, *Understanding and Using Factor Scores: Considerations for the Applied Researcher* Practical Assessment, Research & Evaluation, 2009. **Vol 14**(No 20 ).
411. Yang, H., *Factor Loadings*, in *Encyclopedia of Research Design*, N.J. Salkind, Editor. 2012 SAGE Publications, Inc: Thousand Oaks
412. Holgado-Tello, F.P., et al., *Polychoric versus Pearson correlations in exploratory and confirmatory factor analysis of ordinal variables*. Quality & Quantity, 2010. **44**(1): p. 153-166.
413. Knafl, G.J. and M. Grey, *Factor analysis model evaluation through likelihood cross-validation*. Statistical Methods in Medical Research, 2007. **16**(2): p. 77-102.
414. Hurley, A.E., et al., *Exploratory and confirmatory factor analysis: guidelines, issues, and alternatives*. Journal of Organizational Behavior, 1997. **18**(6): p. 667-683.
415. Heene, M., et al., *Sensitivity of SEM Fit Indexes With Respect to Violations of Uncorrelated Errors*. Structural Equation Modeling-a Multidisciplinary Journal, 2012. **19**(1): p. 36-50.
416. Hu, L.t. and P.M. Bentler, *Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives*. Structural Equation Modeling: A Multidisciplinary Journal 1999. **9**(1).
417. Schumacker, R.E. and R.G. Lomax, *A Beginner's Guide To Structural Equation Modeling*. 4th ed. 2016: Routledge.
418. Putnick, D.L. and M.H. Bornstein, *Measurement invariance conventions and reporting: The state of the art and future directions for psychological research*. Developmental Review, 2016. **41**: p. 71-90.
419. Chen, F.F., *Sensitivity of goodness of fit indexes to lack of measurement invariance*. Structural Equation Modeling-a Multidisciplinary Journal, 2007. **14**(3): p. 464-504.
420. Muthen, B. and T. Asparouhov, *New Methods for the Study of Measurement Invariance with Many Groups*, in *Mplus*. 2013: [www.statmodel.com](http://www.statmodel.com).
421. Gadermann A., G.M., Zumbo B.D, *Ordinal Alpha*, in *Encyclopedia of Quality of Life and Well-Being Research*, M. A.C, Editor. 2014: Springer, Dordrecht.
422. Anne M. Gadermann, Martin Guhn, and B.D. Zumbo, *Estimating ordinal reliability for Likert-type and ordinal item response data: A conceptual, empirical, and practical guide*. Practical Assessment, Research, and Evaluation, 2012. **Volume 17**: p. 1-13.
423. Chalmers, R.P., *On Misconceptions and the Limited Usefulness of Ordinal Alpha*. Educ Psychol Meas, 2018. **78**(6): p. 1056-1071.
424. C Fornell and D. Larcker, *Evaluating Structural Equation Models with Unobservable Variables and Measurement Error*. Journal of Marketing Research, 1981. **18**(1).

425. Muthén, L.K. and B.O. Muthén, *Mplus User's Guide. Eighth Edition.*, M. Muthén, Editor. 2017: Los Angeles, CA.
426. E McElroy, A.V., P Patalay, A Goodman, *Harmonisation of Mental Health Measures in the British Birth Cohorts*, CLOSER, Editor. 2020: CLOSER
427. Hair, J.F., Black, W.C., Babin, B.J., Anderson, R.E., Tatham, R.L., *Multivariate data analysis*. 7th, Ed. ed. Vol. Vol. 7. 2009, Pearson Prentice Hall Upper Saddle River, NJ.
428. UCLA. *Probit regression | Stata Annotated Output*. UCLA: Statistical Consulting Group. 2007 [cited 2022 23/01/22]; Available from: <https://stats.oarc.ucla.edu/stata/output/probit-regression/>.
429. Kline, R.B., *Principles and practice of structural equation modeling*. Third ed ed. 2011, Guilford: The Guilfor Press.
430. Ortuno-Sierra, J., et al., *New evidence of factor structure and measurement invariance of the SDQ across five European nations*. *European Child & Adolescent Psychiatry*, 2015. **24**(12): p. 1523-1534.
431. MacCallum, R.C., et al., *Sample size in factor analysis*. *Psychological Methods*, 1999. **4**(1): p. 84-99.
432. Pearce, A.G.Y.a.S., *A Beginner's Guide to Factor Analysis: Focusing on Exploratory Factor Analysis* *Tutorials in Quantitative Methods for Psychology*, 2013. **9**(2): p. p. 79-94.
433. Gosling, S.D., P.J. Rentfrow, and W.B. Swann, *A very brief measure of the Big-Five personality domains*. *Journal of Research in Personality*, 2003. **37**(6): p. 504-528.
434. Worthington, R.L. and T.A. Whittaker, *Scale development research - A content analysis and recommendations for best practices*. *Counseling Psychologist*, 2006. **34**(6): p. 806-838.
435. Borsboom, D., G.J. Mellenbergh, and J. van Heerden, *The concept of validity*. *Psychological Review*, 2004. **111**(4): p. 1061-1071.
436. Rudnev, M., et al., *Testing Measurement Invariance for a Second-Order Factor. A Cross-National Test of the Alienation Scale*. *Methods Data Analyses*, 2018. **12**(1): p. 47-76.
437. Brown, T.A., *Confirmatory factor analysis for applied research*. 2nd ed ed. 2015, New York: Guilford.: The Guilford Press.
438. Cleridou, K., P. Patalay, and P. Martin, *Does parent-child agreement vary based on presenting problems? Results from a UK clinical sample*. *Child and Adolescent Psychiatry and Mental Health*, 2017. **11**.
439. Johnston, D.W., et al., *The income gradient in childhood mental health: all in the eye of the beholder?* *Journal of the Royal Statistical Society Series a-Statistics in Society*, 2014. **177**(4): p. 807-827.
440. Muthén, L.K. and B.O. Muthén, *Mplus: Statistical Analysis With Latent Variables: User's Guide*. 1998-2017: Los Angeles, CA: Muthén & Muthén.
441. Jones, A.P., et al., *Environmental supportiveness for physical activity in English schoolchildren: a study using Global Positioning Systems*. *Int J Behav Nutr Phys Act*, 2009. **6**: p. 42.
442. Tabachnick, B.G. and L.S. Fidell, *Using Multivariate Statistics*. Fourth Edition ed. 2001, Needham Heights, MA: Allyn & Bacon.
443. Baron, R.M. and D.A. Kenny, *The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations*. *J Pers Soc Psychol*, 1986. **51**(6): p. 1173-82.
444. Mackinnon, D.P. and A.J. Fairchild, *Current Directions in Mediation Analysis*. *Curr Dir Psychol Sci*, 2009. **18**(1): p. 16.
445. Hayes, A.F. and K.J. Preacher, *Quantifying and Testing Indirect Effects in Simple Mediation Models When the Constituent Paths Are Nonlinear*. *Multivariate Behav Res*, 2010. **45**(4): p. 627-60.
446. MacKinnon, D.P., A.J. Fairchild, and M.S. Fritz, *Mediation analysis*. *Annu Rev Psychol*, 2007. **58**: p. 593-614.

447. Imai, K., L. Keele, and D. Tingley, *A general approach to causal mediation analysis*. Psychol Methods, 2010. **15**(4): p. 309-34.
448. Allison, P.D., *Handling Missing Data by Maximum Likelihood*, in *SAS Global Forum 2012*, S. Horizons, Editor. 2012: Haverford, PA, USA.
449. Schober, P., C. Boer, and L.A. Schwarte, *Correlation Coefficients: Appropriate Use and Interpretation*. Anesthesia and Analgesia, 2018. **126**(5): p. 1763-1768.
450. Patalay, P. and C.A. Hardman, *Comorbidity, Codevelopment, and Temporal Associations Between Body Mass Index and Internalizing Symptoms From Early Childhood to Adolescence*. Jama Psychiatry, 2019. **76**(7): p. 721-729.
451. Noonan, R. and S. Fairclough, *Is there a deprivation and maternal education gradient to child obesity and physical activity in England?* Journal of Physical Activity & Health, 2018. **15**(10): p. S65-S65.
452. Geoffroy, M., L. Li, and C. Power. *Psychological distress and body mass index: comorbidity and direction of associations over the life-course*. 2011. J Epidemiol Community Health
453. White, B., et al., *Childhood psychological function and obesity risk across the lifecourse: findings from the 1970 British Cohort Study*. Int J Obes (Lond), 2012. **36**(4): p. 511-6.
454. Jaaskelainen, A., et al., *Stress-related eating, obesity and associated behavioural traits in adolescents: a prospective population-based cohort study*. BMC Public Health, 2014. **14**: p. 321.
455. Laitinen, J., E. Ek, and U. Sovio, *Stress-related eating and drinking behavior and body mass index and predictors of this behavior*. Prev Med, 2002. **34**(1): p. 29-39.
456. Hemmingsson, E., *A new model of the role of psychological and emotional distress in promoting obesity: conceptual review with implications for treatment and prevention*. Obes Rev, 2014. **15**(9): p. 769-79.
457. Kwon, S., et al., *Effects of Adiposity on Physical Activity in Childhood: Iowa Bone Development Study*. Medicine and Science in Sports and Exercise, 2011. **43**(3): p. 443-448.
458. Zhu, W.F., et al., *Is adiposity associated with objectively measured physical activity and sedentary behaviors in older adults?* BMC Geriatrics, 2020. **20**(1).
459. Brandt, V., P. Patalay, and J.K.A. Koerner, *Predicting ADHD symptoms and diagnosis at age 14 from objective activity levels at age 7 in a large UK cohort*. European Child & Adolescent Psychiatry, 2021. **30**(6): p. 877-884.
460. De Crescenzo, F., et al., *The use of actigraphy in the monitoring of sleep and activity in ADHD: A meta-analysis*. Sleep Medicine Reviews, 2016. **26**: p. 9-20.
461. Prasad, V., et al., *Attention-deficit/hyperactivity disorder: variation by socioeconomic deprivation*. Archives of Disease in Childhood, 2019. **104**(8): p. 802-805.
462. Wilkinson, R.G. and K.E. Pickett, *The problems of relative deprivation: Why some societies do better than others*. Social Science & Medicine, 2007. **65**(9): p. 1965-1978.
463. Kim, S., B.J. Lee, and S.W. Kim, *Poverty, income inequality and externalised problem behaviours of children: an empirical study in Korea*. Asia Pacific Journal of Social Work and Development, 2014. **24**(3): p. 158-170.
464. Nieuwenhuis, J., et al., *Being Poorer Than the Rest of the Neighborhood: Relative Deprivation and Problem Behavior of Youth*. Journal of Youth and Adolescence, 2017. **46**(9): p. 1891-1904.
465. Elgar, F.J., et al., *Relative deprivation and risk factors for obesity in Canadian adolescents*. Soc Sci Med, 2016. **152**: p. 111-8.
466. Hoen, P.W., et al., *Positive Affect and Survival in Patients With Stable Coronary Heart Disease: Findings From the Heart and Soul Study*. Journal of Clinical Psychiatry, 2013. **74**(7): p. 716-722.
467. Schipperijn, J., et al., *Associations between physical activity and characteristics of urban green space*. Urban Forestry & Urban Greening, 2013. **12**(1): p. 109-116.

468. Schipperijn, J., et al., *Factors influencing the use of green space: Results from a Danish national representative survey*. Landscape and Urban Planning, 2010. **95**(3): p. 130-137.
469. Libuy, N., et al., *Fast Food and Childhood Obesity: Evidence from the UK (Working Paper)*, C.f.L. Studies, Editor. 2021: UCL Institute of Education.
470. Green, M., et al., *The Association between Fast Food Outlets and Overweight in Adolescents Is Confounded by Neighbourhood Deprivation: A Longitudinal Analysis of the Millennium Cohort Study*. Preprints, 2021(2021090434 ).
471. Dennis, M. and P. James, *Evaluating the relative influence on population health of domestic gardens and green space along a rural-urban gradient*. Landscape and Urban Planning, 2017. **157**: p. 343-351.
472. Betts, K.S., et al., *The Relationship between Maternal Depressive, Anxious, and Stress Symptoms during Pregnancy and Adult Offspring Behavioral and Emotional Problems*. Depression and Anxiety, 2015. **32**(2): p. 82-90.
473. Sutin, A.R., H.A. Flynn, and A. Terracciano, *Maternal cigarette smoking during pregnancy and the trajectory of externalizing and internalizing symptoms across childhood: Similarities and differences across parent, teacher, and self reports*. Journal of Psychiatric Research, 2017. **91**: p. 145-148.
474. Ulmer-Yaniv, A., et al., *Maternal depression alters stress and immune biomarkers in mother and child*. Depression and Anxiety, 2018. **35**(12): p. 1145-1157.
475. Brion, M.J., et al., *Maternal Smoking and Child Psychological Problems: Disentangling Causal and Noncausal Effects*. Pediatrics, 2010. **126**(1): p. E57-E65.
476. Indredavik, M.S., et al., *Prenatal smoking exposure and psychiatric symptoms in adolescence*. Acta Paediatrica, 2007. **96**(3): p. 377-382.
477. Silverwood, R.J., et al., *Association between Younger Age When First Overweight and Increased Risk for CKD*. Journal of the American Society of Nephrology, 2013. **24**(5): p. 813-821.
478. Islam, M.T., et al., *Life-course trajectories of body mass index and subsequent cardiovascular risk among Chinese population*. Plos One, 2019. **14**(10).
479. Stanford, K.I. and L.J. Goodyear, *Exercise and type 2 diabetes: molecular mechanisms regulating glucose uptake in skeletal muscle*. Advances in Physiology Education, 2014. **38**(4): p. 308-314.
480. Gray, L.A., et al., *Family lifestyle dynamics and childhood obesity: evidence from the millennium cohort study*. BMC Public Health, 2018. **18**(1): p. 500.
481. Jivraj, S., et al., *Life Course Neighbourhood Deprivation and Self-Rated Health: Does It Matter Where You Lived in Adolescence and Do Neighbourhood Effects Build Up over Life?* Int J Environ Res Public Health, 2021. **18**(19).
482. Mason, K.E., et al., *Impact of cuts to local government spending on Sure Start children's centres on childhood obesity in England: a longitudinal ecological study*. J Epidemiol Community Health, 2021. **75**(9): p. 860-866.
483. Hunter, P., *The local Living Wage dividend: An analysis of the impact of the Living Wage on ten city regions*. 2018: The Smith Institute.
484. Watson, B., M. Guettabi, and M. Reimer, *Universal Cash Transfers Reduce Childhood Obesity Rates*. SSRN, 2019.
485. Bloomer, E., *Health equity briefing 6 - Local action on health inequalities: Health inequalities and the living wage*, IHE, Editor. 2014: Public Health England.
486. Alliot, O., et al., *Do adolescents' experiences of the barriers to and facilitators of physical activity differ by socioeconomic position? A systematic review of qualitative evidence*. Obes Rev, 2021.
487. Lee, A.C., H.C. Jordan, and J. Horsley, *Value of urban green spaces in promoting healthy living and wellbeing: prospects for planning*. Risk Manag Healthc Policy, 2015. **8**: p. 131-7.

488. Hands, A., et al., *Improving access to greenspace: A new review for 2020*. 2020: Public Health England
489. Adams, J., et al., *Why Are Some Population Interventions for Diet and Obesity More Equitable and Effective Than Others? The Role of Individual Agency*. Plos Medicine, 2016. **13**(4).
490. McGill, R., et al., *Are interventions to promote healthy eating equally effective for all? Systematic review of socioeconomic inequalities in impact (vol 15, 457, 2015)*. BMC Public Health, 2015. **15**.
491. Beauchamp, A., et al., *The effect of obesity prevention interventions according to socioeconomic position: a systematic review*. Obesity Reviews, 2014. **15**(7): p. 541-554.
492. Morris, M.A., et al., *What is the cost of a healthy diet? Using diet data from the UK Women's Cohort Study*. Journal of Epidemiology and Community Health, 2014. **68**(11): p. 1043-1049.
493. Vogel, C., et al., *Examination of how food environment and psychological factors interact in their relationship with dietary behaviours: test of a cross-sectional model*. International Journal of Behavioral Nutrition and Physical Activity, 2019. **16**.
494. Marra, M., et al., *Assessment of Body Composition in Health and Disease Using Bioelectrical Impedance Analysis (BIA) and Dual Energy X-Ray Absorptiometry (DXA): A Critical Overview*. Contrast Media & Molecular Imaging, 2019.
495. Ho-Pham, L.T., U.D.T. Nguyen, and T.V. Nguyen, *Association Between Lean Mass, Fat Mass, and Bone Mineral Density: A Meta-analysis*. Journal of Clinical Endocrinology & Metabolism, 2014. **99**(1): p. 30-38.
496. Littlejohns, T.J., et al., *The UK Biobank imaging enhancement of 100,000 participants: rationale, data collection, management and future directions*. Nat Commun, 2020. **11**(1): p. 2624.
497. Hill, S.E., et al., *Differences in Obesity Among Men of Diverse Racial and Ethnic Background*. American Journal of Mens Health, 2017. **11**(4): p. 984-989.
498. Barrington, D.S., S.A. James, and D.R. Williams, *Socioeconomic Correlates of Obesity in African-American and Caribbean-Black Men and Women*. Journal of Racial and Ethnic Health Disparities, 2020.
499. Smith, N.R., Y.J. Kelly, and J.Y. Nazroo, *The effects of acculturation on obesity rates in ethnic minorities in England: evidence from the Health Survey for England*. Eur J Public Health, 2012. **22**(4): p. 508-13.
500. McLaughlin, K.A., L.M. Hilt, and S. Nolen-Hoeksema, *Racial/ethnic differences in internalizing and externalizing symptoms in adolescents*. J Abnorm Child Psychol, 2007. **35**(5): p. 801-16.
501. Goodman, A., V. Patel, and D.A. Leon, *Child mental health differences amongst ethnic groups in Britain: a systematic review*. BMC Public Health, 2008. **8**: p. 258.
502. Belhadj Kouider, E., U. Koglin, and F. Petermann, *Emotional and behavioral problems in migrant children and adolescents in Europe: a systematic review*. Eur Child Adolesc Psychiatry, 2014. **23**(6): p. 373-91.
503. Roe, J., P.A. Aspinall, and C.W. Thompson, *Understanding Relationships between Health, Ethnicity, Place and the Role of Urban Green Space in Deprived Urban Communities*. International Journal of Environmental Research and Public Health, 2016. **13**(7).
504. McEachan, R.R.C., et al., *Availability, use of, and satisfaction with green space, and children's mental wellbeing at age 4 years in a multicultural, deprived, urban area: results from the Born in Bradford cohort study*. Lancet Planet Health, 2018. **2**(6): p. e244-e254.
505. Bridger Staatz, C. and D. Bann, *Diet – Opportunities for Data Collection*, U.I.o.E. Centre for Longitudinal Studies, Editor. 2021.
506. Sutin, A.R., et al., *Personality and obesity across the adult life span*. J Pers Soc Psychol, 2011. **101**(3): p. 579-92.
507. Gerlach, G., S. Herpertz, and S. Loeber, *Personality traits and obesity: a systematic review*. Obesity Reviews, 2015. **16**(1): p. 32-63.



508. Wimmelman, C.L., et al., *Associations of Personality with Body Mass Index and Obesity in a Large Late Midlife Community Sample*. *Obes Facts*, 2018. **11**(2): p. 129-143.
509. Chapman, B.P., et al., *Personality, socioeconomic status, and all-cause mortality in the United States*. *Am J Epidemiol*, 2010. **171**(1): p. 83-92.
510. Pluess, M. and M. Bartley, *Childhood conscientiousness predicts the social gradient of smoking in adulthood: a life course analysis*. *J Epidemiol Community Health*, 2015. **69**(4): p. 330-8.
511. Elliot, A.J., N.A. Turiano, and B.P. Chapman, *Socioeconomic Status Interacts with Conscientiousness and Neuroticism to Predict Circulating Concentrations of Inflammatory Markers*. *Ann Behav Med*, 2017. **51**(2): p. 240-250.
512. Hawkins, S.S., et al., *Regional differences in overweight: an effect of people or place?* *Archives of Disease in Childhood*, 2008. **93**(5): p. 407-413.