A Public Health Approach to Childhood Obesity: The Role of Econometrics

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ABSTRACT

The increasing prevalence of childhood obesity in the United Kingdom is of great interest to public health policy makers and guidance developers such as NICE, as well as the general public. In order to develop effective policies, the causes of childhood obesity need to be better understood. Analysing the Millennium Cohort Survey, this thesis uses econometric techniques to investigate the relationships between childhood obesity, family lifestyle behaviours and child health outcomes.

The first empirical chapter investigates the causal effects of breastfeeding behaviours on obesity during early childhood, a topic which has been of particular interest to NICE. There is a small but statistically significant influence suggesting that breastfeeding should be one part of a wider effort to reduce obesity by influencing lifestyle, such as Change4Life. These effects appear to increase in magnitude and significance as children get older suggesting that the dynamics of lifestyle and childhood obesity should be investigated further.

The second empirical chapter investigates the relationship between underlying family lifestyle and childhood obesity using a dynamic framework. Childhood obesity is one of the strongest predictors of obesity in adulthood. If lifestyle is learnt in childhood and is persistent then this could exacerbate the problem of childhood obesity. Amongst other findings, this chapter concludes that childhood weight status significantly depends on family lifestyle.

The final empirical chapter extends this model and allows the effects of both family lifestyle and underlying health on childhood obesity to be investigated. Childhood weight is a significant outcome measure of underlying child health after the age of five. The results suggest that policies should target various lifestyle behaviours simultaneously by improving underlying lifestyle through education and improved understanding and enabling families to make positive changes.

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I am grateful to The Centre for Longitudinal Studies, Institute of Education for the use of these data and to the UK Data Archive and Economic and Social Data Service for making them available. However, they bear no responsibility for the analysis or interpretation of these data.

[1] University of London. UCL Institute of Education. Centre for Longitudinal Studies, Millennium Cohort Study. Colchester, Essex: UK Data Archive, January 2012.

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2SLS	Two-Stage Least Squares
AIC	Akaike Information Criterion
ALSPAC	Avon Longitudinal Study of Pregnancy and Children
ANOVA	Analysis of Variance
AR	Autoregressive
AROW	At Risk of Overweight
ATE	Average Treatment Effect
ATT	Average Treatment Effect on the Treated
ATU	Average Treatment Effect on the Untreated
BHPS	British Household Panel Survey
BIC	Bayesian Information Criterion
BIV	Biological Implausible Value
BMI	Body Mass Index
CDC	Centre for Disease Control and Prevention
CDF	Cumulative Distribution Function
CDS	Child Development Supplement
CHQ	Childhood Health Questionnaire
CIA	Conditional Independence Assumption
CSDH	Commission of Social Determinants of Health
CVD	Cardiovascular Disease
DH	Department of Health
EFA	Exploratory Factor Analysis
GAM	General Additive Model
GEE	Generalised Estimating Equations
GPS	Generalised Propensity Score
HDA	Health Development Agency
HSE	Health Survey for England
HTA	Health Technology Appraisal
IID	Independently and Identically Distributed
IOTF	International Obesity Taskforce
IQ	Intelligence Quotient
IV	Instrumental Variable
kg	kilograms
LR	Likelihood Ratio
m	meters
MCS	Millennium Cohort Study
MTA	Multiple Technology Appraisal
NCDS	National Child Development Study
NCHS	National Center for Health Statistics
NHS	National Health Service
NICE	National Institute of Health and Care Excellence
NN	Nearest Neighbour
NS-SEC	National Statistics Socioeconomic Classification
NVQ	National Vocational Qualification
OLS	Ordinary Least Squares
PHE	Public Health England
PROBIT	Promotion of Breastfeeding Intervention Trial
PSM	Propensity Score Matching
RCPCH	Royal College of Paediatric and Child Health
RCT	Randomised Controlled Trial
RESET	Regression Equation Specification Error Test
SAH	Self-assessed Health

SACN	Scientific Advisory Committee on Nutrition
SES	Socioeconomic Status
STA	Single Technology Appraisal
UK	United Kingdom
UNICEF	United Nations Children's Fund
US	United States
WAPCS	Western Australian Pregnancy Cohort Study
WHO	World Health Organisation
ZINB	Zero-Inflated Negative Binomial
ZIP	Zero-Inflated Poisson

I. INTRODUCTION AND RATIONALE

Childhood obesity has been increasing in prevalence in the United Kingdom (UK) over the past three decades, causing high levels of public interest as well as government concern. In order to develop effective policies, the causes and consequences of obesity during childhood need to be fully understood. Surprisingly, relatively little is known about the effects of childhood obesity caused by early childhood influences. Childhood obesity is also a growing public health concern and due to the numbers of obese children a population based public health approach is needed rather than an individual clinical approach.

The World Health Organisation (WHO) defines public health as the prevention of disease, promotion of health and prolonging of life among the population as a whole. The aim of any public health approach is to benefit the population of interest, in this case, as many children in the UK as possible, by tackling the underlying risk factors of a disease at a population level. In England, it is the role of the National Institute of Health and Care Excellence (NICE) to create public health guidance[2]. This guidance is developed independently of the Government by committees of experts and members of the public who review the available evidence in order to make recommendations. NICE produce advice and guidance on a range of public health issues aimed at the public, the National Health Service (NHS), other health bodies and local authorities as well as other organisation in both the public, private and voluntary sectors[3].

The majority of public health guidance developed by NICE uses economic models which predict future health or economic outcomes as consequences of different potential interventions, actions or policies. These include cost-effectiveness models usually based on the principles of cost utility analysis which aim to identify interventions that will have benefits beyond their cost of implementation. These economic models are evidence based wherever possible but inevitably also rely on a number of assumptions where evidence is lacking. Where needed, these assumptions are tested using sensitivity analysis. They collate clinical, medical, economic and policy related evidence from a range of sources. Evidence is often taken from clinical trials and policy evaluation or econometric studies. Much of this evidence is of short-term outcomes; for example, trials do not routinely collect information longer than two years after an intervention. However, for policy purposes it is just as important, if not more, to understand how these effects come about

[2] NICE primarily produces guidance for England, but has agreements to provide certain guidance and recommendations to Scotland, Wales and Northern Ireland also.
[3] NICE also provides guidance on other health and care related topics, such as health technology appraisals (HTAs) and multiple technology appraisals (MTAs) as well as clinical and social care guidelines.

and develop over time. To overcome this problem, econometric models can be used to provide more long-term evidence. NICE guidance and recommendations can benefit greatly from this knowledge, particularly when assessing cost-effectiveness which requires long-term outcomes to be predicted. Where this evidence is not available, economic models must extrapolate and/or make assumptions. The more evidence available on longer-term outcomes relating to any public health guidance, the more accurate the economic assessment or cost-effective analysis can be.

An example of a 'Public Health Approach' is defined by WHO (2015) as a four-step iterative approach. The four steps are outlined as follows:

1. *Surveillance.* What is the problem that we aim to solve? In this case it is childhood obesity.

2. *Determinants.* What are the causes? Are there influences which protect against childhood obesity or which are risk factors for childhood obesity?

3. *Informing interventions.* What policies work and who benefits most from each of these policies? What types of children will benefit most from the proposed interventions?

4. *Implementation.* Carrying out the interventions on a larger scale in order to prevent childhood obesity at a national or international level.

This thesis aims to contribute to steps two and three of this public health approach. The scope of this thesis does not include any analysis which addresses steps one or four, although these are discussed in the introductory sections and literature reviews.

Current practice in public health broadly focuses on evidence relying expert opinion or data from other countries or unrepresentative samples. This thesis uses a large nationally representative dataset which allows analysis to be applicable to the UK population and enables a variety of children with different characteristics to be investigated. The role of econometrics in producing evidence for public health guidance is two-fold. On one hand it can inform public health recommendations and directly influence expert committees to help them in the development of public health guidance. This is generally done by estimating econometric models using observational data in order to identify the effects of past interventions or treatments, usually, but not always, on a single outcome. On the other hand, econometric analysis can be used to inform the economic models which are

then used to create guidance or recommend interventions. Any type of econometric model can be used to feed into these economic models, but certain econometric models, known as structural models, are particularly useful. This is because they estimate systems of equations as well as the correlations between them and thus any subsequent economic model needs to make no assumptions about these correlations. More complex econometric analysis involving multiple outcomes over a period of time using longitudinal data can provide longer-term evidence. This type of model is known in statistics as a structural model and can lead to more solid and robust public health interventions being recommended. These structural models pull together related concepts providing more evidence for economic models and guidance developers. They also allow the distributions of treatment effects to be given more consideration (Carneiro, Hansen, & Heckman, 2003; Heckman & Urzúa, 2010). This means that rather than identifying the average effect of an intervention on a chosen outcome measure or a set of outcome measures independently, it is possible to explore how different individuals might be influenced differentially by potential interventions, allowing different parameter estimates to answer different policy questions using a single model. The need for more robust long-term evidence to feed into economic or cost-effectiveness models is growing as the NHS budget is being increasingly stretched. Therefore it is more important as ever that long-term effects are identified.

There are a number of examples of econometric evidence which have been used in public health guidance over recent years. For example, Pilgrim *et al*. (2010) carried out a systematic review of econometric studies which investigated the long-term influences of teenage pregnancy. Their review only included studies which used econometric techniques which controlled for unobservable confounders. This review emphasised the benefits of long-term outcomes being investigated and used in a public health setting. However, it also highlighted the lack of dynamic models used to investigate outcomes over time; none of the identified studies used outcomes which were measured at more than one point in time. Similarly, most of the studies identified by this review estimated population average effects which limits the evidence available for economic models and does not allow the distribution of effects to be investigated. Other studies identified in this review estimated the local average treatment effect, which is also problematic for NICE (Faria *et al*., 2015) because these studies only estimate the effect for a subgroup of the population. This review of econometric studies was used as evidence in public health guidance on contraception for young people (NICE, 2014b).

Other studies which have used econometric methods and which have been used as evidence to support public health guidance include Brennan *et al*. (2008) and Brown & Taylor (2008). Brennan *et al*. (2008) used econometric analysis to estimate the effects of alcohol pricing and promotion policies on underage drinking, binge drinking and harmful drinking. Their intention was to help inform an economic model where a lack of evidence was identified; previously, there was no econometric analysis in this area which was fit for purpose. In this report, the authors investigated the price elasticities of different types of alcohol using cross-sectional data. Their results were used to inform an economic model which predicted the effects of different alcohol pricing policies. This economic model was then used to update evidence in public health guidance on alcohol-use disorders (NICE, 2014a). However, the economic model was limited in that there was a lack of evidence on long-term influences and as a result the economic model was unable to estimate the long-term effects of alcohol pricing policies without the need for extrapolation and additional assumptions.

Brown & Taylor (2008) carried out econometric analysis on the long-term effects of bullying during childhood on educational attainment and income. Although this article was not originally intended to be used as evidence for NICE or other public health bodies, it nevertheless had an impact on public health guidance. Evidence from this study was used in an economic model by Hummel *et al*. (2009) to provide NICE with an economic cost-effectiveness model for emotional and social wellbeing interventions in secondary schools (NICE, 2012b). Although this study used static models and did not investigate how these influences came about, it provided an important addition to the existing evidence which was otherwise limited to short-term outcomes.

More complex econometric models have also been drawn upon to feed into economic models used in NICE guidance. For example, public health guidance on childhood development (NICE, 2012b) relied heavily on an econometric structural model developed by Hernandez Alava *et al*. (2011) to investigate long-term outcomes which resulted from childhood cognitive, social and emotional development. This report used a range of datasets, including the Millennium Cohort Study (MSC) for which a variety of outcomes were jointly analysed. These outcomes included both child outcome measures (in the MCS) and adult outcomes (in other datasets) and depended on either behaviour and/or cognition in children at different ages. This information was then incorporated into an economic model developed by Hummel *et al*. (2011) allowing the influence of social and emotional wellbeing interventions on outcome measures at age five and on adult

outcomes to be predicted. The dynamic nature of these models provide more information than simpler static models and enable more than one equation to be jointly estimated at multiple time points and allow the analysis of a range of outcomes simultaneously.

This thesis uses a range of econometric techniques to investigate the relationships between childhood obesity, family lifestyle behaviours and child health outcomes. It aims to identify the causes of childhood obesity during early childhood in order to inform policy and tackle the childhood obesity epidemic. It investigates childhood obesity using methods which have not before been used in these settings in an attempt to identify causal relationships. In addition, many of the parameters investigated in this thesis have not previously been considered. This thesis consists of three distinct but related empirical chapters, each analysing data from the Millennium Cohort Study (Centre for Longitudinal Studies, 2000-2008). Each of the chapters have important policy implications and each could be used to enrich future public health guidance provided by NICE and add to the public health debate.

Chapter II investigates the effects of breastfeeding behaviours on childhood obesity throughout the early years of life, using a variety of econometric models. It uses a number of models in order to find the most appropriate set of assumptions for this empirical problem and to check the robustness of the results. The chapter aims to identify causal influences with the intention of informing public health guidance development committees such as those interested in producing guidance to reduce childhood obesity or increase breastfeeding participation. For example, this type of econometric analysis could have been beneficial to committees developing recent public health guidance on how to improve lifestyle to prevent and reduce childhood obesity (NICE, 2013b), tackle obesity in local communities (NICE, 2012c) and improve maternal and child nutrition (NICE, 2008). In addition, any results from this chapter also have the potential to be used in economic models for obesity.

Chapter III and Chapter IV use structural models to investigate the underlying causes of childhood obesity. Chapter III explores the causal influence of underlying family lifestyle on childhood obesity and how this relationship develops over time. Chapter IV introduces underlying childhood health to the model used in Chapter III in order to determine the extent to which poor health during childhood influences childhood obesity. In addition to the contributions to the public health debate described above for Chapter II, these two empirical chapters have the potential to provide economic models with a wealth of

information about a number of different outcomes, over and above childhood obesity. For example, the structural model in Chapter III jointly estimates the weight status of the child and of their parents as well as other outcome measures and as a result, findings from this study could provide important evidence in future public health guidance on obesity in a family context. Similarly, variables relating to diet, exercise and a variety of childhood health conditions are estimated by these models highlighting the wide range of public health guidance which could benefit from these structural models.

Results from the thesis suggest that longer breastfeeding durations reduce childhood BMI and the likelihood of childhood obesity. These effects become more apparent as children get older. The analysis reveals that childhood weight is strongly influenced by family lifestyle which also has strong influences on parental weight status, in particular maternal weight status. Childhood weight is causally influenced by underlying health but not to the same extent as family lifestyle. The results suggest that policies should focus on a wide range of lifestyle behaviours simultaneously by enabling families to make changes and educating them to understand why healthy lifestyles are important and how they impact on health and adiposity. By targeting disadvantaged families, socioeconomic inequalities in health and obesity prevalence during childhood could be reduced.

The remainder of this chapter discusses the recent trends in obesity in the UK. It also explains the reasons for using childhood obesity and overweight definitions which are different to adult definitions. It illustrates the need for high quality research in the area of childhood obesity, obesity policies which are already in place in the UK and describes what this thesis will do to add to the existing literature and contribute to knowledge in this area.

1.1 Obesity and Overweight in Adults

1.1.1 Measuring Adiposity in Adults

The most commonly used and accepted measurement of adult adiposity is Body Mass Index (BMI), which calculated using weight in kilograms (kg) and height in meters (m),

$$BMI = \frac{weight\ (kg)}{height\ (m^2)}. \qquad \text{(I.1)}$$

BMI can then be used to categorise adult individuals into different weight status; for example, underweight, normal weight, overweight and obese. The WHO classifications of weight status in adults are shown in Table I-1.

Table I-1: WHO Adult BMI Thresholds and Weight Categories

Body Mass Index (BMI)	Weight Status
BMI < 18.5	Underweight
18.5 ≤ BMI < 25	Normal weight
25 ≤ BMI < 30	Overweight
30 ≤ BMI < 35	Obese class I
35 ≤ BMI < 40	Obese class II
BMI ≥ 40	Obese class III

Source: World Health Orgnaisation (2011c). Notes: Definitions of weight status in adults.

Definitions of overweight and obesity are designed to identify individuals carrying excess body fat. According to the Department of Health (DH) (2010), an acceptable level of body fat in adults differs between individuals depending on their height and sex. However, neither BMI nor the most commonly used classifications for overweight or obesity in adults take sex into account. Greene *et al*. (2008) explained that the current BMI boundaries do not give the correct evaluation of adiposity of certain types of people. For example, athletic individuals are likely to have more heavy muscle and might fall into the overweight category with much less body fat than someone with a normal build. Similarly, elderly people are likely to have less muscle and therefore might be miscategorised in a lower BMI category when they experience the same health risks as overweight or obese individuals. Individuals misclassified as normal could potentially have health risks which are being ignored. Despite this, BMI remains the most widely used measure of adult adiposity and no better alternative has yet been agreed upon.

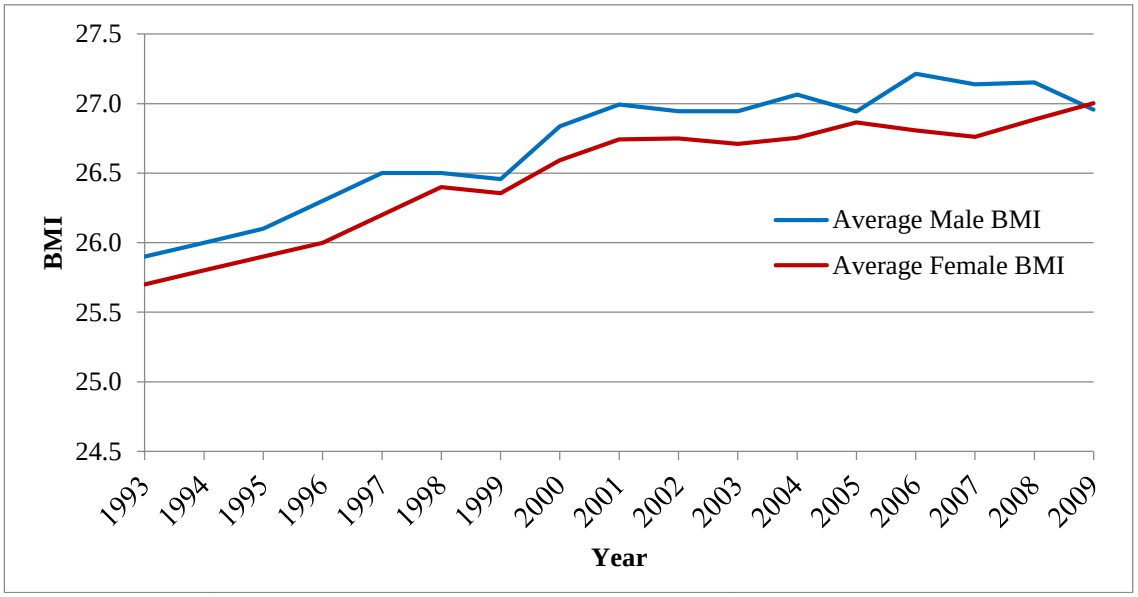
1.1.2 Trends in Adult Adiposity

In 2011, WHO (2011a) estimated that 67.8% and 63.8% of male and female UK adults, respectively, were overweight and 23.7% and 26.3%, respectively, were obese. Obesity has become one of the biggest health problems faced by developed countries and the rate at which obesity is continuing to increase remains alarming. The past twenty years has seen obesity double worldwide and in 2008 an estimated 1.5 billion adults worldwide were classified as overweight or obese. Obesity during childhood has been repeatedly found to be one of the strongest predictors of obesity in adulthood. Therefore, learning

how to tackle obesity in early life is important in the fight against obesity in the entire population.

Using data from the Health Survey for England (HSE) accessed through the National Health Service (NHS) Information Centre website (2008), Figure I-1, Figure I-2 and Figure I-3 show the evolution of body mass index (BMI) in the population of England over recent years. Figure I-1 shows the increase in mean BMI in England for both male and female adults since 1993. The average BMI has steadily increased over this short period of time from under 26 to over 27 and in 2009 the average BMI among women surpassed that of men for the first time; this could be of particular importance to policy makers if the reasons for this change are identified. For example, it could be a result of lifestyle changes in men and women. Figure I-1 shows that since 1993, the average BMI for both men and women has remained above 25, the threshold for the definition of overweight.

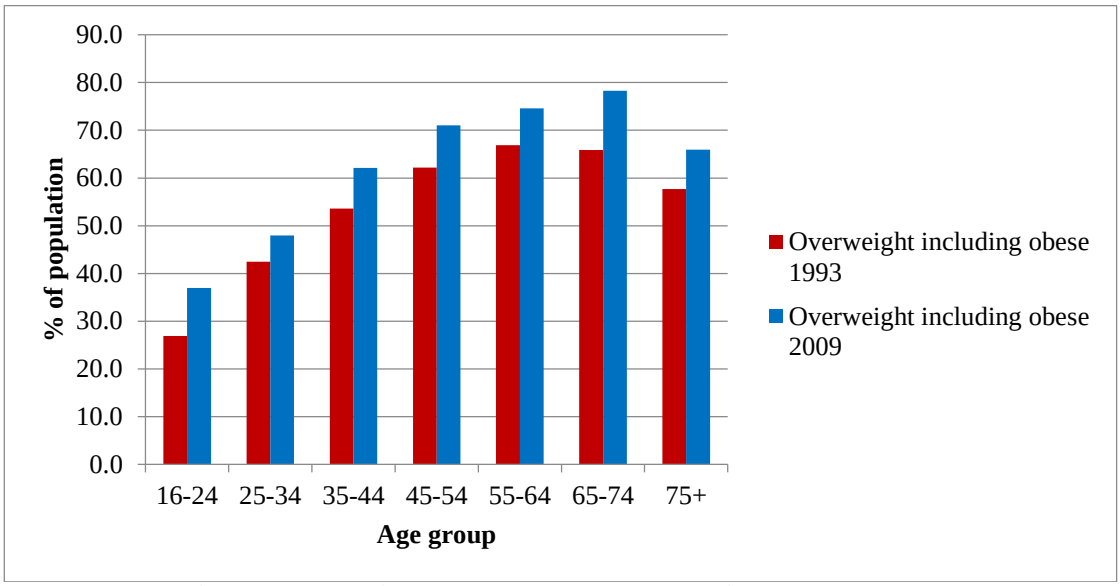
Figure I-1: Average BMI of English Males and Females (1993-2009)



Source: Health Survey for England, NHS Information Centre website, (The NHS Information Centre 2008). Notes: Mean BMI of English Male and Female Adults over the age of sixteen years between 1993 and 2009.

Figure I-2 compares the percentage of adults in England who were considered overweight or obese in 1993 and 2009. During these years, overweight and obesity increased in adults of all ages. In both 1993 and 2009, there is a peak in the population of overweight adults between their fifties and sixties before it decreases in older adults. This peak occurs at an older age in 2009 than in 1993, possibly due to increases in life expectancy between the two periods and the worsening of the obesity epidemic.

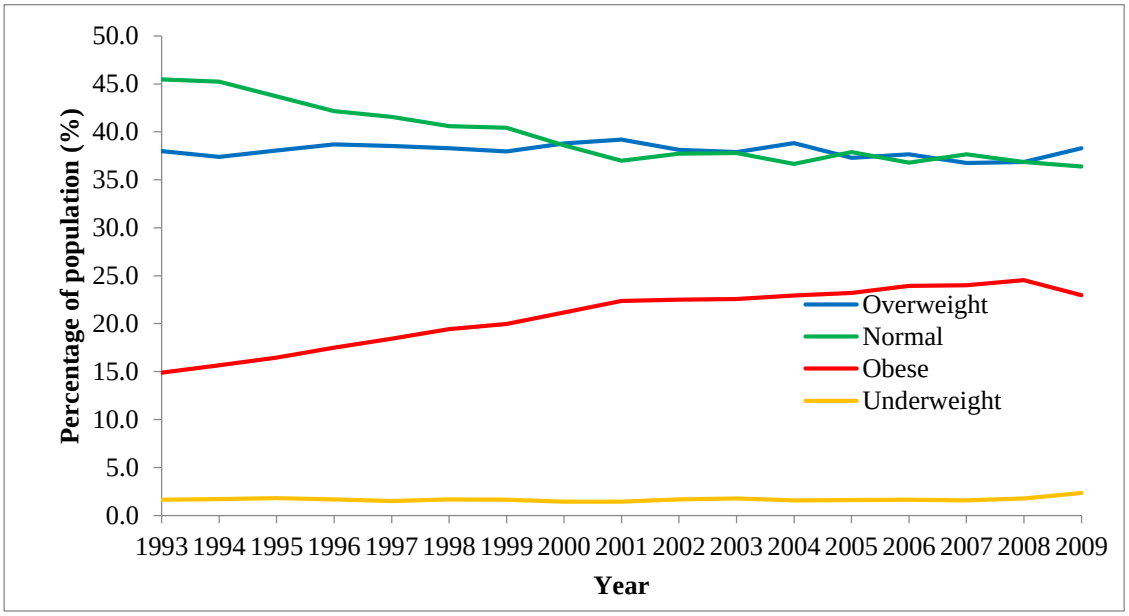
Figure I-2: Percentage of English Adults Overweight or Obese (1993 and 2009)



Source: Health Survey for England, NHS Information Centre website, (The NHS Information Centre 2008). Notes: Percentage of English adults (sixteen years or over) which are considered overweight or obese between 1993 and 2009.

Figure I-3 shows the changes in the percentage of English adults in each weight category between 1993 and 2009. The percentage of underweight and overweight adults remained relatively stable over time, at around 1.5% and 38%, respectively. However, while the percentage of normal weight adults decreased, the percentage of obese adults increased at a similar rate. By 2000, fewer adults were considered to have a healthy weight than were considered to be overweight, suggesting that overweight is becoming 'the norm'.

Figure I-3: Percentage of English Adults by BMI Category (1993 - 2009)



Source: Health Survey for England, NHS Information Centre website, (The NHS Information Centre 2008). Notes: Percentage of English adults (sixteen years and over) by weight status between 1993 and 2009.

Foresight (2008), a scientific think tank which advises the UK government, predicted that, by the year 2050, 60% of UK men and 50% of UK women will be obese, along with 25% of UK children if no action is taken. This massive rise in obesity over a relatively short period of time would place a large burden on the NHS and UK economy. The economic cost of obesity to the wider economy, such as through a loss of productivity is estimated to be £15.6 billion pounds (Public Health England, 2015).

Obesity significantly reduces life expectancy. For example, Dent & Swanston (2010) estimated that it is decreased by approximately three years in the moderately obese and between eight and ten years in the morbidly obese. This reduced life expectancy imposes costs to the economy through lost future income and productivity[4]. The National Obesity Observatory (2010) explained how obesity often decreased quality of life through incontinence, obstructive sleep apnoea, mental health problems, infertility and musculoskeletal pain as well as other co-morbidities. This emphasises the impact of obesity on public health and outlines the numerous co-morbidities which can result from obesity and reduce quality of life.

Obese adults already impose a great demand on health services through increased risk of type 2 diabetes, cardiovascular disease, coronary heart disease, strokes, osteoporosis, hypertension and endometrial, colon and liver cancer (see for example, Wang *et al*., 2011), in addition to the co-morbidities described by National Obesity Observatory (2010). The Department of Health (2008) claimed that drugs and other treatments, diagnoses, doctors' time and preventative treatments directly related to obesity cost the NHS £4.2bn each year. Foresight (2008) predicted that this figure will double by 2050 if nothing is done. In addition to these direct costs, there are also indirect costs associated with obesity including the costs of mortality and co-morbidities. Foresight (2008) estimated that obesity currently costs the UK economy £16bn overall and this is predicted to rise to £50bn by 2050 in the absence of policy intervention. As well as costs to the NHS, obesity-related co-morbidities can cause restricted productivity, leading to days off work and time in hospital which inflicts additional indirect costs to the national economy. The negative influences on both public health and the economy in the UK highlight the need for good quality research into the causes of obesity to inform policy makers on the most effective ways to reduce the obesity prevalence.

[4] Even in retired individuals, obesity and related illnesses can cause a loss of productivity. An example of this is the loss to productivity as a result of not being able to provide child care for grandchildren.

Although most of these co-morbidities are more commonly found in adults, tackling obesity in early life could reduce these problems in the future. Public Health England (2014) identified the need for early intervention and prevention of obesity due to the difficulty in treating it after it is established. For this reason it is extremely important that childhood obesity is at the forefront of obesity policies in order to prevent children from growing up to be obese adults with obesity-related co-morbidities. Reducing childhood obesity may help reduce future obesity in the whole population.

1.2 Childhood Obesity and Overweight

Research into childhood obesity and overweight differs in a number of ways to that of obesity and overweight in adults. BMI fluctuates during childhood in a different way to adults and the causes and consequences of obesity in childhood differ to those in adulthood. There is less research into the causes and consequences of childhood obesity than there is in adults but the evidence has shown that obese children are more likely to become obese adults (Serdula *et al*., 1993). This suggests that in the long-run, identifying the causes of obesity at an early age could help to prevent obesity in people of all ages. This section outlines some of the issues which are specific to researching obesity in children.

1.2.1 Measuring Childhood Adiposity

Quantifying childhood adiposity is a known problem and it is difficult to determine the level of adiposity which puts a child's health at risk. This problem and the fact it has yet to be resolved, is reflected within the literature by the various definitions of childhood overweight and obesity. This section outlines the problems faced when measuring childhood adiposity. It discusses how childhood BMI fluctuates with age and outlines the different ways that childhood obesity and overweight have been defined using growth reference curves. It also discusses the lack of continuity between childhood and adulthood definitions, which is potentially limiting when estimating the long-term effects of childhood obesity.

Childhood BMI and the Adiposity Rebound

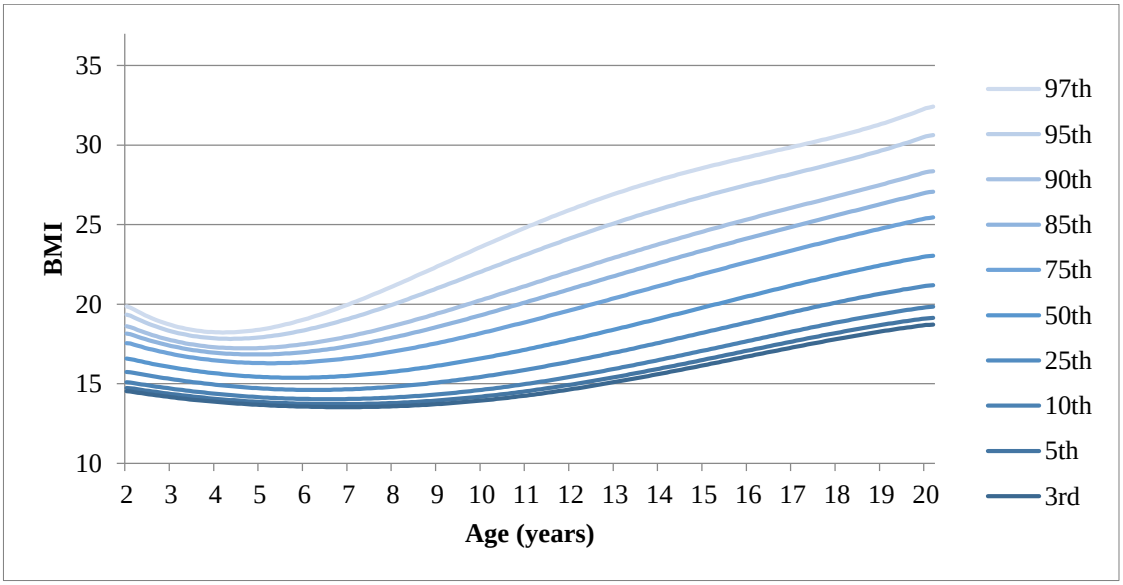
Childhood BMI is measured in the same way as adult BMI (see Equation 2.1) and is often used to measure childhood adiposity. However, childhood BMI suffers from additional

complications to those encountered when measuring adult BMI. Childhood BMI fluctuates depending on their age and sex making it impossible to classify all children over a single BMI threshold as overweight or obese.

The adiposity rebound, a term established by Rolland-Cachera *et al*. (1984), occurs in children around the age of five years when they begin to experience an increase in BMI, after a drop in BMI during early childhood. After the adiposity rebound there is a steady increase in average BMI throughout childhood and adolescence until adult definitions can be used. Rolland-Cachera *et al*. (1984) found that children who experience an early adiposity rebound were at higher risk of overweight later in life. Similarly, Baird *et al*. (2005) claimed that an early increase in weight was widely accepted to predict later obesity.

Figure I-4 and Figure I-5 show how BMI fluctuates during childhood in males and females, respectively. They show the expected BMI throughout childhood for children on different percentiles of the BMI distribution. These charts are from the Centers for Disease Control and Provention (CDC) & the National Center for Health Statistics (NCHS) (2001). These growth reference charts are often referred to as the CDC growth charts and use data on US children from 1963 to 1994[5]. For more information on the data used to produce these charts, see CDC & NCHS (2001).

Figure I-4: BMI Percentiles for US Boys between Two and Twenty Years



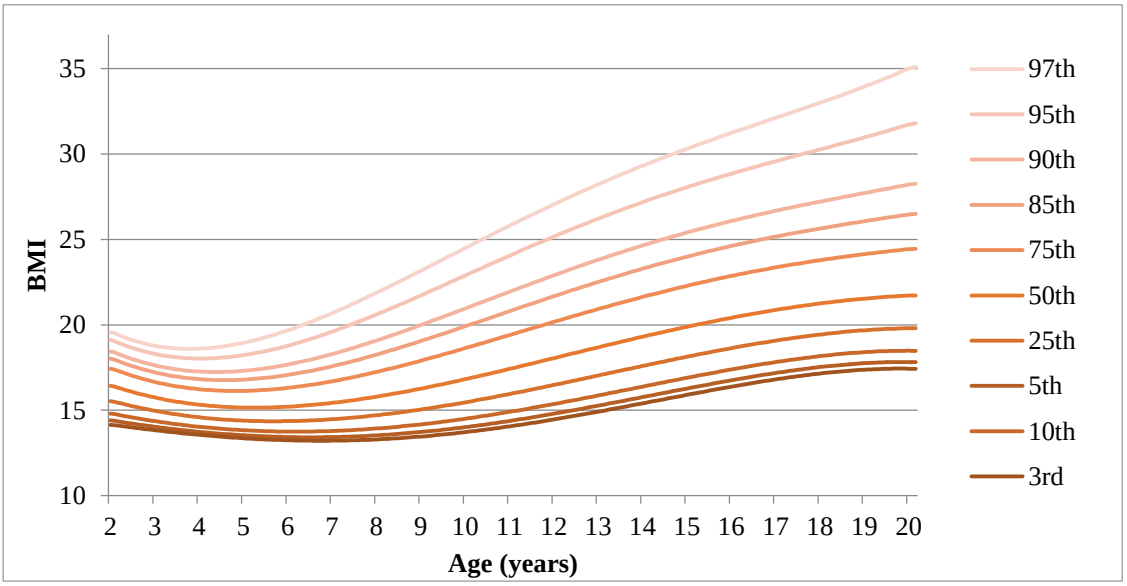
Source: Centers for Disease Control and Provention (CDC) & the National Center for Health Statistics (NCHS) (2001). Notes: Growth charts showing the expected BMI of boys on different BMI percentiles throughout childhood.

[5] Repeated cross-sections rather than following the same individuals.

These figures show that for both males and females, the distribution of BMI throughout childhood does not follow a normal distribution. Those on the 97th percentile were much further from the median than those on the 3rd percentile showing the asymmetric distribution of BMI. It is apparent from these figures that children with a higher BMI during very early childhood were likely to experience an earlier and steeper adiposity rebound.

A healthy childhood BMI differs with age, so comparing children of different ages could give misleading results. As children get older, the standard deviation of the BMI distribution widens and the extreme upper percentiles move further away from the median BMI. If a child is obese or overweight in later childhood, their BMI is further away from the median BMI suggesting that obesity in older children could be more difficult to reverse. Fixed BMI thresholds to define childhood obesity or overweight would fail to identify obesity or overweight in younger children and/or incorrectly identify many older children as obese or overweight.

Figure I-5: BMI Percentiles for US Girls between Two and Twenty Years



Source: Centers for Disease Control and Provention (CDC) & the National Center for Health Statistics (NCHS) (2001). Notes: Growth charts showing the expected BMI of boys on different BMI percentiles throughout childhood.

Growth Reference Curves

Numerous reviews have attempted to compare different measurements of childhood adiposity but they have failed to agree on a preferred measure. In April 2012, the Scientific Advisory Committee on Nutrition (SACN) and the Royal College of Paediatric and Child Health (RCPCH) discussed this issue and reviewed common measurements of

obesity and overweight. Some of the measurements they reviewed, as well as their advantages and disadvantages, are briefly outlined here but a more detailed review can be found in SACN & RCPCH (2012).

Childhood obesity and overweight have most commonly been defined by comparing childhood BMI to sex and age specific references. These references generally use specific populations at given points in time to determine sex and age specific percentiles. Children are then classified as overweight or obese if their BMI is above certain arbitrary percentiles. This means that the prevalence of obesity can be seen to be improving or worsening in relation to the given population at the time the references were created.

The most widely used UK specific classifications of childhood obesity are the 1990 UK BMI reference curves, outlined by Cole *et al*. (1995). The curves are available from birth to the age of 23 years by which time adult BMI definitions can be used. These measurements were not intentionally created to define obese or overweight children; in fact, they were created for the opposite reason. Cole *et al*. (1995) disagreed that children should have fixed BMI thresholds to define obesity or overweight. They argued that arbitrary thresholds to define obesity or overweight would not reflect any increased risk to health. Despite the intentions of the 1990 UK reference curves, they have since been used to create arbitrary BMI thresholds to define obesity and overweight in children.

Another issue arising from these definitions is that different percentiles have been used by different studies. In an attempt to provide guidance on which percentiles to use SACN & RCPCH (2012) suggested that, in a general population, the 95th and 85th percentiles should be used to identify children as 'at high risk of obesity' and 'at high risk of overweight', respectively. In a clinical setting they suggested that the 98th and 91st percentiles should be used to identify obese and overweight children, respectively. However, introducing differing thresholds for general and clinical settings could cause further confusion when interpreting and comparing studies.

The US equivalent of the 1990 UK reference curves are the CDC growth reference charts, see Kuczmarski *et al*. (2002). These charts were created using a US population of two to twenty year olds during 2000. The CDC originally defined 'at risk of overweight' (AROW) and overweight as over the 85th and 95th percentiles, respectively. They used 'AROW' and 'overweight' rather than 'overweight' and 'obese' due to negative connotations which might have been associated with the latter. However, Ogden & Flegal (2010) later argued that the term 'obese' portrayed the correct level of clinical importance

and urgency. Recent childhood obesity literature reflects this changing opinion, with more frequent use of the words 'obese' and 'overweight' in their definitions.

Although the percentiles recommended by the CDC and those recommended by SACN & RCPCH (2012) for a general population are the same, these percentiles refer to different populations and therefore different distributions of BMI meaning that the definitions of overweight and obesity differ between the two. Using a third sample, a different proportion of children could be defined as obese depending on which reference curves were used. Obesity levels vary between populations and over time and so using arbitrary percentiles from an arbitrary sample of a population would not always give the same definition of obesity, even if the same BMI percentiles were used. US references are likely to classify fewer children as obese compared to the UK references when applied to the same sample of children because obesity levels in the US are historically higher than in the UK. Therefore, when US reference curves are applied to UK samples, the extent of obesity problems might be underestimated. Problems also arise when comparing studies which have used different growth reference data.

The time at which the reference data are collected can also influence the number of children which will be defined as overweight or obese. Obesity, in most developed countries, has been increasing over the past three decades. For a given population, references created earlier in time classify more children as obese compared to more recently created references. This inconsistency is demonstrated by Salsberry & Reagan (2005) who compare the 2000 CDC Growth Charts with the 1977 NCHS Growth Charts, both US references using the 95th BMI percentile as a threshold. This highlights the fact that the majority of childhood obesity definitions are arbitrary measures and have little direct relation to health risks.

Lack of Continuity between Childhood and Adult Measurements

Another problem encountered when defining childhood obesity and overweight is that many of the preferred methods, such as those discussed previously, are not in line with adult BMI thresholds for obesity (30kg/m^2) and overweight (25kg/m^2). Obese children are more likely to become obese adults and so a lack of continuity between the two measurements could lead to problems when researching obesity throughout the life-course. Studies such as Cole *et al*. (2000), attempted to create childhood overweight and obesity definitions which align with the more established adult measures. This interpretation of childhood obesity links children with adult health risks associated with

obesity, assuming that nothing is done to reduce the BMI percentile of the child before they reach adulthood. These definitions were created by finding percentiles of childhood BMI which align to the adult BMI thresholds used to define overweight and obesity. However, despite being well-established and widely used, the age at which the adult measures should start to be used remains unclear.

The WHO 2007 growth reference data (Onis *et al*., 2007), available for 5-19 year olds, attempted to address the problem of discontinuity between childhood and adulthood overweight and obesity definitions. These references were a reconstruction of the 1977 references from the NCHS and the WHO which used US data but were intended for international use. This is described in more detail by Onis *et al*. (2007). The WHO 2007 curves were age and sex specific; obesity and overweight were defined as above the 97.7th and 84.1st percentiles of the BMI distribution, respectively. These percentiles were chosen because they aligned with the adult obesity and overweight thresholds at nineteen years. A weakness of the WHO 2007 growth reference data is that it is available only from the age of five years. If the thresholds were defined closer to birth it would be possible to investigate the early life determinants of obesity. It is possible to link the 2007 references with the WHO (2008) growth standards which can fill in the gap between birth and five years. It has also been questioned whether aligning the child and adult measures at nineteen years of age is an appropriate age. At nineteen years old, individuals may not yet have reached their adult BMI levels and further research could give a less arbitrary age at which to make the alignment. However, the WHO 2007 growth charts only hold data on children until the age of nineteen years and so it is not possible to create a later alignment using these data.

Similar measures have been developed by Cole *et al*. (2000). The International Obesity Task Force (IOTF) thresholds for childhood obesity and overweight were created using an international sample from six different countries. For each of these countries, the percentiles which aligned with the BMI thresholds for adults at age eighteen years were identified. Again, it has been questions whether this arbitrary age is appropriate. These national percentiles were then combined to make international age and sex BMI thresholds. The use of international data means that the IOTF thresholds included a variety of ethnic groups in large numbers and remain one of the few obesity measures to do so. As a result, these thresholds have been widely used around the world making it possible to directly compare obesity prevalence between different countries. It is worth noting that no data was taken from African countries which could make these thresholds

less applicable to children of African ethnicities. Reilly (2005) suggested that the IOTF definitions underestimated the prevalence of obesity in UK children, due to the differences in ethnicity and body type represented in the IOTF classifications to the UK population. He suggested that this could lead to an underestimation of the public health crisis caused by childhood obesity but that this was not a problem empirically.

One disadvantage of the childhood obesity measures which align with adult measures is that they only provide the BMI thresholds which align with adult BMI thresholds at specific ages. They do not provide BMI values for other percentiles on the BMI distribution. However, these measures have the advantage that the BMI thresholds they produce can be linked to adult health problems and are more likely to remain relevant over time.

The review by SACN & RCPCH (2012) emphasised the importance of interpretation when using different definitions of childhood obesity, especially when comparing studies. However, the review failed to conclude which set of thresholds were most appropriate and suggested that these issues should be reviewed again in two years' time. Further research could also be done into the health consequences of childhood obesity in order to determine the most meaningful measures of childhood overweight and obesity. The definitions of overweight and obesity used in this thesis will be those designed by Cole *et al*. (2000) and are available in the MCS.

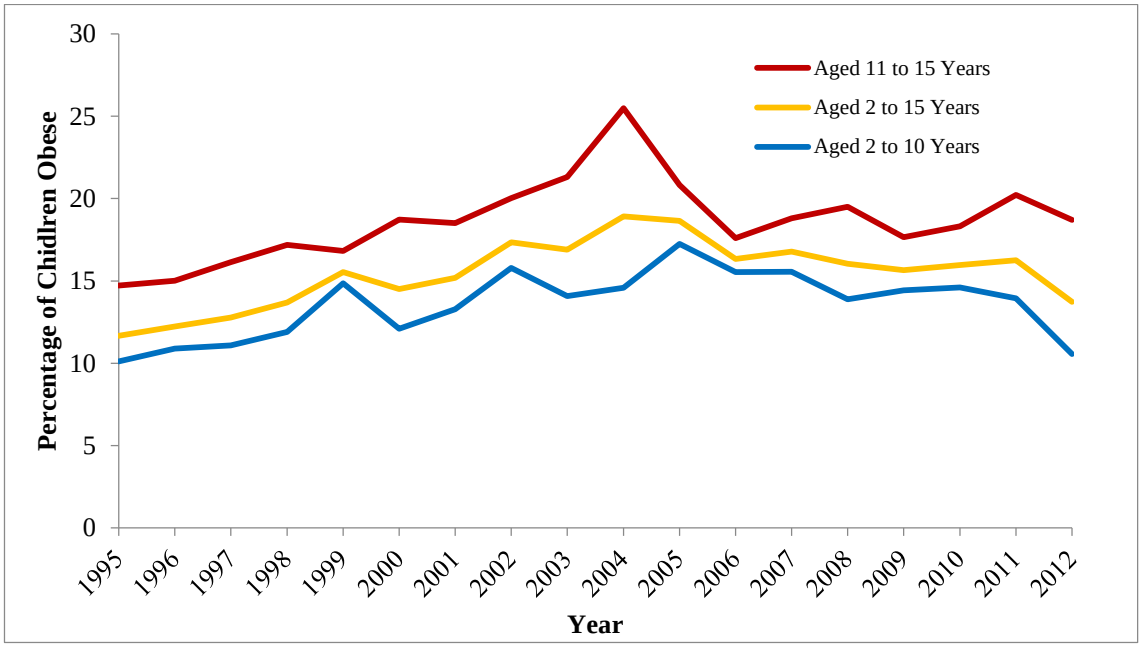
1.2.2 Trends in Childhood Adiposity

According to the HSE, 17% of boys and 15% of girls, between two and fifteen years old in England were obese in 2011[6]. Similar figures are found in the Scottish Health Survey and the Welsh Health Survey. In 2013, the NHS Information Centre for Health and Social Care and Public Health England published data claiming that, during the 2012/2013 academic year, 9.3% of children aged four or five years and 18.9% of children aged ten or eleven years were obese. This could be because children are more likely to be obese as they get older or could show cohort effects where children born earlier are more likely to be obese due to their environment or other external influences. Figure I-6 illustrates the trend in obesity prevalence amongst children in the UK using data from the HSE (2013). There was a steady increase in the prevalence of childhood obesity between 1995 and 2004. Between 2004 and 2012, the prevalence of childhood obesity in England

[6] Here, obesity is defined here as above the 95th percentile using the British 1990 (UK90) growth references. These measures are discussed later.

decreased modestly. A report by the Comptroller[7] and Auditor General (2006) put the reduction in childhood obesity prevalence down to targets set out by the Public Service Agreement in 2004, aiming to stop the increase in childhood obesity prevalence by 2010 (DH, 2004). However, despite this reversal in the trend of childhood obesity levels in England, the percentage of children who are obese remains high, particularly in older children. There is a continued threat that childhood obesity poses to public health and more should be done in order to continue to reduce childhood obesity. Despite the modest decline in childhood obesity prevalence over recent years, BBC news articles, Briggs (2013), have claimed that childhood obesity is a 'ticking time-bomb' and that obesity-related hospital admissions have quadrupled since 2003 suggesting that childhood obesity is an ongoing and worsening problem.

Figure I-6: Percentage of UK Children who are Obese (1995-2012)



Source: Health Survey for England, NHS Information Centre website, (The NHS Information Centre 2008). Notes: Percentage of children (aged two to fifteen years) in the UK with obesity between 1995 and 2012.

Relatively little is known about the relationship between childhood obesity and childhood health. Reilly *et al*. (2003) suggested that many health professionals think childhood obesity causes only cosmetic problems and is not a threat to childhood health. However, they found evidence that childhood obesity was more than just a cosmetic problem. They noted that childhood obesity was associated with co-morbidities during childhood, for example, they found that obese children were at greater risk of cardiovascular problems, similar to those experienced by obese adults. They also observed that obese adolescents

[7] A comptroller is a government official in the UK which is tasked with ensuring the quality of finance and accounting.

were at increased risk of both psychological and psychiatric problems and were more likely to experience poorer social and economic outcomes throughout their lives, even after accounting for intelligence and social class at eleven years of age.

Regardless of the relationship between childhood obesity and health during childhood there is strong evidence that obese children are more likely to become obese adults and, as a result, affect their health in later life. Whitaker *et al*. (1997) found that obesity related diseases during adulthood are much worse in adults who were obese throughout childhood. This suggests that childhood obesity has an influence on adiposity and health in later life. Serdula *et al*. (1993) found that obese and overweight children, in particular adolescents, were significantly more likely to become obese or overweight adults with substantial health risks.

1.3 Obesity Policies in the United Kingdom

An increasing number of policies which focus on the prevention of childhood obesity have been implemented over recent years. Obesity remains high on the public policy agendas of the government and public health bodies such as Public Health England (PHE) and NICE. Recent publications discuss both the current and forecasted costs to the NHS and the wider economy and about the damage to quality of life as a result of obesity, some of which are discussed here. It should be noted that the analysis presented in this thesis does not explore the effectiveness of these policies but aims to inform future policies through evidence based research.

NICE has produced a number of documents containing public health and clinical guidance in relation to obesity and obesity-related diseases and co-morbidities; for example, NICE (2013b) and NICE (2014) provided guidance on managing overweight and obesity in adult and children, respectively. NICE's ongoing interest in obesity emphasises the need for research in order to better understand how to prevent obesity and how doing so might improve public health. For these reasons, a number of strategies and campaigns have been implemented with the aim of preventing the obesity crisis from worsening and reducing the numbers of overweight and obese people in the UK.

In 2004, the Health Minister, Lord Hunt, commissioned NICE and the Health Development Agency (HDA) to develop guidelines on the prevention and management of obesity, some of which are outlined below. This built on earlier work conducted by

the HDA and provided the first systematic review of world evidence on effective strategies of obesity prevention. Research such as this could further understanding into which types of policies are most effective in the fight against obesity. NICE has published a number of guidelines relating to obesity; for example, NICE (2006) gave advice on diet and physical activity and how to help children maintain a healthy weight in which guidance was aimed at the NHS, schools and other institutions on how to help patients, children and employees eat better and stay active. The guidance also gave recommendations on which diet-related drugs should be used and in what circumstances. NICE (2004) gave clinical advice on eating disorders and NICE (2010) provided information on weight management during pregnancy, including ideal weights of mothers as well as babies. NICE (2011b) carried out research into the use of the drug, lorcaserin[8], for use on individuals who were obese and suffered co-morbidities. NICE (2012) worked with local communities, trying to prevent obesity from spreading and NICE (2011c) published guidance on specific co-morbidities relating to obesity, including the prevention of type 2 diabetes. More recently NICE developed public health guidance on lifestyle weight management in adults (NICE, 2014c) and children (NICE, 2013b). The persistent interest in obesity from the government and institutions such as NICE emphasise the continued importance in tackling obesity in the UK.

In 2005, the UK government commissioned a review of obesity which was carried out by Foresight (2008). The review, 'Tackling Obesity: Future Choices', started in October 2007 and used scientific evidence to review the current obesity epidemic and forecast future costs of obesity. It aimed to find a long-term solution to the obesity problem and reduce levels of obesity over time. The report concluded that in order to halt the epidemic, action should be taken on a number of levels; societal, individual and familial. The report suggested that policies implemented to date did not make sufficient progress and that greater efforts must be made in order to prevent the epidemic worsening.

In response to the Foresight (2008) report, the Government issued a new anti-obesity strategy in January 2008, costing £372 million, part of which was promised to fund extra research into obesity. The strategy, outlined by the Cross-Government Obesity Unit (2008) focused on five main topics; these were childhood obesity, healthier food choices, physical activity, incentives for better health and providing support and advice on a personal level. A number of policies were put in place as a result of this anti-obesity

[8] Lorcaserin: A drug used to manipulate appetite in obese patients.

strategy. These policies included, amongst others, working with Ofcom to impose advertising restrictions on certain foods, increasing the number of cycle lanes and bicycle parking spaces along with campaigns for healthier lifestyles such as Change4Life.

Change4Life was started in January 2009 by the DH (2009) in collaboration with the NHS. This public health campaign offers free advice and support for people in England and Wales trying to lose weight as well as educating the public about the consequences of carrying excess fat. With the support of the UK government, the NHS and a number of UK charities and businesses, Change4Life aimed to have a positive and permanent effect on the obesity problem through advertising and education. In addition, Change4Life has a sister intervention aimed specifically at the parents of younger children. Start4Life is aimed at reducing the prevalence of obesity in childhood. It has a particular focus of extending average breastfeeding durations. The range of UK policies already in place demonstrates the interest in childhood obesity prevention but also the scale of the problem and the wide range of policies and interventions which will be needed to tackle the problem.

In 2013, Public Health England (2013) announced that tackling obesity would be one of their priorities for 2013 and 2014 in order to help people live longer and healthier lives. Specifically, they pledged to work with the DH and the NHS to tackle childhood obesity at a national level. This thesis takes a public health approach to the childhood obesity problem by informing public policies on the most effective ways to tackle obesity and how to identify the children and families most at risk. The methods used throughout this thesis will investigate the causes of childhood obesity while identifying characteristics which can be used to target those children most at risk. It aims to ascertain causal relationships of early life determinants on childhood obesity and to help combat the growing problem of childhood obesity by identifying potential policy interventions.

1.4 Millennium Cohort Study

This thesis will analyse data from the Millennium Cohort Study (MCS) obtained from the UK Data Archive, University of Essex during January 2012. The MCS is a large UK birth cohort study following children born in England and Wales between 1st September 2000 and 31st of August 2001 and in Scotland and Northern Ireland between 24th November 2000 and 10th January 2002. Table I-2 shows when each wave of the survey

was, or will be carried out and the average age of the cohort members at the time of interview.

Table I-2: MSC Timeline

Sweep	Year	Age of cohort member
1	2001/2002	9 months
2	2003/2004	3 years
3	2006	5 years
4	2008/2009	7 years
5	2012	11 years
6	2015/2016	14 years
7	2018	17 years

Source: Millennium Cohort Study. Notes: MCS timeline.

The MCS purposefully includes infants born all year round to prevent any bias due to seasonal births. Previous British cohort studies included only individuals born at a certain time of year; for example, the National Child Development Study (NCDS) included only children born in a certain week in 1958. The MCS clustered participants geographically by electoral ward and over-represented children from deprived areas and minority ethnic groups. A total of 398 electoral wards were included in the study and each ward was grouped into one of three categories; ethnic (over 30% of families from an ethnic minority), disadvantaged (poorest 25% of wards which are not considered 'ethnic') and advantaged (all other wards). The 'ethnic' category was only implemented in England. All other countries have only two strata, 'advantaged' or 'disadvantaged', creating nine strata in total. From this study design, 27,201 children were identified using records from the Department for Work and Pensions on child benefits and their families were approached to take part in the study. This was a reasonably robust way of sampling because, at the time, every parent was eligible for child benefits and there was an extremely high uptake. However, 692 eligible families from selected wards were missing in the first wave because they had not yet been added to the child benefit records, in most cases because they had recently arrived or returned to the UK. These families were picked up in the second wave of the MCS but have been removed from analysis in this thesis due to missing variables recorded in the first wave, such as birth weight and infant feeding variables.

Table I-3: Strata and Attrition

		Number of families in sample				
Country	Wards Sampled	Wave 1	Wave 2	Wave 3	Wave 4	% remaining after 4 waves
England	200	11,532	10,050	9,717	8,839	76.65%
Wales	73	2,761	2,261	2,181	2,018	73.09%
Scotland	62	2,336	1,814	1,814	1,628	69.69%
N. Ireland	62	1,923	1,465	1,534	1,372	71.34%
Total	398	18,552	15,590	15,246	13,857	74.69%

Source: Millennium Cohort Study. Notes: MCS sample design strata.

Table I-3 shows the number of wards sampled in each country of the UK, the number of families in the sample during each wave as well as the percentage of families remaining in the study in waves one to four. It was possible for a cohort member to leave and re-join the study.

Table I-4: Millennium Cohort Study UK-wide weighting for Strata

	England	**Wales**	**Scotland**	**N. Ireland**
Advantaged	2.00	0.62	0.93	0.47
Disadvantaged	1.09	0.23	0.57	0.25
Ethnic	0.37	-	-	-

Source: Millennium Cohort Study. Notes: MCS sample design strata weightings.

The over-sampling and attrition which occur in the MCS can easily be accounted for using weights given in the longitudinal family file of the data. Weights are given for the UK (all cohort members), Great Britain (excluding Northern Ireland) and for each individual country. The UK wide weights for each stratum are shown in Table I-4. Weights are also provided for each wave to include attrition which inevitably occurs over time. These weights account for stratification, clustering and sampling as explained by Hansen (2010) and allow the data to represent the entire UK population.

Within each wave of the MCS, the cohort members' main carers were interviewed. In the vast majority of cases this was the cohort member's natural mother. For the purposes of the studies in this thesis, any cohort member whose main carer is not their natural mother will be excluded from the analysis. These observations tend to have missing data on important variables in the context of the present analysis. The MCS also interviewed the partner of the main carer, if there is one, in each wave. Although this was not always the natural father of the cohort member, they are generally a father figure and so no observations will be removed if the partner respondent is not their biological father. The

partner of the cohort members' mothers will be referred to as their fathers for the remainder of this thesis.

Data from the MCS will be analysed throughout the three empirical chapters of this thesis. Within each of these chapters, the variables which will be used and the characteristics of the data specific to each chapter will be discussed further.

1.4.1 Childhood Adiposity Measures in the MCS

There are a range of childhood adiposity measures available in the MCS; some are existing variables and others are generated using existing variables. Childhood adiposity measures were discussed in Section 1.2.1 and different measures are more appropriate for different types of model. Throughout this thesis, different childhood adiposity variables will be used as dependent variables across a range of econometric models. The different adiposity variables taken from the MCS are discussed here and referred to throughout the thesis. As outlined below in more detail, given the different nature of these dependent variables, continuous, binary and ordinal, a range of econometric models are as appropriate.

Childhood BMI

BMI values allow an entire distribution of adiposity to be analysed. Binary variables for overweight or obesity allow the likelihood of having excessive weight to be examined. BMI is calculated using the height and weight of a child in the same way it is calculated in adults, see Equation (I.1). BMI values for each cohort member are available in waves 2, 3 and 4 of the MCS, when the cohort of children was approximately three, five and seven years old, respectively. A small proportion of BMI values were manually calculated (0.44%) in wave 2 where BMI was not available, but where height and weight were recorded. It is unclear why these values were not automatically calculated in the original data.

In the existing literature, models of childhood BMI are most commonly estimated using linear models, where a continuous variable is preferable. As previously discussed, BMI measures for children of different ages are incomparable and so a different mean BMI for children of different ages does not necessarily suggest that children are more or less obese. This was discussed in more detail in Section 1.2.1.

Biologically implausible values (BIVs) for BMI, height and weight, are removed in accordance with the recommendations defined by an expert committee at WHO (1995). This report stated that any plausible height must lie between -5 and +3 z-scores, any plausible weight must lie between -5 and +5 z-scores and any plausible BMI values must be between -4 and +5 z-scores. These BIVs were developed using data from the NCHS and WHO growth charts from 1977.

Childhood Obesity and Overweight

Children in the MCS have also been categorised by their weight status. They are defined as 'normal', 'overweight' or 'obese' using their BMI score and sex and age specific thresholds. This thesis will use the IOTF thresholds developed by Cole *et al*. (2000) which were discussed in more detail in Section 1.2.1 and are already calculated and readily available in the MCS. Two binary variables are available indicating obesity and overweight, including obese[9]. These are

$$\boldsymbol{y}_{obese} = \begin{cases} 0, & BMI < \delta_{obese} \\ 1, & BMI \geq \delta_{obese} \end{cases} \tag{I.2}$$

$$\boldsymbol{y}_{overweight} = \begin{cases} 0, & BMI < \delta_{overweight} \\ 1, & BMI \geq \delta_{overweight} \end{cases} \tag{I.3}$$

respectively and δ_{obese} and $\delta_{overweight}$ are the sex and age specific thresholds for obesity and overweight, respectively, using the IOTF classifications. The IOTF definitions of overweight and obesity are more in line with the established adult definitions, which they align with at the age of eighteen years. This makes it possible to extrapolate whether a child will become an obese or overweight adult if they remain on the same BMI percentile into their adulthood. This is of particular importance as a consequence of the associated health risk associated with obesity and overweight.

Childhood Weight Status

These IOTF thresholds will also be manually combined into a single ordered variable,

$$\boldsymbol{y}_{weight\ status} = \begin{cases} 0, & BMI < \delta_{overweight} \\ 1, & \delta_{overweight} \leq BMI < \delta_{obese} \\ 2, & \delta_{obese} \leq BMI \end{cases} \tag{I.4}$$

[9] The definition of overweight presented here includes children who have a BMI over the threshold $\delta_{overweight}$ and therefore indicates whether a child is overweight or obese.

where δ_{obese} and $\delta_{overweight}$ are the same sex and age specific IOTF thresholds for obesity and overweight[10], respectively.

Summary Statistics

Table I-5 shows the summary statistics of the childhood adiposity dependent variables after data have been cleaned and any BIVs removed. The number of observations, mean (with standard deviation for continuous variables) and median are presented for each variable and for each wave of the data. The proportion of overweight children significantly decreases with the age of the cohort[11]. The proportion of obese children increases with age but this increase is statistically insignificant. Children from disadvantaged backgrounds are over-represented in the data and these children are more likely to be overweight or obese. Once this over-representation is accounted for by weighting, the means are slightly reduced. However, there is very little difference between the means of the weighted and unweighted means (16.78 vs. 16.77, respectively) suggesting that weighting the data will have little empirical influence. Hansen (2012) and Plewis (2007) also suggest that this weighting would make little difference.

Table I-5: Summary Statistics of Childhood Adiposity Variables

Variable	Number of Observations	Mean	Median	Weighted Mean
		3 Years		
BMI (kg/m^2)	12,922	16.77 (1.591)	16.6	16.78
Overweight	12,853	0.2351	-	0.2315
Obese	12,853	0.0520	-	0.0498
		5 Years		
BMI (kg/m^2)	13,474	16.32 (1.689)	16.08	16.30
Overweight	13,474	0.2120	-	0.2053
Obese	13,474	0.0533	-	0.0507
		7 Years		
BMI (kg/m^2)	12,301	16.62 (2.255)	16.18	16.57
Overweight	12,299	0.2032	-	0.1971
Obese	12,299	0.0567	-	0.0527

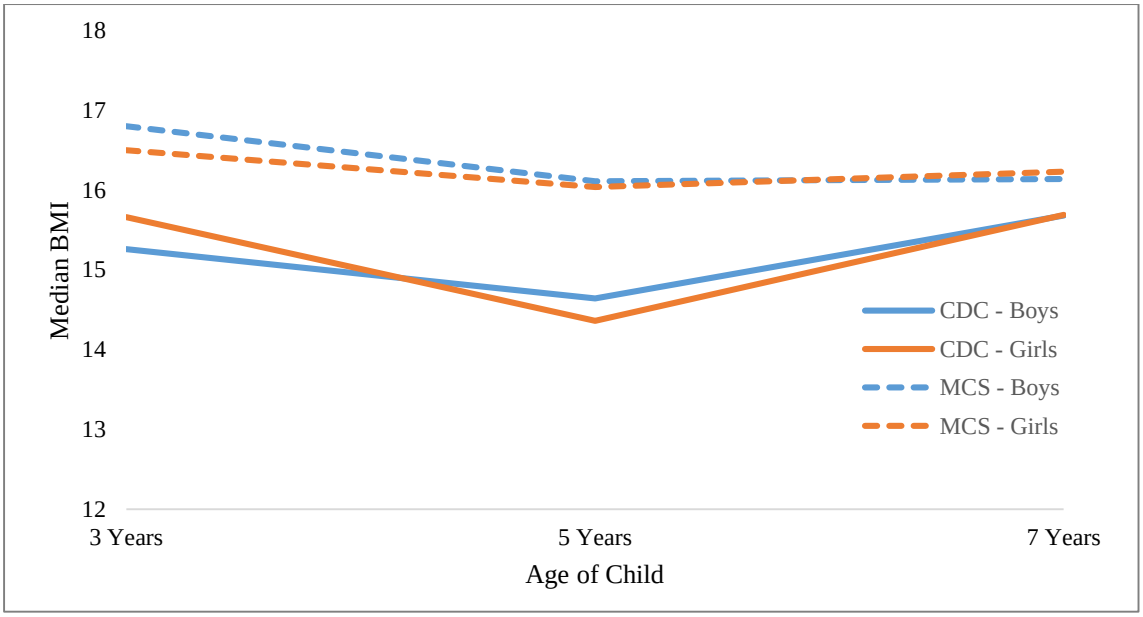
Notes: Data from Millennium Cohort Study. Childhood adiposity summary statistics. Standard deviations in parentheses where applicable.

The medians of the BMI values from the MCS are slightly higher than those displayed in Figure I-4 and Figure I-5, using data from the CDC growth charts. This could be due to

[10] Here, the overweight category does not include obesity.
[11] Significantly different using a χ^2 test for proportions.

the over-representation of disadvantaged children in the MCS or because the MCS contains more recent data on children, since the obesity epidemic has worsened. That said, the median BMIs for both boys and girls follow a similar pattern in both sets of data and there is clear evidence of the adiposity rebound after the age of five years.

Figure I-7: Median BMI by Age and Sex from the CDC Growth Charts and Children from the MCS.



Source: CDC growth charts and Millennium Cohort Study.

The MCS is sufficiently large and has a high enough incidence of obesity and overweight to assume that type II error in the analysis is minimal. Increased type II error often occurs in smaller cohort studies with a low prevalence of the outcome, as explained by Kramer (1981), reducing the statistical power of the analysis.

1.5 Structure and Content of Thesis

This thesis focuses on three distinct but related topics. Its overall aim is to investigate the causes of and influences on obesity throughout early childhood (between birth and seven years of age), using a variety of econometric techniques, in order to inform policy makers and guidance producers such as NICE. It investigates how early life influences, socio-demographics, family environment and health affect childhood adiposity. Using large scale national cohort data and suitable econometric techniques, three empirical chapters will help to inform obesity prevention policies and help to target them towards the appropriate children and families.

The first empirical study explores the extent to which breastfeeding protects against childhood obesity; this will take into account early life and demographic variables as well as other confounding factors, including parental and prenatal variables. A number of public health guidance reports have linked breastfeeding with childhood obesity and suggested that breastfeeding is the most reliable way to provide infants with the best possible nutrition (NICE, 2006; NICE, 2010; NICE, 2012). There have been a number of policies which have aimed to increase the prevalence and duration of breastfeeding. Breastfeeding is known to have a range of benefits to both mother and child and has been found in some studies to be associated with a reduction in childhood obesity. The first empirical chapter of this thesis will use a range of econometric techniques to investigate the effects of breastfeeding on childhood adiposity. The methods will each use different sets of assumptions in an attempt to identify causal effects. These methods include OLS and logit models to allow a comparison with the existing literature, ordered probit models to estimate overweight and obesity in a single model and propensity score matching (PSM) to investigate the causal relationship without the restrictions of a functional form. In order to account for the possible endogeneity of breastfeeding in estimating childhood BMI, instrumental variable (IV) techniques and Roy models will be used; the IV technique will allow the causal relationship of breastfeeding on childhood BMI to be identified in the presence of potential endogeneity and the Roy model allows breastfeeding and childhood adiposity to be simultaneously estimated in the form of a switching regression model. By estimating the effect of breastfeeding on childhood adiposity using this range of methods it will be possible to determine how realistic some of the assumptions that they make are; for example, whether all important confounding factors are accounted for and whether breastfeeding is endogenous after these confounders have been accounted for.

The study finds insufficient evidence that breastfeeding is endogenous once such a wide range of important confounding factors are accounted for. For this reason, results from the models which assume treatment selection only on observable characteristics are used for policy recommendations. There is also evidence that the functional form imposed by the linear models is restrictive. For this reason, any policy recommendations will be based on results from the PSM approach. The results suggest that breastfeeding has a small but statistically significant reduction in childhood BMI and the likelihood of childhood obesity and overweight. These effects increase as children get older, which

might suggest that there are a large number of other influences which might come into play as they grow up.

Although these effects are statistically significant, they are small. This suggests that breastfeeding should have a part in obesity prevention but that policy makers should include breastfeeding as part of a wider obesity intervention targeting multiple lifestyle behaviours.

After looking specifically at the causal effect of breastfeeding, a single potential influence on childhood adiposity, the second empirical chapter encompasses the more complex problems of familial influences on obesity as well as the dynamics of how underlying family lifestyle and childhood obesity develop throughout early childhood. Developing a dynamic latent factor model allows the persistence of family lifestyle and its causal influence on childhood obesity to be investigated. NICE (2010) and NICE (2012) which provided guidance in relation to obesity management whilst pregnant and within the local community, respectively, both acknowledged the need for family based approaches to weight management. NICE (2013b) provided guidance on childhood weight and suggested that childhood weight management services should be family based and have multiple components. Chapter III dynamically models childhood weight status an outcome measure of underlying family lifestyle and uses simulations to estimate probabilities of obesity in children with different characteristics.

Results from this model show that underlying family lifestyle is very persistent suggesting that strong and sustained policies and interventions would be needed in order to have a significant influence. It also suggests that any successful interventions will have long-lasting effects, an important point to bear in mind when considering the cost-effectiveness of interventions. There is evidence that improvements to family lifestyle will provide long-lasting benefits to all family members over a range of outcome measures and that improving family lifestyle for disadvantaged families could help to reduce social inequalities in obesity prevalence.

Finally, in Chapter IV, health is incorporated into the model used in Chapter III. This makes it possible to determine whether there are indirect effects of lifestyle on childhood obesity through the health of the child. Again, this chapter will investigate these relationships throughout early childhood. NICE (2013b) identified a number of potential co-morbidities related to childhood obesity in the existing literature; these included type 2 diabetes, cardiovascular problems, asthma and non-alcoholic fatty liver disease.

However, despite there being plenty of research into the health consequences of childhood obesity, there is a lack of research into how the underlying general health of a child might influence the risk of childhood obesity. The chapter jointly models underlying childhood health and underlying family lifestyle in order to determine how childhood obesity is influenced. This model allows the direct and indirect causal effects of lifestyle on childhood adiposity to be estimated, as well as the causal influence of child health on childhood adiposity.

Underlying childhood health is persistent although not to the same extent as underlying family lifestyle. Family lifestyle is already well established within a family before a child is conceived but health is more responsive to external shocks. That said, child health is at least partly determined at birth by maternal health and lifestyle during pregnancy which could influence child health throughout childhood. Both health and lifestyle at birth have lasting effects on childhood obesity prevalence. Socioeconomic and family background characteristics are associated with childhood adiposity; advantaged children are less likely to be obese. Both underlying family lifestyle and child health are found to be the mechanisms by which social inequalities in obesity prevalence occur. The addition of health in this chapter is important for economic models which aim to estimate the cost-effectiveness of lifestyle interventions. For example, the range of outcome measures in each period of the model means that multiple parameter estimates from this structural model could be used in cost-effectiveness models which account for the variety of benefits which might come about due to lifestyle interventions.

In order to effectively reduce childhood inequalities in obesity prevalence and health, caused by early disadvantage, policy makers need to address underlying family lifestyle by informing and educating parents and enabling them to provide their families with healthier lifestyles. Interventions should be targeted at families with young children as early as possible in order to have the greatest cumulative influence. That said, successful family lifestyle interventions implemented at any stage of early childhood could have long lasting effects, including improved health and reduced obesity. Rather than intervening in specific lifestyle behaviours, policy makers should take a wider approach and help families to understand how improving their lifestyles will have an influence on their family's health and adiposity. Before and during pregnancy, women should be encouraged to lead healthy lifestyles. During infancy breastfeeding should be encouraged, along with a number of other lifestyle behaviours, both generally and specifically relating to early life. Throughout childhood, the lifestyle of all family

members, in particular the mother, should be targeted. Lifestyle improvements which are encouraged should include diet, physical activity and maintaining a healthy weight and these should be encouraged alongside an effort to increase family awareness of the importance of these changes and the long-term effects that they are likely to have. At each of these stages of pregnancy and childhood, policy makers should aim to improve parental knowledge of the benefits of a healthy lifestyle and educate parents about the effects that lifestyle has on obesity, child health and other outcomes. Particular attention should be given to disadvantaged families and families which are more at risk of obesity, unhealthy lifestyle and poor health. Targeting these individuals could help to reduce inequalities in health and obesity during childhood.

In addition to the findings from each of the empirical studies outlined above, the parameter estimates resulting from all three empirical chapters could be important in providing economic models with much needed evidence when modelling childhood obesity, as well as other outcome measures. This is discussed in more detail for each specific study in each of the empirical chapters in the thesis.

This thesis contributes to the existing literature in several ways. Many of the previous studies in this area use small samples taken from specific sections of society which make it difficult to generalise results to a population level. Using UK representative data is important when trying to tackle obesity as a nationwide problem. Each of the empirical chapters make contributions to the literature and to the public health approach to childhood obesity. The thesis is structured as follows: Chapters II, III and IV are standalone empirical chapters which will investigate the three topics outlined above and Chapter V provides a discussion and conclusion to the thesis.

II. CHILDHOOD OBESITY AND INFANT FEEDING

Research Question: What is the influence of breastfeeding on childhood adiposity?

Aims:

- To disentangle the relationship between breastfeeding and childhood adiposity measures by accounting for confounding factors.
- To investigate this relationship using different econometric models and therefore using different sets of assumptions.
- To determine whether or not breastfeeding is endogenous in predicting childhood adiposity measures after confounders are accounted for.
- To determine the most appropriate econometric model in testing this relationship.
- To inform policy makers and identify future research.

2.1 Introduction

The protective effect of breastfeeding on obesity throughout childhood and into later life has been the subject of health research across the developed world over the last thirty years. There is little doubt that breastfeeding and childhood adiposity are inversely correlated but the extent to which this relationship is mediated by confounding social and genetic factors is an important point of contention. Potentially, confounding factors could affect both maternal breastfeeding behaviour and childhood weight causing a correlation between them when there is no causal relationship. For example, Iacovou & Sevilla-Sanz (2010) found that higher parental education significantly increased the duration of partial and exclusive breastfeeding, where exclusive breastfeeding is defined as breastfeeding without supplementary liquids or solids, including formula milk and Lamerz *et al*. (2005) and von Kries *et al*. (1999) found that parental education was a very strong predictor of lower levels of obesity in childhood. Socioeconomic status (SES) has also been found to have a large confounding influence on breastfeeding and obesity. Amir & Donath (2008) found that parents with higher SES breastfed for longer durations and Shrewsbury & Wardle (2008) found that their children were at lower risk of obesity. Many of these confounding factors, for example parental education and SES, are highly correlated, highlighting the difficulty in disentangling the relationship between breastfeeding, adiposity and their confounding factors. Maternal influences have also been found to confound this relationship. Dewey (2003) found that maternal overweight reduced the likelihood of breastfeeding and also increased the likelihood of childhood overweight. Hill & Aldag (1996) found that mothers who smoked breastfed for shorter durations than non-smoking mothers. They put this down to insufficient milk produced by smoking mothers. Toschke *et al*. (2002a) found that maternal smoking whilst pregnant increased the chance of obesity later in childhood. However, it remains unclear whether this effect is due to causal biological factors or a confounding influence.

The current WHO (2011b) recommendations advise exclusive breastfeeding until an infant is six months old. At six months, they recommend that solids are introduced, after which breastfeeding should be continued alongside appropriate foods until the child is at least two years' of age. Prolonged and exclusive breastfeeding are recommended because they have been found to have a wide range of benefits to both the infant and the mother. For example, breastfeeding was found by Iacovou & Sevilla-Sanz (2010) to have a positive causal impact on childhood cognitive development, Oddy *et al*. (2010) found that

it reduced the risk of mental health problems during childhood and Hanson (1998) found that breastfeeding boosted immunity and prevented childhood infections. Breastfeeding has also been linked with an improvement in childhood behaviour by Heikkilä *et al*. (2011). Singhal & Lanigan (2007) claimed that increased rates of breastfeeding could help to reduce health inequalities[12] suggesting that increased breastfeeding duration or exclusivity benefits children from lower SES more than those from higher SES. Studies including Klaus (1998) and Renfrew *et al*. (2000) found that breastfeeding helped mothers to bond with their child and it can be the cheapest and often most convenient type of infant feeding. Vanlandingham *et al*. (1991) recorded the benefits of postpartum contraception that result from breastfeeding and Dewey *et al*. (1993) found that breastfeeding supported maternal weight loss. Many studies have also found that breastfeeding can prevent obesity during childhood and although there is little doubt that there is a correlation between the two, the literature is inconclusive when it comes to how much of this correlation is due to confounders and many findings contradict each other. Renfrew *et al.* (2007) suggested that breastfeeding should be encouraged regardless of possible effects on obesity during childhood due to the other benefits outlined above. That said, any additional evidence of health benefits in breastfed children will give further weight to policies already promoting breastfeeding and could encourage more mothers to breastfeed for the recommended durations.

A number of theories have been presented to explain why breastfeeding might influence childhood adiposity. Li *et al*. (2010) found evidence to support the 'self-regulation theory' which suggests that breastfed infants learn, at an early age, to stop feeding once satisfied. Bottle fed infants are often encouraged to finish any milk they are given despite how much they need. This self-regulation in breastfed infants is thought to persist into childhood and thus prevent overeating and unnecessary weight gain. Li *et al*. (2010) found that infants who consumed breast milk from a bottle did not benefit from reduced BMI, supporting the 'self-regulation' theory. Kramer *et al*. (2004) investigated the 'growth-accelerating theory' and found that formula fed infants experienced accelerated growth during infancy which Koletzko *et al*. (2009) held accountable for an increased risk of obesity later in life. Günther *et al*. (2007) outlined the 'early protein hypothesis' suggesting that formula fed infants consumed a much higher protein level than infants who consumed only breast milk. They suggested that high protein levels early in life

[12] Health inequalities are differences in health between people or groups due to social, geographical, biological or other factors and lead to those who are worst off experiencing poorer health and shorter lives (NICE, 2012a).

induced hormone responses which caused higher levels of insulin to be secreted leading to weight gain. The latter two theories suggest that improvements in formula milk could reduce any potential relationship between infant feeding and childhood obesity.

This chapter aims to disentangle the relationships between breastfeeding, obesity and any confounding factors in order to identify the true impact that breastfeeding has on childhood obesity. The rich, large scale panel data used in this chapter will allow for a more robust estimation than many previous studies[13]. Causal influences are difficult to estimate and there is a large literature on the estimation of causal treatment effects (Blundell & Costa Dias, 2009; Faria *et al*., 2015; Imbens & Wooldridge, 2009). Different models are identified using different assumptions which are often untestable. Therefore, throughout this empirical chapter, a range of models with different assumptions will be used in order to check the appropriateness of each set of assumptions in this specific setting. This includes models that assume selection on observables and those which assume selection on unobservable confounders, as well as other potentially restrictive assumptions. A number of existing studies use a similar approach to this chapter to compare the appropriateness of the assumptions of range of econometric techniques. For example, Vandenberghe & Robin (2004) investigated the effects of private education on attainment using a range of methods and Posner *et al*. (2002) explored the influence of mammography on the stage of cancer at diagnosis. Other studies have discussed the comparison of the models used in this chapter more generally (D'Agostino & D'Agostino, 2007; Faria *et al*., 2015; Heckman & Navarro-Lozano, 2004). This chapter is most similar to the approach taken by Rothstein (2013) who investigated the effects of breastfeeding on cognitive development. The range of econometric techniques used in this chapter have been used before in many different research areas, but to my knowledge has not been done when investigating the effects of breastfeeding on childhood adiposity measures.

First, in line with the majority of current literature, linear and logit regression models will be estimated. Next, ordered probit models will be used and compared to the regression models used throughout the existing literature. Propensity score matching (PSM) will be implemented with a range of binary breastfeeding treatments in order to relax the assumption of a functional form in the outcome equation. Finally, models which assume

[13] Del Bono & Rabe (2012) attempted to investigate this relationship using the MCS and this study will be discussed in more detail later. However, to my knowledge no other study has investigated the influence on breastfeeding on childhood adiposity using the range of methods and assumptions used in this chapter.

selection into treatment on unobservable confounders will be used. These include a linear regression with instrumental variable (IV) technique as well as a restricted version of the Roy model. The range of econometric techniques used in this chapter will allow the relationship between childhood adiposity and breastfeeding to be investigated using a range of dependent variables and under different sets of assumptions in order to determine the most appropriate analysis for policy recommendations.

Results from this chapter show that breastfeeding has a small but statistically significant influence on childhood adiposity. The effects are more pronounced and more statistically significant as children get older suggesting that it will take time for the full effects of breastfeeding on childhood adiposity to become apparent. Longer durations and more exclusive breastfeeding have the largest and most significant effects. Reductions in the likelihood of overweight due to breastfeeding are generally larger than reductions in the likelihood of obesity. Exclusive breastfeeding continued for at least sixteen weeks is found to reduce BMI by up to 0.25 points by the age of seven years. Although this does not appear to be a large decrease in BMI, at this young age the average BMI is low and this is a relatively large proportion of the average BMI. Additionally, any differences in BMI at this young age are likely to become larger as children get older and the distribution becomes wider. There is some evidence that the effects of breastfeeding on adiposity are greater in disadvantaged children which suggests that breastfeeding interventions could help to reduce childhood inequalities in obesity prevalence. As a result of the findings from this chapter, policy makers aiming to reduce childhood obesity should try to improve breastfeeding participation and encourage exclusive and prolonged breastfeeding. However, because the magnitude of these effects are small, they should target breastfeeding alongside a range of other lifestyle interventions. Breastfeeding should be part of wider anti-obesity policies tackling a range of early life influences simultaneously in order to produce a meaningful reduction in childhood obesity. No single lifestyle intervention will be sufficient to prevent childhood obesity completely.

The remainder of this chapter will be structured as follows. Section 2.2 will review the literature, Section 2.3 will describe the data used in the chapter, Section 2.4 will describe the methodology, Section 2.5 will present the results and Section 2.6 will discuss the findings.

2.2 Literature Review

This section illustrates the need for further research into the effects of breastfeeding on childhood adiposity. By reviewing the existing literature, this section will explain the key issues arising from research in this area as well as identifying research gaps.

This review is not a 'systematic review' and does not aim to cover every study which has previously investigated the relationship between breastfeeding and childhood adiposity[14]. Rather, it is a scoping review aiming to identify each type of method which has previously been used to investigate this specific relationship while also identifying research gaps and potential problems with existing studies. Each study which is included is not intended to add further weight to an overall result (as would be the case in a meta-analysis) but is instead intended to add to the knowledge provided by the review, in this case identifying gaps in the research. This approach is known as an 'interpretive review', as opposed to an 'aggregate review' (see pages 21-22 of Booth *et al*., 2012). This interpretive review aims for 'conceptual saturation' rather than saturation of every piece of literature in the area. Therefore the inclusion of every study which appears in a search is not required, only those which add a new line of enquiry, method, idea or concept to the review are included.

In order to fulfil the aims of this review, a technique known as 'berrypicking' was used. This approach was first described by Bates (1989) who explains how this approach allows a review to evolve as new studies are found. This evolution allows the reviewer to follow up ideas or concepts which become apparent as the literature is searched and enables the reviewer to look at more focused aspects of their research in more detail. The berrypicking approach identifies a study or studies which are particularly relevant to the research question and what the review is aiming to ascertain. It then uses six different strategies to identify further relevant literature. These are, *footnote and reference chasing*, *citation searching*, *journal runs, area scanning*, *bibliography, abstracting and indexing services* and *author searching*. Further details on each of these search strategies can be found in the article by Bates (1989).

The berrypicking method was started using the search terms 'breastfeeding' and 'childhood obesity' in google scholar to identify articles which had both of these phrases

[14] Additional articles which address the methodological issues relevant to this chapter but do not specifically investigate the influence of breastfeeding on childhood adiposity will be included later, in the methodology section.

in their titles[15]. From the results which contained both these phrases in their titles, those which were empirical studies were looked at in more detail. Studies were only included if they specifically investigated the effects of infant feeding on some measure of childhood adiposity and if this was apparent from their title or abstract. Due to the interpretive review approach taken, not every study was included as many studies were very similar (for example, there were are large number of very similar studies which used logistic regressions on different populations). The review focuses, where possible, on children born in 1982 onwards[16] and on studies from developed countries most similar to the UK. Table A-1 in Appendix A lists the eighteen studies reviewed in this section along with additional information on each of them.

By investigating the different adiposity and breastfeeding measures, as well as the different datasets and methods used throughout the literature, it is possible to see how the large discrepancy in the findings comes about. This literature review is structured as follows. Section 2.2.1 discusses problems arising from the lack of consistency in outcome variables and breastfeeding definitions. Section 2.2.2 reviews datasets and methodologies which have been used previously and Section 2.2.3 reviews their findings. Each of these issues was identified while carrying out the berry-picking review method. Finally, Section 2.2.4 summarises and concludes the review.

2.2.1 Inconsistency of Measurements

One of the major differences between studies in this literature is the different ways important variables are measured. Different studies use different measures of childhood adiposity as well as different measures of the same concepts, as explained in Section 1.2.1. There are also differences between the measurements used to identify and quantify breastfeeding. The measurements chosen by each study depend upon the model used and data available. These inconsistencies make it difficult to interpret the results of each study and to compare the results of different studies.

[15] Other electronic databases were also used to replicate this initial search and made no difference to the papers selected. Although these search terms were used to identify the initial articles, they were not considered necessary at later stages of the berrypicking method.

[16] The thirty years prior to the start of the review. Breastfeeding habits are constantly changing due to different societal trends, improvements in the availability and quality of formula milk, the working habits of women and changes in the recommended durations of breastfeeding. This review focuses on the last thirty years in an effort to make it as comparable as possible to current breastfeeding behaviours.

Anthropometric Measurements

Section 1.2.1 outlined the different ways of defining childhood obesity. The lack of agreement around which measures are most appropriate means that a variety of measures are used throughout the literature. The definitions of the childhood adiposity used by each study reviewed in this chapter can be found in Table A-1. The majority of studies used childhood overweight and obesity as dependent variables. Most are US studies and used the CDC charts to define overweight and obesity in children. These studies include Bogen *et al*. (2004), Burdette & Whitaker (2007) and Mayer-Davis *et al*. (2006). However, other studies such as Armstrong & Reilly (2002) and Reilly *et al*. (2005) used data from the UK along with the 1990 UK curves. McCrory & Layte (2012) used the IOTF thresholds in an attempt to link childhood obesity with adult measures in order to relate childhood obesity to health risks in later life. The difference between childhood and adulthood obesity measures is an important one and was discussed in more detail in Section 1.2. Del Bono & Rabe (2012) defined an overweight child as one with a BMI over 25. This definition of overweight is more commonly used in adults and as a result is difficult to compare to the majority of childhood studies[17]. The usual thresholds for BMI for children are much lower than 25 and vary by age and sex. See Section 1.2.1 for a discussion of the problems of measuring childhood adiposity and a description of recognised measures. In particular, Figure I-4 and Figure I-5 show it is extremely unusual for children at those ages to have a BMI above 25 and this is more likely to be the result of a severe health problem or measurement error, particularly at the young ages of three and five years when a child with a BMI over 25 is likely to fall in the biologically implausible range, as defined by WHO (1995). Similarly, Brion et al. (2011) used BMI to investigate the effects of breastfeeding in different groups of children. This measure of adiposity is non age or sex specific but was used to compare two cohorts of children each of different ages. This is problematic because different levels of BMI are considered to be healthy in children of different ages.

The range of dependent variables and the range of definitions and percentiles used to create BMI thresholds emphasises the need for a single, commonly accepted definition. Papers in this literature have generally avoided the use of the WHO 2007 charts, most likely because the data are only available from the age of five years which does not allow

[17] It is acknowledged that Del Bono & Rabe (2012) state in footnote 16 on page 35 that other dependent variables relating to childhood adiposity were also used and gave no major differences in results. However, continuous weight might also be problematic in that it does not account for the height of the child.

for childhood adiposity measures in infancy, when breastfeeding is more recent. Additionally, the different measures of childhood adiposity used throughout the literature make it difficult to compare results and could influence the findings of the studies.

The fluctuation in BMI throughout childhood also appears to have an impact on the relationship between breastfeeding and adiposity. Bergmann *et al*. (2003) found that bottle fed infants experienced their adiposity rebound significantly earlier. Burke *et al*. (2005) investigated whether children who gained weight and became obese at twelve months were more likely to be persistently obese until at least eight years. They found that by the age of eight years, breastfeeding no longer had an impact on obesity after this earlier obesity was taken into account and suggested that breastfeeding prevented an increased weight-for-length during early childhood causing early obesity which often continued into throughout childhood. This theory is also supported by the finding of Kramer *et al*. (2004) who found that formula feeding caused a faster growth in infants which slowed to a normal growth as the child got older. Baird *et al*. (2010) described an earlier and more rapid increase in weight or BMI to be a strong predictor of later obesity which suggests that early life factors, such as breastfeeding, could influence obesity if they effect the speed of growth during infancy. These fluctuations in childhood BMI, particularly in relation to the adiposity rebound, suggest that studies investigating childhood obesity should only compare children of similar ages rather than a wider age range of children.

Other studies have used childhood BMI as part of an indexed measure of health. For example, Fitzsimons & Vera-Hernández (2013) investigated the influences of breastfeeding on cognitive development during childhood as well as an indexed measure of health. This indexed measure of health included childhood BMI as part of its composition. However, because childhood BMI was part of a more complex health measures, the specific influence of breastfeeding on childhood BMI cannot be identified from their estimation.

Quality of Anthropometric Data

Problems can also arise depending on how data are collected. Data on childhood obesity are often reported by mothers and bias could occur if certain types of mothers were more likely to under- or over-report childhood weight. Carnell *et al*. (2005) found that mothers with larger children were more likely to underreport the weight of their child. Seghers & Claessens (2010) found that parents became more likely to under-report childhood weight

as their child got older and Livingstone *et al*. (1992) found that childhood weight was more often underreported by parents of female children.

Although most studies in this literature used data reported by parents, some studies chose to avoid the problems that parent-reported data can cause. In a study by Bergmann *et al*. (2003), participants were recruited to the German Multicentre Atopy Study. This study required participants to attend repeated physical examinations where their height and weight were measured accurately by medical professionals. These examinations were carried out until the age of six years and would have prevented bias caused by parent-reported data. However, Bergmann *et al*. (2003) acknowledged the possibility of an unobservable effect on participants caused by repeated communication with the professional observers carrying out the study which could have caused children to gain less or even lose weight. This could have lessened the effects of breastfeeding found in this study and a different type of bias might have occurred. Both parent-reported and professionally measured anthropometric data could be subject to bias and it is important to keep these issues in mind when interpreting results.

Breastfeeding Measurements

Similar problems to those which occur when measuring childhood obesity can also occur when defining breastfeeding. Different definitions of breastfeeding can be found in different studies, making interpretation and comparison between studies more difficult.

Breastfeeding duration is often defined as either partial or exclusive. Partial breastfeeding, such as that used by Jiang & Foster (2012) and Oddy & Sherriff (2003), is usually measured by the age of an infant when they received their last breast milk, irrespective of whether the breast milk was received alongside other liquids or solids. Exclusive breastfeeding, such as that used by Hediger *et al*. (2001), is usually defined as the age of the infant when liquids or solids, other than breast milk, were introduced.

Although breastfeeding duration is a continuous variable, many studies have used breastfeeding categories, by reporting whether or not the infant was still breastfed at given ages. For example, Burke *et al*. (2005) sorted children into categories of partial breastfeeding for ‘less than four months’, ‘five to eight months’, ‘nine to twelve months’ or ‘over twelve months’. Von Kries *et al*. (1999) used categories to sort children by exclusive breastfeeding for the following durations, ‘never’, ‘less than two months’, ‘three to five months’, ‘six to twelve months’ or ‘over twelve months’. These differing

categories could cause confusion and make it difficult to compare the results of different studies. Mayer-Davis *et al*. (2006) categorised both partial and exclusive breastfeeding. Partial breastfeeding was categorised as 'never', 'less than one month', 'one to three months', 'four to six months', 'seven to nine months' or 'over nine months'. However, exclusive breastfeeding was recorded as 'more than six months' or 'less than six months'. This made it difficult to distinguish between the different effects that partial and exclusive breastfeeding might have, even within the same study.

Some studies such as Salsberry & Reagan (2005) and Beyerlein *et al*. (2008) used a single binary variable determining whether the infant was 'ever breastfed', giving no information on exclusivity or duration. Other studies such as Jiang & Foster (2012) used a truncated variable, measuring partial breastfeeding censored at twelve months due to restrictions in data collection. This could affect results because some children are partially breastfed for longer than twelve months. These additional breastfeeding variables add further to confusion when comparing the results of different studies.

As a result of different breastfeeding measurements, studies are often incomparable. Renfrew *et al*. (2005) suggested that researchers should agree upon definitions of breastfeeding which should then be used in any future studies. However, no single breastfeeding measurement has since been decided on and the use of different datasets often makes it difficult to consistently use the same definitions. This could be because different statistical models are used for different types of variables: continuous or binary *etc*.

The time that breastfeeding data was collected is also an important factor to consider. If data were recorded too early in infancy, some children may not have stopped breastfeeding, making it impossible to determine the full duration of breastfeeding. If the data were collected too late, maternal recall may be a problem. For example, Liese *et al*. (2001) investigated the relationship between both partial and exclusive breastfeeding on overweight in nine and ten year olds. Breastfeeding data were recalled by the mother, up to ten years after the birth of their child; recall over such a long period of time could be inaccurate or biased. However, Parsons *et al*. (2003), found evidence to suggest that mothers were likely to recall breastfeeding behaviour with a high accuracy.

Other studies have used different variables to instrument breastfeeding duration. For example, Del Bono & Rabe (2012) used the UNICEF baby friendly Initiative, Fitzsimons & Vera-hernández (2013) used the day and time of birth and Denny & Doyle (2008) used

delivery by Caesarean section. These variables are used in conjunction with the instrumental variable technique and are not used to directly measure breastfeeding duration. The instrumental variable technique is discussed later in Section 2.3.3.

The different breastfeeding variables used in the existing literature are shown in Table A-1. This table also gives the years of birth for children in each study and which country the data come from, making it easier to compare studies where breastfeeding trends, fashions and prevalence might have been similar.

Recommendations for Breastfeeding

Until 2001, the WHO recommended exclusive breastfeeding from birth until an infant was four months old. Since then, they have changed their recommendations to exclusively breastfeed from birth to six months. Fewtrell *et al*. (2011) discussed these recommendations in more detail. Recommendations such as those published by the WHO could influence maternal breastfeeding behaviour and the duration and exclusivity of breastfeeding that they provide. However, the mothers who act upon these types of recommendations are likely to be systematically different from those who do not. Most studies within the literature used participants who were born and breastfed before 2001, meaning that results from these studies might not be representative of more recent infants. This should be considered when comparing these studies to more recent breastfeeding behaviour.

2.2.2 Data and Methods

There have been a number of observational datasets and methodologies used in this literature. It is impossible to carry out randomised controlled trials (RCTs) using breastfeeding as a treatment because it is unethical to randomly prevent or force mothers to breastfeed. Additionally, RCTs might influence the normal behaviour of mothers to differ from how they would behave in the absence of a trial. Duflo *et al*. (2007) explained the potential bias caused by the Hawthorne and John Henry effects which inadvertently affect the behaviour of the treated and control groups, respectively. If a patient is aware that they are part of a randomised controlled trial they might act in a different way than they would otherwise have acted. For example, if a mother was told not to breastfeed as part of a RCT, she might try to make up for the lack of breastfeeding through other behaviours. For these reasons, RCTs might not be the best way to determine the causal effects of a lifestyle choice such as breastfeeding. As a result, data from observational

studies is the best way to proceed, as many studies in this literature have done. The advantages and disadvantages of the data and methodologies which appear in the literature are explained and analysed in the following subsections.

Datasets

One of the main issues found in data used throughout this literature is bias but few studies attempted to correct for this or determine whether it affects results or not. The types of bias in this literature include attrition, recruitment or sample selection bias and bias caused by missing data and these might have had an influence on any results. If a study suffered from bias but its aim was to inform policy at a population level, then sample weighting could produce more generalisable results. However, weighting is unnecessary as long as the results are interpreted appropriately and the study population is known. Very few studies in this literature have acknowledged any potential bias, let alone attempted to correct for it. This could cause results to be misinterpreted, especially if any attrition or missing values are correlated with the adiposity measures used in the analysis.

Throughout the literature, a variety of datasets have been used and each has advantages and disadvantages. Cross-sectional data allows children of different ages to be investigated. However, this makes it more difficult to identify when in childhood the effects are greatest. Cohort data allows a cohort of children to be followed over time and the effects of breastfeeding on childhood obesity at a range of ages for the same cohort can be identified but any results are specific to the cohort. Attrition and missing data is often a problem in some of the datasets and some studies suffer from attrition bias; disadvantaged children are often under-represented. Many of the datasets used here also have small sample sizes.

Salsberry & Reagan (2005) used a cross-section of children born in the US between 1982 and 1996 and similarly, Liese *et al*. (2001) used a cross-section of German children born between 1982 and 1984. Although using cross-sectional data often allows more children of the required age to participate in a study, it can cause problems. Children born at different times might be affected differently by confounding factors or by breastfeeding trends and prevalence. That said, using children who were not born during the same period could mean that bias is reduced when applying results to future cohorts.

Jiang & Foster (2012) used the Child Development Supplement (CDS) of the Panel Study of Income Dynamics, a panel dataset treated here as repeated cross-sections. The CDS

holds data on US children, aged between five and eighteen years, born between 1997 and 2002. Jiang & Foster (2012) included an age variable in their model to account for the different effects experienced by children of different ages. The addition of an age variable could have been problematic due to the fluctuating nature of BMI during childhood. However, Jiang & Foster (2012) avoided this problem by using a generalized propensity score approach which only matched children of similar ages. Despite this, the influence of breastfeeding on childhood BMI might also differ with age and by including children of all ages in their analysis the authors might have missed a specific time in childhood in which this relationship was larger than others. The study could have missed information which could be valuable for policy makers aiming to identify the best times to intervene during childhood in order to prevent obesity. This study also had a large proportion of missing data which meant a large number of participants were excluded from the analysis and consequently the sample could be biased. The data used by Jiang & Foster (2012) also had problems with how data on breastfeeding were recorded. Mothers were only asked about breastfeeding duration thirteen years after the birth of their child which could have led to recall bias. The data was also clustered around certain durations of breastfeeding; mothers tended to round to one, three or six months.

Many studies in this literature used cohort data to overcome bias caused by wide age ranges. For example, McCrory & Layte (2012) used the Growing Up in Ireland Study, a cohort of children born in 1997 and 1998, Oddy & Sherriff (2003) used the Western Australian Pregnancy Cohort Study (WAPCS), born between 1989 and 1992 and Reilly *et al*. (2005) used the Avon Longitudinal Study of Pregnancy and Children (ALSPAC), a cohort of UK children born in 1991 and 1992. Cohort data has the advantage of removing the problems caused by participants being born at different times. However, it is important to keep in mind when comparing the results of studies using cohort data, that each cohort was breastfed at different times and grew up during different years. Each of these factors could influence the results of a study.

Some studies used multiple waves of cohort data in order to follow the same children throughout their childhood. For example, Bergmann *et al.* (2003) used multiple waves of the German Multicentre Atopy Study to investigate how the adiposity of a cohort of childhood was affected by breastfeeding until six years of age. Burke *et al*. (2005) followed a cohort from birth to eight years and investigated the likelihood of overweight in children breastfed for different durations. Using multiple waves of data from the same cohort allows the impact of breastfeeding on adiposity to be investigated throughout

childhood without any bias relating to when the children were born. However, results obtained from one cohort may not be applicable to cohorts from earlier or later years due to the changing attitudes towards breastfeeding and the increasing prevalence of childhood obesity over time.

Attrition could also cause problems when using multiple waves of cohort studies. Bergmann *et al.* (2003) suffered from a high attrition rate, losing almost a third of participants over six years, possibly due to the inconvenience caused to participants by repeated physical examinations. This could have led to bias if the likelihood of dropping out was correlated with the adiposity measures. In this case, the authors carried out a Cochran-Mantel-Haenszel[18] test and claimed that the removed observations had no significant influence on results.

Some studies which used cohort data only used one wave of available data. For example, Reilly *et al*. (2005) used data from the 1998 to 2000 sweep of the ALSPAC, containing data on a cohort of children at seven years old, using a single cross-section of data and not taking full advantage of the multiple waves of available data. This data could also have suffered from attrition; the sample was of seven year old children and certain types of children might have been more likely to remain in the sample up to this age than others.

Some studies under- or over-represented certain groups of participants. Certain groups which were of more interest were often overrepresented so that any analysis had a high enough statistical power to obtain accurate results relating to these groups. For example, Burdette & Whitaker (2007) and Grummer-Strawn & Mei (2004) over-represented children from low-income families and Bogen *et al.* (2004) over-represented children from disadvantaged backgrounds. Disadvantaged children and those from lower-income families have a higher chance of being overweight or obese and a lower chance of being breastfed as infants making them an important group of children for policy makers. These children are generally more likely to drop out of studies when attrition occurs. Conversely, some studies had an under-representation of minority groups. For example, Reilly *et al.* (2005) used data from the ALSPAC which under-represents ethnic minorities. When interpreting results from studies which under-represent certain minority groups, it is important to apply them to the appropriate population. Any results from this study which were related to differences in ethnicity may be difficult to interpret

[18] Cochran-Mantel-Haenszel test uses repeated tests for independence on stratified categorical data.

correctly and results might not be applicable to children from the under-represented groups.

The number of observations in a study could also have influenced the accuracy of any results. Studies with larger sample sizes generally have higher statistical power and a lower type II error; low prevalence of overweight, obesity or breastfeeding would not be as problematic statistically in results from larger studies. The studies in this literature used a variety of sample sizes (the number of observations in each study is given in Table A-1 in Appendix A). Studies with low sample sizes generally focused on more specific populations and had weaker recruitment strategies which could have produced misrepresentative results. Many studies within the literature used large representative datasets. For example, Armstrong & Reilly (2002) used a large representative sample of 32,200 Scottish three year olds, only excluding participants that did not take part in the Child Health Surveillance Programme, a routine health check, or those who had missing or implausible values. Likewise, von Kries *et al*. (1999) used cross-sectional German data from an obligatory health examination consisting of over 9,000 children aged five and six years, in an attempt to keep bias to a minimum. Conversely, Bergmann *et al*. (2003) used a relatively small sample size of 918 children. They used data collected from six areas in Germany; two rural and four urban areas. Limiting data collection to only six areas in a country could cause bias within the sample. This was not acknowledged and nothing was done to correct for this bias to make the data more representative of the entire German population.

Further problems with bias could have been caused by recruitment in many studies. Both Oddy & Sherriff (2003) and Burke *et al*. (2005) used data from the WAPCS which recruited mothers from antenatal clinics between 1989 and 1992. This could have caused bias because the mothers who attended antenatal clinics may have had different demographic and social variables to those who did not. Mayer-Davis *et al.* (2006) and Gillman *et al*. (2001) used data from the Growing up Today Study which recruited children born to a cohort of nurses from the NHANES II study. As a result, all children in the study had at least one parent who had medical training. Results from studies like these could lead to problems in policy making.

Kramer *et al*, (2004) and Kramer *et al*. (2007) used data from the 'Promotion of Breastfeeding Intervention Trial' (PROBIT)[19] to estimate the effects of a randomised promotion of prolonged and exclusive breastfeeding on infant growth and childhood adiposity, respectively[20]. The PROBIT trials were carried out by Kramer *et al*. (2001) and collected data from thirty-one maternity hospitals in Belarus. These hospitals were involved in a cluster-randomised intervention trial based on the 'Baby-Friendly Hospital Initiative' (UNICEF, 2010) to promote breastfeeding created by the WHO and the United Nations Children's Fund (UNICEF). These trials aimed to investigate the effect of this breastfeeding promotion intervention on breastfeeding duration as well as other outcomes such as gastrointestinal, respiratory infection and atopic eczema during infancy. The original trial (Kramer *et al*., 2001) only included one year of follow up and included no information on childhood adiposity. Although childhood adiposity was not one of the original outcomes, Kramer later published a number of papers, including Kramer *et al*. (2003), Kramer *et al*, (2004), Kramer *et al*. (2007), Kramer *et al*. (2009) which used additional observational follow up data and investigated the effect of this breastfeeding intervention on a number of different outcomes by estimating the intention to treat effect (ITE). The PROBIT trials (Kramer *et al*., 2001) are the only RCT to my knowledge which has investigated the effects of breastfeeding interventions. They did not investigate the causal effects of breastfeeding participation but due to the ethical issues discussed previously, RCTs cannot be carried out on the effects of breastfeeding itself. The study by Kramer *et al*. (2007) estimated the ITE to investigate the influence of these breastfeeding promotion interventions on childhood adiposity outcomes. Similarly, Kramer *et al*. (2009) also investigated the ITE of the breastfeeding promotion intervention on childhood obesity. Neither of these studies found that the breastfeeding promotion intervention had any effect on childhood adiposity. However, the parameter estimates from these studies using the PROBIT trials are not directly relevant to the analysis presented in this chapter. The ITE is based on the assignment of the intervention which is randomised rather than whether the treatment is taken up (i.e. whether the child is or is not breastfed). This chapter aims to find the causal effect of breastfeeding itself on childhood adiposity rather than the effect of an intervention. The ITE estimated in these studies are identified not for an entire population but only for mothers who intended to breastfeed and who changed their breastfeeding behaviour as a result of the intervention.

[19] Despite this RCT being carried out in Belarus, a developing country, it is included in this literature review because it is the only RCT in the area of breastfeeding.

[20] Although randomising breastfeeding is generally considered unethical, the randomisation of breastfeeding promotion is not.

Additionally, the PROBIT trials only included mothers who intended to breastfeed and this is not the population of interest in this chapter. Furthermore, the PROBIT trials were carried out in Belarus which is a developing country and is therefore likely to be very different to the UK in its attitudes towards both breastfeeding and obesity. The effects of Chernobyl also affected mothers' attitudes towards breastfeeding. This makes it difficult to base potential UK policy implications on results from these studies.

Many studies in the literature suffered from missing data which could potentially cause biased results. Certain types of people might have been more likely to leave certain questions unanswered. For example, mothers with higher BMIs could have been less likely to report their weight. In this literature, the proportion of missing data varied between studies and observations with important missing data were generally removed from any analysis. For example, Mayer-Davis *et al*. (2006) dropped only 470 out of 16,882 initial participants (2.7%) in their cross-sectional analysis due to missing data, whereas, Jiang & Foster (2012) dropped 292 out of 3,563 participants (8.2%) due to missing data, in a study which was also cross-sectional. The proportion of missing data could be of great importance if data is not missing at random.

Some studies have used larger more nationally representative data such as the Millennium Cohort Study (MSC) (Del Bono & Rabe, 2012; Fitzsimons & Vera-Hernández, 2013) or the National Child Development Study (NCDS) (Denny & Doyle, 2008). However, each of these papers focused on childhood cognitive development rather than childhood adiposity. Although Del Bono & Rabe (2012) and Fitzsimons & Vera-Hernández (2013) referred to childhood overweight and BMI, respectively, Del Bono & Rabe (2012) used an adult definition of overweight and Fitzsimons & Vera-hernández (2013) included BMI only as part of a more complex health index. For this reason, it is not possible to compare the effects found in these studies to those in this chapter. The empirical work in this chapter focuses on childhood adiposity outcomes and therefore adds to the existing literature by using a large nationally representative dataset.

Key Variables

The inconclusive outcome of this literature is most likely due to the lack of clarity concerning confounding factors. It has been consistently found that confounding factors attenuate the relationship between breastfeeding and adiposity, but different studies find this attenuation to different extents. It remains unclear whether this correlation is the result of a causal influence or whether it is a result of confounding factors. The

confounding factors which were accounted for differed between studies depending on the data available, methodology used and the authors' interpretation of the literature.

In order to find the true extent of the causal relationship between breastfeeding and childhood adiposity, it is important that all important variables affecting the relationship are considered. Studies which failed to include important confounding variables could have suffered from omitted variable bias. If one important confounding variable was omitted, the model could have produced biased coefficients for other variables which were picking up the effects of the omitted variable. The impact of an omitted variable within a logit model was illustrated by Cramer (2005) who explained that coefficients in the logit regression were artificially reduced if other important variables were omitted.

Lamerz *et al*. (2005) found that higher parental education had a large, inverse, significant impact on the likelihood of obesity. There was also evidence found by Lindeboom *et al*. (2009) that higher parental education increased the likelihood of breastfeeding. SES has also been found to confound the relationship; children from families with higher SES have been found to have a reduced risk of obesity and higher likelihood of being breastfed. For example, Reilly *et al*. (2005) and Salsberry & Reagan (2005) all found some measure of SES to influence obesity and attenuate the effects of breastfeeding. Von Kries *et al*. (1999) failed to use any measure of SES. They argued that there was no German measure of SES available which was equivalent to those found in Britain because Germans were more reluctant to give information relating to their income. For the same reason, financial variables could not be used within any analysis. This failure to account for SES was criticised soon after by Wadsworth *et al*. (1999) in a letter to the journal.

Ethnicity could also have a large confounding influence on the relationship between breastfeeding and adiposity. As explained by Greene *et al*. (2008), people might be more or less likely to be classified as obese or overweight depending on their ethnicity[21]. Burdette & Whitaker (2007) found that Hispanic children were more likely to be considered obese compared to non-Hispanic, black or white children, possibly due to difference in body shapes. This study also found mothers of white and Hispanic children were more likely to breastfeed than mothers of black children, most likely due to the different cultures experienced by different ethnic groups. Studies in this literature took different views on the importance of ethnicity. Burdette & Whitaker (2007) looked specifically at the relationship between breastfeeding and obesity across different

[21] Greene *et al*. (2008) used an ordinal variable for ethnicity containing three groups: black, white and other.

ethnicities. However, studies such as Reilly *et al*. (2005) under-represented ethnic minorities which could have led to any effects caused by ethnic differences being underestimated or undiscovered. Further research could provide more information on the reasons for, and extent of, any differences in the relationship between breastfeeding and adiposity between ethnic groups including research into a wider variety of ethnicities.

Hediger *et al*. (2001) found a large correlation between maternal BMI and infant feeding method. Women with higher BMIs were less likely to breastfeed, possibly due to health complications. Unsurprisingly, they also found a strong correlation between the BMI of a mother and her child. However, it was unclear whether this was due to genetic similarities or shared eating habits and lifestyles experienced within families.

Smoking during pregnancy has repeatedly been linked with low birth weight. For example, Blake *et al*. (2000) concluded that mothers who smoked whilst pregnant had lighter babies. However, as children got older, smoking during pregnancy appeared to increase childhood weight. Salsberry & Reagan (2005) found maternal smoking during pregnancy increased the BMI of children between the ages of two and eight years. Further research into this area could help to discover why this relationship exists or whether it too is due to confounding factors, such as maternal education. Horta *et al*. (2001) carried out a systematic review looking at the relationship between maternal smoking habits and breastfeeding. They found that maternal smoking, especially during pregnancy, decreased the prevalence and duration of breastfeeding, possibly due to risks associated with smoking whilst breastfeeding which could have put smoking mothers off. Blake *et al*. (2000) also found that non-smoking mothers were likely to be from a higher SES, older and more likely to breastfeed. These characteristics could have further confounding influences.

Statistical Methods

Due to the ethical problems with randomising breastfeeding and the possibility of influencing normal maternal behaviour through Hawthorne or John Henry effects as mentioned earlier and discussed in more detail by Duflo *et al*. (2007), RCTs cannot be used and so observational data have often been used. A variety of statistical techniques have been applied to observational datasets in the existing literature.

Linear models, estimated using Ordinary Least Squares (OLS), have been used in the literature to estimate childhood BMI using information on breastfeeding along with other

factors. Beyerlein *et al*. (2008) used such a model but later criticised it and suggested some major flaws. Modelling mean BMI fails to identify how the entire childhood BMI distribution is affected. The standard deviation and other dynamics of the BMI distribution may hold important information that the mean cannot detect. Breastfeeding could prevent childhood underweight as well as overweight and obesity meaning that the mean BMI of breastfed children may not be significantly different from that of non-breastfed children, even if childhood BMI benefits from breastfeeding. Further statistical analysis would be needed to determine which children benefit most, if at all, in relation to their weight, as a result of breastfeeding. The standard deviation of the BMI percentiles gets larger as children get older and further issues occur when modelling BMI due to its fluctuations during childhood. These inconsistencies means that modelling children of different ages in a single model becomes difficult. Brion *et al*. (2011) used multiple linear regression to compare the effect of breastfeeding on the BMI of two cohorts of different aged children. As discussed earlier, this causes problems to arise because different levels of BMI are considered healthy in children of different ages. Furthermore, the authors set out to find the causal effect of breastfeeding on childhood BMI and although they found a significant effect they concluded that no causal inference could be made because the assumptions of their model were not appropriate.

Kramer *et al*. (2007) used a linear model but failed to identify these problems when modelling mean BMI. The assumptions imposed by linear models might also be problematic. Linear models assume that the data follow a linear functional form, a specific relationship whereby the mean of the outcome variable is linear in parameters. If this model specification is incorrect then it may give false or misleading results. OLS also assumes that all covariates are exogenous, that is, they are uncorrelated with the errors terms. However, if a variable is correlated with the error term and also with the outcome, then the variable is endogenous and an IV might be needed to help in obtaining unbiased estimates. The assumptions of the functional form imposed by linear models mean that they might not be the most appropriate model for this analysis.

In order to investigate how the distribution of BMI is affected by breastfeeding, Beyerlein *et al*. (2008) used a quantile regression. They found that BMI in children above the 90th BMI percentile was reduced and that a small yet statistically significant upward shift in BMI occurred in children below the 30^{th} BMI percentile. The use of the quantile regression highlighted the problems with linear models and the authors insisted that to detect the true relationship between breastfeeding and later BMI, the choice of the

dependent variable and the statistical method must be carefully considered. However, quantile regression models suffer from some of the same problems as the linear regressions. Similar to the linear model discussed previously, this model is also linear in parameters and imposes a functional form on the relationship.

The majority of studies within the literature used logit, or logistic regression, models to estimate the probability of a child being obese, overweight or 'at risk of overweight' (AROW). They did so by using binary variables such as those described in Section 1.2.1. Most of these logit models were adjusted for a variety of confounding factors and many studies provided odds ratios to describe the differences. Logit models suffer from some of the same problems as the other regression models discussed in this section. They assume that the latent variable is a linear combination of the covariates, that the logit function is the correct functional form and that the correct covariates are included in the model. Logit models also suffer from strong exclusion restrictions and if there are endogenous covariates then an IV estimator might be needed to correct for this. The logit model also assumes that observations are a random sample from a population. Studies such as Reilly *et al*. (2005) acknowledged that this could be a problem, although more generally in the literature, these assumptions often went unrecognised and were rarely investigated. Without ensuring that the correct functional form is being used, any results gained using a regression model could be unreliable.

Salsberry & Reagan (2005) used a first-order dynamic logit model to account for whether a child has previously been overweight. By using a model which accounted for previous overweight status, the authors attempted to determine an age at which breastfeeding affected childhood overweight. They used three logit models for three stages of childhood, age three, five and seven years. These dynamic logit models have the same assumptions as general logit models and therefore suffer from the same problems as the models discussed previously.

Burke *et al*. (2005) used Generalised Estimating Equations (GEEs) to determine the effects of breastfeeding on obesity and the pattern this relationship followed throughout childhood. GEEs estimate the parameters in panel versions of generalised linear models allowing for an unobserved correlation between ordinal outcomes over time. GEEs have much fewer assumptions than standard regression models and do not assume homoscedasticity or independent error terms. However, like other models used within the literature, GEEs impose a functional form, again, leading to the same potential

problems discussed previously. Additionally, they do not give a likelihood making it difficult to compare this method with others. For further information on GEEs see Liang & Zeger (1986).

Jiang & Foster (2012) used a generalised propensity score (GPS) approach to estimate the effect of partial breastfeeding duration on BMI. The continuous propensity score used to estimate the length of partial breastfeeding was modelled using a zero-inflated Poisson (ZIP) model to account for the large number of mothers who do not breastfeed at all. Once the propensity scores had been estimated using the ZIP model, they used a linear regression model adjusted for the GPS. They also use a general additive model (GAM) adjusted for the GPS in order to allow for a non-linear relationship between breastfeeding duration and BMI. The ZIP model, used here to estimate the propensity score, has an underlying assumption that data are not over-dispersed. This is because it has restrictive parameters and so it would not have worked well with this type of data. The breastfeeding data used by Jiang & Foster (2012) were likely to be over-dispersed due to clustering and the authors failed to acknowledge this or attempt to check for any over-dispersion. A zero-inflated negative binomial (ZINB) model, which would still account for the large number of mothers who chose not to breastfeed, would have introduced an extra parameter and allowed for over-dispersion. There are a number of further problems with using a ZIP model to estimate the propensity score in this way. A Poisson model should only be used for discrete data and any data it uses should only take integer values. However, in the data used by Jiang & Foster (2012) this was not the case, as can be seen in Figure 1 of their paper. They modelled months of breastfeeding experienced by a child, but Figure 1 shows that not all children were breastfed for a whole number of months. It might have been more sensible for the breastfeeding duration to be measured in weeks rather than months. This would also have allowed those who were breastfed but for less than one month to be more accurately measured rather than having a large jump from never breastfed to one month breastfeeding. A Heckman correction model could have allowed for non-integer values while also accounting for the large proportion of children who were never breastfed. In this study, the duration of breastfeeding was censored at twelve months. However, the Poisson model was not censored accordingly. By failing to censor the ZIP model at twelve months, Jiang & Foster (2012) could have produced inaccurate results. If there were a large number of children breastfed for longer than twelve months which were censored by data collection, then the ZIP model would have been artificially stretched to include the increased number of observations at twelve

months. A large number of observations towards the tail of the ZIP model would distort the distribution leading to shorter durations of breastfeeding being underestimated and longer durations of breastfeeding being overestimated. This means that any effect of breastfeeding could have been over- or under-estimated. There was no mention of model fit when estimating the propensity score using the ZIP model in the study by Jiang & Foster (2012). If the propensity score was modelled incorrectly any results from the GAM model could be unreliable. Alternative models estimating the propensity score or model fit tests could have assessed the robustness of these results.

Some studies in a closely related literature, namely the effects of breastfeeding on childhood cognitive development, have used instrumental variable techniques to identify the local average treatment effect (LATE) of breastfeeding on cognitive development (Del Bono & Rabe, 2012; Denny & Doyle, 2008; Fitzsimons & Vera-hernández, 2013). This is an important method, widely used in the literature investigating the influence of breastfeeding on cognitive ability. The instrumental variable technique is discussed in more detail and in the context of this chapter in Section 2.3.3. The suitability of any instruments vary with different outcome variables. What is a suitable instrument in the context or cognitive development, might or might not be suitable in the context of obesity.

2.2.3 Findings

The results within this literature are wide ranging and many of the findings are directly conflicting. Even studies which used similar data and methodology often produce different results. The results found heavily depend on data, the variables included in the model, how variables are measured and the methodological assumptions made. The context of any findings is important and some effects are only found in certain subgroups of children. Outlined below are some of the findings from previous studies and a summary of the results from the studies outlined in Table A-1, in Appendix A.

A number of studies within the literature, including Hediger *et al*. (2001), McCrory & Layte (2012), Oddy & Sherriff (2003) and Salsberry & Reagan (2005) found no relationship between breastfeeding and childhood adiposity. In most cases, this was because accounting for potential confounding factors removed the relationship between the two. However, other studies found that the relationship remained even after confounding factors were taken into account. These studies included Armstrong & Reilly (2002), Bergmann *et al*. (2003), Burke *et al*. (2005), Gillman *et al*. (2001), Mayer-Davis *et al*. (2006) and von Kries *et al*. (1999). The large differences between results emphasise

the need for further research using a more appropriate statistical method in order to determine whether there is a causal, protective effect of breastfeeding on childhood adiposity and to test whether sufficient confounding variables have been accounted for.

Beyerlein *et al*. (2008) found their results differed between models and dependent variables, even when using the same data. Using a linear model, no relationship was found between childhood BMI and breastfeeding. A logit model estimating the likelihood of overweight also found no relationship. However, a logit model estimating the likelihood of obesity found that children who were breastfed had a lower risk of obesity. Using a quantile regression model, the authors found that only children on certain parts of the BMI distribution were affected by breastfeeding. This could help to explain the inconsistency in results between studies which used different models and measurements.

Different adiposity and breastfeeding variables, as well as different models, can influence the type of effect that is found. For example, von Kries *et al*. (1999) found a dose response using logit models; as duration of breastfeeding increased, the likelihood of being obese decreased. Von Kries *et al*. (1999) had detailed data on breastfeeding duration until twelve months. If however, data on breastfeeding are insufficient, e.g. a single binary breastfeeding variable, then a dose response would be impossible to detect. The differences between dependent and treatment variables across the studies in the literature could be one reason for the variation in the findings.

Other studies such as Bogen *et al*. (2004) aimed to find the lowest duration of breastfeeding required to significantly protect against obesity in later life and whether the use of formula milk alongside breastfeeding lessened any protective effect that breastfeeding might have. Using a logit model they found that partial breastfeeding for at least twenty-six weeks or exclusive breastfeeding for at least sixteen weeks significantly reduced the likelihood of obesity in certain groups of children. These threshold responses show that breastfeeding over a specific duration decreased the likelihood of being obese during childhood. Threshold responses could be useful for policy makers who can then target their policies at increasing breastfeeding to a specific duration. Associating certain durations with positive outcomes for a child may also encourage mothers to continue breastfeeding until they reach target durations.

Some studies only found a relationship between breastfeeding and obesity in certain types of children, for example, in particular ethnic groups. Bogen *et al*. (2004) found an inverse relationship between breastfeeding duration and obesity that only existed in white

children whose mothers did not smoke during pregnancy. Similarly, Grummer-Strawn & Mei (2004) found that the relationship existed only in non-Hispanic white children. Conversely, Burdette & Whitaker (2007) used data on similar children and found that the relationship only existed in Hispanic children. The different effects found in different subgroups of children suggest the need for the appropriate context used when diseminating any results.

The results found by Jiang & Foster (2012) were surprising. They found that an increase in maternal intelligence by one intelligence quotient (IQ) point appears to increase breastfeeding duration by sixteen weeks. The authors claimed that this result was consistent with previous literature but although the literature has suggested that there was a positive relationship, this result was much larger in magnitude. Although it was not the effect of breastfeeding on childhood adiposity which was unusual and somewhat unrealistic in this study, it calls into question the methods that they used. It emphasises the importance of testing model assumptions wherever possible and acknowledging the disadvantages of any methodologies used.

2.2.4 Summary

The results of this review illustrate the need for further and more conclusive research into the effects of breastfeeding on childhood adiposity. The lack of consistency between previous studies and the limited statistical techniques used in much of the literature emphasise the need for an approach which is more carefully considered. The large quantity of literature on the relationship between breastfeeding and childhood adiposity demonstrates the interest and relevance of this research area. Childhood obesity is continuing to increase and further research into possible preventions could help to reverse this trend.

This empirical chapter aims to add to the existing literature in a number of ways. All analyses within the rest of this chapter will use data from a large scale cohort study. The MCS contains data representing the UK population and over-represents participants from ethnic minorities and disadvantaged families which are of interest when aiming to reduce obesity and improve breastfeeding participation. It builds on previous work by Beyerlein *et al*. (2008) to investigate the influence of breastfeeding on childhood adiposity using a range of methods. It extends their work by using a variety of econometric techniques which use assumptions not previously tested in the literature. It uses a range of methods more similar to the literature on breastfeeding and cognitive development and compares

a range of methods, taking a similar approach to that of Rothstein (2013). It also uses instrumental variables to investigate the effects of breastfeeding on childhood adiposity. This method has repeatedly been used in the cognitive development literature (Del Bono & Rabe, 2012; Denny & Doyle, 2008; Fitzsimons & Vera-hernández, 2013).

This chapter investigates the causal relationship between breastfeeding and childhood adiposity to be investigated using a range of different assumptions. This will produce more robust findings, as well as identifying the most appropriate techniques to use to investigate this relationship. Similarly, by using a range of adiposity variables the results found in this study can be compared to those in the literature. The breastfeeding or 'treatment' variables used throughout this chapter will be discussed further in Section 2.4 along with the independent variables considered to be potential confounders in the relationship between breastfeeding and childhood adiposity.

2.3 Methodology

Each of the methods outlined here will use observational data from the MCS due to the problems arising from the use of RCT data, discussed earlier. This chapter will investigate the impact of breastfeeding exclusivity and duration on a range of childhood adiposity measures[22]. It will aim to find robust evidence for the extent to which breastfeeding affects childhood adiposity by using a variety of methods which impose different assumptions.

This chapter takes a similar approach to Rothstein (2012) who compared a range of econometric techniques to investigate the influence of breastfeeding on early cognitive outcomes. Rothstein used weighted least squares (WLS), maternal fixed effects, propensity score matching (PSM) and instrumental variables (IVs) to investigate the relationship. They found little difference between the WLS and the PSM estimates and concluded that functional form was not an important issue. However, the PSM effect that they estimated was the average treatment effect on the treated (ATT) and is not directly comparable to the WLS which estimates the overall average treatment effect (ATE)[23]. Rothstein (2012) did not report the IV results because they were not believed to be valid. This chapter, like Rothstein (2012), uses a range of econometric techniques but rather

[22] A range of outcome measures need to be used because the different econometric techniques require different types of variables.
[23] Treatment effects are discussed in more detail in Section 2.3.2.

than investigating the effects of breastfeeding on cognitive ability, it investigates the effects of breastfeeding on childhood adiposity.

By using a range of different econometric techniques, the relationship between breastfeeding and childhood adiposity can be investigated using different sets of assumptions. Blundell and Costa Dias (2009) and Imbens and Wooldridge (2009) explain in detail the different models which assume selection on no unobservable confounding factors and those which do not as well as the differences between them. They explain how, if selection on unobservable confounding factors exists, then standard techniques such as OLS will produce inconsistent estimators and a model which accounts for the endogeneity of the treatment must be used. Further advantages and disadvantages of each of the econometric methods used in this chapter are discussed later.

This chapter aims to provide robust evidence of whether or not breastfeeding affects BMI, obesity and overweight during different stages of childhood as well as identifying the most appropriate statistical method for estimating a causal effect of breastfeeding on childhood adiposity. The methodologies used throughout this chapter are outlined in this section and a more detailed explanation of the models can be found in most econometrics textbooks, such as Cameron & Travedi (2005). Each methodology covered in this section will be carried out for the second, third and fourth waves of the MCS cross-sectionally to estimate the impact of breastfeeding on childhood adiposity at ages three, five and seven years old, respectively. This allows the parameters to differ for children of different ages and prevents the BMI of children of different ages from being included in a single model. However, although it does have the problem of using slightly different samples of children at each age due to the availability of data in each wave. This section concludes with a discussion of the advantages and disadvantages of the methodologies used in this chapter.

2.3.1 Selection on Observables

Initially, models which assume treatment selection only on observables will be used. These include the regression models used widely within the literature which will be replicated. Depending on the dependent variable, either linear or logit regression models have been used throughout the literature investigating the impact of breastfeeding on a range of childhood adiposity measures. An ordered probit model will then be used with a single dependent variable containing three discrete levels: normal weight, overweight and obese. Using an ordered probit model will allow comparison between the logistic

and normal distributions and act as a robustness check for the results obtained by the logit models in this study and in the existing literature.

Ordinary Least Squares

Linear regression models have been used in the literature to model expected childhood BMI for a given set of independent variables. This linear model can be written

$$\boldsymbol{y} = \boldsymbol{X}'\boldsymbol{\beta} + \boldsymbol{u} \tag{II.1}$$

where $\boldsymbol{y}$ is the outcome vector, $\boldsymbol{X}$ is a matrix of exogenous variables including an intercept, $\boldsymbol{\beta}$ is a vector of coefficients and $\boldsymbol{u}$ is a vector of random error terms. The linear model is usually estimated using OLS.

The marginal effect gives the effect of a change in one variable x_k, by one unit on the outcome $\boldsymbol{y}$. For the linear model, this is found by differentiating $\boldsymbol{y}$ with respect to the independent variable of interest, so that the marginal effect of x_k on $\boldsymbol{y}$ is

$$\frac{\partial \boldsymbol{y}}{\partial x_k} = \frac{\partial(\boldsymbol{X}'\boldsymbol{\beta} + \boldsymbol{u})}{\partial x_k} = \beta_k \tag{II.2}$$

where β_k is the average treatment effect (ATE) of a one unit increase in x_k. In cases where x_k has both a linear and a quadratic term, the marginal effect is

$$\frac{\partial \boldsymbol{y}}{\partial x_k} = \frac{\partial(\boldsymbol{X}'\boldsymbol{\beta} + \boldsymbol{u})}{\partial x_k} = \beta_{k_1} + 2x_k\beta_{k_2}. \tag{II.3}$$

The marginal effects in the linear model are calculated so that it is possible to compare with other models.

Linear regressions impose a functional form which has sometimes been considered restrictive and they assume that a model is linear in parameters. Misspecification of the model could lead to biased estimates of the parameters. If these assumptions are not met then the linear model could produce biased results. It is also possible that breastfeeding reduces BMI in overweight children but also increases BMI in children who would otherwise be underweight. This would mean that the variance of BMI distribution is smaller in breastfed children than in non-breastfed children, breaking the assumption of homoscedasticity.

OLS also assumes that all covariates are exogenous, that is, they are uncorrelated with the error terms in a model. However, if a variable is correlated with the error term due to

a variable omitted from the model, then the variable is endogenous and a model which accounts for selection on unobservable characteristics might be needed to obtain unbiased estimates.

Logit Model

Logit models (or logistic regression models) are frequently used to model binary variables such as obesity or overweight. Logit models will be estimated in this study, making it possible for comparisons to be made with results from previous studies.

Assume an unobservable latent variable $\boldsymbol{y}^*$, such that

$$\boldsymbol{y}^* = \boldsymbol{X}'\boldsymbol{\beta} + \boldsymbol{u} \tag{II.4}$$

where $\boldsymbol{X}$ is a matrix of observed exogenous covariates including an intercept, $\boldsymbol{\beta}$ is a vector of estimated coefficients and $\boldsymbol{u}$ is a vector of random error terms which are independently logistically distributed with a mean of zero and variance one. These error terms differ from those in the linear model which follow a normal distribution. It is assumed that the mean of the latent variable can be written as a linear combination of the parameters.

The observed binary variable $\boldsymbol{y}$ is then defined as

$$\boldsymbol{y} = \begin{cases} 1 & if\ \boldsymbol{y}^* > 0 \\ 0 & if\ \boldsymbol{y}^* \leq 0 \end{cases} \tag{II.5}$$

and consequently

$$P(\boldsymbol{y} = 1|\boldsymbol{X}) = \Lambda(\boldsymbol{X}'\boldsymbol{\beta}). \tag{II.6}$$

where $\Lambda(.)$ is the logistic cumulative distribution function (CDF), such that

$$\Lambda(\boldsymbol{X}'\boldsymbol{\beta}) = \frac{e^{\boldsymbol{X}'\boldsymbol{\beta}}}{1 + e^{\boldsymbol{X}'\boldsymbol{\beta}}}. \tag{II.7}$$

So, although the distribution of $\boldsymbol{y}^*$ is continuous, logit model has the following properties:

$$\begin{aligned} &\lim_{\boldsymbol{X}'\boldsymbol{\beta} \to +\infty} P(\boldsymbol{y} = 1|\boldsymbol{X}) = 1 \\ &\lim_{\boldsymbol{X}'\boldsymbol{\beta} \to -\infty} P(\boldsymbol{y} = 1|\boldsymbol{X}) = 0 \end{aligned} \tag{II.8}$$

so that once estimated, the logit model is non-linear. In contrast with the linear models, the magnitude of an effect varies with individual characteristics. In this non-linear model,

the estimated parameters only provide the significance and sign of an effect but not the magnitude. For this reason, marginal effects will be used to show the effect, at the mean of all covariates, of a change in a single covariate on the conditional probability that $\boldsymbol{y} = 1$, *ceteris paribus*.

Marginal effects are found by calculating the change in the conditional probability that $\boldsymbol{y} = 1$ given the set of independent covariates $\boldsymbol{X}$, that results from a unit change in the k^{th} covariate, x_k. Since $\Lambda'(z) = \Lambda(z)[1 - \Lambda(z)]$, it follows that if x_k has coefficient β_k then

$$\frac{\partial \boldsymbol{p}}{\partial x_k} = \frac{\partial \Lambda(\boldsymbol{X}'\boldsymbol{\beta})}{\partial x_k} = \frac{e^{\boldsymbol{X}'\boldsymbol{\beta}}}{1 + e^{\boldsymbol{X}'\boldsymbol{\beta}}}\left(1 - \frac{e^{\boldsymbol{X}'\boldsymbol{\beta}}}{1 + e^{\boldsymbol{X}'\boldsymbol{\beta}}}\right)\beta_k \qquad \text{(II.9)}$$
$$= \boldsymbol{p}(1 - \boldsymbol{p})\beta_k$$

where $\boldsymbol{p} = P(\boldsymbol{y} = 1|\boldsymbol{X})$ and again, β_k is the ATE of a one unit increase in x_k.

Marginal effects are preferred here over other parameters, such as odds ratios, because they can be calculated for different sets of observable characteristics.

As with the linear model, if this functional form is incorrect then estimates may be biased and give misleading results. This model, in the same way as the linear model, assumes selection into treatment depends only on observable characteristics.

Ordered Probit Model

In order to analyse ordinal discrete outcomes and following Mckelvey & Zavoina (1975), the ordered probit model will be used. The results from the ordered probit models will be compared to those from the logit model. Although logit and probit models give different parameter estimates due to the different error terms and different dependent variables, in practice the predictions they give are often very similar. The ordered probit model has been chosen because it easily takes into account the ordered nature of the dependent variable, weight status.

Similarly to the logit model, assume a latent variable,

$$\boldsymbol{y}^* = \boldsymbol{X}'\boldsymbol{\beta} + \boldsymbol{u} \qquad \text{(II.10)}$$

where $\boldsymbol{y}^*$ is unobserved and can take any value between $-\infty$ and $+\infty$ and $\boldsymbol{u}$ is an error term assumed to follow a standard normal distribution.

Similar to the outcome in the logit model, the observed values, $\boldsymbol{y}$ take only discrete values;

$$\boldsymbol{y} = \begin{cases} 0 & if\ \boldsymbol{y}^* \leq \mu_0 \\ 1 & if\ \mu_0 < \boldsymbol{y}^* \leq \mu_1 \\ 2 & if\ \boldsymbol{y}^* > \mu_1 \end{cases} \qquad \text{(II.11)}$$

where, in the present case, $\boldsymbol{y}$ takes the values 0, 1 and 2 to represent normal weight, overweight and obese, respectively. μ_0 and μ_1 are unknown threshold parameters to be estimated. So that, for example,

$$\begin{aligned} P(\boldsymbol{y} = 0|\boldsymbol{X}) = P(\boldsymbol{y}^* \leq\ \mu_0|\boldsymbol{X}) = P(\boldsymbol{X}'\boldsymbol{\beta} + \boldsymbol{u} \leq \mu_0|\boldsymbol{X}) \\ = P(\boldsymbol{u} \leq \mu_0 - \boldsymbol{X}'\boldsymbol{\beta}|\boldsymbol{X}) = \Phi(\mu_0 - \boldsymbol{X}'\boldsymbol{\beta}) \end{aligned} \qquad \text{(II.12)}$$

where $\Phi(.)$ is the standard normal CDF. Similarly, for $\boldsymbol{y} = 1$ and $\boldsymbol{y} = 2$ the probabilities of $\boldsymbol{y}$ given $\boldsymbol{X}$ are as follows;

$$P(\boldsymbol{y}|\boldsymbol{X}) = \begin{cases} P(\boldsymbol{y} = 0|\boldsymbol{X}) = \Phi(\mu_0 - \boldsymbol{X}'\boldsymbol{\beta}) \\ P(\boldsymbol{y} = 1|\boldsymbol{X}) = \Phi(\mu_1 - \boldsymbol{X}'\boldsymbol{\beta}) - \Phi(\mu_0 - \boldsymbol{X}'\boldsymbol{\beta}) \\ P(\boldsymbol{y} = 2|\boldsymbol{X}) = 1 - \Phi(\mu_1 - \boldsymbol{X}'\boldsymbol{\beta}) \end{cases} \qquad \text{(II.13)}$$

The threshold parameters μ_j, where j is the observed outcome of dependent variable $\boldsymbol{y}$, must be strictly increasing in order to insure that all probabilities are positive, so that

$$\mu_0 < \mu_1. \qquad \text{(II.14)}$$

When x_k has coefficient β_k, the marginal effect of x_k is

$$\frac{\partial P(\boldsymbol{y}|\boldsymbol{X})}{\partial x_k} = \begin{cases} \dfrac{\partial P(y = 0|\boldsymbol{X})}{\partial x_k} = -\phi(\mu_0 - \boldsymbol{X}'\boldsymbol{\beta})\beta_k \\ \dfrac{\partial P(y = 1|\boldsymbol{X})}{\partial x_k} = [\phi(\mu_0 - \boldsymbol{X}'\boldsymbol{\beta}) - \phi(\mu_1 - \boldsymbol{X}'\boldsymbol{\beta})]\beta_k \\ \dfrac{\partial P(y = 2|\boldsymbol{X})}{\partial x_k} = \phi(\mu_1 - \boldsymbol{X}'\boldsymbol{\beta})\beta_k \end{cases} \qquad \text{(II.15)}$$

Assuming that β_k is positive and holding $\boldsymbol{\beta}$ and μ constant, an increase in x_k is equivalent to shifting the distribution of $\boldsymbol{y}^*$ marginally to the right. In doing so, the probability of each outcome will change for some observations but it is impossible to determine which observations. In this case, $P(y = 0|\boldsymbol{X})$ will increase and $P(y = 2|\boldsymbol{X})$ will decrease. However, what happens to $P(y = 1|\boldsymbol{X})$ is ambiguous and depends on the size of the marginal effects for $y = 0$ and $y = 2$ as all marginal effects must sum to zero. The

opposite would be true for a negative β_k which would cause a shift in the distribution of $\boldsymbol{y}^*$ to the left.

As with the logit models, marginal effects are preferred over odds ratios making it possible to look at the effects on individuals with different characteristics. Again, a functional form is imposed by this model and this could be restrictive.

2.3.2 Removing the Functional Form

In each of the methods discussed so far, a functional form is assumed in the outcome equation, imposing a specific relationship between the outcome and the independent variable of interest or 'treatment'. In reality, the functional form in the outcome equation is unknown.

Propensity Score Matching

PSM is a semi-parametric technique which estimates the effects of a treatment, in this case a binary treatment. PSM is semi-parametric and does not require the parametric assumptions of the regression models discussed so far. For this reason, it does not impose a restrictive functional form on the relationship between breastfeeding and childhood adiposity. However, a number of further assumptions are required to identify treatment effects. This technique, in line with the methods discussed in the previous section, assumes selection only on observable characteristics.

PSM is used to investigate the expected difference in an outcome $\boldsymbol{y}$ between treated and untreated observations. It allows treated and untreated observations to be matched using a single score rather than matching on each individual characteristic which is often unfeasible if there are a large number of covariates. Matching allows RCTs to be imitated in the presence of selection bias due to observables without the ethical problems and changes in behaviour associated with RCTs. PSM estimates the effects of a treatment on an outcome after accounting for independent characteristics which influence an individual's likelihood of treatment. The binary treatment $\boldsymbol{d}$ takes the value 1 if an observation is treated and takes the value 0 if the observation is untreated. A range of treatment variables will be investigated using this method and are described later in Section 2.4. The treated and untreated groups are mutually exclusive; if an individual is observed to be treated then they cannot also be observed to be untreated. Consequently, there exists no counterfactual for a treated observation in the absence of treatment, or for an untreated observation in the presence of treatment. PSM uses observations from the

untreated group who have the same likelihood of treatment as observations in the treated group, and vice versa, to estimate these counterfactuals. Studies which have previously used PSM to investigate the effects of breastfeeding on child outcomes include Jiang *et al*. (2011) and Iacovou & Sevilla-Sanz (2010) and Rothstein (2012) which all investigated the effects on cognitive outcomes.

The remainder of this subsection will discuss the propensity score, how it is estimated and the assumptions that it requires. It will introduce the parameters of interest, or treatment effects and the assumptions that they each impose. It will discuss different matching algorithms and the assumptions that are required when matching using a propensity score and it will conclude by discussing how PSM will be used in this study.

The Propensity Score

This chapter will use a propensity score to estimate the probability of treatment for each observation. Propensity scoring is the most common matching method in the econometrics literature and has previously been used to estimate the probability of breastfeeding in a similar way by Iacovou & Sevilla-Sanz (2010) who investigated the effects of breastfeeding on childhood cognitive development. The propensity score is a function of independent variables $\boldsymbol{W}$,

$$p(\boldsymbol{W}) = P(\boldsymbol{d} = 1|\boldsymbol{W}), \qquad \text{(II.16)}$$

where $p(\boldsymbol{W})$ is the propensity score given observable characteristics $\boldsymbol{W}$[24]. This gives the probability of treatment given $\boldsymbol{W}$. Here, probit models will be used to estimate the propensity score for a range of binary breastfeeding treatments.

There are a number of assumptions which must be taken into consideration when using propensity scores. These are explained here and used throughout this section. Firstly, the conditional independence assumption (CIA) states that, conditional on $\boldsymbol{W}$, the outcome is independent of treatment,

$$\boldsymbol{y}_0, \boldsymbol{y}_1 \perp \boldsymbol{d}|\boldsymbol{W}, \qquad \text{(II.17)}$$

where $\boldsymbol{y}_0$ is the outcome in the absence of treatment and $\boldsymbol{y}_1$ is the outcome in the presence

[24] Here, independent variables are represented by vector $\boldsymbol{W}$ rather than $\boldsymbol{X}$. This is because the vector includes variables which influence breastfeeding but are not considered to influence childhood adiposity. Vector $\boldsymbol{W}$ includes independent variables used in the previously discussed regression models $\boldsymbol{X}$ as well as instruments $\boldsymbol{Z}$ used to predict breastfeeding behaviour. This is explained in more detail in Section 2.4.3 which discusses the independent variables used and the models that they are each included in.

of treatment. The CIA states that the outcome is independent of treatment once observable characteristics are accounted for. Different versions of this assumption are needed when estimating different parameters of interest which will be discussed later.

The balancing condition,

$$\boldsymbol{d} \perp \boldsymbol{W}|p(\boldsymbol{W}), \tag{II.18}$$

is an important consequence of the CIA and states that treatment is independent of observable characteristics for any given propensity score. Matching on a propensity score was first proven to succeed by Rosenbaum & Rubin (1983) who showed that, if the CIA assumption is met and there is conditional independence on observable characteristics $\boldsymbol{W}$, then it is possible to also assume that there is also conditional independence on the propensity score,

$$\boldsymbol{y} \perp \boldsymbol{d}|\boldsymbol{W} \Rightarrow \boldsymbol{y} \perp \boldsymbol{d}|p(\boldsymbol{W}). \tag{II.19}$$

This means that matching on the propensity score is sufficient to remove bias caused by each observable characteristic used to estimate the propensity score. A full proof of this theorem can be found in Rosenbaum & Rubin (1983). Equation (II.19) emphasises the importance of the CIA assumption; matching on a propensity score is only valid when the CIA holds and all confounding factors are included in the estimation of the propensity scores. Equation 3.21 also makes it possible to match the treated and untreated samples by their propensity score. This is much simpler than matching the observations on each of the observable characteristics individually because there is a single function $p(\boldsymbol{W})$, rather than a large number of covariates. The property shown in Equation 3.21 is only true as sample sizes approach infinity and so large sample sizes are essential when using PSM.

Treatment Effects

PSM produces three parameters of interest which illustrate the effect a treatment has on an outcome are discussed here. They are the average treatment effect on the treated (ATT), the average treatment effect on the untreated (ATU) and the average treatment effect (ATE). The ATE is the most comparable to the estimated treatment effects provided using the other methods in this chapter. However, each of these treatment effects are estimated in a different way to the previous models because they estimate unobserved counterfactuals in order to obtain a causal inference. PSM allows each of

these parameters to be estimated and the effects of treatment on different subgroups to be predicted. These treatment effects are discussed in more detail here.

The ATT, ATU and ATE are defined as follows:

$$\text{ATT} = E[\boldsymbol{\Delta}|\boldsymbol{d} = 1] = \frac{1}{n_t}\sum_{i=1}^{n_t}[\Delta_i|d_i = 1] \tag{II.20}$$

$$\text{ATU} = E[\boldsymbol{\Delta}|\boldsymbol{d} = 0] = \frac{1}{n_u}\sum_{i=1}^{n_u}[\Delta_i|d_i = 0] \tag{II.21}$$

$$\text{ATE} = E[\boldsymbol{\Delta}|\boldsymbol{d}] = \frac{1}{n}\sum_{i=1}^{n}[\Delta_i] \tag{II.22}$$

where $\boldsymbol{\Delta} = \boldsymbol{y}_1 - \boldsymbol{y}_0$, n is the total number of observations, $n_t = \sum_{i=1}^{n} d_i$ is the number of treated observations and $n_u = n - n_t$ is the number of untreated observations. Each of these treatment effects requires different assumptions in order to be estimated. The assumptions required by each will be discussed below.

The ATT in Equation II.20 gives the difference in outcome between the treated and untreated states, given treatment. However, $\boldsymbol{\Delta} = \boldsymbol{y}_1 - \boldsymbol{y}_0$ is unobservable because it contains a counterfactual. The ATT can be split into an observable part and an unobservable or counterfactual part so that

$$\begin{aligned}\text{ATT} = E[\boldsymbol{\Delta}|\boldsymbol{d} = 1] &= E[\boldsymbol{y}_1 - \boldsymbol{y}_0|\boldsymbol{d} = 1] \\ &= E[\boldsymbol{y}_1|\boldsymbol{d} = 1] - E[\boldsymbol{y}_0|\boldsymbol{d} = 1]\end{aligned} \tag{II.23}$$

where $E[\boldsymbol{y}_1|\boldsymbol{d} = 1]$ is the outcome of the treated given treatment and $E[\boldsymbol{y}_0|\boldsymbol{d} = 1]$ is the counterfactual, that is, the outcome of the untreated given treatment.

A number of assumptions are required in order to estimate the ATT when using PSM. The ignorability assumption,

$$\boldsymbol{y}_0 \perp \boldsymbol{d}|\boldsymbol{W}, \tag{II.24}$$

is required and suggests that there are no omitted variables and hence, no further confounding factors, once all covariates in the model are accounted for. The ignorability assumption, a weaker version of the CIA, makes it possible to draw conclusions about causality rather than a simple correlation or association. This is because the

counterfactual (untreated) outcome is independent of treatment given the covariates $\boldsymbol{W}$.

An assumption of common support is also required to calculate the ATT when using PSM.

$$P[\boldsymbol{d} = 1|\boldsymbol{W}] < 1 \tag{II.25}$$

implies that no covariate $\boldsymbol{W}$ can ensure participation in treatment.

The ATU from Equation (II.21) gives the difference in outcome between the treated and untreated states in the absence of treatment. Again, $\boldsymbol{\Delta}$ is unobservable due to a counterfactual.

$$\begin{aligned} \text{ATU} = E[\boldsymbol{\Delta}|\boldsymbol{d} = 0] &= E[\boldsymbol{y}_1 - \boldsymbol{y}_0|\boldsymbol{d} = 0] \\ &= E[\boldsymbol{y}_1|\boldsymbol{d} = 0] - E[\boldsymbol{y}_0|\boldsymbol{d} = 0] \end{aligned} \tag{II.26}$$

where $E[\boldsymbol{y}_1|\boldsymbol{d} = 0]$ is unobservable and shows the counterfactual outcome of the treated in the absence of treatment and $E[\boldsymbol{y}_0|\boldsymbol{d} = 0]$ is the observable outcome of the untreated.

The ATU requires a weakened version of the CIA,

$$\boldsymbol{y}_1 \perp \boldsymbol{d}|\boldsymbol{W}, \tag{II.27}$$

which states that the outcome of the treated is independent of treatment given observable characteristics $\boldsymbol{W}$. This suggests that there are no omitted variables once these characteristics $\boldsymbol{W}$ are accounted for. Again, this allows a causal relationship to be estimated rather than a simple association.

The ATU also requires an assumption of common support,

$$0 < P[\boldsymbol{d} = 1|\boldsymbol{W}], \tag{II.28}$$

which implies that no covariate included in $\boldsymbol{W}$ can ensure the absence of treatment.

The ATU also requires the conditional mean independence assumption,

$$E[\boldsymbol{y}_0|\boldsymbol{d} = 1, \boldsymbol{W}] = E[\boldsymbol{y}_0|\boldsymbol{d} = 0, \boldsymbol{W}] = E[\boldsymbol{y}_0|\boldsymbol{W}], \tag{II.29}$$

which implies that the outcome of untreated observations does not influence treatment participation.

The ATE from Equation (II.22) is used when the expected gain from treatment on a random member of a population is required. It is an average of the ATT and ATU, weighted by the probability of treatment, so that

$$\begin{aligned}\text{ATE} &= P(\boldsymbol{d}=1)\{ATT\} + P(\boldsymbol{d}=0)\{ATU\} \\ &= P(\boldsymbol{d}=1)\{E[\boldsymbol{y}_1|\boldsymbol{d}=1] - E[\boldsymbol{y}_0|\boldsymbol{d}=1]\} \\ &+ P(\boldsymbol{d}=0)\{E[\boldsymbol{y}_1|\boldsymbol{d}=0] - E[\boldsymbol{y}_0|\boldsymbol{d}=0]\}\end{aligned} \quad \text{(II.30)}$$

where $E[\boldsymbol{y}_1|\boldsymbol{d}=1]$ and $E[\boldsymbol{y}_0|\boldsymbol{d}=0]$ are observable and $E[\boldsymbol{y}_0|\boldsymbol{d}=1]$ and $E[\boldsymbol{y}_1|\boldsymbol{d}=0]$ are counterfactuals.

When using PSM, estimating the ATE requires stronger assumptions than estimating the ATT or ATU. Firstly, the ATE requires the full version of the CIA in Equation (II.19), which states that the outcome is independent of treatment, given observable characteristics $\boldsymbol{W}$. For example, breastfeeding should not depend on the BMI of breastfed or non-breastfed children once all observable characteristics are accounted for.

A stronger assumption of common support is also required to estimate the ATE. This is

$$0 < P[\boldsymbol{d}=1|\boldsymbol{W}] < 1, \quad \text{(II.31)}$$

which states that for every observable characteristics, the probability of treatment lies between 0 and 1. This ensures a region of common support in which the treated and untreated samples overlap. It implies that there is no observable characteristic $\boldsymbol{W}$ which ensures certain participation or abstention from treatment.

Each of the treatment effects outlined here also require the conditional mean independence assumption,

$$E[\boldsymbol{y}_0|\boldsymbol{d}=1,\boldsymbol{W}] = E[\boldsymbol{y}_0|\boldsymbol{d}=0,\boldsymbol{W}] = E[\boldsymbol{y}_0|\boldsymbol{W}], \quad \text{(II.32)}$$

which implies that the outcome of untreated observations does not influence treatment participation and this allows the estimated treatment effects to ascertain a causal effect of the treatment on the outcome. This is because the counterfactual outcomes are independent of treatment, given the observable characteristics.

In the context of this chapter, policy makers will be more interested in the ATE. The reasons for this are discussed in Faria *et al*. (2015), a technical support document created for NICE which discusses the use of different treatment effects. The ATE will give policy makers an estimated gain to a random member of the population if they were to breastfeed, irrespective of whether they are in the treated or untreated groups. This treatment effect will be the most relevant if policies aim to improve breastfeeding in a population wide context of population wide policies. The ATU and ATT will also be

estimated and displayed in Appendix A, but are of less interest in the context of public health guidance.

Matching

In order for the treatment effects to be calculated, the propensity score for each observation is used to match treated and untreated observations. Matching requires a rich set of observable characteristics and also imposes the stable unit treatment value assumption also known as SUTVA. This means that any treatment given does not directly impact on untreated observations; i.e. there are no general equilibrium effects.

There are a number of different algorithms which allow treated observations to be matched with one or more untreated observation. Each algorithm has strengths and weaknesses and it is often unclear which algorithm is the most appropriate. As explained by Smith (2000), matching algorithms will asymptotically produce the same result, as the matches tend to perfect matches. However, the trade-off between bias and variance is important in finite samples. An increased number of untreated observations matched to each treated observation reduces variance but increases the bias in the estimated treatment effects. This is due to an increased likelihood of 'bad matches' as explained by Caliendo & Kopeinig (2008). Conversely, fewer matches will reduce bias but will also increase variance.

In finite samples, results can be very sensitive to the choice of matching algorithm. If different matching algorithms give similar results then the choice of algorithm is generally considered unimportant. For the purposes of this chapter a nearest neighbour algorithm will be used and this algorithm is discussed here. A more thorough explanation of this other matching algorithms can be found in a paper by Caliendo & Kopeinig (2008).

The nearest neighbour (NN) algorithm is the most common matching algorithm. Each treated observation is matched with k 'nearest neighbours', or untreated observation(s) with the closest propensity scores. NN matching can be done with or without replacement. If it is performed with replacement then a control, or an untreated match, can be matched to more than one treated observation. This 'oversampling' is useful if there are fewer untreated observations than there are treated observations. If performed without replacement then a control can only be matched to one treated observation and then cannot be used again. Increasing the number of neighbours will reduce the variance, but increase bias. The reason for using this matching algorithm over other algorithms is

that in this study, the treated groups are often much smaller than the untreated groups; the nearest neighbour algorithm allows each treated observation to be matched with a number of untreated observations and allows more of the available data to be used. Additionally, a calliper allowing only matches between treated and untreated observations which have propensity scores within a certain range of each other, can be implemented to prevent bias matches but this could increase the variance.

Propensity Score Matching, Breastfeeding and Childhood Adiposity

The PSM used in this chapter will investigate the effects of a range of breastfeeding treatments (see Section 2.4.2) on a range of childhood adiposity outcomes (see Section 2.4.1). Initially NN matching with replacement is used. The number of neighbours is the number that gives the best balance between bias and variance. Other types of matching algorithms are also used to perform robustness checks.

The PSM, in this chapter, will be performed using the 'psmatch2' a user-written Stata command by Leuven & Sianesi (2012). This command produces biased estimates because it assumes that the propensity score is known rather than estimated. Using bootstrapping when estimating the asymptotic variance of a matching estimator has been shown by Abadie & Imbens (2008) to also provide biased estimates of standard errors because of the same assumption. Bootstrapping uses asymptotic theory to estimate a distribution around an estimator or test statistic when they are unavailable and bootstrapping allows confidence intervals around the ATE to be estimated. This is done by estimating the ATE a number of times using a randomly drawn set of observations with replacement and estimating the confidence intervals using the results. This makes it possible to see any significant difference in the outcome of different groups of observations, for example, children who were breastfed for different lengths of time. This could help to identify dose responses for the treatments. The post-estimation command *pstest* was also used to investigate the balance on each covariate before and after matching.

The NN estimation of the PSM parameters in this chapter will use bootstrapping with 500 repetitions to calculate standard errors and estimate confidence intervals around the ATE. However, it is important to remember that these standard errors could be biased due to the reasons given above and explained by Abadie & Imbens (2008).

Since this analysis was carried out, a new command, *teffects psmatch* has become available in a more recent version of the software, Stata 13 (2013). This new inbuilt command acknowledges that the propensity scores are estimated and can therefore produce robust standard errors. However, the calculations used to estimate these robust standard errors, given by Abadie & Imbens (2009) and implemented in the new command, require all treated observations to have at least one match within any specified calliper. As a result of this difference between the new (*teffects psmatch*) and old (*psmatch2*) commands, the analysis which was performed using the *psmatch2* command could not be repeated using the *teffects psmatch* command with the same sample of observations. For this reason, it was not possible to re-estimate the results from the PSM using the new command within the time available to complete this thesis. Abadie & Imbens (2009) showed that their robust standard errors were always more efficient when estimating the ATE, resulting in reduced standard errors and more significant estimates[25]. Given that the emphasis in this chapter will be on the ATE in order to inform population wide policy implications and ease of comparison with other models, this is not considered to be detrimental to the main findings.

2.3.3 Unobservables Confounders: The Potential Problem of Endogeneity

Each of the methods outlined above have so far assumed that selection into breastfeeding depends only on observable characteristics. However, this is a potential problem if selection is also determined by unobservable confounding factors. In the regression models used so far[26], childhood adiposity measure $\boldsymbol{y}$ was a function of breastfeeding $\boldsymbol{d}$ and other independent variables $\boldsymbol{X}$, so that

$$\boldsymbol{y} = \boldsymbol{f}(\boldsymbol{d}, \boldsymbol{X}), \tag{II.33}$$

and all explanatory variables were assumed to be exogenous. However, it is possible that breastfeeding $\boldsymbol{d}$ is endogenous. That is, breastfeeding could be correlated with the error term in the model and considered endogenous in the child adiposity equation. This endogeneity could be due to unobservable maternal influences or unobservable initial endowments of the child which affect both the outcome and the propensity to breastfeed.

For this reason, two additional techniques will be used to explore the relationship between breastfeeding and childhood adiposity which account for the possibility that breastfeeding

[25] This is not the same for the ATT and ATU, for which the new command yields robust standard errors which can be either smaller or larger than the previous command.
[26] The regression models used so far are the OLS, logit models and ordered probit models.

is endogenous. These techniques are a two stage instrumental variable (IV) used in a linear model and a restricted version of a Roy model which jointly estimates an outcome and a treatment equation. These techniques are now discussed in more detail, along with how they each tackle the potential problem of endogeneity.

Instrumental Variable Technique

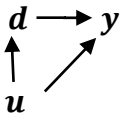
This section outlines the IV technique which could be used to account for the possible endogeneity of breastfeeding variables within the regression models used in this chapter. In doing so, this method, unlike the previous methods discussed, accounts for selection on unobservable as well as observable characteristics. For example, OLS assumes that all independent variables are uncorrelated with the error term in the outcome equation in order to estimate consistent coefficients. If any variable(s) are endogenous then the true causal effect cannot be estimated, only a correlation. The instrumental variable technique aims to produce consistent coefficients by instrumenting the independent variable(s). Exogenous variables are instruments for themselves and endogenous variables require instrument(s) $\boldsymbol{Z}$ which are correlated with the endogenous variable(s) but not with the error term. Instrument(s) $\boldsymbol{Z}$ would not have been included in the previous models' outcome equations because they are assumed to causally influence breastfeeding but not childhood adiposity. Therefore they are included in treatment equations but not in outcome equations throughout this chapter. For this reason, they were included in the estimation of the propensity scores in the PSM and will be included in the first stage of the 2SLS estimation discussed here. This is in line with all econometric textbooks[27] as well as papers which compare econometric methods in a similar way to this chapter, such as Rothstein (2013).

In a simple linear model,

$$\boldsymbol{y} = \boldsymbol{X'\beta} + \boldsymbol{\delta d} + \boldsymbol{u} \qquad \text{(II.34)}$$

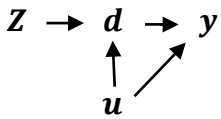
where $\boldsymbol{\delta}$ is assumed to be the ATE of treatment $\boldsymbol{d}$ and is comparable to the ATE of x_k defined in Equation (II.22), the treatment $\boldsymbol{d}$ would be endogenous if it were correlated with the error term $\boldsymbol{u}$. In this case, there is a direct effect on the outcome $\boldsymbol{y}$, through $\boldsymbol{\delta}$ but also an indirect effect on $\boldsymbol{y}$ resulting from the influence of $\boldsymbol{u}$ on $\boldsymbol{d}$. This can produce biased and inconsistent estimates of $\boldsymbol{\delta}$.

[27] For an empirical example see page 92 of 'Mostly Harmless Econometrics' by Angrist & Pischke (2008) which explicitly shows the covariates and instruments included when comparing OLS and 2SLS estimations.



In this study, an IV could help to remove the potentially endogeneity of breastfeeding. It is possible that breastfeeding has a direct effect on childhood BMI but also an indirect effect due to unobserved confounding factors.

A valid instrument(s) $\boldsymbol{Z}$ must not have a direct effect on the dependent variable $\boldsymbol{y}$ or an indirect effect on $\boldsymbol{y}$ through any omitted variable. It must only influence $\boldsymbol{y}$ indirectly through the endogenous variable $\boldsymbol{d}$. A valid instrument must be uncorrelated with the error term, $\boldsymbol{u}$ and a strong instrument is theoretically and causally strongly correlated with the endogenous variable.



In this study, a valid instrument will not have a direct effect on childhood BMI or effect childhood BMI through any unobservable characteristic. Any effect it has on BMI should be indirectly, through its effect on breastfeeding. For an instrument to be strong it should be significantly correlated with the endogenous treatment, in this case breastfeeding. Test for a weak instrument will be reported along with the results of the 2SLS approach.

Once a potential instrument is identified, a two-stage model is estimated using two-stage least squares (2SLS). The 2SLS performs two linear regressions using OLS. First, the endogenous variable is estimated:

$$\text{stage 1:} \quad \boldsymbol{d} = \boldsymbol{W\gamma} + \boldsymbol{\varepsilon} \tag{II.35}$$

$$\boldsymbol{d} = \boldsymbol{X'\gamma}_1 + \boldsymbol{Z\gamma}_2 + \boldsymbol{\varepsilon}$$

where $\boldsymbol{X}$ is the same set of exogenous variables which were included in the previous regression models, and $\boldsymbol{\gamma}_1$ is a matrix of corresponding coefficients, $\boldsymbol{z}$ is the instrumental variable and $\boldsymbol{\gamma}_2$ is the corresponding coefficient and $\boldsymbol{\varepsilon}$ is a standard normally distributed error term vector. The predicted value of the endogenous variable is then used in the outcome equation,

$$\text{stage 2:} \quad \boldsymbol{y} = \boldsymbol{X'\beta}_1 + \widehat{\boldsymbol{d}}\boldsymbol{\beta}_{2SLS} + \boldsymbol{\epsilon} \tag{II.36}$$

where $\boldsymbol{X}$ is the same set of exogenous variables as in the previous stage and $\boldsymbol{\beta}_1$ is a matrix

of corresponding coefficients in this outcome equation, $\widehat{\boldsymbol{d}}$ are the predicted values from the first stage and $\widehat{\boldsymbol{\beta}}_{2SLS}$ is the asymptotically normally distributed 2SLS estimator which is unbiased under the assumption that $\boldsymbol{d}$ is endogenous and that the instrument is strong. Finally, $\boldsymbol{\epsilon}$ is a standard normally distributed vector of error terms.

This IV approach identifies a different treatment effect to the methods discussed so far. In using an instrument, the local average treatment effect (LATE) rather than the ATE is identified. This means that the treatment effect estimated is the average treatment effect for a subpopulation of observations. These 'local' observations are those which are influenced by the chosen instrument to participate in treatment but which otherwise would be untreated. This is not always the subpopulation of interest and means that results using this method are not directly comparable to those found using the other methods because they apply to a different group of people. The LATE also depends heavily on the chosen instrument and differs when different instruments are used because it is identified for a different subpopulation. In contrast, the ATE identifies the average treatment effect over the entire sample and does not depend on the choice of an instrument. The 2SLS estimation will be carried out using the user-written Stata command *ivreg2* (Baum *et al*., 2010).

Despite a lack of literature which uses instrumental variable techniques to investigate the effects of breastfeeding on childhood adiposity, a number of previous studies have investigated the causal influences of breastfeeding on other outcomes, in particular childhood cognitive development. For example, Denny & Doyle (2008) used caesarean sections, Rothstein (2012) used breastfeeding rates by geographical area and differences in state laws in relation to breastfeeding in public and the workplace, Del Bono & Rabe (2012) used distance from the nearest hospital with breastfeeding support from the Baby Friendly Initiative and Fitzsimons & Vera-Hernández (2013) used the day of birth.

Following Denny & Doyle (2008), this chapter will use caesarean section as an instrument for breastfeeding. In order for an instrument to be suitable, it is required to be (a) correlated with the potentially endogenous variable (breastfeeding) and (b) not causally influence the dependent variable (BMI) or be correlated with the error term in the model. In the existing literature, it is well-established that caesarean sections are associated with a lower probability of initiating breastfeeding and shorten the duration of breastfeeding, see for example, Perez-Escamilla *et al*. (1994), Rowe-Murray & Fisher (2002), Perez-Escamilla *et al*. (1996), Merten & Ackermann-Liebrich (2004) and Chien & Tai (2007).

This reduction in breastfeeding initiation and duration is attributed to the delay in skin to skin contact between the mother and child, which can hinder the chances of successful breastfeeding practices. This suggests that caesarean sections satisfy the first condition for a valid instrument.

However, when it comes to the second requirement it is less clear cut. Three things to consider are a) unobservable attitudes which might influence a mothers' choice of Caesarean section as well as the BMI of their children in later childhood, b) unobservable health status which might influence the need for emergency Caesarean section as well as subsequent childhood BMI and c) the influences of Caesarean sections on childhood BMI through the effects on the child's digestive system.

The first two points were discussed in detail by Del Bono & Rabe (2012). They suggested that it is questionable whether or not individual-level characteristics could be completely excluded from the main equation. Although, their main equation used a different outcome to this study, the same theoretical problems might still apply here. In many cases, mothers can choose whether to have a Caesarean section or not and that this could mean that unobservable characteristics, for example attitudes towards health, which could influence both the choice of Caesarean section as well as childhood BMI. As well as attitude variables, there could be more objective health variables which might influence both the occurrence of emergency Caesarean section and subsequent childhood BMI. These might include gestational diabetes, or abnormal growth during gestation.

These issues highlight the problems with two different types of Caesarean section, elective and emergency. The differences between the two were discussed further by Denny and Doyle (2008)[28]. In both of cases, Caesarean section would be an invalid instrument if these unobservable characteristics had an effect on both participation in Caesarean section and childhood BMI. For the reasons discussed above, an ideal instrument would take the form of a policy change or institutional differences, based on either geographical difference (for example different health bodies implementing different breastfeeding interventions policies) or changes overtime (such as national changes which are exogenous to the mother's decision to breastfeed.

In addition, Blaser (2014) suggested that the sterile environment in which Caesarean sections are carried out can influence an infant's digestive system, which in turn could

[28] The analysis using the instrumental variable techniques were split and repeated using only elective and only emergency Caesarean section. This made no substantial difference to the results.

influence their BMI during later childhood. If this were case, the instrument itself would be having a direct influence on the outcome, making it invalid.

In the waves of the Millennium Cohort Study which are investigated in this thesis, over 21% of children were born by Caesarean section (see Table II-7). This is a sufficiently high proportion of observations to warrant its use as an instrument. It is also very similar to national statistics published by WHO (Gibbons *et al*., 2010) which suggest that 22% of births during 2008 in the UK were by Caesarean section. Table 3 in the report by Gibbons *et al*. (2010) shows that the UK has a lower rate of Caesarean sections than similar European countries such as Spain, Germany and Italy as well as the US. It also shows that the number of unnecessary Caesarean sections is also lower in the UK than in similar countries. This suggests that mothers may have less or a choice in the UK about Caesarean sections than in similar countries or that mothers are more aware of the risks associated Caesarean sections. If the data used in this analysis were from the US however, usign Caesarean sections as an instrument for breastfeeding might be even more problematic. In the US over 30% of births were by Caesarean section in 2008 (Gibbons *et al*., 2010) and a greater proportion of these were unnecessary, suggesting that mothers were more often choosing to have a Caesarean section without medical reason.

Empirically, Caesarean sections have previously been found by Ajslev *et al*. (2011) to have no significant influence on childhood overweight and by Flemming *et al*. (2013) to have no causal effect on childhood obesity after pre-pregnancy obesity was accounted for. Although there is some evidence in the literature that finds a significant influence of delivery method on childhood adiposity, they generally do not include the large range of observable characteristics that are included in this study and there is no evidence from the data used in this chapter to support this.

Roy Switching Model

A linear regression model with an endogenous treatment effect will be estimated to further investigate the relationship between breastfeeding and childhood adiposity in the case where breastfeeding is endogenous. This model is described in further detail by Maddala (1983) and is a restricted version of the switching model developed by Roy (1951). This model jointly estimates both the outcome and treatment equations. In doing so, it also estimates the joint variance-covariance matrix of the errors in both equations. This means that it is straightforward to test for any remaining correlation between the errors in the outcome and the treatment equations (i.e. to test for endogeneity of the treatment). This

model has been chosen over a control function approach which would not have allowed the endogenous variable to be binary. For more detail on control function approaches, see Heckman & Robb (1986) or Petrin & Train (2010).

Similar to the IV technique, instruments can be included in the treatment equation when using this switching model. However, in this case it is not necessary to include an instrument to identify the model because the model is already identified parametrically. The same variables W will be included in the estimation for the breastfeeding treatments to those which were used in the first stage of the IV regressions and the propensity score estimations which are also treatment equations.

Assume an unobservable latent treatment variable $\boldsymbol{d}^*$ which underlies the binary treatment variable,

$$\boldsymbol{d}^* = \boldsymbol{W}'\boldsymbol{\gamma} + \boldsymbol{v} \tag{II.37}$$

where $\boldsymbol{W}$ is a vector of exogenous variables predicting selection into treatment, $\boldsymbol{\gamma}$ is a vector of corresponding coefficients and $\boldsymbol{v}$ is a vector of random error terms with a standard normal distribution. Observed binary treatment variable $\boldsymbol{d}$, is defined as

$$\boldsymbol{d} = \begin{cases} 1 & if\ \boldsymbol{d}^* > 0 \\ 0 & if\ \boldsymbol{d}^* \leq 0 \end{cases} \tag{II.38}$$

where $\boldsymbol{d} = 1$ if an individual is treated and $\boldsymbol{d} = 0$ if an individual is untreated. Equations (II.37) and (II.38) represent a probit model and are simultaneously estimated alongside a linear regression model (see Equation (II.1)) using maximum likelihood. Error terms $\boldsymbol{u}$ and $\boldsymbol{v}$ are assumed bivariate normal with mean zero and covariance matrix

$$\begin{bmatrix} \sigma^2 & \rho\sigma \\ \rho\sigma & 1 \end{bmatrix} \tag{II.39}$$

where ρ is the correlation between the two error terms.

Subsequently, a likelihood ratio (LR) test is performed in order to test the null hypothesis that there is no correlation between the errors from the outcome and treatment equations, $\boldsymbol{u}$ and $\boldsymbol{v}$, respectively,

$$H_0: \rho = 0. \qquad \text{(II.40)}$$

This tests for the any evidence of endogeneity in the potentially endogenous variable $\boldsymbol{d}$. If there is no evidence to reject this null hypothesis then it is reasonable to assume selection only on observables.

2.3.4 Advantages and Disadvantages of Methodologies

The models used in this chapter will each impose different assumptions in order to identify the effects of breastfeeding variables on childhood adiposity variables. Each of the methods has advantages and disadvantages resulting from their underlying assumptions.

The regression models outlined in Section 2.3.1 which assume that all independent variables are exogenous, will allow the relationship between breastfeeding and childhood adiposity to be investigated under the assumptions that the functional forms are correctly specified, that selection into breastfeeding depends only on observable characteristics and that the groups are balanced. These models are most similar to those used to test the same relationship in the existing literature and will allow a more direct comparison to be made with them. In comparing these methods to those which relax some of these assumptions it will also be possible to determine how restrictive these assumptions are.

If a functional form is incorrectly specified the parameter estimates from the regression models discussed above could be biased and inconsistent. PSM relaxes the assumption of a functional form for the outcome equation and specifically addresses any problems of overlap in the covariates. PSM is a semi-parametric method used to investigate the causal effect of a treatment on an outcome. It imposes no functional form on the relationship when estimating the outcome. Rather than imposing a specific functional form in the outcome equation, it calculates the difference in the mean outcome in the treated and untreated groups. A functional form is imposed when estimating the propensity scores. This tends to be less restrictive and is less likely to influence the results because it is not used in the estimation of a structural coefficient (Caliendo & Kopeinig, 2008; Smith, 1997). PSM estimates a counterfactual in order to obtain the parameters of interest meaning that the assumption of common support is imposed. This assumption means that conclusions are not extrapolated to parts of the outcome distribution which are not included in the data due to poor common support. However, it also means that some observations might not be included in the analysis because they do not have a

counterfactual match. This is not a problem for the regression models from Section 2.3.1, which use all available observations. That said, regression models can extrapolate to parts of the outcome distribution which are not available in the data usually in the tails of the distribution, which is where relationships tend to be different. This lack of common support can severely bias the results of regression models, even when selection us solely on observables.

The regression models outlined in Section 2.3.1 and the PSM each assume that there are no omitted variables or unobservable characteristics which are correlated with both the probability of treatment and the outcome. If this assumption is incorrect then the estimated parameters could be biased. If it is correct to assume that all important confounding factors are accounted for then the standard regression models will produce unbiased estimates of the treatment effect, so long as common support is not a problem. PSM will estimate a causal treatment effect by removing sample selection bias, as discussed by Dehejia & Wahba (2002) and directly addressing the common support. However, if both the outcome and the treatment are correlated with unobservable influences then the IV technique and the restricted Roy model provide an opportunity to account for this endogeneity. They account for potential endogeneity by assuming selection into treatment results from both observable and unobservable characteristics which are correlated with the outcome. Another advantage of these models is that post-estimation analysis can be used to test for endogeneity of the treatment variables. However, they still impose a potentially restrictive functional form in the outcome question and if all covariates are exogenous then IV estimates are inefficient.

By using a variety of econometric techniques which each impose different sets of assumptions, it will be possible to investigate the relationship between breastfeeding and childhood adiposity using models comparable to the existing literature, models which relax the assumption of a functional form and models which control for the potential endogeneity of treatment. By comparing the results of these different models it will be possible to identify which assumption are important and which make very little difference to the results.

2.4 Data

The analysis in this empirical chapter will use data from the Millennium Cohort Study (MCS) described in Section I. In this section, the variables used throughout the analysis in this chapter will be discussed. First, Section 2.4.1 outlines which of the childhood adiposity measures will be used as the dependent variable within each of the models described in the previous section. Due to the nature of the models, it is not possible to have the same dependent variable across each of the models. This is not ideal and will mean that it is not possible to compare the estimated treatment effects across every model. However, a sufficient number of models using each dependent variable will be used to make it possible to determine the most appropriate model. Section 2.4.2 outlines the binary breastfeeding treatment variables used throughout this study, Section 2.4.3 describes the independent variables or confounding factors which are potentially influencing the relationship between infant feeding and childhood adiposity and Section 2.4.4 discusses what is done with missing data and which observations are excluded from the analysis and problems of attrition.

2.4.1 Dependent Variables

Section 1.2.1 discussed the childhood obesity measures which are widely recognised by the literature and the reasons why adult definitions of obesity cannot be used for children. Section 1.4.1 discussed the IOTF childhood adiposity measures available in the MCS which will be used in this chapter. Different dependent variables are required for the different models used throughout this chapter because different functional forms require different types of outcome variables. Table II-1 gives a summary of which dependent variables are used for each model.

Table II-1: Dependent Variables used in Each Model

Model	BMI (continuous)	Overweight (binary)	Obesity (binary)	Weight Status (ordinal)
OLS	✓			
Logit		✓	✓	
Ordered Probit				✓
PSM	✓	✓	✓	
IV	✓			
Roy	✓			

Source: Variables taken from the MCS. Overweight and Obesity are defined using IOTF definitions.

Where possible, more than one dependent variable is estimated using the same technique. For example, propensity score matching is used to estimate both continuous and binary dependent variables. BMI is the only outcome used in the OLS estimations because OLS requires a continuous outcome variable. Logit models require binary outcomes and so overweight and obesity are used as two distinct outcomes. Ordered probit models require ordinal outcome variables and so weight status is used.

An instrumental variable approach is often used in a case where an endogenous explanatory variable exists to estimate an unbiased estimator. However, if both the endogenous variable and the dependent variable are discrete problems can occur. If this is the case then the standard two-stage least squares approach can fail. For example, Greene & Hensher (2010) explained that the 2SLS estimation is not appropriate in models with discrete dependent variables because its estimation is based upon the moments of the data[29]. For this reason, only BMI is used as an outcome for this technique. The Roy model also requires that the outcome variable be continuous because it is estimated using OLS.

2.4.2 Breastfeeding

The MCS contains a range of questions on infant feeding. From these questions it is possible to create a range of breastfeeding variables. This study will investigate the effects of a range of binary breastfeeding variables or 'treatment'. Binary breastfeeding variables are used because the PSM and Roy models can only be used when the treatment variables are binary. For consistency and comparability, these binary breastfeeding variables are used throughout all the models in this chapter. However, binary exogenous variables are less than ideal in the IV technique when using *ivreg2*. The IV models will also be estimated using continuous breastfeeding durations as a robustness check. This section explains how they are created as well as giving summary statistics on breastfeeding in general.

Mothers were asked *"How old was <child's name> when he/she last had breast milk?"* This chapter uses answers to this question, converted into weeks and recorded as 'partial breastfeeding duration'. Partial breastfeeding duration is the length of time a child is breastfed, irrespective of whether this breastfeeding is supplemented with other liquids

[29] 2SLS runs into problems when the potentially endogenous variable is discrete. This is discussed later.

or solids, including formula milk. The number of weeks a child is partially breastfed is rounded down so that a child must have been breastfed for *at least* one week to be considered as being breastfed for one week.

The MCS also recorded information on when cohort members were introduced to a range of other liquids and solids, including formula milk. Mothers were asked the following set of questions.

"I'm now going to ask when <child's name> first had (other) different types of milk. Please include any eaten with cereal."

"First, how old was he/she when he/she first had Formula milk, such as Cow & Gate or SMA?"

"How old was <child's name> when he/she first had Cow's milk?"
"How old was <child's name> when he/she first had any other type of milk, such as soya milk?"
"How old was <child's name> when he/she first had any solid food such as cereal or rusk?"

From the answers to these questions it was possible to infer the duration of exclusive breastfeeding. Exclusive breastfeeding duration is calculated using the earliest introduction of any liquids or solids other than breast milk. Again, all answers were converted into weeks and rounded down to the number of full weeks.

The mean and standard deviations of these breastfeeding variables are presented in Table II-2. The means of these breastfeeding variables differ between the waves of the MCS because they are from different samples. This is a result of attrition and missing data in each wave. In the first wave of the MCS, the average exclusive and partial breastfeeding durations were 4.979 and 10.94 weeks, respectively. The average duration of both exclusive and partial breastfeeding gets larger in each wave. This suggests that weighting for attrition and missing data might be required to produce more consistent estimates. This will be discussed again later.

Table II-2: Means and Standard Deviations of Continuous Breastfeeding Durations

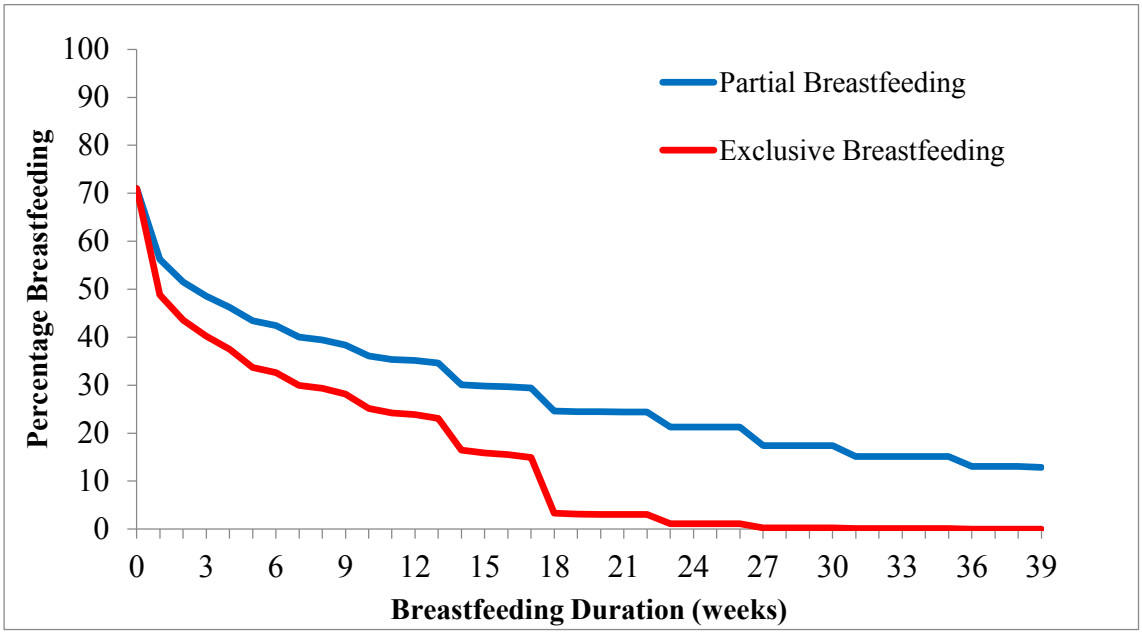
	9 Months	3 Years	5 Years	7 Years
	Exclusive Breastfeeding (weeks)			
Number of observations	17,385	13,970	13,690	12,483
Mean	4.979	5.214	5.215	5.308
(Standard deviation)	(7.006)	(7.075)	(7.089)	(7.097)
	Partial Breastfeeding (weeks)			
Number of observations	17,397	13,979	13,699	12,493
Mean	10.94	11.47	11.51	11.70
(Standard deviation)	(14.86)	(15.03)	(15.06)	(15.11)

Notes: Data from Millennium Cohort Study.

Figure II-1 shows the percentage of children within the first wave of the MCS who were exclusively and partial breastfed by duration, in weeks. The figure shows that less than 50% of children were exclusively breastfed for at least one week and around only 55% were partially breastfed for at least one week. There is a steep drop in the percentage of exclusive breastfeeding after seventeen weeks which could reflect the WHO guidance at the time this cohort were breastfed when WHO recommended four months of exclusive breastfeeding (WHO, 2003). It is worth noting that some of the interviews in the first wave of the MCS were carried out before a cohort member was thirty-nine weeks old and so some of the data may not include the full breastfeeding duration. This is could affect partial breastfeeding duration curve in Figure II-1 but will not affect the results of this study which will only analyse the effects of breastfeeding up to sixteen weeks.

Figure II-1 shows that at four weeks approximately 50% of mothers were still, at least partially, breastfeeding. After four weeks the percentage of mothers who are still breastfeeding remains relatively stable until a small drop after three months possibly due to a spike in the number of women going back to work at three months as a result of maternity legislation. Figure II-1 also shows a sudden drop in exclusive breastfeeding just after sixteen weeks which reflects the WHO recommended breastfeeding durations at the time that this cohort was breastfed. The percentages of women still breastfeeding in the MCS shown in Figure II-1 are similar to those in other reports from a similar time; for example see Dyson *et al*. (2005).

Figure II-1: Percentage of Children Breastfed by Duration



Source: First wave of the Millennium Cohort Study

Mothers of cohort members in the MCS were asked *"Going back to <child's name>. Did you ever try to breastfeed him/her?"* A binary variable was created to indicate whether breastfeeding had ever been initiated. Four additional binary breastfeeding variables are also created. Each of these binary breastfeeding 'treatments' are described in Table II-3. These are the binary treatment variables which will be used throughout this chapter. Binary variables are used to indicate whether a child was breastfed for at least four or sixteen weeks, both partially and exclusively. These durations were chosen because they show the importance of breastfeeding at two stages of infancy. By four weeks, less than half of mothers were still exclusively breastfeeding and the number who are partially breastfeeding had halved from those who first initiated it. However, at four weeks there remains a large sample of treated observations. At sixteen weeks there was a steep decrease in the proportion of mothers who continued to breastfeed, either exclusively or partially, making it an interesting threshold to analyse. This is most likely to be because at the time that this cohort was born, the WHO recommendations suggested that mothers should breastfeeding exclusively for four weeks.

Table II-3: Sample Sizes for Treatment Variables

		Number of non-missing observations		
Treated	**Untreated**	**Age 3**	**Age 5**	**Age 7**
Ever breastfed	Never Breastfed	13,979	13,699	12,493
Breastfed partially for at least 4 weeks	Never Breastfed	11,028	10,825	9,892
Breastfed partially for at least 16 weeks	Never Breastfed	8,665	8,493	7,757
Breastfed exclusively for at least 4 weeks	Never Breastfed	9,771	9,574	8,763
Breastfed exclusively for at least 16 weeks	Never Breastfed	6,569	6,444	5,850

Notes: Data from Millennium Cohort Study. If an observations is considered neither treated nor untreated then they are considered to have a missing value for that variable.

By using the same five treatment variables throughout this chapter, the estimated treatment effects will be comparable, across the different methods and different assumptions. For each variable, observations are considered 'treated' if they reached the corresponding exclusive or partial breastfeeding duration. They are considered 'untreated' if they were never breastfed, and any observations which are neither treated nor untreated are considered as missing and removed from the analysis, in accordance with Scott *et al*. (2012). This restricts the sample size in some cases but ensures that the control groups are consistent across all analyses. It also means that the analysis is more in line with randomised controlled trials in which the control group would generally be completely untreated, rather than on a lower dose of treatment. These variables will allow a range of breastfeeding behaviours to be investigated by identifying both length and exclusivity of breastfeeding. Using binary breastfeeding treatments also allows nonlinear relationships and discontinuities to be investigated.

Table II-4: Summary Percentages for Treatment Variables

	Percentage Meeting Criteria			
Breastfeeding Criteria	**9 Months**	**Age 3**	**Age 5**	**Age 7**
Ever breastfed	66.88%	68.97%	68.95%	69.48%
Breastfed partially for at least 4 weeks	45.92%	47.86%	47.97%	48.66%
Breastfed partially for at least 16 weeks	29.42%	30.96%	30.95%	31.57%
Breastfed exclusively for at least 4 weeks	37.16%	38.90%	38.88%	39.66%
Breastfed exclusively for at least 16 weeks	15.28%	15.98%	16.01%	16.33%

Notes: Data from Millennium Cohort Study. The percentage of children meeting these breastfeeding criteria accounts for all observations, including those removed because they were neither treated nor untreated, in order to make comparisons with national statistics. The number of observations for partial and exclusive breastfeeding is the same as in Table II-2. The number of observations for 'ever breastfed' is the same as that for partial breastfeeding in each wave of data.

Table II-4 shows that 66.88% of cohort members, whose mothers responded to the infant feeding questions were initially breastfed. Unfortunately, due to the differences in breastfeeding variables used throughout the literature, as discussed in Section 2.2.1, it is difficult to directly compare these descriptive statistics with those in other studies. However, the data shown in Table II-4 is similar to national data. For example, the Infant Feeding Survey 2000, published by the DH (2002), found 69% of babies were breastfed initially during 2000 suggesting that this data from the MCS is a realistic and reliable representation of the UK population at the time. The number of breastfed children in the MCS is slightly lower than the estimated national average at the time and this could be due to the over-representation of disadvantaged children in the survey.

Table II-5 shows the mean BMI for children meeting each of the five breastfeeding criteria investigated here, as well as for those who were never breastfed. Irrespective of breastfeeding group, there is a dip in BMI at five years of age in line with the dip in BMI that children experience before their adiposity rebound.

Table II-5: Binary Breastfeeding Treatments and BMI at 9 Months

	Mean BMI (std. dev.)		
	3 Years	**5 Years**	**7 Years**
Never Breastfed	16.85 (1.649)	16.44 (1.721)	16.76 (2.345)
N	3,986	4,166	3,733
Ever breastfed	16.75 (1.564)	16.26 (1.671)	16.55 (2.212)
N	8,936	9,307	8,567
Partially for at least 4 weeks	16.72 (1.554)	16.23 (1.646)	16.49 (2.168)
N	6,226	6,492	6,011
Partially for at least 16 weeks	16.71 (1.535)	16.18 (1.613)	16.43 (2.10)
N	4,036	4,190	3,905
Exclusively for at least 4 weeks	16.73 (1.531)	16.21 (1.609)	16.46 (2.133)
N	5,066	5,267	4,898
Exclusively for at least 16 weeks	16.65 (1.505)	16.09 (1.548)	16.33 (2.036)
N	2,090	2,167	2,022

Notes: Data from Millennium Cohort Study. Mean BMI for 'treated' observations with standard deviations in parentheses.

There is a difference in BMI at each age between children who were breastfed for different durations and for children who were exclusively or partially breastfed. Children breastfed exclusively and for longer, have a lower BMI than children who were not. This difference in BMI becomes wider as children get older. The differences in BMI seen here appear to be small, but for children of such a young age these are important differences.

Any differences in BMI at this age could result in large differences later on in life (Serdula *et al.*, 1993). Also the standard deviation of BMI gets wider as the cohort of children get older; this is in accordance with the percentile charts shown in Figure I-4 and Figure I-5 of Section 1.2.1.

Table II-6 shows the percentage of children who were overweight and obese for each breastfeeding criteria and by age.

Table II-6: Binary Breastfeeding Treatments and Weight Status

	3 Years	5 Years	7 Years
Never breastfed			
Percentage Obese	5.78%	5.90%	6.67%
Percentage Overweight	20.09%	17.86%	16.02%
N	3,986	4,166	3,733
Ever breastfed			
Percentage Obese	4.94%	5.07%	5.23%
Percentage Overweight	17.52%	14.99%	14.06%
N	8,936	9,307	8,567
Partially for at least 4 weeks			
Percentage Obese	4.74%	4.91%	5.14%
Percentage Overweight	17.17%	14.71%	13.34%
N	6,226	6,492	6,011
Partially for at least 16 weeks			
Percentage Obese	4.51%	4.54%	4.46%
Percentage Overweight	17.31%	14.58%	13.42%
N	4,036	4,190	3,905
Exclusively for at least 4 weeks			
Percentage Obese	4.54%	4.27%	4.80%
Percentage Overweight	17.34%	14.42%	12.87%
N	5,066	5,267	4,898
Exclusively for at least 16 weeks			
Percentage Obese	3.93%	3.60%	3.81%
Percentage Overweight	15.79%	13.57%	12.61%
N	2,090	2,167	2,022

Notes: Data from Millennium Cohort Study. Overweight and obesity are defined here using the IOTF classifications. Overweight does not included obese.

The differences in the percentage of children who are classed as overweight or obese is more apparent than the differences in BMI, particularly by the age of seven years when the variance of BMI is larger. Simply initiating breastfeeding is associated with a reduction in both overweight and obesity and breastfeeding for longer and exclusively are associated with the largest reductions in obesity prevalence.

2.4.3 Independent Variables

*Independent Variables (**X**)*

There are a number of independent variables used throughout this chapter. These include variables which are considered within much of the literature to be confounding, such as

maternal education and parental SES. Other independent variables considered in this chapter include demographic, parental and birth related variables. Each of the independent variables described here are included in each of the models used throughout this chapter and are represented by vector $\boldsymbol{X}$.

Table A-2, in Appendix A, shows a list of the independent variables and a description of each of them. The time-invariant variables are generally birth or pregnancy related variables so more accurate responses are expected closer to birth. For this reason, time-invariant variables from as close to the time of birth as possible will be used. In cases where time-invariant variables are missing or implausible in the first wave, the value in the first subsequent wave with a valid value is used, where available. Variables which change over time also from as close to birth as possible because characteristics around this time are most likely to influence maternal breastfeeding choices. Changing characteristics are not able to influence breastfeeding retrospectively.

Based on the existing literature range of standard independent variables are included in the analysis, namely sex, ethnicity (binary variables indicating white, black, Asian and other), mother's marital status and age at the cohort member's birth, as well as the families housing tenure are included as independent variables. A binary variable was also created, indicating whether or not the child lived with both biological parents during the first wave of the MCS.

Following Lamerz *et al*. (2005) and von Kries *et al*. (1999) maternal education is controlled for. Maternal education was measured on a five point scale indicates whether a mother has 'no qualifications', 'GCSEs (grade A*-C)', 'A-levels', 'Degree level', 'higher than degree level' or vocational qualification equivalents. Qualifications were converted into their National Vocational Qualification (NVQ) equivalent levels. High and low maternal education levels were derived for use in this chapter; a mother with at least one degree, the equivalent of NVQ level 4 and above is classed as having high education and a mother who received no qualifications after compulsory education, is classed as having low education.

In accordance with Shrewsbury & Wardle (2008), who report an inverse association between obesity risk and socio-economic status, this chapter includes SES in the set of explanatory variables. Socioeconomic status (SES) was recorded for both parents of MCS cohort members, wherever possible. In the first wave, SES was derived on the five point National Statistics Socioeconomic Classification (NS-SEC) scale. The highest

available SES level of each of the cohort members' parents is used as a proxy for the cohort members' SES at birth. The NS-SEC five point scale includes 'managerial or professional', 'intermediate', 'small employer or self-employed' and 'semi-routine or routine'. A further category to indicate 'long-term unemployed' was also added. Two dummy variables indicating high and low SES have been created using the NS-SEC scale. High SES is defined as 'managerial or professional' and low SES was defined as 'semi-routine, routine or long-term unemployed'.

In accordance with Mizutani *et al*. (2007), data on birth weight was available and all entries were converted into kilograms to create a single continuous variable. Birth weight was given by the mother, in all cases considered in this analysis. Mothers were asked to consult their 'red book' wherever possible; the red book holds medical information from birth to four years old.

Following Dewey (2003) and Mizutani *et al*. (2007), this chapter accounts for maternal weight status as it is potentially an important indicator of childhood obesity. Mothers were asked in the first wave of the MCS, about their height and weight before their pregnancy. For the purpose of this chapter, all weights are converted into kilograms and all heights into meters. From these values pre-pregnancy BMI was calculated and a binary variable was then created to indicate whether the mother was obese before her pregnancy. There could be a problem with recall bias here as mothers might not remember their weight before they were pregnancy. This could lead to both missing and incorrect data being recorded.

Smoking during pregnancy has previously been found to be an important determinant of obesity in childhood, see for example, Toschke *et al*. (2002a) and Mizutani *et al*. (2007). If a cohort member's mother had ever smoked then they were asked *"about how many cigarettes a day were you usually smoking just before you became pregnant with <child's name>?"* and *"did you change the amount you smoked during your pregnancy?"* If they did change their smoking habits when pregnant then they were also asked *"In what month of the pregnancy did you make this change?"* and *"How many cigarettes a day did you usually smoke after you made this change?"* From the answers to these questions, three binary variables were created. Each of the three binary variables indicated whether the mother smoked during the first, second or third pregnancy trimesters, respectively.

This chapter also controls for maternal alcohol consumption habits. In the previous literature alcohol consumption has been found to have a negative or insignificant effect

on childhood weight status, see for example, Strauss (1997) and Mizutani *et al*. (2007), respectively. Mothers were asked "*Thinking back to when you were pregnant with <child's name>, which of these best describes how often you usually drank then? Every day, 5-6 times a week, 3-4 times a week, 1-2 times a week, 1-2 times a month, less than once a month or never?"* If they drank once or twice a week or more often they were also asked *"And in an average week, how many units did you drink then?"* and if they drank less than once a week they were asked *"And on the days when you did drink alcohol, on average how many units did you drink in a day?"* From the answers to these questions the average number of units which a mother consumed whilst pregnant on a day which she did consume alcohol was calculated.

In accordance with Iacovou & Sevilla-Sanz (2010), two additional binary variables are included to indicate whether a mother was in care at the age of sixteen and whether the mother has a longstanding illness. Iacovou & Sevilla-Sanz (2010) used these variables among others to estimate to propensity to breastfeed in a similar study investigating cognitive outcomes. Mothers were asked *"Were you in care at the time you left school?"* A binary variable was created to indicate whether they replied *"yes"* to this question. They were also asked "*Do you have a longstanding illness, disability or infirmity. By longstanding I mean anything that has troubled you over a period of time or that is likely to affect you over a period of time*?" A binary variable was created to indicate whether a cohort member's mother answered *"Yes"* to this question in the first wave of the MCS.

A child's birth order is not included in the independent variables in this study. Although birth order is found to have a significant influence on BMI in teenagers and in later life, there is evidence that BMI in younger children is unaffected (Hawkins *et al*., 2009). Maternal employment is also not included in the independent variables in this chapter because there is such a small proportion of mothers in the MCS who return to work before their child is sixteen weeks old. So it is not expected to influence the breastfeeding variables in this chapter.

*Instrumental Variable (**Z**)*

As discussed in the methodology section, an additional variable is included when predicting treatment (breastfeeding) rather than the outcome (childhood obesity). Any variables which are used as an instrument for breastfeeding should only be included when estimating breastfeeding treatment and never included in equations estimating the childhood adiposity outcomes. This is in line with all econometric textbooks and a clear

example of how instruments included in 2SLS estimation should not be included in regressions such as OLS can be found on page 92 of 'Mostly Harmless Econometrics' by Angrist & Pischke (2008) which explicitly shows the covariates and instruments included when comparing OLS and 2SLS estimations. Rothstein (2013) who compared a range of methods in a similar way to this chapter, also does not include her suggested instruments in the standard regression techniques that she implements. Any instruments can also be included in the treatment equation in the PSM and should be included in the Roy models. This is in accordance with econometric theory and literature; for example, Heckman & Navarro-Lozano (2004) explained that matching makes no distinction between a potential instrument and any other independent variables because they are not entered into the outcome equation, only the treatment equation. They explain the differences in exclusion restrictions when using matching and selection models such as IV. When using an IV technique one makes the assumption that the instrument(s) $\boldsymbol{Z}$ do not causally influence the outcome but that they do have a causal influence on the treatment which is the outcome when predicting the propensity score.

A possible IV to instrument for breastfeeding is whether or not the cohort member was born by caesarean section, similar to those used by Denny & Doyle (2010) in the cognitive development literature. The use of Caesarean sections as an instrument for breastfeeding in this setting will be discussed further along with tests for instrument strength and validity in the results section. An additional binary variable indicating the method of delivery during child birth will be included in the IV regressions and Roy models. Mothers were also asked *"What type of delivery did you have?"* A binary variable was created indicating whether a cohort member was born by caesarean section or not. Cohort members are defined as having been born by caesarean if their mothers answered 'planned caesarean' or 'emergency caesarean'. A single binary variable for Caesarean section is created for use in this chapter. The appropriateness and validity of this instrument will be discussed further in the results section, Section 2.5.6.

Considering $\boldsymbol{X}$ and $\boldsymbol{Z}$ together ($\boldsymbol{W}$)

When predicting breastfeeding treatments rather than the childhood adiposity outcomes, for example in predicting the propensity scores, there is no distinction between $\boldsymbol{X}$ and $\boldsymbol{Z}$ as described earlier and discussed by Heckman & Navarro-Lozano (2004). In these cases, independent variables are referred to as vector $\boldsymbol{W}$ which represents all independent variables in $\boldsymbol{X}$ as well as the instrument(s) $\boldsymbol{Z}$.

Descriptive statistics of the independent variables, including the instruments[30], across each wave are given in Table II-7. The table shows how the mean of each covariate changes with the sample over time, due to attrition. It shows descriptive statistics after the data has been cleaned but for all available observations for each variable. The summary statistics remain relatively steady over time indicating that attrition does not have a large influence on the mean or standard deviations of any independent variables investigated in this chapter. Reviews of the dataset also suggest that attrition will not be a significant problem in most empirical studies (Hansen, 2012; Plewis, 2007).

[30] The breastfeeding variable are not included in $\boldsymbol{X}$ or $\boldsymbol{Z}$ and are discussed separately.

Table II-7: Descriptive Statistics of Independent Variables

	9 Months		3 Years		5 Years		7 Years	
Variable	*N*	Mean (s.d.)	*N*	Mean (s.d.)	*N*	Mean (s.d.)	*N*	Mean (s.d.)
High education*	17,401	0.2904	13,979	0.3128	13,700	0.3130	12,494	0.3202
Low education*	17,401	0.5680	13,979	0.5422	13,700	0.5418	12,494	0.5319
High SES*	17,235	0.1788	13,858	0.1944	13,590	0.1919	12,401	0.2006
Low SES*	17,235	0.5466	13,858	0.5126	13,590	0.5169	12,401	0.5041
Male*	17,401	0.5139	13,979	0.5083	13,700	0.5097	12,494	0.5054
Age (days)	17,401	42.21 (2.201)	13,972	163.8 (10.72)	13,700	272.2 (12.95)	12,494	377.1 (12.85)
Black*	17,370	0.0350	13,954	0.0288	13,674	0.0303	12,470	0.0301
Asian*	17,370	0.1045	13,954	0.0978	13,674	0.0965	12,470	0.0953
Other*	17,370	0.0352	13,854	0.0315	13,674	0.0320	12,470	0.0302
Home Owner*	17,232	0.5838	13,872	0.6211	13,588	0.6127	12,394	0.6268
Private Renter*	17,232	0.0899	13,872	0.0809	13,588	0.0841	12,394	0.0824
Natural Parents*	17,401	0.8248	13,979	0.8458	13,700	0.8429	12,494	0.8490
Birth weight	17,382	3.365 (0.5587)	13,965	3.377 (0.5575)	13,686	3.372 (0.5574)	12,483	3.376 (0.5558)
Premature*	17,231	0.0665	13,860	0.0644	13,580	0.0655	12,383	0.0645
Log Hospital Stay	17,401	1.124 (0.6068)	13,979	1.125 (0.6088)	13,700	1.126 (0.6071)	12,494	1.130 (0.6085)
Planned Pregnancy*	17,372	0.5410	13,962	0.5617	13,678	0.5570	12,478	0.5650
Mother married*	16,699	0.5770	13,379	0.6008	13,109	0.5949	11,955	0.6033
Mother obese*	16,269	0.0686	13,124	0.0791	12,877	0.0721	11,726	0.0701
Mother age at birth	17,382	28.24 (5.949)	13,970	28.63 (5.872)	13,693	28.56 (5.878)	12,485	28.68 (5.853)
Smoking 1st Trimester*	17,386	0.2478	13,967	0.2417	13,692	0.2422	12,485	0.2407
Smoking 2nd Trimester*	17,386	0.0269	13,934	0.0249	13,659	0.0245	12,451	0.0239
Smoking 3rd Trimester*	17,386	0.0756	13,935	0.0701	13,660	0.0709	12,452	0.0680
Alcohol units a day	17,398	0.2845 (1.003)	13,972	0.2906 (0.9718)	13,689	0.2862 (0.9642)	12,484	0.2850 (0.9551)
Mother in Care leaving school*	17,398	0.0095	13,979	0.0089	13,698	0.0085	12,493	0.0074
Illness*	17,395	0.2090	13,974	0.2171	13,694	0.2144	12,490	0.2145
Caesarean Section*	17,376	0.2131	13,958	0.2177	13,683	0.2127	12,478	0.2151

Notes: Data from Millennium Cohort Study. Mean with standard deviation in parentheses for all available data. *Binary variable.

2.4.4 Missing and Excluded Observations

Some observations from the MCS have been excluded from the analysis in this chapter for a number of reasons. The number of observations excluded from the sample in each wave of the data are shown in Table II-8.

Table II-8: Number of Observations Excluded

Variable	9 Months	3 Years	5 Years	7 Years
Original Sample	18,552	15,808	15,460	14,043
Late entry	0	699	573	500
Multiple birth	256	413	409	351
Mother's BMI*	819	80	666	698
Birth weight	846	690	747	673
Hospital stay	459	362	428	369
Gestation length	834	679	734	664
Child's BMI*	-	669	768	683
Number after exclusions	16,219	13,979	13,700	12,494
(% removed)	(12.58)	(11.57)	(11.38)	(11.03)
Missing observations	1,151	2,779	1,956	1,787
# observations in sample	15,068	11,200	11,744	10,707
(% of original obs.)	(81.22)	(70.85)	(75.96)	(76.24)

Notes: Data from Millennium Cohort Study. Number of excluded observations for each reason and further missing observations. Values are for number of children, not families. Observations can be missing in more than one variable. *implausible or missing height, weight or BMI. Childhood BMI is not measured at nine months.

The 692 families (699 children) which entered the study during the second wave because they were not identified in the initial wave were not included in the analysis in this chapter. These observations are removed from the analysis in this study due to a lack of information on breastfeeding and early life variables.

In accordance with Oddy & Sherriff (2003) and Burke *et al*. (2005), children from multiple births have also been removed due to the different breastfeeding experiences mothers have when caring for more than one infant. These studies argued that babies from multiple births were likely to have very different breastfeeding experiences because mothers found it more difficult to breastfeed more than one child due to insufficient milk and time restraints. Childhood adiposity could also be systematically different in children from multiple births.

Any children who remained in hospital immediately after birth for over fourteen days are also excluded. This is because these babies are likely to be very different and have further health problems and breastfeeding behaviours could be influenced in these cases. BMI may be affected if the child was suffering from an illness which might have prevented healthy growth. Any cohort member with a gestational period less than 196 days has also been excluded from analysis because a baby born before twenty eight weeks is considered

'extremely preterm' by WHO (2012) and this could affect the child's growth as well as their ability to breastfeed.

Some variables have been removed due to implausibility. For example, observations have been dropped if mother's height and/or weight were over three standard deviations away from the mean because this is likely to result in an implausible BMI. Implausible birth weights were excluded using the WHO recommendations discussed earlier in Section 1.4.1. This is in line with McCrory & Layte (2012) who screened their data for biologically implausible values for height and weight prior to analyses.

Any 'main responders' from the MCS who were not a cohort member's natural mother have not been included. However, these observations have been removed due to missing data for other important variables and so this does not add to the number of missing observations.

Observations which suffer from item-non-response will also be removed from the analysis. It is assumed that missing data are missing at random. Results were robust to the use of sampling weights. Sampling weights are available in the MCS for attrition and non-response. Item-non-response weights were created specifically for this purpose. Weighting was carried out on the OLS and logit models where the *svy* command in Stata allowed the easy use of weighting. This made very little difference to the estimated parameters and so it is assumed that sample design, attrition and item-non-response do not have a significant influence on the results throughout this chapter. This is in line with Plewis (2007) and Hansen (2012) who found that these weighting adjustments would have little influence on the majority of analyses.

Table II-9, Table II-10 and Table II-11 show the descriptive statistics of the outcome variables in the final samples, breastfeeding treatments and independent variables, respectively.

Table II-9: Descriptive Statistics of Childhood Adiposity Variables

Variable	**3 Years**	**5 Years**	**7 Years**
BMI	16.78 (1.561)	16.31 (1.679)	16.60 (2.224)
Overweight*	0.2334	0.2103	0.2016
Obesity*	0.0498	0.0516	0.0539
N	11,200	11,744	10,707

Notes: Data from Millennium Cohort Study. Mean with standard deviation in parentheses. *Binary variable.

Table II-9 shows a dip in BMI at age five years. This is in line with the dip experienced by young children before their adiposity rebound. The proportion of children who are overweight decreases with age and at the same time the proportion of obese children increases. This is in accordance with data from the MCS before observations with missing or excluded values were removed (see Table I-5).

Table II-10: Means of Breastfeeding Variables

Variable	**9 Months**	**3 Years**	**5 Years**	**7 Years**
Ever breastfed*	0.6682	0.6923	0.6920	0.6982
N	15,068	11,200	11,744	10,707
Four weeks partial*	0.5804	0.6104	0.6104	0.6187
N	11,913	8,845	9,283	8,474
Four weeks exclusive*	0.5304	0.5630	0.5621	0.5716
N	10,645	7,885	8,259	7,542
Sixteen weeks partial*	0.4702	0.5041	0.5030	0.5136
N	9,435	6,949	7,278	6,643
Sixteen weeks exclusive*	0.3182	0.3486	0.3472	0.3571
N	7,332	5,290	5,541	5,026

Notes: Data from Millennium Cohort Study. *Binary variable.

Table II-10 shows that children who were breastfed were more likely to remain in the MCS cohort. Breastfed children are more likely to be from more educated families with higher SES and these families are less likely to drop out of the study or provide missing or implausible answers to questionnaires. However, a rich set of variables which could be causing this bias are included in the analysis in this chapter and so it is not thought to be a problem.

Table II-11 shows that some of the independent variables are related to attrition or affected by missing variables being removed. As explained above, high SES and high education are associated with remaining in the sample. After the initial wave of the MCS at nine months, the sample size used in this study drops but remains relatively stable in the three waves of data which are investigated in this study.

Table II-11: Descriptive Statistics of Independent Variables

Variable	**9 Months**	**3 Years**	**5 Years**	**7 Years**
High education*	0.2989	0.3262	0.3252	0.3332
Low education*	0.5551	0.5233	0.5249	0.5146
High SES*	0.1849	0.2028	0.1992	0.2090
Low SES*	0.5330	0.4968	0.5019	0.4874
Male*	0.5145	0.5024	0.5077	0.5023
Black*	0.0296	0.0236	0.0246	0.0242
Asian*	0.0898	0.0829	0.0827	0.0805
Other*	0.0350	0.0320	0.0320	0.0305
Home Owner*	0.5927	0.6354	0.6238	0.6391
Private Renter*	0.0901	0.0812	0.0840	0.0816
Natural Parents*	0.8239	0.8483	0.8428	0.8495
Birth weight	3.367 (0.5535)	3.379 (0.5493)	3.375 (0.5536)	3.381 (0.5494)
Premature*	0.0664	0.0635	0.0652	0.0639
Log Hospital Stay	1.126 (0.6070)	1.125 (0.6083)	1.126 (0.6071)	1.128 (0.6082)
Planned Pregnancy*	0.5438	0.5679	0.5617	0.5706
Mother married*	0.5771	0.6051	0.5969	0.6051
Mother obese*	0.0688	0.0792	0.0730	0.0707
Mother age at birth	33.67 (0.5535)	28.50 (5.764)	28.41 (5.768)	28.55 (5.753)
Smoking 1st Trimester*	0.2534	0.2470	0.2457	0.2433
Smoking 2nd Trimester*	0.0280	0.0252	0.0250	0.0255
Smoking 3rd Trimester*	0.0755	0.0685	0.0707	0.0669
Alcohol units a day	0.2902 (0.9826)	0.2960 (0.9929)	0.2949 (0.9945)	0.2940 (0.9854)
Mother in Care when leaving school*	0.0096	0.0090	0.0083	0.0072
Illness*	0.2069	0.2143	0.2125	0.2137
Caesarean Section*	0.2098	0.2139	0.2079	0.2095
N	15,068	11,200	11,744	10,707

Notes: Data from Millennium Cohort Study. Mean with standard deviation in parentheses. *Binary variable.

2.5 Results

This section will describe the results found using the methods outlined in Section 2.3 to investigate the causal relationship between breastfeeding and childhood adiposity. Firstly, Section 2.5.1 will discuss the results from the linear models. Next, Section 2.5.2 will outline the results of the logit models estimating both overweight and obesity and Section 2.5.3 will discuss results from the ordered probit models estimating weight status. Section 2.5.4 will compare and review the performance of these regression models before Section 2.5.5 will provide the results from the PSM relaxing the assumption of a functional form with BMI, overweight and obesity as outcomes. Next, Section 2.5.6 and Section 2.5.7 will outline the results of the IV regressions and Roy models, respectively to provide results under the assumption of selection on unobservable characteristics which are correlated with childhood adiposity.

2.5.1 Ordinary Least Squares

Table II-12 displays a summary of results from the OLS regression used to estimate BMI in children at ages three, five and seven years and a full set of these OLS regressions which contain parameter estimates for all covariates $\boldsymbol{X}$ can be found in Table A-3, Table A-4 and Table A-5 of Appendix A, respectively.

Table II-12: Summary of Results using OLS

		OLS				
		(1)	(2)	(3)	(4)	(5)
Age 3		-0.0582	-0.0626	-0.1062**	-0.0618	-0.1721***
		(0.0437)	(0.0380)	(0.0446)	(0.0393)	(0.0538)
	N	11,200	8,845	6,949	7,885	5,290
Age 5		-0.0889**	-0.1195***	-0.1886***	-0.1309***	-0.2645***
		(0.0356)	(0.0398)	(0.0465)	(0.0411)	(0.0556)
	N	11,744	9,283	7,278	8,259	5,541
Age 7		-0.1182**	-0.1846***	-0.2609***	-0.1953***	-0.3408***
		(0.0495)	(0.0551)	(0.0641)	(0.0572)	(0.0774)
	N	10,707	8,474	6,643	7,542	5,026

Notes: Data from Millennium Cohort Study. * p < 0.1, ** p < 0.05, *** p < 0.01. OLS regressions vary by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Overall, the OLS results generally show a reduction in BMI for breastfed children. However, these results take time to become significant. At the age of three years there is no significant effect unless breastfeeding is prolonged. Even then it is only exclusive breastfeeding which produces an effect which is significant at 99% confidence. Prolonged exclusive breastfeeding reduces the expected BMI of a three year old child by

0.17 BMI points. Although this does not sound like a large reduction, at this young age this is a relatively high percentage (4.23%) of the average BMI and is likely to lead to larger differences later in childhood.

All binary breastfeeding treatments investigated here have an effect on childhood BMI at ages five and seven when using OLS. As the cohort get older, these effects generally increase in magnitude. Similarly, longer durations of breastfeeding produce larger differences in BMI, as does exclusive breastfeeding compared to partial breastfeeding. By the age of seven years, a child which was exclusively breastfed for at least sixteen weeks has an average BMI 0.34 points lower than child who was never breastfed.

Breastfeeding initiation has a significant effect on the BMI of five and seven year olds. This suggests that the model might not have a great fit because one would not expect to see a reduction in BMI simply due to breastfeeding being initiated. This is in accordance with the anti-tests described by Jones (2007).

The results found here using OLS conflict with other studies which use linear regressions. For example, Beyerlein *et al.* (2008) found no evidence that breastfeeding initiation influenced BMI in five to seven year old German children and Oddy & Sherriff (2003) found no significant relationship between partial breastfeeding duration and BMI in Australian children up to the age of six years. These differences could be due to the different datasets; these studies do not use data from the UK and both analyse data on cohorts born earlier who were less likely to and be obese or overweight than children in the MCS.

2.5.2 Logit Models

Table II-13 and Table II-14 display summaries of the results using logit models to estimate childhood obesity and overweight, respectively. These tables also show the marginal effects estimated at the mean of each covariate $\boldsymbol{X}$. A full set of the results from the logit models which show parameter estimates for all covariates $\boldsymbol{X}$ predicting obesity and overweight in children at age three, five and seven years is given in Table A-6, Table A-7 and Table A-8 of Appendix A, respectively.

Table II-13: Summary of Results from Logit Models Estimating Obesity

		Logit Models				
		(1)	(2)	(3)	(4)	(5)
		Coefficients (standard error)				
Age 3		-0.0986	-0.110	-0.145	-0.123	-0.370**
		(0.102)	(0.117)	(0.141)	(0.123)	(0.177)
	N	11,200	8,845	6,949	7,885	5,290
Age 5		-0.0916	-0.133	-0.243	-0.161	-0.412**
		(0.0980)	(0.112)	(0.138)	(0.119)	(0.176)
	N	11,744	9,283	7,278	8,259	5,541
Age 7		-0.242*	-0.286*	-0.500***	-0.317**	-0.704***
		(0.0995)	(0.113)	(0.139)	(0.120)	(0.178)
	N	10,707	8,474	6,643	7,542	5,026
		Marginal Effects (standard error)				
Age 3		-0.0042	-0.0046	-0.0061	-0.0051	-0.0152**
		(0.0044)	(0.0049)	(0.0059)	(0.0051)	(0.0072)
	N	11,200	8,845	6,949	7,885	5,290
Age 5		-0.0039	-0.0056	-0.0098*	-0.0065	-0.0163**
		(0.0042)	(0.0047)	(0.0056)	(0.0049)	(0.0069)
	N	11,744	9,283	7,278	8,259	5,541
Age 7		-0.0108**	-0.0128**	-0.0216***	-0.0139***	-0.0301***
		(0.0044)	(0.050)	(0.0059)	(0.0052)	(0.0074)
	N	10,707	8,474	6,643	7,542	5,026

Notes: Data from Millennium Cohort Study. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Marginal effects evaluated at means from logit models. Estimations vary by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

The logit models estimating obesity show a similar pattern to the results from the OLS regressions. Breastfeeding produces a general reduction in the likelihood of obesity which is particularly apparent when breastfeeding is pronged and exclusive. However, these effects take longer to become significant and it is only by the age of seven years that the effects of breastfeeding become significant at a 99% level. Even then, the effects are small. There is only a 3% reduction in the chance of obesity in the average seven year old when breastfeeding is prolonged and exclusive.

As well as becoming more significant, the effects of breastfeeding in this model become larger in magnitude as children get older. This suggests that the full effects on adiposity which result from breastfeeding might take time to become apparent. It is possible that the effects only start to occur after the adiposity rebound. The standard deviations of the BMI distribution and the distribution in BMI percentiles also become wider after the adiposity rebound and this increase in variability could mean that any effects will be easier to identify.

The parameter estimates from these logit models are generally less significant than those from the OLS regressions. This could indicate that children at the upper extreme of the

BMI distribution might not be affected to the same extent from breastfeeding as the average child would.

Table II-14: Summary of Results from Logit Models Estimating Overweight

		Logit Models				
		(1)	(2)	(3)	(4)	(5)
				Coefficients (standard error)		
Age 3		-0.159**	-0.176**	-0.224**	-0.170**	-0.403***
		(0.0532)	(0.0604)	(0.0714)	(0.0628)	(0.0881)
	N	11200	8845	6949	7885	5290
Age 5		-0.160**	-0.210***	-0.288***	-0.214***	-0.405***
		(0.0541)	(0.0615)	(0.0731)	(0.0643)	(0.0900)
	N	11744	9283	7278	8259	5541
Age 7		-0.152**	-0.229***	-0.254***	-0.242***	-0.362***
		(0.0574)	(0.0652)	(0.0771)	(0.0683)	(0.0951)
	N	10707	8474	6643	7542	5026
				Marginal Effects (standard error)		
Age 3		-0.0277***	-0.0305***	-0.0391***	-0.0297***	-0.0702***
		(0.0092)	(0.0104)	(0.0125)	(0.0110)	(0.0153)
	N	11200	8845	6949	7885	5290
Age 5		-0.0255***	-0.0334***	-0.0458***	-0.0340***	-0.0646***
		(0.0086)	(0.0098)	(0.0116)	(0.0102)	(0.0143)
	N	11744	9283	7278	8259	5541
Age 7		-0.0236***	-0.0350***	-0.0391***	-0.0367***	-0.0557***
		(0.0089)	(0.0100)	(0.0118)	(0.0104)	(0.0146)
	N	10707	8474	6643	7542	5026

Notes: Data from Millennium Cohort Study. * p < 0.1, ** p < 0.05, *** p < 0.01. Marginal effects evaluated at means from logit models. Estimations vary by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Similarly to the parameter estimates from the logit model predicting obesity, these results show a larger and more significant reduction in overweight when breastfeeding is prolonged and exclusive. However, unlike the results of the logit models predicting obesity, those predicting overweight show a significant effect much earlier in childhood. This supports the idea that children at the upper extreme of BMI percentiles benefit less from breastfeeding, that breastfeeding does not have a strong enough effect to prevent obesity but can influence overweight. This could however, be due to the lower proportion of children who are considered obese than those considered to be overweight, making any effect more difficult to identify.

Unlike the previous results found in this chapter, the magnitude of the effects of breastfeeding on overweight do not appear to increase in age. When breastfeeding is pronged and exclusive, the magnitude decreases as the children get older, falling from a 7% reduction in the risk of overweight at three years of age to only 5.5% at seven years old. That said, the estimated reductions in overweight are still larger in magnitude than the estimated reductions in obesity by the age of seven years, using the same models.

Initiating breastfeeding has a significant effect on the likelihood of overweight at all ages investigated here and on obesity in seven year olds. Similar to the OLS results, this suggests that these models could be a poor fit for the data, in accordance with the anti-tests outlined by Jones (2007).

In order to allow a better comparison between models, age and sex were included in the independent variables of the logit models discussed here. The logit models were repeated for the same samples, but excluding age and sex from the independent variables. These were performed as robustness checks because the dependent variables in the logit models included age and sex in their calculation. The results found when excluding age and sex were very similar and showed no significant difference in the effects of breastfeeding on adiposity to those displayed in this thesis.

2.5.3 Ordered Probit Models

Table II-15 shows a summary of result from ordered probit models estimating weight category measured using an ordinal dependent variable. Table A-12, Table A-13 and Table A-14 show the full sets of parameter estimates for the ordered probit models estimating weight status is three, five and seven year old children, respectively.

These results are very similar to those from the logit models above. Breastfeeding produces a larger reduction in both obesity and overweight if it is prolonged and exclusive. The effects on obesity increase in magnitude as children get older but the effects of prolonged and exclusive breastfeeding on the likelihood of overweight get smaller as children get older. The anti-tests outlined by Jones (2007) suggest that the models are not predicting either obesity or overweight well. The magnitude of the effects on obesity are similar here to those estimated by the logit models but the effects on overweight are slightly lower when using the ordered probit models. However, there are some differences between the results from logit models and the ordered probit models. When estimating the likelihood of obesity, the logit model found no significant effects until the age of seven, however, in the ordered probit models, the results are significant throughout.

Table II-15: Summary of Results from Ordered Probit Models Estimating Weight Category

		Ordered Probit				
		(1)	(2)	(3)	(4)	(5)
		Coefficients				
Age 3		-0.0843***	-0.0923***	-0.117***	-0.0908**	-0.221***
		(0.0300)	(0.0339)	(0.0399)	(0.0352)	(0.0489)
	N	11200	8845	6949	7885	5290
Age 5		-0.0834***	-0.110***	-0.157***	-0.115***	-0.225***
		(0.0301)	(0.0341)	(0.0404)	(0.0356)	(0.0494)
	N	11744	9283	7278	8259	5541
Age 7		-0.0915***	-0.130***	-0.162***	-0.139***	-0.229***
		(0.0318)	(0.0360)	(0.0424)	(0.0376)	(0.0517)
	N	10707	8474	6643	7542	5026
		Marginal Effects – Obesity				
Age 3		-0.0079***	-0.0086***	-0.0110***	-0.0084**	-0.0203***
		(0.0028)	(0.0032)	(0.0037)	(0.0033)	(0.0046)
	N	11200	8845	6949	7885	5290
Age 5		-0.0079***	-0.0103***	-0.0143***	-0.0106***	-0.0203***
		(0.0029)	(0.0032)	(0.0037)	(0.0033)	(0.0045)
	N	11744	9283	7278	8259	5541
Age 7		-0.0091***	-0.0129***	-0.0157***	-0.0136***	-0.0222***
		(0.0032)	(0.0036)	(0.0041)	(0.0037)	(0.0051)
	N	10707	8474	6643	7542	5026
		Marginal Effects – Overweight				
Age 3		-0.0174***	-0.0191***	-0.0245***	-0.0190***	-0.0463***
		(0.0062)	(0.0070)	(0.0083)	(0.0074)	(0.0103)
	N	11200	8845	6949	7885	5290
Age 5		-0.0155***	-0.0205***	-0.0296***	-0.0218***	-0.0428***
		(0.0056)	(0.0064)	(0.0077)	(0.0067)	(0.0095)
	N	11744	9283	7278	8259	5541
Age 7		-0.0160***	-0.0224***	-0.0285***	-0.0239***	-0.0404***
		(0.0056)	(0.0062)	(0.0075)	(0.0065)	(0.0092)
	N	10707	8474	6643	7542	5026

Notes: Data from Millennium Cohort Study. Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Marginal effects evaluated at means from ordered probit models. Estimations vary by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Again, the analysis using the ordered probit models was repeated for the same sample but excluding age and sex because age and sex are already accounted for in the ordered dependent variable. The results were robust to the exclusion of these two independent variables and this made no significant difference to the effects of breastfeeding on weight status displayed in this thesis.

Advantaged and Disadvantaged Children

In each of the regression models used so far, breastfeeding appears to have small effects on childhood adiposity. Each of the models has suggested that breastfeeding should be both prolonged and exclusive to have the largest effects. However, so far the models have only been used to show the effects of breastfeeding on childhood adiposity in children with average characteristics. It could be that different types of children might

experience different effects. Table II-16 shows the characteristics of two hypothetical children; one of these children comes from an advantaged background and the other from a disadvantaged background.

Table II-16: Hypothetical Characteristics for Marginal Effects

Variable	Advantaged	Disadvantaged
SES	High	Low
Maternal education	High	Low
Both natural parents	Yes	No
Housing status	Home owners (incl. mortgage)	Neither own nor rent privately
Mother married	Yes	No
Mother smoked during pregnancy	Never	Throughout
Mother obese before pregnancy	No	Yes
Pregnancy planned	Yes	No

Notes: Marginal effects for each of these hypothetical children will be estimated for the logit and ordered probit models. Marginal effects will be calculated at the mean of all other independent variables contained in $\boldsymbol{X}$.

Table II-17: Marginal Effects of Breastfeeding on Obesity in Advantaged and Disadvantaged Children

	Advantaged		Disadvantaged	
	Logit	Ordered Probit	Logit	Ordered Probit
		3 Years		
Breastfeeding Initiation (N = 11,200)	-0.0033	-0.0069***	-0.0173	-0.0207***
	(0.0035)	(0.0026)	(0.0177)	(0.0074)
Partial 4 weeks (N = 8,845)	-0.0038	-0.0085***	-0.0184	-0.0211***
	(0.0042)	(0.0029)	(0.0193)	(0.0078)
Partial 16 weeks (N = 6,949)	-0.0053	-0.0103***	-0.0227	-0.0272***
	(0.0054)	(0.0039)	(0.0217)	(0.0094)
Exclusive 4 weeks (N = 7,885)	-0.0040	-0.0074**	-0.0230	-0.0210**
	(0.0042)	(0.0031)	(0.0227)	(0.0082)
Exclusive 16 weeks (N = 5,290)	-0.0134*	-0.0205***	-0.0581**	-0.0497***
	(0.0077)	(0.0056)	(0.0297)	(0.0125)
		5 Years		
Breastfeeding Initiation (N = 11,744)	-0.0026	-0.0063***	-0.0199	-0.0263***
	(0.0029)	(0.0024)	(0.0211)	(0.0094)
Partial 4 weeks (N = 9,283)	-0.040	-0.0085***	-0.0285	-0.0337***
	(0.0035)	(0.0029)	(0.0237)	(0.0104)
Partial 16 weeks (N = 7,278)	-0.0069	-0.0122***	-0.0540*	-0.0494***
	(0.0044)	(0.0036)	(0.0298)	(0.0126)
Exclusive 4 weeks (N = 8,259)	-0.0045	-0.0085***	-0.0358	-0.0360***
	(0.0036)	(0.0029)	(0.0261)	(0.0110)
Exclusive 16 weeks (N = 5,541)	-0.0121*	-0.0177***	-0.0923**	-0.0732***
	(0.0064)	(0.0049)	(0.0384)	(0.0162)
		7 Years		
Breastfeeding Initiation (N = 10,707)	-0.0073**	-0.0066***	-0.0531**	-0.0315***
	(0.0033)	(0.0024)	(0.0217)	(0.0109)
Partial 4 weeks (N = 8,474)	-0.0101**	-0.0101***	-0.0600**	-0.0438***
	(0.0045)	(0.0031)	(0.0237)	(0.0120)
Partial 16 weeks (N = 6,643)	-0.0196***	-0.0123***	-0.1098***	-0.0561***
	(0.0068)	(0.0037)	(0.0299)	(0.0144)
Exclusive 4 weeks (N = 7,542)	-0.0109**	-0.0103***	-0.0697***	-0.0475***
	(0.0047)	(0.0032)	(0.0260)	(0.0127)
Exclusive 16 weeks (N = 5,026)	-0.0306***	-0.0172***	-0.1604***	-0.0800***
	(0.0109)	(0.0049)	(0.0387)	(0.0179)

Notes: Data from Millennium Cohort Study. * p < 0.1, ** p < 0.05, *** p < 0.01. Marginal effects evaluated at means, standard deviations in parentheses.

Similar to the results for the average child, both the hypothetically advantaged and disadvantaged children benefit from a reduction adiposity when breastfeeding is prolonged and exclusive. Both the logit and ordered probit show a larger reduction in the likelihood of obesity in disadvantaged children than in advantaged children as a result of breastfeeding. The differences in the effects on the two hypothetical children are large. The effects are up to five times larger in disadvantaged children showing a reduction of up to a 16% in the likelihood of obesity in disadvantaged children compared to a 3% reduction advantaged children. This difference in effect is visible across all breastfeeding treatments and suggests that breastfeeding could help to reduce inequalities in obesity prevalence between advantaged and disadvantaged children.

2.5.4 Regression Model Performance

Within the literature, regression models have repeatedly been used to estimate the effects of breastfeeding on childhood adiposity. However, very few studies have mentioned the performance or model fit of their regressions or shown that they do not violate any important assumptions. For this reason, goodness-of-fit tests are carried out. The Ramsey Regression Equation Specification Error Test (RESET)[31] which tests whether the general specification of a regression model is correct and the link test[32] for model specification will be carried out on the regression models discussed so far, where possible.

The RESET tests are displayed below the regression results for the OLS models displayed in Table II-18.

Table II-18: RESET tests for Misspecification in OLS Regressions

	Ramsey RESET Tests				
	(1)	(2)	(3)	(4)	(5)
Age 3	2.71**	1.57	1.29	3.80***	3.26**
Age 5	1.21	0.94	1.21	0.65	2.41
Age 7	0.75	0.58	0.17	0.57	0.42

Notes: Data from Millennium Cohort Study. * p < 0.1, ** p < 0.05, *** p < 0.01. Test statistics are shown for each binary treatment variable at each age; the binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

[31] The Ramsey RESET test is used to determine whether $(\boldsymbol{\beta X})^2$, $(\boldsymbol{\beta X})^3$,…, $(\boldsymbol{\beta X})^k$ have any power in explaining y by estimating $\boldsymbol{y} = (\boldsymbol{\beta X})^2 + (\boldsymbol{\beta X})^3 + \cdots + (\boldsymbol{\beta X})^k + \boldsymbol{\epsilon}$. An F-test is carried out to test the null hypothesis that the parameter estimates are equal to zero. If the null hypothesis is rejected then the model suffers from misspecification.

[32] The link test regresses the outcome on its predicted values and the squares of its predicated values, $\boldsymbol{y} = \boldsymbol{\alpha} + \beta_1 \hat{\boldsymbol{y}} + \beta_2 \widehat{\boldsymbol{y}^2} + \boldsymbol{\epsilon}$. The null hypothesis is $\beta_2 = 0$. If β_2 is significant, the null hypothesis is rejected and the model is misspecified. It is also expected that β_1 should be significant, i.e. that the predicted values should estimate y with statistical significance.

The results of these tests show some evidence of misspecification in the linear models estimating BMI in three year olds. This supports findings by Beyerlein *et al*. (2008) that the relationship is non-linear. They give no evidence of misspecification in those estimating BMI in five or seven year old children. However, there is evidence that the relationships are non-linear because the logit models suggest that there are different effects at different parts of the BMI distribution, i.e. at the levels of BMI which define obesity and overweight.

Link tests for misspecification in the logit models are displayed in Table II-19. These test for any significance of the squared fitted values but in this case find no evidence that the linear or logit models are misspecified.

Table II-19: Link Tests from Standard Regression Models

		Link Tests for Logit Models Estimating Overweight				
		(1)	(2)	(3)	(4)	(5)
Age 3	$\hat{y}$	1.001***	0.0976	0.8563***	1.065***	0.0484
	$\hat{y}^2$	0.1665	-0.1721	-0.0640	0.0292	-0.1850*
Age 5	$\hat{y}$	0.9924***	1.038***	0.9357***	0.9912***	0.9289***
	$\hat{y}^2$	-0.0033	0.0162	-0.0272	-0.0037	-0.0297
Age 7	$\hat{y}$	0.9088***	0.9322***	0.6465***	0.8744***	0.7787***
	$\hat{y}^2$	-0.0393	-0.0281	-0.1472*	-0.0514	-0.0916
		Link Tests for Logit Models Estimating Obesity				
Age 3	$\hat{y}$	0.4424	1.0713***	1.061***	0.3225	0.0048
	$\hat{y}^2$	-0.1074	0.0315	0.0269	-0.1271	-0.1874
Age 5	$\hat{y}$	0.5991	0.6424	0.4864	0.6828	0.8060
	$\hat{y}^2$	-0.0790	-0.0695	-0.0987	-0.0602	-0.0371
Age 7	$\hat{y}$	0.5363	0.5954	0.6301	0.6914	0.8839*
	$\hat{y}^2$	-0.0932	-0.0812	-0.0723	-0.0611	-0.0228

Notes: Data from Millennium Cohort Study. * p < 0.1, ** p < 0.05, *** p < 0.01. Binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

2.5.5 Propensity Score Matching

This section will outline the results found using PSM. PSM was carried out for each of the binary breastfeeding treatment variables in Table II-3 for children at ages three, five and seven years. The results are discussed here. First the results of the probit models predicting breastfeeding treatments are discussed and then the non-parametric matching to estimate the treatment effects.

Estimating the Propensity Scores

The propensity scores for each of the binary treatments at each age of the cohort study were estimated using probit models, as described in Section 2.3.2. The probit models used to estimate the propensity scores using data from wave 2 of the MCS are given in

Table II-20. The table also provides the marginal effects of each of the independent variables on the likelihood of each treatment. Results were similar in sign, magnitude and significance, across all three waves. This was expected because covariates in the probit models are recorded early in life for every wave and therefore should not vary as the children get older. However, the samples differ with the age of the cohort due to the data available. This shows evidence that losing observations does not change the relationship between the observable characteristics and breastfeeding variables.

A probit model has been used here to predict the likelihood of participation in each of the breastfeeding treatments. Although this is a parametric model and imposes a functional form, as discussed earlier it is not used to estimate any structural coefficients. This was discussed in more detail by Caliendo & Kopeinig (2008) and Smith (1997). As a result, these parameter estimates have no meaning in estimating the outcome and so their magnitude is not discussed here. However, their significance in predicting the propensity score is still important.

As expected, higher maternal education and similarly, higher SES, lead to an increased likelihood of prolonged and exclusive breastfeeding, *ceteris paribus*. These variables are consistently significant across all breastfeeding durations. Ethnicity also has a very significant impact on breastfeeding across all treatment variables. Black, Asian and 'other' ethnicities all have a higher likelihood of being breastfed exclusively and for longer than white children, *ceteris paribus*. This could be due to different cultural behaviours. Although birth weight has a large influence on childhood adiposity, shown by the regression results, it does not play a large part in predicting breastfeeding behaviour. Prematurity does not significantly influence the likelihood that a child will be breastfed initially, *ceteris paribus*[33]. However, there is some evidence that it reduces the likelihood of being breastfed for at least sixteen weeks, *ceteris paribus*, both exclusively and partially. Birth weight and prematurity are highly correlated and could be capturing the same effect on breastfeeding leading to undetected or less significant effects. An increased hospital stay increases the likelihood of breastfeeding initiation in a child, *ceteris paribus*. This could be because mothers are more likely to initiate breastfeeding if they are being given advice and support by midwives while still in the hospital. Longer hospital stays also increase the chance of being breastfed until at least four and sixteen

[33] The infants who were very premature (less than 28 weeks gestation) were removed from the sample because their start in life would be very different to the majority of children.

weeks, irrespective of exclusivity as well as increasing the likelihood of being partially breastfed for four weeks, *ceteris paribus*.

Table II-20: Probit Models Estimating Propensity Scores of Breastfeeding Treatments at Age 3

	Probit model estimating Breastfeeding				
	(1)	(2)	(3)	(4)	(5)
Age	-0.00180	-0.00231	-0.000925	-0.00191	-0.00143
	(0.00124)	(0.00141)	(0.00165)	(0.00148)	(0.00198)
Sex	0.0348	0.0357	0.0249	0.0265	-0.0474
	(0.0268)	(0.0301)	(0.0350)	(0.0314)	(0.0417)
Black	1.246***	1.483***	1.637***	1.244***	1.428***
	(0.126)	(0.133)	(0.148)	(0.147)	(0.181)
Asian	0.681***	0.787***	0.852***	0.656***	0.821***
	(0.0572)	(0.0624)	(0.0705)	(0.0667)	(0.0818)
Other	0.756***	0.864***	0.956***	0.788***	0.889***
	(0.0888)	(0.0976)	(0.111)	(0.103)	(0.131)
high education	0.339***	0.365***	0.399***	0.357***	0.426***
	(0.0454)	(0.0493)	(0.0553)	(0.0512)	(0.0655)
low education	-0.254***	-0.330***	-0.406***	-0.334***	-0.366***
	(0.0384)	(0.0430)	(0.0495)	(0.0447)	(0.0596)
high SES	0.257***	0.308***	0.340***	0.321***	0.356***
	(0.0458)	(0.0490)	(0.0539)	(0.0508)	(0.0624)
low SES	-0.274***	-0.304***	-0.366***	-0.293***	-0.343***
	(0.0325)	(0.0365)	(0.0422)	(0.0380)	(0.0503)
live with both natural parents	0.276***	0.288***	0.333***	0.263***	0.325***
	(0.0429)	(0.0505)	(0.0625)	(0.0528)	(0.0779)
mother married	0.0319	0.0561	0.0470	0.0633	0.110*
	(0.0346)	(0.0388)	(0.0451)	(0.0405)	(0.0538)
home owners	0.0947*	0.0948*	0.0726	0.0972*	0.0527
	(0.0376)	(0.0430)	(0.0509)	(0.0451)	(0.0614)
private renters	0.180***	0.220***	0.270***	0.223***	0.219*
	(0.0517)	(0.0595)	(0.0707)	(0.0622)	(0.0871)
birth weight	-0.0110	-0.00594	0.0301	-0.0108	0.0179
	(0.0276)	(0.0311)	(0.0367)	(0.0328)	(0.0439)
hospital stay (log)	0.129***	0.0948**	0.0641	0.0864**	0.0442
	(0.0258)	(0.0290)	(0.0340)	(0.0304)	(0.0410)
planned pregnancy	0.0939**	0.108**	0.0974*	0.0995**	0.0583
	(0.0299)	(0.0335)	(0.0388)	(0.0349)	(0.0460)
Premature	-0.0807	-0.0992	-0.245**	-0.162*	-0.266**
	(0.0601)	(0.0684)	(0.0830)	(0.0726)	(0.0995)
mother obese	-0.0273	-0.110	-0.282***	-0.139*	-0.379***
	(0.0488)	(0.0560)	(0.0685)	(0.0592)	(0.0858)
mother age at birth	0.0117***	0.0247***	0.0358***	0.0256***	0.0433***
	(0.00270)	(0.00306)	(0.00359)	(0.00319)	(0.00429)
smoker 1st trimester	-0.0790*	-0.168***	-0.344***	-0.183***	-0.353***
	(0.0335)	(0.0384)	(0.0457)	(0.0400)	(0.0551)
smoker 2nd trimester	-0.335***	-0.415***	-0.454***	-0.371***	-0.577***
	(0.0826)	(0.0981)	(0.119)	(0.100)	(0.158)
smoker 3rd trimester	-0.341***	-0.454***	-0.652***	-0.474***	-0.741***
	(0.0532)	(0.0633)	(0.0807)	(0.0664)	(0.104)
alcohol during pregnancy	-0.000174	-0.00106	0.0148	0.000330	0.00984
	(0.0129)	(0.0151)	(0.0169)	(0.0155)	(0.0228)
mother in care at 16 years	-0.0299	-0.116	-0.146	-0.126	0.123
	(0.132)	(0.162)	(0.210)	(0.171)	(0.233)
maternal longstanding illness	0.0522	0.0138	-0.0245	-0.0118	-0.120**
	(0.0326)	(0.0371)	(0.0435)	(0.0389)	(0.0531)
Caesarean Section delivery	-0.118***	-0.138***	-0.169***	-0.178***	-0.168***
	(0.0382)	(0.0430)	(0.0502)	(0.0455)	(0.0603)
Constant	0.122	-0.372	-1.213***	-0.479	-1.632***
	(0.247)	(0.281)	(0.331)	(0.294)	(0.395)
N	11,200	8,845	6,949	7,885	5,290

Notes: Data from Millennium Cohort Study. Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Probit model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Mother's marital status has no significant influence on breastfeeding initiation, *ceteris* paribus. However, married mothers are more likely to breastfeed exclusively for longer. It is worth noting that this variable is highly correlated with a child living with both natural parents and this possible multicollinearity could lead to inflated standard errors. Maternal obesity has no significant effect on whether breastfeeding is initiated, *ceteris paribus*. However, mothers who were obese before their pregnancy are significantly less likely to carry on any form of breastfeeding until four weeks, *ceteris paribus*. They are also significantly less likely to breastfeed until sixteen weeks. This effect could be because obese women are often unable to continue breastfeeding due to insufficient milk or other health problems. Amir & Donath (2007) suggested that the negative relationship between maternal obesity and breastfeeding initiation and duration could be down to behavioural, cultural or psychological reasons. Older mothers are found to be more likely to initiate breastfeeding, as well as continue breastfeeding longer and more exclusively than younger mothers, *ceteris paribus*. Smoking during any stage of pregnancy reduces breastfeeding initiation as well as exclusive and partial duration, *ceteris paribus*. This is could be because mothers who smoke during pregnancy continue to smoke after the birth of their child and is consistent with the literature; Lee *et al*. (2005) found that mothers who smoked throughout their pregnancy were less likely to initiate breastfeeding. Breastfeeding is significantly less likely to be initiated in children who were born by caesarean section than children born by another delivery method, *ceteris paribus*. They also have a reduced likelihood of being breastfed, exclusively or partially to four or sixteen weeks.

Results from link tests for misspecification in the probit models are displayed in Table II-21. They show no evidence of misspecification in the probit models suggesting that that independent variables are specified correctly[34].

[34] The model fit is not important here. As discussed earlier, the functional form makes little empirical difference when estimating the propensity scores. However, it is important to be confident that all important variables and possible non-linearities are accounted for in the estimation of the propensity score.

Table II-21: Link Tests in Calculating the Propensity Score

		Link Tests of Probit Models Estimating Propensity to Participate in Treatment				
		(1)	(2)	(3)	(4)	(5)
Age 3	$\hat{y}$	1.002***	1.002***	0.9999***	0.9937***	1.014***
	$\hat{y}^2$	-0.0016	-0.0034	-0.0036	0.0204	0.0254
Age 5	$\hat{y}$	1.019***	1.012***	1.000***	0.9987***	1.009***
	$\hat{y}^2$	-0.0192	-0.0228	-0.0214	0.0041	0.0307
Age 7	$\hat{y}$	1.011***	1.009***	1.000***	0.9982***	1.006***
	$\hat{y}^2$	0.0455	-0.0162	-0.0099	0.0049	0.0094

Notes: Data from Millennium Cohort Study. * p < 0.1, ** p < 0.05, *** p < 0.01. Binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Matching on the Propensity Scores

After estimating the propensity scores, treated and untreated observations were matched using the NN matching algorithm with replacement. The full results of the PSM analysis can be found in Table A-15, Table A-16 and Table A-17 showing the estimated differences in BMI, probability of overweight and probability of obesity, respectively, between the treated and untreated groups. These results tables show the number of nearest neighbours used as well as any calliper imposed on each set of matches. The number of nearest neighbours was chosen by considering the numbers in the treated and untreated groups followed by trial and error in an attempt to find the best balance between bias and variance. Each model initially imposed a calliper of 0.01 but again this was changed by trial and error in some cases to achieve the best balance between bias and variance. The sample sizes and percentage of common support in each model are also displayed in these tables for each of the estimated treatment effects. They also give the estimates for the ATT, ATU and ATE along with the standard errors and 95% confidence interval for the ATE estimate. The confidence intervals for the ATEs were estimated using bootstrapping with 500 repetitions. The ATU and ATT are displayed in the appendix because they might be important for policy makers wishing to focus on the differences between breastfed and non-breastfed children. This could be useful when investigating the impact of breastfeeding on inequalities. However, the PSM analysis discussed here focuses on the effect of treatment on an average child which is more comparable to the other models and more informative for policy makers who wish to identify the expected treatment effect on a randomly selected member of the population. For example, Chang4Life aims to improve lifestyle and reduce obesity in children and families from all backgrounds. This is because any national interventions such as this will affect mothers and babies in both the treated and untreated groups.

A summary of the results estimating the ATEs is shown in Table II-22. The results show that breastfeeding generally has an inverse effect on childhood adiposity, which increases with age. However, these effects are not always statistically significant, particularly in younger children. This reduction in BMI and in the probability of overweight and obesity is generally greater in magnitude and more statistically significant as the duration of breastfeeding increases.

Table II-22: ATEs on Adiposity Outcomes using PSM

Treatment	Age 3	Age 5	Age 7
	ATE BMI		
	(bootstrapped standard error[c])		
Ever breastfed	-0.0392 (0.0419)	-0.0782 (0.0456)	-0.1591** (0.0672)
N	9,330	9,996	8,372
> 4 weeks partial breastfeeding	-0.0333 (0.0470)	-0.1086** (0.0535)	-0.1665** (0.0767)
N	7,877	6,858	6,168
> 16 weeks partial breastfeeding	-0.0086 (0.0077)	-0.1772** (0.0686)	-0.2416*** (0.0761)
N	6,949	4,841	6,534
> 4 weeks exclusive breastfeeding	-0.0602 (0.0421)	-0.1401*** (0.0484)	-0.2072*** (0.0743)
N	7,451	7,829	7,167
> 16 weeks exclusive breastfeeding	-0.1592** (0.0785)	-0.2031** (0.0824)	-0.2762** (0.1077)
N	5,183	5,423	4,948
	ATE Overweight		
	(bootstrapped standard error[c])		
Ever breastfed	-0.0171 (0.0118)	-0.0313*** (0.0106)	-0.0329*** (0.0115)
N	9,483	9,996	9,717
> 4 weeks partial breastfeeding	-0.0284** (0.0129)	0.0308** (0.0127)	-0.0315** (0.0125)
N	8,445	8,953	6,867
> 16 weeks partial breastfeeding	-0.0415*** (0.0151)	-0.0505*** (0.0149)	-0.0327** (0.0148)
N	5,543	5,394	6,534
> 4 weeks exclusive breastfeeding	-0.0342** (0.0132)	-0.0446*** (0.0121)	-0.0414*** (0.0131)
N	7,823	7,829	7,509
> 16 weeks exclusive breastfeeding	-0.0607*** (0.0201)	-0.0566*** (0.0193)	-0.0350*** (0.0105)
N	5,183	5,423	4,948
	ATE Obesity		
	(bootstrapped standard error[c])		
Ever breastfed	0.0011 (0.0061)	-0.0026 (0.0059)	-0.0179*** (0.0067)
N	9,413	11,136	9,717
> 4 weeks partial breastfeeding	-0.0031 (0.0060)	-0.0040 (0.0062)	-0.0209*** (0.0068)
N	8,445	8,953	8,033
> 16 weeks partial breastfeeding	-0.0091 (0.0077)	-0.0104 (0.0082)	-0.0243*** (0.0085)
N	5,543	4,841	4,861
> 4 weeks exclusive breastfeeding	-0.0042 (0.0069)	-0.0080 (0.0063)	-0.0176** (0.0083)
N	7,844	8,083	7,510
> 16 weeks exclusive breastfeeding	-0.0167** (0.0080)	-0.0052 (0.0103)	-0.0247*** (0.0096)
N	5,183	5,423	4,948

Notes: Data from Millennium Cohort Study. * p < 0.1, ** p < 0.05, *** p < 0.01. [c]bootstrap standard error (500 repetitions). Standard errors assume propensity score is known.

If a child was ever breastfed, irrespective of exclusivity or duration, BMI generally remains unaffected, *ceteris paribus*. However, there is some evidence that BMI in seven year olds is reduced by 0.16 in breastfed children. There is some evidence to suggest that the probability of obesity and overweight is lower in older children if breastfeeding was initiated. The probability of overweight appears to be reduced by the age of five in children who were ever breastfed, *ceteris paribus*. The probability of obesity is not significantly reduced until the age of seven years. By the age of seven years, breastfeeding reduced the likelihood of obesity and overweight by 1.8% and 3.3%, respectively. The fact that the effects of breastfeeding initiation on childhood adiposity becomes significant as children get older could suggest that the effect of breastfeeding initiation on childhood adiposity is small and could take time to become apparent. However, it could also suggest that simply initiating breastfeeding is not enough to reduce childhood adiposity and breastfeeding should be prolonged in order to produce a significant result.

Neither exclusive nor partial breastfeeding for four weeks significantly reduces BMI in three year olds, these effects on BMI only appear later in childhood. Exclusive breastfeeding for at least four weeks produces a larger and more significant reduction in the BMI of five year olds, than partial breastfeeding, *ceteris paribus*. The same is true in seven year olds. Breastfeeding exclusively for at least sixteen weeks, produces a significant reduction in childhood BMI at all ages. These effects increase in magnitude as children get older. By the age of seven, partial breastfeeding for at least sixteen weeks has a more significant impact than exclusive breastfeeding when compared to children who were never breastfed, *ceteris paribus*. However, the magnitude of the effect is larger for seven year olds than for five year olds.

The PSM and the linear models estimating BMI provide similar results, particularly when estimating the effects of shorter durations of breastfeeding. For prolonged and exclusive breastfeeding PSM provides smaller parameter estimates than the linear models. This could be because the functional forms imposed by the linear models is restrictive, particularly at longer durations.

Using PSM, both exclusive and partial breastfeeding for at least four weeks have no significant influence on the likelihood of obesity in three or five year olds, *ceteris paribus*. However, both provide a significant reduction in the likelihood of overweight in three and five year olds, *ceteris paribus*. The effects of exclusive and partial breastfeeding on

overweight in three and five year olds are not significantly different from each other. By the age of seven years, children breastfed for at least four weeks have a lower probability of overweight or obesity compared to those who were never breastfed, *ceteris paribus*. This effect is more statistically significant for partial breastfeeding than for exclusive breastfeeding but the magnitudes of these effects are not significantly different from each other. Breastfeeding for at least sixteen weeks produces a highly significant reduction in the probability of overweight amongst three and five year olds. However, the significance of these effects diminishes as the children get older. By the age of seven, there is little evidence that exclusive breastfeeding for at least sixteen weeks has an effect on the likelihood of overweight compared to children who were never breastfed, *ceteris paribus*. This could be due to the relatively small proportion of children in the MCS who were breastfed exclusively for sixteen weeks or more.

The effects of exclusive and partial breastfeeding on childhood adiposity are not significantly different from each other[35]. However, there are noticeable differences between the effects and the results are similar in sign, magnitude and significance to those found using the logit models.

The probability of obesity is unaffected by breastfeeding until the age of seven years. However, the probability of overweight is reduced by breastfeeding from the age of three years, an effect which appears to start diminishing by the age of seven years. This suggests that there could be additional factors affecting childhood adiposity as children get older. It also suggests that different parts of the BMI distribution are affected by breastfeeding in different ways.

PSM was also performed using the NN algorithm using binary treatment variables which included all observations which were not 'treated' within the control group[36]. The PSM results were robust to either sample and as expected, effects were slightly smaller when using this inclusive sample.

In order to determine how well the PSM can analyse the data used in this chapter a number of model checks were carried out. First, t-tests were performed to test for bias between the treated and untreated samples after matching. The t-tests were carried out for each covariate and for each set of matches. The tests showed no significant difference between

[35] Standard errors are inflated because they are calculated assuming that the propensity scores are known rather than estimated.
[36] Here, if an observation was not considered 'treated' it was considered 'untreated rather than only including children who were never breastfed in the control group.

the means of any of the independent variables in the two groups and provided no evidence of bias. A joint significance test of all independent variables, the LR test was also carried out for each set of PSM analysis. These tests were each insignificant at a 5% significance level, again suggesting that bias is not a cause for concern.

The common support between the treated and untreated groups was never less than 65% and in most cases was over 90%. This suggests that the number of observations dropped, due to unmatchable observations was relatively low, especially in comparison to other studies using similar techniques. For example, Iacovou & Sevilla-Sanz (2010) had a common support of 65% in their final model.

All results displayed here using PSM are robust to matching algorithm. The same matches were also performed using radius matching and Epanechnikov kernel matching and results were found to be very similar to those presented here.

Similar to the previous methods analysing binary outcomes, the results from PSM estimating overweight and obesity are robust to the exclusion of age and sex which are included in the calculations of these dependent variables, that is, the exclusion of these variables in estimating the propensity score does not change the effect of breastfeeding on the adiposity outcomes.

2.5.6 Instrumental Variable Estimation

This section discusses the results using the IV technique, both the first and second stage results. First however, it discusses the use of caesarean sections as an instrument as well as discussing other variables which have been used as instruments for breastfeeding in related literatures.

Potential Instruments

Table II-23 shows the parameter estimates for delivery by caesarean section if it were to be included in the outcome equation of a standard OLS regression. This is displayed here only to illustrate how it influences the outcome directly after other independent variables are accounted for. It is assumed, that it is a valid instrument[37] and is not included in the outcome equations, only the treatment equations for each of the main analyses in this chapter. The results from these illustrative OLS regressions show that conditional on

[37] The validity of an instrument cannot be tested. It involves estimating the correlation between the instrument and an unobservable. That is why, the only thing that one can do is to present a good theoretical and empirical case that an instrument is valid.

independent variables included in $\boldsymbol{X}$, delivery by Caesarean section has little influence on the outcome, BMI.

Table II-23: Standard OLS Regressions including the Instrument

	Estimated coefficients of the effect of the instrument on BMI				
	(1)	(2)	(3)	(4)	(5)
Age 3	0.0311	0.0189	0.0431	0.0070	0.0007
	(0.0337)	(0.0460)	(0.0526)	(0.0494)	(0.0617)
N	11200	8845	6949	7885	5290
Age 5	0.0717*	0.0955*	0.1105*	0.0880*	0.1395**
	(0.0428)	(0.0488)	(0.0566)	(0.0518)	(0.0640)
N	11744	9283	7278	8259	5541
Age 7	0.0847	0.1059	0.1391*	0.1206*	0.1601*
	(0.0592)	(0.0667)	(0.0761)	(0.0720)	(0.0899)
N	10707	8474	6643	7542	5026

Notes: Data from Millennium Cohort Study. Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Showing estimated parameters for delivery by caesarean section on BMI using standard OLS regression; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks. These parameter estimates are conditional on all other independent variables included in $\boldsymbol{X}$.

Although there is some indication of significance at a 90% significant level, the estimated effect of caesarean sections on childhood BMI is generally weak. It is therefore assumed that delivery by caesarean section is a valid instrument. Including delivery by Caesarean section in the OLS regressions also makes no significant difference to the parameter estimates for other independent variables.

2SLS First Stage Results

Table II-24 shows a summary of the first stage results from the IV regressions. It shows the estimated effects of caesarean sections on each of the breastfeeding treatments[38]. A full set of results for the first stage of the 2SLS estimations which show the parameter estimates for all independent variables contained in $\boldsymbol{W}$ when estimating breastfeeding treatments in three, five and seven year olds are displayed in Table A-18, Table A-19 and Table A-20, respectively, in Appendix A.

[38] Although the first stage estimates a binary variable using OLS here, very similar results are found when using a logit or probit model for the first stage estimation and when estimating continuous breastfeeding durations using an instrumental variable, for both exclusive and partial breastfeeding.

Table II-24: Summary of First Stage IV Results

	2SLS – First Stage Results estimating Breastfeeding Treatments				
	(1)	(2)	(3)	(4)	(5)
Age 3	-0.0362***	-0.0441***	-0.0511***	-0.0565***	-0.0462***
	(0.0113)	(0.0852)	(0.0142)	(0.0141)	(0.0158)
N	11200	8845	6949	7885	5290
Age 5	-0.0441***	-0.0546***	-0.0655***	-0.0667***	-0.0589***
	(0.011)	(0.0126)	(0.0139)	(0.0138)	(0.0155)
N	11744	9283	7278	8259	5541
Age 7	-0.0331***	-0.0422***	-0.0504***	-0.0505***	-0.0290***
	(0.0116)	(0.1437)	(0.0146)	(0.0145)	(0.1750)
N	10707	8474	6643	7542	5026

Notes: Data from Millennium Cohort Study. Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Showing estimated parameters for delivery by caesarean section in stage one of IV model estimating breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

The first stage results displayed in Table II-24 show that Caesarean sections have a significant and negative influence on each of the breastfeeding treatments. This is as expected and in line with the results found in the first stage of analysis by Denny & Doyle (2008). Denny and Doyle (2008) used a continuous breastfeeding variable for three and five year olds and as a result the first stage result presented here are not directly comparable with those from their study. However, both sets of results produce statistically significant estimates for the effects of Caesarean sections on breastfeeding and similar results are found using the data from this chapter when continuous breastfeeding duration outcomes were used. For seven year olds, Denny and Doyle (2008) used a binary breastfeeding variable indicating whether a child was ever breastfed. The first stage results estimating this variable are similar to those found in this chapter. Results from this chapter showed a slightly larger effect of Caesarean sections on breastfeeding initiation. Denny and Doyle found that emergency Caesarean sections had more effect on breastfeeding initiation than elective Caesarean sections.

The effect of caesarean sections on breastfeeding treatments are relatively stable across the different ages of children, which is as expected and suggests that attrition is not affecting these results.

Tests for Weak Instruments

The Cragg-Donald Wald tests for the first stage of each of these models are shown in Table II-25. The show the F-statistics for the first stage of the 2SLS regression.

Table II-25: Cragg-Donald Wald Tests for Weak Instruments

	Cragg-Donald Wald F tests for Weak Instruments F-statistic (p-value)				
	(1)	(2)	(3)	(4)	(5)
Age 3	10.23	11.72	13.03	15.99	8.58
	(0.0014)	(0.0006)	(0.0003)	(0.0001)	(0.0034)
Age 5	15.84	18.78	22.23	23.23	14.54
	(0.0001)	(0.0000)	(0.0000)	(0.0000)	(0.0001)
Age 7	8.23	10.26	11.96	12.12	5.97
	(0.0041)	(0.0014)	(0.0005)	(0.0005)	(0.0146)

Notes: Data from Millennium Cohort Study. F statistics with p-values in parentheses. F-statistics are taken from stage one of IV model estimating delivery by caesarean section, varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

The Cragg-Donald Wald test suggests that F-statistics should be above 10 to reject the null hypothesis of a weak instrument. Table II-25 shows that the majority of F-statistics are higher than 10 and have a p-value less than 0.01. This suggests that there is little evidence of the instrument being weak. However, there is some evidence that the instrument is weak at the age of seven when breastfeeding is initiated and at the ages of three and seven when breastfeeding is prolonged and exclusive.

Stock & Yogo (2002) provide critical values for maximal bias. If the F-statistics are over these critical values then there is no evidence of the instrument being weak. The critical values given by Stock & Yogo (2002) for a 5% significance level are 16.38 and 8.96 for 10% and 15% maximal bias, respectively. At a 15% maximal bias, these tests show evidence that the instrument is weak under the same circumstances as it is in the Cragg-Donald test. However, all but three F-statistics in Table II-25 are below the 10% maximal bias critical value, showing some evidence for a weak instrument. Interestingly, there is less evidence of a weak instrument in the models for five year olds.

In this case, there are no over-identifying restrictions because it is assumed that only one variables (breastfeeding) is endogenous. Nevertheless, the Anderson-Rubin test for over-identification is carried out. This tests the null hypothesis that the endogenous coefficients (in this case the relevant breastfeeding treatment) are jointly equal to zero in the outcome equation and that the over-identifying restrictions are valid. Results from these tests are displayed in Table II-26. The test substitutes the estimated treatment equation into the estimated outcome equation, so that

$$\hat{\boldsymbol{y}} - \hat{\boldsymbol{x}}\beta_0 = \hat{\boldsymbol{Z}}'\boldsymbol{\varphi} + \boldsymbol{u} \qquad \text{(II.41)}$$

where $\varphi = (\beta - \beta_0)$ and $\boldsymbol{u} = \boldsymbol{\epsilon} + \boldsymbol{\varepsilon}(\beta - \beta_0)$. The null hypothesis that $\beta = \beta_0$ can then be rejected or otherwise by testing whether $\varphi = 0$. Very similar results were found using the Stock-Wright test for the same null hypothesis.

Table II-26: Anderson-Rubin Tests for Weak Instruments

	Anderson-Rubin tests for Weak Instruments				
	(1)	(2)	(3)	(4)	(5)
Age 3	0.67	0.22	0.85	0.05	0.02
	(0.4114)	(0.6372)	(0.3565)	(0.8311)	(0.8890)
Age 5	3.12*	4.44**	4.95**	3.48*	4.776**
	(0.0771)	(0.0351)	(0.0261)	(0.0621)	(0.0289)
Age 7	2.24	2.90*	3.98**	3.28*	3.72*
	(0.1346)	(0.0889)	(0.0460)	(0.0703)	(0.0538)

Notes: Data from Millennium Cohort Study. Anderson-Rubin Wald Test statistics with p-values from F distribution in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. F statistics are taken from stage one of IV model estimating delivery by caesarean section, varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

There is no evidence to reject the null hypothesis in the models estimating the BMI of three year old children. However, there is evidence that the instruments are weak or the exclusion restrictions invalid when estimating BMI in five year olds, particularly when breastfeeding is prolonged. There is some evidence to reject the null in the models for seven year old children if breastfeeding is prolonged.

2SLS Second Stage Results

Table II-27 shows the second stage results for the IV regressions estimating BMI.

Table II-27: Summary of Second Stage IV Results

	2SLS – Second Stage Results estimating BMI				
	(1)	(2)	(3)	(4)	(5)
Age 3	-0.916	-0.492	-0.949	-0.186	-0.187
	(1.145)	(1.049)	(1.053)	(0.872)	(1.331)
N	11200	8845	6949	7885	5290
Age 5	-1.712	-1.863	-1.871*	-1.447	-2.626*
	(1.050)	(0.970)	(0.912)	(0.821)	(1.246)
N	11744	9283	7278	8259	5541
Age 7	-2.672	-2.692	-3.014	-2.582	-4.328
	(1.992)	(1.762)	(1.702)	(1.579)	(2.765)
N	10707	8474	6643	7542	5026

Notes: Data from Millennium Cohort Study. Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Stage two of IV model estimating BMI varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks. Instrument is a binary variable indicating delivery by caesarean section.

It shows a summary of the second stage results from the models estimating BMI using an IV. Breastfeeding initiation, instrumented by caesarean section, has no significant effect on BMI at any age investigated here, although the magnitudes of the coefficients do

increase with age. Despite the lack of significant effect found in this model, the results are intuitive. Simply initiating breastfeeding is not expected to influence childhood adiposity once confounding factors are accounted for but prolonged breastfeeding does appear to reduce childhood BMI. However, unlike the previous models, there is no evidence of a statistically significant reduction in childhood adiposity as a result of any duration of exclusive or partial breastfeeding. This is most likely due to the large standard errors in these models. The inflated standard errors are probably due to the weak instrument. The evidence suggesting a weak instrument was not overwhelming but these tests are only indicative and caution should be taken when interpreting these parameter estimates, particularly due to the large standard errors.

The Local Average Treatment Effect (LATE)

The LATE estimated using the instrumental variable technique in this chapter identifies the average effect of treatment in children whose mother's breastfeeding behaviour is induced to change as a result of having a caesarean section. This means that the treatment effects are not directly comparable with those resulting from the previous methods. This also causes problems for policy makers which are unlikely to have a particular interest in this specific subpopulation but are more often interested in the ATE (Faria *et al*., 2015; Heckman, 1997).

Tests on the Endogeneity of Instruments

The endogeneity of the breastfeeding treatments in the outcome equations are tested for using a comparison of the Sargan-Hansen statistics in the OLS regressions and the IV regressions. This test which is included as part for the -*ivreg2*- command in Stata is an alternative to the Durbin-Wu-Hausman test for endogeneity and tests the null hypothesis that the regressor being tested for endogeneity can be treated as exogenous variables.

Table II-28: Test for Endogenous Treatments

	Tests for Endogeneity of Treatments				
	(1)	(2)	(3)	(4)	(5)
Age 3	0.594	0.170	0.675	0.020	0.000
	(0.4410)	(0.6799)	(0.4114)	(0.8864)	(0.9913)
Age 5	2.820*	3.912**	4.036**	2.899*	4.776**
	(0.0931)	(0.0480)	(0.0445)	(0.0887)	(0.0289)
Age 7	2.053	2.525	3.350*	2.817*	3.189*
	(0.1519)	(0.1120)	(0.0672)	(0.0933)	(0.0741)

Notes: Data from Millennium Cohort Study. Test statistics with p-values in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01 Test statistics are shown for each binary treatment variable at each age; the binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

At the age of three years, there is no evidence of breastfeeding being endogenous in the outcome equation predicting BMI. There is some evidence that exclusive breastfeeding is endogenous in the outcome equations in five year old children, evidence which increases with exclusivity and duration. By the age of seven years there is again little evidence to reject the null hypothesis of endogenous breastfeeding. These results suggest that in the majority of cases, there is little or no evidence that breastfeeding is endogenous and that an OLS regression would be preferable over the IV technique. However, in cases where there is some evidence that breastfeeding is endogenous, the evidence is weak and it is worth looking at the results from both estimation methods and considering the strength of the instrument. There is most evidence that breastfeeding is endogenous when predicting BMI for five year olds. This could be because both breastfeeding and BMI are associated with dip in BMI before the adiposity rebound which occurs around this age.

Despite the controversy of the instrument used in this section, the results do follow a similar pattern to the previous models. Although they follow the same pattern, the estimated effects are larger in magnitude than the previous models, despite the lack of significance. This lack of significance caused by inflated standard errors suggests that Caesarean section is a weak instrument.

There is also insufficient evidence that breastfeeding is endogenous which suggests that an instrument might not be needed and that the parameter estimates from the model which assume selection into treatment only on observables might be more appropriate. If breastfeeding is exogenous then it would be inappropriate to use the IV estimates for policy purposes[39]. Although using instruments allows causal effects to be identified, the IV estimates are inefficient. For this reason, it is better to use alternative models for policy purposes unless there is sufficient evidence of endogeneity.

Further tests on the endogeneity of breastfeeding in childhood adiposity equations are required. For this reason, post-estimation endogeneity tests will also be carried out after the Roy models, which are identified parametrically and do not depend solely on a reliable instrument for identification.

2.5.7 Roy Model

This restricted version of the Roy model simultaneously estimates an outcome (childhood BMI) and a treatment (binary breastfeeding treatments) using maximum likelihood. It

[39] This is in addition to the fact that policy makers are less interested in the LATE, as discussed previously.

allows the error terms in each of the equations to be correlated and this correlation is also estimated. The Roy model makes it possible to test whether or not breastfeeding is endogenous in predicting childhood BMI after conditioning on the observable variables. Unlike the 2SLS approach the Roy model is parametrically identified and does not require the inclusion of instrumental variables. That said, the inclusion of any instrumental variables will strengthen their identification.

Table A-24, Table A-25 and Table A-26 show the full set of results from the Roy models for children aged three, five and seven, respectively. The treatment effects ($\boldsymbol{\delta}$) in effects Equation (II.34) from these models are summarised in Table II-29.

Table II-29: Roy Model Estimated Treatment Effects

	BMI (outcome equations)				
	(1)	(2)	(3)	(4)	(5)
Age 3	0.101	0.0477	-0.419	-0.000959	-0.587*
	(0.191)	(0.259)	(0.260)	(0.291)	(0.235)
N	11,200	8,845	6,949	7,885	5,290
Age 5	-0.0443	-0.291	-0.607*	-0.242	-0.693**
	(0.200)	(0.296)	(0.252)	(0.261)	(0.229)
N	11,744	9,283	7,278	8,259	5,541
Age 7	0.0104	-0.197	-0.629	-0.196	-0.960**
	(0.230)	(0.307)	(0.341)	(0.318)	(0.303)
N	10,707	8,474	6,643	7,542	5,026

Notes: Data from Millennium Cohort Study. Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Roy model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks. Outcome equation estimating BMI.

The results from the restricted Roy switching models are consistent with the results from the linear regression with IV in that the estimated treatment effects for initiating breastfeeding are insignificant in children of all ages. Partial breastfeeding and breastfeeding for only four weeks also have no significant influence on BMI when using a Roy model. However, unlike the IV results, these results suggest that exclusive breastfeeding has a statistically significant effect on BMI when it is continued for at least sixteen weeks. These results, like those produced by the standard regression results, suggest that any effects of breastfeeding might only be apparent when children get older and that breastfeeding must be prolonged in order to make a significant difference to childhood adiposity. However, the results from the Roy model are larger in magnitude but less statistically significant than those from the standard linear models using OLS to estimate BMI.

The results from the probit models predicting the binary breastfeeding treatments which are simultaneously estimated with the linear regressions are displayed in the bottom half

of Table A-24, Table A-25 and Table A-26 in Appendix A for children at ages three, five and seven years, respectively. Results from these probit models are very similar to those used to estimate the propensity scores in the PSM analysis. These effects are summarised in Table II-30. Delivery by Caesarean section produces a small but consistent and statistically significant reduction in the likelihood of each breastfeeding treatment and across all ages.

Table II-30: Roy Model Predicting Treatment

	Breastfeeding (treatment equations)				
	(1)	(2)	(3)	(4)	(5)
Age 3	-0.117**	-0.137**	-0.169***	-0.178***	-0.165**
	(0.0382)	(0.0430)	(0.0499)	(0.0455)	(0.0599)
N	11,200	8,845	6,949	7,885	5,290
Age 5	-0.146***	-0.180***	-0.231***	-0.217***	-0.226***
	(0.0378)	(0.0427)	(0.0493)	(0.0451)	(0.0590)
N	11,744	9,283	7,278	8,259	5,541
Age 7	-0.107**	-0.134**	-0.174***	-0.162***	-0.152*
	(0.0395)	(0.0445)	(0.0515)	(0.0470)	(0.0613)
N	10,707	8,474	6,643	7,542	5,026

Notes: Data from Millennium Cohort Study. Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Roy model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks. Outcome equation estimating BMI.

The correlation between the error terms of the linear and probit regression models which are estimated simultaneously can determine whether the breastfeeding treatments are still endogenous after conditioning on the covariates by testing the null hypothesis of exogeneity, see Equation (II.40). Table II-31 shows the results of likelihood ratio tests to test the null hypothesis that there is no correlation between the error terms in the two equations estimated simultaneously in the Roy model[40]. The table shows the likelihood ratio χ^2 value for each test and gives the p-value for the test in parentheses.

Table II-31: LR Test for Endogeneity

	Likelihood Ratio Test for Endogeneity in the Roy Model χ^2 (LR p-value)				
	(1)	(2)	(3)	(4)	(5)
Age 3	0.64	0.18	1.21	0.05	2.21
	(0.4233)	(0.6711)	(0.2704)	(0.8313)	(0.1376)
Age 5	0.05	0.38	2.27	0.19	2.41
	(0.8244)	(0.5384)	(0.1320)	(0.6646)	(0.1203)
Age 7	0.31	0.00	0.99	0.00	2.83
	(0.5783)	(0.9659)	(0.3190)	(0.9986)	(0.0923)

Notes: Data from Millennium Cohort Study. * p < 0.1, ** p < 0.05, *** p < 0.01. Binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

[40] H_0: no correlation between outcome and treatment error terms, $\rho = 0$, *i.e.* breastfeeding is exogenous.

The LR tests show no evidence that breastfeeding is endogenous in the outcome equation once the other independent variables are accounted for. This suggests that all important confounding factors are accounted for within this chapter and that the models which assume selection on observables are preferable.

Taking all the evidence into consideration, there is little evidence of any remaining endogeneity after conditioning on rich set of variables available in the data. Both the IV technique and the Roy models have problems; the IV technique relies on a strong and valid instrument and the Roy models on parametric assumptions for identification. However, overall the evidence seems to suggest that selection on observables is supported by the data and additional variables are unlikely to affect the parameter of interest (the ATE) using the preferred method (PSM) because it does not use a parametric regression for the outcome. For this reason, breastfeeding is considered to be exogenous once all the confounding factors in this chapter are accounted for.

2.5.8 Summary of Results

This chapter investigated the effect of breastfeeding on childhood adiposity under different sets of assumptions imposed by the different models. First, the regression analysis investigated the relationship using the models and assumptions which have been commonly used throughout the literature. These regression models assumed selection into treatment only on observable characteristics as well as imposing a functional form on the relationship. Next, PSM was used in an attempt to identify the causal relationship of breastfeeding on childhood adiposity without using the restrictive functional form imposed by the regression models and by considering explicitly the potential problems of common support. Finally, the chapter went on to analyse the relationship using models which assumed selection on unobservable characteristics which are correlated with the outcome to account for potentially endogenous treatment effects using IV techniques[41]. However, these models also imposed a functional form as do the standard regression models. They also require the use of a strong and valid instrument which are in practice often difficult to find. Similar to the IV regressions, Roy models were used. These models jointly estimated the outcome, using a linear regression, and the potentially endogenous treatment, using a probit model. Although, like the IV technique, these restricted versions of the Roy model impose a functional form and allow selection on

[41] All analysis was not significantly changed when using the restricted sample of common support, imposed by the PSM.

unobservables which are correlated with the outcome, they differ in that they are parametrically identified and so do not rely solely on the strength of instrument for identification but would cause problems if the functional form was misspecified.

The results from the parametric models in this chapter did not significantly differ when they were restricted to the sample included in the PSM (common support). There was also no evidence that the relationship between breastfeeding and childhood adiposity differed between girls and boys[42].

The PSM analysis, the IV analysis and the Roy model each use different assumptions to deal with potential problems with the standard regression models. Therefore it is not possible to choose the most appropriate method purely on their theoretical merits and shortcomings. Each model has advantages and disadvantages and different models would be appropriate in different settings and with different datasets and sets of conditioning variables. There is not one model which is best in every situation. For this reason, the choice of the best estimates found in this chapter is an empirical one.

The standard regression techniques rely on a correctly specified functional form. If this is misspecified then estimated coefficients could be biased. In this chapter, there was no evidence of misspecification in the regression models using post-estimation tests. However, the effects of breastfeeding appear very different in the estimating of obesity and overweight, two cut-offs at different points of the BMI distribution. This provides some evidence of a non-linear relationship between breastfeeding and BMI. A non-linear relationship is also supported by Beyerlein *et al.* (2008). These regressions models also assume that selection into treatment depends only on observable characteristics and that there is sufficient common support between treated and untreated observations.

In order to account for the potentially endogenous effect of breastfeeding in predicting childhood adiposity, two additional models were used. The instrumental variable technique relies on the strength and validity of a good instrument in order to be identified. Furthermore, the IV estimates are inefficient and estimate the LATE rather than an ATE meaning that the treatment effect is identified for an unknown subsample of the population. This could be problematic when using results for policy purposes (Basu *et al*., 2007) and the average effect for a known subsample or an entire population would be of more practical use for policy makers and guidance providers such as NICE. This

[42] The sample was split into male and female and the OLS and logit models were used to analyse these sub samples. There was no significant difference between the two samples.

problem with the IV analysis in this setting is attributable to the method, rather than the chosen instrument. However, the instrument is also found to be weak and therefore adds to the inappropriateness of the IV technique when the aim is to inform policy makers about the potential effects of interventions. The Roy model also accounts for the potential endogeneity of breastfeeding in predicting childhood BMI. However, it relies on parametric assumptions for identification and similar to the standard regression models, if it is misspecified then it produces biased results. Both the IV technique and the Roy models make it possible to test for the endogeneity of treatment. When considering all the evidence from both models there is little indication of endogeneity after all the covariates are accounted for[43]. This suggests that models which rely on selection into treatment only on observable characteristics are preferred to the IV technique and the Roy model. This also means that the inflated standard errors in the IV analysis which could potentially be due to a weak instrument do not need to be relied upon.

PSM has the advantage that it does not rely on a functional form and so has no parametric specification for the estimated treatment effect. However, it does impose the assumption that there are no remaining unobservables which influence both the treatment and the outcome after confounders have been accounted for. As there was little evidence found for the endogeneity of breastfeeding once the covariates were accounted for, this is thought to be a reasonable assumptions. PSM also directly addresses the issue of common support which could severely bias the standard regression models. For these reasons, policy recommendations will be based on the results estimated using the PSM approach. A further advantage of this approach is that it can be used to investigate all three childhood adiposity outcomes: BMI, overweight and obesity. The results found using PSM are very similar to those using the conventional regression models, although this small difference gets modestly larger when breastfeeding is prolonged and exclusive, particularly when estimating BMI. This small differences might be attributable to the small number of observations which are excluded due to poor common support and could mean that the regression models are extrapolating poorly to the tails of the distribution and causing bias.

Results from this chapter suggest that breastfeeding produces a small but statistically significant reduction in childhood BMI and in the likelihood of childhood obesity and overweight. These effects increase as children get older and are stronger when breastfeeding is prolonged and exclusive. The likelihood of overweight is reduced to a

[43] This was also the case when the sample was restricted to the common support imposed by the PSM. This suggests that it is not simply imposing the common support which implies that there is no selection on unobservables.

greater extent than the likelihood of obesity as a result of breastfeeding. These results suggest that when creating policies aiming to reduce childhood obesity, policy makers should target breastfeeding as one part of a wider intervention by tackling a range of lifestyle influences.

2.6 Discussion and Conclusion

Results suggest that breastfeeding has a small but significant influence on childhood adiposity. The effects get larger and more significant as children get older and when breastfeeding is exclusive or prolonged. By the age of seven years, prolonged and exclusive breastfeeding accounts for a 0.28 drop in BMI, a 3.5% drop in the likelihood of overweight and a 2.5% reduction in the likelihood of obesity, *ceteris paribus*.

Even when applying methods which accounted for confounding factors, this study found that the causal effects of breastfeeding on childhood adiposity were insufficient to singlehandedly prevent childhood obesity. However, the small effect that breastfeeding was found to have suggests that breastfeeding should be included as part of any wider early life interventions which aim to reduce childhood BMI. It has also been shown that even small differences in adiposity at this age can lead to increasingly large differences as children get older.

This section discusses these results and their implications in more detail. Section 2.6.1 compares the methods and results from this chapter with the existing literature. Section 2.6.2 discusses the policy implications of the results and Section 2.6.3 discusses the limitations of this empirical chapter.

2.6.1 Comparisons with Existing Literature

The methods used in this chapter have added to existing research into the effects on breastfeeding on childhood adiposity in a number of ways. Unlike the existing literature, this study has used a range of methods which have allowed assumptions made by different models about the relationship to be assessed. For example, the IV technique and the Roy model allowed the endogeneity of breastfeeding to be tested in order to determine whether all important confounders had been accounted for. This shows that by using a rich set of variables such as those available in the MCS assuming selection into treatment only on observables can be sufficient to estimate a causal effect. This approach is similar to that

taken by Rothstein (2013) who investigated the effects of breastfeeding on cognitive outcomes and builds on the work of Beyerlein *et al*. (2008) who used multiple methods in this setting but did not use the same econometric techniques with the variety of assumptions as those used in this chapter.

Results from this study showed that when investigating the relationship between breastfeeding and childhood adiposity, in particular BMI, an appropriate statistical method should be used. The study shows that testing for endogeneity and investigating the fit of functional forms is important and to my knowledge, has not before been done in this particular setting.

This study found that breastfeeding had a stronger influence on the likelihood of overweight than on the likelihood of obesity. This contradicts findings from Beyerlein *et al.* (2008) who claimed that it was the children at the upper and lower tails of the BMI distribution who benefitted more from breastfeeding in relation to their BMI; they found that the largest reduction in BMI was in children who were the most obese. The fact that this study found that there was a different effect of breastfeeding on childhood overweight to that on childhood obesity, suggested that the relationship is different at different parts of the BMI distribution. This calls into question the functional form in the linear models, despite post-estimation tests finding no evidence of misspecification. Beyerlein *et al.* (2008) also suggested that the relationship was non-linear and went on to use a quantile regression. This also imposes, albeit a less restrictive, functional form. The PSM used in this chapter build on this work by reducing the reliance on functional form and directly addressing the issues of common support.

Although the effects found in this study were small, many were statistically significant, contradicting findings from a number of studies which found insignificant effects. For example, Oddy & Sherriff (2003), Jiang & Foster (2012), McCrory & Layte (2012), Reilly *et al*. (2005), Salsberry & Reagan (2005) and Kramer *et al*. (2007) all found no relationship between breastfeeding and recognised measures of childhood adiposity. The results also contradict Burke *et al*. (2005) who found that breastfeeding had a significant effect on adiposity in young children but that this effect became insignificant by the age of eight years. The difference between the results of this study and those listed above could be due to the large number of confounding factors which are accounted for in this study, removing any potential endogeneity of breastfeeding or because they were estimating different treatment effects. For example, Kramer *et al*. (2007) estimated an

intention to treat effect rather than the direct effect of breastfeeding on childhood adiposity.

Conversely, results from this chapter support studies such as Bergmann *et al*. (2003) who found a statistically significant reduction in BMI as a result of breastfeeding once a child was four years old. The results also support Armstrong & Reilly (2002), Gillman *et al*. (2001) and Mayer-Davis *et al*. (2006) who found that breastfed children are less likely to be overweight or obese. The findings from this study support studies such as Liese *et al*. (2001), who found that, although a relationship remained between breastfeeding and childhood adiposity, it was largely attenuated by confounding factors. The results from this study are not directly comparable with these studies because they each use different definitions of childhood obesity and overweight and look specifically at different durations and definitions of breastfeeding. However, the results from this study appear to show a slightly larger effect than other studies which have previously found a significant effect.

Throughout the chapter, data from the MCS was used in order to provide analysis which was representative to the UK population and could be used to inform UK policy makers. The MCS is a rich and nationally representative dataset with detailed information on infant feeding methods and childhood adiposity measures, as well as a wide range of possible confounding factors. Many studies within the existing literature used small samples taken from specific geographical locations or from specific institutions such as doctors' surgeries and in many cases the results might not be generalizable to the entire population. Other studies in similar settings have investigated the influences of breastfeeding on childhood outcomes using large representative datasets, but they are less common in the literature investigating the influences on childhood adiposity specifically.

2.6.2 Policy Implications

These results suggested that if policy makers can encourage mothers who would otherwise have never breastfed, to breastfeed exclusively for sixteen weeks, the BMI of their child would be reduced by 0.28 BMI points by the age of seven, *ceteris paribus*, an effect which becomes larger as children get older. Although these results appear small, they are larger than previous studies have found and represent around a 1.8% reduction in relation to the average BMI at this very early age. If policy makers can encourage mothers to breastfeed exclusively for longer, then any reduction which is apparent at this young age could produce a much larger effect as children get older and the distribution

of BMI widens. Exclusive breastfeeding for sixteen weeks would also reduce their likelihood of being overweight and obese, by 3.5% and 2.5%, respectively, compared to those who are never breastfed. These are the children who policy makers would be hoping to influence the most.

The majority of the effects are gained when infants are breastfed for at least four weeks, after which an additional twelve weeks of breastfeeding adds a relatively small amount to the overall effect, this is true for both partial and exclusive breastfeeding[44]. Although it seems like the marginal benefit of breastfeeding reduces with longer durations, there is still a positive effect and so breastfeeding should continue to be encouraged.

The small effects of breastfeeding on childhood adiposity are unsurprising; one would not expect breastfeeding to single-handedly solve the childhood obesity epidemic. However, if breastfeeding is encouraged alongside a range of other lifestyle interventions during early life it might be possible to collectively produce larger reductions in childhood BMI. The significant effects found in this chapter suggest that breastfeeding could be an important part of a wider lifestyle intervention which tackles a number of lifestyle behaviours in order to reduce childhood obesity. Further research into a range of early life influences and lifestyle behaviours could improve the understanding of how more complex lifestyle interventions could reduce obesity in childhood.

At the time that the data on breastfeeding in the MCS were collected, the WHO recommended that mothers should breastfeed their children exclusively for four months. Results from this chapter show that exclusive breastfeeding still has an additional influence on childhood adiposity when carried out for sixteen weeks or more. The results suggest that the current WHO recommendation of exclusive breastfeeding for six months should continue. However, due to the non-robust standard errors it is not possible to ascertain from these results whether these effects are statistically different from one another and therefore a dose response cannot be identified or ruled out.

Current WHO recommendations also suggest that partial breastfeeding should continue alongside other liquids and solids until a child is at least two years old. The results of this chapter suggest that partial breastfeeding continues to have an increasing effect on childhood adiposity until at least sixteen weeks. Further research into longer breastfeeding durations of partial breastfeeding would be required in order to identify

[44] It is unclear whether this additional effect is significant or not because the standard errors are not robust.

whether this effect will continue to increase or not, and robust standard errors would be needed in order to determine if any further increase was significant or not. The foods eaten alongside partial breastfeeding could also help to determine whether it is continued breastfeeding which has a biological effect or whether the types of mothers who breastfeed for longer are also those more likely to feed their children the better types of food. Within the MCS, very few mothers continued to partially breastfeed their children for two years[45]. Data from a different population who were more likely to breastfeed for longer durations, or more recent data relating to a period with the more recent WHO recommendations, could facilitate research into more prolonged partial breastfeeding and additional research is needed to make policy recommendations for longer durations of breastfeeding.

The results of this study contribute to public health research by taking a population-wide approach in order to estimate the average effects of breastfeeding on childhood adiposity. The findings from this chapter could potentially be useful for guidance developers such as NICE. The expert committees set up by NICE to help improve public health guidance in the areas of both childhood obesity and breastfeeding behaviours could make use of this research. In addition, the parameter estimates found in this chapter are arguably more robust than those found in previous studies and could be used in economic models for obesity or breastfeeding, as discussed in Chapter I.

Breastfeeding is also known to have a variety of other benefits and policy makers should continue to encourage mothers to prolong breastfeeding, regardless of its impact on childhood adiposity. Even if the benefits in relation to childhood adiposity are small, they are an addition to a range of other breastfeeding related beneficial outcomes, for both the mother and infant. The evidence provided by this chapter should be used alongside existing evidence available in the related areas.

2.6.3 Limitations and Future Research

Although this chapter contributes to the literature in a number of ways, it does suffer from limitations, some of which are discussed here.

The investigation into the effects of breastfeeding is limited in that one cannot randomise breastfeeding as one would a treatment in an RCT. A randomised breastfeeding treatment

[45] Less than 0.1% of mothers said they were still breastfeeding their child when they reached two years old. Responses taken from second wave of the MCS.

would provide sufficient common support over the entire outcome distribution. Using observational data creates a common support which may not support the entire sample but is the best alternative and PSM prevents estimations based on incomparable observations. This method also removes any John Henry and Hawthorne effects (Duflo *et al*., (2007). The MCS provides a rich set of observable characteristics, giving a much better set of variables than those used in many of the previous studies in the literature. The methods which took into account the potential endogeneity of breastfeeding found little evidence of selection into breastfeeding on unobservables which were correlated with childhood adiposity. Propensity score matching allows the causal relationship of breastfeeding on childhood adiposity to be estimated, but the method itself has limitations. For example, it allows only one parameter to be estimated for the effect of the treatment on the outcome. There are no parameter estimates for the effects of the remaining independent variables on the outcome which might also provide interesting results. However, the aim of this chapter was simply to identify the causal effects of breastfeeding while taking into account the other independent variables. Studies which are interested the effects of a range of independent variables would require a different statistical approach. The standard errors of the treatment effects estimated using PSM in this chapter are non-robust which prevents dose responses and direct comparisons between different effects to be statistically compared. Abadie & Imbens (2008) have shown that when robust standard errors are calculated using more recent software, these standard errors are reduced, meaning that these results would only become more significant. This gives further weight to the results but still does not allow an accurate comparison between different durations of breastfeeding etc.

The estimation using IV techniques suffers from the lack of a convincingly strong instrument. The instrument used in this chapter was delivery by caesarean section, similar to Denny & Doyle (2008), but as highlighted by Del Bono & Rabe (2012) it could be argued that individual-level instruments cannot be completely independent of the outcome equations. However, the Roy models presented no evidence that breastfeeding was endogenous and there was little evidence of endogeneity in the IV models. This suggests that once the rich set of variables in the MCS were accounted for and it was reasonable to assume selection only on observables.

Although the MCS includes a range of variables which are important in this chapter and is representative of the UK population, it also has limitations. The MCS has a significantly lower response rate than the previous British birth cohort studies. This is

likely to be due to the manner in which the participants were recruited, as explained by Plewis (2007). The MCS recruited participants through administrative child benefit records rather than through the NHS. This could have potentially lead to a lower response rate because parents may consider studies relating to the NHS and child health care as more important. Future research could also investigate the relationship between breastfeeding and childhood adiposity in children later in childhood as more additional waves of the MCS are collected and released. Data from subsequent waves would have allowed the relationship between breastfeeding and adiposity in later childhood and adolescence to be investigated. This would be an interesting extension to this chapter, considering how the relationship between breastfeeding and childhood adiposity got stronger as the children get older. The cohort nature of the data also means that the MCS only holds information on children born around the millennium and as the obesity epidemic and breastfeeding behaviours and trends change, the results based on these children might differ for children born at a different time. Repeating this analysis on data from other cohorts could determine how representative these result are to children born in different years.

In order to recommend breastfeeding for longer durations of partial breastfeeding, on the grounds of benefits to childhood adiposity, further research should be carried out into the benefits of longer durations of breastfeeding. The available data limits the length of duration of partial breastfeeding which can be investigated. Although the average age of cohort member during the first interviews is nine months, many are younger and sixteen weeks is the longest duration which can be investigated without reducing the sample size. There is also a relatively small proportion of mothers which breastfeed for this length of time, probably due to the recommendations at the time. Data which contains information on mothers more likely to breastfeed for longer might be better in identifying the effects of prolonged partial breastfeeding.

The MCS data contained no information on reasons for mothers not breastfeeding. Additional information on whether a mother chose not to breastfeed, her reason for doing so or whether there were any medical reasons that a mother could not breastfeed would have been useful in this analysis and could have provided more detailed policy recommendations.

As children get older, there will be many other influences on childhood adiposity which come into play. This could be the reason that breastfeeding only accounts for a small

difference in childhood adiposity. Future research into how childhood adiposity develops over time and how family lifestyle influences childhood adiposity more generally could help to understand these effects.

Although this chapter helps to disentangle the relationship between breastfeeding and childhood adiposity and provide causal inference, it cannot provide any information about why an effect might occur. There have been many theories suggesting different reasons for a relationship between breastfeeding and subsequent obesity (these were discussed in Section 2.1), but these can neither be confirmed nor rejected by the findings from this chapter. Further research into why breastfeeding reduces childhood adiposity could help policy makers to improve infant feeding in future interventions. Future research could also include analysis of the effects of changing the WHO recommended breastfeeding durations. Policy evaluation techniques could be implemented to determine whether the change in recommendations in the 2001 had a significant impact on breastfeeding duration or on childhood obesity.

Despite the numerous benefits of breastfeeding, there are potential disadvantages which might discourage women from breastfeeding, for example mothers returning to work. Renfrew *et al.* (2007) suggested that further research was needed into the barriers to breastfeeding including sore nipples and insufficient milk. Little research has been done into possible detriments of breastfeeding meaning that the reasons for mothers choosing not to breastfeed are not yet systematically understood.

Regardless of the limitations of this study, it offers an improvement, both in terms of breadth of the study as well as the techniques used as assumptions tested compared to the existing literature which investigates the same relationship. It provides an in-depth investigation into the relationship between breastfeeding and childhood adiposity under a wider range of assumptions allowing the most appropriate statistical method to be identified. Many of the methods used in this study have previously been used in related areas (see Iacovou & Sevilla-Sanz, 2010 and Del Bono & Rabe, 2012) but never to my knowledge to investigate the specific causal relationship of breastfeeding on any recognised childhood adiposity measure. Similarly, previous studies have compared different econometric techniques with a range of assumptions to more thoroughly explore the effects of breastfeeding on childhood outcomes (Rothstein, 2012) but again this has not previously been done specifically for the effects on childhood adiposity outcomes. Beyerlein *et al*. (2008) compares results from a range of methods but all of these methods

impose some sort of functional form and none account for the potential endogeneity of breastfeeding. This empirical chapter builds on their work to include additional econometric methods. This study overcomes many of the limitations found in previous studies by investigating the causal relationship of breastfeeding on childhood adiposity outcomes, whilst relaxing the assumptions of functional form and selection into treatment and assessing what assumptions are necessary in this particular case.

III. CHILDHOOD OBESITY AND UNDERLYING FAMILY LIFESTYLE

Research Questions:

- What is the causal effect of underlying family lifestyle on childhood weight status at each stage of early childhood?
- How does underlying family lifestyle evolve during early childhood?

Aims:

- To identify the underlying lifestyle in a family by exploiting the large number of variables available in the data.
- To explore the evolution of family lifestyle and its causal persistence during early childhood.
- To identify the extent to which family lifestyle mediates the relationship between socioeconomic and family background characteristics and childhood obesity.
- To provide evidence for policy makers and guidance providers interested in reducing childhood obesity through lifestyle interventions and to provide more long-term evidence for use in economic models.
- To explain how this causal effect is identified.

3.1 Introduction

The existing empirical literature has acknowledged that there is a link between family lifestyle and obesity, including childhood obesity. Consequently, the UK Government has implemented campaigns to improve the lifestyles of families in the UK in an attempt to help people lead more healthy lives. For example, the public health program Change4Life (started in 2009) aimed to change family lifestyles in order to tackle obesity and other health issues (DH, 2009). However, there is a lack of research into how this type of wide ranging intervention might help to improve childhood outcomes, including childhood adiposity. This chapter aims to inform future programs in order to improve them and enable them to be targeted at families who might need more help or benefit more from any interventions. By understanding the mechanisms by which these influences work, more evidence based policies can be developed. Specifically, the empirical analysis will identify the effects of underlying family lifestyle on childhood adiposity, how this underlying family lifestyle evolves over time, as well as a range of other parameters which allow this underlying family lifestyle to be directly estimated. This approach will inform policy makers about which children are likely to benefit most from interventions targeted at underlying family lifestyle and the long term effects of interventions which successfully improve family lifestyle.

This chapter will bring together various ideas from the existing lifestyle literature in order to determine how lifestyle is related to childhood adiposity. It will incorporate a range of mechanisms which have been observed in the previous literature. It will use multiple lifestyle outcomes (Balia & Jones, 2008) and include outcomes for different family members (Brown *et al*., 2013; Brown & Roberts, 2013). It will also allow underlying family lifestyle to be persistent over time (Ashenden *et al*., 1997).

The treatment effects of breastfeeding on childhood adiposity, estimated in Chapter III, were small but statistically significant suggesting that, in order to successfully tackle the childhood obesity epidemic, breastfeeding should be part of a wider early life intervention where a range of lifestyle behaviours should be addressed. The methods used in Chapter II identify only average effects, and although PSM provided these average effects for the treated and untreated subpopulations, the results are still limited when informing policy. The methods used in this chapter can explain much more in a single model by simultaneously estimating a range of parameters. As a result, this can be used to find answers to much more ambitious research questions than techniques which identify only

one parameter, such as the models in Chapter II (see for example Heckman & Urzúa, 2010).

This empirical chapter will investigate a range of lifestyle related outcomes. These outcomes will be highly correlated with each other because they each depend on an unobservable factor, family lifestyle, which underlies them. By simultaneously estimating multiple outcome equations, it is possible to identify this unobservable underlying factor and in doing so account for its effect on each of these outcomes. Moreover, the correlation between childhood obesity and parental obesity (Brown *et al*., 2013; Brown & Roberts, 2013), as well as between the lifestyles of family members (Golan & Weizman, 2001 and Lindsay *et al*., 2006) is well established in the literature. This suggests that there is a shared family lifestyle and children learn their lifestyle from their family and that all lifestyle outcomes and behaviours observed in a family are likely to be influenced by the same unobservable characteristics. Furthermore, the influence of these unobservable characteristics on childhood adiposity and the other outcome measures are themselves of interest. This study will use both childhood and parental adiposity, among other observable outcomes of family lifestyle in order to identify underlying family lifestyle throughout early childhood. These outcomes each measure some aspect of underlying lifestyle and are each influenced by the underlying factor. For this reason, they are often referred to as outcome measures (Cunha & Heckman, 2008; Ermisch, 2008; Hernández Alava *et al*., 2013).

The previous chapter used static models to investigate a single cause of childhood adiposity. For policy purposes, it is also important to know more about how effects come about and how they develop over time in order to be able to infer long time effects. This chapter takes a different approach to the previous chapter and estimates a dynamic model which enables a better understanding of how policies and interventions might help to reduce childhood obesity in the long run. Family lifestyle in one period of the life course is expected to be a strong indicator of family lifestyle in the next period because lifestyle habits tend to be persistent over time. As children grow up, family lifestyles are passed on from parent to child so family lifestyle is also expected to be persistent across generations. In this study, a dynamic relationship will be achieved by creating a structural model. The structural model imposes a relationship between the latent factors described above, in this case imposing an auto-regressive relationship on the latent factor for family lifestyle.

This dynamic framework will require more parametric assumptions than the PSM in the previous chapter in order to identify the more complex model. However, Heckman & Urzúa (2010) explain why these structural models are better than single parameter models for use in policy development because they allow different means to be estimated for observations with differing characteristics allowing the outcomes of different types of children to be investigated over time.

In summary, this empirical chapter builds on existing work in several ways. The methods used to estimate underlying family lifestyle, latent factor models, allow for a more comprehensive measure of underlying lifestyle by accounting for measurement error. This work builds on that of Balia & Jones (2008) who estimated a range of lifestyle outcomes using a multivariate probit model by estimating this underlying lifestyle itself, rather than just removing it from each of the outcome equations. The dynamic investigation into family lifestyle and how it influences childhood adiposity is also an important contribution to knowledge and builds on the many existing cross-sectional studies.

This study also adds to the evidence which could be used in economic models with the need for fewer assumptions and more robust extrapolation. The parameters which estimate the relationship between the latent factor and the outcome measures (the factor loadings) allow the identification of the latent factor at each time period and estimate their influence on the outcome measures. Although they are not all of primary interest in this chapter, future studies or economic models could utilise the results of this study as evidence of how underlying family lifestyle might influence other outcomes. In addition, the dynamic nature of the model allows more long-term evidence to be produced. This is evidence which is lacking in the existing literature and is of great importance to guidance developers such as NICE.

The analysis in this study shows that interventions which can successfully improve underlying family lifestyle could significantly reduce the risk of obesity and overweight in children and in their parents, as well as improving a range of other lifestyle outcomes. The persistent nature of underlying family lifestyle which is found suggests the need for strong policies which will be sufficient to shift the underlying trend of family lifestyle. Policies should be implemented as soon as possible during childhood and should be sustained throughout early childhood in order to have the greatest cumulative effects. The persistence of family lifestyle also suggests that any interventions which do have a

significant influence on underlying lifestyle will have long-lasting effects on childhood adiposity in addition to other lifestyle improvements for all family members. Simulations from the model show that the relationship between childhood obesity and socioeconomic or family background variables is heavily mediated by family lifestyle.

The remainder of this chapter is structured as follows. Section 3.2 will review the relevant literature and identify the contribution of this empirical chapter to the existing literature. Section 3.3 will discuss the dynamic factor model and how it will be used to provide simulated results. Section 3.4 will discuss the variables from the MCS used within the model. Section 3.5 will present the results from the dynamic factor model as well as the results from the simulations which use the model parameters. Finally, Section 3.6 will discuss the findings from this chapter and potential policy implications.

3.2 Literature Review

This section outlines the existing literature relating to lifestyle, including how it has been previously defined and measured and how it relates to adiposity. It goes on to explore the literature surrounding the demographic and social determinants of family and childhood lifestyle before investigating related policies and interventions which could be informed by this empirical chapter.

Due to the number of different aims to this chapter (outlined on page 139) and because the chapter seeks to jointly identify a number of causal parameters, the literature is broken down into a series of relevant sub-sections. For this reason, an 'investigative' approach which responds to new relevant concepts or lines of enquiry as they become apparent by reviewing the literature is used, as described by Gough *et al*. (2012). This approach was followed until no new lines of enquiry relevant to the research aims or the proposed model emerged. As in the previous chapter, this review is not intended to be a 'systematic review' and does not aim to cover every piece of related literature. It is a scoping review undertaken to identify gaps in the literature and issues with current measures, definitions and methodologies in existing studies. The review, as in the previous chapter, uses an 'interpretive' approach rather than an 'aggregate' approach, as described by Booth *et al*., (2012), to fulfil the aims of the literature review in the most efficient way.

The 'berrypicking' method outlined in the previous chapter and by Bates (1989) was used to extend the search from the previous chapter. This investigative approach (Gough *et*

al., 2012) allows the review to evolve as additional studies are found. These are additional studies which were not included in the previous review because they did not specifically investigate breastfeeding, but wider definitions and determinants of lifestyle, and the relationship between lifestyle and obesity. With the aims of the chapter in mind, the investigative approach used in this review identified the following concepts: *measures and definitions of lifestyle*, *determinants of lifestyle*, *family lifestyle and its relationship with childhood obesity* and *the dynamics of lifestyle*.

Due to the interpretive approach taken in this review, not every study related to this topic is included but instead enough studies are included to give an overview of each of the important issues identified during the review. Throughout the review, studies are prioritised depending on their relevance to each of the sub-sections of the review. Those most applicable to a UK population or similar setting are identified using their titles and abstracts and those which appear to add conceptually to the review are investigated in more detail and are included in the review where appropriate[46].

The remainder of this section is structured as follows. Section 3.2.1 discusses different lifestyle variables used throughout the literature and how previous studies have defined lifestyle. Section 3.2.2 explores the literature on the determinants of underlying family lifestyle. Section 3.2.3 reviews the empirical literature investigating relationships between lifestyle and obesity, specifically focusing on family lifestyle and childhood adiposity, whilst Section 3.2.4 considers the use of dynamic modelling of lifestyle and related variables in children. Section 3.2.5 considers existing lifestyle interventions and related policies and, finally, Section 3.2.6 outlines the original contribution to the existing literature of the empirical analysis presented in this chapter.

3.2.1 Lifestyle Variables and Definitions

One of the main problems when trying to estimate lifestyle, either family or individual, is that definitions vary. Contoyannis & Jones (2004) defined lifestyle as 'a set of behaviours which are considered to influence health and are generally considered to involve a considerable amount of free choice' and Mcleod & Ruseski (2013) explained that lifestyle choices, or 'health behaviours', were widely recognised by economists and epidemiologists as important non-medical health determinants. Many studies have aimed to measure individual lifestyle using health-related behaviours, such as smoking habits,

[46] Some articles were relevant in more than one of the identified sub-sections of the review.

alcohol consumption, participation in regular exercise and eating habits. However, no single observable variable can measure underlying family lifestyle perfectly.

The Alameda County Study identified seven lifestyle factors which it named the 'Alameda Seven'. The study was started in Alameda County, California in 1965 and recorded information on diet, exercise, weight-for-height, smoking, alcohol, sleep and stress. For a comprehensive review of this study see Housman & Dorman (2005). Although these seven lifestyle factors are not all directly applicable to children, these behaviours in parents could be used as indicators of a family lifestyle, opposed to the child lifestyle explicitly. Furthermore, the study was carried out in the US and so the lifestyle factors which were identified as a result of this study could be different in the context of UK families. However, they are a good starting point when investigating family lifestyle and are well established within the literature.

Many studies have focussed on diet, physical activity or sedentary behaviour when measuring lifestyle, especially when investigating the relationship between lifestyle and obesity. For example, Reilly *et al*. (2005), Bauer *et al*. (2011), Haug *et al*. (2009) and Janssen *et al*. (2005) each looked at the effects of physical activity, time spent watching television and diet in order to estimate the impact of lifestyle on childhood obesity. Childhood lifestyle, physical activity and dietary behaviours are heavily influenced by parental lifestyle variables (Bauer *et al*., 2011). For example, if parents take their children to playgrounds, parks or sporting events they are likely to be more physically active. Similarly, childhood diet, especially in younger childhood, is heavily dependent on parental influences. Parental lifestyle factors are also used when investigating the relationship between lifestyle and childhood adiposity. Mizutani *et al*. (2007) argued that smoking during pregnancy, a maternal lifestyle choice, could affect the weight of a child as they grew up. They used smoking during pregnancy as a proxy for underlying parental lifestyle during pregnancy.

Francis *et al*. (2003) concluded that the effect of television watching on childhood overweight was indirect and mediated through snacking which increased whilst watching television, suggesting that it is not sedentary behaviour, but diet, which has the largest effect on childhood adiposity. They found that this effect was also mediated by parental obesity. They also claimed that generations of children have watched television and the obesity epidemic began later than increases in television viewing so other factors must be influencing childhood overweight.

Conversely, in a cross country analysis, Janssen *et al*. (2005) suggested that physical activity should be the focus of policies aiming to reduce childhood obesity rather than focusing on diet. They found that countries where children participated in less physical activity and more sedentary behaviours had a higher prevalence of childhood obesity. The lifestyle variables they used in their analysis included dietary and physical activity variables such as fruit and vegetable intake and time spent watching television. They suggested that the WHO should take a 'leadership role' in the fight against childhood obesity due to its increasing prevalence worldwide.

It is also important to acknowledge correlations between the observable lifestyle behaviours of family members. Brown & Roberts (2013) investigated the strong correlation between maternal and adolescent BMI and found that observable characteristics accounted for only 11.2% of this correlation suggesting that the remaining correlation was partly due to genetics and other unobservable shared environments or underlying attitude. Furthermore, Brown & Roberts (2013) also suggested that the association between inactivity and adiposity in adolescents was embedded within the lifestyle of a family. Similarly, Brown *et al*. (2013) investigated the relationship between obesity in married couples and found a strong correlation between the BMIs of spouses. They put this relationship down to shared environmental and social influences.

Lifestyle is complex and not directly observable or measurable. In order to overcome this problem Balia & Jones (2008) used a multivariate probit model to simultaneously estimate a range of dependent lifestyle variables including smoking, alcohol consumption, whether an individual eats breakfast, sleeping patterns, obesity and exercise. This allows a more complete investigation of lifestyle to be estimated and does not focus simply on a single lifestyle behaviour. However, this approach is unable to estimate the underlying lifestyle (the cause of endogeneity) in order to determine how this underlying lifestyle influences observable lifestyle outcomes. This chapter will take a similar approach to that of Balia & Jones (2008) by jointly estimating a range of equations analysing lifestyle outcomes. At the same time it will identify a time-varying latent factor to represent the unobservable underlying family lifestyle which has an influence on each of them. In doing so, it is acknowledged that there are a wide range of theories and definitions of lifestyle used by different disciplines. Studies such as Cockerham *et al*. (1986), Barker & Osmond (1987) and Graham (2004) suggested that it is not lifestyle *per se* which affects health but socioeconomic variables such as housing, overcrowding and the lack of take up of free health care. The following section outlines some of these social

variables which are identified in the literature as influencing lifestyle and health-related behaviours.

3.2.2 Determinants of Family Lifestyle

The determinants of family lifestyle have received an increased amount of attention in recent years where a particular focus has been on the relationship with poor health outcomes[47]. In the existing literature, social factors influence lifestyle behaviours. For example, SES, education and income among other factors have been found to influence different lifestyle variables.

Wardle & Steptoe (2003) suggested that lifestyle is the combination of a range of lifestyle variables including smoking, physical activity and diet. They investigated each of these lifestyle variables separately and found that, in the UK, individuals with higher SES were more likely to eat healthily, exercise and were less likely to smoke. These individuals were more likely to make conscious lifestyle decisions and were less likely to believe that bad health was simply a consequence of chance, suggesting that it is differences in attitudes towards health that cause lifestyles to differ by SES.

Semmler *et al*. (2009) found that the effects of SES on childhood overweight were mediated through parental obesity suggesting that parental lifestyle could influence this relationship. Children with at least one obese parent were more likely to be overweight if they were from families with low SES than if they were from families with high SES. However, in families with no obese parents, SES had no statistically significant effect on childhood overweight. Semmler *et al*. (2009) used maternal education to proxy for family SES but did not account for paternal education, family income or employment status, all of which could further affect the relationship. These variables tend to be highly correlated with maternal education and might therefore capture the effects of family SES more accurately. The methodology used in this paper does not properly consider the assumptions made by the models used. Differences between the BMI z-scores in groups of children with different family characteristics were analysed using t-tests and analysis of variance (ANOVA), without accounting for any potential covariates. The ANOVA only accounts for age, sex and clustering for twins and failed to account for important lifestyle variables. While this study does not directly investigate childhood or family

[47] This is the focus of the next empirical chapter and so is not discussed in great detail here.

lifestyle, the links between childhood and parental adiposity suggest that families share a common lifestyle, particularly in families with lower SES.

Rhee *et al*. (2005) investigated the determinants of parental decisions to actively make changes to their family's lifestyle, specifically relating to diet and physical activity. They found that social factors played a large part in a parents 'readiness to change' in helping overweight children to lose weight, suggesting that parents from lower SES were less likely to change their lifestyles. Policies targeting parents which are more likely to be ready to change their lifestyles could make policies more effective. Different policies could be targeted at parents of different levels of readiness in order to maximise their impact. However, targeting parents who are more ready to make lifestyle changes could further the differences in lifestyle between families from high and low SES.

Currie (2011) found that mothers with lower SES were less able to provide a healthy environment for their child whilst pregnant. For example, mothers with low SES were more likely to smoke and drink during pregnancy. However, Currie (2011) did not investigate how the foetal environment affected the subsequent lifestyle of a child or family but the study suggested that children from different backgrounds experienced different environments and family lifestyles, even before birth.

Crosnoe (2012) measured family instability using a count of how many times a family structure changed. This included changes in a step-parent, single-parenthood, parental marital status as well as other changes in family structure. This study suggested that parenting situations had an impact on emotional health and as a result could affect childhood adiposity. Moreover, Cunha & Heckman (2009) found that single-parent households were less able to invest in their children and suggested that this could be due to differences in time constraints between single-parent and two-parent households, or the lack of resources available to single-parent households.

Vázquez-Nava *et al*. (2013) investigated the effects of family structure and maternal education on sedentary lifestyle in children between six and twelve years old. Although they looked only at a binary outcome variable indicating a sedentary lifestyle, this variable was created using information on a number of activities. These included time spent watching television, time and frequency of playing sport and time spent playing video games. The study found that children not living with both their natural, married parents were less likely to have sedentary lifestyles. Other studies such as McConley *et al*. (2011) and Quarmby *et al*. (2011) also found that family structure influenced different

lifestyle factors. McConley *et al*. (2011) found that children living with both natural parents were less likely to participate in risky lifestyle behaviours such as smoking. Quarmby *et al*. (2011) also found that family structure influenced sedentary lifestyles in children. They found that children living in two-parent families experienced more opportunities to participate in physical activity both with and without their parents participation. These two-parent families included step-families as well as natural parent couples, suggesting that time constraints, experienced by single parents, affected family lifestyle rather than having both natural parents in the household. Vázquez-Nava *et al*. (2013) found that, unlike family structure, maternal education did not have a statistically significant effect on sedentary lifestyle in children. However, other studies have found maternal education to influence specific aspects of childhood lifestyle. For example, Cribb *et al*. (2011) found that maternal education had a significant influence on the diets of ten year old children. Children with less educated mothers consumed more fast food and children with more educated mothers ate more fruit and vegetables. However, Cribb *et al*. (2011) used a one-way ANOVA to test the effects of maternal education on childhood diet and did not account for other lifestyle, demographic or socioeconomic factors which could influence this relationship. The subsequent section outlines a range of studies which explore the relationship between these lifestyle behaviours outlined above and their relationships to weight status.

3.2.3 Family Lifestyle and Childhood Obesity

A number of studies have investigated the influence that specific family lifestyle variables and other family behaviours have on adiposity during childhood. For example, Haug *et al*. (2009) and Janssen *et al*. (2005) used cross country data to examine childhood obesity levels and how lifestyle behaviours affected childhood adiposity in secondary school aged children. Both used logistic regression models to estimate the probability of obesity in children using the International Obesity Taskforce (IOTF) definitions of childhood obesity. Janssen *et al*. (2005) used a series of logistic regressions, one for each country analysed, whereas Haug *et al*. (2009) used only one multi-level logistic regression model to analyse all countries simultaneously. Both Haug *et al*. (2009) and Janssen *et al*. (2005) found that physical activity reduced the probability of obesity in childhood and both found some evidence that watching more television increased the likelihood of obesity. Janssen *et al*. (2005) also found that, contradictory to prior expectations, increases in the number of times a child consumes sweets reduced the likelihood of obesity in children in some countries, including England, Scotland and Wales. However, the portion sizes of sweets

consumed was not recorded, only the number of occasions when sweets were consumed, which could have led to this unexpected result. When children are young, parents will have a large influence over what their children eat and how frequently, as well as the amount of exercise that their children participate in. As children get older, parents might have less influence but it is expected that children will 'learn' their eating habits from their parents or families and continue to have the same underlying lifestyle throughout childhood.

Other studies investigating the relationship between lifestyle and childhood adiposity focused on other lifestyle behaviours, not specifically diet and exercise. Mizutani *et al*. (2007) investigated whether maternal smoking during pregnancy affected the BMI of Japanese five year olds. In accordance with Haug *et al*. (2009) and Janssen *et al*. (2005), Mizutani *et al*. (2007) used logistic regression models to estimate the probability of childhood overweight and obesity, controlling for a range of other lifestyle factors including continued smoking, drinking, eating habits and exercise. They found that children were more likely to be overweight or obese at the age of five years if their mothers smoked during pregnancy. This could have been due to biological factors or because parents with less healthy lifestyles were likely to feed their children less healthy food or provide them with a less healthy environment. However, a causal influence of smoking on overweight or obesity would be difficult to identify here because the study did not account for problems with self-selection and a lack of randomisation. The correlation between maternal smoking and childhood adiposity is only attenuated slightly by the confounding factors accounted for in this study. This attenuation is greater for the probability of childhood obesity than childhood overweight. After accounting for other lifestyle and demographic factors, a mother who smoked during pregnancy was twice as likely to have an overweight five year old and three times as likely to have an obese five year old.

Reilly *et al*. (2005), also using a logistic regression, investigated the relationship between early lifestyle factors and childhood obesity in UK seven year olds. They found that increased TV watching and parental obesity increased the likelihood of childhood obesity. However, they suggested that these relationships could be due to a shared familial environment. This is an important justification for the model used later in this chapter which will assume that families have a shared underlying lifestyle. Reilly *et al*. (2005) also found that low birth weight, smoking during pregnancy, lack of breastfeeding, early weaning, poor early eating habits and poor sleeping patterns all produced an increased

risk of childhood obesity. They claimed that early life factors played a crucial part in the prevention of childhood obesity.

Bauer *et al*. (2011) investigated the influences of lifestyle factors later in childhood on the BMI of girls between the ages of fourteen and twenty years in the US. They used a linear multilevel model to estimate the effects of a range of childhood and parental lifestyle behaviours on self-reported adolescent BMI. Parents were asked about the lifestyle of their child and how it was influenced by family discipline and habits. They found positive correlations between parent and child lifestyle variables, including their physical activity, time spent watching television, diet, weight and body composition. They found that although parental lifestyle had an influence on childhood lifestyle, adolescent weight was not directly affected by parental influences suggesting that by the age of fourteen children became responsible for their own lifestyles and that different indicators of lifestyle become important as children grow up. Bauer *et al*. (2011) used a US data set with a large proportion of participants from ethnic minorities (71%) which could produce different results to those found using UK data. Families from different ethnicities might lead different types of lifestyles due to cultural differences and their weight could be affected by cultural or genetic differences. Results found by Bauer *et al*. (2011) suggested that families share a common lifestyle and that lifestyle behaviours are learned by children from their parents. As well as lifestyle, Bauer *et al*. (2011) also found parental and childhood adiposity to be strongly related. This could be due to a shared family lifestyle which influences both parental and childhood adiposity rather than an intergenerational influence. Although the study only used a small number of parent-child dyads, it provided further evidence that parental lifestyle is an indicator of childhood lifestyle, even if there is no causal effect, and they suggested that parents play an important part in determining childhood BMI.

Giles-Corti *et al*. (2003) used a cross-sectional dataset from Western Australia to investigate the relationship between lifestyle factors and obesity in adults of working age in sedentary jobs. They used logistic regression models to predict overweight and obesity using a selection of demographic, socioeconomic and lifestyle variables. They found that sedentary activities such as watching television were strong predictors of overweight and obesity and that physical activity reduced the likelihood of overweight or obesity. Contrary to much of the other literature, SES had no effect on overweight or obesity in their models. However, the logistic regression models used in their study cannot provide a causal inference and any relationship found is an association. Further research into

causal lifestyle behaviours of childhood obesity is needed in order to properly inform policy makers of the most effective interventions. A potential problem relating to the studies outlined above is the fact that they analyse cross sectional data, or fail to exploit the panel nature of any data analysed. The next section discusses the existing literature which investigates lifestyle and related concepts using a dynamic framework.

3.2.4 Dynamic Modelling of Lifestyle

Within the existing literature a limited number of studies explore the determinants of lifestyle using a dynamic framework. Given the persistent nature of lifestyle (Gilleskie & Strumpf, 2005; Stringhini *et al*., 2010) it is argued that previous family lifestyle should be allowed to influence current family lifestyle and therefore that family lifestyle should be investigated over time. For example, Stringhini *et al*. (2010) emphasised the importance of investigating lifestyle behaviours over time and not just assuming that they are time-invariant. Stringhini *et al*. (2010) found that diet, physical activity and alcohol consumption varied over time. They found that the confounding nature of these lifestyle variables on the relationship between SES and mortality was more prevalent when multiple lifestyle variables were investigated over time compared to a single time point. Smoking did not have this effect, perhaps due to its habitual nature.

When investigating lifestyle dynamically it is important to acknowledge different approaches which are taken by different disciplines. There is a growing literature which uses a lifecycle approach, often used within epidemiology. A lifecycle approach suggests that advantage and disadvantage, in a socioeconomic context, cluster cross-sectionally and accumulate longitudinally, as described by Graham & Power (2004). This is also true of lifestyle behaviours; risky lifestyle behaviours cluster cross-sectionally and their effects can accumulate over time. Braveman (2014) explained how the life-course approach allows health in later life to be influenced by previous experiences, not just dynamically over a lifetime but also through generations. These experiences could include lifestyle variables, which are expected to influence later health and be persistent across generations.

The majority of research investigating the dynamics of lifestyle focuses on a particular lifestyle behaviour or outcome rather than on overall individual or family lifestyle. Single-item proxies for lifestyle or lifestyle-related variables are readily available in many datasets and simplify analysis. For example, Gilleskie & Strumpf (2005) investigated the persistence of smoking behaviour in US adolescents using data from the National

Education Longitudinal Study, 1988 to 1992. They explored whether current smoking behaviour was caused by previous smoking behaviour or whether this relationship was due to unobserved heterogeneity. They used lagged smoking behaviour to predict current smoking behaviour and included expectations of future smoking behaviour as well as the past, present and expected price of cigarettes. They found that previous smoking behaviour influenced current smoking behaviour and that individual heterogeneity was not the only cause of the persistence of smoking behaviour. They also used simulations to estimate the impact that price changes might have on future cigarette consumption and found that price increases could lead to a reduction in smoking. This reduction appeared to occur as a result of fewer smokers rather than a reduction in the number of cigarettes consumed by each smoker.

Balia & Jones (2008) used data from the British Health and Lifestyle Survey to investigate the impact of health and lifestyle on mortality. Similarly to Contoyannis & Jones (2004), they defined lifestyle as behaviours which were influenced by both choice and circumstance and suggested that lifestyle choices were influenced by the extent to which an individual discounts the future causing unobservable heterogeneity. They used a dynamic multivariate probit model to measure individual lifestyle outcomes, using a range of observable behavioural variables. These included smoking, drinking, sleeping patterns, obesity, physical activity and breakfasting habits. They allowed the probability of mortality to depend on initial health and lifestyle variables and similarly they allowed health to depend on previous lifestyle variables. Balia & Jones (2008) found that individuals who were not obese had a lower probability of death and fewer morbidities. They also found that individuals who exercised more regularly were healthier. Contrary to the majority of existing literature, they also found that eating breakfast had a positive impact on the risk of mortality once selection on unobservables was accounted for. However, this coefficient was insignificant and eating breakfast was also found to be endogenous. This study only used individuals over the age of forty due to low mortality rates in younger people. Consequently, parental lifestyle had little effect on the sample and the authors found evidence supporting the exclusion of parental lifestyle variables using likelihood criteria. Although Balia & Jones (2008) investigated the effects of lifestyle on morbidity and mortality over time, they did not investigate the persistence of lifestyle or how an underlying lifestyle might influence obesity over time.

Like Balia & Jones (2008), Cunha & Heckman (2008) also estimated a range of observable variables simultaneously. However, Cunha & Heckman (2008) did so using

a dynamic latent factor model to investigate the dynamics of cognitive and non-cognitive skill formation during childhood and investigated the effects of family environment and parental investments on these skill formations throughout childhood. Although they investigated skill formation rather than lifestyle evaluation, they emphasised the importance of family and parental influences on childhood outcomes. As a result, the authors could identify the most appropriate stages of childhood in which to target policies aimed at parents in order to increase parental investment and have the largest impact on childhood outcomes, in this case human capital. This chapter will use a similar methodology to that used by Cunha & Heckman (2008). Rather than dynamically modelling cognitive and non-cognitive skills throughout childhood, this chapter will dynamically investigate underlying family lifestyle throughout childhood and look more closely at its influence on childhood obesity. The following section explores the literature relating to existing policy interventions directed at family and childhood lifestyle, as opposed to the determinants or consequences of underlying family lifestyle or single-item lifestyle behaviours.

3.2.5 Family Lifestyle Interventions

Improving family lifestyles remains high on policy agendas for health departments across the developed world, see for example Sure Start, Change4life and Start4life. This section discusses existing UK policies and national interventions, what they aim to do and who they are targeted at. It also highlights existing studies that explore the effect of policy interventions on lifestyle choices. This section serves to put the research implemented in this thesis into context and show how the results from this chapter can further inform potential policy interventions, as opposed to estimating the influence existing policies might have. Initially this section outlines the existing policies relating to family lifestyle and subsequently goes on to explore the effects of past policies.

Existing UK Lifestyle Policies and Interventions

Behaviour change interventions are preventative strategies which aim to promote positive behaviours or choices. They can be aimed at individuals, families or communities. It is assumed that these positive health messages encourage people to adopt improved health and lifestyle behaviours (or reduce poor ones) increasing the likelihood of good health. There are a number of national level interventions which have been developed over recent years in the UK. However, the majority of these have not been evaluated and so it is hard to determine their effectiveness. A number of recent policies in the UK have aimed to

help families, specifically those with younger children, to live healthier lifestyles. These include Change4Life, Start4Life and the Healthy Start program, amongst others. Each of these policies attempted to directly tackle the lifestyle of parents, children or the entire family.

Change4Life is a national marketing campaign which aims to reduce obesity in the population by encouraging behaviour change (Department of Health, 2009). It is part of a wider government strategy aimed at reducing obesity, set out by the Cross-Government Obesity Unit, the Department of Health and the Department of Children Schools and Families (2008). Start4Life is aimed specifically at parents of infants with an aim of reducing the prevalence of obesity in childhood. It has a particular focus of extending average breastfeeding durations. Again, this is a national campaign (throughout England) run alongside Change4Life.

The Department of Health's Healthy Start program is targeted at pregnant women, families on low incomes and teenage mothers. It provides these families with vouchers for fresh milk, fruit and vegetables as well as infant formula milk as well as supplying vitamins for both mothers and children. It also provides information on breastfeeding and eating healthily. The initiatives implemented by the Health Start program were created using an evidence-based approach and many took guidance from NICE, as well as other scientific and public health bodies. For example, the committee on Medical Aspects of Food Nutrition Policy and Scientific Advisory Committee on Nutrition recommended the use of vitamins and the methods of implementation of the Healthy Start program were developed using recommendations from NICE (2008) guidance on maternal and child nutrition. The Healthy Start program is intended to work alongside the Start4Life campaign and there is an emphasis given by the Department for Health to ensure that the messages given by each of these initiatives are consistent with each other.

In addition, local authorities as opposed to national bodies, are increasingly tasked with tackling health problems because a growing number of services which influence health behaviours are falling under their control. For example, NICE (2012) guidance asserts the importance of developing a sustainable, community-wide approach to obesity, and the National Obesity Observatory recommends weight management interventions as part of a wider approach to the development of local care pathways for obesity (Cavill & Ells, 2010).

Despite the numerous policies implemented to date which aimed to improve childhood health and lifestyle, much of the current literature has suggested that more needs to be done and policies should be targeted at specific children, parents and families who are most at risk. There are also a number of studies which have criticised existing health policies, for example, Fitzpatrick (2001) suggested that the majority of 'health policies' aimed to control the lifestyles of individuals and how they lived, rather than to improve health. They suggested that any health benefits were of secondary importance. The criticism of health policies is not a new occurrence. Coulter (1987) suggested that health policies and health care systems widened the gap between social groups due to the lack of knowledge and uptake in lower SES groups.

Health inequalities play a large part in influencing childhood health, including obesity prevalence. In 2008, WHO published a report into health inequities entitled 'Closing the Gap in a Generation', written by the Commission on Social Determinants of Health (CSDH) (2008). The report focused on a global population and tackled issues such as lifestyle, or health behaviours, education, geographical environment, employment and policy targeting, amongst others. Although this report investigated the issues surrounding global health inequity and inequality in children and adults, UK children are affected by many of the issues that the report identified. The report outlined a range of lifestyle variables including smoking, alcohol consumption, physical activity, diet and nutrition, as well as a range of physical, social and environmental factors. These variables could be considered to be influenced by an unobserved underlying lifestyle. The CSDH took the approach that it was not lifestyle choices which influenced health but the wider social and environmental conditions which affected lifestyle behaviours. They therefore encouraged policy makers to target these wider social determinants rather than the lifestyle behaviours themselves.

Further research into the performance of these policies aiming to reduce inequalities and how they improve observable outcomes in UK families are needed. However, there is research evaluating the performance of some smaller interventions and policies; these are discussed below.

Intervention Evaluation

In the existing literature there have been a range of methods used to explore the effects of policy interventions on lifestyle. For example, RCTs have also been used to investigate the effects of lifestyle interventions on childhood obesity, similar to the PROBIT trials

discussed in the previous chapter. For example the Cochrane review on obesity interventions reviewed 55 international studies and found, despite many studies being successful in improving the nutrition or physical activity of children, relatively few studies found a significant effect of the interventions on childhood adiposity (Summerbell *et al*., 2009). Moreover, McCallum *et al*. (2007) analysed data from an Australian RCT which aimed to identify whether participating in the 'live eat play' intervention reduced the likelihood of childhood obesity. This intervention was carried out by GPs in Australia between 2002 and 2004. Treated participants were provided with four GP consultations over three months in order to discuss healthy lifestyle changes. The RCT analysed the data collected over twelve months following the intervention. McCallum *et al*. (2007) found no difference between the mean BMI of the intervention and control groups but found that parents of children in the intervention group reported more improvements in childhood nutrition compared to those in the control group. If these changes in childhood nutrition were long lasting then it is possible that a reduction in BMI could result from the intervention later in childhood. However, they acknowledged that the RCT could have been limited by the delivery of the intervention; there was no check on how well the intervention was delivered by the GPs.

Ho *et al*. (2012) reviewed the effectiveness of some lifestyle interventions, which aimed to reduce childhood obesity, in a meta-analysis. These lifestyle interventions were predominantly dietary and exercise related interventions. They found that lifestyle interventions were generally effective in reducing BMI in children. They also suggested that incorporating diet into any lifestyle intervention was essential in reducing obesity.

Analysing data from an RCT, Boutelle *et al*. (2011) aimed to establish whether parent only lifestyle interventions were less effective than parent and child interventions in reducing childhood obesity. Using a linear mixed-model with a random effect error term to account for potential heterogeneity caused by clustering between treatment groups, they found that parent only lifestyle interventions were no less effective than interventions targeting both parents and children. This provides further evidence to support the idea that parental lifestyles play an important role in determining child outcomes and supports the argument for an underlying family lifestyle which is learned by the child.

One potential problem with studies which analyse the effectiveness of lifestyle interventions, such as those outlined here, is that they could influence the normal behaviour of the participants. These effects, known as the Hawthorne and John Henry

effects, were mentioned in the previous chapter and explained in more detail by Duflo *et al*. (2007).

Consequently, the empirical analysis presented in this chapter aims to help inform potential lifestyle interventions by identifying both the most effective time for intervention as well as identifying the most at risk individuals.

3.2.6 Summary

This review highlights the need for further research into the relationship between lifestyle and childhood adiposity using a more comprehensive measure of underlying lifestyle. Studies such as Reilly et al. (2005) and Boutelle et al. (2011) highlighted the need for lifestyle to be measured at a family level when investigating childhood lifestyle. Although some studies have investigated the relationship between lifestyle behaviours and adiposity, there is a gap in research investigating an underlying more general attitude towards lifestyle. The lifestyle variables discussed in Section 3.2.1 were generally specific single-item lifestyle behaviours, correlated with lifestyle but not encompassing the wide range of behaviours influenced by an overall underlying family lifestyle definition. No single-item lifestyle behaviour can perfectly measure underlying family lifestyle, they each have measurement error. This chapter builds on work by Balia & Jones (2008) who used a multivariate probit model to simultaneously estimate a range of lifestyle behaviours. However, while their method accounts for the endogeneity of unobservables in the correlation of error terms, it does not directly estimate the cause of this endogeneity or the effect that this underlying factor has on each of the lifestyle outcomes. Similar to Balia & Jones (2008), this chapter jointly estimates a range of lifestyle outcome measures but extends this work by using a latent factor, similar to those used by Cunha & Heckman (2008), to measure underlying family lifestyle from birth to the age of seven years. It will also assume that this underlying family lifestyle can be altered by interventions as well as social circumstance. It will investigate the extent to which childhood adiposity, as well as other observable family lifestyle variables are influenced by this underlying family lifestyle at each period in the model.

In this chapter, it is assumed that underlying lifestyle will be affected by circumstance and social determinants. It is also assumed that this 'lifestyle' is to some extent, learnt by children from their parents, especially during the early years of life. A further discussion of the definition of underlying family lifestyle which is assumed in this chapter is provided in Section 3.3 which discusses the methodology used in this chapter. This is

because the statistical analysis used in this chapter has implications on how underling lifestyle must be defined.

Similarly to the previous empirical chapter, much of the literature discussed in this review used logistic regressions models and cross-sectional data. This does not allow the relationships between lifestyle and childhood obesity to be investigated dynamically and so an appropriate time for intervention cannot be assessed. Some studies used RCTs to investigate the effects of lifestyle interventions on childhood adiposity at different ages but they rarely have follow up periods long enough to investigate the effects of any intervention throughout childhood. This highlights the need for longitudinal studies in this area of research and cohort data will allow more long term childhood outcomes to be investigated. By dynamically modelling underlying family lifestyle, it will be possible to determine the effect that this underlying family lifestyle has on future lifestyle and on childhood adiposity throughout childhood, as well as other observable family lifestyle behaviours.

The existing literature emphasised the importance of accounting for socioeconomic influences, such as SES and maternal education, on lifestyle behaviours and outcomes, including obesity. Wardle & Steptoe (2003) found that observable lifestyle behaviours differed by SES and other socioeconomic characteristics because of the influence that these social characteristics had on an underlying attitude towards healthy behaviours. In accordance with this finding, this chapter will allow socioeconomic variables to influence underlying family lifestyle therefore allowing them to have an indirect influence on the single-item lifestyle behaviours.

The report by the CSDH (2008), discussed previously, suggested that policy makers should consider how children from different backgrounds might be affected differently by interventions. In allowing socioeconomic and family background variables to influence the underlying family lifestyle which will be the focus of interventions, this chapter will enable the identification of children most at risk of childhood obesity, those who will benefit the most to changes in underlying family lifestyle and how to most effectively reduce lifestyle inequalities amongst children.

Additionally, this study will use a larger data set than those most commonly used in previous longitudinal analysis in this area of research. The MCS represents families across the UK and contains a wide range of variables which could be used to identify

underlying family lifestyle, as well as social determinants and childhood adiposity variables.

In summary, this chapter will contribute to the existing literature in several distinct ways. It will use a dynamic latent factor to construct a measure of underlying family lifestyle which evolves over time and explore how this underlying construct is related to childhood weight status. An important contribution of this chapter is the use of a dynamic modelling approach to explore the relationships between lifestyle and childhood adiposity. Underlying family lifestyle will be modelled dynamically to estimate how family lifestyle in one period influences family lifestyle in the next period, allowing the persistence of family lifestyle to be investigated. It will use a large nationally representative survey, which includes socio-economic information on both parents and children, allowing for a wide range of confounding factors to be considered. It will simultaneously estimate social influences on underlying family lifestyle allowing the effects of underlying family lifestyle to be investigated for children with a range of different socioeconomic and family background characteristics.

3.3 Methodology

This section describes the dynamic latent factor model to be estimated in order to investigate the persistence of underlying family lifestyle. This is a complex model which simultaneously estimates a system of equations in order to identify a range of parameters rather than the single average treatment effect that the models in the previous chapter identified. This methodology has previously been used in a range of related literatures. For example, Heckman (2012) discussed how this type of model could be used to investigate cognitive and non-cognitive ability and health during childhood. James Heckman has previously used similar latent factor models with a number of co-authors and in a wide range of settings: these include Heckman *et al*. (2006), Heckman (2007), Conti *et al*. (2010) and Cunha *et al*. (2010) amongst others. Latent factor models have also been used by Deb & Trivedi (2006) to explore selection effects in the utilization of health care, Hernandez & Popli (2013) who investigated parental input and Morciano *et al*. (2014) who investigated standard of living and disability.

By using a dynamic latent factor model to estimate underlying family lifestyle, this chapter builds on work by Balia & Jones (2008) who simultaneously estimated a range

of observable lifestyle behaviours using a multivariate probit model. Although their model, like the latent factor estimated in this chapter, jointly estimates a variety of lifestyle outcomes, there is one important difference. Balia & Jones (2008) account for the effect from unobservables through the correlation of the error terms in each equation but they do not directly estimate the underlying variable which is causing the correlation. The methodology used in this chapter jointly estimates lifestyle outcomes, while at the same time estimating (or measuring) the underlying unobservable family lifestyle which is one of the sources of correlation across the outcome measures in the model.

The research questions at the start of this chapter explained that the purpose of this study is to identify the causal influence of underlying family lifestyle on childhood weight status but also the evolution of this underlying family lifestyle during the early years of childhood. The only way to jointly estimate both of these effects is to use a structural model which uses a range of lifestyle outcome measures. Using only a single proxy for lifestyle would lead to biased estimates caused by measurement error. By using a structural equation which estimates the evolution of this latent factor over time, the persistence of this underlying family lifestyle can be investigated and it is possible to explore the implications of early versus late lifestyle interventions. The parameters from this type of model could be utilised by economic or cost-effectiveness models in order to determine the effectiveness of money spent at different stages of lifestyle under the restrictions of scarce resources. For this reason, this study is interested in both the child weight equation as well as the structural model which imposes the dynamic relationship, i.e. the part of the model which estimates the relationships between latent factors. Using the results of a dynamic latent factor model, simulations can then be used to predict childhood adiposity outcomes for children from different types of family and socioeconomic backgrounds *etc*.

The remainder of this section is structured as follows. Section 3.3.1 outlines the dynamic latent factor model, how it identifies underlying family lifestyle and is able to estimate its causal influence on childhood adiposity. Section 3.3.2 discusses the factor scores estimated by the model and Section 3.3.3 explains the use of simulations from the model.

3.3.1 A Dynamic Latent Factor Model of Family Lifestyle

Many statistical models used in the existing lifestyle literature do not account for the wide variety of variables which together make up a more comprehensive measure of underlying family lifestyle. Generally, one lifestyle variable is used as a proxy for overall lifestyle.

However, as outlined in Section 3.2, lifestyle is multidimensional and a range of observable lifestyle variables could be used to identify underlying lifestyle. No single-item lifestyle measure can perfectly measure underlying lifestyle, especially when investigating underlying family lifestyle which involves the lifestyle variables of more than one family member. This suggests that in order to create a comprehensive lifestyle factor, a range of observable lifestyle variables should be considered. This will avoid focussing on one particular lifestyle variable of an individual and will allow new exploration of the underlying lifestyle of a whole family.

Latent Factors, Endogeneity and Causality

If childhood obesity is treated as a single dependent variable, i.e. the only outcome in the model which is influenced by a range of other observable lifestyle behaviours then there is likely to be a problem of endogeneity within the model. Many lifestyle variables are affected by the same unobservable characteristics and so it is important to consider problems that might arise due to endogeneity.

Assume an equation estimating childhood adiposity $\boldsymbol{y}$ as a function of other variables lifestyle indicators $\boldsymbol{I}$ (for example maternal adiposity) so that

$$\boldsymbol{y} = \boldsymbol{f}(\boldsymbol{I}, \boldsymbol{\varepsilon}). \qquad \text{(III.1)}$$

Childhood adiposity is known to be strongly correlated with other lifestyle outcomes and so it is expected that a significant effect of $\boldsymbol{I}$ on $\boldsymbol{y}$ would be found. In order for a model such as that in Equation (III.1), to produce unbiased estimates, one would have to assume that the lifestyle indicators in $\boldsymbol{I}$ are exogenous, that they are uncorrelated with any unobservable error within the model. However, both child and other lifestyle outcomes (e.g. maternal adiposity) are likely to be influenced by the same unobservable characteristics, say $\boldsymbol{\theta}$. Theoretically, all lifestyle outcomes ($\boldsymbol{y}$ and $\boldsymbol{I}$**)** are expected to be influenced by some 'underlying family lifestyle' which encompasses attitudes towards lifestyle and the general lifestyle behaviours of a family. However, this underlying family lifestyle is unobservable. It is therefore, this underlying family lifestyle $\boldsymbol{\theta}$, which is the source of endogeneity in the model. If this unobservable cause of endogeneity were to be included,

$$\boldsymbol{y} = \boldsymbol{f}(\boldsymbol{I}, \boldsymbol{\theta}, \boldsymbol{\varepsilon}) \qquad \text{(III.2)}$$

then the effects of $\boldsymbol{I}$ on $\boldsymbol{y}$ might no longer be significant.

In this context, the source of endogeneity itself is of interest. Many public health interventions have focused on improving the general lifestyle of a family and so the effect that this underlying family lifestyle has on lifestyle outcomes and how underlying family lifestyle evolves during early childhood are of interest in this chapter. For this reason, estimating $\boldsymbol{\theta}$ itself is important. Due to the unobservable nature of $\boldsymbol{\theta}$ the only way to estimate it is using a common factor model.

A common factor model allows the range of lifestyle indicators, also known as outcomes or outcome measures to be used in the identification of the latent factor which represents underlying family lifestyle, in effect measuring the underlying concept. The outcome of interest, $\boldsymbol{y}$ will be jointly estimated alongside each of the indictors $\boldsymbol{I}$. In this model, both $\boldsymbol{y}$ and $\boldsymbol{I}$ are dependent variables, collectively measure the underlying concept. This determines a causal relationship of underlying family lifestyle on childhood adiposity $\boldsymbol{y}$.

The underlying lifestyle factor is unobserved but the MCS has a number of observable lifestyle variables for the cohort member as well as for their mother and father. These observable lifestyle characteristics are used to identify a latent factor to represent underlying family lifestyle in the same way that Cunha & Heckman (2008) used latent factors to model the dynamics of human skills formation. This method allows underlying unobserved variables to be estimated using multiple observable variables which are considered to be directly influenced by the underlying factor[48].

A common factor model does not use observable variables to measure the 'true score' of the factor. In these models, the factor being measured is an underlying concept, one which can never be directly measured by any single variable without the presence of measurement error. This common factor, or latent factor, model is written

$$\boldsymbol{I_t} = \boldsymbol{\lambda_t \theta_t} + \boldsymbol{\xi}_t. \qquad \text{(III.3)}$$

Equation (III.3) is a vector of equations, each estimating a different lifestyle indicator. The vector of indicators $\boldsymbol{I}$ are are not interpreted as direct measures of this latent factor and vector of error terms $\boldsymbol{\xi}$ are assumed to be independent of the underlying factor $\boldsymbol{\theta}$. Each of the indicators are considered to measure different aspects of the underlying concept but also include an 'item-specific' part. That is, each indicator can be broken down into a common and specific part. The common part is that which indicates the relationship between the underlying factor and the indicator. A vector of factor loadings

[48] This is the same idea as that used in measurement error models.

$\boldsymbol{\lambda}$ represents the sensitivity of the corresponding indicator to a change in the underlying factor. Now that the underlying factor can itself be estimated, the endogeneity it causes is accounted for and the causal effect of the factor on the outcome of interest can be estimated. These dependent variables are in effect collectively measuring the underlying factor.

The error term $\boldsymbol{\xi}_t$ can be broken down into two parts, a 'factor specific' $\boldsymbol{s}_t$ part and a 'measurement' error $\boldsymbol{e}_t$, so that

$$\boldsymbol{\xi}_t = \boldsymbol{s}_t + \boldsymbol{e}_t. \qquad \text{(III.4)}$$

This common factor is so-called because it is a common determinant of each of the indicators used in its estimation. It is explained further by Skrondal & Rabe-Hesketh (2004) along with the differences between these models and other types of measurement models, including the congeneric measurement model (Jöreskog, 1971) which, although statistically similar[49] to the common factor model, has a different interpretation. Using factor models in a situation where the indicators are considered to influence the factor would be a misspecification and these variables should not be considered as part of a common factor model, but can be included as independent variables influencing the latent factor. This is also discussed by Skrondal & Rabe-Hesketh (2004) in more detail.

The type of model used in this chapter has been used in other studies for a range of different purposes and can be tailored to individual problems or research questions. For example, Hancock *et al*. (2015) identified a latent factor for disability using three different datasets in order to determine whether survey data give consistent measures of the underlying disability concept in relation to the receipt of attendance allowance benefit[50]. Hancock *et al*. (2015) used a static latent factor model in order to answer their research question. They separate the receipt of attendance allowance from the vector of other disability outcomes because this outcome is inherently different from the others. This model is an example of how the causal effect of a latent factor on the outcome of interest can be identified.

Other studies have also been interested in the dynamic evolution of an underlying factor. For example, Cunha & Heckman (2008) used a dynamic factor model in order to

[49] The error term of the common factor model can be broken down into the common and item-specific parts, the congeneric model has only the item-specific measurement error.

[50] The equation estimating the receipt of attendance allowance is part of their latent concept and is one of their outcome measures. They have separated it from the vector of other outcome measures to emphasise its importance in their study but it has the same interpretation as the other equations in their disability measurement model.

investigate the evolution of cognitive and non-cognitive development in which the identification of the latent factors representing them each is central. This paper investigates the evolution of these child development factors during early childhood and how parental investment influences them over time. This model differs from that in this chapter because the focus is on how the latent factors are influenced rather than how they influence observable outcomes. In order to 'anchor' their factors, Cunha & Heckman (2008) estimate the effects of these factors on adult outcomes which allows the scale of the factors to be identified. This is discussed later in greater detail.

This chapter aims to, amongst other things, identify the causal influence of underlying family lifestyle on childhood adiposity and as a result, a common latent factor approach, described above, is the most appropriate method. It is the only method which allows the causal effects of underlying family lifestyle on childhood adiposity to be identified as well as the evolution of this latent factor over time. Heckman & Urzúa (2010) also discuss the advantages of using structural models rather than IV models, particularly when using the results for policy purposes. The structural models can predict how policies which have not yet been implemented might affect a range of observable outcomes in individuals with different characteristics.

In this chapter, a range of dependent variables will be used to indicate underlying family lifestyle in each period. These include the outcome of interest $\boldsymbol{y}$ as well as a range of other indicators $\boldsymbol{I}$. Collectively these observable variables will be referred to here as outcome measures and will be denoted using vector $\boldsymbol{Y}$ due to their identical statistical nature. This is in accordance with Cunha & Heckman (2008) and Cunha *et al*. (2010) and is the terminology that is used throughout the remainder of this thesis. These outcome measures are also often referred to as indicators (Skrondal & Rabe-Hesketh, 2004) because they can be used as an indication of the underlying factor, response variables (Muthen, 1984) because they respond to the underlying factor, or outcomes (Heckman *et al*., 2006). These terms are used interchangeably throughout the literature but it is important to remember that in this chapter, although each of these dependent variables are referred to as outcome measures, childhood adiposity is the outcome of interest and the other outcome measures are used to measure the underlying factor, i.e. they are indicators of family lifestyle.

These outcome measures are correlated with each other, an assumption which is not restricted by the model. Although the majority of studies that use these structural models

in econometrics aim to identify the causal effect of latent factors on future or adult outcomes, it is just as conceivable to identify the causal influence of the latent factor on the outcome of interest, in this case childhood adiposity (for other examples see Hancock *et al*. (2015) or Hernandez Alava *et al*. (2011). Indicators of family lifestyle are related to the latent factor, according to the following equations. These are in accordance with the common latent factor models discussed by Skrondal & Rabe-Hesketh (2004). Latent indicators depend on the underlying factor,

$$\boldsymbol{I}_t^* = \boldsymbol{\lambda}_t \boldsymbol{\theta}_t + \boldsymbol{\xi}_t \qquad \text{(III.5)}$$

where $\boldsymbol{I}_t^*$ is the unobserved latent variable underlying each indicator $\boldsymbol{I}_t$ and $\boldsymbol{\lambda}_t$ is a vector of factor loadings indicating the sensitivity of the latent indicator $\boldsymbol{I}_t^*$ to a change in the underlying family lifestyle factor $\boldsymbol{\theta}_t$. The error terms are assumed to be normally distributed; $\boldsymbol{\xi}_t \sim N(0, \sigma_{\xi t})$ where $\sigma_{\xi t}$ is the standard deviation at time t. Probit models are used to show the relationship between binary indicators and underlying latent family lifestyle. Similarly, ordered probit models are used to show the relationship between ordinal indicators and underlying latent family lifestyle. The latent variables underlying each of these binary and ordinal indicators are those in Equation (III.5). Continuous indicators are equal to their corresponding underlying latent indicator, so that $\boldsymbol{I}_t = \boldsymbol{I}_t^*$.

The outcomes of interest (child weight in the initial wave and child weight status in subsequent waves) are also estimated jointly with the indicators. In the same way as Equation (III.5), the outcome of interest has an underlying latent variable

$$\boldsymbol{y}_t^* = \boldsymbol{\rho}_t \boldsymbol{\theta}_t + \boldsymbol{\delta}_t \boldsymbol{W}_t + \boldsymbol{\epsilon}_t \qquad \text{(III.6)}$$

where $\boldsymbol{y}_t^*$ represents the unobserved latent variable underlying to outcome of interest at time t, $\boldsymbol{\rho}_t$ is the sensitivity of this outcome to the latent factor at time t analogous to the factor loadings $\boldsymbol{\lambda}_t$ and $\boldsymbol{\epsilon}_t$ is an error term analogous to $\boldsymbol{\xi}_t$ in Equation (III.5). Again, when this outcome is continuous[51] is equal to its corresponding underlying latent value, so that $\boldsymbol{y}_t = \boldsymbol{y}_t^*$. In addition, the model allows independent variables in matrix $\boldsymbol{W}_t$ to influence this outcome of interest $\boldsymbol{y}_t^*$ where $\boldsymbol{\delta}_t$ is a vector of corresponding time-varying coefficients. These or other independent variables could also be allowed to influence indicators $\boldsymbol{I}$ in the same way. However, due to the large number of time-varying

[51] In this case, the only continuous outcome of interest is child weight in the initial period.

parameters being estimated in the model already independent variables are only included in the parts of the model which are most important in answering the research questions.

By jointly estimating these outcome measures ($\boldsymbol{y}$ and $\boldsymbol{I}$) for underlying family lifestyle in each period, it is possible to estimate the causal effect of lifestyle on childhood adiposity in each period. This is done by ensuring that potential endogeneity is addressed. If underlying family lifestyle was identified using only the indicators $\boldsymbol{I}_t$, the endogeneity between the outcomes $\boldsymbol{y}_t$ and the underlying factor $\boldsymbol{\theta}_t$ would remain.

For simplicity, Equations (III.5) and (III.6) are stacked together into a vector of k outcome measures $\boldsymbol{Y}$, which each depend on underlying family lifestyle, such that the vector of latent variables underlying the outcome measures is

$$\boldsymbol{Y}^*_{kt} = \boldsymbol{\lambda}_{kt}\boldsymbol{\theta}_t + \boldsymbol{\delta}_{kt}\boldsymbol{W}_{kt} + \boldsymbol{\xi}_{kt} \tag{III.7}$$

where $\boldsymbol{\lambda}_{kt}$ is the factor loading for the k^{th} outcome measure and $\boldsymbol{W}_{kt}$ is a vector of independent variables which, in this chapter, affect only the outcome of interest, with corresponding coefficient vector $\boldsymbol{\delta}_{kt}$.

A set of threshold parameters, $\boldsymbol{\tau}^j_{kt}$ are simultaneously estimated for each of the binary and ordinal outcome measures at time t for $j = 0, \dots, J_k$ where J_k is the number of categories in outcome measure k at time t and $\boldsymbol{\tau}^0_{kt} = -\infty$ and $\boldsymbol{\tau}^J_{kt} = +\infty$. Thresholds are strictly increasing so that

$$\boldsymbol{\tau}^0_{kt} < \boldsymbol{\tau}^1_{kt} < \cdots < \boldsymbol{\tau}^{J-1}_{kt} < \boldsymbol{\tau}^J_{kt}. \tag{III.8}$$

Outcome measure k takes the value j when the latent outcome measure lies between thresholds $j-1$ and j;

$$\boldsymbol{\tau}^{j-1}_{kt} < \boldsymbol{Y}^*_{kit} \leq \boldsymbol{\tau}^j_{kt} \quad \Rightarrow \quad \boldsymbol{Y}_{kt} = j. \tag{III.9}$$

This model allows different outcome measures $\boldsymbol{Y}_{kt}$ to measure underlying family lifestyle at different stages of childhood. This is important because, although family lifestyle is well established by the time a child is born, it could manifest itself in different ways and different outcome measures might better indicate underlying family lifestyle at different stages of early childhood. The outcome measures of lifestyle at a given time are correlated with each other because they are each influenced by underlying family lifestyle $\boldsymbol{\theta}_t$. It is assumed that there is no remaining correlation between these measures

once the influences of underlying family lifestyle have been accounted for. Lifestyle outcome measures will be chosen for each period of the model using exploratory factor analysis (EFA) to investigate which of the lifestyle variables, informed by the literature, are most correlated with each other and are most representative of an underlying family lifestyle.

The Structural Model: The Relationship between Latent Factors

A 'full latent variable model' (see Byrne, 2012) consists of two parts. They are the measurement model (in this case the common factor model discussed above) and a structural model, described here. The structural model is one which illustrates the relationships between the latent factors, in this case it models the evolution over time of the latent factor underlying family lifestyle. This definition of the structural model is consistent across the literature when discussing structural equation models (Byrne, 2012; Skrondal & Rabe-Hesketh, 2004). In this chapter, the structural model is the dynamic process because it is the process by which family lifestyle changes over time which provides the relationship between each latent factor. The structural model which estimates a causal relationship between the latent factors is often of the most interest to a study. In this chapter, the causal relationship between the latent factor and the outcome of interest is also of particular interest and the structural model can show how this effect accumulates over time. Both the structural and measurement (or latent factor) parts of this model help to answer the research questions asked at the start of this chapter.

Initial latent family lifestyle, $\boldsymbol{\theta}_0$ is assumed to be

$$\boldsymbol{\theta}_0 = \boldsymbol{X}_0'\boldsymbol{\beta}_0 + \boldsymbol{u}_0, \tag{III.10}$$

where $\boldsymbol{X}_0$ is a vector of independent variables influencing initial lifestyle, $\boldsymbol{\beta}_0$ is a vector of estimated coefficients corresponding to these independent variables and $\boldsymbol{u}_0$ is a vector of normally, independently and identically distributed (IID) error terms with zero mean and variance σ_u. The error term also includes a time-invariant individual random effect $\boldsymbol{\eta}$, where $\boldsymbol{u}_0 = \boldsymbol{\eta} + \boldsymbol{\varepsilon}_{i0}$. This random effect is essentially a family effect. However, there can be no distinction made between errors at the individual-level or the family-level. This is because the analysis is only carried out for one child in each family. For this reason, this error term will be referred to as an individual random effect throughout the remainder of this thesis.

Family lifestyle is assumed to evolve over time according to a dynamic process, so that

$$\boldsymbol{\theta}_t = \alpha_t \boldsymbol{\theta}_{t-1} + \boldsymbol{X}_t^{'} \boldsymbol{\beta}_t + \boldsymbol{\eta} + \boldsymbol{\varepsilon}_t \qquad t = 1 \ldots 3 \tag{III.11}$$

where $\boldsymbol{\theta}_{t-1}$ is the latent family lifestyle in the previous period and α_t are the coefficients for the lagged latent factor. $\boldsymbol{X}_t$ is a vector of independent variables influencing family lifestyle at time t and $\boldsymbol{\beta}_t$ is a vector of corresponding coefficients. The error term in this dynamic process is decomposed into a time-varying error term, $\boldsymbol{\varepsilon}_t \sim N(0, \sigma_\varepsilon)$ which is IID and a time-invariant unobserved individual random effect, $\boldsymbol{\eta} \sim N(0, \sigma_\eta)$. The unobserved random effect is correlated with the individual random effect in the error term in the initial conditions, $\boldsymbol{u}_0$ in Equation (III.10).

The individual random effect $\boldsymbol{\eta}$ was found to be insignificant and was consequently removed from the final model. This insignificant individual random effect suggests that there are no remaining time-invariant individual effects in the evolution of family lifestyle.

The independent variables represented by $\boldsymbol{X}_t$ can differ over time. Some of these variables will be included only in the initial conditions and others will be included in each time period. Table III-2 in the data section discusses these variables and which period they are each included in.

Identifying Assumptions

For model identification one can either fix the variance of the error terms in the structural model or fix one of the factor loadings to an arbitrary constant[52] (see Skrondal & Rabe-Hesketh (2004) for more discussion on these methods of identification). In this study the variance of the error term, $\boldsymbol{u}_0$ in Equation (III.10) (σ_u) is fixed at 0.05 and the variance of error terms, $\boldsymbol{\varepsilon}_t$ in Equation (III.11) (σ_ε) are fixed at 0.01. This is known as factor standardisation and the magnitudes of these variances are arbitrary but allow the model to be identified. The method of identifying the latent factor is arbitrary and has no influence on model results[53]. It is not possible to identify both the means and the intercepts in Equations (III.10) and (III.11) because the factors $\boldsymbol{\theta}_t$ are latent variables and because both the dependent variable and the error terms in these equations are

[52] This method of identification is sometimes referred to as anchoring (Skrondal & Rabe-Hesketh, 2004), but should not be confused with anchoring the latent factors to adult outcomes.
[53] The same results were found when fixing one factor loading equal to 1 in each latent factor.

unobservable[54]. In the same way that probit models are identified by restricting the variance to one (normalisation), so is the structural part of this model. These arbitrary restrictions do not have any influence on model predictions.

An alternative method of identification is the 'anchoring' method used by Cunha & Heckman (2008) and Cunha *et al*. (2010), amongst others. This method uses continuous adult outcomes in order to identify the latent factors. By simultaneously estimating the existing model with a continuous adult outcome, the latent factors have numerical meaning in relation to the adult outcome and to each other. This outcome must be a future outcome with a meaningful and continuous metric in order to pin down the scale of the factor. This is not the method of identification which is used in this study.

In theory, all of the parameters estimated by this model could be time-varying but empirically some of the parameters may have to be fixed in order for the model to converge. These parameters are fixed for empirical estimation rather than identification; these variables are not needed for the model to be theoretically identified. This will be discussed further in Section 3.5 with the model results.

The underlying family lifestyle in each period changes independently of all outcome measures. Underlying family lifestyle is a conditioning variable in each of these equations and therefore any changes in the underlying factor are assumed to influence each of the outcome measures.

The error terms in the measurement models, Equation (III.7), are assumed to have zero mean and be independent of each other across observations, time periods and independent of the latent factor.

There may or may not be a relationship between independent variables $\boldsymbol{W}$ in Equation (III.7) and $\boldsymbol{X}$ in Equations (III.10) and (III.11). Any relationship between them would not affect this model unless perfect multicollinearity exists. In this case, since there are no variables included in both vectors $\boldsymbol{X}$ and $\boldsymbol{W}$, it is assumed that multicollinearity is not a problem[55]. The variables included in both vectors are discussed later in Section 3.4.2.

[54] The same problem occurs in an ordered probit model, where the parameter estimates have no numerical meaning and arbitrary identifying assumptions are made. In both the structural model here and an ordered probit model, fixing parameters at different arbitrary values in order to identify the model will produce different parameter estimates. However, these different values make no empirical or practical difference to either model.

[55] In principle, vectors $\boldsymbol{X}$ and $\boldsymbol{W}$ could contain the same variables and influence all outcome measures as well as family lifestyle directly, but exclusion restrictions would be needed in order to identify each of the separate effects that the independent variables were having.

Assuming identification, it is reasonable to assume that family lifestyle, as it is defined and identified here, has a contemporaneous influence on childhood adiposity. The latent factor can be interpreted as representing the normal underlying family lifestyle in recent weeks or months that has led to the outcome measures in Equation (III.7). Although the latent factor is estimated by outcomes which are observed at time t, this underlying lifestyle has already had an influence on these outcome measures. There is no way of identifying the exact time that this measured lifestyle occurs.

Despite the interpretation of the latent factor discussed above, an additional model will also be estimated which will assume that underlying family lifestyle has a lagged influence on child weight status. In this additional model, a change in lifestyle which has a contemporaneous influence on lifestyle outcome measures including parental weight status does not have an effect on child weight status until the next period. These models will then be compared using the Akaike and Bayesian Information Criteria (the AIC and BIC, respectively) in order to determine which model has the best fit. This model is identical to that above but Equation (III.6) is replaced with Equation (III.12).

$$\boldsymbol{y}_t^* = \boldsymbol{\rho}_t \boldsymbol{\theta}_{t-1} + \boldsymbol{\delta}_t \boldsymbol{W}_t + \boldsymbol{\epsilon}_t \qquad \text{(III.12)}$$

The results from this model will be compared to the original model outlined above in the results section but, based on the published literature, are not expected to fit the data as well. The models described in this section are complicated due to the large number of parameters that they estimate. For this reason, a number of different ways of representing the results will be used to aid the models interpretation. This includes analysis using standardised parameters, factor scores and simulations. These are discussed below.

Standardised Parameters

The standardised parameters will be provided in the results section and give a more intuitive representation of the results than the unstandarised parameters given as standard in the model. These parameters are standardised using the variances of independent variables $\boldsymbol{X}$ and outcome measures $\boldsymbol{Y}$. For example, linear regression $\boldsymbol{Y} = \boldsymbol{Xb} + \boldsymbol{u}$, has standardised parameters

$$\boldsymbol{b}_{std} = \boldsymbol{b} \cdot \frac{\sigma_{\boldsymbol{X}}}{\sigma_{\boldsymbol{Y}}} \qquad \text{(III.13)}$$

where $\boldsymbol{b}$ is the vector of unstandardised parameters, and $\sigma_{\boldsymbol{X}}$ and $\sigma_{\boldsymbol{Y}}$ are the standard deviations of $\boldsymbol{X}$ and $\boldsymbol{Y}$, respectively. These standardised parameters are more comparable

than the unstandardised parameters and show that, for a change in $\boldsymbol{X}$ by one standard deviation, $\boldsymbol{Y}$ is estimated to change by $\boldsymbol{b}_{std}$ standard deviations.

The dynamic latent factor model is estimated by simulated maximum likelihood using expectation maximisation algorithm and Monte Carlo integration with 3,000 integration points. Robust standard errors are computed using a sandwich estimator. The model is estimated using Mplus 6.1 (Muthen & Muthen, 2011).

3.3.2 Factor Scores

Factor scores are the numerical values of the underlying factors for each observation and are estimated using the observable characteristics of each observation. The factor scores have no cardinal scale or numerical interpretation, but can be used to pinpoint where on the distribution of family lifestyle each observation lies.

The factor scores estimated by the dynamic factor model of underlying family lifestyle at each period of childhood are investigated here. The estimated values for these latent factors, or factor scores, have no numerical meaning because there is no natural metric for underlying family lifestyle and therefore cannot be used to compare family lifestyle across time. This also means that the persistence parameter, α_t in Equation (III.11) does not have a direct numerical interpretation because the factor scores are not on the same scale in each period. One way around this would be to anchor the latent factor, $\boldsymbol{\theta}$, to an adult outcome which could be used to provide more intuitive and comparable scales to the factor scores, such as is done by Cunha *et al*. (2010). However, the Millennium Cohort study does not have any adult outcomes available because the cohort of children are not yet adults. This limits the direct comparison of the factor scores over time and the direct interpretation of the persistence parameter α_t.

However, the factor scores do allow the relative standing of family lifestyle to be identified. It is the ranking of the factors scores and how easy it is for families to move up or down these rankings which provide the meaningful information. Factor scores are estimated using posterior distributions where

$$\boldsymbol{Y}^* = \boldsymbol{\lambda\vartheta} + \boldsymbol{\delta W} + \boldsymbol{\xi} \qquad \text{(III.14)}$$

where $\boldsymbol{Y}^*$ is a vector of both observed and latent responses, including the latent variable underlying child adiposity $\boldsymbol{y}^*$. Across all time periods, $\boldsymbol{\vartheta}$ is a four-dimensional[56] vector of latent family lifestyle factors and $\boldsymbol{\lambda}$ is a matrix of corresponding factor loadings. Additionally, $\boldsymbol{W}$ is a vector of independent variables with a corresponding vector of estimated coefficients $\boldsymbol{\delta}$, again across all time periods, and $\boldsymbol{\xi}$ is a vector of residual errors. Additionally,

$$\boldsymbol{\vartheta} = \boldsymbol{B\vartheta} + \boldsymbol{\beta X} + \boldsymbol{e} \tag{III.15}$$

where $\boldsymbol{\vartheta}$ is a vector of the latent factor in each period, $\boldsymbol{B}$ is a four-by-four parameter matrix of the slopes for regressions of latent factor on itself at each time point, $\boldsymbol{X}$ is a vector of independent variables with corresponding coefficients, $\boldsymbol{\beta}$, and $\boldsymbol{e} = \boldsymbol{\eta} + \boldsymbol{\beta}$ is a vector of error terms made up of an unobserved individual random effect and residual errors. It is assumed that $\boldsymbol{B}$ has diagonal elements zero and that $(\boldsymbol{I}_4 - \boldsymbol{B})$ is non-singular.

The expected mean of $\boldsymbol{\vartheta}$ given $\boldsymbol{X}$ is then

$$E(\boldsymbol{\vartheta}|\boldsymbol{X}) = (\boldsymbol{I}_4 - \boldsymbol{B})^{-1}\boldsymbol{\beta X} = \mu \tag{III.16}$$

and has conditional variance

$$Var(\boldsymbol{\vartheta}|\boldsymbol{X}) = (\boldsymbol{I}_4 - \boldsymbol{B})^{-1}\psi(\boldsymbol{I}_4 - \boldsymbol{B})^{-1} = \Sigma \tag{III.17}$$

where $\psi = Var(\boldsymbol{\varepsilon})$.

The posterior distribution of $\boldsymbol{\vartheta}$, given $\boldsymbol{Y}$ and $\boldsymbol{X}$, is

$$g(\boldsymbol{\vartheta}|\boldsymbol{Y},\boldsymbol{X}) \propto \phi(\boldsymbol{\vartheta}|\boldsymbol{X})\mathrm{f}(\boldsymbol{X},\mathbf{Y}|\boldsymbol{\vartheta X}) \tag{III.18}$$

where $\phi(\boldsymbol{\vartheta}|\boldsymbol{X})$ is multivariate normal with mean vector, μ and covariance matrix Σ.

Families are ranked in order of their factor scores in each period of the model making it possible to investigate the persistence of underlying family lifestyle. It is also possible to investigate the intra-distributional dynamics of family lifestyle, for example, whether families at one end of the distribution find it easier to move around this distribution than families at the other end.

Although the factors scores in this chapter are not anchored to adult outcomes, as in Cunha *et al*. (2010) it is still possible to make comparisons over time and by using factor

[56] The latent factor in each time period adds a dimension. There is one latent factor in each of the four periods in the model, resulting in four dimensions.

score percentiles or simulations (discussed in the following section). It is still possible to determine how easily families move up or down the rankings of family lifestyle.

3.3.3 Predictions

This section outlines how parameter estimates from the dynamic latent factor model will be used to simulate the likely outcomes of children and families from the sample and for those with different sets of hypothetical characteristics. The latent factor within the model, defined by the joint estimation of Equations (III.7) to (III.11), need to be integrated out of the likelihood function in order to be estimated. This requires the computation of a four-dimensional integration. To avoid the complexities of these integrals, simulations are used to approximate them.

Using simulations, it is possible to predict outcomes for children with specific characteristics or from specific backgrounds and determine which children will benefit most from family lifestyle interventions, for example, which children are expected to lose more weight as result of changes to their lifestyle. The simulations which are presented in this chapter are similar to those presented by Heckman et al. (2006) in their article and in the corresponding web appendix. The authors simulated outcome measures both at time t and in the future. This highlights the capabilities of this type of model to predict a range of observable outcomes, both contemporaneous outcome measures influenced by the underlying latent factors and future outcomes when they are available in the data[57].

When predicting an expected value or probability for the outcome of interest $\boldsymbol{y}$, conditional on independent variables $\boldsymbol{X}$ and $\boldsymbol{W}$, there is a conditional distribution,

$$f(\boldsymbol{y}|\boldsymbol{X},\boldsymbol{W}) = \int f(\boldsymbol{y}|\boldsymbol{\vartheta},\boldsymbol{W}) \cdot f(\boldsymbol{\vartheta}|\boldsymbol{X}) d\boldsymbol{\vartheta}. \qquad \text{(III.19)}$$

Conditional on independent characteristics $\boldsymbol{X}$ and $\boldsymbol{W}$, the expected value of $\boldsymbol{y}$ is the mean of that conditional distribution,

$$E(\boldsymbol{y}|\boldsymbol{X},\boldsymbol{W}) = \int \boldsymbol{y} \left[\int f(\boldsymbol{y}|\boldsymbol{\vartheta},\boldsymbol{W}) \cdot f(\boldsymbol{\vartheta}|\boldsymbol{X}) d\boldsymbol{\vartheta}\right] d\boldsymbol{y}. \qquad \text{(III.20)}$$

For continuous variables Equation (III.14) is integrated over all values of $\boldsymbol{y}$ and for discrete variables the sum of the integrals for each of the values of $\boldsymbol{y}$ is calculated. These

[57] As mentioned earlier, this study does not include any future adult outcomes because they are not available in the data.

calculations allow, for example, childhood weight status to be predicted for children at specific ages, conditional on independent variables.

When predicting $\boldsymbol{y}$ conditional on indicator I_k, as well as independent characteristics, the conditional distribution is

$$f(\boldsymbol{y}|\, I_k, \boldsymbol{X}, \boldsymbol{W}) = \frac{\int f(\boldsymbol{y},\, I_k|\boldsymbol{\vartheta}, \boldsymbol{W}) \cdot f(\boldsymbol{\vartheta}|\boldsymbol{X}) d\boldsymbol{\vartheta}}{\int f(\, I_k|\boldsymbol{\vartheta}, \boldsymbol{W}) \cdot f(\boldsymbol{\vartheta}|\boldsymbol{X}) d\boldsymbol{\vartheta}} \qquad \text{(III.21)}$$

and so, conditional on independent characteristics, $\boldsymbol{X}$ and $\boldsymbol{W}$, as well as indicator I_k, the expected value of $\boldsymbol{y}$ is

$$E(\boldsymbol{y}|\, I_k, \boldsymbol{X}, \boldsymbol{W}) = \int \boldsymbol{y} \left[\frac{\int f(\boldsymbol{y},\, I_k|\boldsymbol{\vartheta}, \boldsymbol{W}) \cdot f(\boldsymbol{\vartheta}|\boldsymbol{X}) d\boldsymbol{\vartheta}}{\int f(\, I_k|\boldsymbol{\vartheta}, \boldsymbol{W}) \cdot f(\boldsymbol{\vartheta}|\boldsymbol{X}) d\boldsymbol{\vartheta}} \right] d\boldsymbol{y}. \qquad \text{(III.22)}$$

Again, for continuous variables, Equation (III.14), is integrated over all values of $\boldsymbol{y}$ and for discrete values, the sum of the integrals for each value of $\boldsymbol{y}$ is calculated. These calculations allow childhood weight or weight status to be predicted for specific children at certain ages, conditional on independent variables as well as other indicators such as parental weight status. Similarly, maternal weight status could be predicted conditional on independent variables and child weight status. These conditioning variables do not have a causal influence on the predicted outcome and for this reason it is not these variables which this study aims to provide policy information about. They are simply conditioning variables included in order to determine which children's adiposity is most affected by their family lifestyle.

These simulations will use parameter estimates from the dynamic latent factor model estimated in Mplus 6.1 and simulations in this chapter are estimated using a user-written program in Stata 12[58].

3.4 Data

As in the previous empirical chapter, this chapter uses data from the first four waves of the MCS. For a detailed description of the MCS design, see Chapter I.

[58] The Stata program referred to here was written by me specifically for the analysis in this thesis.

3.4.1 Latent Factors for Family Lifestyle

For each latent family lifestyle factor, a range of different outcome measures are used to indicate family lifestyle. These outcome measures are chosen in accordance with the definition of family lifestyle outlined in Section 3.1, as well as the existing lifestyle literature and using EFA. EFA determines the way in which observable variables group together. Outcome measures used to indicate an underlying factor are each expected to be highly correlated with each other and with the factor itself. A number of potential variables which could represent part of an underlying lifestyle factor are included in the EFA. Those identified as being highly correlated with the factor best representing an underlying construct for family lifestyle during each period are included in the latent factors of family lifestyle.

During the early years of childhood, parental lifestyle variables are expected to be the most prominent outcome measures of family lifestyle whilst the child will have little influence. The first period of the model contains the initial conditions, lifestyle variables experienced by the family between the beginning of a pregnancy and until the child is nine months old. The variables used to measure underlying family lifestyle in each period are outlined below. First, the initial conditions are discussed followed by the variables used to measure the subsequent underlying family lifestyle factors.

Initial Conditions for Family Lifestyle

The first wave of data is used to determine the initial conditions, that is, the conditions influencing family lifestyle at the start of childhood. These measures are available in the MCS from around the time of birth or soon after. Many of these variables are similar to those used in estimating the propensity scores in the previous empirical chapter and exclusive breastfeeding behaviour is included in the model as an outcome measure of initial underlying family lifestyle. The variables used as lifestyle measures in each period, including this initial period, are displayed in Table III-1.

Table III-1: Outcome Measures of Underlying Family Lifestyle

Outcomes of Latent Factors (Y or Y^* in Equation (III.7))			
Initial Family Lifestyle	**Family Lifestyle at 3 Years**	**Family Lifestyle at 5 Years**	**Family Lifestyle at 7 Years**
Weight at nine months (kg)	Weight category	Weight category	Weight category
Maternal pre-pregnancy weight category	Maternal weight category	Maternal weight category	Maternal weight category
Father's Weight Category	Paternal weight category	Paternal weight category	Paternal weight category
Mother's Smoking Behaviour whilst pregnant	Mother is a smoker	Mother is a smoker	Mother is a smoker
Planned pregnancy	More than three hours of TV/computer per day	More than three hours of TV/computer per day	More than three hours of TV/computer per day
Breastfeeding behaviour	Regular meals	Regular meals	Eats breakfast everyday
-	-	Times per week plays sport	Times per week plays sport
-	-	Goes to playground or park at least once a week	Goes to playground or park at least once a week
-	-	-	Unhealthy snacks between meals

Source: All variables are from or derived from the Millennium Cohort Study.

During the first wave of interviews in the MCS, childhood weight was recorded. In this chapter, all weights have been converted into kilograms. As in the previous chapter, childhood weight has been trimmed to remove any biologically implausible values (BIVs)[59]. Similarly, maternal pre-pregnancy weight status is determined using maternal pre-pregnancy BMI, in accordance with the previous chapters. The same is done to determine paternal weight status during the first wave of MSC interviews. A categorical variable indicating maternal smoking behaviour before and during pregnancy is created. The categories used include '*not smoked in the past two years'*, '*smoked in the two years leading up to their pregnancy but quit once they found out they were pregnant'* and '*smoked throughout pregnancy'*. A binary variable identical to that used in the previous chapter is used to indicate whether a pregnancy was planned or not. Due to the wide range of breastfeeding durations experienced by the cohort members, an ordinal categorical variable is created splitting the cohort members into five groups depending on length of exclusive breastfeeding. These groups are '*never breastfed'*, '*exclusively*

[59] Any values not lying between -5 and +5 z-scores are considered implausible.

breastfed for up to four weeks', '*between four and thirteen weeks'*, '*between fourteen and seventeen weeks'* and '*over seventeen weeks'*. The variable uses answers from the same questions on infant feeding from the MCS used in the previous chapter.

Each of these variables are observable outcomes of the latent factor representing the initial family lifestyle of a child when $t = 0$.

Subsequent Family Lifestyle Outcome Measures

Variables taken from subsequent waves of the data are used to indicate family lifestyle throughout later stages of childhood and are also displayed in Table III-1. Latent factors for family lifestyle are created for children at ages three, five and seven years old. The outcome measures used to indicate underlying family lifestyle are allowed, by the model, to differ throughout childhood; as children get older, their underlying family lifestyle is identified by different lifestyle variables. These variables are represented by $\boldsymbol{Y}_t$ in Equations (III.7) when $t > 0$.

Childhood weight status is used as a measure of underlying family lifestyle at each subsequent stage of childhood. By using categories rather than BMI, allows weight in kilograms, height, age and sex to be accounted for. Both maternal and paternal weight status at the times of each interview are included in as underlying family lifestyle outcome measures for all periods. Maternal smoking status is a behavioural outcome of underlying family lifestyle in each period. A binary variable will indicate whether or not the mother is currently a smoker at the time of each MCS interview. A binary variable indicating whether or not a child watches television or plays computer games for more than three hours each day is an outcome of family lifestyle at ages three, five and seven years. Similarly, a binary variable indicating whether a child has regular meal times is an outcome of underlying family lifestyle for children aged three and five years. At age seven, a binary variable indicating the consumption of unhealthy snacks between meals is introduced. The number of times each week that a child participates in sport, either '*never'*, '*once'*, '*twice'*, '*three times'* or '*four or more times',* is an outcome of family lifestyle in children aged five and seven years. In addition, whether or not the child visits parks or playgrounds at least once a week is a family lifestyle outcome in five and seven year old children. By the age of seven, information is available on the number of times a week a child eats breakfast and a binary variable is included to indicate whether or not a child consumes breakfast on a daily basis.

3.4.2 Independent Variables

Independent variables are included in the model when estimating the latent lifestyle factors. These are variables which influence latent family lifestyle over and above their influence on individual outcome measures. The literature suggests that social and family background variables influence family lifestyle.

Maternal education, family SES and family structure are each included as independent variables which are allowed to directly influence underlying family lifestyle, represented by $\boldsymbol{X}_t$ in Equations (III.10) and (III.11). These independent variables influence outcome measures through their influence on underlying family lifestyle. Table III-2 displays the independent variables used in the model to directly influence underlying family lifestyle at each time period t. It also shows the periods in which each of these variables are allowed to influence family lifestyle.

Table III-2: Independent Determinants of Underlying Family Lifestyle

Determinants of Underlying Family Lifestyle, *X* in Equations (III.10) **and** (III.11)			
Initial Lifestyle	**Lifestyle Age 3**	**Lifestyle Age 5**	**Lifestyle Age 7**
Single parent family at birth	Currently single parent	Currently single parent	Currently single parent
Maternal education at birth	-	-	-
High family SES at birth	-	-	-
Low family SES at birth	-	-	-

Source: All variables are from or derived from the Millennium Cohort Study.

It is expected that children who have more highly educated mothers will experience a healthier family lifestyle than those with less well educated mothers. Marmot & Bell (2012) suggested that parental education had an influence on health-related behaviours and healthy lifestyles and that improving education could help to reduce health inequalities. In the data, maternal education remains relatively constant over the stages of childhood investigated in this chapter. Only 588 (6.9%) mothers in the sample gained additional qualifications during period included in the analysis of this chapter. Mothers might find it difficult to improve their education while bringing up small children. For this reason, the model allows maternal education to influence underlying family lifestyle in the initial period. Maternal education has a continued influence on family lifestyle throughout childhood due to the autoregressive (AR) nature of the dynamic process in the

model. Maternal education will be measured on a five point scale relating to NVQ levels or equivalent academic qualifications, as described in the previous empirical chapter.

Additionally, two binary variables indicating *high family SES* and *low family SES* are allowed to influence initial latent family lifestyle. Family SES is defined in the same way as in the previous empirical chapter and is the highest SES of the main or partner respondent in the MCS and the baseline category is *average SES*. Of the families included in the final sample, 3,041 (35.9%) experienced at least one change in SES during the periods analysed in this model. However, a large part of this variation in family SES was due to changes in family structure, where the family SES shifts from paternal SES to maternal SES due to a father, or father-figure, leaving the household. Similarly to maternal education, the family SES variables are allowed to directly influence initial family lifestyle. Any effect of SES on subsequent family lifestyle is picked up indirectly through its effects on initial family lifestyle due to the AR nature of the model.

The final independent variable predicting underlying family lifestyle is family structure. A binary variable indicating whether a family is currently a single-parent or two-parent family is allowed to influence underlying family lifestyle in each period of the model. It is assumed that single-parent families have additional time constraints which could make it more difficult to provide a healthy family lifestyle. Single parents might have less time to take their child to sporting events or less time to cook healthy meals and as a result of these time constraints, children could spend more time watching television or participating in sedentary activities. Allowing family structure to influence family lifestyle in all periods of the model allows the effects of changes in family structure on underlying family lifestyle and on specific lifestyle outcomes to be investigated. Of the families in the sample, 1,132 (13.4%) of them experienced at least one change in family structure between the birth of the cohort member and the interviews when the cohort member is seven years old.

Lifestyle is expected to be persistent and changes in underlying family lifestyle are rare. This suggests that family lifestyle essentially drives itself rather than being driven by individual independent variables. There is little evidence in the literature that suggests that tackling individual variables will change this underlying lifestyle but, as with any model, it is possible that there are other variables which might have a contemporaneous influence on underlying family lifestyle. However, these independent variables are not the main focus of this chapter and are only used in the simulations in order to condition

on different characteristics. Variables which might influence family lifestyle but have not been included in this model could be time-varying or time-invariant. Although it is likely that there are other independent variables which have an effect on family lifestyle, there is a trade-off between the number of additional variables that can be identified and allowing for time-varying parameters[60]. These additional independent variables are not the main interest in this chapter and so it is considered more important to allow all parameters to vary over time. However, any time-varying or time-invariant effects which are not included in the model are included in the error terms. The individual random effect $\boldsymbol{\eta}$, in Equations (III.10) and (III.11) accounts for any omitted time-invariant variables[61]. Time-varying influences on family lifestyle which are not included in the model are also accounted for through the time-varying error term $\boldsymbol{\varepsilon}_t$ in Equations (III.10) and (III.11). This accounts for any unexpected shocks to underlying family lifestyle. This includes potential shocks such as a family member being diagnosed with type II diabetes which could have an influence on the underlying lifestyle of a family. It is acknowledged that there are time-varying parameters which influence family lifestyle and are not included in this study. If relevant time-varying variables which are highly correlated over time and are therefore correlated with family lifestyle are excluded from the model, the persistence parameter α_t could be biased. This is because these omitted variables would create a correlation between the latent factor and the error term at time t. The direction of this bias will depend on whether the variable is positively or negatively correlated over time. The exclusion of these variable will not be as problematic as the exclusion of time invariant variables which have a correlation of one over time. Any variables which are highly correlated (close to time-invariant) would be accounted for in the individual random effect $\boldsymbol{\eta}$ discussed above. Some bias might remain from variables which are omitted and are more weakly correlated over time, but this bias is not expected to be high due to the weaker correlation.

In addition, the random family effect $\boldsymbol{\eta}$, in Equations (III.10) and (III.11) accounts for any omitted time invariant variables. The error terms in Equations (III.10) and (III.11) account for any omitted influences on family lifestyle. Although this means that the effects of some independent variables on family lifestyle are not estimated, these effects are not the main interest of this chapter and allowing parameters to change over time is considered more important.

[60] This refers to all time-varying parameters, not only the time-varying independent variables.
[61] This is later found to be insignificant and is removed from the final model.

In addition to the independent variables outlined above, another set of independent variables are included to influence the childhood adiposity variables in each period. These are represented by $\boldsymbol{W}_t$ in Equation (III.7). Table III-3 displays the independent variables used in the model to directly influence childhood adiposity. It also shows the periods in which each of these independent variables is included in the model.

Table III-3: Independent Determinants of Childhood Adiposity Measures

Determinants of Childhood Adiposity Measures (*W* in Equation (III.7))			
Weight During 1st Interview (kg)	**Weight Status Age 3**	**Weight Status Age 5**	**Weight Status Age 7**
Ethnicity	Ethnicity	Ethnicity	Ethnicity
Sex	-	-	-
Age at first interview (weeks)	-	-	-

Source: All variables are from or derived from the Millennium Cohort Study.

Ethnicity is indicated by the same set of binary variables used in the previous empirical chapter: '*black'*, '*Asian'* or '*other ethnicity'* and '*white*' is the baseline ethnicity. These ethnicity variables are allowed to directly influence childhood adiposity at each period in the model. This will allow any difference in lifestyle or lifestyle outcomes between ethnic groups to be identified, as well as the stage in childhood at which these differences occur. This could lead to important policy implications. NICE (2013a) guidance has already been published which investigates the differences in obesity and BMI between adults of different ethnic backgrounds but there is a lack of research in this area when it comes to childhood.

Age and sex are both allowed to influence child weight in the initial period. Boys and girls have different mean weights at this young age[62], as well as throughout childhood. However, in subsequent waves, age and sex specific thresholds are used to determine weight status[63]. It is possible that there is an additional effect of sex on the trajectories of weight status, over and above the differences in thresholds. However, it is assumed that these additional influence will be relatively small in comparison to the differences in weight and of less interest to policy makers than the differences between ethnic groups.

[62] Boys are expected to weigh more than girls, *ceteris paribus*.
[63] The IOTF thresholds used here are discussed in more detail in Section 1.2.1, but all measures of weight status in children are age and sex specific.

Relationships between Independent Variables

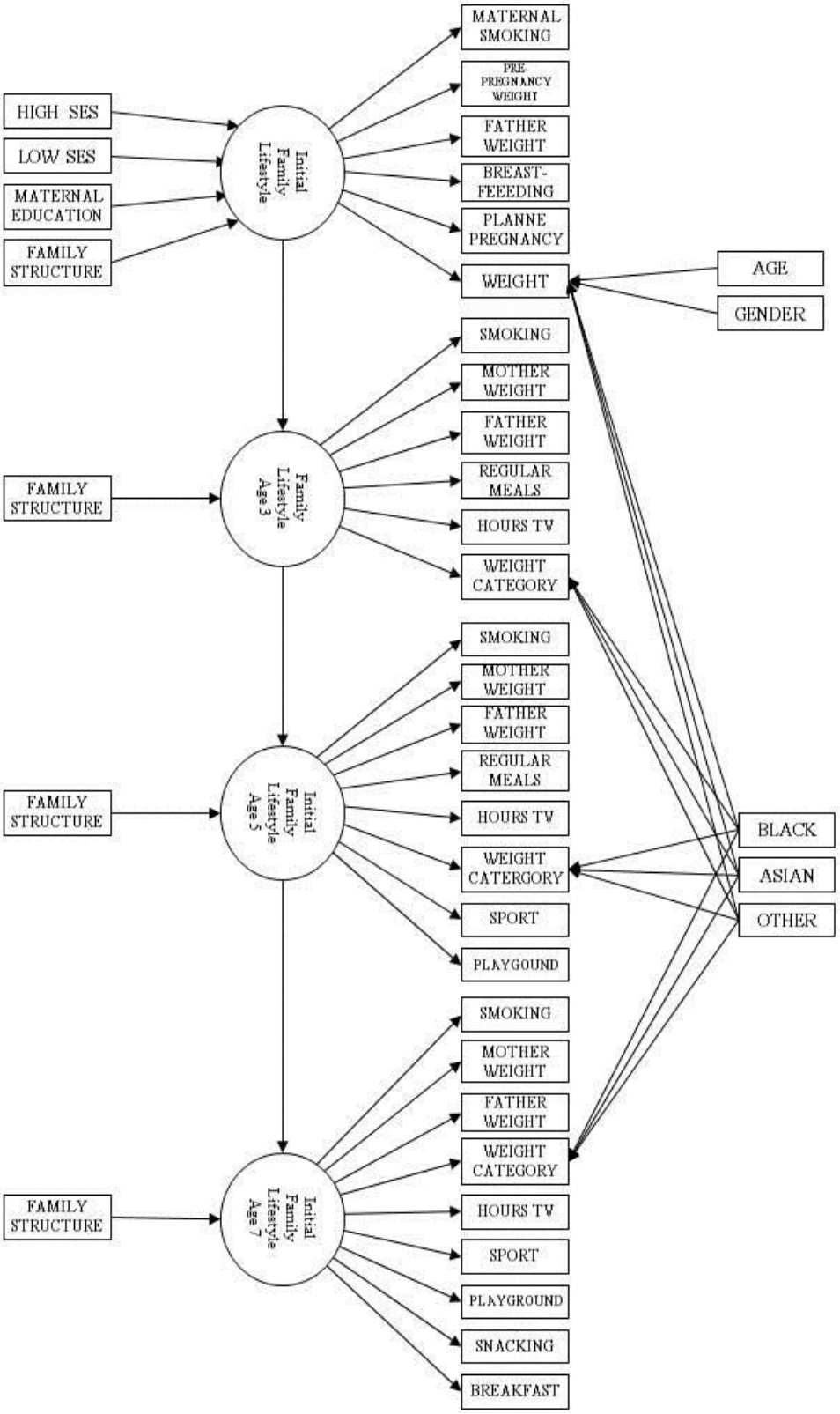
As discussed in Section 3.3.1 there are no independent variables which are included in both vector $\boldsymbol{X}$ and vector $\boldsymbol{W}$, even though in principle there could be. Age, sex and ethnicity of the child are not expected to have any direct influence on underlying family lifestyle. Any effect they have on childhood adiposity is assumed to be a direct effect. For example, there is a large amount of literature which suggests that ethnicity has a large influence on weight, both during childhood and throughout life. However, the literature suggests that these differences are largely biological rather than environmental. For this reason, and to avoid difficulties in identification, ethnicity is included only as an independent variable influencing childhood weight and not directly influencing underlying family lifestyle. Conversely, SES, maternal education and family structure are not expected to have a direct influence on child childhood adiposity. Any effect they do have on childhood adiposity is assumed to be through their influence on underlying family lifestyle. If any variable were to be included in both these variables then exclusion restrictions might be necessary. This is not an issue in this chapter.

Variables Amenable to Policy Change

This chapter estimates a complex dynamic factor model to be able to identify the unobserved variable, family lifestyle. This is the variable that most recent policy interventions aiming at reducing obesity are targeting nowadays. Interventions such as Change4Life aim to improve family lifestyle through changing attitudes and parental willingness to change. These are the types of interventions that are of policy interest in this chapter. However, the independent variables discussed here will help to inform policy makers because, using simulations it will be possible to condition on these variables in order to determine the characteristics of children who will benefit most from any family lifestyle interventions and make it possible to see the effects that potential family lifestyle policies might have on inequalities.

A visual representation of the model is displayed in Figure III-1. Latent factors are represented using oval shapes and observable variables are represented using rectangles. In line with standard path diagrams, the arrows between variables, both observed and latent, show the causal directions of any effects estimated by the model.

Figure III-1: Path Diagram



3.4.3 Missing and Excluded Observations

The methodology used in this chapter requires a balanced panel. This means that any observations which are missing in a single wave cannot be included in the analysis. Removing the 692 families which were not included in the first wave and those who were lost to follow up in later waves, results in a balanced sample with 11,484 families.

Again, twins and triplets are removed because these families are likely to have different patterns of behaviour to families with single children, especially when considering their initial conditions, for example, birth weight and breastfeeding behaviours. However, this only results in one further family being removed from the sample, leaving 11,483 observations. Any children weighing less than 2.5 kg at birth have been removed from the analysis because they are likely to have very serious health problems causing their families to have very different lifestyle from other families. This led to a further 699 children being removed, leaving a sample of 10,784 observations. A further 670 observations were removed from the sample because the child was taken to a special care unit (SCU) immediately after their birth. It is again expected that families with a child in such a condition will not experience a 'normal' lifestyle. This leaves a balanced sample of 10,114 observations. One benefit of using latent factors models is that observations are not dropped due to item-non-response in the outcome measures. The latent factors can still be estimated using any remaining outcome measures, provided that there are enough non-missing outcome measures for the same individual. In this analysis, no observations are removed as a result of missing outcome measures. This is a strength of the latent factor models and the majority of other methods would result in further observations being removed and could lead to further bias.

Observations are dropped from this analysis when independent variables included in vector $\boldsymbol{X}$ or vector $\boldsymbol{W}$ are missing. This includes variables predicting latent family lifestyle or childhood adiposity. In total 1,652 observations were dropped due to item-non-response in independent variables. These are SES (468 missing observations), maternal education (1,167 observations) and ethnicity (17 observations). This leaves a balanced panel consisting of 8,462 observations which are included in the model. The proportion of observations removed from analysis for this reason is relatively small and, as explained in the previous chapter, weighting to account for this item-non-response or for sample design or attrition makes little difference to the majority of studies (Hansen, 2012; Plewis, 2007). Missing data is also assumed to be missing at random.

In accordance with the WHO BIV values used in the previous chapter, children with biologically implausible height, weight or BMI and biologically implausible maternal or paternal BMIs are recorded as missing. However, due to the benefits of the model described above, this did not result in the removal of any observations. Although this methodology required a balanced panel meaning that attrition could have an influence on the observations included in the model. However, if attrition were a problem, then it would also be a problem for any other study using this data set and as discussed above attrition in the MCS has been shown to make little difference to results (Hansen, 2012; Plewis, 2007).

The summary statistics for each of the variables in the final sample used in the model are displayed in Table B-1 in Appendix B.

3.5 Results

Three different specifications of the final model were implemented. Initially, a model is estimated with constant parameters across all periods. In this model, all outcome measures $\boldsymbol{Y}$ which appear in more than one period of the model, had constant parameters, including factor loadings and threshold parameters. Independent variables influencing underlying family lifestyle or childhood adiposity and which appear in more than one period also had fixed parameters. Next, an unrestricted model was estimated, with a greater number of time-varying parameters. In this model, factor loadings, threshold parameters and independent variable coefficients were allowed to vary over time. In this unrestricted model, all parameters were freed apart from the AR component (α_t) and the factor loadings and threshold parameters ($\boldsymbol{\lambda}_{kt}$ and τ_{kt}^{j}, respectively) relating to maternal and paternal weight categories. These parameter estimates remained restricted over time due to empirical problems with convergence in the model which occurs when they were freed. This is likely due to empirical identification problems when so many parameters are allowed to be time-varying. This is not considered particularly restrictive because a standard AR model would restrict all parameters to be time-invariant. Additionally, this model only investigates the dynamic relationship over a small number of years and so it is reasonable to assume that some of the parameters may not change considerably, particularly variables which are likely to be more established. Because the AR process is restricted to be time-invariant, it is assumed that the dynamic evolution of family lifestyle is also already well established before a child is born.

A LR test was carried out to determine whether the unrestricted model was an improvement on the restricted model. Additionally, AIC and BIC values are presented in Table III-4 to compare the models for goodness of fit. The restricted model estimated sixty parameters with a log-likelihood of $-146{,}207$. The unrestricted model estimated ninety-two parameters with a log-likelihood of $-145{,}620$. The likelihood ratio test can then be performed, which produces an LR statistic $\Lambda = 1174$, following a χ^2-distribution with $32\ (92 - 60)$ degrees of freedom. The test statistic, Λ is higher than the critical value, χ^2_{32} of 46.194 with a p-value of less than 0.0001. The LR test provides strong evidence to reject the null hypothesis in favour of the alternative and the unrestricted model has a better fit. The AIC and BIC also suggest that the unrestricted model has a better fit, indicated by the lower values. This is in accordance with Cunha & Heckman (2008) who suggested that time-invariant parameters are not applicable when analysing data on children because they are constantly developing and changing. As a result of these model fit tests, the remainder of this section focuses on parameters from the unrestricted model.

Next, a further model was run which allowed the delayed response of child weight status to changes in underlying family lifestyle. As discussed in Section 3.3.1, this model assumes that family lifestyle which has already influenced other outcome measures including parental weight status, does not have an influence on child weight status until the next period. This could be thought of as counterintuitive because one would expect that adiposity would react faster in children than in adults and there is no theoretical reason why underlying family lifestyle should take longer to influence childhood adiposity than parental adiposity, for example. The output from this model is displayed in Table B-2 of Appendix B which shows the Mplus output for the estimated parameters under this specification[64]. The AIC and BIC of this model with a lagged effect on child weight, are also shown in Table III-4 along with its log-likelihood and degrees of freedom.

[64] A number of different specifications which had a lagged effect of underlying family lifestyle on child weight status were estimated. None of them fit the data as well as the original model for which the full set of results are displayed and discussed throughout this chapter. The model shown here is that which is as close to the final specification as possible; it includes the same number of parameters and the same dependent variables. This means that the degrees of freedom are the same and that it is more directly comparable to the final model. All additional models estimated assuming a lagged influence of underlying family lifestyle on child weight status produced very similar results.

Table III-4: Model Fit Statistics

	Restricted Model	Unrestricted Model	Unrestricted Model with lagged effect on child weight
Log-likelihood	-146,207.243	**-145,603.609**	-145,712.564
Degrees of Freedom (df)	60	**92**	92
AIC	292,534.486	**291,391.219**	291,609.129
BIC	292,957.087	**292,039.206**	292,257.116
N		8,462	

Notes: Data from Millennium Cohort Study. Bold represents the specification of choice which provides the best fit to the data.

The AIC and BIC are both lowest in the unrestricted model for which underlying family lifestyle influences contemporaneously influences child weight status suggesting that this model has a better fit than the others. This makes sense conceptually as well as empirically because if underlying family lifestyle has already influenced maternal and paternal weight status as well as other family lifestyle indicators then it is also likely to have already influenced child weight status. For this reason, it is the first model which is used throughout the remainder of this chapter.

The remainder of this section is structured as follows. Section 3.5.1 discusses the estimated parameters. Section 3.5.2 discusses the factor scores. Section 3.5.3 explores the predicted outcomes from the model using simulated data for children and families with different hypothetical characteristics.

3.5.1 Parameter Estimates

Table III-5 shows the estimated factor loadings for each of the underlying family lifestyle outcome measures in each period. These factor loadings represent the strength of association between the corresponding outcome measures and the underlying factor.

It is not possible to directly compare the factor loadings across time or across outcome measures because the scale of the factors are arbitrarily identified. However, the sign of the coefficients can still provide information. The quantifying of underlying family lifestyle will be discussed later.

Table III-5: Estimated Factor Loadings

	Factor Loading, **λ** (Equations (III.7)) (Standard Error)			
Dependent Variable	Initial	Age Three Years	Age Five Years	Age Seven Years
Weight (kg)	-0.051*** (0.007)	-	-	-
Weight Category	-	-1.205*** (0.079)	-1.535*** (0.080)	-1.518*** (0.078)
Maternal Weight Category¥	-8.527*** (0.321)	-12.574*** (0.418)	-12.574*** (0.418)	-12.574*** (0.418)
Father's Weight Category	-1.393*** (0.102)	-1.215*** (0.088)	-1.215*** (0.088)	-1.215*** (0.088)
Mother's Smoking Behaviour€	-0.739*** (0.105)	-0.757*** (0.101)	-0.697*** (0.092)	-0.643*** (0.083)
Planned Pregnancy	0.712*** (0.079)	-	-	-
Breastfeeding Behaviour	1.056*** (0.064)	-	-	-
Regular Meals	-	0.577*** (0.091)	0.648*** (0.090)	-
Over Three Hours TV per day	-	-0.867*** (0.076)	-0.545*** (0.070)	-0.431*** (0.062)
Sport	-	-	0.669*** (0.053)	0.561*** (0.047)
Playground/Park	-	-	0.154*** (0.057)	0.182*** (0.051)
Unhealthy Snacks	-	-	-	-0.290*** (0.056)
Regular Breakfast	-	-	-	0.553*** (0.082)
N	8,462	8,462	8,462	8,462

Notes: Data from Millennium Cohort Study. Parameter estimates taken from dynamic factor model, this table shows factor loadings from the factor models. * p < 0.1, ** p < 0.05, *** p < 0.01, ¥for initial conditions this is pre-pregnancy weight category, €for initial conditions this is smoking behaviour during pregnancy.

Each of the factor loadings is statistically significant and has the expected sign. Childhood weight status has a consistently negative response to positive changes in latent family lifestyle. Parental weight statuses are negatively associated with changes in latent family lifestyle. Maternal weight status is consistently the outcome measure with the largest factor loading, much larger than other factor loadings, suggesting that underlying family lifestyle is heavily associated with maternal adiposity. As discussed previously, the size of these factor loadings are not directly comparable with each other but a difference of this size shows some indication that maternal influences are strongly associated with underlying family lifestyle. The estimates for the thresholds for all ordinal lifestyle outcome measures can be found in the appendix in Table B-3. These parameters are important when calculating the predictions of the model but explain very little on their own.

Table III-6 shows the estimated coefficients of the independent variables in Equations (III.10) and (III.11) which influence latent family lifestyle, both in the initial period and in subsequent periods.

Table III-6: Estimated Coefficients in Predicting Latent Family Lifestyle

	Coefficient (scalar α and vector **β** Equations (III.10) and (III.11)) (Standard Error)			
Independent Variable	Initial Family Lifestyle	Family Lifestyle Age 3	Family Lifestyle Age 5	Family Lifestyle Age 7
		A		
Previous Latent Family Lifestyle	-	1.094*** (0.007)	1.094*** (0.007)	1.094*** (0.007)
		β		
Currently High SES	0.028*** (0.008)	-	-	-
Currently Low SES	-0.072*** (0.008)	-	-	-
Maternal Education at Birth	0.013*** (0.003)	-	-	-
Single Parent	-0.044*** (0.010)	-0.002 (0.007)	-0.003 (0.005)	-0.012** (0.005)
N	8,462	8,462	8,462	8,462

Notes: Data from Millennium Cohort Study. Parameter estimates taken from a dynamic factor model. This table shows the autoregressive parameter on lifestyle and the coefficients for independent variables directly influencing underlying family lifestyle. * p < 0.1, ** p < 0.05, *** p < 0.01.

. The AR coefficient, α, positive and statistically significant suggesting that underlying family lifestyle is persistent over time and that a family lifestyle at the time of a child's birth is likely to have a large influence on family lifestyle throughout their childhood. The determinants of family lifestyle in this model are consistent with the literature. Family SES, maternal education and being from a single-parent family each have a statistically significant effect on initial latent family lifestyle. Families with higher SES have healthier lifestyles in the initial period and those with a lower SES on average have a less healthy family lifestyle, *ceteris paribus*. Single-parent families appear to have less healthy lifestyles, *ceteris paribus*, across all periods. However, this effect is only significant in the initial conditions and when the child is seven years old.

Table III-7 shows the estimated coefficients of the independent variables which influence childhood adiposity measures. The parameter estimates given here represent coefficients in Equation (III.7).

Table III-7: Estimated Coefficients in Childhood Adiposity

	Coefficient ($\boldsymbol{\delta}$ Equations (III.7)) (Standard Error)			
Independent Variable	Weight at 9 Months (kg)	Weight Category Age 3	Weight Category Age 5	Weight Category Age 7
Male	0.066*** (0.003)	-	-	-
Age (weeks)	0.004*** (0.001)	-	-	-
Black	-0.010 (0.012)	0.186 (0.113)	0.352*** (0.103)	0.339*** (0.101)
Asian	-0.077*** (0.007)	-0.262*** (0.083)	-0.091 (0.080)	0.096 (0.076)
Other	-0.028*** (0.009)	-0.011 (0.092)	-0.041 (0.097)	0.058 (0.098)
N	8,462	8,462	8,462	8,462

Notes: Data from Millennium Cohort Study. Parameter estimates taken from a dynamic factor model. This table shows the coefficients of the independent variables directly influencing childhood adiposity. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table III-7 suggests that nine month old male children weigh more than their female counterparts with the same underlying lifestyle, *ceteris paribus*. As expected, children who are older are expected to weigh slightly more during the first MCS interview. Asian children are expected to weigh less than white children at nine months old, *ceteris paribus*. However, there is no significant difference between the expected weights of black and white children at nine months old. At the age of three, Asian children are still lighter on average but by the age of five and seven years there is no longer a significant difference in weight between white and Asian children. Conversely, by the age of five years, black children are significantly heavier than their white counterparts, *ceteris paribus*.

The standardized parameters for the factor loadings calculated using in Equation (III.13), are displayed in Table III-8. They show that underlying family lifestyle in every period is heavily associated with maternal weight status. As children grow up, their own weight status becomes a better indicator or measure of underlying family lifestyle, suggesting that childhood obesity is more dependent on family lifestyle as children get older. Interestingly, once these parameters are standardised, eating breakfast regularly does not have a significant relationship with underlying family lifestyle.

Table III-8: Standardised Factor Loadings

	Standardised Factor Loadings (Standard Error)			
Dependent Variable	Initial	Age Three Years	Age Five Years	Age Seven Years
Weight (kg)	-0.094*** (0.012)	-	-	-
Weight Category	-	-0.293*** (0.017)	-0.394*** (0.017)	-0.423*** (0.017)
Maternal Weight Category¥	-0.891*** (0.007)	-0.955*** (0.003)	-0.962*** (0.002)	-0.968*** (0.002)
Fathers' Weight Category	-0.306*** (0.020)	-0.296*** (0.019)	-0.323*** (0.020)	-0.351*** (0.021)
Mother's Smoking Behaviour	-0.166*** (0.024)	-0.186*** (0.025)	-0.188*** (0.025)	-0.191*** (0.025)
Planned Pregnancy	0.160*** (0.018)	-	-	-
Breastfeeding Behaviour	0.236*** (0.014)	-	-	-
Regular Meals	-	0.143*** (0.023)	0.176*** (0.024)	-
Over Three Hours TV per day	-	-0.214*** (0.018)	-0.150*** (0.019)	-0.131*** (0.019)
Sport	-	-	0.183*** (0.014)	0.168*** (0.014)
Playground/Park	-	-	0.043** (0.016)	0.056*** (0.016)
Unhealthy Snacks	-	-	-	-0.088*** (0.017)
Regular Breakfast	-	-	-	-0.011 (0.091)
N	8,462	8,462	8,462	8,462

Notes: Data from Millennium Cohort Study. Parameter estimates taken from a dynamic factor model. This table shows the standardised factor loadings for each of the measurement models. * p < 0.1, ** p < 0.05, *** p < 0.01.

The standardised parameters for the AR process of previous lifestyle on current lifestyle show that family lifestyle is very persistent and that if a child is born into a family with an unhealthy lifestyle, their lifestyle is likely to remain unhealthy. These are displayed in Table III-9.

Table III-9: Standardised AR Parameters

	Standardised AR Parameters (Standard Error)			
Independent Variable	Initial Family Lifestyle	Family Lifestyle Age 3	Family Lifestyle Age 5	Family Lifestyle Age 7
		α		
Previous Latent Family Lifestyle	-	0.992*** (0.001)	0.993*** (0.000)	0.994*** (0.001)
N	8,462	8,462	8,462	8,462

Notes: Data from Millennium Cohort Study. Parameter estimates taken from a dynamic factor model. This table shows the standardised autoregressive parameter estimate of lifestyle. * p < 0.1, ** p < 0.05, *** p < 0.01.

3.5.2 Factor Scores

The factor scores outlined in Section 3.3.2 provide numerical values for the lifestyles of each of these families. Although these factor scores have no measurable meaning and cannot be compared across time periods, they can be used to rank children in terms of their family lifestyle to determine where each family lies on a lifestyle distribution. Families with higher factor scores have 'healthier' lifestyle than families with lower factor scores.

The factor scores show that there is little movement by families across the lifestyle distribution. If families start off at the higher end of the lifestyle distribution they are likely to remain at the higher end. Similarly, if they start at the lower end of the distribution then they are also likely to remain at the lower end. This is in accordance with the persistence parameter in Table III-9.

Table III-10 shows the proportion of families remaining in certain parts of the lifestyle distribution throughout childhood. When a child is seven years of age, 87.43% of families which were above the ninety-fifth percentile on the lifestyle distribution in the initial period remain above the ninety-fifth percentile.

Table III-10: Proportion of Families Remaining in Initial Lifestyle Percentile Group

	Proportion Remaining in Percentile		
Initial percentile	3 Years	5 Years	7 Years
$\geq 95^{th}$	95.48%	91.27%	87.43%
$\geq 90^{th}$	95.94%	92.77%	88.96%
$< 10^{th}$	99.99%	99.99%	99.76%
$< 5^{th}$	>99.99%	>99.99%	99.99%
N	8,462	8,462	8,462

Notes: Data from Millennium Cohort Study. Results taken from the factor scores from a dynamic latent factor model.

However, families which are initially in the lowest five percentiles almost never improve their lifestyle; 99.99% of them remain in the lowest five percentiles when the child is seven years old. These figures suggest that there is more mobility at the upper end of the lifestyle distribution than at the lower end. That said, the amount of movement is still relatively low in all parts of the distribution. This is of great importance to policy makers aiming to improve the underlying lifestyles in families with the unhealthiest lifestyles because it suggests that those most in need of help to improve their lifestyle might be those most difficult to influence.

Table III-11 shows the proportion of families remaining in the upper and lower quartiles and the inter-quartile range of the lifestyle distribution throughout childhood.

Table III-11: Proportion of Families Remaining in Initial Lifestyle Percentile Group (2)

	Proportion Remaining in Percentile		
Initial percentile	3 Years	5 Years	7 Years
$\geq 75^{th}$	95.70%	93.84%	91.52%
Inter-quartile range	97.57%	96.46%	94.98%
$< 25^{th}$	>99.99%	>99.99%	>99.99%
N	8,462	8,462	8,462

Notes: Data from Millennium Cohort Study. Results taken from the factor scores from a dynamic latent factor model.

Families who are in the unhealthiest 25% of the lifestyle distribution when a child is born are likely to remain in this quartile as their child grows up, at least until the age of seven years. There is more movement at the upper end of the distribution when families are more likely to move down the distribution. Of the families which are in the inter-quartile range when their child is born, 5.02% improve their relative lifestyle and are in the upper quartile when their child is seven years old. In contrast, less than 0.01% move into the lower quartile. Again, this suggests that lifestyle is persistent and could be difficult to change using interventions, particularly in families with unhealthy lifestyles but also that successful interventions could have a big cumulative effect.

Table III-12 shows the correlations between the factor scores in each period of the model. The correlations between all factors scores are extremely high, giving further evidence of the persistence of family lifestyle and suggesting that policy makers might find it difficult to improve lifestyle.

Table III-12: Correlations between Factors Scores

Correlation	$\boldsymbol{\theta_0}$	$\boldsymbol{\theta_1}$	$\boldsymbol{\theta_2}$	$\boldsymbol{\theta_3}$
$\boldsymbol{\theta_0}$	1	-	-	-
$\boldsymbol{\theta_1}$	0.999	1	-	-
$\boldsymbol{\theta_2}$	0.997	0.999	1	-
$\boldsymbol{\theta_3}$	0.982	0.986	0.991	1
N	8,462	8,462	8,462	8,462

Notes: Data from Millennium Cohort Study. Correlations taken from the factor scores from a dynamic latent factor model.

Table III-13 shows some of the differences in characteristics between those ranked as having the 'healthiest' and 'unhealthiest' lifestyles. The table shows that children from families with the healthiest lifestyles weigh less at the age of nine months and are less often obese during childhood than those from families with the least healthy lifestyles.

Table III-13: Differences between High and Low Family Lifestyle Rankings

	Initial Family Lifestyle Ranking	
Variable	$\geq 95^{th}$ percentile	$< 5^{th}$ percentile
Percentage Male	49.58%	51.34%
Mean Weight (kg)	8.784	8.935
(standard deviation)	(1.444)	(1.513)
Percentage Obese Age 3	4.05%	6.01%
Percentage Obese Age 5	2.06%	6.44%
Percentage Obese Age 7	2.37%	8.37%
High SES	83.99%	0.42%
Low SES	0.14%	98.73%
N	8,462	8,462

Notes: Data from Millennium Cohort Study. Results taken from the factor scores from a dynamic latent factor model.

The difference between children at each end of this distribution appears to increase as children get older. Children from families with the unhealthiest lifestyles become more likely to be obese as they get older, suggesting that policies aiming to reduce childhood obesity should be targeted at families with unhealthy lifestyles in order to have the largest impact.

3.5.3 Predictions

As discussed in Section 3.3.3, simulations are used to approximate predictions from the model in order to avoid complex computations with multiple integrals. This section outlines the results from simulations using parameter estimates from the dynamic latent factor model to estimate the posterior distributions illustrated in Equation (III.18). For cases which use the entire sample to estimate expected outcomes, 250 simulations were used. In other cases, the model is used to simulate outcomes for hypothetical children with specific independent characteristics. In these cases, 10,000 simulations are used for each hypothetical child in order to obtain expected outcomes. The characteristics of these hypothetical children can be found in Table III-14.

Considering children and families from different backgrounds makes it possible to determine which families will benefit most from changes in their lifestyle. Policy makers are interested in reducing inequalities. Consequently, it is important to distinguish between children who grow up in advantaged or disadvantaged families, to determine which family background characteristics have most influence on underlying lifestyle and to explore how the influence of underlying family lifestyle on childhood weight status differs between children.

Table III-14: Independent Characteristics of Hypothetical Children

						Family Structure			
Child Number	**Description**	**Sex**	**Ethnicity**	**Maternal Education**	**Family SES**	**9 Months**	**3 Years**	**5 Years**	**7 Years**
1	white male	male	**white**	A-level	Average	couple	couple	couple	couple
2	white female	female	**white**	A-level	Average	couple	couple	couple	couple
3	Asian male	male	**Asian**	A-level	Average	couple	couple	couple	couple
4	Asian female	female	**Asian**	A-level	Average	couple	couple	couple	couple
5	black male	male	**black**	A-level	Average	couple	couple	couple	couple
6	black female	female	**black**	A-level	Average	couple	couple	couple	couple
7	other male	male	**other**	A-level	Average	couple	couple	couple	couple
8	other female	female	**other**	A-level	Average	couple	couple	couple	couple
9	two parents	male	white	A-level	Average	**couple**	**couple**	**couple**	**couple**
10	one parent	male	white	A-level	Average	**single**	**single**	**single**	**single**
11	two to one	male	white	A-level	Average	**couple**	**couple**	**single**	**single**
12	one to two	male	white	A-level	Average	**single**	**single**	**couple**	**couple**
13	one then fluctuating	male	white	A-level	Average	**single**	**couple**	**single**	**couple**
14	two then fluctuating	male	white	A-level	Average	**couple**	**single**	**couple**	**single**
15	advantaged female	female	white	**higher degree**	**High**	**couple**	**couple**	**couple**	**couple**
16	disadvantaged female	female	white	**compulsory only**	**Low**	**single**	**single**	**single**	**single**
17	high SES female	female	white	A-level	**High**	couple	couple	couple	couple
18	low SES female	female	white	A-level	**Low**	couple	couple	couple	couple

Notes: Variable from or derived from Millennium Cohort Study.

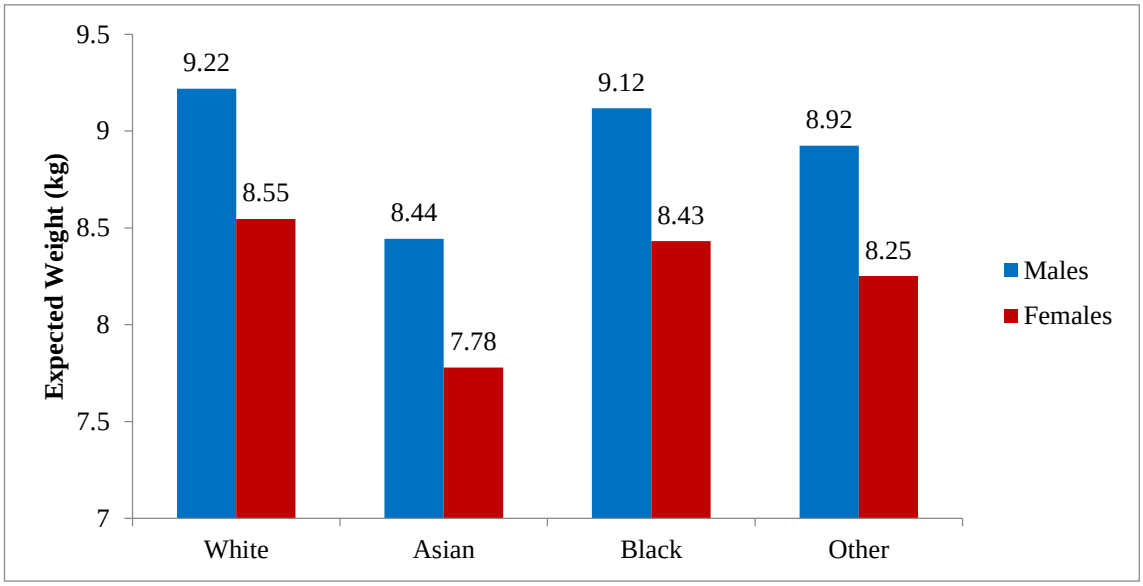
Simulations will be carried out using the parameters from the latent factor model described in Section 3.3. This section shows predictions using simulations which investigate the influence of underlying family lifestyle on child weight status in children from different ethnic social and family backgrounds. It then conditions on maternal lifestyle during and after pregnancy as well as diet and physical activity during early childhood. These simulations show just some of the policy relevant predictions that are made possible by the model outlined in Section 3.3.1. They show the expected place on the distributions of lifestyle and childhood adiposity of children with different characteristics and from different backgrounds. Due to the computational complexity in calculating these means, confidence intervals are not calculated here. However, the vast majority of parameter estimates from the final model used in these predictions were

significant and the literature suggests that even small differences in adiposity at a young age can lead to large inequalities in later life. Therefore it is reasonable to assume that even small differences in these predications are likely to be important.

Ethnicity

Differences in adiposity between ethnic groups have been known to exist for some time. However, it is not clear how, or when, these differences come about but is potentially due to biological or genetic differences. Figure III-2 shows the expected weight in kilograms at the age of nine months for eight hypothetical children (children 1-8 in Table III-14). Each of these children is from a two-parent family with average SES and had a mother educated to A-level or equivalent at their time of birth, but they differ by ethnicity.

Figure III-2: Expected Mean Weight (kg) at first MCS Interview by Ethnicity

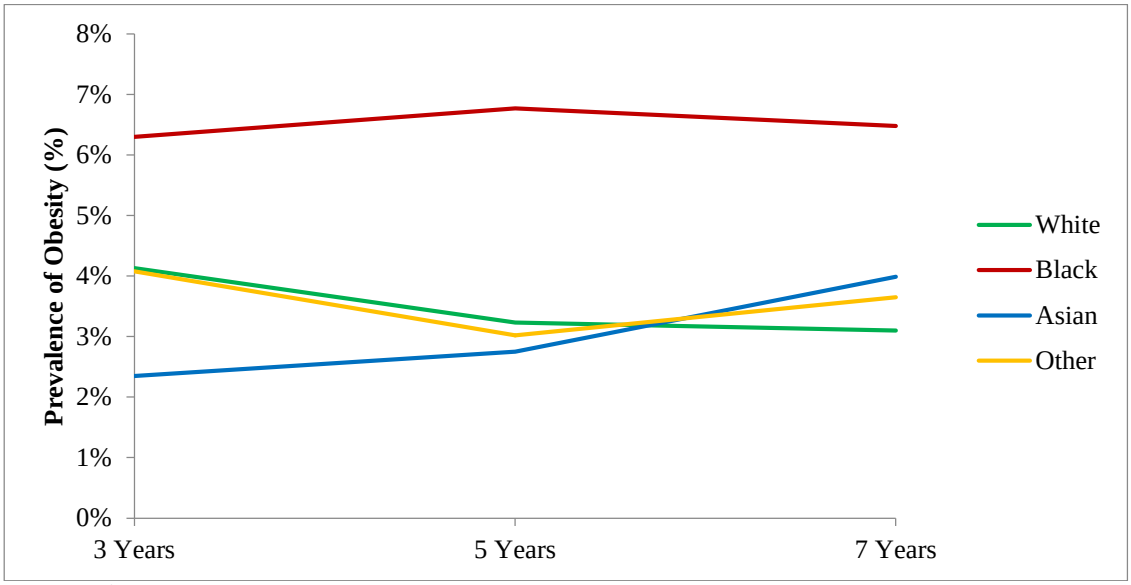


Notes: Data from Millennium Cohort Study.

As expected, males are heavier than females for children of all ethnicities. White children, on average, are the heaviest at nine months, followed closely by black children and then by children of other ethnicities. Asian children, both male and female are around half a kilogram lighter than their counterparts from the other ethnic groups. This is a substantial difference at this young age and is in line with existing literature. Although Asian children are, on average, lighter than children from other ethnicities at nine months, the dynamics of childhood adiposity could be different for children of different ethnicities and Asian children might not remain lighter than children of other ethnicities throughout childhood. It is important, therefore, to investigate the differences between childhood adiposity later in childhood.

Figure III-3 shows the likelihood of obesity in the four hypothetical boys from each ethnicity[65]. Using simulated data, the expected likelihood of obesity is higher in the black child than in the children of other ethnicities, a result which is consistent throughout early childhood. The Asian child has the lowest likelihood of obesity at the age of three years. However, his expected likelihood of obestiy increases with age at a steeper pace than the other children and by the age of seven he is more likely to be obese than the white child.

Figure III-3: Expected Prevalence of Obesity in Male Children by Ethnicity



Notes: Data from Millennium Cohort Study.

Table III-15 shows the expected percentile of the underlying family lifestyle distribution of the hypothetical boys shown in Figure III-3. The children from each of the ethnic groups are from families on very similar expected lifestyle percentiles. This suggests that, after taking into account family background characteristics, any differences in childhood adiposity between ethnic groups are a result of biological or developmental influences rather than difference in lifestyle.

Table III-15: Expected Lifestyle Percentiles by Ethnicity

	Lifestyle Percentiles			
	White	Black	Asain	Other
9 months	63.08	63.00	63.15	62.96
3 Years	61.81	61.79	61.95	61.75
5 Years	61.11	61.04	61.22	61.03
7 Years	60.61	60.54	60.65	60.54
N		8,462		

Notes: Data from Millennium Cohort Study. Results taken from the factor scores from a dynamic latent factor model.

[65] Similar results are found in girls.

The distributions of underlying family lifestyle ($\boldsymbol{\theta}$) at the age of seven are displayed in Figure B-1 in Appendix B. In line with the table above, they show little difference in the average family lifestyle or in the standard deviation of family lifestyle between children from different ethnicities.

NICE (2013) produced guidance suggesting that black and Asian adults are at increased risk of a range of health conditions and mortality at a lower BMI than their white counterparts. Although this guidance does not specifically refer to children, the increased risk of obesity in black children could potentially be problematic. If health risks occur in black children at a lower BMI than children of other ethnicities in the same way as they do in adults, then an even higher proportion of black children could be at risk in relation to their health than those shown by these results. For this reason, black children could be an important group for policy makers aiming to address childhood obesity. However, if it is not differences in underlying family lifestyle which are causing the different obesity prevalence between ethnic groups then further research might be need in order to determine how best to reduce these differences. Similarly, if the likelihood of obesity in Asian children continues to increase into later childhood this could be an important indication to policy makers that interventions should also be targeted at Asian children as well as black children. Any interventions aimed at Asian children should be implemented at a very young age, in order to have the greatest impact, before their risk of obesity increases.

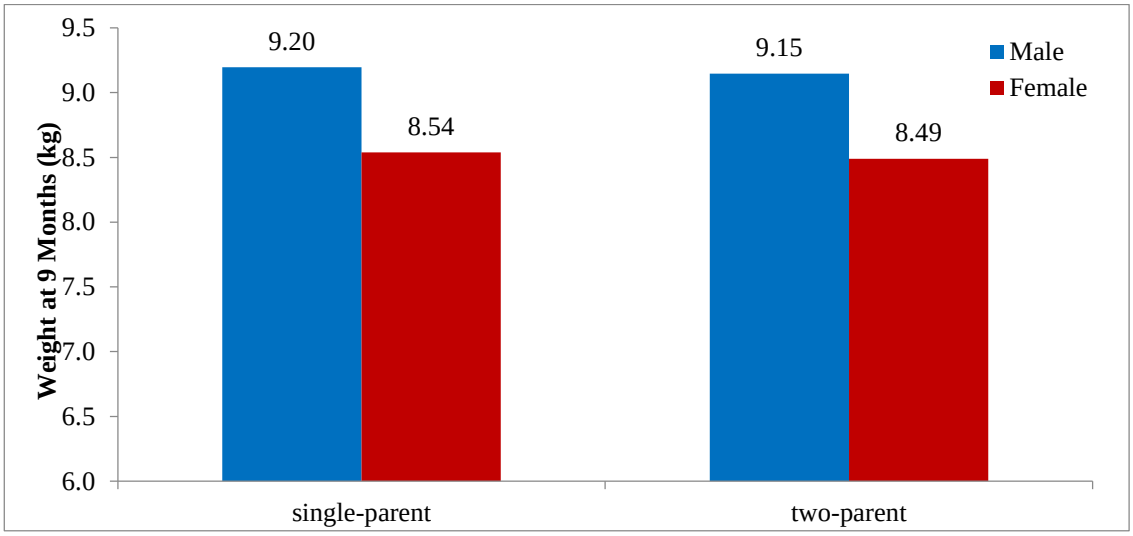
Family Background

As discussed previously, family structure could influence underlying family lifestyle due to the amount of available time that parents can spend providing their child with healthier lifestyles. Single parent families could inadvertently have unhealthier lifestyles because they might have less time to cook healthy meals, or to take their children to the park or sporting events. Moreover, children from single parent households are often breastfed less, spend more time in front of the television or have fewer regular meals. For example, Cunha & Heckman (2009) found that two-parent families were able to invest more into their children and suggest that this might be due to time preferences or the availability of resources. These time constraints could affect many of the family lifestyle outcomes used within the dynamic factor model in this chapter.

Figure III-4 shows the expected weight in kilograms at nine months of age for children in the sample who were in single-parent and two-parent families in the first wave. The

graph suggests that there is little difference between the mean weights of children with different family structure at this early age. Unlike the effects of ethnicity on childhood adiposity, the effects of family structure are related to underlying family lifestyle. The mean lifestyle of a single-parent family is on the 24.01th percentile and the mean for a two-parent family is on the 53.47th percentile. This is a large difference in relative lifestyle which translates only to a small difference in expected weight at nine months.

Figure III-4: Expected Weight at Nine Months by Family Structure

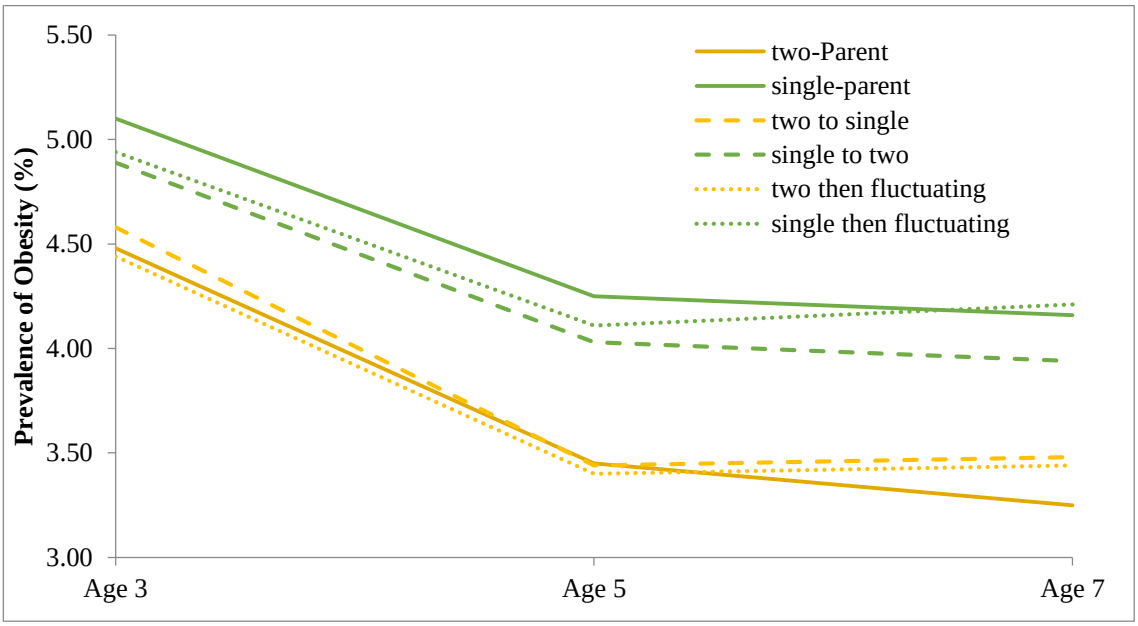


Notes: Data from Millennium Cohort Study.

However, because family lifestyle is persistent and the standard deviation of childhood BMI also increases as children get older, the effects on childhood adiposity could accumulate and create larger differences in later childhood.

It is also possible that changes in family structure during childhood could influence childhood weight status through its influences on family lifestyle. Figure III-5 shows the dynamics of obesity likelihood in hypothetical children from differing family structures.

Figure III-5: Expected Prevalence of Obesity by Family Structure (2)



Notes: Data from Millennium Cohort Study.

Each of these hypothetical children are white males from families with middle SES and have mothers with A-level education or equivalent. The children shown in this figure vary only by family structure. The figure includes a 'consistently two-parent' family, a 'consistently single-parent' family, a family which 'change from two-parent to single-parent' and two families with fluctuating structure. The latter two children are from families which change in structure more than once during the first four interviews and start life in a single-parent or two-parent family (children 13 and 14, respectively).

The simulations indicate that the expected likelihood of obesity is consistently higher at three years of age. This could be because obesity in three year olds is over-diagnosed using the available definitions. Children from families which are consistently two-parent families are persistently at a lower risk of obesity than those from consistently single-parent families. The difference in the prevalence of obesity between these two groups of children also appears to increase as children get older. There is also a difference in the prevalence of obesity between families who change from a single-parent to a two-parent family and *vice versa*. Children from families which become two-parent families after being a single-parent family have a decreasing likelihood of obesity as they get older. However, in children from families which change from two-parent families to single parent families, the obesity prevalence appears to increase between the ages of five and seven years. In line with (Crosnoe, 2012), this suggests that relationship breakdowns in early childhood increase the likelihood that a child will become obese, possibly due to

emotional health. Changes from single-parent families to two-parent families do not appear to produce the same increase in obesity prevalence.

There are two distinct groups which can be identified by examining this figure, children who start life in a single-parent family and children who start life in a two-parent family. This suggests that family structure is most important at the start of life when single-parents might struggle finding time to provide a healthy lifestyle for their family. The fact that family structure at the very start of life appears to have the most influence on obesity prevalence, along with the model parameters described at the beginning of this section, suggests that the AR process has a much stronger effect than subsequent social or family influences. Family structure at the start of childhood has a continued effect throughout childhood due to the persistent nature of family lifestyle and subsequent family structure has a relatively modest influence on childhood adiposity in comparison.

Table III-16: Expected Lifestyle Percentiles by Family Structure

	Lifestyle Percentiles					
	Two-parent	Single-parent	two- to single-parent	single- to two-parent	Two then fluctuate	Single then fluctuate
3 Years	61.67	40.47	61.23	40.85	60.93	41.08
5 Years	60.97	38.44	59.66	39.71	59.61	39.79
7 Years	60.53	34.06	54.87	39.23	57.90	36.26
N	8,462					

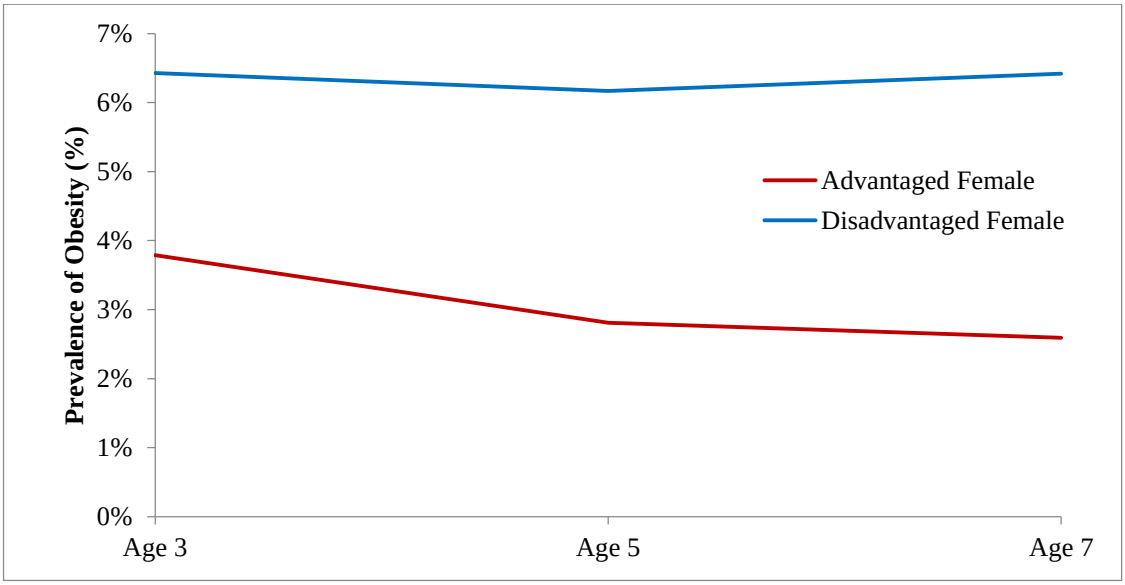
Notes: Data from Millennium Cohort Study. Results taken from the factor scores from a dynamic latent factor model.

Table III-16 shows that a typical two-parent family will remain roughly in the same place in the distribution whereas a typical single-parent family will keep falling further down the distribution. For this reason, the gap between the lifestyles of two-parent and single-parent families increases as children get older. It appears that family structure influences childhood weight through its influence on lifestyle. It also suggests that relative family lifestyle suffers when families split up. Together with Figure III-5 these results suggest that although changes in family structure throughout childhood can influence on childhood adiposity and lifestyle, family structure during the very early years appear to be very important and could have a large and long-lasting effect on family lifestyle and as a result the likelihood of childhood obesity. Figure B-2 in Appendix B shows the kernel densities for the distributions of underlying family lifestyle ($\boldsymbol{\theta}$) at the age of seven years for each hypothetical child represented in Figure III-5. The distributions show that children who start life in a two-parent family have a healthier expected lifestyle, regardless of subsequence family structure, again suggesting that family structure during the earlier years of childhood are extremely important. Providing more support to single-

parents to improve their lifestyle through interventions which enable them to make healthier lifestyle choices within their time constraints, including reducing their own BMI, could help to reduce this inequality. The effects of maternal education and family SES at the start of life on childhood obesity prevalence and underlying family lifestyle throughout childhood are similar to those shown here for family structure.

As well as conditioning on these social variables in isolation, the effects of multidimensional measures of childhood advantage or disadvantage are now explored. Children 15 and 16 in Table III-14 show the independent characteristics of two hypothetical children: an advantaged and a disadvantaged child. By looking at the different adiposity outcomes of these hypothetical children, it is possible to identify the collective impact that these independent variables have on childhood adiposity outcomes through their effects on family lifestyle. The advantaged child starts life in a two-parent family with high SES and has a highly educated mother. Conversely, the disadvantaged child starts life in a single-parent family with low SES and has a mother with a low level of education. Both children are white females.

Figure III-6: Expected Prevalence of Obesity for Advantaged and Disadvantaged Child



Notes: Data from Millennium Cohort Study.

Figure III-6 shows the prevalence of obesity in these advantaged and disadvantaged girls at ages three, five and seven years. In line with prior expectations, advantaged children have a lower risk of obesity than disadvantaged children, an observation which is consistent over time. The difference in obesity prevalence is already noticeable by the age of three years when children from the most disadvantaged backgrounds are around 50% more likely to be obese than those from the most advantaged backgrounds. The

differences in obesity prevalence between the advantaged and disadvantaged child increases as they get older suggesting that these differences could further increase by later childhood and adulthood. The simulations suggest that policy makers should therefore target interventions at disadvantaged children and families in order to reduce these inequalities in obesity prevalence. The differences in childhood adiposity seen between advantaged and disadvantaged children are much larger than those between children who differ only by one of the independent social variables used in the model. Consequently, policy makers should focus on a range of social determinants when targeting their interventions at certain children in order to help those most at risk of obesity through improvements to underlying family lifestyle.

Table III-17 shows the expected percentile of underlying family lifestyle for the advantaged and the disadvantaged child represented in Figure III-6, at the age of seven years. The figure shows a very large difference in the relative underlying family lifestyle of children from different backgrounds. Moreover, the kernel density distributions in Figure B-3 in Appendix B show that there is very little overlap in the distributions of family lifestyle in advantaged and disadvantaged children. This suggests that the family background characteristics, SES, maternal education and family structure, are good at identifying groups that policy makers might want to target. Again, the standard deviations of these distributions are very similar. Unlike the differences in obesity prevalence between ethnic groups, the differences seen here between advantaged and disadvantages children could be reduced if underlying family lifestyle in disadvantaged children is improved.

Table III-17: Expected Lifestyle Percentiles in Advantaged and Disadvantaged Children

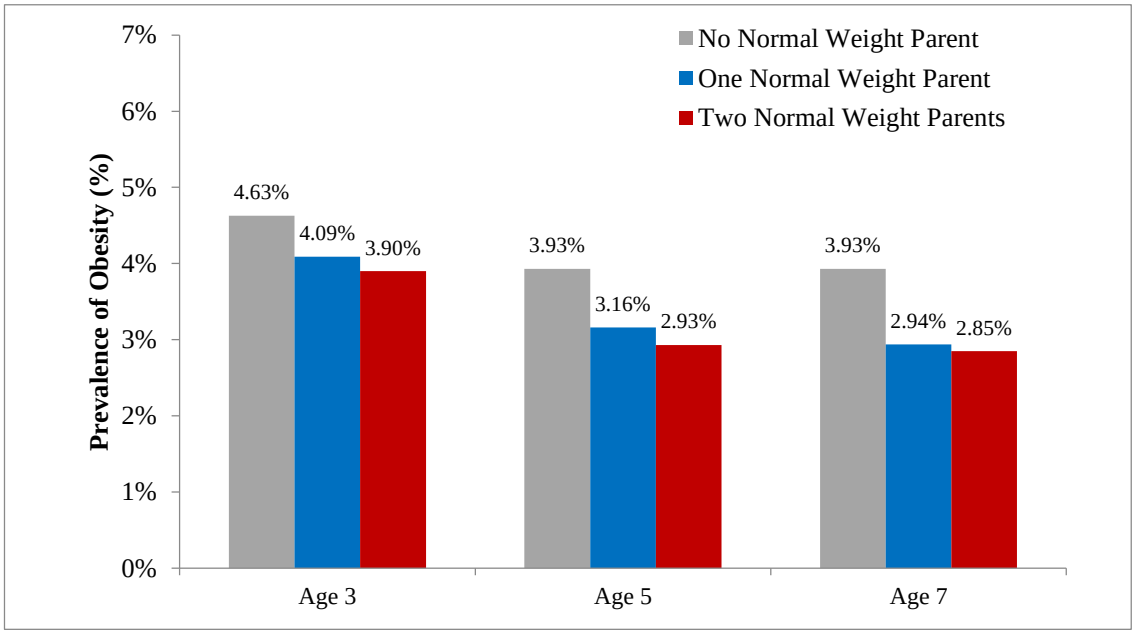
	Lifestyle Percentiles	
	Advantaged	Disadvantaged
9 months	85.63	7.50
3 Years	84.96	6.86
5 Years	84.59	6.29
7 Years	84.39	4.97
N	8,462	

Notes: Data from Millennium Cohort Study. Results taken from the factor scores from a dynamic latent factor model.

This suggests that the differences which are observed in obesity prevalence between advantaged and disadvantaged children are largely due to differences in the underlying lifestyle of their families. This further emphasises the importance of targeting children from disadvantaged backgrounds when aiming to reduce inequalities in obesity prevalence through the use of lifestyle interventions.

The association between the obesity of family members is now considered. Obesity in family members has been found to be highly correlated (Brown *et al.*, 2013; Brown & Roberts, 2013). If children learn their underlying lifestyle from their parents then this is likely to be true from a very young age. Figure III-7 and Figure III-8 show the expected obesity likelihood for two hypothetical children, both white females from two-parent families with mothers educated to A-level or equivalent. They are from families with high and low SES, respectively (children 17 and 18 in Table III-14). The figures show the expected likelihood of obesity in these children conditional on parental obesity. If an association exists between parental and child obesity, as it does in the literature, then parental obesity could be used as an outcome measure to identify children and families in need of support in relation to their underlying lifestyle.

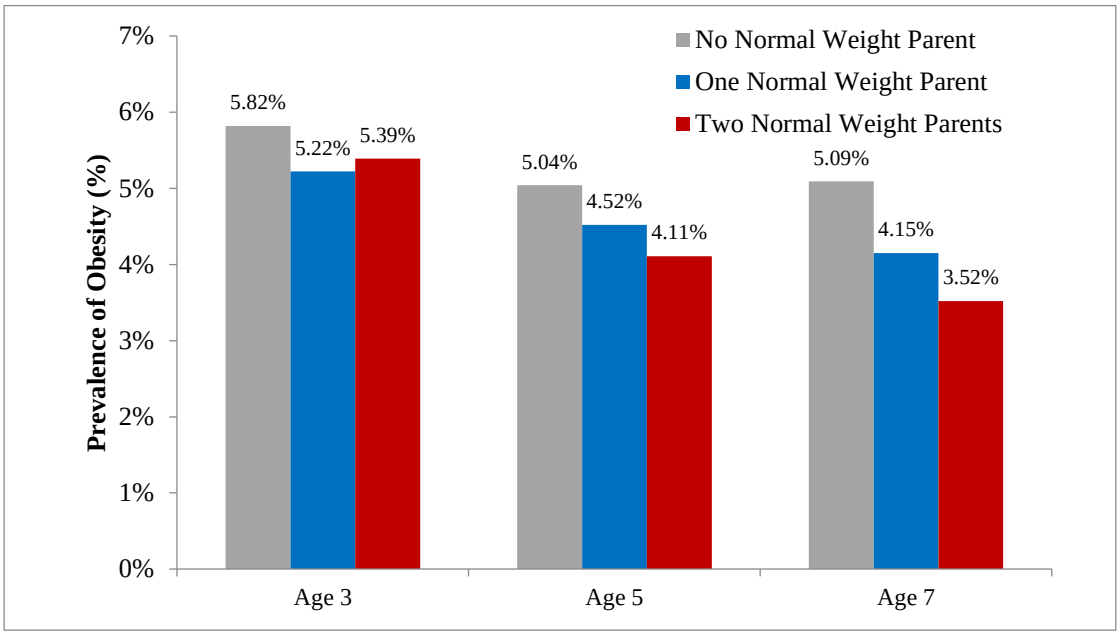
Figure III-7: Expected Childhood Obesity and Parental Weight Status (High SES)



Notes: Data from Millennium Cohort Study.

Figure III-7 shows that children from families with high SES are more likely to be obese if both of their parents are overweight or obese than if they have normal weight parents. There is a smaller difference between the expected obesity prevalence in children with one and two normal weight parents; the marginal effect of a second normal weight parent is much smaller.

Figure III-8: Expected Childhood Obesity and Parental Weight Status (Low SES)



Notes: Data from Millennium Cohort Study.

Figure III-8 shows that the child from the family with low SES is generally more likely to be obese than her counterpart from the family with high SES, regardless of parental weight status.

Again, the child from the family with low SES is more likely to be obese if both her parents are overweight or obese. Interestingly, at the age of three years, the child from a family with low SES is more likely to be obese if both her parents are a normal weight than if only one of her parents is a normal weight. This outlying result is not found in males but suggests that the differences in childhood obesity prevalence which result from family lifestyle that are apparent later in childhood are not always established at this young age. Aside from this, the results were similar for male children and for children from other ethnic groups. It is worth remembering that both Figure III-7 and Figure III-8 show the expected results for children from two-parent families. Children from single-parent families with no normal weight parents or only one normal weight parent could have different outcomes to those shown above.

Table III-18 and Table III-19 show the expected percentiles of family lifestyle for the children from families with high and low SES, respectively, conditional on parental obesity.

Table III-18: Expected Lifestyle Percentiles by Parental Weight in High SES

	Lifestyle Percentiles		
	Two Normal Weight Parents	One Normal Weight Parent	No Normal Weight Parent
3 Years	78.48	75.00	51.41
5 Years	78.27	75.19	59.64
7 Years	77.88	74.65	47.60
N		8,462	

Notes: Data from Millennium Cohort Study. Results taken from the factor scores from a dynamic latent factor model.

In both children, having at least one normal weight parent is associated with having a relatively healthier lifestyle. In line with the findings from the previous simulations, there is also a large difference between the lifestyle percentiles of children from families with high and low SES.

Table III-19: Expected Lifestyle Percentiles by Parental Weight in Low SES

	Lifestyle Percentiles		
	Two Normal Weight Parents	One Normal Weight Parent	No Normal Weight Parent
3 Years	39.49	32.58	19.52
5 Years	40.05	32.87	18.97
7 Years	40.54	33.18	18.44
N		8,462	

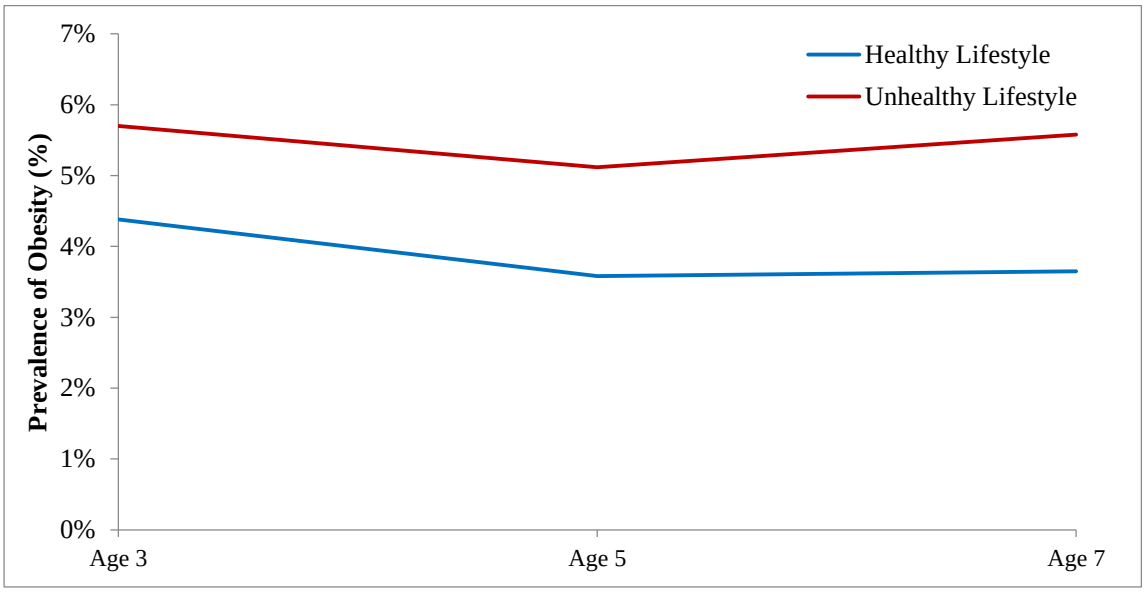
Notes: Data from Millennium Cohort Study. Results taken from the factor scores from a dynamic latent factor model.

These results are also reflected in the kernel density distributions of underlying family lifestyle displayed in Figure B-4 in Appendix B. The simulated distributions each have a similar variance but family lifestyle is expected to be healthier in children with a higher number of normal weight parents and those from families with higher SES. These results suggest that policy makers should focus their policies on mechanisms which influence an entire family rather than those specific to one member of a family.

Maternal Lifestyle

Maternal choices during and around the time of pregnancy could also influence obesity throughout childhood. Figure III-9 shows the expected prevalence of obesity in children at ages three, five and seven years, whose mothers had a healthy lifestyle during pregnancy and those whose mothers had an unhealthy lifestyle. Mothers who planned their pregnancy, did not smoke during pregnancy and were not overweight or obese immediately before their pregnancy are considered to have had a healthy lifestyle during pregnancy. Those who smoked throughout their pregnancy and were overweight or obese immediately before their pregnancy were considered to have an unhealthy lifestyle during pregnancy. Figure III-9 shows this relationship estimated using the entire sample, where other characteristics are at their sample value.

Figure III-9: Expected Maternal Lifestyle and Childhood Obesity



Notes: Data from Millennium Cohort Study.

Children whose mothers had a healthy lifestyle during pregnancy are less likely to be obese than those whose mothers had unhealthy lifestyle. These differences in obesity prevalence also appear to increase as children get older, similar to the differences resulting from family structure. Again, there is a large difference in the percentiles of family lifestyle for these two groups of children.

Table III-20 shows the mean percentiles of family lifestyle for children born after healthy and unhealthy pregnancies. It shows that there is a large difference in relative lifestyle between families whose mothers had healthy and unhealthy lifestyles during pregnancy. Figure B-5 shows the kernel density distributions for underlying family lifestyle at seven years of age in children whose mothers had a healthy and unhealthy lifestyle during pregnancy. The distributions appear to be skewed in opposite directions and those from healthy pregnancies are expected to have a relatively healthier underlying family lifestyle.

Table III-20: Expected Lifestyle Percentiles by Lifestyle during Pregnancy

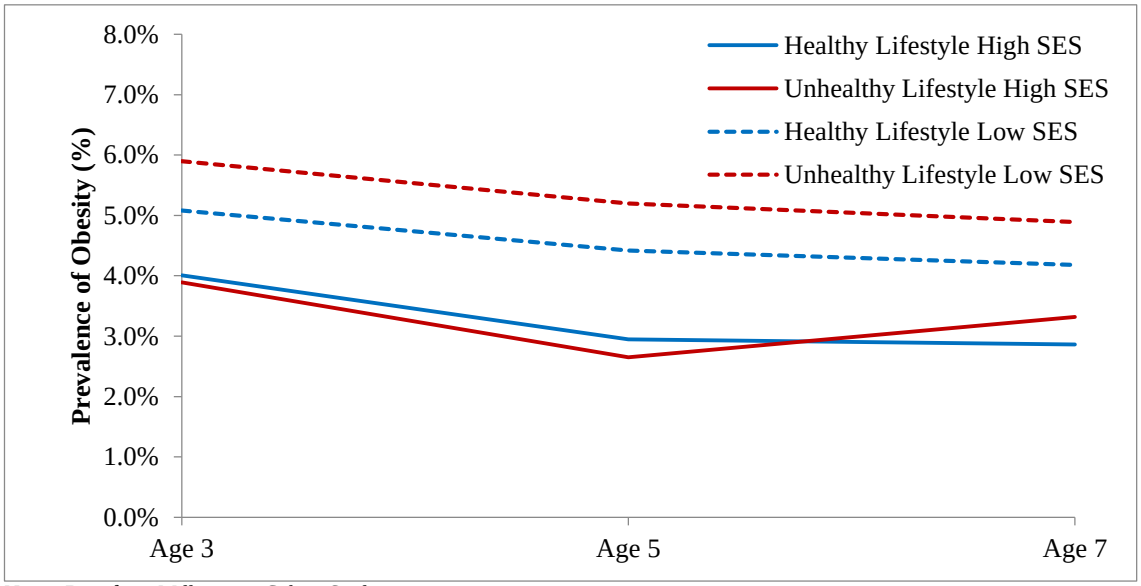
	Lifestyle Percentiles	
	Healthy Pregnancy	Unhealthy Pregnancy
3 Years	56.57	19.66
5 Years	56.13	19.46
7 Years	55.57	19.08
N	8,462	

Notes: Data from Millennium Cohort Study. Results taken from the factor scores from a dynamic latent factor model.

Previous studies have found that this relationship is attenuated for by confounding factors. For example, Currie (2011) found that SES and maternal lifestyle were highly correlated suggesting that SES could confound this relationship. Figure III-10 shows the expected likelihood of obesity in children by maternal lifestyle in children from families with high

SES and low SES. These figures use independent characteristics for two hypothetical children (children 17 and 18 in Table III-14) who are white females from two-parent families and have mothers with A-level education, but whose families differ in SES. In accordance with Currie (2011), the association between maternal lifestyle and childhood obesity appears to be different in children from families with high and low SES. In children from families with low SES, an unhealthy maternal lifestyle is consistently associated with a higher likelihood of childhood obesity. For children from families with high SES, a healthy maternal lifestyle during pregnancy appears to have little correlation with childhood obesity prevalence, and even appears to be associated with an increased risk of childhood obesity in three and five year olds. By the age of seven, there is very little difference between the expected prevalence of childhood obesity in children with mothers who had healthy or unhealthy lifestyles during pregnancy.

Figure III-10: Expected Maternal Lifestyle and Childhood Obesity (2)



Notes: Data from Millennium Cohort Study.

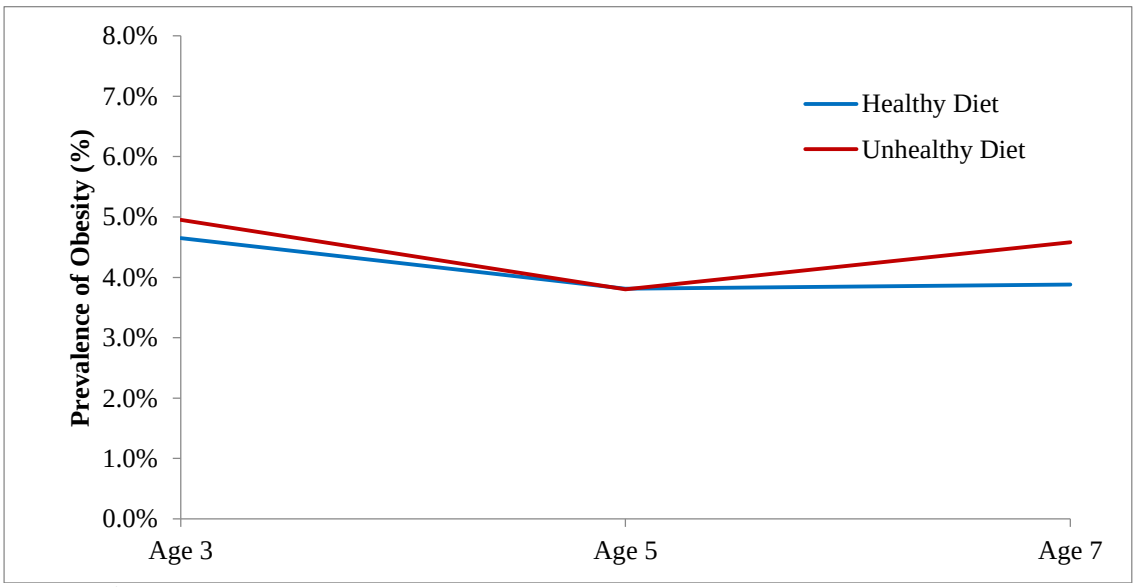
Figure III-10 shows that the effects of lifestyle during pregnancy on childhood weight status are much larger in children from families with low SES. In these families, children whose mothers had a healthy lifestyle during pregnancy had a reduced risk of obesity throughout early childhood. However, children from families with high SES did not see the same reduction in obesity risk associated with a healthy maternal lifestyle during pregnant. This could be important for policy makers who are aiming to reduce inequalities during childhood or provide better outcomes for children from low socioeconomic backgrounds. Again, results are found to be similar in male children and children from different ethnic groups. Similar analysis to this was carried out looking only at differences between mothers that smoked during pregnancy and those who did

not. A large difference in underlying family lifestyle was found, however, this relationship did not translate into differences in the likelihood of obesity during childhood. This suggests that it is maternal weight status before pregnancy is more strongly correlated with childhood weight status than maternal smoking behaviour.

Diet and Physical Activity

Children's eating habits could be associated with their likelihood of obesity as they grow up. Figure III-11 shows the expected likelihood of obesity throughout early childhood for two types of children, using data from the full simulated sample. They differ in that one group of children had a 'good diet' (they had regular meal times at ages three and five, ate breakfast regularly and did not eat unhealthy snacks between meals). The second group had a 'bad diet' (they ate unhealthy snacks and did not have regular meal times). Figure III-11 shows the expected prevalence of obesity throughout early childhood in children with healthy and unhealthy diets.

Figure III-11: Expected Diet and Childhood Obesity



Notes: Data from Millennium Cohort Study.

There appears to be little difference in the likelihood of obesity associated with diet at any stage of childhood. However, by the age of seven years, the difference between the two groups does appear to start increasing. If this increase continues then diet could be associated with childhood obesity during later childhood.

Table III-21 shows that there is some difference in the underlying family lifestyle between children with healthy and children with unhealthy diets. Even if diet is not heavily

associated with weight at this young age, having a healthy diet during childhood is associated with a healthy underlying lifestyle and should be encouraged regardless of its relationship with childhood obesity. The influence of underlying family lifestyle on diet is expected to remain throughout childhood and is expected to get stronger as children grow older.

Table III-21: Expected Lifestyle Percentiles by Diet

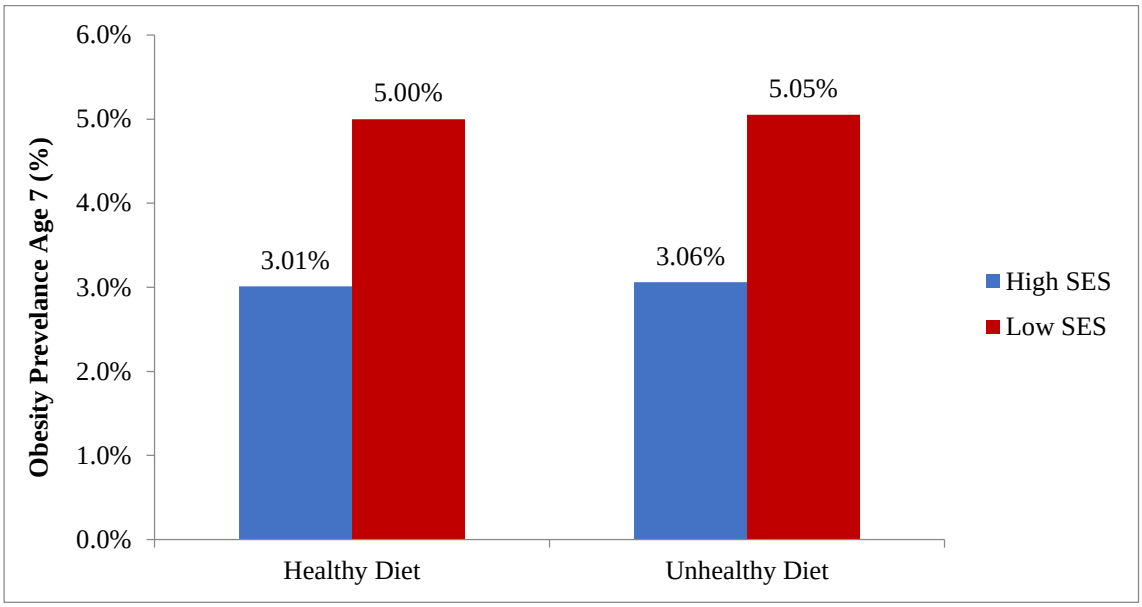
	Lifestyle Percentiles	
	Healthy Diet	Unhealthy Diet
3 Years	51.80	42.63
5 Years	51.09	42.31
7 Years	49.95	41.29
N	8,462	

Notes: Data from Millennium Cohort Study. Results taken from the factor scores from a dynamic latent factor model.

Figure B-6 in Appendix B shows the kernel density distributions for underlying family lifestyle in seven year old children with healthy and unhealthy diets. In line with Table III-21, there is a small difference in the expected family lifestyle between the two groups. However, Figure B-6 in Appendix B also shows that the children with a healthy diet have a much wider spread in underlying family lifestyle than those with an unhealthy diet.

Figure III-12 shows the relationship between diet and childhood obesity in seven year old children from families with high and low SES. The association between obesity risk and diet are much smaller than the influence of SES on obesity through the effects of lifestyle. Diet does not appear to have much association with obesity risk at all. However, looking back at Figure III-11 it could be that the differences in obesity prevalence between those with healthy and unhealthy diets get larger as children get older.

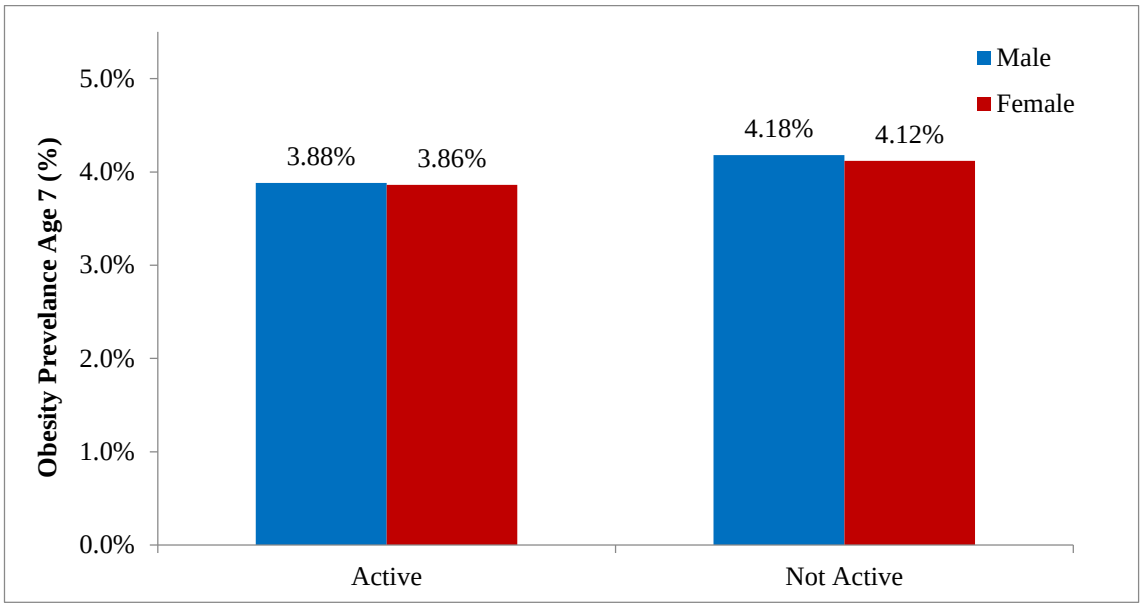
Figure III-12: Expected Diet and Childhood Obesity by SES at 7 Years



Notes: Data from Millennium Cohort Study.

As well as diet, the amount of physical activity that a child participates is expected to be associated with their adiposity. By the age of seven years, the amount of physical activity that a child engages with can vary widely between children. However, in this sample, physical activity does not have a strong association with childhood obesity prevalence. An active child is considered to be one who plays sport at least once a week, who regularly visits the playground and who does not spend more than three hours a day watching TV or playing computer games. An inactive child is one who never plays sport, does not visit the playground or park and who watches TV or plays computer games for three or more hours each day. Figure III-13 shows the expected prevalence of obesity for children who are active and inactive, by sex. The figure shows a small difference in the prevalence of obesity between males and females at the age of seven years. The small association between physical activity and childhood obesity appears in both boys and girls.

Figure III-13: Expected Physical Activity and Childhood Obesity at 7 Years



Notes: Data from Millennium Cohort Study.

Differences in underlying family lifestyle between active and inactive children are displayed in Table III-22 and are similar to differences between children with healthy and unhealthy diets. The differences in obesity prevalence and underlying family lifestyle between active and inactive children appear to be slightly stronger in boys than in girls, suggesting that boys might benefit more from interventions encouraging physical activity.

Table III-22: Expected Lifestyle Percentiles at Seven Years by Physical Activity

	Lifestyle Percentiles	
	Active	Inactive
Male	50.38	42.86
Female	50.28	43.29
N	8,462	

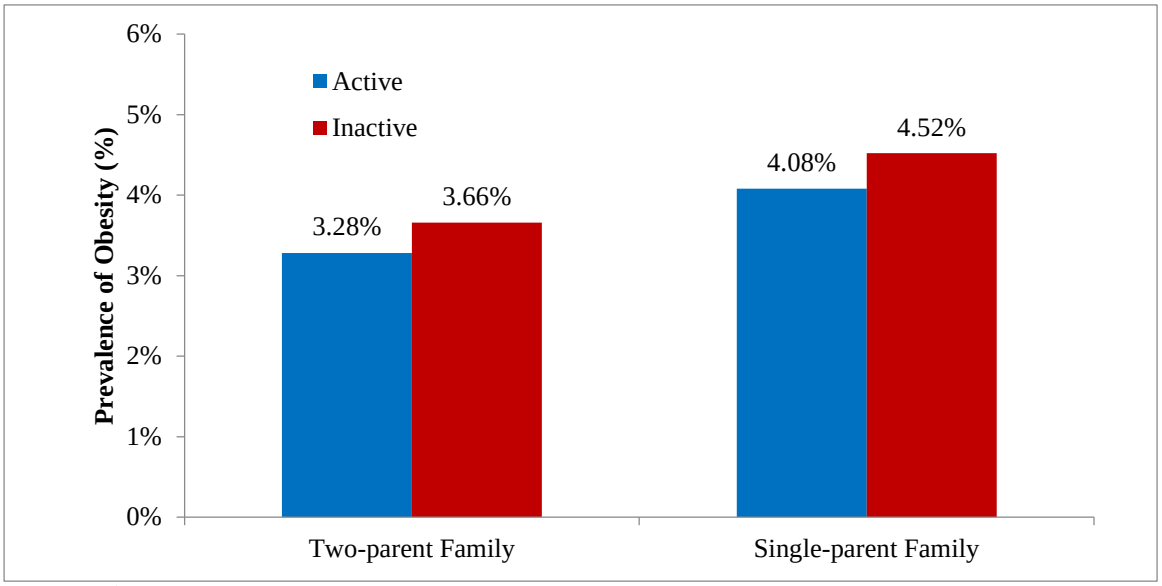
Notes: Data from Millennium Cohort Study. Results taken from the factor scores from a dynamic latent factor model.

Figure B-7 in Appendix B shows the kernel density distributions of underlying family lifestyle. The distributions for both active and inactive children appear to be very similar which explains the weak association between physical activity and childhood obesity.

Vázquez-Nava *et al.* (2013) found that family structure influenced physical activity in children and so it is important to account for this when investigating the influence of family lifestyle on physical activity and childhood obesity. It could be that physical activity in children with different family structures might be influenced differently by underlying lifestyle. Figure III-14 shows the expected likelihood of obesity in two different hypothetical children with differing family structures when they participate in exercise and when they do not. Both of these hypothetical children are white males from

families with middle SES and have a mother educated to A-level or equivalent (children 9 and 10 in Table III-14).

Figure III-14: Expected Physical Activity and Childhood Obesity by SES at 7 Years



Notes: Data from Millennium Cohort Study.

Figure III-14 shows that once family background characteristics are conditioned on, the association between childhood obesity and physical activity becomes larger. The association between physical activity and childhood obesity is similar in children from single- and two-parent families suggesting that any association between physical activity and childhood obesity is not a result of family structure. That said, family structure still has a large influence on both underlying family lifestyle and childhood obesity prevalence.

3.5.4 Summary of Results

The parameter estimates in Section 3.5.1 showed that each of the outcome measures used to identify the underlying factor were significant, suggesting that underlying family lifestyle is measured by each of them in every period. Childhood adiposity has a negative factor loading in all waves meaning that improvements to family lifestyle significantly and causally reduce the risk of obesity and overweight in children at all ages of early childhood. The standardised parameters also show that maternal weight status is heavily influenced by underlying family lifestyle, more so than child weight status or paternal weight status. This suggests that maternal behaviours play a large part in how family lifestyle is identified and that mothers are most sensitive to any changes in the lifestyle of the family. The persistence parameter α, the AR process of underlying family lifestyle, although having no direct numerical meaning, is positive and significant suggesting that

family lifestyle is difficult to change and persists over time. The analysis of the factor scores displayed in Section 3.5.2 is further evidence of the persistence of family lifestyle and shows the immobility of families to move up the rankings of family lifestyle. This immobility is particularly prevalent in families at the lower end of the lifestyle ranking. They also show how families at the lower end of the lifestyle rankings are more likely to have low SES and are more likely to have obese children.

By conditioning on both independent variables and outcome measures, it is possible to see the characteristics of children and families who have the healthiest and unhealthiest lifestyles and the children who are more or less likely to be obese. This information is essential in targeting interventions at the appropriate families. The simulations shown in Section 3.5.3 show that ethnicity has a direct influence on child weight status which differs by the age of the child. However, these differences do not appear to be a result of changes in underlying family lifestyle. Underlying family lifestyle is more strongly associated with childhood obesity than the other outcome measures individually. This emphasises the need for wider ranging interventions that do not focus only on one aspect of lifestyle. In order to effectively reduce childhood obesity in the largest number of children, disadvantaged families and those with characteristics which indicate poor lifestyle should be targeted.

Overall, the results show that interventions which successfully improve underlying family lifestyle are likely to also be successful in causally reducing childhood. Moreover, these changes in family lifestyle will improve other observable outcome measures of lifestyle, such as reducing parental obesity and improving childhood diet and physical activity. The persistent nature of underlying family lifestyle suggests that interventions aimed at family lifestyle will need strong policies which produce a shock to the underlying family lifestyle to change the trajectory of family lifestyle. These should be carried out over a long period of time and begin as early in childhood as possible in order to have the greatest possible cumulative influence. The persistence of family lifestyle also suggests that any interventions which can successfully improve family lifestyle are likely to induce long-lasting improvements in the lifestyle of the family and therefore reduce the likelihood of obesity and overweight in all family members.

3.6 Discussion and Conclusion

This chapter aimed to investigate the causal influence of underlying family lifestyle on childhood adiposity over time, while also estimating the persistence of underlying family lifestyle. In addition, the extent to which underlying family lifestyle mediates the effect of socioeconomic and family background influences on childhood adiposity was also explored. The empirical analysis directly estimated underlying family lifestyle and determined how persistent it is during early childhood. These aims were met by using a dynamic latent factor model to investigate the evolution of underlying family lifestyle as well as its influence on childhood adiposity throughout early childhood. This approach made it possible to rank families in terms of their underlying lifestyle. The parameter estimates from the model were then used to perform simulations to determine the likely outcomes of children with different characteristics and investigated how the effects of these characteristics are mediated by family lifestyle.

This study contributes to the existing literature in several ways. First, the latent factors used in each period allowed a range of outcome measures to be used to estimate an underlying family lifestyle. These latent factors provide a more comprehensive measure of lifestyle compared to single-item lifestyle variables, such as those used by many studies within the existing literature, see Reilly *et al*. (2005), Bauer *et al*. (2011), Haug *et al*. (2009) and Janssen *et al*. (2005). The use of latent factors also builds on work by Balia & Jones (2008) who use a multivariate probit model to simultaneously estimate a range of lifestyle behaviours but who do not estimate the unobservable underlying family lifestyle. Furthermore, this study contributes to the existing literature by using a dynamic model of lifestyle. Previous studies, see for example Janssen *et al*. (2005), Haug *et al*. (2009) and Giles-Corti *et al*. (2003), among others, investigated lifestyle variables using static or cross-sectional models. The dynamic nature of the latent factor model allows the evolution and persistence of family lifestyle to be explored during early childhood making it possible to investigate the effects of early life and family background influences on childhood adiposity. In addition, this chapter uses a large dataset which is representative of children and families in the UK. To my knowledge there is no other study which investigates the effects of underlying family lifestyle on childhood outcomes using such a large number of children.

The results found in this chapter support the use of a dynamic latent factor model. The persistent nature of family lifestyle which is found using this model emphasises the need

for family lifestyle to be modelled dynamically. The fact that underlying family lifestyle had a significant influence on each of the outcome measures used to identify it, illustrates the endogenous influence that it would have in a model which did not properly account for this. This demonstrates the importance and appropriateness of using common latent factor models to estimate underlying family lifestyle at each period.

3.6.1 Policy Implications

The policy implications from this study relate to interventions which influence the underlying family lifestyle, in particular those which aim to reduce childhood obesity. The underlying family lifestyle factors, although unobservable, has an influence on the observable lifestyle outcome measures and it is interventions aimed at changing the underlying lifestyle itself which this study aims to address. Interventions which have targeted underlying family lifestyle in the past include Change4Life which aims to improve the underlying lifestyle of all family members in order to reduce childhood obesity as well as improve other observable lifestyle outcomes. These types of interventions are of increased interest to policy makers in recent years due to the lack of causal evidence which suggests that targeting single-item observable variables does not produce a large enough reduction in childhood obesity or improvement in other observable lifestyle outcomes. This type of lifestyle intervention focuses on changing the attitudes and beliefs about lifestyle and improving willingness to change.

From the simulations, it is apparent that the largest influence on family lifestyle was found to be previous family lifestyle, again suggesting that interventions aiming to improve family lifestyle should be implemented as early as possible to have the most influence. This persistence of underlying family lifestyle suggests that any exogenous shock to family lifestyle, caused by an intervention or otherwise, which successfully improves underlying lifestyle, will have long-lasting influences on childhood adiposity as well as the other observable outcome measures for all family members. For this reason, policy makers should consider the long-term and multiple benefits when estimating the expected benefits of any interventions. Policy makers should consider the influences of underlying family lifestyle on the variety of outcome measures rather than focusing only on the benefits to child weight status. The persistence of family lifestyle also means that any interventions which aim to improve family lifestyle will need to be substantial or sustained, in order to cause a significant and permanent improvement family lifestyle. Long-term interventions are likely to be required in order to have a large enough effect to reduce the prevalence of childhood obesity to a meaningful extent. Given that there is

evidence that any effect of family lifestyle will be cumulative, it is likely that these policies will see bigger long term improvements that those tackling individual observable lifestyle outcomes.

Analysis of the factor scores showed that families rarely move up or down the lifestyle distribution over time. This lack of mobility around the family lifestyle distribution suggests that interventions should be targeted at families before children are born or as soon as possible in early infancy. Once a child has 'learned' the family lifestyle it could become their own individual lifestyle and could be more difficult to change in later life, or even later in childhood. There was an increasing association between childhood obesity and underlying family lifestyle as children got older suggesting that later in childhood, obesity might be even more dependent on underlying family lifestyle. This again suggests that interventions should be aimed at families as soon as possible when their children are as young as possible. Although interventions carried out before a child is born might be the most effective in reducing childhood obesity, targeting families before child birth and or very early in infancy is not always possible. The model in this chapter also provided evidence that despite earlier interventions being most effective lifestyle interventions later in childhood, if successful, could still influence childhood adiposity.

The standardised factor loadings displayed in Section 3.5.1 showed that underlying family lifestyle has a significant negative influence on the likelihood of obesity in all family members. In particular, maternal weight status was very strongly influenced by family lifestyle. This suggests that the mother is a large driver of underlying family lifestyle but also illustrates how all family members could benefit from interventions which target the family as a while. An example of a family wide intervention is Change4Life.

Current UK policies such as Change4Life have identified the need to target families rather than individuals when aiming to improve childhood outcomes. Results from this chapter support the use of interventions such as those which aim to tackle attitudes towards lifestyle and educate families about how they can improve their lifestyles and what the benefits of doing so might be. Encouraging change in specific lifestyle behaviours cannot singlehandedly address the obesity epidemic, nor can tackling social determinants of underlying lifestyle. Consequently, policies which simultaneously target a range of lifestyle behaviours could be one potential way of effectively reducing the prevalence of childhood obesity. Moreover, if these policies improve other observable lifestyle

outcome measures they could also reduce inequalities in obesity prevalence between advantaged and disadvantaged families. The observable socioeconomic and family background variables included in the model provide help to policy makers in identifying which groups of people might benefit most or be in most need of this type of lifestyle interventions. Their inclusion in the model allows different effects to be estimated for different groups of children. Single-parent families from low SES backgrounds with less educated mothers generally have unhealthier lifestyles and policy makers could target interventions at children from these families.

As discussed in Chapter I, as well as the direct impact that this study might have on policy, the findings could also have an indirect impact on policy, such as through NICE guidance, as a result of the more complex and comprehensive dynamic model that it estimates. The multiple outcome measures used in each period of the model have policy implications which go beyond just childhood obesity policies. In addition to the analysis displayed in this chapter, parameter estimates from this model could be used as evidence for a range of public health lifestyle-related interventions. Because this model estimates a variety of parameters for the effects of underlying family lifestyle on each of the outcome measures, the evidence it provides could be used by policy makers aiming to improve parental obesity, increase physical activity in children or improve any one or more of the outcome measures of lifestyle. The simultaneous estimation of the system of equations included in the measurement models can also provide economic models with estimates of correlations between these equations. This allows for economic models which rely on fewer assumptions.

By estimating the same outcome measures over a period of time using longitudinal data, this study provides more long-term evidence than many other studies in the area and could lead to stronger public health guidance. The dynamic nature of the model is also important for providing economic models with information that can be used to identify the most cost-effective interventions while using fewer extrapolations.

The simulations using estimates from the structural model in this chapter show only a fraction of the potential of this model and are a small demonstration of what this structural model has the potential to be used for.

3.6.2 Limitations and Future Research

This study contributes to the existing literature in that it directly estimates underlying family lifestyle and models it dynamically. However, there are limitations to this study

and there are a number of areas in which future research could build upon or add to this work.

Limitations in the data prevent the factor scores from being 'anchored' meaning that the scale of the factors scores over time are not directly comparable and that the AR process for family lifestyle has no numerical interpretation. Although this study gets around these problems by using rankings and percentiles as well as simulations, data which included information on adult outcomes would make it possible to anchor the factor scores. This might allow these parameters to be more easily interpreted without the need for simulations or for the percentiles of the lifestyle factors to be calculated. However, other recent studies, see for example Hancock *et al*. (2015) and Gladwell *et al*. (2015), have also used models which are not anchored to outcomes in this way and this lack of 'anchoring' does not affect the simulated predictions at all.

Similarly, data from before birth would also have been useful in that family lifestyle could have been identified before the birth of a child. This might have allowed the effects of having a child on a family's lifestyle to be investigated. More detailed data on siblings might also have been useful and future research from later waves which contain such data could focus on the differences between individual and family effects.

The family lifestyle which is identified in this chapter is that which has led to the manifestation of the observable outcome measures used to measure it. It is therefore by definition, the lifestyle of the family in the weeks and months leading up to these outcome measures being observed. However, some outcome measures may take longer to be affected by family lifestyle than others. The MCS data has limitations in that variables are only reported approximately every two years. Therefore the model is constrained by the frequency of the survey waves; the variables are all observed during a single time point.

The results suggested that family lifestyle is persistant and is already well established by the time a child reaches seven years old. However, as children become adolescents and increasingly interact with people outside the family home, they might be less influcenced by their family's lifestyle and could develop a more personal, individual lifestyle as they become more independent. Further research could investigate how the dynamic path of lifestyle changes throughout childhood and adolescence when they begin to make their own lifestyle decisions. Balia & Jones (2008) found that parental lifestyle decisions had no significant influence on an individual lifestyle in adults over the age of forty,

suggesting that by the time an individual reaches adulthood, they are no longer influenced by the lifestyles of their parents and the lifestyle that they learned as they were growing up is already well established. Future research into the intergenerational transmissions of lifestyle could be useful to policymakers wishing to identify different ways that interventions might be able to prevent unhealthy lifestyles from being passed from parent to child.

The effects of ethnicity appear to change overtime suggesting that further research investigating later stages of childhood could reveal bigger differences between children of different ethnicities. The effects of ethnicity on child weight status were not found to be mediated by family lifestyle and so further research into the reasons for the differences in adiposity in children of different ethnicities could be of interest. Further research into the different co-morbidity risks experienced as a result of obesity in children of different ethnicities could also help to identify more clearly which children should be targetted by anti-obesity policies. This could also help inform future NICE guidance to build on the current NICE (2013) recommendations.

Results from the simulations in this chapter consistently show the prevalence of obesity to be generally higher in three year olds than in children aged five and seven years. Therefore, many of the children classified as obese at three years old will no longer be obese two years later. This suggests that the IOTF childhood obesity classifications used in this study could be overly sensitive for very young children. Identifying children as obese at such a young age is only necessary if the obesity is a risk to their health. It could be that this risk is being exaggerated at the age of three if many of those identified as obese are no longer obese by the age of five. Further research to test the robustness of the model used in this study to the use of different childhood obesity definitions could add further weight to the results of this chapter. Similarly, further research into the classifications of childhood obesity could help to identify a more appropriate way of defining childhood obesity in very young children, specifically before the adiposity rebound.

This chapter investigated the dynamic relationship between family lifestyle and a range of lifestyle outcome measures, in particular childhood adiposity. However, there is a disagreement in the existing literature as to whether or not childhood adiposity is related to childhood health. It is well documented that obesity in adulthood leads to increased risks of mortality and co-morbidities but there is no conclusive evidence to suggest that

childhood obesity is significantly associated with physical health during childhood[66]. Further research into how the relationships investigated within this chapter are related to health during childhood could help policy makers determine how important improving lifestyle during childhood and reducing childhood obesity are for subsequent health. Improvements in family lifestyle have been associated with better child health as well as reductions in childhood obesity; see for example, Case *et al*. (2002), Currie (2011) and Contoyannis & Jones (2004). The relationship between health and childhood obesity is unclear. Many studies have found an association but Daniels (2006) explained that causal effects are difficult to identify. Further research into these relationships could help to disentangle some of the reasons behind these associations. Reilly *et al*. (2003) explained that many health professionals think of obesity during childhood as a cosmetic problem with no real health consequences. This could be because childhood obesity does not itself cause poor health during childhood but is instead an observable outcome measure of poor health. It could be that underlying family lifestyle is a confounding factor and influences both child health and childhood obesity causing the association between them.

[66] There are well-established links between childhood obesity and mental health.

IV. CHILDHOOD OBESITY, FAMILY LIFESTYLE AND CHILD HEALTH

Research Questions:

- What is the causal effect of child health on childhood adiposity at each stage of early childhood?
- How persistent is child health throughout early childhood?
- What are the causal effects of underlying family lifestyle on child health and on childhood adiposity?

Aims:

- To identify a more comprehensive measure of child health.
- To build on the model from the previous chapter by introducing underlying child health as an additional set of latent factors.
- To determine how underlying family lifestyle causally influences childhood adiposity, both directly, and indirectly through its effect on child health.
- To provide long-term evidence for policy makers and public health guidance providers and for use in economics models.

4.1 Introduction

The previous empirical chapter investigated how childhood adiposity and other lifestyle outcome measures are influenced by underlying family lifestyle. The chapter discussed the policy implications brought about by the results of the empirical analysis and, via simulations, identified families which should be targeted by these policies in order to reduce childhood obesity. Many family lifestyle interventions that have recently been introduced by the Government and Public Health Bodies, such as Change4Life, have aimed to simultaneously reduce obesity and improve the lifestyles of families, particularly in young children, with the ultimate purpose of improving health. Health is an important outcome of any public health lifestyle intervention. This chapter aims to identify the extent to which changes in family lifestyle can influence childhood health and the extent to which improvements in both family lifestyle and child health can help to reduce childhood obesity.

The relationship between child health and obesity is not well-established. Deckelbaum & Williams (2001) suggested that co-morbidities experienced by obese children are the same as those experienced by obese adults. However, Reilly *et al*. (2003) explained that childhood obesity is seen by health professionals as a purely cosmetic problem with no real health consequences. However, a number of studies have found an association between childhood obesity and health during childhood, for example see Reilly *et al*. (2003), Must & Strauss (1999) and Verbeeten *et al*. (2011), amongst others. Despite these conflicting arguments, little research has been done into the causal effects of health on childhood obesity. Moreover, it is possible that family lifestyle has both a direct influence on childhood adiposity and an indirect influence through the effect that family lifestyle has on childhood health. The analysis presented in this chapter aims to explore these relationships and identify causal relationships between them.

The model estimated in this chapter will introduce an additional set of latent factors representing child health, again following Cunha & Heckman (2008). A second dynamic equation, for latent child health, is added to the dynamic model for lifestyle from the previous chapter. Child health is identified using several measures available in the MCS dataset. In this model, underlying family lifestyle in one period will be allowed to influence both family lifestyle and child health in the next period. Similarly, child health in one period will influence child health in the next period. Childhood adiposity will be included as an outcome of both underlying family lifestyle and child health. This will

allow policy makers to identify the most appropriate mechanisms by which these interventions work. As in the previous chapter, this structural model enables the identification of a range of parameters and make it possible to predict the expected outcomes for children with differing characteristics to be estimated using a single model.

Estimating each of these parameters jointly is important for understanding both the effect of lifestyle interventions on childhood adiposity and the potential spill-over effect of lifestyle on health even at this young age. Lifestyle is expected to have a larger influence on childhood weight, earlier in childhood, but if there is also an indirect effect of lifestyle on childhood adiposity through health then any cost-effectiveness analyses should take into account both the benefits to child health and the indirect benefits to childhood weight. Improving family lifestyle policies such as Change4Life could reduce childhood obesity but could also improve underlying childhood health. This type of structural model can also be of great benefit to public health guidance providers, such as NICE. It allows individual level variables to be investigated at a population level while allowing effects to be estimated for individuals with different characteristics. If the influences of lifestyle on health and obesity are better understood, the NHS could benefit from a more efficient allocation of scarce resources at a time when NHS budgets are being stretched.

The analysis in this study shows that interventions which can successfully improve underlying family lifestyle can reduce the risk of childhood obesity as well as improving child health. Any improvements family lifestyle could also reduce the risk of childhood obesity indirectly through the effect on child health, although this effect is relatively smaller. The findings suggest that lifestyle interventions will be most effective very early in childhood because their effects will be cumulative. They also suggest that any economic models for lifestyle interventions and childhood obesity should also take into account further benefits to child health.

The remainder of this chapter is structured as follows. Section 4.2 will review the relevant literature and identify how the empirical analysis presented in this chapter contributes to the existing literature. Section 4.3 will discuss the methodology and Section 4.4 will outline the data analysed. Section 4.5 will present simulations using the model parameters and Section 4.6 will discuss the implications of the findings.

4.2 Literature Review

Due to the several different aims of this chapter (outlined on page 223) and because the chapter seeks to jointly estimate an even larger number of parameters than the previous chapter, the literature review is again broken down into a series of relevant sub-sections. This is done using the same methods as those used in the previous chapter. This is the 'investigative' approach outlined by Gough *et al*. (2012). See Section 3.2 in previous chapter for more details. This review follows a number of leads from the previous study which were not relevant to lifestyle but are to child health. Studies which focus on health and were not necessarily included in the previous two reviews, were found using the methods described in the previous chapter.

The review identified the following concepts which were used as subsections of the review: *measures of childhood health, determinants of childhood health, childhood health and obesity prevalence, inequalities in childhood health and in childhood obesity* and *dynamic modelling of child health*. Similar to the previous review, studies most applicable to a UK population or similar setting were identified using their titles and abstracts and those which appeared to add conceptually to the review were investigated in more detail and included in the review where appropriate[67]. Additionally, the review focused, wherever possible, on studies which included some discussion of lifestyle and on those which did not duplicate concepts which had been made apparent in the previous review.

This section outlines the existing literature on childhood health and family lifestyle in relation to childhood adiposity. Section 4.2.1 discusses the different measures of childhood health used in the existing literature. Section 4.2.2 investigates the determinants of childhood health, including how lifestyle influences child health. Section 4.2.3 reviews the existing literature on the links between childhood health and childhood obesity. Section 4.2.4 discusses inequalities during childhood, particularly in health and adiposity. Section 4.2.5 investigates the use of dynamic modelling of health. Finally, Section 4.2.6 summarises this review of the literature.

4.2.1 Measures of Childhood Health

Similar to underlying family lifestyle, childhood health is not directly observable and there is no single established measure of the underlying health of a child. Almond *et al*.

[67] Some articles were relevant in more than one of the identified sub-sections of the review.

(2005) suggested that there was a lack of research investigating different health measures and that more should be done in order to determine a universal measure of child health.

Within the current literature there are a variety of variables and methods which are used in an attempt to quantify childhood health. There are specific measures for childhood health which have been developed and aim to measure health-related quality of life, for example the Child Health Utility 9D (Stevens, 2010). However, these types of measures are not included in typical large panel or cohort surveys such as the MCS and so many observational studies need to find alternative ways to estimate child health.

The most commonly used variables to measure childhood health within the literature using survey data are parent-reported health measures. Once children are old enough, self-reported health often becomes available in these data sets. Parent-reported childhood health is widely used within the existing literature, in a variety of contexts. For example, Case *et al*. (2002), Starfield *et al*. (2002), Currie *et al.* (2007) and Hobcraft & Kiernan (2010) all used some measure of parent-reported health. Case *et al*. (2002) compared maternal reports of childhood health with reports from doctors and concluded that, on average, doctors reported better health than mothers, suggesting that maternal opinion of their child's health might not be accurate. However, unlike the mothers, the doctors had no previous contact with the children in this study and were given no information on their previous health. If this study had been carried out using family doctors who had a previous knowledge of the children's underlying health or who had a summary of any previous doctor's visits then their diagnosis may have been more similar to the mothers' reports.

Many studies use single-item measures to proxy for childhood health, the most common in young children being birth weight. Birth weight is commonly recorded, is readily available in many data sets and provides a proxy for health at birth, before other factors can influence childhood health. A binary variable indicating low birth weight (< 2500g) was used by Currie (2011) to indicate poor health at birth. The study investigated the effects of pollution on health, concluding that pollution damaged the health of unborn children and caused low birth weight. Currie (2011) also found that a binary variable indicating a premature birth gave similar results. However, Almond *et al*. (2005) argued that birth weight is unlikely to capture all aspects of health at birth. They claim that shocks in the first trimester of pregnancy have been found to have greater effects on birth weight than shocks during the third trimester, indicating that birth weight is only picking up on certain aspects of foetal health. Also, birth weight can only be used as a proxy for

health at birth. When investigating the health of older children or when dynamically modelling health, birth weight is of little interest. It could be argued that birth weight is a measure of both initial adiposity and initial health and so it could play an important part in investigating the relationship between the two.

Case & Paxson (2008) argued that childhood height can be an indicator of early deprivation and therefore has a strong relationship with health; they claimed that if young children were taller, then they were less deprived. This relationship could be because more healthy children might have growth spurts earlier in childhood or experience their adiposity rebound at different ages. There is no evidence to support this relationship in older children or adults and consequently height could be an unreliable proxy for health in later childhood. Height would also be an inappropriate proxy for health if jointly modelling health and BMI due to height being part of the calculation for BMI.

Case *et al*. (2002) suggested that many health measures in the current literature are highly correlated with each other. They illustrated that chronic conditions, bed days and episodes of hospitalisation were likely to be positively related and suggested that using a range of variables linked with health could measure overall health more accurately than a single variable. Multiple-item measures are often used to measure certain aspects of childhood health; for example, the Childhood Health Questionnaire (CHQ) measures the general quality of life of five to eighteen year old children. The CHQ has been adapted for use in a number of countries around the world and a number of studies have tested the validity of these adaptations. For example, Raat *et al*. (2002) investigated the reliability of the Dutch CHQ and found that it worked well in predominantly healthy populations and Waters *et al*. (2000) concluded that the Australian CHQ was a valid measure of childhood health and well-being.

The data needed to use measures such as the CHQs are not always available in observational data and alternative multiple-item measures are often necessary. For example, Heckman (2012) endorsed the use of latent factors to measure childhood health with a range of observational childhood health measures. He explained that the use of dynamic latent factors to explore childhood health makes use of the proxy nature of a wide range of variables often used to measure childhood health, including those described above, such as birth weight. The use of latent factors to measure underlying childhood health could allow a more accurate measurement than using single-item measures. It could also help to identify which single-item measures are the most effective measures of underlying health at different stages of childhood.

Hillemeier *et al*. (2013) used latent class analysis to measure health status. They used a range of health indicating variables to measure childhood health and estimated a latent class model with eight classes. The health measures used to estimate the probability of latent class membership included asthma, obesity and overweight, regular medication, autism and ADHD, diabetes and hearing problems. The fact that there are eight classes shows the range of heterogeneity of health states that can be determined using multiple measures. The analysis carried out in this empirical chapter will use a dynamic latent factor model, in line with Heckman (2012), in order to estimate child health using a range of measures.

4.2.2 Determinants of Childhood Health

The determinants of childhood health are likely to vary throughout childhood. For example, health at birth is likely to be influenced by parental health and maternal behaviours before and during pregnancy, whereas health in later childhood is likely to be affected by different family lifestyle behaviours at different stages of childhood.

Much of the existing literature exploring childhood health used health measures recorded at birth as proxies for childhood health. Many studies found that health at birth is a strong predictor of health throughout childhood. Currie (2011) claimed that factors influencing health inequalities are apparent even before the birth of a child and that health at birth can be influenced by environment and behaviour, including parental lifestyle behaviours. Initial childhood health could be influenced by maternal health and lifestyle during pregnancy. For example, Currie (2011) found that maternal health at the time of birth had a significant and positive effect on the health of a child at birth, whilst Hobcraft & Kiernan (2010) found that low birth weight was a strong predictor of bad health at birth. However, a causal relationship between birth weight and poor health has not been established; it is unclear whether low birth weight is a determinant of poor health at birth or whether it is a result of poor health during pregnancy. Case *et al*. (2002) found that the effect of poor health at birth was attenuated as individuals got older suggesting that other factors need to be considered when modelling health after birth and throughout childhood.

Currie (2011) found that mothers from a lower SES were less able to provide a healthy environment for their child during pregnancy. For example, mothers with low SES were more likely to smoke and drink during pregnancy. Currie (2011) also found that higher maternal education reduced the likelihood of low birth weight and put this down to a

reduction in smoking behaviour and better environmental conditions during pregnancy. Currie (2011) also claimed that disadvantaged families on low incomes are disproportionately exposed to environmental hazards and that this accounted for part of the reduction in birth weight suffered by children from families with lower SES.

Currie & Moretti (2003) used an instrumental variable (IV) approach to estimate the effects of maternal education on childhood health in the USA. They used college openings within the local area of a family to instrument for maternal education, under the assumptions that maternal education in areas where colleges opened would increase but that the college openings would not directly influence childhood health. However, this approach proved to be controversial because of the different effect college openings had on the education of black and white mothers. Currie (2011) later discussed the possibility of using an IV approach to account for the possible endogeneity of maternal education as well as SES in predicting childhood health.

A number of studies have also explored the relationship between parent and child health. For example, Case *et al*. (2002), analysed data from a range of US sources and suggested that intergenerational transmission of SES caused the relationship between parent and child health. They discussed the possibility that intergenerational transmission of health could be due to shared genetics through the susceptibility of disease or due to shared environmental factors. These environmental factors could include lifestyle, socioeconomic or financial factors which are not always observable or measurable. They also discussed the possibility that ill or unhealthy parents might provide a lower quality of care which could cause their children to be less healthy, in effect, passing on their poor health. When including parental health in predicting the health of a child it could therefore be important to account for possible confounding factors such as SES. Case *et al*. (2002) split the participants of their study into two groups, children with biological parents and children with adoptive parents. They then looked at health-income gradients estimating self-reported health on a one-to-five scale using ordered probit models. They claimed that if intergenerational transmission of health was a result of genetics, the gradient should only appear in biological children and not in those who were adopted. They found no difference between the gradients of the two groups and concluded that intergenerational transmission of health was due to income socioeconomic similarities rather than genetic similarities. Case *et al*. (2002) also found that maternal health is a better predictor of childhood health than paternal health.

Underlying family lifestyle is associated with childhood health. However, Contoyannis & Jones (2004) found that lifestyle variables were endogenous in predicting health, due to confounding factors correlated with both health and lifestyle, for example family SES and parental education. They also found that when lifestyle factors were accounted for, much of SES-health and education-health gradients were attenuated, illustrating the importance of accounting for a range of lifestyle factors before drawing any conclusions about health gradients. Many of the lifestyle behaviours which are most likely to directly influence health are also the behaviours which are likely to influence childhood adiposity. For example, Case *et al*. (2002) highlighted the importance of diet in influencing childhood health and suggested that wealthier parents are more able to purchase healthy, balanced diets for their children. The authors claimed that if more health related behaviours or parental lifestyle factors were accounted for, the health inequalities resulting from differences in family income could disappear.

4.2.3 Childhood Health and Obesity

The relationship between health and obesity is complex and there is a large amount of research on this relationship among adults. However, when it comes to children there is a lack of research into this relationship, if and how this relationship occurs and the direction of any effect.

Reilly *et al*. (2003) claimed that the majority of health professionals believed that the consequences of obesity experienced in childhood were purely cosmetic and that this perception needed to be changed. However, there remains a lack of evidence of a causal effect of childhood obesity on poor child health. Many studies have found an association between health and obesity during childhood but the statistical techniques used do not allow a causal influence to be determined. It is possible that certain health conditions, such as asthma, could increase the likelihood of childhood obesity through, for example, being less able to do exercise. It is also possible that poor underlying health could increase both the risk of childhood obesity as well as other co-morbidities which are often associated with obesity during childhood.

Must & Strauss (1999) concluded that the majority of co-morbidities suffered as a result of childhood obesity were only present in children that were severely obese, at the extreme upper end of the BMI distribution. However, they pointed out that the number of children experiencing these health consequences is increasing due to the increasing prevalence of childhood obesity.

Cardiovascular disease (CVD) has repeatedly been linked with excess weight in adults: see for example, Pérez Pérez et al. (2007) and Poirier & Eckel (2002). Other studies, such as Daniels (2006) identified an association between obesity and CVD during childhood. Reilly (2005) carried out a systematic review and found childhood obesity to be related to risk factors associated with CVD such as high blood pressure, hypertension and diabetes. If these risk factors are apparent during childhood then the chances of developing CVD in both childhood and adulthood will be increased, as well as the prevalence of heart attacks or stroke. Saha *et al*. (2011) also found that childhood obesity could cause risk factors for CVD in Indian children between six and eleven years old. However, they had a very small sample size compared with other observational studies. The study also used statistical methods such as t-tests, chi-squared tests and Pearson's correlation coefficients; none of which account for any confounding variables which could influence both childhood obesity and CVD risk factors. Therefore their claim of a causal effect is not proven; only an association can be determined from these tests. The assumption made by the existing literature that childhood obesity is the cause of other health problems could be misleading and this lack of causal inference in the existing literature needs to be addressed.

Childhood obesity has also been linked with an increased risk of type I diabetes during childhood by Verbeeten *et al*. (2011) who concluded that there was a likely, but not conclusive, association. Again, they could not identify a causal relationship between the two and the association could be the result of confounding factors. Reilly *et al*. (2003) found evidence for a relationship between childhood obesity and asthma and later, Reilly (2005) emphasised the importance of any relationship between childhood obesity and asthma due to the increasing prevalence of both obesity and asthma in the UK.

In addition to causing health problems which are visible during childhood, childhood obesity has also been found to increase the likelihood of obesity during adulthood. For example, Must & Strauss (1999) and Reilly *et al*. (2003), amongst others, found a persistence in obesity through childhood and into adulthood. The health consequences of obesity in adulthood are much more established than those of childhood obesity; adult obesity can lead to much more severe conditions, including mortality. However, his systematic review also found evidence that childhood obesity, particularly during adolescents had an impact on health in adulthood, regardless of whether obesity persisted into adulthood. These associations were not necessarily causal and Daniels (2006) explained that identifying any causal effect of childhood obesity on subsequent adult

health was problematic. There are a lack of longitudinal datasets which follow subjects throughout childhood and adulthood. Those which do exist often suffer from attrition or end before many of the co-morbidities of interest might arise.

There is evidence in the existing literature that poor health and childhood obesity are associated with each other. However, it is unclear whether childhood obesity causes poor health in childhood or whether obesity is an observable outcome of poor underlying health. Anti-obesity interventions largely focus on lifestyle behaviours and improvements to these lifestyle behaviours could improve general health as well as reduce obesity causing a confounding effect. Further research is needed into whether the association between health and obesity during childhood remains once family lifestyle is accounted for.

4.2.4 Childhood Inequalities

The UK government commissioned a strategic review of health inequalities in England which was carried out by Marmot (2010). This review suggested six areas for improvement, including giving every child the best start in life and aiming to enable all children, young people and adults to maximise their capabilities and take control of their lifestyles. Marmot & Bell (2012) discussed the findings of the Marmot Review (2010) as well as the CSDH (2008) report discussed in the previous chapter and suggested that in order to prevent ill-health, more needed to be done to tackle the social determinants of health as opposed to primary prevention measures, such as lifestyle interventions. They concluded that by doing so, health inequalities could be reduced. Marmot & Bell (2012) found evidence of health gradients in income, employment and education. They also explained that in the UK, health care was free at the point of delivery and, as a result, an income-health gradient should not exist. However, this did not take into account the fact that people on lower incomes might get ill more frequently and the authors acknowledged that the health system could not remove health inequalities singlehandedly. Marmot & Bell (2012) also investigated the SES-health gradient and concluded that it existed because of confounding factors which were present more frequently in lower SES groups and which worsened health; these included smoking, inactivity and bad nutrition. They also found that alcohol consumption was greater among higher SES groups but that hospital admissions relating to alcohol abuse were higher in lower SES groups. They suggested that parental education influenced health behaviours and lifestyles and improving parental education could help to narrow health inequalities. Factors indicating parental lifestyle such as parental smoking and drinking behaviours also influenced

childhood health. Although these reviews of health inequalities focussed on childhood health as an outcome, childhood obesity is one of these outcomes and is also subject to inequality.

Marmot & Bell (2012) claimed that the NHS focussed primarily on curing ill-health and that only around 4% of NHS funds were spent on the prevention of ill-health. They suggested that policies targeted at the prevention of ill-health could be more effective. This might also apply to childhood obesity; if more were done to prevent children from become overweight or obese, perhaps by improving their lifestyle at a young age, then the costs to the NHS could be much lower if fewer children became obese adults in need of obesity-related health care.

Currie (2011) emphasised the importance of targeting health policies at appropriate individuals in order to maximise benefits; for example, targeting children of white educated mothers in the US could increase the uptake of an intervention but could also exacerbate the situation by widening inequalities. Currie (2011) also advised that mothers should not be forgotten by policy makers aiming to improve childhood health or reduce health inequality. She claimed that maternal lifestyle could affect childhood health even before pregnancy and recommended that women should be targeted before having children. The links between maternal health and child health were investigated further by Almond & Currie (2011) who discussed the 'fetal origins hypothesis' and how it fits in with the economic literature. Deckelbaum & Williams (2001) also suggested that women of childbearing age should be targeted by policies aiming to improve childhood health. They claimed that policies should aim to prevent excessive weight gain during pregnancy, support breastfeeding and encourage parents to feed their children appropriate foods whilst weaning. These studies emphasised the importance of addressing maternal lifestyle behaviours before, during and after pregnancy.

Policy makers might also need to consider family income when constructing interventions. The Marmot Review (2010) discussed a threshold income for a 'healthy lifestyle', suggesting that income should be sufficient to provide satisfactory nutrition, healthcare and hygiene. However, it might not be enough to ensure that everyone has the income needed to live a healthy lifestyle if they do not have the knowledge necessary to provide the healthy lifestyle for themselves or for their children. Simply providing individuals with a threshold income to provide a healthy lifestyle could have the adverse effects unless they were also provided with education and information about healthy

living. Reinhold & Jurges (2012) suggested that there was a second income threshold over which income no longer improved health.

The policies described in this section generally focus on lifestyle behaviours which might influence health, specifically with a focus on family or parental lifestyle. However, there have been many criticisms of this type of policy and many studies have suggested that more needs to be done and that new approaches should be tried. Graham (2004a) highlighted the importance of considering inequalities when creating policies aimed at improving health in order to create a policy making approach which was 'determinants-oriented'. It is also important to acknowledge the literature on victim blaming when creating policies which are related to lifestyle. Ryan (1971) explained at length the problems that can occur when victims are blamed for their circumstance. He explained that blaming individuals for their misfortune or unfortunate circumstance could further worsen the problem. When creating lifestyle policies it is important to identify the true cause of the wider social problems rather than focussing on individual behaviours or characteristics. That said, these characteristics need to be identified in order to determine the mechanisms by which social determinants affect underlying lifestyle or health. This chapter aims to inform policy makers about which children would benefit most from family lifestyle interventions and which groups of children should be targeted in order to produce the greatest reduction in childhood obesity prevalence as well as the largest improvement in child health. Independent socioeconomic factors will also be allowed to influence underlying lifestyle, allowing policy makers to identify the wider social determinants.

4.2.5 Dynamic Modelling of Health

Many studies in the existing literature have each acknowledged the importance of dynamic health models. Persistence in health over time could be due to a number of causes; these are explained in more detail by Jones *et al*. (2004) but are briefly outlined here. Many health issues are long-lasting, causing the same health problems to be correlated with underlying health over a number of time points. Another possibility is that characteristics which influence health are often persistent; for example, SES does not tend to vary over time and has been shown to influence health. Similarly, poverty, educational attainment and nutrition are all characteristics which are often persistent themselves and could have an effect on health throughout the life-course. Understanding how health develops over time will be important in estimating a dynamic model and determining the causes of this development will be central to including the appropriate

independent variables. Inequalities in health between individuals from different socioeconomic backgrounds could account for the persistent nature of health found by many existing studies. However, the reasons for these health inequalities can be complicated and often controversial. Jones *et al*. (2004) explained that direct links from SES to health could arise because of, for example, poverty or lack of disposable income. Reverse causality could also occur if an individual's health effects their earnings or their ability to work. There could also be confounding influences which affect both health and SES, such as education; the well-educated might be more likely to comply with medical advice and also achieve a higher SES. The large existing literature surrounding this issue is discussed further by Adams *et al*. (2003). From a policy perspective it is important to remember that SES might influence health but that social mobility is often low and helping families to improve their SES could be more productive than directly targeting their health. Mackenbach (2012) argued that rich countries with good welfare states still have persistent health inequalities and gave a list of nine possible explanations for this. This review does not explore the different theories explaining health inequalities but it is important to consider the large number of possible mechanisms by which health inequalities occur and persist.

A number of economists have modelled health dynamically. In 1972, Grossman developed the first dynamic model for health. The Grossman model treated 'good health' as a commodity and investigated the demand for this commodity as well as the demand for medical care. Grossman (1972) assumed that an individual's initial stock of health was inherited from their parents and as individuals got older their commodity of good health depreciates. In the Grossman model, an individual's health can be improved throughout life by investments. Individual health depends on past health, after the initial inherited health and on investment in health capital minus any health depreciation. The model assumed that improved health produces more productive economic activity and therefore increases income. Good health also improves leisure time which increases utility. The Grossman model takes the view that an individual chooses the length of their life through their investment in health, through their consumption of medical care and also through health-related lifestyle behaviours. Health is also allowed to depend on environmental factors, such as education and social class. The value of the commodity 'good health' depends on many things as well as the price of health care. The 'shadow price' of good health rises with age, assuming that health depreciates with age, and decreases with education, assuming that individuals with higher levels of education are better at producing good health. When the stock of good health falls below a specified

level, then death occurs. Results from the Grossman model found that as the "shadow price" of good health increases, the demand for health decreases, due to the downward sloping demand curve. However, the demand for medical care increases, possibly due to higher levels of bad health. The Grossman model treats health as a form of human capital but unlike other forms of human capital health affects time spent earning money as well as producing commodities, whereas for example, education would affect productivity when earning money. Grossman (1972) stated that the depreciation of health only started after a certain age. Therefore, the depreciation of health is unlikely to play an important role in this study which looks specifically at children; we can assume that the depreciation of health, in the cohort followed in this study, has not yet started.

Later, Heckman (2012) discussed the developmental origins of health. Like Grossman (1972), Heckman (2012) suggested that health should be modelled dynamically from an early age. However, unlike Grossman, Heckman (2012) took more of a life-cycle approach to health during childhood, a view supported by Case *et al*. (2002), Smith (2004) and Smith (2009), who found that health during childhood influenced future health as well as other outcomes such as labour market outcomes in later life. The approach that Heckman (2012) took is similar to many studies form the epidemiology literature, for example see Davey-Smith (2007). Heckman (2012) implied that modelling health at birth or even pre-birth, needs to take into account how this early health is determined. He did not assume that initial health was simply hereditary but that initial health was a consequence of both genetics and circumstance and can be measured using a latent factor comprising of a range of health outcomes. Heckman (2012) suggested an econometric approach based on dynamic latent factors and claimed to combine health literature with capability formation literature, bringing together ideas from health economics and epidemiology. This method recognised the proxy nature of health outcomes to identify underlying individual health which is not directly observable. The study also suggested that both families and environments can play an important role in determining individual health suggesting that family lifestyle might be an important determinant of childhood health. He also suggested that a good policy for health could also be a good policy for family life, an important point when investigating the relationship between health and family lifestyle. Heckman (2012) suggested the investigation of the dynamics of health at regular intervals in order to enable policy makers to target the interventions at children of the appropriate age. This would allow policies aimed at prevention and policies aimed at remediation to be compared.

Conti & Heckman (2012) discussed this developmental approach to health in more detail. Specifically, they explored the relationships between cognitive and non-cognitive skills and health during childhood. They claimed that the majority of previous literature investigating the effects of early life experiences had found that the timing of interventions was fundamental to a successful health policy. They suggested that early life interventions are more effective than later interventions aimed at remediation; however, they discussed the importance of not abandoning children who did not have access to the most appropriate early life interventions. The authors explained that the latent dynamic factor framework acknowledges the multiple dimensions of the variables used to measure the latent factors and is an important progression from the existing literature. Using results from this methodology, they suggested three lessons for policy makers; these were to target all aspects of the child's environment and personality rather than just their health directly, to start early in childhood, before birth if possible and that prevention is more effective than remediation.

Jones & Nicolás (2004) aimed to develop a method which enabled the comparison of indices of inequality in health which are based on both short and long-run measures of health and income. Pure health inequality is measured by the Gini-coefficient, whereas the income-related health inequality is measured by the concentration index. The authors emphasised the importance of longitudinal data when analysing income-related health inequality as it can reveal important relationships which cannot be identified using cross-sectional data. Using longitudinal data, Jones & Nicolás (2004) aimed to develop a measurement tool for the change in measured income-related health inequality. Their results indicated that in the presence of systematic differences in health between those moving up or down the income distribution, long-run income related health inequality differed from results obtained using a short-run framework from a series of cross-sectional analysis. The study went on to illustrate the methods developed by considering the dynamics between income and mental health in Britain. Jones & Nicolás (2004) acknowledged that the contribution of unobserved factors could outweigh the contributions of the regressors and suggested that a more sophisticated econometric specification could potentially overcome this problem. This study emphasised the importance of using longitudinal data and modelling health dynamically and also the importance of accounting for individual heterogeneity when dynamically modelling health. The model used in this chapter takes a similar approach to that of Jones & Nicolás (2004).

Contoyannis *et al*. (2004) explored the persistence of self-assessed health (SAH) outcomes using the first eight waves of the British Household Panel Survey (BHPS). The study aimed to decompose the persistence of SAH into state dependence and unobserved heterogeneity, whilst also exploring the consequences of health related attrition. In addition, the study considered the relationship between SAH and household income. In this context, a dynamic modelling approach allowed for the impact of persistent unobservable characteristics which might influence both household income and health. SAH was measured on a five point scale, where one indicates "*very poor*" health and five represented "*excellent*" health. Due to the ordinal nature of the dependent variables, the study used a dynamic ordered probit model along with the Wooldridge method for the initial conditions. The study used the natural logarithm of the equivalised annual household income, whilst accounting for a variety of other variables including marital status, highest educational level, ethnicity, size of household and the age of any children in the household. The study also included a series of time dummies in order to account for aggregate health shocks.

Contoyannis *et al*. (2004) found that, for both men and women, very poor initial health was the main source of health related attrition bias; those with very poor initial health were more likely to have non-response in subsequent waves. However, only a small percentage of individuals reported being in very poor health in the initial wave (1.5% of males and 1.9% of females). The results of the dynamic ordered probit model indicated that response rates were positively related to better health but suggested that attrition did not impose a large bias on the estimated coefficients. SAH was highly state dependent and was influenced by unobserved heterogeneity. The study reported that unobservable heterogeneity was accountable for 30% of the variation in health. Considering the impact of income on SAH, the authors reported that permanent income, measured by the mean household income over the eight waves had a larger impact on SAH than household income in the current period. The result was found to be stronger for females than for males.

This study by Contoyannis *et al*. (2004) used a dynamic model for health which aimed to overcome problems of unobserved heterogeneity, including problems suffered by static or fixed effects models when using self-reported measures such as SAH. The dynamic methodology allowed unobservable heterogeneity to be separated from the true state dependence of health. However, this study did not take into account any health-related behaviours or lifestyle variables. Lifestyle variables are likely to be correlated with

education, SES and income and could mediate the relationship between education or income and health. It is also possible that underlying lifestyle could bring about an unobservable individual effect due to its persistent nature shown in the previous empirical chapter.

Similarly, Contoyannis *et al*. (2004a) investigated the dynamics of health in a similar way to the analysis of transitions into and out of poverty by Jenkins (2000). Again, the authors made use of the panel element of the BHPS by allowing for state dependence, permanent unobserved effects and a transitory error component in order to decompose the persistence of health. They estimated a range of models including a static model and dynamic models for health with both Heckman (1981) and Wooldridge (2000) initial conditions and with and without a random error term. They found a substantial persistence in health over time and concluded that state dependence was very important. They also found that the model which did not allow for dynamics resulted in an overestimation in the proportion of persistence in health which was due to time-invariant unobservable factors. They proposed that any shocks to health would have a long lasting effect; a result which could be very important for policy implications. These health shocks also appeared to have a more permanent effect on men than on women. The authors also investigated the effect of deprivation on health and found that permanent deprivation had a negative effect on health which was ten times larger than temporary deprivation.

Contoyannis & Li (2011) investigated the dynamics of health during childhood and adolescence in an attempt to discover any systematic differences in the social mobility of health across groups of individuals with different characteristics. They used a dynamic ordered probit model with an individual random effect to estimate SAH. They investigated the dynamic effects of family SES variables on SAH for children with different neighbourhood characteristics. They accounted for state dependence using a lagged dependent variable and estimated models with and without a time-invariant random effect to account for unobserved heterogeneity. They concluded that the individual random effect improved the model fit, implying that unobserved individual characteristics were important in explaining the persistent nature of childhood health. In accordance with Contoyannis *et al*. (2004a), they found that poor health is generally more permanent than transitory. They found that family SES has a stronger influence on health and that good health is more persistent in neighbourhoods which have a higher proportion of wealthy individuals and better educated mothers. This could have been because better

educated mothers may be more equipped to make lifestyle changes to improve their child's health after a period of poor health.

Jones *et al*. (2010) explored the long-term effects of cognitive skills, social adjustment and schooling on health and lifestyle. Using a non-parametric matching technique and parametric regression techniques to analyse data from the National Child Development Study (NCDS), the authors aimed to identify a causal relationship of education on health. Jones *et al*. (2010) accounted for childhood health, measured using indicators of morbidities in children between the ages of seven and eleven years, as well as parental circumstances captured by social class and years of schooling. The study also accounted for childhood cognitive and non-cognitive skills which were collected before the children attended secondary school as well as local area characteristics, such as local unemployment rates. Adult physical and mental health were measured at the age of forty-six years, using self-reported data. The study reported that non-cognitive skills are strongly related with health and social adjustment. Children who suffered problems with social adjustment were more likely to suffer both physical and mental illnesses as an adult. In addition, these children were more likely to smoke in later life. However, the paper found that cognitive skills at age seven years did not significantly predict health in later life after non-cognitive skills were accounted for. SES measured using paternal occupation was related to both health and health related behaviours. Educational attainment had an impact on health related behaviours, with higher educational attainment reducing smoking and drinking and improving diet. Educational attainment, however, only influenced adult health if the individual attended or would have attended grammar school. Although this study analysed data from two time periods, it did not dynamically model health; it did not investigate the effects of previous health on health in the current period. A model which uses more waves of data and which includes a lagged health term could allow the determinants of health to be dynamically investigated more thoroughly.

Analysing the Health and Lifestyle Survey, Contoyannis & Jones (2004) explored the mediating effects of lifestyle in the relationship between SES and health. The authors developed a simple economic model in which health was influenced by lifestyle and lifestyle was determined by preferences, budget, time and unobservable characteristics. Health was measured using a binary indicator for self-assessed health (SAH) and the Alameda Seven[68] were used as lifestyle proxies, with the exception of stress because no suitable measure was found in the data. A binary variable indicating whether the

[68] Alameda Seven: diet, exercise, weight-for-height, smoking, alcohol, sleep and stress.

individual ate breakfast was used as a proxy for diet. In addition, smoking, alcohol consumption, optimal sleeping habits and obesity were all measured using binary variables. Estimating a multivariate probit model on discrete measures of health status and lifestyle behaviours, the study found evidence to suggest that sleeping well, exercising, and being a non-smoker in 1984 had a statistically significant and positive influence on the probability of reporting excellent health in 1991. Eating breakfast and alcohol consumption in 1984 did not influence SAH in 1991. The study suggested that a univariate probit model would severely underestimate the effects of lifestyle behaviours on health and emphasised the importance of accounting for unobserved heterogeneity. The study reported that unobserved characteristics which increased the likelihood of being a smoker, also lead to higher levels of SAH and greater participation in exercise. As a consequence, the absence of accounting for unobserved individual heterogeneity in the univariate probit specification could have led to underestimation of the effects of lifestyle behaviours on health. The estimates from a randomised treatment of lifestyle factors would not suffer from this bias. Although this study used a range of variables to measure lifestyle, the variables are treated separately, rather than being used to measure an underlying lifestyle. These lifestyle measures are also used to measure adult lifestyle and might not be as applicable when researching children. However, when attempting to measure family lifestyle, many of these adult measures of lifestyle might be useful for measuring parental lifestyle.

Mcleod & Ruseski (2013) aimed to identify the longitudinal relationship between participation in physical activity and health outcomes. Analysing eight waves of data from the Canadian National Population Health Survey, the study used a random effects ordered probit model and a dynamic unobserved effect probit model. Initially, the study presented a Grossman health production framework to explore the theoretical relationship between physical health activity and health status. Physical health activity was measured using the respondents' self-reported frequency and intensity of physical activity. Based on these measures, four binary variables were constructed in order to capture different levels of physical activity; these were active participation, moderately active participation, inactive participation and daily participation. The respondent's health outcome was based on physical health and the presence of chronic conditions. Seven binary variables were defined, capturing whether or not the respondent had specific chronic conditions including, diabetes or heart disease. In addition, SAH was also included as a binary health measure indicating whether or not the respondent reported being in poor or fair health. The study also accounted for a variety of demographic and

socio-economic conditions. The descriptive statistics suggested that the majority of respondents were physically inactive; however, participation rates in physical activity increased over the eight waves included in the study. These statistics also indicated that the prevalence of the seven chronic conditions had increased; for example, between 1994 and 2008, there was a 400% and 280% increase in the prevalence of diabetes and heart disease, respectively. The results from the random effects probit model suggested that participation in physical activity had a limited impact on health. However, physical activity participation did reduce the probability of the individual reporting fair or poor health. Interestingly, participation in daily physical activity actually increased the likelihood of having arthritis, implying that physical activity participation could have adverse health effects. It is more likely that there was some sort of endogeneity at work here and selection into daily physical activity was influenced by confounding factors which might also have affected health. It is also possible that there is reverse causality and that health has a causal effect on physical activity. The results from the dynamic probit model suggested that participation in physical activity reduces the incidence of a range of health outcomes in the next period, including high blood pressure, arthritis and heart disease. Previous participation in physical activity reduced the likelihood an individual reporting fair or poor health. The impact of physical activity on future health emphasises the importance of modelling health and health-related behaviours dynamically.

Tubeuf *et al*. (2012) explored the long term effects of early life conditions on later health outcomes. They explored the potentially mediating effects of education and lifestyle between early life conditions and later health. The study analysed data from the NCDS, the 1958 British Cohort study, using a dynamic model to explore this relationship. SAH was collapsed into a binary variable indicating good health or above compared to poor or fair health. A binary variable was also created to indicate whether an individual had any qualifications at least at O-level or above. This variable was measured when the respondent was in adolescence and it was anticipated that this education variable could influence health outcomes in later life. Binary lifestyle variables were created to indicate whether an individual was a smoker, a prudent drinker and obese, as well as variables indicating their level of exercise over the past four weeks and whether they consume fruit or vegetables at least once a week. In addition, the study controls for a series of early life environmental conditions experienced by the respondent including father's social class, the presence of financial hardship, parental education and illness. The study also considered whether the participants' parents were smokers, whether they had a chronic

condition at the age of sixteen, were born with a low birth weight or whether they were obese at the age of sixteen. Tubeuf *et al*. (2012) used a random effects dynamic probit model to investigate the dynamic evolution of health. The results indicated the early life conditions were important determinants of SAH in adulthood. Once the effects were decomposed into direct and indirect effects, the study found that the absence of a father at time of birth and the presence of financial hardship had large direct effects on adult health. Being obese at the age of sixteen was found to negatively influence adult health, both directly and indirectly through individual lifestyle. This suggested that adiposity is related to both health and lifestyle. This study also found that previous lifestyle had a greater effect on health than current lifestyle suggesting that it takes time for the effects of lifestyle to become apparent and emphasising the need for a dynamic model.

4.2.6 Summary

This chapter incorporates the lifestyle model of the previous chapter into a dynamic model for health with the aim of determining how childhood adiposity is affected by both family lifestyle and health. In accordance with the methodology described in Heckman (2012), latent factors will be used to estimate health as well as family lifestyle in each period of the model. Previous research has shown that family lifestyle and environment are strong predictors of childhood health. This new dynamic latent factor model will jointly model child health and family lifestyle with the aim of identifying the causes of childhood obesity. It will allow childhood weight status to be influenced by underlying family lifestyle, both directly and indirectly through underlying child health. This will allow the association between health and childhood obesity which is observed in many studies to be investigated while accounting for underlying family lifestyle.

The latent factors allow for a more comprehensive measure of childhood health than many of the single-item measures used in the literature and will incorporate a range of health indicators including parent-assessed health as well as more objective measures. This is also the case for underlying family lifestyle, as in the previous chapter. It also allows the influence of family lifestyle on child health to be estimated meaning that future economic models could take into account these additional benefits, as well as any reductions in childhood obesity.

Much of the previous literature which modelled health dynamically, both in adults and children, did not start the dynamic process at the same stage in life for each individual. The model estimated in this chapter will have the advantage of starting the dynamic

process of both health and lifestyle very close to birth for all individuals because a range of variables from this period of childhood are available in the MCS and can be used to measure initial family lifestyle and initial child health.

The ability to separate the contributions of true state dependence and unobserved heterogeneity is of particular importance when attempting to identify the causal effects of health in one period on health in the next period. This is emphasised in the literature. True state dependence occurs here when health is causally dependent on previous health. Unobserved heterogeneity can cause a correlation between health states in different periods but this association is not causal. Dynamic health models have an advantage over static health models because they make it possible to account for unobservable heterogeneity and identify true health state dependence. The literature suggests that both of these mechanisms are important in identifying the persistence of health.

4.3 Methodology

This section describes how child health will be introduced to extend the dynamic factor model used in Chapter IV. The method is similar to that suggested by Heckman (2012) on the development of child health and uses a model similar to Cunha & Heckman (2008). This chapter uses latent factors to measure child health in each period as well as family lifestyle in order to remove measurement error and prevent biased estimates.

The structural model estimated in this chapter allows the dynamic relationships between family lifestyle, child health and childhood obesity to be estimated in a single model. In this model, family lifestyle is allowed to influence child health both directly, and indirectly through its effects on child health. As in the previous chapter, the interpretation of the parameters is difficult in these complex models. For this reason, the analysis of factor scores and predictions using simulations are used to highlight some of the important results.

The remainder of this section is structured as follows. Section 4.3.1 will describe the dynamic latent factor model estimated in this chapter, how it identifies child health and family lifestyle and how both of these unobservable factors causally influence childhood adiposity. Section 4.3.2 will then discuss the factor scores and Section 4.3.3 will outline how predictions will be made using simulations in order to determine how different children will be affected by potential obesity policies and lifestyle interventions.

4.3.1 A Dynamic Latent Factor Model

The dynamic latent factor model used in this chapter will build on the model used in the previous chapter. It will do so by adding further latent factors, one in each period, to represent child health identified using a range of child health outcome measures. This model is similar to that used by Cunha & Heckman (2008).

Latent Factors, Endogeneity and Causality

The outcome measures of child health in each period are jointly estimated in order to identify underlying child health which influences each of these outcome measures and is the source of endogeneity between them. In addition to the latent factors representing underlying family lifestyle $\boldsymbol{\theta}^L$, there are also latent factors representing child health in each period $\boldsymbol{\theta}^H$. As in the previous chapter, all outcome measures are jointly estimated along with the existing model. This allows the sources of the endogeneity between the outcome of interest and the indicator variables to be accounted for and allows the causal influence of each underlying factor on the outcome of interest to be identified.

The latent variable underlying the indicators of family lifestyle are influenced by an underlying family lifestyle, so that

$$\boldsymbol{I}_t^{*L} = \boldsymbol{\lambda}_t \boldsymbol{\theta}_t^L + \boldsymbol{\xi}_t^L \tag{IV.1}$$

in the same way as in the previous chapter. Similarly, the latent variables underlying the indicators for child health are influenced by underlying child health, so that

$$\boldsymbol{I}_t^{*H} = \boldsymbol{\zeta}_t \boldsymbol{\theta}_t^H + \boldsymbol{\xi}_t^H, \tag{IV.2}$$

where $\boldsymbol{\zeta}_t$ is a vector of factor loadings corresponding to the underlying child health factor at time t. Probit models are used to model binary indicators and ordered probit models are used to model ordinal indicators. For continuous indicators, the observed dependent variables are equal to their corresponding underlying latent value, so that $\boldsymbol{I}_t^L = \boldsymbol{I}_t^{*L}$ and $\boldsymbol{I}_t^H = \boldsymbol{I}_t^{*H}$.

The outcome of interest, child weight status is estimated in the same way as those above. Childhood adiposity depends on both health and family lifestyle and is written

$$\boldsymbol{y}_t^* = \boldsymbol{\rho}_t^L \boldsymbol{\theta}_t^L + \boldsymbol{\rho}_t^H \boldsymbol{\theta}_t^H + \boldsymbol{\delta}_t \boldsymbol{W}_t + \boldsymbol{\xi}_t^y \tag{IV.3}$$

where $\boldsymbol{y}_t^*$ represents the unobserved latent variable underlying the outcome of interest at time t, $\boldsymbol{\rho}_t$ is the sensitivity of this outcome (factor loading) to the latent factor for child health at time t, and $\boldsymbol{\xi}_t^y$ is an error term analogous to the error terms in Equations (IV.1) and (IV.2). In the same way as the previous chapter, the model allows independent variables to influence childhood adiposity, where $\boldsymbol{W}_t$ is a matrix of independent variables influencing $\boldsymbol{y}_t^*$ and $\boldsymbol{\delta}_t$ is a vector of corresponding time-varying coefficients. In the initial wave, continuous childhood weight is the outcome of interest and so $\boldsymbol{y}_t = \boldsymbol{y}_t^*$. In subsequent waves, the outcomes of interest are child weight status. These are ordinal variables and are estimated using ordered probit models.

For simplicity, Equations (IV.1), (IV.2) and (IV.3) are stacked into a single vector of equations. This makes notation more compact and allows them to be easily written in matrix form. The vector of stacked equations is

$$\boldsymbol{Y}_t^* = \boldsymbol{\lambda}_t \boldsymbol{\theta}_t^L + \boldsymbol{\zeta}_t \boldsymbol{\theta}_t^H + \boldsymbol{\delta}_t \boldsymbol{W}_t + \boldsymbol{\xi}_t \qquad \text{(IV.4)}$$

where $\boldsymbol{Y}_t$ is a vector of outcome measures at time t. Corresponding vectors of factor loadings $\boldsymbol{\lambda}_t$ and $\boldsymbol{\zeta}_t$ indicate the sensitivity outcome measures to underlying family lifestyle and underlying child health, respectively. $\boldsymbol{W}_t$ is a matrix of independent variables which influence outcome measures at time t, and $\boldsymbol{\delta}_t$ is a matrix of corresponding coefficients. The vector of error terms $\boldsymbol{\xi}_t \sim N(0, \sigma_{\xi_t})$, is IID normally distributed with variance estimated in the model. Again, for continuous outcome measures, a linear regression is used ($\boldsymbol{Y}_t = \boldsymbol{Y}_t^*$) and discrete outcome measures are modelled using probit or ordered probit models. As in the previous chapter, the threshold parameters $\boldsymbol{\tau}_{kt}^j$, are jointly estimated for each of the discrete outcome measures and are strictly increasing (see Equations (III.8) and (III.9) in Chapter III).

The outcome measures are allowed to differ over time so that the measures underlying child health and family lifestyle are indicated by different observable variables as children get older. Not all outcome measures will be influenced by both underlying child health and underlying family lifestyle; most will be influenced by just one of the latent factors. However, childhood adiposity will depend on both family lifestyle and child health in order to determine the mechanisms by which it is influenced. Similarly to the outcome measures used to estimate underlying family lifestyle, those used to estimate underlying child health are identified using EFA. The measures of child health are expected to be highly correlated with each other because they are each influenced by the same underlying child health factor. The EFA identified a distinct difference between the variables

measuring underlying lifestyle and those measuring underlying child health. Childhood adiposity loaded into both factors.

The Structural Model: Estimation of the Relationship between Latent Factors

In the same way as the model in the previous chapter, the latent factors are related to each other by the structural model (Skrondal & Rabe-Hesketh, 2004).

In the initial period, underlying family lifestyle is

$$\boldsymbol{\theta}_0^L = \boldsymbol{X}_0^{'}\boldsymbol{\beta}_0 + \boldsymbol{u}_0, \tag{IV.5}$$

as defined in the previous chapter and in addition underlying child health is

$$\boldsymbol{\theta}_0^H = \boldsymbol{Z}_0^{'}\boldsymbol{\varrho}_0 + \boldsymbol{\eta} + \boldsymbol{\epsilon}_0, \tag{IV.6}$$

where $\boldsymbol{Z}_{i0}$ is a vector of independent variables which influence child health at birth and $\boldsymbol{\varrho}_0$ is a vector of corresponding coefficients. The error term, $\boldsymbol{\epsilon}_0 \sim N(0, \sigma_\epsilon)$ is IID normally distributed. A time-invariant unobserved individual random effect, $\boldsymbol{\eta} \sim N(0, \sigma_\eta)$, is also included to account for any unobserved characteristics which might influence child health.

Similarly to underlying family lifestyle which evolves over time

$$\boldsymbol{\theta}_t^L = \alpha_t \boldsymbol{\theta}_{t-1}^L + \boldsymbol{X}_t^{'}\boldsymbol{\beta}_t + \boldsymbol{\eta} + \boldsymbol{\varepsilon}_t \qquad t = 1 \ldots 3 \tag{IV.7}$$

as in the previous chapter, underlying child health also evolves over time, so that

$$\boldsymbol{\theta}_t^H = \gamma_t \boldsymbol{\theta}_{t-1}^H + \varphi_t \boldsymbol{\theta}_{t-1}^L + \boldsymbol{Z}_t^{'}\boldsymbol{\varrho}_t + \boldsymbol{\eta} + \boldsymbol{\epsilon}_t \quad t = 1 \ldots 3 \tag{IV.8}$$

where $\boldsymbol{\theta}_{t-1}^H$ is underlying child health in the previous period. Current underlying child health depends on both underlying child health in the previous period and underlying family lifestyle in the previous period[69]. The autoregressive (AR) and lagged coefficients, γ_t and φ_t, for child health and family lifestyle, respectively, are allowed to vary over time in order to determine more accurately how child health evolves over time and at what age interventions will be most effective. $\boldsymbol{Z}_t$ is a matrix of independent variables which influence child health at time t and $\boldsymbol{\varrho}_t$is a matrix of corresponding coefficients. The

[69] An attempt was also made to introduce a lagged response of family lifestyle from child health, such that Equation (IV.7) was replaced by $\boldsymbol{\theta}_{it}^L = \alpha_t \boldsymbol{\theta}_{it-1}^L + \varsigma_t \boldsymbol{\theta}_{it-1}^H + \boldsymbol{X}_{it}^{'}\boldsymbol{\beta}_t + \boldsymbol{\eta}_i + \boldsymbol{\varepsilon}_{it}$. However, this model would not converge when this parameter was fixed or when it was freed over time. This will be discussed later but the lack of convergence is due to the data rather than the identification methods.

unobserved individual random effect $\boldsymbol{\eta} \sim N(0, \sigma_\eta)$, is again included to account for unobserved time-invariant characteristics influencing child health, such as genetics.

In order to account for exogenous shocks, the addition of a correlation between the error terms of the underlying family lifestyle and child health equations in the same period was added to the model. This allows any unexpected shocks that influence the health of a child to also contemporaneously influence the lifestyle of a family. This correlation was found to be insignificant and was therefore not included in the final model. Any exogenous shock to child health was found to have no significant influence on family lifestyle in the same period.

As well as accounting for exogenous shocks, potential cross directional influences between the two dynamic processes were investigated. It is possible that child health in one period might influence family lifestyle in the next. Specifications of the model where lagged child health was included in the dynamic family lifestyle equation were estimated. This model replaced Equation (IV.7) with Equation (IV.9), so that

$$\boldsymbol{\theta}_t^L = \alpha_t \boldsymbol{\theta}_{t-1}^L + \boldsymbol{\varsigma}_t \boldsymbol{\theta}_{t-1}^H + \boldsymbol{X}_t' \boldsymbol{\beta}_t + \boldsymbol{\eta} + \boldsymbol{\varepsilon}_t \qquad t = 1 \ldots 3. \qquad \text{(IV.9)}$$

However, although this model is theoretically identified, it failed to converge. The lack of convergence is because there were not enough data to identify these additional parameters[70].

Identifying Assumptions

In line with the model in Chapter III, the variance of $\boldsymbol{u}_0$ in Equation (IV.5) is fixed at 0.05 and the variance of $\boldsymbol{\varepsilon}_t$ in Equation (IV.7) is fixed at 0.01, for model identification. In addition, the variance of error term $\boldsymbol{\epsilon}_t$ in Equations (IV.6) and (IV.8) is fixed at 0.05, also for model identification. The variance of the error terms for the estimation of discrete variables, $\boldsymbol{\xi}_t$ in Equation (IV.4) is also fixed at 1 to allow identification of the probit and ordered probit models. Again, the values of these variances are arbitrary and do not influence the model empirically. The method of identification used in this chapter is similar to that of an ordered probit model, in that numerical meaning is not given to each variable, but both types of models still provide meaningful information and predictions and this identification has no impact on the simulated predications or policy

[70] This version of the model would not converge when the AR parameter ς_t was fixed over time or when it was freed. A range of specifications fixing and freeing different parameters were attempted, but all failed to converge.

recommendations. Similar to the previous chapter, some variables are fixed for empirical identification. These will be discussed later in Section 4.5.

In the final model estimated in this chapter, there is no independent variable which appears in both vector $\boldsymbol{W}$ in Equation (IV.4) and the two vectors of independent variables influencing the latent factors $\boldsymbol{X}$ or $\boldsymbol{Z}$ in Equations (IV.5) to (IV.8). The only assumption made about the relationship between them is that there is no perfect multicollinearity between them. Theoretically, there could be independent variables which influence both childhood adiposity and the latent factors. However, the literature suggested that it was different variables which were expected to affect each of them. Some independent variables are included in both $\boldsymbol{X}$ and $\boldsymbol{Z}$ but this is not a problem because they each effect different latent factors. The variables included in each of these vectors are discussed later in Section 4.4.2.

The theoretical identification of this model, as well as the model accounting for cross directional influences between health and lifestyle in Equation (IV.9), was proven by Cunha & Heckman (2008) (page 747) who gave a detailed explanation of how all parameters in this dynamic model are identified. These identification assumptions[71] include assumptions involving the error term $\boldsymbol{\xi}_t$ from Equation IV.4 as well as the error term $\boldsymbol{\varepsilon}_t$ from Equation IV.9 and were included in the set of restrictions discussed in the methodology. They are summarised as follows:

- ξ_t are mean zero and independent across agents and over time for all time periods, all outcome measures.
- ξ_t are mean zero and independent of all latent factor in all time periods.
- ξ_t are mean zero and independent across latent factors.
- ε_t is the sum of a factor-specific error and a measurement error, as explained in Equation III.4 in the previous chapter. The measurement error $\boldsymbol{e}_t$ is independent of the factor-specific error term, the latent factors at time t and $\boldsymbol{e}_\tau$, where $t \neq \tau$, conditional on any independent variables.

The proof by Cunha & Heckman (2008) shows how all models in this chapter are theoretically identified, without the need for independent variables which influence child health but do not influence family lifestyle are not needed for identification. In a static

[71] Greek letters used in the paper by Cunha & Heckman (2008) have been changed in line with those used in this study.

model which jointly estimated both health and lifestyle and which allowed health and lifestyle to contemporaneously influence each other, exclusion restrictions would apply in order to identify the model. However, in this type of dynamic model, the parameters are identified instead by the restrictions imposed on the covariance matrix of the error terms. It must be assumed therefore, that the lack of convergence in the model which includes a cross dependence between health and lifestyle is due to a lack of empirical identification rather than theoretical identification. A larger number of observations or more time periods might enable this model converge but is not possible using the data available in this study.

The latent variables within the model defined by Equations (IV.4) to (IV.8) need to be integrated out of the likelihood function. This requires the computation of an eight-dimensional integration which is performed using Monte Carlo integration with 8,000 integration points and with a sandwich estimator to compute robust standard errors[72]. A Fisher Score algorithm is used to calculate the gradient of the likelihood with respect to the parameters. As in the previous chapter, the model is estimated using Mplus 6.1 (Muthen & Muthen, 2011).

The model described in this section is even more complex than that in the previous chapter due to the large number of parameters it estimates. Consequently, a number of different ways of representing the results will be used to aid interpretation of the findings. This includes analysis using standardised parameters outlined in Equation (III.13) as well as factor scores and simulations which are described below.

4.3.2 Factor Scores

The latent factors estimated in this dynamic latent factor model quantify both underlying family lifestyle and underlying child health, in each period of the model. Percentiles are used to give numerical interpretation to these factors, as discussed in the previous chapter.

Factor scores are estimated using posterior distributions as they were in the previous chapter in Equation (III.14).

$$\boldsymbol{Y}^{*} = \boldsymbol{\Lambda\vartheta} + \boldsymbol{\delta W} + \boldsymbol{\xi} \qquad \text{(IV.10)}$$

[72] Eight-dimensional integration would be required because there were two latent factors in each of the four periods, resulting in eight dimensions.

The only difference is that here $\boldsymbol{\vartheta}$ is now an eight-dimensional vector of both latent family lifestyle factors and latent child health factors.

Additionally,

$$\boldsymbol{\vartheta} = \boldsymbol{B\vartheta} + \boldsymbol{\beta X} + \boldsymbol{\varrho Z} + \boldsymbol{e} \tag{IV.11}$$

differs from Equation (III.15) in Chapter III in that $\boldsymbol{B}$ is now an eight-by-eight parameter matrix and there is the addition of $\boldsymbol{\varrho Z}$ where $\boldsymbol{Z}$ is a vector of independent variables influencing child health factors with corresponding coefficients, $\boldsymbol{\varrho}$.

The vector of error terms,

$$e = \boldsymbol{\eta} + \boldsymbol{\varepsilon} + \boldsymbol{\epsilon} \tag{IV.12}$$

is made up of the unobserved individual random effect $\boldsymbol{\eta}$, the residual error term $\boldsymbol{\epsilon}$ from Equations (IV.6) and (IV.8) and the residual error terms, $\boldsymbol{u}$ and $\boldsymbol{\varepsilon}$ from Equations (IV.7) and (IV.10), respectively. It is assumed that $\boldsymbol{B}$ is non-singular.

The expected mean of $\boldsymbol{\vartheta}$ given $\boldsymbol{X}$ and $\boldsymbol{Z}$ is therefore

$$E(\boldsymbol{\vartheta}|\boldsymbol{X},\boldsymbol{Z}) = (\boldsymbol{I}_4 - \boldsymbol{B})^{-1}(\boldsymbol{\beta X} + \boldsymbol{\rho Z}) = \mu \tag{IV.13}$$

and has conditional variance

$$Var(\boldsymbol{\vartheta}|\boldsymbol{X},\boldsymbol{Z}) = (\boldsymbol{I}_4 - \boldsymbol{B})^{-1}\psi(\boldsymbol{I}_4 - \boldsymbol{B})^{-1} = \Sigma \tag{IV.14}$$

where $\psi = Var(\boldsymbol{e})$.

It follows that posterior distribution of $\boldsymbol{\vartheta}$, given $\boldsymbol{Y}$, $\boldsymbol{X}$ and $\boldsymbol{Z}$, is

$$g(\boldsymbol{\vartheta}|\boldsymbol{Y},\boldsymbol{X},\boldsymbol{Z}) \propto \phi(\boldsymbol{\vartheta}|\boldsymbol{X},\boldsymbol{Z})\mathrm{f}(\boldsymbol{Y}|\boldsymbol{\vartheta},\boldsymbol{X},\boldsymbol{Z}) \tag{IV.15}$$

where $\phi(\boldsymbol{\vartheta}|\boldsymbol{X},\boldsymbol{Z})$ is multivariate normal with mean vector, μ and covariance matrix Σ.

Families can then be ranked in order of their factor scores for each of the latent factors in each time period. This will allow the mobility of child health as well as family lifestyle to be investigated, determining how difficult it is for families to change their underlying lifestyle or their child's health as well as the relationship between the two distributions.

4.3.3 Predictions

Simulations are used to predict child and family outcomes in the same way as in Chapter III. The calculations presented below require the computation of several integrals and so they are approximated using simulations.

The conditional distribution of the outcome of interest, $\boldsymbol{y}$ shown in Equation (III.19) in Chapter III is now also conditional on independent variables $\boldsymbol{Z}$, as well as $\boldsymbol{X}$ and $\boldsymbol{W}$, so that

$$f(\boldsymbol{y}|\boldsymbol{X},\boldsymbol{Z},\boldsymbol{W}) = \int f(\boldsymbol{y}|\boldsymbol{\vartheta},\boldsymbol{W}) \cdot f(\boldsymbol{\vartheta}|\boldsymbol{X},\boldsymbol{Z}) d\boldsymbol{\vartheta} \qquad \text{(IV.16)}$$

meaning that, conditional on these independent characteristics, the expected value of $\boldsymbol{y}$ is

$$E(\boldsymbol{y}|\boldsymbol{X},\boldsymbol{Z},\boldsymbol{W}) = \int \boldsymbol{y} \left[\int f(\boldsymbol{y}|\boldsymbol{\vartheta},\boldsymbol{W}) \cdot f(\boldsymbol{\vartheta}|\boldsymbol{X},\boldsymbol{Z}) d\boldsymbol{\vartheta}\right] d\boldsymbol{y}. \qquad \text{(IV.17)}$$

Equation (IV.16) is integrated over all values of $\boldsymbol{y}$ to predict outcomes of continuous variables. The sum of the integrals for each value of discrete variable $\boldsymbol{y}$ is calculated in Equation (IV.17). As described in Chapter III, these calculations allow childhood adiposity to be predicted for children at specific ages, conditional on independent variables.

Similarly, the distribution of outcome $\boldsymbol{y}$ conditional on independent characteristics and other outcome variables, say I_k, is given by

$$f(\boldsymbol{y}|\, I_k, \boldsymbol{X},\boldsymbol{Z},\boldsymbol{W}) = \frac{\int f(\boldsymbol{y},\, I_k|\boldsymbol{\vartheta},\boldsymbol{W}) \cdot f(\boldsymbol{\vartheta}|\boldsymbol{X},\boldsymbol{Z}) d\boldsymbol{\vartheta}}{\int f(\, I_k|\boldsymbol{\vartheta},\boldsymbol{W}) \cdot f(\boldsymbol{\vartheta}|\boldsymbol{X},\boldsymbol{Z}) d\boldsymbol{\vartheta}} \qquad \text{(IV.18)}$$

and the mean of that distribution, that is, the prediction is given by

$$E(\boldsymbol{y}|\, I_k, \boldsymbol{X},\boldsymbol{Z},\boldsymbol{W}) = \int \boldsymbol{y} \left[\frac{\int f(\boldsymbol{y},\, I_k|\boldsymbol{\vartheta},\boldsymbol{W}) \cdot f(\boldsymbol{\vartheta}|\boldsymbol{X},\boldsymbol{Z}) d\boldsymbol{\vartheta}}{\int f(\, I_k|\boldsymbol{\vartheta},\boldsymbol{W}) \cdot f(\boldsymbol{\vartheta}|\boldsymbol{X},\boldsymbol{Z}) d\boldsymbol{\vartheta}}\right] d\boldsymbol{y}. \qquad \text{(IV.19)}$$

Again, for continuous variables, Equation (IV.18), is integrated over all values of $\boldsymbol{y}$ and for discrete values, the sum of the integrals for each value of $\boldsymbol{y}$ is calculated for Equation (IV.19). These calculations will allow, for example, childhood adiposity to be predicted for specific children at specific ages, conditional on independent variables as well as lifestyle or health indicators such as parental weight status or health problems.

Simulations are used to approximate these equations because they require the computation of several integrals. These simulations use parameter estimates from the dynamic latent factor model outlined in Section 4.3.1. Simulations in this chapter are calculated using a user-written program[73] in Mata 13 written specifically for this purpose.

4.4 Data

Many of the variables used in this empirical chapter are identical to those used in the previous chapter. The outcome measures used to identify the latent factors for underlying family lifestyle will be the same as those used in the previous chapter (Table III-1). The independent variables which influence underlying family lifestyle as well as those which influence childhood adiposity will also be the same as in the previous model (Table III-2 and Table III-3, respectively). These parameters will be re-estimated jointly with the additional parameters introduced in this chapter.

Section 4.4.1 will discuss the variables used to measure child health using latent factors and Section 4.4.2 will outline the independent variables which are allowed to influence these latent factors for child health. Section 4.4.3 will then describe the excluded observations due to missing or outlying data.

4.4.1 Latent Factors for Child Health

As children grow up the type of illnesses that will best identify health change. For this reason, a different set of health outcome measures is used to identify underlying child health in each period. In the same way as those chosen to indicate underlying family lifestyle, outcome measures of child health are chosen in accordance with the literature and using EFA. This section outlines the measures of child health used in the initial period of the model to identify health at birth. It then describes the outcome measures used to indicate child health over the subsequent periods.

Initial Conditions for Health

The variables used to identify initial child health taken from the first wave of the MCS relate to pregnancy, birth or soon after, in the first nine months of infancy. These are displayed in the first column of Table IV-1.

[73] This Mata program was written by me specifically for the predictions in this chapter.

Birth weight in kilograms and gestational time in weeks minus the average (39 weeks) are taken from variables derived within the MCS. A binary variable indicating whether or not a child was in a special care unit (SCU) immediately after birth is also included.

Mothers were asked about a range of health problems during infancy: '*We would now like to know about any health problems for which <child's name> has been taken to the GP, Health Centre or Health visitor, or to Casualty, or you have called the NHS direct. How many separate health problems, if any, has <child's name> had, not counting any accidents or injuries?*' If a child had experienced any health problems then mothers were asked, '*What was this problem?*' or '*What were these problems?*' The answers to these questions were then used to create binary variables indicating whether a child had ever experienced chest infections, asthma or wheezing, feeding problems or growing problems. Mothers were also asked '*Were there any problems with his/her hearing?*' and a binary variable was created to indicate whether a child had ever had any hearing problems. These variables are outcome measures of initial child health and are represented by $\boldsymbol{Y}_t$ in Equation (IV.4), when $t = 0$.

Table IV-1: Outcome Measures of Underlying Child Health and Family Lifestyle

Outcome Measures of Latent Factors (*Y*, Equation (IV.4))			
Initial Period	**3 Years**	**5 Years**	**7 Years**
Family Lifestyle and Child Health Outcome Measures			
-	Weight category	Weight category	Weight category
-	Mother is a smoker	-	-
Child Health Outcome Measures Only			
Hearing Problems	Hearing Problems	Hearing Problems	Hearing Problems
Birth Weight (kg)	Long Standing Illness	Long Standing Illness	Long Standing Illness
Chest Infections	Hospitalised	Hospitalised	Hospitalised
Gestation Time	Headaches or Sickness	Headaches or Sickness	Headaches or Sickness
Asthma	Asthma	Medication	Medication
Special Care Unit	Speech/Language Problems	General Health	General Health
Feeding Problems	-	-	Autism/Asperger's
Growing Problems	-	-	Measles
Family Lifestyle Outcome Measures Only			
Weight at nine months (kg)	Maternal weight category	Maternal weight category	Maternal weight category
Maternal pre-pregnancy weight category	Paternal weight category	Paternal weight category	Paternal weight category
Father's Weight Category	More than three hours of TV/computer per day	Mother is a smoker	Mother is a smoker
Mother's Smoking Behaviour whilst pregnant	Regular meals	More than three hours of TV/computer per day	More than three hours of TV/computer per day
Planned pregnancy	-	Regular meals	Eats breakfast everyday
Breastfeeding behaviour	-	Times per week plays sport	Times per week plays sport
-	-	Goes to playground or park at least once a week	Goes to playground or park at least once a week
-	-	-	Unhealthy snacks between meals

Notes: Variable from or derived from Millennium Cohort Study.

Table IV-1 shows that maternal smoking is included as an outcome of both family lifestyle and child health in the second wave of the data. The reason for this is purely

statistical. Maternal smoking loaded onto both the lifestyle and health factors in the EFA in the data from this wave.

Subsequent Child Health Outcome Measures

Variables taken from subsequent waves of the MCS data are used to indicate child health as children get older. The outcome measures used to indicate underlying child health differ throughout childhood. As children get older their underlying health is identified more appropriately by different outcome measures. Childhood weight status is used as an outcome measure of underlying child health in each subsequent period. This is in addition to childhood weight status being used as an outcome measure of underlying family lifestyle in each period of the model. The child health outcome measures used to identify the underlying child health latent factor in each subsequent period of the model are also displayed in Table IV-1.

Binary variables indicating asthma, hearing problems, speech or language problems measles and diagnosed Autism, Asperger's Syndrome or other autistic spectrum disorder were included as outcome measures in at least one subsequent period of the model. Parents were also asked if their child had any longstanding illness. When their child was three they were asked *'Does <child's name> have long-term conditions that have been diagnosed by a health professional? By long-term I mean anything that <child's name> has had for at least 3 months or is expected to continue for at least the next 3 months?'* Similarly when their child was five and seven years old they were asked, *'Does <child's name> have any longstanding illnesses, disability or infirmity? By longstanding I mean anything that have troubled <child's name> for a period of time or is likely to affect <child's name> over a period of time.'* The answers to these questions were used to create further binary variables indicating whether or not the child had a longstanding illness at each age.

During each wave, parents were presented with this statement about their child: *'Often complains of headaches, stomach-aches or sickness'*. They were asked to choose form the following responses. *'Not true', 'Somewhat true', 'Certainly true'* or *'Can't say'*. A binary variable indicating whether this statement was somewhat or certainly true was generated.

In the third and fourth waves, mothers were asked '*Is <child's name> currently taking any medicines on a regular basis that were prescribed by a doctor or hospital? By medicines I mean any pills, syrups or other liquids, inhalers, patches, creams,*

suppositories or injections. By regular I mean every day for two weeks or more. Please don't include any 'over the counter' medicines.' A binary variable for each of these waves was generated to indicate whether the child was taking regular medication.

During each interview, mothers were also asked '*Since we saw you last, has <child's name> been admitted to hospital because of an illness or health problem apart from any hospital admissions you have not already told me about?*' Using the answers to these questions, ordinal variables were created for children at three, five and seven years old to indicate whether a child had never been hospitalised, hospitalised once or hospitalised more than once due to illness since the previous interview. Parent-assessed health was also included when children were five and seven years old. An ordinal variable was included to indicate whether the child's health was '*excellent*', '*very good*', '*good*', '*fair*' or '*poor*'.

4.4.2 Independent Variables

Table IV-2 shows which latent factors are influenced, in each wave, by which independent variables. The independent variables influencing underlying family lifestyle and underlying child health are represented by $\boldsymbol{X}$ in Equations (IV.5) and (IV.7) and $\boldsymbol{Z}$ in Equations (IV.6) and (IV.8), respectively.

The independent variables influencing each of the latent family lifestyle factors remain the same as those in the previous chapter and these variables are measured in the same way as described in Chapter III. In addition to these, SES also influences child health in the initial period. By allowing SES to influence child health, this model will make it possible to examine health inequalities in children from different social backgrounds. There is a large existing literature on health inequalities emphasising the importance and interest around this topic. However, there is a lack of empirical research into these health inequalities in relation to children, particularly during early childhood. The model will allow the effects of SES on child health to be investigated and enable policy makers to identify whether earlier childhood interventions are better in reducing health inequalities. Family structure and maternal education are not allowed to directly influence child health at any age. Instead they can indirectly influence child health through their effects on underlying family lifestyle. Of the families included in the final sample, 1,206 (13.4%) experienced at least one change in family structure and 3,213 (35.6) experienced at least one change in SES during the first four waves of the MCS. Only 622 (6.9%) of mothers in the sample gained additional education during the observation period.

As with the model in the previous chapter, it is acknowledged that there are time-varying parameters which influence family lifestyle and are not included in this study. The same bias in the persistence parameters as that discussed in the previous chapter could be caused by omitted time-varying independent variables but again this is not expected to be very problematic.

Table IV-2: Independent Determinants of Underlying Child Health

Initial Period	Age 3	Age 5	Age 7
Independent Variable Influencing Child Health (**Z** in Equations (IV.6) and (IV.8))			
High family SES	-	-	-
Low family SES	-	-	-
Independent Variable Influencing Family Lifestyle (**X** in Equations (IV.5) and (IV.7))			
Maternal education	-	-	-
High family SES	-	-	-
Low family SES	-	-	-
Single parent family	Single parent family	Single parent family	Single parent family

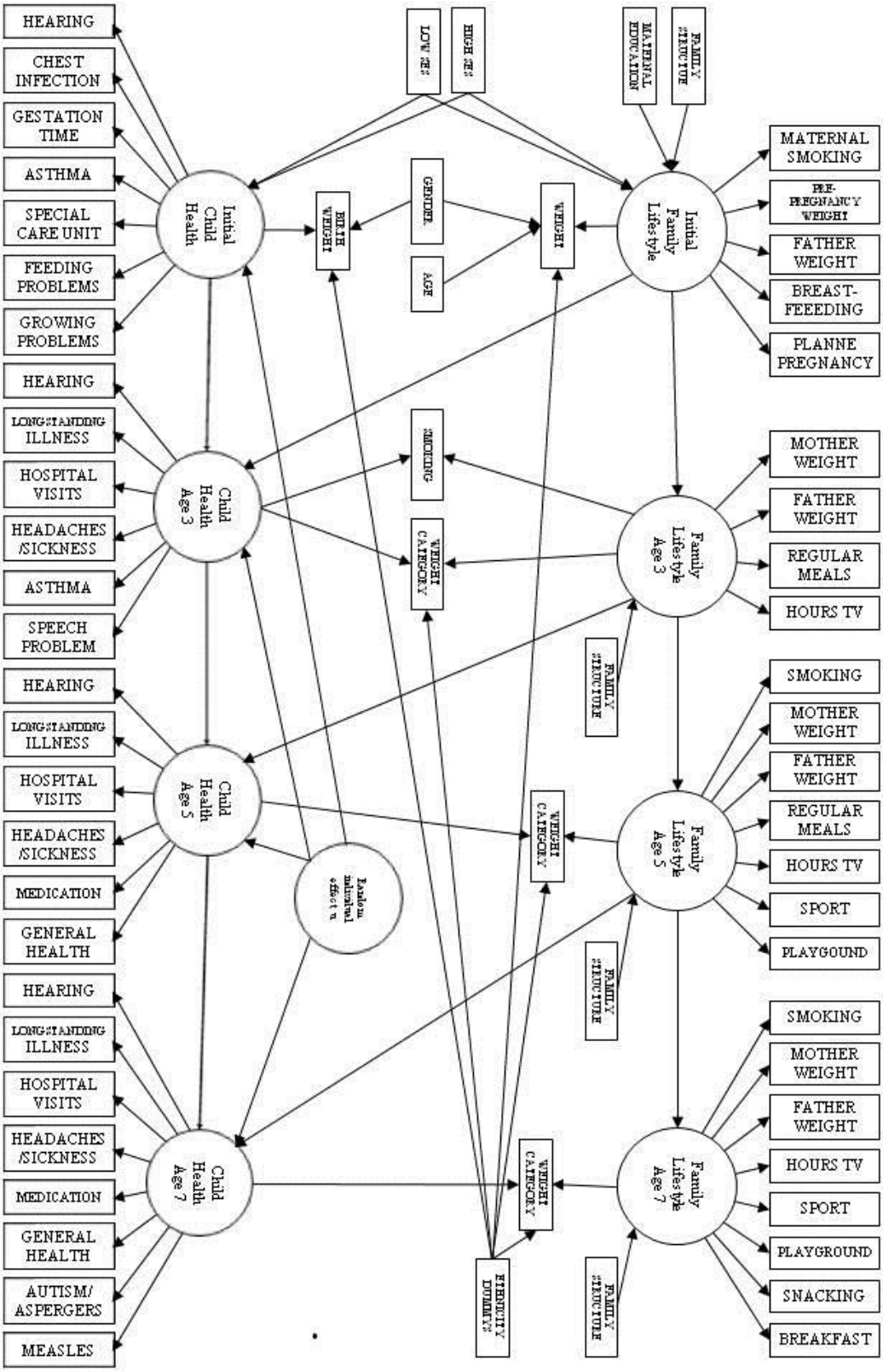
Notes: Variables from or derived from Millennium Cohort Study.

Independent variables which influence childhood adiposity are the same as those used in the previous chapter and are represented by $\boldsymbol{W}$ in Equation (IV.4). These were displayed in Table III-3 in Chapter III. Ethnicity, age and sex account for differences in the weight of a child during the initial period. Ethnicity is also a determinant of childhood weight status in each of the subsequent periods. As discussed in the previous chapter, age and sex are already accounted for in the childhood weight status definitions and so do not directly influence it in the model. Birth weight is included in this chapter as an outcome measure of initial child health and ethnicity and sex are also included as independent variable of birth weight[74].

A non-technical representation of the structural model is displayed in Figure IV-1. In line with standard practice, this path diagram uses rectangles to represent observable variables and ovals to represent latent variables. The directions of the arrows show the causal direction of any effects.

[74] Age is not included here because there is no variation in age.

Figure IV-1: Path Diagram



4.4.3 Missing and Excluded Observations

The observations with missing values of independent variables were the same as those in the previous chapter. A total of 1,770 observations were dropped due to missing independent variables and these are assumed to be missing at random. Observations which were excluded from analysis in the previous chapter were also removed from the analysis in this chapter, for the same reasons. The only exception is that no children were excluded as a result of spending time in a SCU. This is because, although having a child in a SCU might dramatically change a family's lifestyle, being in a SCU immediately after birth is an important indicator of underlying health at birth. This means an additional 552 observations are included. This leaves a balanced panel consisting of 9,014 observations followed over each period. Summary statistics for this sample can be found in Table C-1 in Appendix C.

4.5 Results

Two specifications of the model were implemented, one with all parameters fixed across time and another with some of these parameters allowed to vary over time. In the less restrictive model, all parameters are freed apart from the AR component for lifestyle, α which was found in the previous chapter to be persistent. Attempts were made to free this parameter but this prevented model convergence. The parameter estimates in the third and fourth waves of the data were also fixed to be time-invariant because these parameters were consistently very similar in the two waves. It is assumed that this was because children at the ages of five and seven years will be more alike than at the other ages considered in the model. These children are considered to be in a different stage of childhood than the younger children after they have started school. The stages of childhood used in this model are displayed in Table IV-3. This idea of stages of childhood was suggested by Heckman (2012) who described these stages as sensitivity periods. This allows the persistence parameters in the model to remain flexible and vary over time, while being easier to identify empirically due to the restriction of certain parameters.

Table IV-3: Stages of Childhood

Stage of Childhood	Age of Children	Wave of MCS
Stage 1: Birth/Infancy	9 Months	Wave 1
Stage 2: Early Childhood	3 Years	Wave 2
Stage 3: Early School	5 and 7 Years	Waves 3 and 4

As for the previous chapter, an additional model was run which allowed a delayed response of child weight status to result from changes in underlying family lifestyle and underlying child health. In this model, underlying family lifestyle which has already, by definition influenced maternal and paternal weight status, as well as other lifestyle behaviours takes time to influence child weight status. Similarly, the underlying child health which has already influenced all other child health outcome measures, takes longer to influence childhood weight status. The output from this model is displayed in Table C-2 in Appendix C which shows the Mplus output for the estimated parameters under this specification. The AIC and BIC of the original model and the model with a lagged effect on child weight status are shown in Table IV-4 along with their log-likelihood and degrees of freedom.

Table IV-4: Model Fit Statistics

	Model with contemporaneous effect on child weight	Model with lagged effect on child weight
Log-likelihood	**-207,669.924**	-207,674.936
Degrees of Freedom (df)	**143**	143
AIC	**415,625.847**	415,635.872
BIC	**416,642.509**	416,652.534
N	9,014	

Source: Millennium Cohort Study. Notes: Bold represents the specification of choice which provides the best fit to the data.

The AIC and BIC are both lowest in the model for which underlying family lifestyle and child health influence child weight status contemporaneously with other family members and other indicators of lifestyle and health. As in the previous chapter, this makes sense both conceptually and empirically. Underlying lifestyle causally influences childhood adiposity in the same time period as it has an influence on parental adiposity and other lifestyle indicators. Similarly, underlying child health causally influences childhood adiposity in the same period that it has an influence on other health indicators. The remainder of this chapter will focus on the results found using the model with contemporaneous causal effects.

As in the previous chapter, an individual random effect for family lifestyle was found to have no significant effect on underlying family lifestyle and was therefore not included in the final model. However, the individual random effect on child health, $\boldsymbol{\eta}$ in Equations (IV.6) and (IV.8), was found to have a significant influence on underlying child health and was included in the final version of the model. The fully-restricted model did not

converge making it impossible to compare the model fit using a LR test as in the previous chapter. The lack of convergence in the fully-restricted model supports the idea that there are different stages of childhood and that the parameters need to reflect these changes as children get older.

The remainder of this section is structured as follows. Section 4.5.1 displays the parameter estimates from the final dynamic factor model, including factor loadings, and AR components and standardised parameters. Section 4.5.2 discusses the factor scores and gives the likely characteristics of individuals and families for children with different levels of health. Section 4.5.3 explores some predicted adiposity outcome measures from the model using simulated data for children and families with different hypothetical characteristics.

4.5.1 Parameter Estimates

The estimated factor loadings for each of the underlying family lifestyle outcome measures are presented in Table C-3, in Appendix C. They are very similar in size, sign and significance to those in the previous chapter so are not included in the main text. They are estimated from a different, although very similar, sample. The estimated factor loadings for each of the outcome measures for underlying child health in each period are displayed in Table IV-5.

Again, it is not possible to compare these factor loadings across time periods or between outcome measures due to the different arbitrary scales of the latent factors caused by the method of identification. All factor loadings have the expected sign and all except one are statistically significant. Child weight status has an insignificant factor loading for the latent health factor at the age of three years. This suggests that, as expected at this age, lifestyle has more influence on weight status than health does. Despite this, health has a consistently negative coefficient in the childhood weight category equation, suggesting that improvement in childhood health might lead to a reduction in childhood obesity. For example, children with asthma might not always be able to exercise as often causing them to put on weight. In accordance with existing literature, birth weight is positively related to childhood health, healthier babies are born heavier. The estimates for the thresholds for ordinal outcome measures of family lifestyle and child health can be found in Table C-4 and Table C-5, respectively, in Appendix C.

Table IV-5: Estimated Factor Loadings for Child Health

	Factor Loading ζ, Equation (IV.4) (Standard Error)			
Dependent Variable	Initial	Age Three Years	Age Five Years	Age Seven Years
Birth Weight (kg)	0.040*** (0.009)	-	-	-
Weight Category	-	-0.071 (0.067)	-0.138** (0.060)	-0.138** (0.060)
Asthma	-5.366*** (1.288)	-3.005*** (0.396)	-	-
Hearing Problems	-0.760*** (0.155)	-1.185*** (0.128)	-0.900*** (0.056)	-0.900*** (0.056)
Gestational Length	0.092*** (0.019)	-	-	-
Chest Infection	-0.653*** (0.097)	-	-	-
Special Care Unit	-1.096*** (0.204)	-	-	-
Feeding Problems	-0.395** (0.169)	-	-	-
Growing Problems	-0.842*** (0.275)	-	-	-
Longstanding Illness	-	-2.143*** (0.183)	-4.958*** (0.276)	-4.958*** (0.276)
Hospitalised	-	-1.326*** (0.115)	-1.287*** (0.066)	-1.287*** (0.066)
Headaches/Sickness	-	-0.688*** (0.127)	-0.951*** (0.081)	-0.951*** (0.081)
Mother Smokes	-	-0.527*** (0.071)	-	-
Speech/Language Problems	-	-0.789*** (0.088)	-	-
Medication	-	-	-3.341*** (0.173)	-3.341*** (0.173)
Self-assessed Health	-	-	-2.270*** (0.094)	-2.270*** (0.094)
Autism	-	-	-	-1.413*** (0.116)
Measles	-	-	-	-0.184*** (0.062)

Source: Millennium Cohort Study. Factor loadings on child health taken from a dynamic latent factor model. Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Table IV-6 shows the estimated coefficients of the independent variables which influence latent family lifestyle, $\boldsymbol{\beta}$ in Equations (IV.5) and (IV.7), and those which influence child health, $\boldsymbol{\varrho}$ in Equations (IV.6) and (IV.8) both in the initial period and subsequent periods.

Table IV-6: Parameter Estimates of Independent Variables on Family Lifestyle and Child Health, $\boldsymbol{\beta}$ and $\boldsymbol{\varrho}$

	Coefficient (standard error)							
	$\boldsymbol{\beta}$, Equations (IV.5) and (IV.7)				$\boldsymbol{\varrho}$, Equations (IV.6) and (IV.8)			
Independent Variable	Initial Family Lifestyle	Family Lifestyle Age 3	Family Lifestyle Age 5	Family Lifestyle Age 7	Initial Child Health	Child Health Age 3	Child Health Age 5	Child Health Age 7
High SES	0.038*** (0.007)	-	-	-	-0.003 (0.012)	-	-	-
Low SES	-0.053*** (0.007)	-	-	-	-0.068*** (0.013)	-	-	-
Maternal Education at Birth	0.022*** (0.003)	-	-	-	-	-	-	-
Single Parent	-0.044*** (0.009)	0.009 (0.007)	-0.002 (0.003)		-	-	-	-

Source: Millennium Cohort Study. Notes: Independent variables influencing latent factors in a dynamic latent factor model. * p < 0.1, ** p < 0.05, *** p < 0.01.

The parameter estimates of the independent variables influencing latent family lifestyle are similar to those found in the previous chapter. Maternal education and family structure each have a significant influence on initial family lifestyle, with the expected sign. Although family structure changes over time for many of the cohort members, it does not have a significant influence on underlying family lifestyle after the initial period. Being from a family with low SES has a significant and negative influence on both child health and family lifestyle in the initial period. Although being from a family with high SES has a significant positive influence on family lifestyle, it does not appear to have a significant influence on child health. This could be because children from families with middle and high SES have similar levels of underlying initial health.

Table IV-7: Autoregressive Parameter Estimates

	Autoregressive Coefficients (standard error)		
	Family Lifestyle, $\boldsymbol{\theta}_t^L$	Child Health, $\boldsymbol{\theta}_t^H$	
		Age 3	Age 5 and 7
	$\boldsymbol{\alpha}$	$\boldsymbol{\varphi}$	
Previous Family Lifestyle, $\boldsymbol{\theta}_{t-1}^{L}$	1.115*** (0.011)	0.160*** (0.027)	0.040*** (0.014)
		$\boldsymbol{\gamma}$	
Previous Child Health, $\boldsymbol{\theta}_{t-1}^{H}$	-	1.218*** (0.069)	0.815*** (0.046)

Source: Millennium Cohort Study. Autoregressive coefficients from a dynamic latent factor model. Notes: * p < 0.1, ** p < 0.05, *** p < 0.01.

The AR parameters from the model are displayed in Table IV-7. The parameter estimates given here represent the scalar, α in Equation (IV.7) and the vectors of coefficients, $\boldsymbol{\gamma}$ and $\boldsymbol{\varphi}$ in Equation (IV.8).

The lagged effect of underlying family lifestyle is similar to that found in the previous chapter. Family lifestyle in the previous period appears to be a stronger indicator of current family lifestyle than any of the family background variables discussed above.

The effect of previous family lifestyle on child health is significant in all periods of the model and suggests that targeting family lifestyle early in childhood could have a large cumulative effect on child health. The most effective child health and obesity interventions should tackle family lifestyle in families with young children of all ages, particularly around during pregnancy and around birth. This is in line with the results in the previous chapter.

Previous child health has a significant and positive influence on current child health in all stages of the model, indicating that good health in the previous period is associated with good health in the current period. Any improvements made to child health from birth, or even during pregnancy, could improve the health of a child over many years. This might suggest that policy makers should target interventions at improving health at birth and the health of young babies in order to give children the best chance of a healthy childhood, potentially by targeting pregnant women and families before children are born.

Table IV-8 shows the estimated coefficients of the independent variables which influence adiposity measures throughout childhood. The parameter estimates given here represent the estimated coefficients $\boldsymbol{\delta}$ in Equation (IV.4).

Table IV-8: Parameter Estimates of Independent Variables Predicting Adiposity Measures, δ

	Coefficient, δ in Equation (IV.4) (Standard Error)			
Independent Variable	Birth Weight (kg)	Initial weight (kg) 9 Months	Weight Category 3 Years	Weight Category 5 and 7 Years
Male	0.012*** (0.001)	0.066*** (0.003)	-	-
Age (weeks)	-	0.004*** (0.001)	-	-
Black	-0.007* (0.004)	-0.010 (0.012)	0.163 (0.110)	0.317*** (0.092)
Asian	-0.032*** (0.002)	-0.073*** (0.007)	-0.233*** (0.079)	0.002 (0.070)
Other	-0.016*** (0.003)	-0.028*** (0.008)	-0.001 (0.090)	0.000 (0.087)

Source: Millennium Cohort Study. Independent variables coefficients influencing childhood adiposity variables in a dynamic latent factor model. Notes: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Male children are heavier at birth and at nine months old than their female counterparts, *ceteris paribus*. In accordance with the previous chapter, children who are older during the first MCS interviews weighed more, *ceteris paribus*. There was no significant difference between the adiposity of black and white children until they reached five years old, when it appears that black children started to put on weight faster than white children. Conversely, Asian children weighed less at birth and at nine months old than white children and were also more likely to be obese or overweight at the age of three years; this is consistent with results from the previous chapter and these differences between white and Asian children become insignificant as children get older.

The standardized parameters for the factor loadings are displayed in Table IV-9, for each of the latent child health factors. Underlying child health appears to have only a small influence on weight status which is only significant once a child reaches five years of age. Although small, this standardised coefficient does get larger and more statistically significant as children get older. This is as expected since, at birth, heavier babies are considered to be healthier.

Table IV-9: Standardised Factor Loadings for Latent Child Health Factors

	Standardised Factor Loadings (Standard Error)			
Dependent Variable	Initial	Age 3 Years	Age 5 Years	Age 7 Years
Birth Weight (kg)	0.196*** (0.040)	-	-	-
Weight Category	-	-0.025 (0.024)	-0.049** (0.021)	-0.050** (0.022)
Asthma	-0.777*** (0.075)	-0.743*** (0.044)	-	-
Hearing Problems	-0.172*** (0.034)	-0.401*** (0.030)	-0.328*** (0.017)	-0.339*** (0.018)
Gestational Length	0.207*** (0.041)	-	-	-
Chest Infection	-0.148*** (0.021)	-	-	-
Special Care Unit	-0.244*** (0.041)	-	-	-
Feeding Problems	-0.090** (0.038)	-	-	-
Growing Problems	-0.190*** (0.060)	-	-	-
Longstanding Illness	-	-0.620*** (0.020)	-0.886*** (0.009)	-0.893*** (0.008)
Hospitalised	-	-0.440*** (0.019)	-0.445*** (0.016)	-0.457*** (0.015)
Headaches/Sickness	-	-0.246*** (0.040)	-0.345*** (0.024)	-0.355*** (0.025)
Mother Smokes	-	-0.187*** (0.020)	-	-
Speech/Language Problems	-	-0.279*** (0.023)	-	-
Medication	-	-	-0.790*** (0.011)	-0800*** (0.011)
Self-assessed Health	-	-	-0.659*** (0.010)	-0.672*** (0.010)
Autism	-	-	-	-0.492*** (0.029)
Measles	-	-	-	-0.073*** (0.024)

Source: Millennium Cohort Study. Notes: Standardised factor loadings on child health in a dynamic latent factor model. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

The standardised parameters for the AR processes of previous family lifestyle on current child health and family lifestyle are displayed in Table IV-10.

Table IV-10: Standardised AR Processes

	Standardised Autoregressive Coefficients (Standard Error)					
	Family Lifestyle, $\boldsymbol{\theta}_t^L$			Child Health, $\boldsymbol{\theta}_t^H$		
	Age 3	Age 5	Age 7	Age 3	Age 5	Age 7
		$\boldsymbol{\alpha}$			$\boldsymbol{\varphi}$	
Previous Family Lifestyle, $\boldsymbol{\theta}_{t-1}^{L}$	0.993*** (0.001)	0.994*** (0.000)	0.995*** (0.000)	0.100*** (0.017)	0.027*** (0.009)	0.029*** (0.010)
					$\boldsymbol{\gamma}$	
Previous Child Health, $\boldsymbol{\theta}_{t-1}^{H}$	-	-	-	0.758*** (0.011)	0.779*** (0.020)	0.788*** (0.029)

Source: Millennium Cohort Study. Notes: Standardised autoregressive coefficients from a dynamic latent factor model. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Child health, as well as family lifestyle, is persistent throughout childhood. Table IV-10 shows that child health is influenced by underlying family lifestyle during early childhood. The effect is small, compared to the effect of previous health, but highly significant. This is expected but although lifestyle interventions might not be the best way to improve general health in young children, any additional effects that interventions aiming to reduce obesity might have are still important. These effects could accumulate over time to give substantial long term health consequences resulting from family lifestyle. Any significant influence on child health, no matter how small, could increase in magnitude as the children get older and produce large differences by the time these children reach adulthood.

4.5.2 Factor Scores

The factor scores used in this chapter, described in Section 4.3.2, are discussed here. As in the previous chapter, the factor scores have no numerical meaning and cannot be compared. However, they can be used to rank children in terms of their family lifestyle and their health. Families with higher family lifestyle factors score have 'healthier lifestyle' than families with lower factor scores and children with higher child health factors are healthier than those with lower scores.

Table IV-11: Proportion of Families Remaining in Initial Child Health Percentile Group

	Proportion Remaining in Health Percentile		
Initial percentile	3 Years	5 Years	7 Years
$\geq 95^{th}$	49.83%	35.46%	27.10%
$\geq 90^{th}$	57.33%	44.15%	35.87%
$< 10^{th}$	60.00%	46.83%	38.34%
$< 5^{th}$	52.55%	38.20%	29.50%

Source: Millennium Cohort Study. Notes: Results taken from factor scores for latent health in a dynamic latent factor model.

Table IV-11 shows the proportion of children remaining in certain percentile groups when ranked in order of child health. For example, 27.10% of all children above the 95th percentile on the family lifestyle distribution in the initial period remain above the 95th percentile by the age of seven years. Similar to the model in the previous chapter, family lifestyle is found to be persistent over time. These figures suggest that childhood health is not as persistent as family lifestyle, possibly because there are more or larger shocks to health than there are to family lifestyle or because child health is developing whereas family lifestyle is already well established. There does not appear to be much difference in the intra-distributional dynamics of family health between those ranked in the higher percentiles of child health to those in the lower percentiles.

Table IV-12: Proportion of Families Remaining in Initial Child Health Percentile Group (2)

	Proportion Remaining in Health Percentile		
Initial percentile	3 Years	5 Years	7 Years
$\geq 75^{th}$	69.90%	59.84%	52.65%
Interquartile range	70.74%	62.23%	57.98%
$< 25^{th}$	70.98%	60.51%	53.74%

Source: Millennium Cohort Study. Notes: Results taken from factor scores for latent health in a dynamic latent factor model.

Table IV-12 shows the proportion of children remaining in the upper and lower quartiles and the inter-quartile range of the health distribution throughout childhood. Child health appears to be much more adaptable than family lifestyle with little difference in the mobility of child health in each of the quartiles. Of the children who start life in the inter-quartile range, they are almost equally as likely to move to the upper or lower quartiles by the age of seven; 20.84% move to the upper quartile and 21.18% to the lower quartile. Despite there being more movement around the relative distribution of child health compared to the movement of family lifestyle, those who start life with relatively poor health are more likely to remain in relatively poor health throughout childhood.

Table IV-13 shows the correlations between the factor scores across each period in the model. As found in the previous chapter, underlying family lifestyle is highly correlated over time. Child health appears to be less correlated over time, again suggesting that underlying child health is less persistent than underlying family lifestyle.

Table IV-13: Correlations between Factors Scores

Correlation	θ_0^H	θ_1^H	θ_2^H	θ_3^H	θ_0^L	θ_1^L	θ_2^L	θ_3^L
θ_0^H	1	-	-	-	-	-	-	-
θ_1^H	0.8315	1	-	-	-	-	-	-
θ_2^H	0.6967	0.8377	1	-	-	-	-	-
θ_3^H	0.5856	0.7040	0.8359	1	-	-	-	-
θ_0^L	0.3446	0.4056	0.3710	0.3464	1	-	-	-
θ_1^L	0.3445	0.4054	0.3709	0.3463	0.9994	1	-	-
θ_2^L	0.3446	0.4056	0.3709	0.3464	0.9995	0.9999	1	-
θ_3^L	0.3448	0.4056	0.3710	0.3464	0.9995	0.9998	0.9999	1

Source: Millennium Cohort Study. Notes: Results taken from factor scores for latent health in a dynamic latent factor model.

Underlying child health and underlying family lifestyle are positively correlated across all time periods suggesting that improvements in family lifestyle are associated with improvements in child health. These correlations between the different factors are not as strong as those within factors.

Table IV-14 shows some of the differences in characteristics between children in excellent health (above the 95th health percentile) and children in very poor relative health (below the 5th health percentile). Existing literature suggests that children who are heavier at birth are generally healthier but here there is little difference in birth weight between the healthiest and unhealthiest children. There is also very little difference between the weight of children at nine months old. By the age of three years, the healthiest five percentiles of children are less likely to be obese than the unhealthiest five percentiles. This suggests that unhealthy children put weight on more quickly between nine months and three years. This difference in the likelihood of obesity in healthy and unhealthy children continues to widen as children get older. Table IV-14 also shows that children in excellent health are much more likely to be from families with high SES and vice versa. Children with excellent health are also more likely to be from families with the healthiest lifestyles.

Table IV-14: Differences between Children with Excellent and Poor Health

	Initial Child Health Ranking	
Variable	Excellent Health	Poor Health
Percentage Male	51.01%	50.73%
Birth weight (kg)	3.9597	3.9483
(standard deviation)	(0.0257)	(0.0255)
Weight (kg)	9.8607	9.8568
(standard deviation)	(0.1417)	(0.1407)
Percentage Obese Age 3	4.22%	5.67%
Percentage Obese Age 5	3.38%	5.21%
Percentage Obese Age 7	3.21%	5.14%
High SES	61.91%	5.10%
Low SES	2.68%	92.78%
$\geq 95^{th}$ initial lifestyle percentile	7.87%	0.67%
$< 5^{th}$ initial lifestyle percentile	0.63%	11.18%

Source: Millennium Cohort Study. Notes: Results taken from factor scores for latent health in a dynamic latent factor model.

The differences in obesity prevalence between children in the top and bottom five percentiles of health rankings are not as large as the differences between the top and bottom five percent of the family lifestyle rankings. This suggests that family lifestyle has more influence over childhood adiposity than health does. However, in order to correctly compare the effects of the different factors on childhood adiposity, and the mechanisms through which they have an effect, simulations are needed to approximate the posterior distributions outlined in Equations (IV.16) to (IV.19).

4.5.3 Predictions

In line with Chapter III, simulations are used to approximate the conditional distributions from the model in order to avoid the complex computations in Equations (IV.16) to (IV.19). For cases which use the entire sample to estimate the expected effect on a random observation, 250 simulations are used. The model is also used to simulate outcomes for hypothetical children with specific independent characteristics, for which 100,000 simulations are used to obtain the expected outcomes. The characteristics of these hypothetical children can be found in Table IV-15.

Table IV-15: Independent Characteristics of Hypothetical Children

Child Number	Description	Sex	Ethnicity	Maternal Education	Family SES	Family Structure
1	Highly educated	male	white	**higher degree**	average	couple
2	Poorly educated	male	white	**compulsory only**	average	couple
3	Advantaged	female	white	**higher degree**	**high**	**couple**
4	Disadvantaged	female	white	**compulsory only**	**low**	**single**

Source: Variables taken from or derived from MCS.

Considering children from different family backgrounds makes it possible to investigate the different effects that health and lifestyle outcomes can have on different types of children.

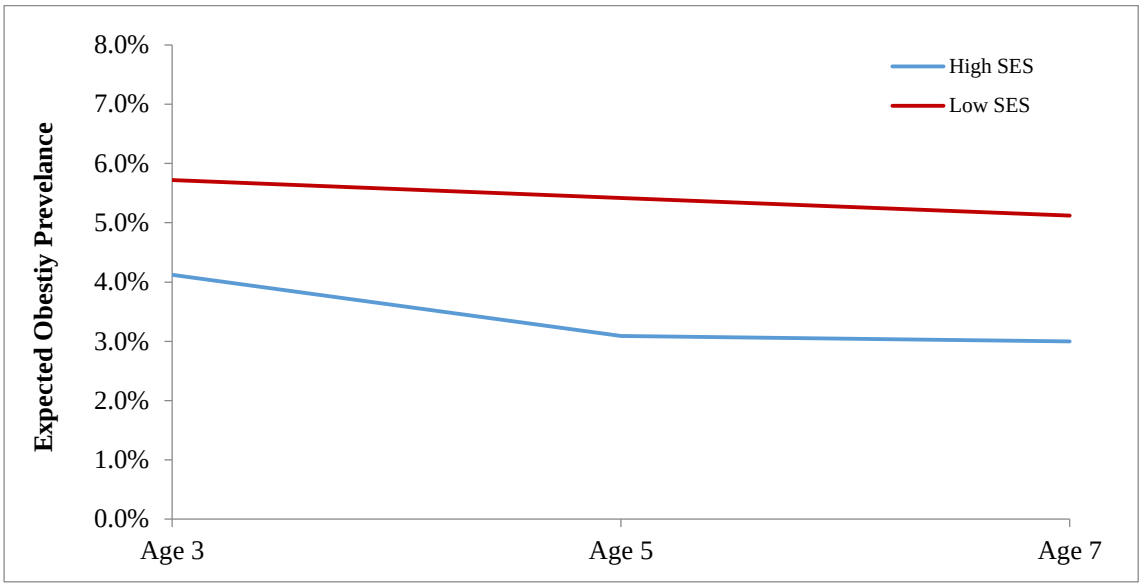
This section investigates how childhood health inequalities are associated with differences in childhood obesity prevalence. It then explores the correlations between birth weight, health and subsequent childhood obesity. Next, it investigates this relationship between family lifestyle, child health and childhood adiposity conditional on diet and physical activity. Finally, it explores the same relationships conditional on infant feeding and growth characteristics.

Health Inequalities and Childhood Obesity

The literature on health inequalities in adults is well established and there is a growing literature on childhood health inequalities. However, it remains unclear whether differences in obesity prevalence between children from difference socioeconomic backgrounds can be explained by health inequalities.

Figure IV-2 shows the expected prevalence of obesity during early childhood in children from high and low SES. Children from families with low SES are at greater risk of obesity throughout childhood, however, the risk of obesity in these children appears to be decreasing as they get older.

Figure IV-2: Expected Childhood Obesity and Socioeconomic Status



Source: Millennium Cohort Study.

The figure shows a clear association between SES and childhood obesity, suggesting that health inequalities might be observed through differences in obesity prevalence. Table IV-16 shows the expected health and lifestyle percentiles conditional on being from families with high and low SES at birth. They give an indication of the health and lifestyle of these children in relation to the rest of the sample.

Table IV-16: SES and Expected Factor Percentiles

	Health Percentiles		Lifestyle Percentiles	
	High SES	Low SES	High SES	Low SES
3 Years	62.43	31.74	71.64	26.85
5 Years	61.04	34.21	71.64	26.85
7 Years	59.82	36.24	71.64	26.84

Source: Millennium Cohort Study. Notes: Results taken from factor scores in a dynamic latent factor model.

The differences in obesity prevalence between children from families with different socioeconomic backgrounds are also reflected in the differences in where they lie on the health and lifestyle distributions. The difference in health percentiles between children from families with high and low SES widens as they get older. This suggests that health inequalities worsen throughout childhood and could lead to greater disparities in obesity prevalence as children approach adulthood. Figure C-1, in Appendix C, shows the kernel density distributions of underlying child health, at the age of seven years, in children from families with high and low SES. The spread of the distributions is very similar. The differences in lifestyle percentiles, although larger than the differences in health percentiles, do not widen over time. This is consistent with the findings from the previous

chapter which showed that lifestyle was persistent over time and that families tend not to move up or down the underlying lifestyle distribution.

The differences shown here between children from different socioeconomic backgrounds do not account for any other independent characteristics. These results represent health inequalities over the sample used to estimate the model in order to show the extent of health inequalities between difference socioeconomic groups. The causes of the health and obesity inequalities shown here are unknown and could be confounded by variables such as maternal education or family structure.

Birth Weight and Child Health

Many studies, such as Currie (2011) and Hobcraft & Kiernan (2010) used birth weight as a proxy for health; higher birth weight has been repeatedly found to be positively correlated with being healthier at birth. Table IV-17 shows the correlations between birth weight and the factor score for health in each wave of the MCS analysed in this study.

Table IV-17: Correlation between Birth Weight and Child Health

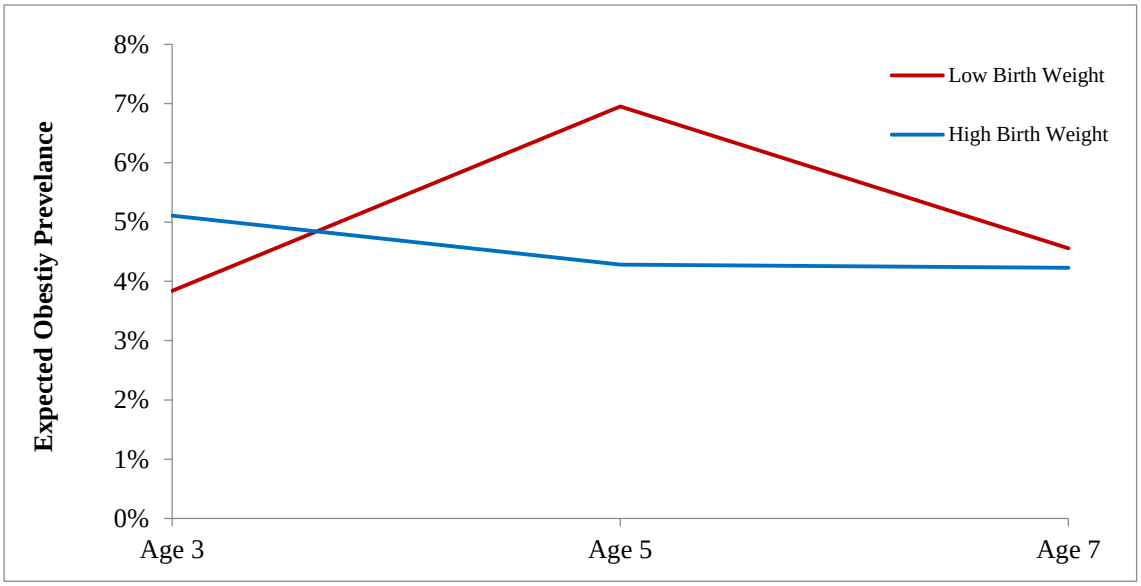
	Factor Score for Health			
Age of Child	9 Months	3 Years	5 Years	7 Years
Correlation[¥] with birth weight	0.1126^{***}	0.0967^{***}	0.0819^{***}	0.0693^{***}

Source: Millennium Cohort Study. Notes: Correlations taken from a dynamic latent factor model. * p < 0.1, ** p < 0.05, *** p < 0.01. [¥] Pearson's correlation coefficient.

The results show a positive and significant correlation between birth weight and health at birth (using the factor score). This gives further evidence that low birth weight is associated with poorer health at birth. The correlation remains significant between birth weight and childhood health as children get older but the correlation becomes weaker over time. This supports the use of latent health factors in which a number of different health outcomes can be used to measure health. The positive correlation was expected due to the persistence of health in the model, represented by the AR component, $\boldsymbol{\gamma}$ in Equation (IV.8), and provides further support for interventions to be aimed at children as young as possible.

Figure IV-3 shows the expected obesity prevalence throughout early childhood for children with low and high birth weights where high and low birth weight are defined as one standard deviations above or below the mean, respectively[75].

Figure IV-3: Expected Birth Weight and Subsequent Childhood Obesity



Source: Millennium Cohort Study.

Children with a lower birth weight are at a lesser risk of obesity at the age of three years. However, at five years, around the time of their adiposity rebound, their risk of obesity is much greater than children with a high birth weight. This could be because they experience their adiposity rebound earlier than other children. This phenomenon was found by Cole *et al*. (1995) and Whitaker *et al*. (1998) to be an indicator of obesity in later childhood and into adulthood. By the age of seven years, the difference in obesity prevalence between those with high and low birth weights has decreased. This could be due to children with low birth weights experiencing later adiposity rebounds causing them to have a relatively lower BMI at the age of seven. Further research into the association between birth weight and adiposity later in childhood could determine long-lasting effects of birth weight.

[75] Low birth weight is less than 2.981 kg (17.13% of observations) and high birth weight is above 3.913 kg (15.46% of observations).

Table IV-18: Expected Health Percentiles in Children with differing Birth Weight

	Health Percentiles		
	Average birth weight	High birth weight	Low birth weight
9 months	51.89	53.32	38.23
3 Years	51.62	52.47	40.17
5 Years	51.35	52.07	42.62
7 Years	51.09	52.23	43.03

Source: Millennium Cohort Study. Notes: Results taken from factor scores in a dynamic latent factor model.

Table IV-18 shows the expected percentile on the underlying health distribution throughout early childhood of children born with an average, high and low birth weight. Children born with a low birth weight are expected to have a poorer underlying health throughout childhood compared to children with average or high birth weights. However, the difference in health between those with the highest and lowest birth weights decreases as children get older, suggesting that the association between birth weight and health diminishes with age or that there is a lot of intra-distributional mobility. This is similar to the findings shown in Table IV-17. Table IV-18 also shows that children with high birth weights are expected to be on a higher health percentile than those with average birth weight and this difference does not appear to dissipate over time. Figure C-2 displays the kernel densities of the underlying childhood health distributions in children with high and low birth weights at the age of seven years. Similar to those from different SES, the spread of these distributions are very similar.

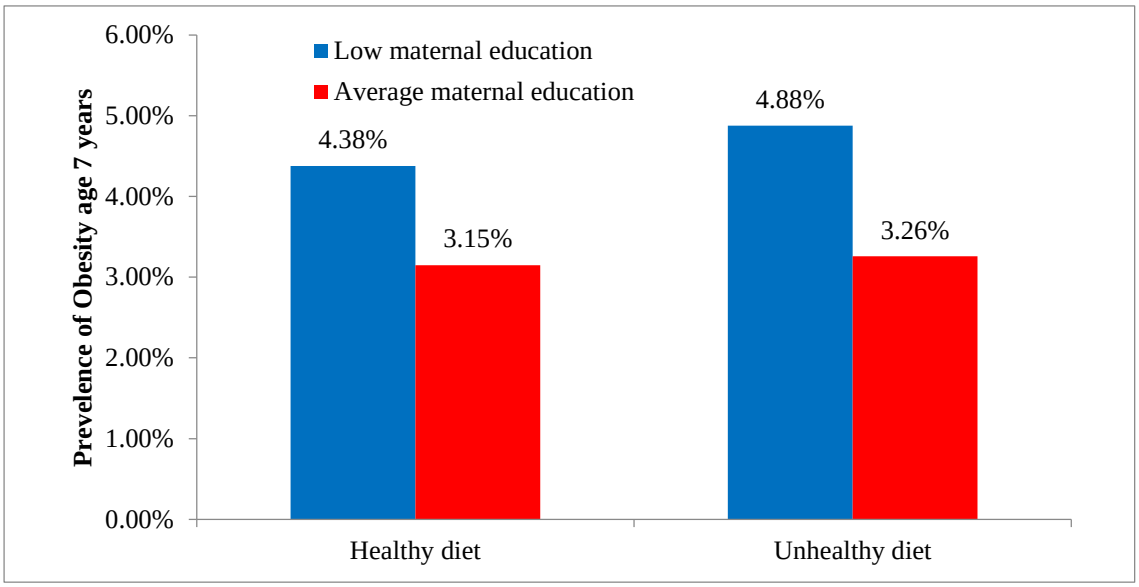
Diet and Physical Activity

Results from Chapter III showed that, although not to the same extent as social factors, diet and physical activity were associated with childhood obesity through the common effect of underlying family lifestyle. The model used in this chapter finds similar relationships between diet, physical activity and childhood obesity. In addition, using the model estimated in this chapter, it is possible to investigate how child health, as well as underlying family lifestyle, might be influencing this relationship.

Figure IV-4 shows the expected prevalence of obesity conditional on diet, in two hypothetical seven year old male children; one who has a highly educated mother and one with a poorly educated mother (child 1 and 2, respectively, in Table IV-15). Cribb *et al*. (2011) found that maternal education and children's diet were related and suggested that maternal education could influence child health through their diet. Healthy and unhealthy diets are defined in the same way as in Chapter III; a 'good diet' consists of regular meal

times regular breakfasts and no unhealthy snacking and a 'bad diet' includes unhealthy snacks and irregular meal times.

Figure IV-4: Expected Diet, Childhood Obesity and Maternal Education at 7 Years



Source: Millennium Cohort Study.

The influence of maternal education on the relationship between diet and obesity prevalence is similar to that of SES found in the previous chapter. Children with less educated mothers have a greater likelihood of being obese, particularly when their diet is poor. Having a poor diet appears to exacerbate inequalities in childhood obesity. This is unsurprising as less educated mothers are less likely to provide their children with a healthy diet[76]. The differences in obesity prevalence shown in Figure IV-4 show how inequalities in obesity prevalence are wider in children with unhealthy diets. This increased inequality might be wider still in children which are disadvantaged with respect to more than one family background variable.

Table IV-19 shows the percentile of the health distribution these children are expected to be on at the age of seven years. Amongst children with highly educated mothers, having an unhealthy diet appears to improve health. However, this unexpected result might be due to the very low numbers of children with highly educated mothers who have an unhealthy diet. This could also be what is causing the differing likelihood of obesity in children with highly educated mothers in Figure IV-4. Children with less educated

[76] High maternal education is not included in this graph because the number of highly educated mothers who provide their children with a healthy diet is so small and the number of simulations required to stabilise the numbers is too high.

mothers appear to benefit from a healthy diet, as expected. This implies that improving diets in children with less educated mothers could reduce health inequalities.

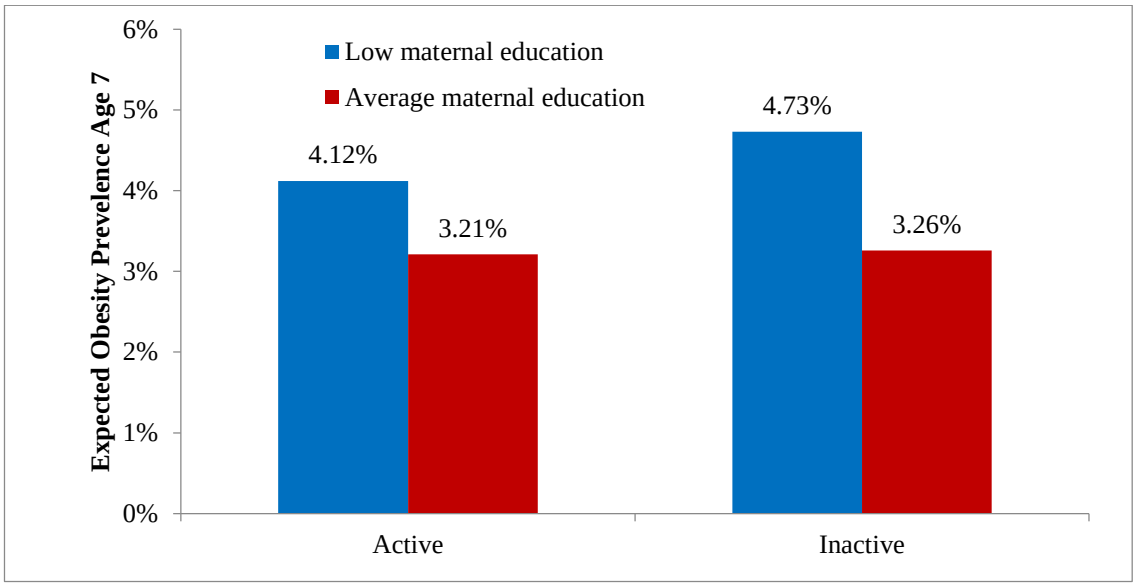
Table IV-19: Expected Health Percentiles by Child's Diet

	Health Percentiles (Age 7)	
	Average maternal education	Low maternal education
Healthy Diet	63.51	57.14
Unhealthy Diet	67.97	52.71

Source: Millennium Cohort Study. Notes: Results taken from factor scores in a dynamic latent factor model.

Figure IV-5 shows the relationship between childhood obesity prevalence, physical activity and maternal education. Active and inactive children are defined in the same way as in Chapter III, that is, an 'active child' participates in sport at least once a week, regularly visits the park or playground and has less than three hours screen time a day and an 'inactive child' never plays sport or visits the park and watches TV or plays computer games for at least three hours a day. Similar to diet, physical activity appears to be more associated with inequalities in obesity prevalence in children with less educated mothers, compared to those with mothers with average education.

Figure IV-5: Expected Physical Activity, Childhood Obesity and Maternal Education at 7 Years



Source: Millennium Cohort Study.

Table IV-20 shows that there is little difference in expected health percentiles between active and inactive children; this result is found across both high and low levels of maternal education. However, the differences in expected health percentile between children with mothers who have average and low education is larger.

Table IV-20: Expected Health Percentiles by Child's Physical Activity at 7 Years

	Health Percentiles	
	High maternal education	Low maternal education
Active	64.55	57.20
Inactive	63.45	57.69

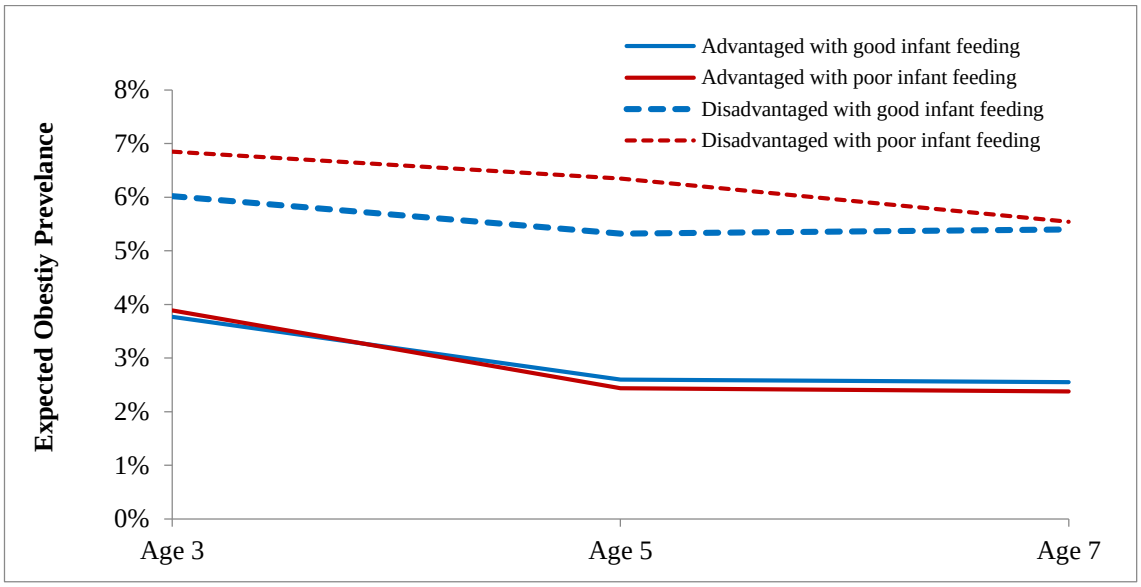
Source: Millennium Cohort Study. Notes: Results taken from factor scores in a dynamic latent factor model.

Similarly, the kernel density distributions displayed in Figure C-4, in Appendix C, show little difference between the distributions of underlying child health in active and inactive children.

Infant Feeding and Growth

Chapter II showed the importance of early life characteristics in determining adiposity in later childhood. This section investigates the relationship between early life feeding and growing variables and subsequent obesity. Here, a child with healthy infant feeding and growing characteristics is defined as one who was exclusively breastfed for at least four weeks and who did not suffer from feeding or growing problems during infancy. A child with unhealthy infant feeding is defined as one who suffered from feeding and/or growing problems and for whom breastfeeding was never initiated. WHO (2003) suggested that infant feeding, in particular breastfeeding, could help to reduce health inequalities during childhood. For this reason, the relationship between infant feeding characteristics and subsequent childhood health obesity is investigated for two hypothetical female children, one from an advantaged background and one from a disadvantaged background (child 3 and 4, respectively, in Table IV-15).

Figure IV-6: Expected Infant Feeding and Childhood Obesity in Advantaged and Disadvantaged Children



Source: Millennium Cohort Study.

Figure IV-6 shows the expected risk of obesity for the two hypothetical children outlined above conditional on infant feeding variables. It shows that for the advantaged child, infant feeding and growing has little association with obesity prevalence. Infant feeding appears to be more associated with adiposity in the disadvantaged child. Here, poor infant feeding and growing characteristics are associated with a higher likelihood of obesity, particularly at the ages of three and five years. This suggests that any improvements made to infant feeding and growing could help to reduce inequalities in childhood obesity (which is part of latent health). This supports the claim by WHO (2008) discussed earlier. That said, infant feeding appears to have little influence on obesity prevalence by the age of seven years after controlling for other background variables. This could be a sign that the effects of poor feeding and growing in infancy diminish throughout early childhood, or that the effects of diet later in childhood are more important and overtake the benefits of breastfeeding. As can be seen in previous results, the largest difference in the likelihood of obesity is between advantaged and disadvantaged children, rather than conditioning on outcome measures.

Table IV-21 and Table IV-22 show the health and lifestyle percentiles in the corresponding distributions, respectively, that the hypothetically advantaged and disadvantaged children are expected to be on at seven years of age, conditional on their infant feeding and growing variables.

Table IV-21: Expected Health Percentiles by Infant Feeding and Growing at 7 Years

	Health Percentiles	
	Advantaged	Disadvantaged
Healthy Infant Feeding/Growing	67.05	30.26
Unhealthy Infant Feeding/Growing	65.29	28.22

Source: Millennium Cohort Study. Notes: Results taken from factor scores in a dynamic latent factor model.

As expected, Table IV-21 shows the large health inequalities between advantaged and disadvantaged children. There is also a small difference in the health percentiles of children who experienced healthy and unhealthy infant growing and feeding. Children who had a healthy feeding and growing experience were generally two percentiles higher on the health distribution, regardless of their social and family background. Again, this suggests that infant feeding could help to reduce health inequalities in accordance with WHO (2008) if it is encouraged in disadvantaged families.

Table IV-22: Expected Infant Feeding and Lifestyle Percentiles at 7 Years

	Lifestyle Percentiles	
	Advantaged	Disadvantaged
Healthy Infant Feeding/Growing	89.02	15.21
Unhealthy Infant Feeding/Growing	87.36	13.75

Source: Millennium Cohort Study. Notes: Results taken from factor scores in a dynamic latent factor model.

Table IV-22 shows a similar pattern for the lifestyle distributions. There is an even wider gap between the expected percentiles of the lifestyle distribution for the advantaged and disadvantaged child than there was for the health distribution. Again, there is a small difference in the percentiles due to the infant feeding variables. Children with healthy infant feeding and growing are on a higher percentile of underlying family lifestyle. This difference appears to be less than two percentiles in both the advantaged and disadvantaged child.

These results suggest that the association between infant feeding and obesity, health or family lifestyle diminish during early childhood. Figure C-5 shows the kernel densities, for both health and lifestyle at the age of seven years, for all children in the sample who had a healthy or unhealthy infant feeding experience. The distributions look very similar in children with both types of infant feeding experience again suggesting that the effects of infant feeding on health and lifestyle, as well as obesity risk, diminish during early childhood.

4.6 Discussion

This chapter uses a dynamic latent factor model to investigate the relationship between underlying family lifestyle, underlying child health and childhood obesity, as well as a number of other health and lifestyle outcome measures. The model used in this chapter allows the identification of child health free of measurement error, as well as that of family lifestyle. The analysis in this chapter shows that there are a number of other health outcomes which are also correlated with underlying child health and in order to achieve a more inclusive measure of health, a wider range of outcome measures should be used. This allows a more general and inclusive definition of child health to be considered.

The additional complexity and extra parameters in this model do not change the finding that the evolution of family lifestyle is persistent. Again, this suggests that even small improvements to underlying family lifestyle could have important benefits to child health as the influences accumulate over time.

There is also a production function in health (Grossman, 1972) although this is not as strong as that of family lifestyle. This could be because family lifestyle is already well established when a child is born and that health is more responsive to external shocks. Childhood health is found to be persistent and the model suggests that child health, similar to family lifestyle, is at least partly determined before birth. This suggests that maternal health and lifestyle during pregnancy could have a large influence on the health of a child throughout childhood and even into adulthood. The AR processes for health, both from previous child health and previous family lifestyle were statistically significant throughout early childhood. Any improvements made to family lifestyle or child health could continue to have a significant influence on future child health and therefore on childhood obesity.

While the results in this chapter find that lifestyle still plays an important role in determining childhood obesity, family lifestyle also has a significant but relatively smaller influence on child health. Therefore, improving family lifestyle through interventions could both decrease obesity prevalence and improve child health. It is also important to condition on lifestyle in order to estimate the true causal effect of child health on childhood adiposity due to the correlation between child health and family lifestyle. This is because conditioning on family lifestyle deals with the endogeneity that it causes. After conditioning on family lifestyle, there remains a significant effect of child health on childhood obesity.

Similar to the previous chapter and existing studies, social determinants appear to be associated with childhood obesity. Childhood adiposity is more strongly associated with socioeconomic and family background characteristics than with other outcome measures of health and lifestyle, such as maternal lifestyle during pregnancy. Moreover, the mechanisms by which these social determinants influence childhood adiposity appear to be through their influence on underlying family lifestyle. Maternal education and family structure at birth have a significant influence on childhood obesity, through their impact on underlying family lifestyle. Similarly, family socioeconomic status influences childhood adiposity through its effects on both underlying family lifestyle and child health. This suggests that health and lifestyle inequalities at birth have lasting influences on childhood obesity prevalence. For example, birth weight is significantly, but weakly, correlated with health throughout childhood (see Table IV-17). This suggests, in line with the previous chapter, that lifestyle interventions should be implemented before and during pregnancy in order to improve child health as early as possible, as well as to reduce obesity prevalence and inequalities.

Inequalities are apparent in findings throughout this thesis, through differences in health and childhood obesity prevalence. Differences in obesity prevalence between children from disadvantaged or advantaged backgrounds, or from different socioeconomic or educational groups appear to get wider over time. There is not one obvious remedy to reduce these inequalities, but by tackling underlying family lifestyle through a number of interventions targeted at disadvantaged families and children could help to reduce these inequalities. Only by targeting families' understanding of why health and lifestyle behaviours are important will we see the cumulative effects which are needed to meaningfully reduce the health inequalities caused by early disadvantage. This supports results from the previous chapter which suggested that policies should teach mothers how to eat healthy meals and participate in more exercise and improve the lifestyles of her family, as well as providing help for families to enable them to do so and educating them so they understand why these changes are important.

Results from this chapter also show that diet and physical activity were positively associated with child obesity in children whose mothers are less educated. This suggests that interventions which improve the diet and activity levels of disadvantaged children, potentially by improving understanding lifestyle, will also improve child health and reduce the likelihood of obesity. Similar to the association between health and child weight status, the distribution of health was also associated with diet and physical activity.

This illustrates the fact that interventions which are successful in reducing childhood obesity and child health through improving underlying family lifestyle will also help to improve diet and physical activity, as well as the other outcome measures of both lifestyle and health.

Infant feeding and growing appears to have an influence on both underlying child health and underlying family lifestyle in both advantaged and disadvantaged children. However, infant feeding does not appear to have the same influence on childhood obesity prevalence in advantaged and disadvantaged children. Strong infant feeding and growing appears to reduce obesity prevalence to a greater extent in disadvantaged children, suggesting that helping mothers with breastfeeding and preventing feeding problems in their infants could help to reduce inequalities in obesity prevalence.

4.6.1 Policy Implications

The policy implications from this study relate to interventions which aim to reduce childhood obesity prevalence through improvements to family lifestyle and child health. For example, interventions such as Change4Life and Sure Start which aim to improve families' understanding of why lifestyle is important and how it has real influences on child health are expected to be successful. The underlying family lifestyle and child health factors are unobservable but they are identified by this model and it is these underlying factors which policy makers should focus on. They are themselves independent variables in the equations predicting the outcome measures and it is the underlying concepts identified by the model which this chapter suggests that policies and interventions should tackle.

As in the previous chapter, the complex dynamic model used in this study has the potential to contribute evidence to a variety of public health policies in more than one way. Each of the parameter estimates from the model could be used individually to influence committees of experts or decision makers, for example, those who develop NICE guidance. In addition, one or more of the multiple parameter estimates identified by the model could be used in economic models of obesity, lifestyle or health, to provide more comprehensive, long-term evidence on potential interventions. By estimating the same outcome measures of lifestyle and health over a period of time using longitudinal data, this study provides more long-term evidence than existing studies in this literature and could lead to stronger public health guidance. The same as the model in the previous chapter, this type of dynamic model is essential in providing this long-term evidence

whilst reducing the number of assumptions and extrapolations in economic models. This type of dynamic structural model allows estimated treatment effects to vary for different types of children or families as opposed to estimating a single average treatment effect for an entire population. In doing so, this model, in conjunction with economic models, could allow a range of policy questions to be answered using robust evidence from a single econometric model.

The predictions illustrated in this chapter, using parameter estimates from the structural model show a small example of what the model can be used for. There are a vast number of other predictions which could have been demonstrated in this thesis, both relating to childhood adiposity and relating to other lifestyle and health outcomes and the model has the ability to provide long-term evidence for an countless number of public health debates, not only the childhood obesity epidemic.

In addition to the contribution that the model estimated in this chapter could make to economic or cost-effectiveness models, the parameter estimates can themselves provide information about how and by which mechanisms, interventions might have an effect. For example, policies which are successful in improving underlying family lifestyle will reduce childhood obesity both directly and through improving child health. Families rarely move up or down the lifestyle distribution suggesting that interventions will have to be substantial but that successful ones are likely to have long-last influences on health or adiposity. The distribution of child health, although also persistent, appears to be more amenable to policy interventions. Policies which improve underlying child health might also inadvertently produce a reduction in childhood obesity and *vice versa*. Despite the fact that child health is more fluid than family lifestyle, there are still inequalities in health and obesity prevalence between advantaged and disadvantaged children. By targeting children who are more likely to be obese or in poor health, along with their families, lifestyle interventions might help to reduce these inequalities.

4.6.2 Limitations and Future Research

This chapter investigated the infleunce of family lifestyle on child health. However, it is possible that there is cross state dependence and that child health could have some influence on family lifestyle, particularly in families with children who have illnesses which limit their every day activities. Further research into the influence of child health on family lifestyle could help to estimate or rule out the possibility of this cross state dependence. Within this chapter, an attempt was made to estimate a causal effect of child

health on family lifestyle but the model would not converge with this additional effect. Future research with larger datasets might shed more light on the potential relationship between child health and future family lifestyle and might be more able any cross state dependence. However, if child health were to suffer a negative shock which changed family lifestyle then one would expect this effect to be apparent immediately and a contemperaneous correlation between the error terms in the family lifestyle and child health equations was insignificant. This suggests that the majority of the relationship between health and lifestyle was already accounted for.

The persistence shown by the AR process for the child health factors suggests that child health is at least partly determined during pregnancy. Further research into whether the persistence of health remains throughout childhood and into adolescence and adulthood could inform policy makers hoping to improve health in later years. Similarly, further research into maternal, and possibly paternal, health before the birth of a child and particularly during and immediately before pregnancy, could help policy makers to understand which aspects of parental health are more or less likely to be passed on to children through generations.

Additional research using this model could be carried out. An economic model for childhood obesity could also extend this research providing further evidence for guidance providers.

Further research into which outcome measures best represent underlying health at different stages of childhood, particularly later in childhood, might benefit any future analysis using techniques similar to those used in this study. Research using different datasets which hold information on different types of childhood illnesses could allow a more thorough investigation into how childhood health is observed and how it can be best measured.

Similar to the limitations of the MCS discussed in the previous empirical chapter, the model in this study is limited by the frequency of waves available in the MCS. Underlying health might take longer to influence some outcome measures than it does to influence others and the MCS restricted the observations of health outcomes to every two years. The results from this study, as in the previous study suggest that attitudes towards a healthy lifestyle are important and that this is what interventions should focus on. For this reason, any additional information on attitudinal variables might have been useful. For example questions on whether mothers thought that it was important to provide a

healthy lifestyle for their child could have helped inform the analysis. In addition, the MCS is relatively reliant on mothers to answer the majority of questions in the dataset about their child. Administrative data on birth weights and adiposity variables in each period could have provided more objective or reliable measures.

Another potentially limiting factor is that the MCS has no available data on adult outcomes, something which future research could consider. If these adult outcomes were available then the underlying factors could be anchored to them using an alternative method of identification seen in Cunha *et al*. (2010). This means that the factor scores have no numerical interpretation and percentiles of the distribution of each factor have to be used. This does not affect how the simulations from the model work and the dynamic latent factor model still provides a large amount of useful information.

V. DISCUSSION

This chapter discusses the key findings of the empirical analysis presented in Chapters II, III and IV and concludes the thesis. Section 5.1 summarises the aims, data, methodology of the thesis and its findings. Section 0 discusses possible policy implications arising from the thesis, who should be targeted and who should benefit most from any potential interventions. Section 5.3 identifies areas of possible future research and Section 5.4 concludes the thesis.

5.1 Summary

This thesis aimed to provide a better understanding of the early life causes of childhood obesity in the UK. Specifically, it investigated the causes of childhood adiposity from three perspectives. First, it investigated the influences of breastfeeding on adiposity during early childhood in order to inform policy makers aiming to prevent childhood obesity at an early age. Second, it investigated how underlying family lifestyle is related to childhood obesity over time and aimed to identify the most appropriate types of lifestyle interventions. Finally, the thesis built directly on work from the second empirical chapter and introduced underlying child health to the model in order to determine the relationships between underlying family lifestyle, childhood adiposity and health. By investigating these relationships and the mechanisms behind them, policy makers, schools

and families might benefit from a better understanding of how to reduce the risk of childhood obesity and overweight.

Chapter I gave an introduction to the thesis, provided definitions of different adiposity measures in adults and children and how and why they differ. It also outlined current obesity policies and described the data used throughout this thesis. Data from the Millennium Cohort Study was used throughout all empirical analysis in this thesis. This introductory chapter was followed by three empirical chapters, each of which contained a standalone econometric study and together illustrated a story of the early life causes of childhood obesity. This final chapter, Chapter V, provides an overall discussion and summary of the thesis.

The first empirical chapter, Chapter II, investigated the effects of breastfeeding behaviours on a number of different childhood adiposity measures using a range of econometric techniques. Initially, techniques which had previously been implemented in the existing literature were used. These included linear regression and logit models. In addition to these widely used techniques, a number of other techniques were also implemented in order to investigate the relationship under different assumptions. Ordered probit models were included in order to investigate both childhood overweight and obesity in a single dependent variable. Propensity score matching (PSM) allowed the relationship to be tested without imposing a functional form on the relationship between the outcome and the treatment, unlike many of the commonly used regression techniques. The functional form on the relationship is unknown and imposing the incorrect functional form can produce biased estimates. However, like the techniques commonly implemented in the literature, it assumed that selection into treatment did not depend on unobservable characteristics which were correlated with the outcome (childhood adiposity). Two additional methods were also carried out which accounted for the possible selection on unobservables which were correlated with childhood adiposity. These were an IV technique applied to the outcome equation and a structural model jointly estimating the outcome and the treatment equation (Roy model). The evidence suggested that the assumption of selection on observables was sufficient in this particular case. For this reason, the PSM results were used in the policy implication discussions. Results indicated that breastfeeding, particularly when prolonged and exclusive, could help to reduce childhood obesity. The effects of breastfeeding on childhood adiposity were significant but small, suggesting that breastfeeding should be included as part of a wider early-life approach to reducing childhood obesity. These results got modestly

larger and more significant as the children got older, suggesting that the effects might take time to become apparent.

Chapter III investigated the causal relationship of underlying family lifestyle on childhood weight status whilst accounting for social characteristics such as SES and maternal education. This followed on from the findings of the previous chapter which suggested that additional factors potentially influenced obesity as children grew up. The econometric model estimated in this chapter identified a latent factor representing underlying family lifestyle in each wave of the MCS using a range of observable outcome measures. This built on work by Balia & Jones (2008) and allowed the identification of underlying family lifestyle, free from measurement error. The outcome measures used to measure family lifestyle included observable lifestyle behaviours and outcomes of children as well as their parents and these changed with the age of the child. Using a latent factor in this way allowed a variety of outcome measures to be used, rather than relying on a single-item measure as much of the existing literature had done previously. These outcome measures included childhood weight status (the outcome of interest), as well as parental weight statuses, allowing the adiposity of different family members to be influenced by underlying family lifestyle. The result was the estimation of a structural model to form a dynamic process of underlying family lifestyle. This dynamic latent factor model was then used to estimate probabilistic outcomes for children and families with different sets of characteristics. Results showed that family lifestyle was persistent over time and suggested that targeting family lifestyle before the birth of a child could be most effective due to the lack of mobility around the distribution of family lifestyle.

The final empirical chapter, Chapter IV extended the model used in the previous empirical chapter by introducing child health in each period, as an additional dynamic process. Child health was estimated using a further latent factor identified in each time period as suggested by Heckman (2012). This allowed the identification of underlying child health as well as family lifestyle while removing measurement error from both. Underlying family lifestyle was again persistent and the extra parameters added in this chapter did not significantly change the results. Underlying child health was also found to be persistent, in accordance with the health production function outlined by Grossman (1972). However, this dynamic process is not as strong as that of family lifestyle. The important role that family lifestyle plays in determining childhood obesity is still apparent in this model but family lifestyle interventions also influence child health. Any cost-effectiveness or economic models investigating childhood obesity and family lifestyle

should consider these additional benefits. In addition, after conditioning on lifestyle, there remains a health effect on childhood obesity.

The results from Chapter IV suggested that even in disadvantaged children, the effects of infant feeding diminish as children reach the age of seven years, after the adiposity rebound. This is contradictory to the results from Chapter II which found that the effects of breastfeeding on childhood adiposity increase as children get older. This contradiction could be due to a number of reasons. First, Chapter IV, different groups of children are being compared and the composition of these groups changes over time. Chapter IV chapter identifies a different effect for a different group of observations compared to the methods used in Chapter II. Second, problems with infant feeding and growing are included in the analysis of infant feeding in Chapter IV, as well as breastfeeding behaviour and this difference in the 'treatment' variable could cause differences in results. Parents whose children have growing or feeding problems during infancy might overcompensate in later childhood by giving them more food. Chapter II concentrates on a different type of parameter to that in the other two empirical chapters. Chapter II focuses on results using an average treatment effect (ATE) for the entire sample. Chapters III and IV use more complex structural models which allow the ATE to be investigated for a number for parameters. In addition, the latter two studies allow the results to be easily simulated for different groups of children with different observable characteristics. This allows a more in depth investigation of inequalities to be carried out. This would not have been possible in Chapter II without estimating additional models. The parameter estimates which were identified in both the final models in Chapter III and IV were similar, indicating that the models were robust and well estimated. The predictions which came from each of the models were similar, also suggesting that the parameter estimates were reliable.

5.2 Policy Implications and the Public Health Approach

Each empirical chapter has its own policy implications which are summarised here but discussed in more detail in the individual chapters. The first empirical chapter in this thesis suggested that policies makers aiming to reduce childhood obesity should encourage breastfeeding as part of a wider strategy, encouraging a range of improvements in family lifestyle behaviours during infancy. The results from this chapter indicate that reductions in obesity prevalence and BMI were greatest when breastfeeding was

prolonged and exclusive. Although the effects of breastfeeding on childhood adiposity were found to be small, many were statistically significant. This provides support for the current WHO recommendation for six months of exclusive breastfeeding.

The second empirical chapter emphasised the importance of accounting for a range of family lifestyle behaviours when investigating childhood lifestyle and adiposity. Maternal weight status, in particular, had a strong association with underlying family lifestyle and was highly correlated with childhood weight status. Policy makers should target all members of a family to improve underlying family lifestyle in order to prevent children from becoming obese. This supports research by Brown & Roberts (2013) and Bauer *et al*. (2011) who also suggested that families rather than individuals should be targeted. In order to reduce inequalities in obesity prevalence, as well as decrease childhood obesity rates in the population, policies should focus on children from lower SES and disadvantaged backgrounds. Interventions such as Change4Life which target the family as a whole and focus on marketing campaigns and education should be continued. They should focus on education, in particular for disadvantaged mothers, on how to improve their lifestyles and lose and maintain weight. The most important point to take away from these results is that interventions should focus on attitudes and education rather than changing specific observable behaviours. By changing attitudes towards healthy lifestyles, a range of lifestyle behaviours should improve. However, this thesis does not aim to determine which interventions will most effectively change family lifestyle, only to establish the link between family lifestyle and childhood adiposity.

The results from the final empirical chapter showed that improved child health reduces childhood obesity. This relationship substantiated claims by Reilly *et al*. (2003) and Deckelbaum & Williams (2001) that childhood obesity was not merely a cosmetic problem and that childhood obesity could indicate poor health during childhood. Underlying family lifestyle influenced childhood adiposity, not only directly, but also through its effect on child health, suggesting that family lifestyle interventions could not only reduce childhood obesity but also improve underlying child health. Policy makers aiming to reduce childhood obesity should consider the additional benefits to child health that their policies might cause. These different effects are also important considerations for determining the cost-effectiveness of interventions. Any policies aiming to improve underlying child health should be implemented as early as possible in childhood due to the persistence of child health; however, any improvements to health later in childhood could still have beneficial effects on future health as well as obesity risk.

The analyses presented in this thesis indicate that interventions should begin as early as possible in childhood, even before and during pregnancy. However, improvements to lifestyle are likely to be beneficial at any stage during early childhood and should continue to be encouraged. A range of lifestyle behaviours should be addressed simultaneously by targeting the underlying family lifestyle in order to improve underlying family lifestyle for all family members. During and immediately before pregnancy, women should be encouraged to have a healthy lifestyle and pursue their best possible health. During infancy, breastfeeding should be encouraged, along with a number of other lifestyle behaviours connected with early life. Throughout childhood, the lifestyle of all family members, particularly the mother, should be targeted, these should include diet, physical activity and maintaining a healthy weight. At each stage of pregnancy and childhood, policy makers should aim to improve parental knowledge of the benefits of these lifestyle changes and help educate parents to understand the effects on obesity, child health and other outcomes that these changes could have for their family.

Particular attention should be given to disadvantaged children and their families, who are more at risk of obesity, unhealthy lifestyle and poor health. In targeting these individuals, inequalities in health and obesity could be reduced. For interventions to be successful they should be substantial. Policies should aim to improve lifestyle in several ways by providing help for families to enable them to make these changes as well as education on how these changes might improve their health or reduce obesity. Families with a deeper knowledge of, and better attitude towards, healthy lifestyles are more likely to be able to make changes to improve their lifestyle and in doing so reduce their risks of obesity and improve other lifestyle indicators. Family lifestyle is persistent and any policies aiming to change it should also be persistent and target families throughout childhood. Improvements in family lifestyle and child health can both reduce the likelihood of childhood obesity but due to their persistence, the full extent of the effects of any intervention is cumulative and the full effects might not be apparent until later in childhood and even adulthood.

In summary, the main policy advice which results from this thesis is summarised here.

- Prolonged and exclusive breastfeeding should be encouraged as part of a wider early life intervention which tackles obesity through a range of mechanisms.
- Lifestyle interventions which aim to reduce childhood obesity should be focussed on the entire family, not just the child.

- Families should be educated about how to improve their lifestyles as well as the benefits of doing so in relation to adiposity and child health.
- Families should receive help to enable them to better improve their lifestyle and their health.
- Family lifestyle interventions should begin as early as possible in childhood and continue throughout early childhood in order to have the greatest cumulative influence on child health and adiposity.
- Policy makers should focus their attention on disadvantaged children and families.
- Policy makers should consider the wider benefits to other outcome measures and to child health when aiming to reduce childhood obesity through family lifestyle interventions.

As well as the policy implications outlined above, the empirical studies within this thesis could provide valuable information for future economic or cost-effectiveness models. The parameter estimates found in each of the studies could be used in economic models for obesity, breastfeeding and other lifestyle and child health outcome measures. In particular, the parameter estimates from the second and third empirical studies which used structural models can provide valuable long-term evidence for economic models which require less assumptions to be made. The fact that these models estimate a system of equations jointly means that less assumptions about the correlations between these equations because they are already estimated by the econometric model. Structural models also give a more comprehensive picture of the links between the different variables and concepts. This has important policy implications because any guidance developed as a result of these economic models will be based on more robust and more long-term evidence.

5.3 Future Research

This thesis presented the applications of a range of econometric techniques to investigate childhood adiposity, family lifestyle and child health outcomes. There is a lack of econometric evidence covering childhood obesity and further use of econometric methods applied to large nationally representative datasets could be useful in helping policy

makers to target interventions at appropriate children and families. This could help to reduce childhood obesity prevalence and inequality in a more efficient and effective way.

Similar research to that carried out in this thesis, using longer observation periods covering later childhood, adolescence and into adulthood could produce important findings. This could help to determine whether or not the persistence of lifestyle remains as children grow up and leave their family home, whether adult health is determined during childhood and whether policies aimed at reducing childhood obesity are expected to continue to have an effect later in life. The most recent wave of the MCS data contains information on the cohort at eleven years of age and could be used to extend the analysis presented in this thesis. Data on the Millennium Cohort sample at age fourteen is also expected to be released in 2016. Investigating how school environments and more independence outside the family home influences childhood obesity and lifestyle choices could provide interesting policy implications. In addition, other large panel or cohort datasets hold information on participants from childhood into adulthood and could enable these relationships to be investigated over longer periods of time. This could allow the investigation of when childhood lifestyle and adiposity outcomes become independent of family outcomes.

Further research into the social determinants of lifestyle could also be of interest to policy makers. All three empirical chapters in this thesis showed that SES and family background characteristics influenced childhood obesity through family lifestyle behaviours and child health. Investigation into a wider range of socioeconomic and family background characteristics could help policy makers target interventions more effectively.

This thesis has demonstrated the need for additional research into childhood obesity definitions, particularly in very early childhood. More consistent definitions of childhood obesity and overweight would be beneficial to childhood obesity researchers as well as policy makers and medical professionals. More could be done to improve the understanding of why adult obesity and overweight definitions are impractical for use in childhood research.

Additional research into the differences in obesity prevalence in children of different ethnicities and between male and female children could help policy makers to target polices at the most appropriate children. Recent NICE (2013) guidance has discussed the differences in obesity between adults of different ethnicities and research into the

differences between ethnic groups during childhood could help to inform similar NICE guidance for children. Research into both the different adiposity outcomes between ethnic groups, as well as the reasons for these differences could be informative.

Research into how childhood obesity policies should be implemented could be useful. The need to reduce childhood obesity is well established and policy makers have been aiming to reduce childhood obesity for some time. However, it remains difficult to bring about change in family lifestyle as was shown by the persistent nature of family lifestyle found in Chapters III and IV. More research into how to make interventions effective could provide valuable evidence for policy makers. Additionally, research into the effectiveness of interventions that have already been implanted could provide essential direction for future interventions.

5.4 Conclusion

Childhood obesity is a key concern in the UK, as it is in many developed countries and figures suggest that childhood obesity prevalence remains high. As a result, research into childhood obesity continues to be a top priority for researchers, policy makers and the UK government.

This thesis furthers the understanding of the causes of childhood obesity and how they develop during early childhood. It has highlighted that in order to reduce the childhood obesity and the inequalities in obesity prevalence between advantaged and disadvantaged children, policy makers cannot concentrate only on one intervention but must tackle several issues for children and their families as well as improving understanding and attitudes towards lifestyle. These inequalities widen as children get older implying that family lifestyle during early childhood and early disadvantage could have long-term effects on obesity and health. This makes it important to tackle obesity in disadvantaged children as young as possible. The results also emphasised that family lifestyle is an important determinant of childhood adiposity, not only directly but also indirectly through its effects on child health, even at a young age.

This thesis contributes to the public health debate around childhood obesity by building on the existing childhood obesity literature. It uses a range of econometric techniques which have not before been used in this context. Together the chapters of this thesis outline a range of policy implications aimed at reducing childhood obesity, suggest a

number of areas for future research and provide a range of parameter estimates for future use in economic or cost-effectiveness models illustrating how this econometric approach can be used in a variety of public health problems, including the childhood obesity epidemic.

REFERENCES

Abadie, A., & Imbens, G. W. (2008). On the Failure of the Bootstrap for Matching Estimators. *Econometrica*, *76*(6), 1537–1557.

Abadie, A., & Imbens, G. W. (2009). *Matching on the Estimated Propensity Score* (No. w15301). *National Bureau of Economic Research*. (No. w15301) National Bureau of Economic Research.

Adams, P., Hurd, M. D., McFadden, D., Merrill, A., & Ribeiro, T. (2003). Healthy, wealthy, and wise? Tests for direct causal paths between health and socioeconomic status. *Journal of Econometrics*, *112*(1), 3–56.

Ajslev, T. a, Andersen, C. S., Gamborg, M., Sørensen, T. I. a, & Jess, T. (2011). Childhood overweight after establishment of the gut microbiota: the role of delivery mode, pre-pregnancy weight and early administration of antibiotics. *International Journal of Obesity*, *35*, 522–529.

Almond, D., Chay, K. Y., & Lee, D. S. (2005). The Costs of Low Birth Weight. *The Quarterly Journal of Economics*, *120*(3), 1031–1083.

Almond, D., & Currie, J. (2011). Killing Me Softly: The Fetal Origins Hypothesis. *Journal of Economic Perspectives*, *25*(3), 153–172.

Amir, L. H., & Donath, S. M. (2007). A Systematic Review of Maternal Obesity and Breastfeeding Intention Initiation and Duration. *BMC Pregnancy and Childbirth*, *7*(9).

Amir, L. H., & Donath, S. M. (2008). Socioeconomic Status an rates of breastfeeding in Australia: evidence from three recent national health surveys. *Medical Journal of Australia*, *189*(5), 254–256.

Angrist J. D. and J. S. Pischke. (2008). *Mostly Harmless Econometrics : An Empiricist's Companion*. *Massachusettts Institute of Technology and The London school of Economics*.

Armstrong, J., & Reilly, J. J. (2002). Breastfeeding and Lowering the Risk of Childhood Obesity. *Lancet*, *359*, 2003–2004.

Ashenden, R., Silagy, C., & Weller, D. (1997). A Systematic Review of the effectiveness of Promoting Lifestyle Change in General Practice. *Family Practice*, *14*(2), 160–176.

Baird, J., Fisher, D., Lucas, P., Kleijnen, J., & Roberts, H. (2005). Being big or growing fast: systematic review of size and growth in infancy and later obesity. *British Medical Journal*, *331*(929), 4–9.

Balia, S., & Jones, A. M. (2008). Mortality, lifestyle and socio-economic status. *Journal of Health Economics*, *27*(1), 1–26.

Barker, D., & Osmond, C. (1987). Inequalities in Health in Britain: Specific Explanations in Three Lancashire Towns. *British Medical Journal*, *294*, 749–752.

Basu, A., Heckman, J. J., Navarro-Lozano, S., & Urzua, S. (2007). Use of Instrumental Variables inf the Presenece of Heterogeneity and Self-Selection: An Application to treatments of Breat Cancer Patients. *Health Economics*, *16*, 1133–1157.

Bates, M. J. (1989). The design of browsing and berrypicking techniques for the online search interface. *Online Review*, *13*(5), 407–424.

Bauer, K. W., Neumark-Sztainer, D., Fulkerson, J. a, Hannan, P. J., & Story, M. (2011). Familial correlates of adolescent girls' physical activity, television use, dietary intake, weight, and body composition. *The International Journal of Behavioral Nutrition and Physical Activity*, *8*(1), 25–34.

Baum, C., Schaffer, M. E., & Stillman, S. (2010). ivreg2: Stata module for extended instrumental variables/2SLS, GMM and AC/HAC, LIML and k-class regression.

Bergmann, K., Bergmann, R., Von Kries, R., Böhm, O., Richter, R., Dudenhausen, J. W., & Wahn, U. (2003). Early Determinants of Childhood Overweight and Adiposity in a Birth Cohort Study: Role of Breast-Feeding. *International Journal of Obesity*, *27*, 162–172.

Beyerlein, A., Toschke, A. M., & von Kries, R. (2008). Breastfeeding and Childhood Obesity: Shift of the Entire BMI Distribution or Only the Upper Parts? *Obesity*, *16*(12), 2730–2733.

Blake, K. V, Gurrin, L. C., Evans, S. F., Beilin, L. J., Landau, L. I., Stanley, F. J., & Newnham, J. P. (2000). Maternal cigarette smoking during pregnancy, low birth weight and subsequent blood pressure in early childhood. *Early Human Development*, *57*(2), 137–147.

Blaser, M. J. (2014). *Missing Microbes: How the Overuse of Antibiotics Is Fueling Our Modern Plagues*. United States: Henry Holt Company.

Blundell, R., & Costa Dias, M. (2009). Alternative Approaches to Evaluation in Empirical Microeconomics. *Journal of Human Resources*, *44*(3), 465–640.

Bogen, D. L., Hanusa, B. H., & Whitaker, R. C. (2004). The Effect of Breast-Feeding with and without Formula Use on the Risk of Obesity at 4 Years of Age. *Obesity Research*, *12*(9), 1527–1535.

Booth, A., Papaioannou, D., & Sutton, A. (2012). *Systematic Approaches to a Successful Literature Review*.

Boutelle, K. N., Cafri, G., & Crow, S. J. (2011). Parent-only treatment for childhood obesity: a randomized controlled trial. *Obesity*, *19*(3), 574–580.

Braveman, P. (2014). What is health equity: and how does a life-course approach take us further toward it? *Maternal and Child Health Journal*, *18*(2), 366–372.

Brennan, A., Purshouse, R., Taylor, K., & Rafia, R. (2008). *Independent Review of The Effects of Alcohol Pricing and Promotion Part B: Modelling the Potential Impact of Pricing and Promotion Policies for Alcohol in England*. *Report for the Department of Health*. Retrieved from http://apply.clearing.shef.ac.uk/polopoly_fs/1.95621!/file/PartB.pdf

Briggs, H. (2013). Rise in Child Obesity-Related Hospital Admissions. Retrieved June 10, 2014, from http://www.bbc.co.uk/news/health-22849112

Brion, M.-J. a, Lawlor, D. a, Matijasevich, A., Horta, B., Anselmi, L., Araujo, C. L., … Smith, G. D. (2011). What are the causal effects of breastfeeding on IQ, obesity and blood pressure? Evidence from comparing high-income with middle-income cohorts. *International Journal of Epidemiology*, *40*(3), 670–680.

Brown, H., Hole, A. R., & Roberts, J. (2013). Going the Same "Weigh": Spousal Correlations in Obestiy in the UK. *Applied Economics*, *46*(2), 153–166.

Brown, H., & Roberts, J. (2013). Born to be wide? Exploring correlations in mother and adolescent body mass index. *Economics Letters*, *120*(3), 413–415.

Brown, S., & Taylor, K. (2008). Bullying, education and earnings: Evidence from the National Child Development Study. *Economics of Education Review*, *27*(4), 387–401.

Burdette, H. L., & Whitaker, R. C. (2007). Differences by Race and Ethnicity in the Relationship between Breastfeeding and Obesity in Preschool Children. *Ethnicity and Disease*, *17*(3), 467–470.

Burke, V., Beilin, L. J., Simmer, K., Oddy, W. H., Blake, K. V, Doherty, D., … Stanley, F. J. (2005). Breastfeeding and Overweight: Longitudinal Analysis in an Australian Birth Cohort. *Journal of Pediatrics*, *147*(1), 56–61.

Byrne, B. M. (2012). The Full Latent Variable Model. In *Structural Equation Modeling with Mplus: Basic Concepts, Applications and Programming* (pp. 6–7). Hove, East Sussex: Taylor and Francis Group LLC.

Caliendo, M., & Kopeinig, S. (2008). Some Practical Guidance for the Implemenation of Propensity Score Matching. *Journal of Economic Surveys*, *22*(1), 31–72.

Cameron, A. C., & Travedi, P. K. (2005). *Microeconometrics: Methods and Applications*. New York: Cambridge University Press.

Carneiro, P., Hansen, K. T., & Heckman, J. J. (2003). Estimating Distributions of Treatment Effects with an Application to the Returns to Schooling. *International Economic Review*, *44*(2), 361–422.

Carnell, S., Edwards, C., Croker, H., Boniface, D., & Wardle, J. (2005). Parental perceptions of overweight in 3-5 y olds. *International Journal of Obesity*, *29*(4), 353–355.

Case, A., Lubotsky, D., & Paxson, C. (2002). Economic Status and Health in Childhood: The Origins of the Gradient. *The American Economic Review*, *92*(5), 1308–1334.

Case, A., & Paxson, C. (2008). Height, Health and Cognitive Function at Older Ages. *The American Economic Review*, *98*(2), 463–467.

Cavill, N., & Ells, L. (2010). *Treating adult obesity through lifestyle change interventions A briefing paper for commissioners*. *National Obesity Observatory*. Oxford.

Centers for Disease Control and Prevention (CDC), & National Center for Health Statstics (NCHS). (2001). Growth Charts - Data Table of BMI-for-age Charts. Retrieved October 16, 2012, from http://www.cdc.gov/growthcharts/html_charts/bmiagerev.htm

Cockerham, W. C., Lueschen, G., Kunz, G., & Spaeth, J. L. (1986). Social stratification and self-management of health. *Journal of Health and Social Behavior*, *27*(1), 1–14.

COI for the Department of Health. (2008). Health is Global: A UK Government Strategy 2008-13. Crown Copyright. Retrieved from www.dh.gov.uk/publications

Cole, T. J., Bellizzi, M. C., Flegal, K. M., & Dietz, W. H. (2000). Establishing a Standard Definition for Child Overweight and Obesity Worldwide: International Survey. *British Medical Journal*, *320*, 1240–1245.

Cole, T. J., Freeman, J. V, & Preece, M. A. (1995). Body mass index reference curves for the UK, 1990. *Archives of Disease in Childhood*, *73*(1), 25–29.

Commission on Social Determinants of Health. (2008). *Closing the gap in a generation: Health equity through action on the social determinants of health*. Geneva, World Health Organisation.

Conti, G., & Heckman, J. J. (2013). The Developmental Approach to Child and Adult Health. *Pediatrics*, *131*(supplement), s133–s141.

Conti, G., Heckman, J. J., & Urzua, S. (2010). *Early Endowment, Education and Health*.

Contoyannis, P., & Jones, A. M. (2004). Socio-economic Status, Health and Lifestyle. *Journal of Health Economics*, *23*(5), 965–995.

Contoyannis, P., Jones, A. M., & Rice, N. (2004a). Simulation-Based Inference in Dynamic Panel Probit Models: An Application to Health. *Empirical Economics*, *29*(1), 49–77.

Contoyannis, P., Jones, A. M., & Rice, N. (2004b). The dynamics of health in the British Household Panel Survey. *Journal of Applied Econometrics*, *19*(4), 473–503.

Contoyannis, P., & Li, J. (2011). The Evolution of Health Outcomes from Childhood to Adolescence. *Journal of Health Economics*, *30*(1), 11–32.

Coulter, A. (1987). Lifestyles and social class: implications for primary care. *The Journal of the Royal College of General Practitioners*, *37*(305), 533–536.

Cramer, J. S. (2005). *Omitted Variables and Mis-specified Disturbances in the Logit Model* (No. TI 2005-084/4). *Tinbergen Institute Discussion Paper* (Vol. TI 2005 -). (No. 05-084/4). Tinbergen Instituate Discussion Paper.

Cribb, V. L., Jones, L. R., Rogers, I. S., Ness, A. R., & Emmett, P. M. (2011). Is maternal education level associated with diet in 10-year-old children? *Public Health Nutrition*, *14*(11), 2037–2048.

Crosnoe, R. (2012). Obesity, family instability, and socioemotional health in adolescence. *Economics and Human Biology*, *10*(4), 375–384.

Cross-Government Obesity Unit; Department of Health and Department of Children Schools and Families. (2008). Healthy Weight, Healthy Lives: A Cross-Government Strategy for England. Crown Copyright.

Cunha, F., & Heckman, J. J. (2008). Formulating, Identifying and Estimating the Technology of Cognitive and Noncognitive Skill Formation. *Journal of Human Resources*, *43*(4), 738–782.

Cunha, F., & Heckman, J. J. (2009). The Economics and Psychology of Inequality and Human Development. *Journal of the European Economic Association*, *7*(2-3), 320–364.

Cunha, F., Heckman, J., & Schennach, S. (2010). Estimating the Technology of Cognitive and Noncognitive Skill Formation. *Econometrica : Journal of the Econometric Society*, *78*(3), 883–931.

Currie, J. (2011). *Inequality at Birth: Some Causes and Consequences* (No. w16798). (No. w16798) National Bureau of Economic Research.

Currie, J., & Moretti, E. (2003). Mother's education and the intergenerational

transmission of human capital: evidence from college openings. *The Quarterly Journal of Economics*, *118*(4), 1495–1532.

D'Agostino, R. B., & D'Agostino, R. B. (2007). Using observational data to estimate treatment effects. *JAMA: The Journal of the American Medical Association*, *297*(3), 314–316.

Daniels, S. R. (2006). The Consequences of Childhood Overweight and Obesity. *The Future of Children*, *16*(1), 47–67.

Deb, P., & Trivedi, P. K. (2006). Specification and simulated likelihood estimation of a non-normal treatment-outcome model with selection: Application to health care utilization. *Econometrics Journal*, *9*(2), 307–331.

Deckelbaum, R. J., & Williams, C. L. (2001). Childhood obesity: the health issue. *Obesity Research*, *9*(supplement 4), 239s–243s.

Dehejia, R. H., & Wahba, S. (2002). Propensity Score-Matching Methods for Non-experimental Causal Studies. *The Review of Economics and Statistics*, *84*(1), 151–161.

Del Bono, E., & Rabe, B. (2012). *Breastfeeding and child cognitive outcomes: Evidence from a hospital-based breastfeeding support policy* (No. 2012-29). *Institute for Social and Economic Research Working Paper Series*. Essex.

Denny, K., & Doyle, O. (2008). The Causal Effect of Breastfeeding on Children's Cognitive Development: A Quasi-Experimental Design. *Archives of General Psychiatry*, *65*(5), 578–584.

Dent, M., & Swanston, D. (2010). Briefing Note: Obesity and life expectancy. Retrieved November 7, 2011, from http://www.noo.org.uk/NOO_pub/briefing_papers

Department of Health. (2002). Infant Feeding Survey 2000. Retrieved March 20, 2013, from http://webarchive.nationalarchives.gov.uk/+/www.dh.gov.uk/en/Publicationsandstatistics/Pressreleases/DH_4013168

Department of Health. (2004). Spending Review 2004 Public Service Agreement. Retrieved from http://webarchive.nationalarchives.gov.uk/+/www.dh.gov.uk/en/Aboutus/HowDHworks/Servicestandardsandcommitments/DHPublicServiceAgreement/DH_4106188

Department of Health. (2010). Obesity - NHS Choices. Retrieved November 7, 2011, from http://www.nhs.uk/Conditions/Obesity/Pages/Introduction.aspx

Dewey, K. G. (2003). Is Breastfeeding Protective Against Child Obesity? *Journal of Human Lactation*, *19*(1), 9–18.

Dewey, K., Heinig, M., & Nommsen, L. (1993). Maternal Weight-loss Patterns During Prolonged Lactation. *The American Journal of Clinical Nutrition*, *58*(2), 162–166.

DH. (2009). *Change4Life Marketing Strategy. In Support of ealthy Weight, Healthy Lives*. London.

Duflo, E., Glennerster, R., & Kremer, M. (2007). Using Randomization in Development Economics Research: A Toolkit. *Handbook of Development Economics*, *4*, 3895–3962.

Dyson, L., Renfrew, M., Mcfadden, A., Herbert, G., & Thomas, J. (2005). *Promotion of breastfeeding initiation and duration: Evidence into practice briefing*. Retrieved from http://www.nice.org.uk/niceMedia/pdf/EAB_Breastfeeding_final_version.pdf

Ermisch, J. (2008). Origins of Social Immobility and Inequality: Parenting and Early Child Development. *National Institute Economic Review*, *205*, 62–71.

Faria, R., Hernandez Alava, M., Manca, A., & Wailoo, A. J. (2015). *NICE DSU Technical Support Document 17: The use of observational data to inform estimates of treatment effectiveness in technology appraisal: Methods for comparative individual patient data*.

Fewtrell, M., Wilson, D. C., Booth, I., & Lucas, A. (2011). When to wean? How good is the evidence for six months' exclusive breastfeeding. *British Medical Journal*, *342*, 209–212.

Fitzpatrick, M. (2001). *Doctors and the Regulation of Lifestyle*. *The Tyranny of Health* (1st ed.). New York: Routledge.

Fitzsimons, E., & Vera-hernández, M. (2013). *Food for Thought? Breastfeeding and Child Development*. London.

Flemming, K., Woolcott, C. G., Allen, A. C., Veugelers, P. J., & Kuhle, S. (2013). The association between caesarean section and childhood obesity revisited: a cohort study. *Archives of Disease in Childhood*, *98*, 526–32.

Foresight. (2008). *Tackling Obesity: Future Choices. One Year Review*. London: Government Office for Science.

Francis, L. A., Lee, Y., & Birch, L. L. (2003). Parental weight status and girls' television viewing, snacking, and body mass indexes. *Obesity Research*, *11*(1), 143–151.

Gibbons, L., Belizán, J. M., Lauer, J. a, Betrán, A. P., Merialdi, M., & Althabe, F. (2010). The Global Numbers and Costs of Additionally Needed and Unnecessary Caesarean Sections Performed per Year: Overuse as a Barrier to Universal Coverage. *World Health Report (2010) Background Papers*, 1–31.

Giles-Corti, B., Macintyre, S., Clarkson, J. P., Pikora, T., & Donovan, R. J. (2003). Environmental and Lifestyle Factors Associated with Overweight and Obesity in Perth, Australia. *American Journal of Health Promotion*, *18*(1), 93–102.

Gilleskie, D. B., & Strumpf, K. S. (2005). The Behavioral Dynamics of Youth Smoking. *The Journal of Human Resources*, *40*(4), 822–866.

Gillman, M. W., Rifas-Shiman, S. L., Camargo, C. A., Berkey, C. S., & Frazier, A. L. (2001). Risk of Overweight Among Adolescents who were Breastfed as Infants. *Journal of the American Medical Association*, *285*(19), 2461–2467.

Gladwell, D., Popli, G., & Tsuchiya, A. (2015). *Estimating the impact of health on NEET status* (No. 2015016). Sheffield SERPS WP.

Golan, M., & Weizman, A. (2001). Familial Approach to the Treatment of Childhood Obesity: Conceptual Model. *Journal of Nutrition Education*, *33*(2), 102–107.

Gough, D., Oliver, S., & Thomas, J. (2012). *An Introduction to Systematic Reviews*. London: SAGE Publications Ltd.

Graham, H. (2004a). Social determinants and their unequal distribution: clarifying policy understandings. *The Milbank Quarterly*, *82*(1), 101–124.

Graham, H. (2004b). Tackling Inequalities in Health in England: Remedying Health Disadvantages, Narrowing Health Gaps or Reducing Health Gradients? *Journal of Social Policy*, *33*(1), 115–131.

Graham, H., & Power, C. (2004). *Childhood disadvantage and adult health: a lifecourse framework*. *Health Development Agency*. London: Health Development Agency.

Greene, W., Harris, M., Hollingsworth, B., & Maitra, P. (2008). *A Bivariate latent class correlated generalized ordered Probit model with an application to modelling observed obesity levels* (No. 2451/26027). (No. EC-08-18) NYU.

Greene, W., & Hensher, D. (2010). Endgeneity of a RHS Variable. In *Modelling Ordered Choices: A Primer* (pp. 70–72).

Grossman, M. (1972). On the Concept of Health Capital and the Demand for Health. *Journal of Political Economy*, 223–255.

Grummer-Strawn, L. M., & Mei, Z. (2004). Does Breastfeeding Protect Against Pediatric Overweight? Analysis of Longitudinal Data From the Centers for Disease Control and Prevention Pediatric Nutrition Surveillance System. *Pediatrics*, *113*(2), e81–e86.

Günther, A. L. B., Remer, T., Kroke, A., & Buyken, A. E. (2007). Early protein intake and later obesity risk: which protein sources at which time points throughout infancy and childhood are important for body mass index and body fat percentage at 7 y of age? *The American Journal of Clinical Nutrition*, *86*(6), 1765–1772.

Hancock, R., Morciano, M., & Pudney, S. (2015). Do household surveys give a coherent view of disability benefit targeting?: a multisurvey latent variable analysis for the older population in Great Britain. *Journal of the Royal Statistical Society: Series A (Statistics in Society)*, *178*(4), 815–836.

Hansen, K. (2010). *Millennium Cohort Study First, Second, Third and Fourth Surveys - A Guide to the Datasets*. Centre for Longitudinal Studies, Institute of Education, University of London.

Hansen, K. (2012). *Millennium Cohort Study: First, Second, Thurd and Fourth Surveys: A Guide to the Datasets (Seventh Edition)*. London.

Hanson, L. A. (1998). Breastfeeding provides passive and likely long-lasting active immunity. *Annals of Allergy, Asthma, & Immunology*, *81*(6), 523–537.

Haug, E., Rasmussen, M., Samdal, O., Iannotti, R., Kelly, C., Borraccino, A., … Ahluwalia, N. (2009). Overweight in school-aged children and its relationship with demographic and lifestyle factors: results from the WHO-Collaborative Health Behaviour in School-aged Children (HBSC) study. *International Journal of Public Health*, *54*(supplement 2), 167–179.

Hawkins, S. S., Cole, T. J., & Law, C. (2009). An ecological systems approach to examining risk factors for early childhood overweight: findings from the UK Millennium Cohort Study. *Journal of Epidemiology and Community Health*, *63*(2), 147–55.

Health Survey for England. (2013). *2012 Trend Tables: Child Trend Tables*. Retrieved from www.hscic.gov.uk/pubs/hse2012trend

Heckman, J. J. (1981). *The Incidental Parameters Problem and the Problem of Initial Conditions in Estimating a Discrete Time-Discrete Data Stochastic Process*. MIT

Press, Cambridge, MA: MIT Press.

Heckman, J. J. (1997). Instrumental Variables : A Study of Implicit Behavioral Assumptions Used in Making Program Evaluations. *Journal of Human Resources*, *32*(3), 441–462.

Heckman, J. J. (2007). The economics, technology, and neuroscience of human capability formation. *Proceedings of the National Academy of Sciences of the United States of America*, *104*(33), 13250–13255.

Heckman, J. J. (2012). The developmental origins of health. *Health Economics*, *21*(1), 24–29.

Heckman, J. J., & Navarro-Lozano, S. (2004). Using Matching Instrumental Variables and Control Functions to Estimate Economic Choice Models. *The Review of Economics and Statistics*, *86*(1), 30–57.

Heckman, J. J., & Robb, R. (1986). *Alternative Methods for Solving the Problem of Selection Bias in Evaluating the Impact of Treatments on Outcomes*. *Drawing Inferences from Self-Selected Samples*. Springer New York.

Heckman, J. J., Stixrud, J., & Urzua, S. (2006). The Effects of Cognitive and Noncognitive Abilities on Labor Market Outcomes and Social Behavior: Web Appendix. *Journal of Labor Economics*, *24*(3), 411–482. http://doi.org/10.1086/504455

Heckman, J. J., & Urzúa, S. (2010). Comparing IV with structural models: What simple IV can and cannot identify. *Journal of Econometrics*, *156*(1), 27–37.

Hediger, M. L., Overpeck, M. D., Kuczmarski, R. J., & Ruan, W. J. (2001). Association between infant breastfeeding and overweight in young children. *Journal of the American Medical Association*, *285*(19), 2453–2460.

Heikkilä, K., Sacker, A., Kelly, Y., Renfrew, M. J., & Quigley, M. (2011). Breast feeding and child behaviour in the Millennium Cohort Study. *Archives of Disease in Childhood*, *96*(7), 635–642.

Hernandez Alava, M., & Popli, G. (2013). *Children's Development and Parental Input: Evidence from the UK Millennium Cohort Study* (No. 13/03). Sheffield.

Hernandez Alava, M., Popli, G., Hummel, S., & Chilcott, J. (2011). *Economic Outcomes of Early Years Programmes and Interventions Designed to Promote Cognitive, Social and Emotional Development among Vulnerable Children and Families.* London.

Hernández Alava, M., Sampson, C. J., & Wailoo, A. J. (2013). Happy and healthy: a joint model of health and life satisfaction.

Hill, P. D., & Aldag, J. C. (1996). Smoking and breastfeeding status. *Research in Nursing & Health*, *19*(2), 125–132.

Hillemeier, M. M., Lanza, S. T., Landale, N. S., & Oropesa, R. S. (2013). Measuring early childhood health and health disparities: a new approach. *Maternal and Child Health Journal*, *17*(10), 1852–1861.

Ho, M., Garnett, S. P., Baur, L., Burrows, T., Stewart, L., Neve, M., & Collins, C. (2012). Effectiveness of lifestyle interventions in child obesity: systematic review with meta-analysis. *Pediatrics*, *130*(6), 1647–1671.

Hobcraft, J., & Kiernan, K. (2010). *Predictive factors from age 3 and infancy for poor*

child outcomes at age 5 relating to children's development, behaviour and health: evidence from the Millennium Cohort Study. *University of York*. University of York, York.

Horta, B. L., Kramer, M. S., & Platt, R. W. (2001). Maternal smoking and the risk of early weaning: a meta-analysis. *American Journal of Public Health*, *91*(2), 304–307.

Housman, J., & Dorman, S. (2005). The Alameda County Study: A Systematic, Chronological Review. *American Journal of Health Education*, *36*(5), 302–308.

Hummel, S., Chilcott, J., Rawdin, A., & Strong, M. (2011). *Economic Outcomes of Early Years Programmes and Interventions Designed to Promote Cognitive, Social and Emotional Development among Vulnerable Children and Families. Part 2 - Economic Model*.

Hummel, S., Naylor, P., Chilcott, J., Guillaume, L., Wilkinson, A., Blank, L., … Goyder, E. (2009). *Social and emotional wellbeing in secondary education*.

Iacovou, M., & Sevilla-Sanz, A. (2010). *The Effect of Breastfeeding on Children's Cognitive Development* (No. 2010-40). (No. 2010-40) ISER, Uni. Essex.

Imbens, G. W., & Wooldridge, J. M. (2009). Recent Developments in the Econometrics of Program Evaluation. *Journal of Economic Literature*, *47*(1), 5–86.

Janssen, I., Katzmarzyk, P. T., Boyce, W. F., Vereecken, C., Mulvihill, C., Roberts, C., … Pickett, W. (2005). Comparison of overweight and obesity prevalence in school-aged youth from 34 countries and their relationships with physical activity and dietary patterns. *Obesity Reviews*, *6*(2), 123–132.

Jenkins, S. P. (2000). Modelling household income dynamics. *Journal of Population Economics*, *13*(4), 529–567.

Jiang, M., & Foster, E. M. (2012). Duration of Breastfeeding and Childhood Obesity: A Generalized Propensity Score Approach. *Health Services Research*, *48*(2), 628–651.

Jiang, M., Foster, E. M., & Gibson-Davis, C. M. (2011). Breastfeeding and the child cognitive outcomes: a propensity score matching approach. *Maternal and Child Health Journal*, *15*(8), 1296–307. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/20848171

Jones, A. M. (2007). Indetification of Treatment effects in Health Economics. *Health Economics*, *16*(2007), 1127–1131.

Jones, A. M., & Nicolás, A. L. (2004). Measurement and explanation of socioeconomic inequality in health with longitudinal data. *Health Economics*, *13*(10), 1015–1030.

Jones, A. M., Rice, N., & Contoyannis, P. (2004). Reasons for Persistence in Health. In A. M. Jones (Ed.), *The Elgar Companion to Health Economics* (pp. 18–19).

Jones, A. M., Rice, N., & Dias, P. R. (2010). *Long-term effects of cognitive skills, social adjustment and schooling on health and lifestyle: evidence from a reform of selective schooling* (No. 10/11). (No. 10/11) University of York, York.

Jöreskog, K. G. (1971). Statistical analysis of sets of congeneric tests. *Psychometrika*, *36*(2), 109–133.

Klaus, M. (1998). Mother and infant: early emotional ties. *Pediatrics*, *102*(5 supplement E), 1244–1246.

Koletzko, B., Von Kries, R., Monasterolo, R. C., Subı, E., & Scaglioni, S. (2009). Can infant feeding choices modulate later obesity risk? *American Journal of Clinical Nutrition*, *89*(2), 1502–1509.

Kramer, M. S. (1981). Do breast-feeding and delayed introduction of solid foods protect against subsequent obesity? *The Journal of Pediatrics*, *98*(6), 883–887.

Kramer, M. S., Chalmers, B., Hodnett, E. D., Sevkovskaya, Z., & Dzikovich, I. (2001). Promotion of Breastfeeding Intervention Trial (PROBIT): a randomized trial in the Republic of Belarus. *Journal of the American Medical Association*, *285*(4), 413–420.

Kramer, M. S., Guo, T., Platt, R. W., Sevkovskaya, Z., & Dzikovich, I. (2003). Infant growth and health outcomes associated with 3 compared with 6 mo of exclusive breastfeeding. *The American Journal of Clinical Nutrition*, *78*(2), 291–5.

Kramer, M. S., Guo, T., Platt, R. W., Vanilovich, I., & Sevkovskaya, Z. (2004). Feeding effects on growth during infancy. *The Journal of Pediatrics*, *145*(5), 600–605.

Kramer, M. S., Matush, L., Vanilovich, I., Platt, R. W., & Bogdanovich, N. (2007). Effects of Prolonged and Exclusive Breastfeeding on Child Height, Weight, Adiposity, and Blood Pressure at Age 6.5 y: Evidence from a Large Randomized Trial. *American Journal of Clinical Nutrition*, *86*(6), 1717–1721.

Kramer, M. S., Matush, L., Vanilovich, I., Platt, R. W., Bogdanovich, N., Sevkovskaya, Z., … Shapiro, S. (2009). A Randomized Breast-feeding Promotion Intervention Did Not Reduce Child Obesity. *Journal of Nutrition*, *139*, 417–421.

Kuczmarski, R. J., Ogden, C. L., Guo, S. S., Grummer-Strawn, L. M., Flegal, K. M., Mei, Z., … Johnson, C. L. (2002). *2000 CDC Growth Charts for the United States: methods and development. Vital and health statistics. Series 11, Data from the national health survey*. Washington DC.

Lamerz, A., Kuepper-Nybelen, J., Wehle, C., Bruning, N., & Trost-Brinkhues, G. (2005). Social class, parental education, and obesity prevalence in a study of six-year-old children in Germany. *International Journal of Obesity*, *29*(4), 373–380.

Lee, H. J., Rubio, M. R., Elo, I. T., Mccollum, K. F., Chung, E. K., & Culhane, J. F. (2005). Factors Associated with Intention to Breastfeed Among Low-Income, Inner-City Pregnant Women. *Maternal and Child Health Journalealth Journal*, *9*(3), 253–261.

Leuven, E., & Sianesi, B. (2012). Stata Module to Perform Full Mahalanobis and Propensity Score Matching, Common Support Graphing, and Covariate Imbalance Testing. Statistical Software Components.

Li, R., Fein, S. B., & Grummer-Strawn, L. M. (2010). Do infants fed from bottles lack self-regulation of milk intake compared with directly breastfed infants? *Pediatrics*, *125*(6), e1386–1393.

Liang, K.-Y., & Zeger, S. L. (1986). Longitudinal data analysis using generalized linear models. *Biometrika Trust*, *73*(1), 13–22.

Liese, A. D., Hirsch, T., von Mutius, E., Keil, U., & Leupold, W. (2001). Inverse Association of Overweight and Breast Feeding in 9 to 10-Y-Old Children in Germany. *International Journal of Obesity*, *25*(11), 1644–1650.

Lindeboom, M., Llena-Nozal, A., & van der Klaauw, B. (2009). Parental education and child health: evidence from a schooling reform. *Journal of Health Economics*,

28(1), 109–131.

Lindsay, A. C., Sussner, K. M., Kim, J., & Gortmaker, S. L. (2006). The Role of Parents in Preventing Childhood Obesity. *The Future of Children*, *16*(1), 169–186.

Livingstone, M. B., Prentice, A. M., Coward, W. a, Strain, J. J., & Black, a E. (1992). Validation of estimates of energy intake by weighed dietary record and diet history in children and adolescents. *The American Journal of Child Nutrition*, *56*(1), 29–35.

Mackenbach, J. P. (2012). The persistence of health inequalities in modern welfare states: the explanation of a paradox. *Social Science & Medicine*, *75*(4), 761–769.

Maddala, G. S. (1983). Models with Self-selectivity. In *Limited-dependent and Qualitative Variables in Econometrics* (pp. 257–290). Cambridge University Press.

Marmot, M. (2010). *Fair society, healthy lives*. The Marmot Review, Strategic review of health inequalities in England post-2010.

Marmot, M., & Bell, R. (2012). Fair society, healthy lives. *Public Health*, *126*(supplement), s4–10.

Mayer-Davis, E. J., Rifas-Shiman, S. L., Zhou, L., Hu, F. B., & Colditz, G. A. (2006). Breast-Feeding and Risk for Childhood Obesity: Does Maternal Diabetes or Obesity Status Matter? *Diabetes Care*, *29*(10), 2231–2237.

McCallum, Z., Wake, M., Gerner, B., Baur, L. a, Gibbons, K., Gold, L., … Waters, E. (2007). Outcome data from the LEAP (Live, Eat and Play) trial: a randomized controlled trial of a primary care intervention for childhood overweight/mild obesity. *International Journal of Obesity*, *31*(4), 630–636.

McConley, R. L., Mrug, S., Gilliland, M. J., Lowry, R., Elliott, M. N., Schuster, M. a, … Franklin, F. a. (2011). Mediators of maternal depression and family structure on child BMI: parenting quality and risk factors for child overweight. *Obesity*, *19*(2), 345–352.

McCrory, C., & Layte, R. (2012). Breastfeeding and Risk of Overweight and Obesity at Nine-Years of Age. *Social Science & Medicine*, *75*(2), 323–330.

Mckelvey, R. D., & Zavoina, W. (1975). A Statistical Model for the Analysis of Ordinal Level Dependent Variables. *Journal of Mathematical Sociology*, *4*(1), 103–120.

Mcleod, L., & Ruseski, J. E. (2013). *Longitudinal Relationship between Participation in Physical Activity and Health*. *University of Alberta*. Univerisity of Alberta.

Mizutani, T., Suzuki, K., Kondo, N., & Yamagata, Z. (2007). Association of maternal lifestyles including smoking during pregnancy with childhood obesity. *Obesity*, *15*(12), 3133–3139.

Morciano, M., Hancock, R., & Pudney, S. (2014). Disability Costs and Equivalence Scales in the Older Population in Great Britain. *Review of Income and Wealth*, *62*(3), 494–514.

Must, A., & Strauss, R. (1999). Risks and Consequences of Childhood and Adolescent Obesity. *International Journal of Obesity Related Metabolic Disorders*, *23*(2), s2–11.

Muthen, B. (1984). A General Structural Equation Model with Dichotomous Ordered Categorical and Continuous Latent Varaible Indicators. *Psychometrika*, *49*(1), 115–132.

Muthen, L. K., & Muthen, B. O. (2011). Mplus 6.1. Los Angeles, CA: Muthen & Muthen.

National Obesity Observatory (NOO). (2010). Epidemiology of Morbid Obesity: National Obesity Observatory. Retrieved November 15, 2011, from http://www.noo.org.uk/NOO_about_obesity/morbid_obesity

NICE. (2006). *NICE Clinical Guideline 43: Obesity guidance on the prevention, identification, assessment and management of overweight and obesity in adults and children*. London: National Institute for Health and Care Excellence (NICE).

NICE. (2008). *NICE Public Health Guidance 11: Maternal and Child Nutrition*. London: National Institute for Health and Care Excellence (NICE): National Institute for Health and Care Excellence (NICE). Retrieved from http://guidance.nice.org.uk/PH11

NICE. (2010). *NICE Public Health Guidance 27: Dietary interventions and physical activity interventions for weight management before, during and after pregnancy*. London: National Institute for Health and Care Excellence (NICE).

NICE. (2011a). *NICE Public Health Guidance 35: Preventing type II diabetes: Population and community-level interventions in high-risk groups and the general population:* London: National Institute for Health and Care Excellence (NICE).

NICE. (2011b). Obesity (with co-morbidity) - lorcaserin. NICE. Retrieved from http://guidance.nice.org.uk/TA/Wave21/19

NICE. (2012a). *Health inequalities and population health*. *Local Government Public Health Briefings*. Retrieved from http://publications.nice.org.uk/health-inequalities-and-population-health-phb4

NICE. (2012b). *NICE Public Health Guidance 40: Social And Emotional Wellbeing: Early Years*. London: National Institute for Health and Care Excellence (NICE).

NICE. (2012c). *NICE Public Health Guidance 42: Obesity: Working with Local Communities*. London: National Institute for Health and Care Excellence (NICE).

NICE. (2013a). *NICE Public Health Guidance 46: Assessing Body Mass Index and Waist Circumfrence Thresholds for Intervening to Prevent Ill Health and Premature Death Among Adults from Black, Asian and Other Minority Ethnic Groups in the UK*. London: National Institute for Health and Care Excellence (NICE).

NICE. (2013b). *NICE Public Health Guidance 47: Managing overweight and obesity among children and young people: lifestyle weight management services*. London: National Institute for Health and Care Excellence (NICE).

NICE. (2014a). *NICE Public Health Guidance 24: Alcohol-use disorders: preventing harmful drinking*. London: National Institute for Health and Care Excellence (NICE).

NICE. (2014b). *NICE Public Health Guidance 51: Contraceptive services with a focus on young people up to the age of 25*. London: National Institute for Health and Care Excellence (NICE).

NICE. (2014c). *NICE Public Health Guidance 53: Managing overweight and obesity in adults – lifestyle weight management services*. London: National Institute for Health and Care Excellence (NICE).

NICE: National Collaborating Centre for Mental Health. (2004). *NICE Clinical*

Guidance 9: Eating Disorders. London: The British Society and Gaskell.

Oddy, W. H., Kendall, G. E., Li, J., Jacoby, P., & Robinson, M. (2010). The long-term effects of breastfeeding on child and adolescent mental health: a pregnancy cohort study followed for 14 years. *The Journal of Pediatrics*, *156*(4), 568–574.

Oddy, W. H., & Sherriff, J. L. (2003). Breastfeeding, Body Mass Index, Asthma and Atopy in Children. *Asia-Pacific Journal of Public Health*, *15*(supplement), s15–s17.

Ogden, C. L., & Flegal, K. M. (2010). *Changes in Terminology for Childhood Overweight and Obesity*. National Health Statistics Reports, US Dept of Health and Human Services.

Onis, M. D., Onyango, A. W., Borghi, E., Siyam, A., Nishida, C., & Siekmann, J. (2007). Development of a WHO growth reference for school-aged children and adolescents. *Bulletin of the World Health Organization*, *85*(9), 660–667.

Parsons, T. J., Power, C., & Manor, O. (2003). Infant feeding and obesity through the lifecourse. *Archives of Disease in Children*, *88*(9), 793–794.

Pérez Pérez, A., Ybarra Muñoz, J., Blay Cortés, V., & de Pablos Velasco, P. (2007). Obesity and Cardiovascular Disease. *Public Health Nutrition*, *10*(10A), 1156–1163.

Petrin, A., & Train, K. (2010). A Control Function Approach to Endogeneity in Consumer Choice Models. *Journal of Marketing Research*, *47*(1), 3–13.

Pilgrim, H., Hernandez Alava, M., Blank, L., Payne, N., Guillaume, L., & Baxter, S. (2010). *Systematic review of the long term outcomes associated with teenage pregnancy within the UK*. Retrieved from http://www.nice.org.uk/guidance/ph51/documents/contraceptive-services-for-socially-disadvantaged-young-people-additional-consultation-on-the-evidence-review-of-teenage-pregnancy-outcomes2

Plewis, I. (2007). Non-Response in a Birth Cohort Study: The Case of the Millennium Cohort Study. *International Journal of Social Research Methodology*, *10*(5), 325–334.

Poirier, P., & Eckel, R. H. (2002). Obesity and Cardiovascular Disease. *Current Atherosclerosis Reports*, *4*(6), 448–453.

Posner, M. a., Ash, A. S., Freund, K. M., Moskowitz, M. a., & Shwartz, M. (2002). Comparing standard regression, propensity score matching, and instrumental varibles methods for determining the influence of mammography on stage of diagnosis. *Health Services and Outcomes Research Methodology*, *2*(3-4), 279–290.

Public Health England. (2014). Health Risks of Childhood Obesity. Retrieved from http://www.noo.org.uk/NOO_about_obesity/child_obesity/Health_risks

Public Health England. (2015). Ecnomics of Obesity. Retrieved July 2, 2015, from https://www.noo.org.uk/NOO_about_obesity/economics

Quarmby, T., Dagkas, S., & Bridge, M. (2011). Associations between children's physical activities, sedentary behaviours and family structure: a sequential mixed methods approach. *Health Education Research*, *26*(1), 63–76.

Raat, H., Bonsel, G. J., Essink-Bot, M. L., Landgraf, J. M., & Gemke, R. J. B. J. (2002). Reliability and validity of comprehensive health status measures in children: The Child Health Questionnaire in relation to the Health Utilities Index. *Journal of*

Clinical Epidemiology, *55*(1), 67–76.

Reilly, J. J. (2005). Descriptive Epidemiology and Health Consequences of Childhood Obesity. *Best Practice & Research. Clinical Endocrinology & Metabolism*, *19*(3), 327–341.

Reilly, J. J., Armstrong, J., Dorosty, A. R., Emmett, P. M., & Ness, A. (2005). Early life risk factors for obesity in childhood: cohort study. *British Medical Journal*, *330*, 1357–1363.

Reilly, J. J., Methven, E., McDowell, Z. C., Hacking, B., Alexander, D., Stewart, L., & Kelnar, C. J. H. (2003). Health consequences of obesity. *Archives of Disease in Childhood*, *88*(9), 748–752.

Reinhold, S., & Jurges, H. (2012). Parental income and child health in Germany. *Health Economics*, *21*(5), 562–579.

Renfrew, M. J., Dyson, L., Wallance, L., D'Souza, L., & McCormick, F. (2005). *Breastfeeding for Longer - What Works? Systematic Review Summary*. National Institute for Clinical Excellence (NICE).

Renfrew, M. J., Lang, S., & Woolridge, M. W. (2000). Early Versus Delayed Initiation of Breastfeeding. *Cochrane Database Syst Rev*, *2*.

Renfrew, M. J., Spiby, H., D'Souza, L., Wallace, L. M., & Dyson, L. (2007). Rethinking Research in Breast-Feeding: a Critique of the Evidence Base Identified in a Systematic Review of Interventions to Promote and Support Breast-Feeding. *Public Health Nutrition*, *10*(7), 726–732.

Rhee, K. E., De Lago, C. W., Arscott-Mills, T., Mehta, S. D., & Davis, R. K. (2005). Factors associated with parental readiness to make changes for overweight children. *Pediatrics*, *116*(1), e94–101.

Rolland-Cachera, M. F., Deheeger, M., Bellisle, F., Sempé, M., & Guilloud-Bataille, M. (1984). Adiposity Rebound in Children: a Simple Indicator for Predicting Obesity. *American Journal of Clinical Nutrition*, *39*(1), 129–135.

Rosenbaum, P. R., & Rubin, D. B. (1983). The Central Role of the Propensity Score in Observational Studies for Causal Effects. *Biometrica*, *70*(1), 41–55.

Rothstein, D. (2013). Breastfeeding and children's early cognitive outcomes. *Review of Economics and Statistics*, *95*(3), 919–931.

Roy, A. D. (1951). Some Thoughts on the Distribution of Earnings. *Oxford Economic Papers*, *3*(2), 135–146.

Ryan, W. (1971). *Blaming the Victim* (First Edit). Pantheon Books.

Saha, A. K., Sarkar, N., & Chatterjee, T. (2011). Health Consequences of Childhood Obesity. *Indian Journal of Pediatrics*, *78*(11), 1349–1355.

Salsberry, P. J., & Reagan, P. B. (2005). Dynamics of Early Childhood Overweight. *Journal of Pedicatrics*, *116*(6), 1329–1338.

Scientific Advisory Committee on Nutrition, & Royal College of Paediatrics and Child Health. (2012). *Consideration of issues around the use of BMI centile thresholds for defining underweight, overweight and obesity in children aged 2-18 years in the UK*. SACN & RCPCH Report.

Scott, J. A., Ng, S. Y., & Cobiac, L. (2012). The relationship between breastfeeding and weight status in a national sample of Australian children and adolescents. *BMC*

Public Health, *12*(1), 107. http://doi.org/10.1186/1471-2458-12-107

Seghers, J., & Claessens, A. L. (2010). Bias in self-reported height and weight in preadolescents. *The Journal of Pediatrics*, *157*(6), 911–916.

Semmler, C., Ashcroft, J., van Jaarsveld, C. H. M., Carnell, S., & Wardle, J. (2009). Development of overweight in children in relation to parental weight and socioeconomic status. *Obesity*, *17*(4), 814–820.

Serdula, M. K., Ivery, D., Coates, R. J., Freedman, D. S., Williamson, D. F., & Byers, T. (1993). Do Obese Children Become Obese Adults? A Review of the Literature. *Preventive Medicine*, *22*(2), 167–177.

Shrewsbury, V., & Wardle, J. (2008). Socioeconomic status and adiposity in childhood: a systematic review of cross-sectional studies 1990-2005. *Obesity*, *16*(2), 275–284.

Singhal, A., & Lanigan, J. (2007). Breastfeeding, early growth and later obesity. *Obesity Reviews*, *8*(supplement 1), 51–54.

Skrondal, A., & Rabe-Hesketh, S. (2004). Factor Models and Item Response Models. In N. Keiding, B. Morgan, T. Speed, & P. van der Heijden (Eds.), *Generalized Latent Variable Modeling: Multilevel, Longitudinal and Structural Equation Models* (pp. 49–94). Florida: Chapman and Hall CRC.

Smith, G. D. (2007). Life-course approaches to inequalities in adult chronic disease risk. *The Proceedings of the Nutrition Society*, *66*(2), 216–236.

Smith, H. L. (1997). Matching with Multiple Controls to Estimate Treatment Effects in Observational Studies. *Sociological Methodology*, *27*, 325–353.

Smith, J. (2000). A Critical Survey of Empirical Methods for Evaluating Active Labor Market Policies. *Schweizerische Zeitschrift Fuer Volkswirtschaft Und Statistik*, *136*(3), 1–22.

Smith, J. P. (2004). Unraveling the SES-Health Connection. *Population and Development Review*, 108–132.

Smith, J. P. (2009). The Impact of Childhood Health on Adult Labor Market Outcomes. *The Review of Economics and Statistics*, *91*(3), 478–489.

Starfield, B., Robertson, J., & Riley, A. W. (2002). Social class gradients and health in childhood. *Ambulatory Pediatrics*, *2*(4), 238–246.

StataCorp. (2013). Stata Statistical Software: Release 13. College Station, TX: StataCorp LP.

Stevens, K. (2010). Valuation of the Child Health Utility Index 9D (CHU9D). *Pharmacoeconomics*, *30*(8), 729–747. Retrieved from http://eprints.whiterose.ac.uk/11056/

Stock, J. H., & Yogo, M. (2002). Testing for Weak Instruments in Linear IV Regression. *The National Bureau of Economic Research*, (Technical working paper No. 284), 1–73.

Strauss, R. S. (1997). Effects of the intrauterine environment on childhood growth. *British Medical Bulletin*, *53*(1), 81–95.

Stringhini, S., Sabia, S., Shipley, M., Brunner, E., Nabi, H., Kivimaki, M., & Singh-Manoux, A. (2010). Association of socioeconomic position with health behaviors and mortality. *Journal of the American Medical Association*, *303*(12), 1159–1166.

Summerbell, C., Waters, E., Edmunds, L., Kelly, S., Brown, T., & Campbell, K. (2009). Interventions for preventing obesity in children (Review). *The Cochrane Library*, (1), 1–80.

The Comptroller and Auditor General. (2006). *Tackling Child Obesity – First Steps*. London.

The NHS Information Centre for Health and Social Care. (2008). Health Survey for England - 2008 trend tables. Retrieved November 9, 2011, from http://www.ic.nhs.uk/pubs/hse08trends

Toschke, A. M., Koletzko, B., Slikker, W., Hermann, M., & von Kries, R. (2002). Childhood obesity is associated with maternal smoking in pregnancy. *European Journal of Pediatrics*, *161*(8), 445–448.

Tubeuf, S., Jusot, F., & Bricard, D. (2012). Mediating Role of Education and Lifestlye in the Relationship between Early-life Conditions and Health: Evidence from the 1958 Bristich Cohort. *Health Economics*, *21*(Suppl. 1), 129–150.

UNCEF. (2010). Baby Friendly Initiative. Retrieved August 11, 2015, from http://www.unicef.org.uk/BabyFriendly/

University of London UCL. Institute of Education. Centre for Longitudinal Studies. (n.d.). Millennium Cohort Study: Surveys one to four. Colchester, Essex: UK Data Archive [distributor].

Vandenberghe, V., & Robin, S. (2004). Evaluating the effectiveness of private education across countries: A comparison of methods. *Labour Economics*, *11*(4), 487–506.

Vanlandingham, M., Trussell, J., Grummer-strawn, L., & Vanlandingham, B. M. (1991). Contraception and Health Benefits Contraceptive of Breastfeeding: A Review of the Recent Evidence. *International Family Planning Perspectives*, *17*(4), 131–136.

Vázquez-Nava, F., Treviño-Garcia-Manzo, N., Vázquez-Rodríguez, C. F., & Vázquez-Rodríguez, E. M. (2013). Association between family structure, maternal education level, and maternal employment with sedentary lifestyle in primary school-age children. *Jornal de Pediatria*, *89*(2), 145–150.

Verbeeten, K. C., Elks, C. E., Daneman, D., & Ong, K. K. (2011). Association between Childhood Obesity and Subsequent Type 1 Diabetes: a Systematic Review and Meta-Analysis. *Diabetic Medicine*, *28*(1), 10–18.

von Kries, R., Koletzko, B., Sauerwald, T., von Mutius, E., & Barnert, D. (1999). Breast Feeding and Obesity: Cross Sectional Study. *British Medical Journal*, *319*, 147–150.

Wadsworth, M., Marshall, S., Hardy, R., & Paul, A. (1999). Breast feeding and obesity. Relationship may be accounted for by social factors. *British Medical Journal*, *319*, 1576.

Wang, Y. C., McPherson, K., Marsh, T., Gortmaker, S. L., & Brown, M. (2011). Health and economic burden of the projected obesity trends in the USA and the UK. *The Lancet*, *378*(9793), 815–825. http://doi.org/10.1016/S0140-6736(11)60814-3

Wardle, J., & Steptoe, A. (2003). Socioeconomic differences in attitudes and beliefs about healthy lifestyles. *Journal of Epidemiology and Community Health*, *57*(6), 440–443.

Waters, E., Doyle, J., Wolfe, R., Wright, M., Wake, M., & Salmon, L. (2000). Influence of Parental Gender and Self-Reported Health and Illness on Parent-Reported Child Health. *Pediatrics*, *106*(6), 1422–1428.

Whitaker, R. C., Pepe, M. S., Wright, J. A., Seidel, K. D., & Dietz, W. H. (1998). Early Adiposity Rebound and the Risk of Adult Obesity. *Pediatrics*, *101*(3), e5.

Whitaker, R. C., Wright, J. A., Pepe, M. S., Seidel, K. D., & Dietz, W. H. (1997). Predicting Obesity in Young Adulthood from Childhood and Parental Obesity. *The New England Journal of Medicine*, *337*(13), 869–873.

Wooldridge, J. M. (2000). A framework for estimating dynamic, unobserved effects panel data models with possible feedback to future explanatory variables. *Economics Letters*, *68*(3), 245–250.

World Health Organisation (WHO). (1995). *Physical Status: the Use and Interpretations of Anthropometry*. Geneva.

World Health Organisation (WHO). (2011a). WHO| Exclusive breastfeeding. Retrieved December 14, 2011, from http://www.who.int/nutrition/topics/exclusive_breastfeeding/en/

World Health Organisation (WHO). (2011b). WHO| Global Database on Body Mass Index. Retrieved November 7, 2011, from http://apps.who.int/bmi/index.jsp?introPage=intro_3.html

World Health Organisation (WHO). (2011c). WHO| Obesity and Overweight: Fact Sheet No.311. Retrieved November 7, 2011, from http://www.who.int/mediacentre/factsheets/fs311/en/

World Health Organisation (WHO). (2015). WHO | The Public Health Approach. Retrieved July 3, 2015, from http://www.who.int/violenceprevention/approach/public_health/en/

World Health Organization (WHO). (2003). *Global Strategy for Infant and Young Child Feeding*. Geneva, World Health Organisation.

World Health Organization (WHO). (2008). The New WHO Child Growth Standards. *Paediatria Croatica Supplement*, *52*(Suppl 1), 13–17.

World Health Organization (WHO). (2012). WHO| Preterm birth: Fact Sheet No. 363. Retrieved July 17, 2012, from http://www.who.int/mediacentre/factsheets/fs363/en/index.html

APPENDICES

A. Appendix: Childhood Adiposity and Infant Feeding

Table A-1: Studies included in the Literature Review

Author (year)	Outcome Variable	Breastfeeding Variable(s)	Country	Age of Children	Year(s) of Birth	Data	Sample Size	Model	Result
Armstrong & Reilly (2002)	Overweight (BMI > 95th percentile) - 1990 UK references	Infant exclusively breastfed vs. exclusively formula fed between 6-8 weeks (binary)	Scotland	3 years	1995 - 1996	Cohort	32,200	Logit Models	Exclusively breastfed children less likely to become overweight than exclusively formula fed children.
	Obese (BMI > 98th percentile) - 1990 UK references								Exclusively breastfed children less likely to become obese than exclusively formula fed children.
Bergmann *et al.* (2003)	BMI	Partial (< 3 months, > 3 months)	Germany	Up to 6 years	1990	Cohort	918	Univariate Comparison	Inverse relationship after 4 years.
	Overweight (BMI > 90th percentile) - Rolland Cachera							Logit Models	Breastfeeding decreases likelihood of overweight.
	Obese (BMI > 97th percentile) - Rolland Cachera								Breastfeeding decreases likelihood of obesity.
Beyerlein *et al.* (2008)	BMI	Ever breastfed (binary)	Germany	5 - 7 years	1992 - 1998	Cross-sectional	9368	Linear Regression	No change in mean but reduced standard deviation.
	Overweight (BMI > 90th percentile)*							Logit Models	No relationship found.

	Obese (BMI > 97th percentile)*								Breastfeeding reduced the likelihood of childhood obesity.
	BMI Quantile							Quantile Regression	Breastfeeding reduced BMI over 90th percentile, increased BMI under 3rd percentile.
Bogen *et al.* (2004)	Obese (BMI > 95th percentile) -CDC growth charts	Partial (0, <8, 8-15, 16-26, >26 weeks) Exclusive (0, 8-15, 16-26, >26 weeks)	USA	4 - 5 years	1994 - 2001	Cross-sectional	73,458	Logit Model	Inverse relationship in white children whose mothers did not smoke during pregnancy.
Brion *et al*. (2011)	BMI (No age sex specific measure)	Partial Breastfeeding (0-1, 1-3, 3-6, 6+ months)	UK/ Brazil	9 years/ 11 years	1991 - 1993	Cohort	4,852/ 1,085	Linear Regression	Association found but authors assume no causal inference.
Burdette & Whitaker (2007)	Obese (BMI > 95th percentile) -CDC growth charts	Partial (0, <4, >4 months)	USA	3 years	1998 - 2000	Cohort	2,146	Logit Model	Breastfeeding found to protect from obesity only in Hispanic children.
Burke *et al.* (2005)	Overweight (BMI > 95th percentile) - National Centre for Health Statistics	Partial (<4, 5-8, 9-12, >12 months)	Australia	Up to 8 years	1989 - 1992	Cohort	2,087	Logit Model/ GEE	Significant inverse relationship in early years, relationship becomes insignificant by age 8.
Del Bono & Rabe (2012)	Overweight (adult definitions, BMI>25)	UNICEF Baby Friendly Initiative (Instrument)	UK	3 - 7 years	2000 - 2001	Cohort	9,524	Instrumental Variable	No significant effect of breastfeeding on overweight.
Denny & Doyle (2008)	No adiposity measure included	Caesarean Section (Instrument)	UK	3 - 11 years	1958, 2000 - 2001	Cohort	4,923 - 11,792	Instrumental Variable	N/A

Fitzsimons & Vera-hernández (2013)	BMI included as part of a wider health index	Day/Time of birth (Instrument)	UK	3 - 7 years	2000 - 2001	Cohort	3,424 - 5.989	Instrumental Variable	N/A
Gillman *et al.* (2001)	At risk of overweight (BMI > 85th percentile) -CDC growth charts	Wholly or mostly breastfed (binary)	USA	9 – 14 years	1982 - 1987	Cross-sectional	15,341	Logit Models	Reduced risk in those breastfed for longer.
	Overweight (BMI > 95th percentile) -CDC growth charts								Reduced risk in those breastfed for longer.
Grummer-Strawn & Mei (2004)	Obese (BMI > 95th percentile) -CDC growth charts	Partial (0, 1-2, 3-5, 6-11, ≥12 months)	USA	4 years	1988 - 1992	Cohort	12,587	Logit Model	Dose response found only in non-Hispanic white children.
Hediger *et al.* (2001)	At risk of overweight (BMI > 85th percentile) -CDC growth charts	Ever breastfed (binary) Exclusive (months)	USA	3 – 6 years	1982 - 1992	Cross-sectional	2,685	Logit Models	No dose or threshold response. Never breastfed more at risk.
	Overweight (BMI > 95th percentile) -CDC growth charts								No dose or threshold response. Never breastfed more at risk.
Jiang & Foster (2012)	BMI	Partial (months)	USA	5 – 18 years	1984 - 1997	Cross-sectional	2,907	Generalised Propensity Score Approach	No relationship after accounting for confounders.
	Obesity (BMI > 95th percentile) -CDC growth charts								No relationship after accounting for confounders.

Kramer *et al.* (2007)	BMI	Promotion of exclusive and prolonged breastfeeding (binary)	Belarus	6 years	1996 - 1997	Cohort	13,889	Linear Regression within a Randomised Intervention Trial	No relationship between breastfeeding promotion and BMI.
Liese *et al.* (2001)	Overweight (BMI > 90th percentile) - German BMI-for-age reference values	Partial (0, <6, 6-12, >12 months) Exclusive (0, <2, 2-4, 5-6, >6 months)	Germany	9 – 10 years	1982 - 1984	Cross-sectional	2,108	Logit Model	Inverse relationship but largely attenuated by confounders.
Mayer-Davis *et al.* (2006)	At risk of overweight (BMI > 85th percentile) -CDC growth charts	Partial (0, <1, 1-3, 4-6, 7-9, >9 months) Exclusive for at least 6 months (binary)	USA	9 – 14 years	1982 - 1987	Cross-sectional	15,253	Logit Models	Exclusively breastfed children are at lower risk than those exclusively formula fed.
	Overweight (BMI > 95th percentile) -CDC growth charts								Exclusively breastfed children are at lower risk than those exclusively formula fed.
McCrory & Layte (2012)	Overweight - IOTF references	Partial (0, <4, 5-8, 9-12, 13-25, >26)	Ireland	9 years	1997 - 1998	Cohort	7,798	Logit Models	No relationship after accounting for confounders.
	Obesity - IOTF references								No relationship after accounting for confounders.
Oddy and Sherriff (2003)	BMI	Partial (months)	Australia	Up to 6 years	1989 - 1992	Cohort	2,602	Linear Regression	No relationship after accounting for confounders.
Reilly *et al.* (2005)	Obese (BMI > 95th percentile) - 1990 UK references	Exclusive (0, <2, >2 months)	UK	7 years	1991 - 1992	Cohort	909	Logit Model	No relationship after accounting for confounders.

Salsberry & Reagan (2005)	Overweight (BMI > 95th percentile) -CDC growth charts	Ever breastfed (binary)	USA	2 – 8 years	1982 – 1996	Cross-sectional	3,022	Logit Model/ Markov Model	No relationship after accounting for confounders.
Scott *et al*. (2012)	Weight status - IOTF references	Breastfed for at least 6 months compared to never breastfed	Australia	9 - 16 years	1991 - 2005	Cross-sectional	2,066	Ordered Logit Model	Significant protective effect of breastfeeding on later obesity and overweight.
von Kries *et al.* (1999)	Overweight (BMI > 90th percentile)*	Ever breastfed (binary) Exclusive (0, <2, 3-5, 6-12, >12 months)	Germany	5 – 6 years	1992 - 1993	Cohort	9,357	Logit Model	Dose response.
	Obese (BMI > 97th percentile)*								Dose response.

Notes: *percentiles refer to data used in the study or from the population the sample is taken from. Studies included in this table are those included in the literature review which specifically investigate the effect of breastfeeding on some recognised measure of childhood adiposity. Journal articles which investigate other relationships in the area of breastfeeding or adiposity and which have relevant econometric methods.

Table A-2: Description of Independent Variables

Variable	Description
	Confounding Variables
High education*	Mother has at least one degree
Low education*	Mother received no qualifications after compulsory education
High SES*	Family SES at birth was low (NS-SEC)
Low SES*	Family SES at birth was high (NS-SEC)
	Demographic Variables
Male*	Child is male
Black*¥	Mother considers child to be of any black background
Asian*¥	Mother considers child to be of any Asian background
Other*¥	Mother considers child not to be white, black or Asian
Home Owner*	Owns outright or has mortgage on own home
Private Renter*	Rents home privately or has shared equity
Natural Parents*	Lived with both natural parents during first wave
	Birth Variables
Birth weight	Weight at birth (kg)
Premature*	Child was born before 37 weeks gestation
Caesarean Section*	Infant was delivered by Caesarean section
Log Hospital Stay	Log of number of days in hospital
Planned Pregnancy*	Pregnancy was planned
	Maternal Variables
Mother married*	Mother married during first wave
Mother obese*	Mother obese before pregnancy
Mother age at birth	Age of natural mother at birth of child
Smoking 1st Trimester*	Mother smoked during 1st trimester of pregnancy
Smoking 2nd Trimester*	Mother smoked during 2nd trimester of pregnancy
Smoking 3rd Trimester*	Mother smoked during 3rd trimester of pregnancy
Alcohol units a day	Units of alcohol drank on an average drinking day during pregnancy
Mother in Care as Child*	Mother was in care when leaving school
Illness*	Mother's had a long standing illness around the time of birth

Source: Millennium Cohort Study. Notes: * Binary variable. Description given takes the value 1, otherwise 0. ¥ Omitted category is 'white'. € Omitted category is 'house or bungalow'.

Table A-3: Linear Models Estimating BMI in Three Year Olds

	BMI				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	-0.0582	-0.0626	-0.106*	-0.0618	-0.172**
	(0.0437)	(0.0380)	(0.0446)	(0.0393)	(0.0538)
Age	-0.00186	-0.00110	-0.000217	-0.00202	-0.0000669
	(0.00135)	(0.00153)	(0.00172)	(0.00161)	(0.00193)
Sex	0.178***	0.154***	0.144***	0.164***	0.153***
	(0.0287)	(0.0323)	(0.0366)	(0.0341)	(0.0423)
Black	0.217*	0.223*	0.264*	0.197	0.108
	(0.0974)	(0.104)	(0.121)	(0.128)	(0.175)
Asian	-0.488***	-0.517***	-0.531***	-0.480***	-0.492***
	(0.0573)	(0.0653)	(0.0770)	(0.0728)	(0.0935)
Other	-0.106	-0.0457	-0.0120	-0.0221	-0.189
	(0.0819)	(0.0918)	(0.106)	(0.101)	(0.134)
high education	0.0600	0.0570	0.0978	0.0744	0.149*
	(0.0472)	(0.0533)	(0.0616)	(0.0566)	(0.0746)
low education	-0.0129	-0.0156	-0.0192	0.00636	-0.00460
	(0.0429)	(0.0489)	(0.0560)	(0.0514)	(0.0644)
high SES	0.0404	0.0378	0.0524	0.0543	0.0324
	(0.0438)	(0.0489)	(0.0560)	(0.0524)	(0.0697)
low SES	0.0748*	0.0894*	0.0784	0.0583	0.0393
	(0.0360)	(0.0413)	(0.0473)	(0.0434)	(0.0542)
live with both natural parents	-0.0363	-0.0659	-0.0801	-0.0551	-0.0493
	(0.0487)	(0.0553)	(0.0616)	(0.0574)	(0.0673)
mother married	-0.0614	-0.0441	-0.0375	-0.0812	-0.0790
	(0.0378)	(0.0428)	(0.0486)	(0.0450)	(0.0560)
home owners	-0.0843*	-0.0759	-0.0701	-0.0939	-0.103
	(0.0417)	(0.0477)	(0.0541)	(0.0504)	(0.0611)
private renters	-0.132*	-0.0961	-0.0565	-0.0725	0.0149
	(0.0574)	(0.0654)	(0.0733)	(0.0685)	(0.0828)
birth weight	0.606***	0.634***	0.632***	0.636***	0.628***
	(0.0296)	(0.0334)	(0.0383)	(0.0357)	(0.0444)
hospital stay (log)	0.0331	0.0210	0.00215	0.0263	0.0169
	(0.0241)	(0.0272)	(0.0310)	(0.0288)	(0.0360)
planned pregnancy	0.00529	0.0127	-0.00760	0.0112	0.00337
	(0.0324)	(0.0366)	(0.0415)	(0.0386)	(0.0477)
Premature	0.388***	0.466***	0.368***	0.474***	0.348***
	(0.0651)	(0.0739)	(0.0854)	(0.0794)	(0.0965)
mother obese	0.472***	0.445***	0.470***	0.492***	0.538***
	(0.0533)	(0.0617)	(0.0718)	(0.0657)	(0.0822)
mother age at birth	0.00570	0.00242	0.00206	0.00342	0.00487
	(0.00293)	(0.00332)	(0.00376)	(0.00350)	(0.00428)
smoker 1st trimester	0.177***	0.173***	0.162**	0.165***	0.145**
	(0.0373)	(0.0431)	(0.0494)	(0.0450)	(0.0555)
smoker 2nd trimester	0.0401	0.0245	0.0244	-0.00844	0.00483
	(0.0949)	(0.106)	(0.116)	(0.108)	(0.125)
smoker 3rd trimester	0.254***	0.251***	0.260***	0.279***	0.273***
	(0.0615)	(0.0699)	(0.0775)	(0.0719)	(0.0828)
alcohol during pregnancy	-0.0159	-0.0135	-0.0205	-0.0199	-0.0113
	(0.0145)	(0.0164)	(0.0175)	(0.0168)	(0.0206)
mother in care at 16 years	-0.0157	-0.0562	-0.0217	-0.106	-0.0863
	(0.152)	(0.175)	(0.194)	(0.182)	(0.205)
maternal longstanding illness	-0.0334	-0.0436	-0.0451	-0.0448	-0.0572
	(0.0350)	(0.0400)	(0.0456)	(0.0424)	(0.0527)
Constant	14.78***	14.67***	14.59***	14.80***	14.50***
	(0.269)	(0.304)	(0.343)	(0.320)	(0.387)
N	11200	8845	6949	7885	5290

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. OLS regressions varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Table A-4: Linear Model Estimating BMI in Five Year Olds

	BMI				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	-0.0889**	-0.119**	-0.189***	-0.131**	-0.172**
	(0.0356)	(0.0398)	(0.0465)	(0.0411)	(0.0538)
Age	-0.000869	-0.000780	-0.000319	-0.000460	-0.0000669
	(0.00116)	(0.00129)	(0.00145)	(0.00136)	(0.00193)
Sex	-0.0196	-0.0361	-0.0622	-0.0363	0.153***
	(0.0301)	(0.0337)	(0.0380)	(0.0355)	(0.0423)
Black	0.674***	0.710***	0.815***	0.682***	0.108
	(0.100)	(0.106)	(0.120)	(0.128)	(0.175)
Asian	-0.337***	-0.361***	-0.379***	-0.441***	-0.492***
	(0.0603)	(0.0681)	(0.0802)	(0.0764)	(0.0935)
Other	-0.0591	-0.0118	0.0715	-0.0657	-0.189
	(0.0862)	(0.0947)	(0.109)	(0.104)	(0.134)
high education	0.00863	0.0249	0.0355	0.0421	0.149*
	(0.0498)	(0.0558)	(0.0646)	(0.0592)	(0.0746)
low education	0.0213	0.0122	0.0113	0.0328	-0.00460
	(0.0452)	(0.0512)	(0.0585)	(0.0537)	(0.0644)
high SES	0.0502	0.0426	0.0441	0.0426	0.0324
	(0.0463)	(0.0512)	(0.0586)	(0.0548)	(0.0697)
low SES	0.103**	0.118**	0.0945	0.0996*	0.0393
	(0.0380)	(0.0433)	(0.0493)	(0.0455)	(0.0542)
live with both natural parents	-0.00750	-0.0273	-0.0361	-0.00396	-0.0493
	(0.0505)	(0.0568)	(0.0630)	(0.0588)	(0.0673)
mother married	-0.0590	-0.0389	-0.0106	-0.0673	-0.0790
	(0.0397)	(0.0446)	(0.0503)	(0.0467)	(0.0560)
home owners	-0.0702	-0.0709	-0.0602	-0.0749	-0.103
	(0.0437)	(0.0495)	(0.0556)	(0.0521)	(0.0611)
private renters	-0.151*	-0.101	-0.0528	-0.0874	0.0149
	(0.0592)	(0.0669)	(0.0744)	(0.0698)	(0.0828)
birth weight	0.635***	0.659***	0.659***	0.648***	0.628***
	(0.0309)	(0.0346)	(0.0395)	(0.0369)	(0.0444)
hospital stay (log)	0.0886***	0.0838**	0.0669*	0.0907**	0.0169
	(0.0254)	(0.0284)	(0.0323)	(0.0300)	(0.0360)
planned pregnancy	-0.0191	0.00984	0.0222	0.00579	0.00337
	(0.0341)	(0.0382)	(0.0431)	(0.0402)	(0.0477)
Premature	0.396***	0.425***	0.363***	0.451***	0.348***
	(0.0676)	(0.0759)	(0.0875)	(0.0816)	(0.0965)
mother obese	0.737***	0.678***	0.700***	0.681***	0.538***
	(0.0581)	(0.0670)	(0.0779)	(0.0713)	(0.0822)
mother age at birth	0.00191	0.0000315	0.000348	0.000678	0.00487
	(0.00309)	(0.00346)	(0.00390)	(0.00364)	(0.00428)
smoker 1st trimester	0.247***	0.255***	0.249***	0.253***	0.145**
	(0.0391)	(0.0447)	(0.0509)	(0.0466)	(0.0555)
smoker 2nd trimester	0.147	0.154	0.218	0.135	0.00483
	(0.0999)	(0.112)	(0.120)	(0.114)	(0.125)
smoker 3rd trimester	0.318***	0.314***	0.344***	0.336***	0.273***
	(0.0638)	(0.0711)	(0.0781)	(0.0730)	(0.0828)
alcohol during pregnancy	-0.0157	-0.0129	-0.0148	-0.0166	-0.0113
	(0.0152)	(0.0169)	(0.0181)	(0.0173)	(0.0206)
mother in care at 16 years	-0.137	-0.223	-0.262	-0.214	-0.0863
	(0.166)	(0.189)	(0.217)	(0.198)	(0.205)
maternal longstanding illness	0.0343	0.0408	0.0348	0.0303	-0.0572
	(0.0369)	(0.0418)	(0.0475)	(0.0442)	(0.0527)
Constant	14.20***	14.15***	14.03***	14.07***	14.50***
	(0.352)	(0.392)	(0.443)	(0.414)	(0.387)
N	11744	9283	7278	8259	5290

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. OLS regressions varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Table A-5: Linear Model Estimating BMI in Seven year Olds

	BMI				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	-0.118**	-0.185***	-0.261***	-0.195***	-0.265***
	(0.0495)	(0.0551)	(0.0641)	(0.0572)	(0.0556)
Age	0.0100***	0.0105***	0.0114***	0.0107***	-0.000934
	(0.00162)	(0.00180)	(0.00203)	(0.00191)	(0.00166)
Sex	-0.166***	-0.198***	-0.212***	-0.206***	-0.0724
	(0.0419)	(0.0468)	(0.0527)	(0.0496)	(0.0436)
Black	0.928***	1.056***	1.111***	1.050***	0.688***
	(0.140)	(0.148)	(0.169)	(0.181)	(0.170)
Asian	-0.118	-0.117	-0.108	-0.206	-0.327***
	(0.0847)	(0.0959)	(0.112)	(0.107)	(0.0980)
Other	0.102	0.137	0.189	0.0721	-0.170
	(0.122)	(0.134)	(0.150)	(0.146)	(0.135)
high education	-0.0241	0.00432	0.0471	0.0308	0.0196
	(0.0687)	(0.0768)	(0.0886)	(0.0819)	(0.0774)
low education	0.0503	0.0448	0.0304	0.0595	-0.0265
	(0.0626)	(0.0708)	(0.0808)	(0.0747)	(0.0667)
high SES	0.00529	0.0120	-0.0409	0.0123	0.0521
	(0.0633)	(0.0699)	(0.0800)	(0.0752)	(0.0727)
low SES	0.122*	0.129*	0.0783	0.124	0.101
	(0.0529)	(0.0601)	(0.0686)	(0.0634)	(0.0564)
live with both natural parents	-0.0217	-0.0378	-0.0568	0.00148	-0.0431
	(0.0713)	(0.0799)	(0.0886)	(0.0832)	(0.0681)
mother married	-0.119*	-0.0901	-0.0442	-0.117	-0.0617
	(0.0548)	(0.0614)	(0.0693)	(0.0647)	(0.0574)
home owners	-0.0835	-0.0815	-0.0780	-0.0903	-0.0667
	(0.0612)	(0.0694)	(0.0783)	(0.0735)	(0.0624)
private renters	-0.0396	0.0444	0.153	0.0632	-0.0418
	(0.0836)	(0.0941)	(0.104)	(0.0986)	(0.0829)
birth weight	0.694***	0.750***	0.752***	0.719***	0.670***
	(0.0433)	(0.0483)	(0.0549)	(0.0516)	(0.0453)
hospital stay (log)	0.0939**	0.0792*	0.0450	0.0833*	0.105**
	(0.0352)	(0.0393)	(0.0446)	(0.0417)	(0.0372)
planned pregnancy	-0.0569	-0.0323	-0.00967	-0.0303	0.0439
	(0.0473)	(0.0529)	(0.0597)	(0.0560)	(0.0491)
Premature	0.430***	0.540***	0.497***	0.495***	0.301**
	(0.0950)	(0.106)	(0.123)	(0.116)	(0.0980)
mother obese	1.273***	1.199***	1.224***	1.207***	0.735***
	(0.0820)	(0.0947)	(0.110)	(0.101)	(0.0885)
mother age at birth	0.0135**	0.0128**	0.0121*	0.0110*	0.00214
	(0.00430)	(0.00481)	(0.00543)	(0.00509)	(0.00441)
smoker 1st trimester	0.355***	0.334***	0.343***	0.328***	0.250***
	(0.0544)	(0.0620)	(0.0707)	(0.0651)	(0.0566)
smoker 2nd trimester	0.373**	0.394*	0.554***	0.397*	0.161
	(0.138)	(0.154)	(0.166)	(0.157)	(0.129)
smoker 3rd trimester	0.436***	0.478***	0.517***	0.500***	0.382***
	(0.0905)	(0.101)	(0.111)	(0.104)	(0.0828)
alcohol during pregnancy	-0.0462*	-0.0487*	-0.0605*	-0.0576*	-0.0153
	(0.0214)	(0.0240)	(0.0261)	(0.0248)	(0.0211)
mother in care at 16 years	-0.342	-0.394	-0.571	-0.429	-0.297
	(0.248)	(0.274)	(0.307)	(0.285)	(0.224)
maternal longstanding illness	0.0593	0.0734	0.0650	0.0534	0.0486
	(0.0512)	(0.0577)	(0.0657)	(0.0614)	(0.0546)
Constant	9.995***	9.656***	9.351***	9.712***	14.13***
	(0.652)	(0.726)	(0.819)	(0.770)	(0.506)
N	10707	8474	6643	7542	5541

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. OLS regressions varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Table A-6: Logit Model Estimating Overweight in Three Year Olds

	Overweight				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	-0.159**	-0.176**	-0.224**	-0.170**	-0.403***
	(0.0532)	(0.0604)	(0.0714)	(0.0628)	(0.0881)
Age	0.00188	0.00278	0.00394	0.00248	0.00401
	(0.00215)	(0.00240)	(0.00265)	(0.00253)	(0.00297)
Sex	-0.210***	-0.249***	-0.274***	-0.232***	-0.254***
	(0.0460)	(0.0519)	(0.0584)	(0.0547)	(0.0672)
Black	0.393**	0.408**	0.563**	0.361	0.398
	(0.145)	(0.156)	(0.178)	(0.194)	(0.266)
Asian	-0.305**	-0.361**	-0.398**	-0.306*	-0.348*
	(0.105)	(0.121)	(0.144)	(0.134)	(0.175)
Other	0.0761	0.107	0.141	0.131	-0.0502
	(0.132)	(0.147)	(0.168)	(0.161)	(0.223)
high education	0.0390	0.0798	0.0944	0.0888	0.190
	(0.0755)	(0.0853)	(0.0977)	(0.0907)	(0.118)
low education	-0.0572	-0.0542	-0.117	-0.0366	-0.0430
	(0.0686)	(0.0782)	(0.0887)	(0.0821)	(0.102)
high SES	-0.0227	-0.0346	0.0174	-0.0197	0.0495
	(0.0708)	(0.0793)	(0.0902)	(0.0849)	(0.112)
low SES	0.121*	0.160*	0.203**	0.129	0.125
	(0.0578)	(0.0662)	(0.0756)	(0.0696)	(0.0863)
live with both natural parents	-0.0605	-0.0905	-0.0909	-0.0837	-0.0799
	(0.0762)	(0.0861)	(0.0952)	(0.0894)	(0.104)
mother married	-0.129*	-0.0700	-0.0662	-0.118	-0.0599
	(0.0598)	(0.0679)	(0.0765)	(0.0712)	(0.0877)
home owners	-0.00479	0.00446	0.0715	0.0103	0.0197
	(0.0667)	(0.0761)	(0.0857)	(0.0802)	(0.0962)
private renters	-0.183	-0.135	-0.0668	-0.0819	0.0526
	(0.0945)	(0.106)	(0.117)	(0.110)	(0.128)
birth weight	0.752***	0.786***	0.765***	0.778***	0.756***
	(0.0482)	(0.0545)	(0.0621)	(0.0583)	(0.0712)
hospital stay (log)	0.114**	0.110*	0.0832	0.0845	0.0796
	(0.0385)	(0.0434)	(0.0491)	(0.0459)	(0.0568)
planned pregnancy	0.000980	-0.00525	-0.0257	0.00555	0.0118
	(0.0520)	(0.0587)	(0.0660)	(0.0619)	(0.0756)
Premature	0.510***	0.559***	0.518***	0.590***	0.505***
	(0.105)	(0.118)	(0.136)	(0.126)	(0.152)
mother obese	0.503***	0.415***	0.381***	0.492***	0.479***
	(0.0771)	(0.0899)	(0.105)	(0.0951)	(0.118)
mother age at birth	0.00686	0.00203	0.000555	0.0000110	0.00352
	(0.00467)	(0.00529)	(0.00597)	(0.00558)	(0.00674)
smoker 1st trimester	0.159**	0.170*	0.214**	0.162*	0.196*
	(0.0587)	(0.0674)	(0.0767)	(0.0706)	(0.0859)
smoker 2nd trimester	0.144	0.109	0.138	0.0359	0.0608
	(0.150)	(0.168)	(0.183)	(0.174)	(0.199)
smoker 3rd trimester	0.268**	0.264*	0.310**	0.266*	0.315*
	(0.0953)	(0.108)	(0.118)	(0.111)	(0.125)
alcohol during pregnancy	-0.0116	-0.00943	-0.00526	-0.00918	0.0170
	(0.0237)	(0.0263)	(0.0276)	(0.0269)	(0.0308)
mother in care at 16 years	0.0327	0.135	0.241	0.0391	0.144
	(0.241)	(0.270)	(0.291)	(0.287)	(0.311)
maternal longstanding illness	-0.0792	-0.0898	-0.0951	-0.0842	-0.140
	(0.0565)	(0.0644)	(0.0730)	(0.0682)	(0.0845)
Constant	-4.192***	-4.345***	-4.421***	-4.189***	-4.510***
	(0.430)	(0.484)	(0.540)	(0.509)	(0.607)
N	11200	8845	6949	7885	5290

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Logit model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Table A-7: Logit Model Estimating Overweight in Five Year Olds

	Overweight				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	-0.160**	-0.210***	-0.288***	-0.214***	-0.405***
	(0.0541)	(0.0615)	(0.0731)	(0.0643)	(0.0900)
Age	-0.00102	-0.000253	-0.000115	-0.000260	-0.00136
	(0.00180)	(0.00202)	(0.00227)	(0.00213)	(0.00260)
Sex	-0.378***	-0.430***	-0.441***	-0.427***	-0.430***
	(0.0469)	(0.0529)	(0.0599)	(0.0562)	(0.0687)
Black	0.842***	0.885***	0.918***	0.868***	0.843***
	(0.135)	(0.144)	(0.166)	(0.175)	(0.238)
Asian	0.0503	0.0713	0.0803	0.0617	0.206
	(0.0998)	(0.114)	(0.135)	(0.129)	(0.162)
Other	0.185	0.294*	0.379*	0.213	0.0837
	(0.131)	(0.143)	(0.163)	(0.160)	(0.217)
high education	-0.0224	-0.0161	-0.0163	0.0184	-0.0640
	(0.0779)	(0.0879)	(0.102)	(0.0943)	(0.122)
low education	0.00582	-0.0145	-0.0519	0.0200	-0.0990
	(0.0699)	(0.0794)	(0.0906)	(0.0843)	(0.102)
high SES	0.0725	0.0281	0.0378	0.0170	0.104
	(0.0732)	(0.0818)	(0.0940)	(0.0880)	(0.117)
low SES	0.123*	0.0974	0.0865	0.0608	0.119
	(0.0592)	(0.0677)	(0.0772)	(0.0715)	(0.0886)
live with both natural parents	-0.0661	-0.109	-0.120	-0.105	-0.167
	(0.0756)	(0.0854)	(0.0950)	(0.0890)	(0.103)
mother married	-0.0835	-0.0565	-0.0395	-0.0877	-0.0577
	(0.0609)	(0.0691)	(0.0783)	(0.0727)	(0.0894)
home owners	-0.0230	-0.00130	0.0260	0.00103	0.0192
	(0.0666)	(0.0759)	(0.0856)	(0.0805)	(0.0958)
private renters	-0.263**	-0.197	-0.136	-0.189	-0.177
	(0.0949)	(0.107)	(0.118)	(0.112)	(0.132)
birth weight	0.685***	0.716***	0.727***	0.715***	0.743***
	(0.0484)	(0.0547)	(0.0626)	(0.0587)	(0.0717)
hospital stay (log)	0.133***	0.139**	0.124*	0.142**	0.152**
	(0.0395)	(0.0444)	(0.0506)	(0.0472)	(0.0582)
planned pregnancy	-0.0863	-0.0361	-0.0113	-0.0647	0.0564
	(0.0525)	(0.0594)	(0.0673)	(0.0629)	(0.0768)
Premature	0.412***	0.434***	0.382**	0.419**	0.253
	(0.105)	(0.119)	(0.138)	(0.129)	(0.157)
mother obese	0.775***	0.689***	0.721***	0.725***	0.688***
	(0.0776)	(0.0908)	(0.105)	(0.0969)	(0.120)
mother age at birth	0.00588	0.00475	0.00222	0.00592	0.00785
	(0.00473)	(0.00535)	(0.00605)	(0.00565)	(0.00681)
smoker 1st trimester	0.260***	0.295***	0.315***	0.313***	0.357***
	(0.0594)	(0.0681)	(0.0778)	(0.0714)	(0.0866)
smoker 2nd trimester	0.200	0.231	0.321	0.185	0.373
	(0.151)	(0.170)	(0.181)	(0.175)	(0.193)
smoker 3rd trimester	0.371***	0.395***	0.406***	0.437***	0.477***
	(0.0940)	(0.105)	(0.116)	(0.108)	(0.123)
alcohol during pregnancy	-0.0215	-0.0288	-0.0252	-0.0285	-0.0236
	(0.0247)	(0.0280)	(0.0300)	(0.0289)	(0.0341)
mother in care at 16 years	-0.145	-0.385	-0.672	-0.443	-0.877*
	(0.260)	(0.317)	(0.397)	(0.339)	(0.444)
maternal longstanding illness	0.00137	-0.00998	0.0370	-0.0187	0.0529
	(0.0568)	(0.0648)	(0.0733)	(0.0691)	(0.0838)
Constant	-3.520***	-3.775***	-3.779***	-3.798***	-3.664***
	(0.546)	(0.613)	(0.695)	(0.651)	(0.791)
N	11744	9283	7278	8259	5541

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Logit model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Table A-8: Logit Model Estimating Overweight in Seven Year Olds

	Overweight				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	-0.152**	-0.229***	-0.254***	-0.242***	-0.362***
	(0.0574)	(0.0652)	(0.0771)	(0.0683)	(0.0951)
Age	0.00276	0.00259	0.00345	0.00286	0.00256
	(0.00192)	(0.00216)	(0.00244)	(0.00229)	(0.00282)
Sex	-0.377***	-0.411***	-0.432***	-0.413***	-0.516***
	(0.0499)	(0.0565)	(0.0638)	(0.0601)	(0.0738)
Black	0.806***	0.934***	0.998***	1.044***	1.063***
	(0.144)	(0.153)	(0.175)	(0.184)	(0.245)
Asian	0.311**	0.369**	0.471***	0.378**	0.488**
	(0.100)	(0.115)	(0.131)	(0.128)	(0.158)
Other	0.240	0.335*	0.403*	0.236	0.243
	(0.139)	(0.153)	(0.170)	(0.171)	(0.218)
high education	-0.0693	-0.0535	-0.0774	-0.0679	-0.100
	(0.0829)	(0.0937)	(0.108)	(0.101)	(0.131)
low education	0.0539	0.00703	-0.0120	0.0157	-0.0165
	(0.0738)	(0.0843)	(0.0956)	(0.0893)	(0.109)
high SES	0.0520	0.0292	-0.0245	0.0459	-0.0123
	(0.0776)	(0.0869)	(0.100)	(0.0945)	(0.126)
low SES	0.0939	0.0749	0.0378	0.0716	0.0681
	(0.0627)	(0.0720)	(0.0817)	(0.0763)	(0.0935)
live with both natural parents	-0.0378	-0.0446	-0.0955	-0.00311	-0.0811
	(0.0805)	(0.0912)	(0.101)	(0.0950)	(0.109)
mother married	-0.101	-0.0476	-0.0225	-0.0981	-0.0792
	(0.0644)	(0.0734)	(0.0832)	(0.0773)	(0.0949)
home owners	-0.0869	-0.0801	-0.124	-0.0971	-0.154
	(0.0703)	(0.0805)	(0.0906)	(0.0853)	(0.102)
private renters	-0.0853	-0.0269	0.0628	0.00171	0.0481
	(0.0969)	(0.110)	(0.119)	(0.114)	(0.133)
birth weight	0.535***	0.580***	0.579***	0.562***	0.568***
	(0.0514)	(0.0581)	(0.0661)	(0.0624)	(0.0757)
hospital stay (log)	0.0855*	0.0509	0.0310	0.0428	0.0740
	(0.0418)	(0.0471)	(0.0535)	(0.0502)	(0.0618)
planned pregnancy	-0.0714	-0.0761	0.0125	-0.0729	0.0497
	(0.0555)	(0.0629)	(0.0713)	(0.0669)	(0.0817)
Premature	0.405***	0.520***	0.495***	0.456***	0.435**
	(0.111)	(0.124)	(0.144)	(0.136)	(0.162)
mother obese	0.919***	0.825***	0.842***	0.851***	0.888***
	(0.0817)	(0.0961)	(0.111)	(0.103)	(0.127)
mother age at birth	0.0106*	0.00933	0.00779	0.00856	0.0106
	(0.00500)	(0.00567)	(0.00642)	(0.00602)	(0.00725)
smoker 1st trimester	0.276***	0.278***	0.283***	0.267***	0.212*
	(0.0631)	(0.0728)	(0.0833)	(0.0768)	(0.0933)
smoker 2nd trimester	0.334*	0.337	0.407*	0.362*	0.427*
	(0.154)	(0.174)	(0.186)	(0.177)	(0.196)
smoker 3rd trimester	0.327**	0.421***	0.455***	0.435***	0.431**
	(0.102)	(0.113)	(0.123)	(0.117)	(0.132)
alcohol during pregnancy	-0.0605*	-0.0683*	-0.0701	-0.0783*	-0.0677
	(0.0298)	(0.0343)	(0.0381)	(0.0366)	(0.0414)
mother in care at 16 years	-0.414	-0.511	-0.964*	-0.580	-0.914
	(0.320)	(0.367)	(0.479)	(0.389)	(0.481)
maternal longstanding illness	0.0389	0.0555	0.0994	0.0342	0.0401
	(0.0598)	(0.0681)	(0.0770)	(0.0729)	(0.0892)
Constant	-4.483***	-4.480***	-4.707***	-4.493***	-4.379***
	(0.772)	(0.871)	(0.984)	(0.926)	(1.135)
N	10707	8474	6643	7542	5026

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Logit model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Table A-9: Logit Model Estimating Obesity in Three Year Olds

	Obesity				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	-0.0986	-0.110	-0.145	-0.123	-0.370*
	(0.102)	(0.117)	(0.141)	(0.123)	(0.177)
Age	0.00446	0.00580	0.00858	0.00170	0.00652
	(0.00389)	(0.00427)	(0.00464)	(0.00481)	(0.00541)
Sex	-0.195*	-0.226*	-0.185	-0.189	-0.191
	(0.0885)	(0.1000)	(0.113)	(0.107)	(0.131)
Black	0.917***	1.008***	1.232***	0.972***	1.027**
	(0.207)	(0.224)	(0.249)	(0.279)	(0.374)
Asian	0.252	0.302	0.221	0.431	0.222
	(0.183)	(0.204)	(0.246)	(0.225)	(0.299)
Other	-0.0381	-0.132	-0.157	-0.0101	-0.379
	(0.264)	(0.317)	(0.371)	(0.333)	(0.518)
high education	0.159	0.169	0.171	0.253	0.221
	(0.154)	(0.179)	(0.214)	(0.195)	(0.258)
low education	0.147	0.230	0.399*	0.328	0.373
	(0.139)	(0.162)	(0.191)	(0.176)	(0.222)
high SES	0.00246	0.0860	0.180	0.0879	0.187
	(0.141)	(0.160)	(0.182)	(0.172)	(0.223)
low SES	0.0611	0.0986	0.0236	-0.00639	-0.134
	(0.112)	(0.130)	(0.149)	(0.138)	(0.168)
live with both natural parents	0.0967	0.0517	0.0702	0.0456	0.109
	(0.139)	(0.158)	(0.174)	(0.163)	(0.192)
mother married	-0.168	-0.0503	-0.154	-0.157	-0.158
	(0.114)	(0.132)	(0.148)	(0.139)	(0.169)
home owners	-0.430***	-0.340*	-0.327*	-0.396**	-0.444*
	(0.120)	(0.138)	(0.156)	(0.147)	(0.178)
private renters	-0.496**	-0.405	-0.377	-0.300	-0.143
	(0.188)	(0.210)	(0.232)	(0.213)	(0.242)
birth weight	0.535***	0.622***	0.570***	0.616***	0.485***
	(0.0893)	(0.101)	(0.116)	(0.109)	(0.134)
hospital stay (log)	0.0819	0.0676	0.0615	0.0760	0.0729
	(0.0739)	(0.0834)	(0.0948)	(0.0892)	(0.110)
planned pregnancy	0.0204	-0.0509	0.0394	-0.0735	-0.0717
	(0.0991)	(0.112)	(0.127)	(0.119)	(0.145)
Premature	0.622***	0.711***	0.639**	0.713**	0.488
	(0.183)	(0.205)	(0.236)	(0.218)	(0.271)
mother obese	0.798***	0.743***	0.764***	0.844***	0.930***
	(0.123)	(0.144)	(0.166)	(0.152)	(0.184)
mother age at birth	0.0270**	0.0178	0.0180	0.0234*	0.0313*
	(0.00865)	(0.00988)	(0.0111)	(0.0104)	(0.0124)
smoker 1st trimester	0.275*	0.295*	0.171	0.305*	0.0282
	(0.112)	(0.129)	(0.150)	(0.136)	(0.171)
smoker 2nd trimester	0.277	0.130	0.0585	0.168	0.116
	(0.276)	(0.329)	(0.364)	(0.331)	(0.371)
smoker 3rd trimester	0.284	0.412*	0.449*	0.449*	0.410
	(0.177)	(0.194)	(0.208)	(0.200)	(0.222)
alcohol during pregnancy	0.00984	-0.0141	-0.0204	-0.0318	-0.000573
	(0.0423)	(0.0529)	(0.0581)	(0.0575)	(0.0610)
mother in care at 16 years	-0.156	-0.129	0.0658	-0.258	0.0130
	(0.471)	(0.529)	(0.537)	(0.599)	(0.606)
maternal longstanding illness	-0.0999	-0.0768	-0.214	-0.138	-0.322
	(0.109)	(0.124)	(0.146)	(0.134)	(0.172)
Constant	-6.351***	-6.739***	-7.067***	-6.172***	-6.576***
	(0.793)	(0.885)	(0.982)	(0.974)	(1.134)
N	11200	8845	6949	7885	5290

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Logit model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Table A-10: Logit Model Estimating Obesity in Five Year Olds

	Obesity				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	-0.0916	-0.133	-0.243	-0.161	-0.412*
	(0.0980)	(0.112)	(0.138)	(0.119)	(0.176)
Age	0.00352	0.00322	0.00552	0.00400	0.00617
	(0.00328)	(0.00369)	(0.00420)	(0.00394)	(0.00480)
Sex	-0.272**	-0.258**	-0.305**	-0.303**	-0.340**
	(0.0854)	(0.0965)	(0.111)	(0.104)	(0.128)
Black	1.057***	1.193***	1.381***	1.104***	1.351***
	(0.201)	(0.214)	(0.245)	(0.269)	(0.354)
Asian	0.588***	0.638***	0.487*	0.581**	0.528
	(0.163)	(0.184)	(0.230)	(0.215)	(0.284)
Other	0.241	0.382	0.512	0.338	0.0423
	(0.235)	(0.256)	(0.291)	(0.288)	(0.433)
high education	-0.00204	0.0319	-0.0249	0.101	-0.146
	(0.152)	(0.175)	(0.211)	(0.194)	(0.249)
low education	0.229	0.251	0.341	0.345*	0.175
	(0.133)	(0.154)	(0.182)	(0.170)	(0.202)
high SES	0.0649	0.0911	0.0768	0.0976	0.277
	(0.143)	(0.162)	(0.191)	(0.177)	(0.237)
low SES	0.207	0.277*	0.201	0.259	0.281
	(0.109)	(0.127)	(0.146)	(0.137)	(0.171)
live with both natural parents	-0.0835	-0.0620	-0.0483	-0.0132	-0.0885
	(0.131)	(0.149)	(0.168)	(0.158)	(0.184)
mother married	-0.199	-0.166	-0.118	-0.252	-0.264
	(0.110)	(0.126)	(0.144)	(0.133)	(0.163)
home owners	0.0125	0.00777	-0.00593	0.0223	0.143
	(0.117)	(0.133)	(0.152)	(0.143)	(0.173)
private renters	-0.209	-0.0987	0.0223	-0.0408	0.277
	(0.173)	(0.190)	(0.207)	(0.199)	(0.222)
birth weight	0.592***	0.609***	0.569***	0.642***	0.659***
	(0.0851)	(0.0966)	(0.112)	(0.105)	(0.128)
hospital stay (log)	0.159*	0.126	0.129	0.153	0.207
	(0.0723)	(0.0816)	(0.0940)	(0.0877)	(0.108)
planned pregnancy	-0.0915	-0.00861	0.161	0.0406	0.205
	(0.0947)	(0.107)	(0.124)	(0.115)	(0.141)
Premature	0.454*	0.441*	0.440	0.553*	0.379
	(0.181)	(0.208)	(0.240)	(0.219)	(0.274)
mother obese	1.030***	0.946***	0.953***	0.968***	1.074***
	(0.116)	(0.137)	(0.160)	(0.148)	(0.177)
mother age at birth	0.0235**	0.0198*	0.0239*	0.0251*	0.0265*
	(0.00831)	(0.00945)	(0.0108)	(0.0101)	(0.0122)
smoker 1st trimester	0.422***	0.417***	0.494***	0.452***	0.482**
	(0.106)	(0.123)	(0.143)	(0.131)	(0.160)
smoker 2nd trimester	0.242	0.284	0.461	0.389	0.427
	(0.275)	(0.307)	(0.324)	(0.309)	(0.356)
smoker 3rd trimester	0.460**	0.526**	0.592**	0.596**	0.716***
	(0.164)	(0.181)	(0.200)	(0.186)	(0.211)
alcohol during pregnancy	-0.00206	-0.00899	-0.0361	-0.00554	-0.0616
	(0.0428)	(0.0488)	(0.0588)	(0.0488)	(0.0702)
mother in care at 16 years	-0.428	-0.525	-0.253	-0.324	-0.0419
	(0.522)	(0.604)	(0.615)	(0.600)	(0.609)
maternal longstanding illness	0.000202	0.0463	-0.0210	0.0630	0.0265
	(0.102)	(0.116)	(0.136)	(0.124)	(0.153)
Constant	-6.972***	-6.978***	-7.698***	-7.597***	-8.306***
	(0.997)	(1.122)	(1.288)	(1.209)	(1.470)
N	11744	9283	7278	8259	5541

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Logit model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Table A-11: Logit Model Estimating Obesity in Seven Year Olds

	Obesity				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	-0.242*	-0.286*	-0.500***	-0.317**	-0.704***
	(0.0995)	(0.113)	(0.139)	(0.120)	(0.178)
Age	0.00294	0.00292	0.00633	0.00384	0.00644
	(0.00340)	(0.00380)	(0.00434)	(0.00405)	(0.00501)
Sex	-0.297***	-0.321**	-0.276*	-0.321**	-0.303*
	(0.0879)	(0.0987)	(0.113)	(0.106)	(0.129)
Black	1.035***	1.155***	1.371***	1.190***	1.299***
	(0.216)	(0.228)	(0.263)	(0.277)	(0.385)
Asian	0.686***	0.730***	0.769***	0.693***	0.873***
	(0.164)	(0.187)	(0.221)	(0.207)	(0.256)
Other	0.532*	0.649**	0.680*	0.473	0.392
	(0.224)	(0.242)	(0.278)	(0.282)	(0.384)
high education	-0.128	-0.0257	-0.0633	0.0821	0.170
	(0.151)	(0.174)	(0.202)	(0.189)	(0.240)
low education	0.0824	0.200	0.170	0.218	0.125
	(0.131)	(0.154)	(0.176)	(0.166)	(0.204)
high SES	0.0684	0.163	0.196	0.129	0.243
	(0.144)	(0.158)	(0.180)	(0.173)	(0.224)
low SES	0.184	0.198	0.0412	0.234	0.162
	(0.111)	(0.128)	(0.146)	(0.137)	(0.168)
live with both natural parents	-0.0249	-0.136	-0.186	-0.121	-0.201
	(0.137)	(0.153)	(0.175)	(0.162)	(0.192)
mother married	-0.251*	-0.181	-0.0715	-0.163	-0.0749
	(0.112)	(0.127)	(0.147)	(0.135)	(0.167)
home owners	0.101	0.130	0.169	0.148	0.300
	(0.121)	(0.138)	(0.159)	(0.147)	(0.179)
private renters	0.0379	0.163	0.333	0.194	0.481*
	(0.169)	(0.185)	(0.202)	(0.195)	(0.222)
birth weight	0.431***	0.527***	0.493***	0.439***	0.503***
	(0.0882)	(0.0987)	(0.114)	(0.106)	(0.130)
hospital stay (log)	0.114	0.0783	-0.00350	0.133	0.0752
	(0.0740)	(0.0826)	(0.0947)	(0.0887)	(0.109)
planned pregnancy	-0.0769	0.0149	0.103	0.00257	0.0467
	(0.0966)	(0.109)	(0.126)	(0.117)	(0.143)
Premature	0.476**	0.627**	0.491*	0.548*	0.434
	(0.183)	(0.202)	(0.243)	(0.219)	(0.274)
mother obese	1.171***	1.081***	1.024***	1.114***	1.123***
	(0.118)	(0.138)	(0.163)	(0.148)	(0.181)
mother age at birth	0.0274**	0.0236*	0.0261*	0.0230*	0.0221
	(0.00856)	(0.00967)	(0.0112)	(0.0103)	(0.0126)
smoker 1st trimester	0.411***	0.379**	0.444**	0.426**	0.489**
	(0.110)	(0.126)	(0.146)	(0.133)	(0.163)
smoker 2nd trimester	0.149	0.0958	0.401	0.204	0.491
	(0.292)	(0.330)	(0.337)	(0.332)	(0.358)
smoker 3rd trimester	0.548**	0.585**	0.705***	0.614**	0.847***
	(0.167)	(0.184)	(0.201)	(0.191)	(0.213)
alcohol during pregnancy	-0.0937	-0.0862	-0.144	-0.146	-0.249*
	(0.0614)	(0.0672)	(0.0861)	(0.0805)	(0.114)
mother in care at 16 years	-0.410	-0.223	-1.134	-0.590	-1.052
	(0.596)	(0.601)	(1.018)	(0.729)	(1.020)
maternal longstanding illness	0.0361	0.0961	0.0683	0.0966	0.119
	(0.104)	(0.116)	(0.135)	(0.124)	(0.152)
Constant	-6.455***	-6.776***	-7.916***	-6.940***	-8.124***
	(1.366)	(1.531)	(1.759)	(1.635)	(2.022)
N	10707	8474	6643	7542	5026

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Logit model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Table A-12: Breastfeeding on Weight Status at 3 Years using Ordered Probit Models

	Weight Status				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	-0.0843**	-0.0923**	-0.117**	-0.0908**	-0.221***
	(0.0300)	(0.0339)	(0.0399)	(0.0352)	(0.0489)
Age	0.00122	0.00185	0.00266	0.00127	0.00247
	(0.00121)	(0.00136)	(0.00150)	(0.00143)	(0.00168)
Sex	-0.116***	-0.137***	-0.147***	-0.127***	-0.137***
	(0.0258)	(0.0291)	(0.0328)	(0.0308)	(0.0377)
Black	0.302***	0.318***	0.426***	0.284**	0.296*
	(0.0821)	(0.0879)	(0.101)	(0.110)	(0.149)
Asian	-0.121*	-0.140*	-0.168*	-0.102	-0.139
	(0.0563)	(0.0642)	(0.0763)	(0.0715)	(0.0922)
Other	0.0221	0.0334	0.0546	0.0522	-0.0515
	(0.0742)	(0.0830)	(0.0948)	(0.0908)	(0.124)
high education	0.0302	0.0523	0.0579	0.0607	0.107
	(0.0425)	(0.0481)	(0.0551)	(0.0511)	(0.0667)
low education	-0.0161	-0.00465	-0.0258	0.0105	0.00927
	(0.0385)	(0.0440)	(0.0500)	(0.0462)	(0.0575)
high SES	-0.0111	-0.00925	0.0214	-0.00244	0.0380
	(0.0397)	(0.0444)	(0.0506)	(0.0476)	(0.0626)
low SES	0.0610	0.0834*	0.0983*	0.0589	0.0466
	(0.0324)	(0.0370)	(0.0423)	(0.0390)	(0.0482)
live with both natural parents	-0.0119	-0.0295	-0.0308	-0.0278	-0.0235
	(0.0431)	(0.0487)	(0.0539)	(0.0506)	(0.0587)
mother married	-0.0764*	-0.0399	-0.0470	-0.0729	-0.0447
	(0.0336)	(0.0381)	(0.0430)	(0.0400)	(0.0493)
home owners	-0.0485	-0.0359	-0.00222	-0.0376	-0.0388
	(0.0373)	(0.0426)	(0.0481)	(0.0451)	(0.0541)
private renters	-0.136**	-0.105	-0.0700	-0.0711	0.00680
	(0.0527)	(0.0593)	(0.0657)	(0.0618)	(0.0724)
birth weight	0.402***	0.425***	0.413***	0.422***	0.400***
	(0.0267)	(0.0302)	(0.0345)	(0.0324)	(0.0395)
hospital stay (log)	0.0618**	0.0582*	0.0437	0.0467	0.0422
	(0.0217)	(0.0243)	(0.0276)	(0.0258)	(0.0319)
planned pregnancy	0.00279	-0.00595	-0.00908	-0.000660	0.00331
	(0.0292)	(0.0329)	(0.0371)	(0.0348)	(0.0426)
Premature	0.294***	0.325***	0.299***	0.342***	0.279**
	(0.0587)	(0.0661)	(0.0759)	(0.0707)	(0.0851)
mother obese	0.322***	0.274***	0.264***	0.320***	0.329***
	(0.0442)	(0.0515)	(0.0599)	(0.0546)	(0.0674)
mother age at birth	0.00571*	0.00250	0.00191	0.00213	0.00471
	(0.00262)	(0.00297)	(0.00335)	(0.00313)	(0.00377)
smoker 1st trimester	0.100**	0.105**	0.117**	0.102*	0.0946
	(0.0331)	(0.0380)	(0.0434)	(0.0398)	(0.0485)
smoker 2nd trimester	0.0916	0.0611	0.0754	0.0299	0.0431
	(0.0843)	(0.0945)	(0.103)	(0.0971)	(0.111)
smoker 3rd trimester	0.151**	0.158**	0.188**	0.164**	0.185**
	(0.0539)	(0.0609)	(0.0668)	(0.0626)	(0.0709)
alcohol during pregnancy	-0.00485	-0.00517	-0.00396	-0.00616	0.00804
	(0.0131)	(0.0146)	(0.0154)	(0.0150)	(0.0172)
mother in care at 16 years	0.00469	0.0541	0.119	-0.000501	0.0647
	(0.136)	(0.153)	(0.166)	(0.162)	(0.177)
maternal longstanding illness	-0.0437	-0.0462	-0.0593	-0.0478	-0.0871
	(0.0316)	(0.0360)	(0.0409)	(0.0382)	(0.0472)
cut1 constant	2.428***	2.543***	2.614***	2.407***	2.607***
	(0.242)	(0.272)	(0.305)	(0.287)	(0.342)
cut2 constant	3.376***	3.494***	3.573***	3.371***	3.571***
	(0.243)	(0.273)	(0.306)	(0.288)	(0.344)
N	11200	8845	6949	7885	5290

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Ordered probit model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Table A-13: Breastfeeding on Weight Status at 5 Years using Ordered Probit Models

	Weight Status				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	-0.0834**	-0.110**	-0.157***	-0.115**	-0.225***
	(0.0301)	(0.0341)	(0.0404)	(0.0356)	(0.0494)
Age	-0.000140	0.000229	0.000519	0.000305	-0.00000599
	(0.000997)	(0.00112)	(0.00126)	(0.00118)	(0.00145)
Sex	-0.200***	-0.224***	-0.233***	-0.227***	-0.230***
	(0.0260)	(0.0293)	(0.0331)	(0.0311)	(0.0380)
Black	0.505***	0.539***	0.573***	0.515***	0.524***
	(0.0768)	(0.0820)	(0.0944)	(0.0995)	(0.135)
Asian	0.0894	0.105	0.0946	0.0955	0.161
	(0.0537)	(0.0611)	(0.0726)	(0.0694)	(0.0877)
Other	0.104	0.168*	0.226*	0.123	0.0441
	(0.0731)	(0.0800)	(0.0915)	(0.0894)	(0.119)
high education	-0.0115	-0.00638	-0.00818	0.0144	-0.0388
	(0.0432)	(0.0487)	(0.0565)	(0.0523)	(0.0678)
low education	0.0233	0.0157	0.00446	0.0375	-0.0308
	(0.0388)	(0.0441)	(0.0504)	(0.0467)	(0.0571)
high SES	0.0405	0.0235	0.0261	0.0200	0.0715
	(0.0404)	(0.0451)	(0.0518)	(0.0486)	(0.0647)
low SES	0.0759*	0.0701	0.0580	0.0500	0.0808
	(0.0327)	(0.0374)	(0.0427)	(0.0395)	(0.0490)
live with both natural parents	-0.0322	-0.0508	-0.0531	-0.0416	-0.0821
	(0.0423)	(0.0478)	(0.0532)	(0.0499)	(0.0577)
mother married	-0.0608	-0.0461	-0.0348	-0.0702	-0.0553
	(0.0339)	(0.0384)	(0.0434)	(0.0404)	(0.0496)
home owners	-0.0159	-0.00798	0.00304	-0.00747	0.0111
	(0.0370)	(0.0421)	(0.0475)	(0.0447)	(0.0533)
private renters	-0.149**	-0.108	-0.0682	-0.102	-0.0646
	(0.0521)	(0.0587)	(0.0650)	(0.0616)	(0.0727)
birth weight	0.372***	0.388***	0.392***	0.391***	0.407***
	(0.0266)	(0.0301)	(0.0345)	(0.0323)	(0.0396)
hospital stay (log)	0.0789***	0.0793**	0.0699*	0.0834**	0.0889**
	(0.0218)	(0.0245)	(0.0279)	(0.0261)	(0.0321)
planned pregnancy	-0.0463	-0.0137	0.0117	-0.0222	0.0534
	(0.0292)	(0.0330)	(0.0374)	(0.0350)	(0.0427)
Premature	0.228***	0.239***	0.218**	0.244***	0.150
	(0.0583)	(0.0659)	(0.0762)	(0.0711)	(0.0864)
mother obese	0.477***	0.425***	0.441***	0.444***	0.443***
	(0.0444)	(0.0520)	(0.0601)	(0.0554)	(0.0684)
mother age at birth	0.00521*	0.00429	0.00354	0.00528	0.00627
	(0.00263)	(0.00297)	(0.00335)	(0.00314)	(0.00377)
smoker 1st trimester	0.158***	0.173***	0.191***	0.183***	0.209***
	(0.0331)	(0.0380)	(0.0434)	(0.0398)	(0.0482)
smoker 2nd trimester	0.118	0.133	0.196	0.120	0.211
	(0.0841)	(0.0946)	(0.101)	(0.0974)	(0.108)
smoker 3rd trimester	0.210***	0.225***	0.237***	0.251***	0.282***
	(0.0528)	(0.0592)	(0.0651)	(0.0609)	(0.0689)
alcohol during pregnancy	-0.0104	-0.0140	-0.0158	-0.0135	-0.0168
	(0.0136)	(0.0153)	(0.0165)	(0.0157)	(0.0189)
mother in care at 16 years	-0.112	-0.231	-0.344	-0.236	-0.406
	(0.145)	(0.172)	(0.208)	(0.182)	(0.224)
maternal longstanding illness	0.000665	-0.00180	0.0160	-0.00386	0.0316
	(0.0316)	(0.0360)	(0.0408)	(0.0383)	(0.0467)
cut1	2.199***	2.324***	2.398***	2.387***	2.391***
_cons	(0.304)	(0.340)	(0.386)	(0.361)	(0.441)
cut2	3.058***	3.186***	3.276***	3.263***	3.277***
_cons	(0.304)	(0.341)	(0.387)	(0.362)	(0.442)
N	11744	9283	7278	8259	5541

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Ordered probit model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Table A-14: Breastfeeding on Weight Status at 7 Years using Ordered Probit Models

	Weight Status				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	-0.0915**	-0.130***	-0.162***	-0.139***	-0.229***
	(0.0318)	(0.0360)	(0.0424)	(0.0376)	(0.0517)
Age	0.00150	0.00150	0.00235	0.00170	0.00191
	(0.00106)	(0.00120)	(0.00135)	(0.00127)	(0.00156)
Sex	-0.202***	-0.220***	-0.226***	-0.220***	-0.267***
	(0.0275)	(0.0310)	(0.0351)	(0.0330)	(0.0405)
Black	0.482***	0.555***	0.603***	0.602***	0.618***
	(0.0820)	(0.0872)	(0.0995)	(0.105)	(0.140)
Asian	0.217***	0.251***	0.302***	0.254***	0.325***
	(0.0551)	(0.0630)	(0.0729)	(0.0705)	(0.0876)
Other	0.165*	0.222**	0.257**	0.156	0.156
	(0.0776)	(0.0850)	(0.0951)	(0.0945)	(0.120)
high education	-0.0513	-0.0341	-0.0456	-0.0322	-0.0388
	(0.0455)	(0.0514)	(0.0594)	(0.0552)	(0.0718)
low education	0.0297	0.0192	0.00929	0.0244	0.00306
	(0.0407)	(0.0465)	(0.0530)	(0.0492)	(0.0605)
high SES	0.0319	0.0306	0.00607	0.0339	0.0158
	(0.0424)	(0.0474)	(0.0545)	(0.0513)	(0.0684)
low SES	0.0595	0.0522	0.0219	0.0511	0.0438
	(0.0345)	(0.0397)	(0.0452)	(0.0421)	(0.0517)
live with both natural parents	-0.0186	-0.0300	-0.0582	-0.00676	-0.0526
	(0.0450)	(0.0510)	(0.0565)	(0.0532)	(0.0613)
mother married	-0.0709*	-0.0423	-0.0187	-0.0650	-0.0469
	(0.0357)	(0.0406)	(0.0461)	(0.0429)	(0.0527)
home owners	-0.0328	-0.0251	-0.0446	-0.0306	-0.0435
	(0.0390)	(0.0446)	(0.0503)	(0.0473)	(0.0565)
private renters	-0.0335	0.00605	0.0645	0.0217	0.0726
	(0.0537)	(0.0608)	(0.0665)	(0.0635)	(0.0745)
birth weight	0.287***	0.317***	0.316***	0.302***	0.310***
	(0.0283)	(0.0320)	(0.0366)	(0.0344)	(0.0420)
hospital stay (log)	0.0494*	0.0296	0.0114	0.0303	0.0364
	(0.0230)	(0.0259)	(0.0296)	(0.0276)	(0.0341)
planned pregnancy	-0.0390	-0.0326	0.0164	-0.0331	0.0317
	(0.0307)	(0.0348)	(0.0394)	(0.0369)	(0.0452)
Premature	0.227***	0.296***	0.275***	0.260***	0.243**
	(0.0614)	(0.0690)	(0.0800)	(0.0755)	(0.0900)
mother obese	0.558***	0.504***	0.506***	0.521***	0.541***
	(0.0469)	(0.0551)	(0.0638)	(0.0589)	(0.0725)
mother age at birth	0.00751**	0.00641*	0.00612	0.00589	0.00684
	(0.00277)	(0.00313)	(0.00355)	(0.00333)	(0.00401)
smoker 1st trimester	0.163***	0.161***	0.171***	0.160***	0.140**
	(0.0350)	(0.0403)	(0.0462)	(0.0425)	(0.0517)
smoker 2nd trimester	0.174*	0.167	0.233*	0.191	0.254*
	(0.0863)	(0.0975)	(0.105)	(0.0991)	(0.110)
smoker 3rd trimester	0.197***	0.245***	0.273***	0.255***	0.278***
	(0.0571)	(0.0635)	(0.0694)	(0.0655)	(0.0738)
alcohol during pregnancy	-0.0360*	-0.0387*	-0.0442*	-0.0473*	-0.0478*
	(0.0160)	(0.0183)	(0.0207)	(0.0197)	(0.0229)
mother in care at 16 years	-0.208	-0.230	-0.514*	-0.290	-0.484*
	(0.171)	(0.192)	(0.242)	(0.204)	(0.245)
maternal longstanding illness	0.0225	0.0363	0.0540	0.0264	0.0312
	(0.0332)	(0.0378)	(0.0429)	(0.0404)	(0.0495)
cut1	2.572***	2.631***	2.906***	2.647***	2.741***
constant	(0.427)	(0.482)	(0.547)	(0.512)	(0.628)
Cut2	3.376***	3.423***	3.717***	3.441***	3.553***
Constant	(0.427)	(0.483)	(0.548)	(0.513)	(0.629)
N	10707	8474	6643	7542	5026

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Logit model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks.

Table A-15: Propensity Score Matching with Outcome BMI

Treatment	# NN (calliper)	ATT (s.e.^)	ATT sample size (com. support)	ATU	ATU sample size (com. support)	ATE (s.e.€ ^)	ATE sample size (com. support)	ATE 95% CI
Age 3								
Ever breastfed	1	-0.0448	6,196	-0.0282	3,134	-0.0392	9,330	(-0.1214,
	(0.00024)	(0.0518)	(79.9%)	(0.0223)	(90.9%)	(0.0419)	(83.3%)	0.0430)
Partial Breastfeeding								
> 4 weeks compared to never	2	-0.0174	4,724	-0.0570*	3,153	-0.0333	7,877	(-0.1254,
breastfed	(0.0005)	(0.0585)	(87.5%)	(0.0298)	(91.5%)	(0.0470)	(89.1%)	0.0589)
> 16 weeks compared to never	3	-0.0088	2,835	-0.0083	2,700	-0.0086	5,602	(-0.2291,
breastfed	(0.0005)	(0.0087)	(80.9%)	(0.0068)	(78.4%)	(0.0077)	(80.2%)	-0.0013)
Exclusive breastfeeding								
> 4 weeks compared to never	3	-0.0512	4,178	-0.0231	3,279	-0.0388	7,457	(-0.1446,
breastfed	(0.001)	(0.0580)	(94.1%)	(0.0318)	(95.2%)	(0.0465)	(94.6%)	0.0204)
> 16 weeks compared to never	3	-0.1310*	1,822	-0.1746**	3,361	-0.1592**	5,183	(-0.3131,
breastfed	(0.01)	(0.0790)	(98.8%)	(0.0768)	(97.5%)	(0.0785)	(98.0%)	-0.0054)
Age 5								
Ever breastfed	1	-0.0837	6,726	-0.0669**	3,270	-0.0782	9,996	(-0.1675,
	(0.00025)	(0.0535)	(82.8%)	(0.0294)	(90.4%)	(0.0456)	(85.1%)	0.0112)
Partial Breastfeeding								
> 4 weeks compared to never	2	-0.0977*	4,080	-0.1246*	2,778	-0.1086**	6,858	(-0.2135,
breastfed	(0.00025)	(0.0569)	(72.0%)	(0.0749)	(76.8%)	(0.0535)	(73.9%)	0.0036)
> 16 weeks compared to never	3	-0.1809***	2,439	-0.1735**	2,402	-0.1772**	4,841	(-0.3117,
breastfed	(0.0003)	(0.0651)	(66.6%)	(0.0722)	(66.4%)	(0.0686)	(66.5%)	-0.0428)
Exclusive breastfeeding								
> 4 weeks compared to never	3	-0.1623***	4,363	-0.1121***	3,466	-0.1401***	7,829	(-0.2349,
breastfed	(0.0009)	(0.0597)	(94.0%)	(0.0342)	(95.8%)	(0.0484)	(94.8%)	-0.0453)
> 16 weeks compared to never	3	-0.2176***	1,883	-0.1954**	3,540	-0.2031**	5,423	(-0.3646,
breastfed	(0.01)	(0.0794)	(97.9%)	(0.0840)	(97.9%)	(0.0824)	(97.9%)	-0.0415)
Age 7								
Ever breastfed	1	-0.1880**	5,565	-0.1019**	2,807	-0.1591**	8,372	(-0.2908,
	(0.0002)	(0.0773)	(74.4%)	(0.0472)	(86.9%)	(0.0672)	(78.2%)	0.0274)
Partial Breastfeeding								
> 4 weeks compared to never	2	-0.1542*	3,697	-0.1850***	2,471	-0.1665**	6,168	(-0.3168,
breastfed	(0.00025)	(0.0841)	(70.1%)	(0.0656)	(76.5%)	(0.0767)	(8,474%)	-0.0162)
> 16 weeks compared to never	3	-0.2139**	3,360	-0.2709***	3,174	-0.2416***	6,534	(-0.3908,
breastfed	(0.0003)	(0.1019)	(98.5%)	(0.0488)	(98.2%)	(0.0761)	(98.4%)	-0.0924)
Exclusive breastfeeding								
> 4 weeks compared to never	3	-0.1845**	4,062	-0.2370***	3,105	-0.2072***	7,167	(-0.3528,
breastfed	(0.001)	(0.0867)	(94.2%)	(0.0581)	(96.1%)	(0.0743)	(95.0%)	-0.0616)
> 16 weeks compared to never	3	-0.3674***	1,762	-0.2258**	3,186	-0.2762**	4,948	(-0.4873,
breastfed	(0.01)	(0.1131)	(98.2%)	(0.1047)	(98.6%)	(0.1077)	(98.4%)	-0.0652)

Source: Millennium Cohort Study. Notes: * p < 0.1, ** p < 0.05, *** p < 0.01. €bootstrap standard error (500 repetitions). ^Standard errors assume propensity score is known.

Table A-16: Propensity Score Matching with Binary Outcome Obesity

Treatment	# NN (calliper)	ATT (s.e.^)	ATT sample size (com. support)	ATU (s.e.)	ATU sample size (com. support)	ATE (s.e.[€] ^)	ATE sample size (com. support)	ATE 95% CI
Age 3								
Ever breastfed	1 (0.00026)	-0.0037 (0.0068)	6,269 (80.8%)	-0.0041 (0.0047)	3,144 (91.2%)	0.0011 (0.0061)	9,413 (84.0%)	(-0.0108, 0.0130)
Partial Breastfeeding								
> 4 weeks compared to never breastfed	3 (0.001)	-0.0018 (0.0084)	5,116 (94.8%)	-0.0052** (0.0023)	3,329 (96.6%)	-0.0031 (0.0060)	8,445 (95.5%)	(-0.0149, -0.0087)
> 16 weeks compared to never breastfed	3 (0.0005)	-0.0048 (0.0089)	2,820 (80.5%)	-0.0136** (0.0065)	2,723 (79.0%)	-0.0091 (0.0077)	5,543 (79.8%)	(-0.0242, -0.0059)
Exclusive breastfeeding								
> 4 weeks compared to never breastfed	3 (0.001)	-0.0006 (0.0083)	4,405 (99.2%)	-0.0087* (0.0051)	3,439 (99.8%)	-0.0042 (0.0069)	7,844 (99.5%)	(-0.0176, -0.0093)
> 16 weeks compared to never breastfed	3 (0.01)	-0.0088 (0.0102)	1,822 (98.8%)	-0.0209*** (0.0068)	3,361 (97.5%)	-0.0167** (0.0080)	5,183 (98.0%)	(-0.032, -0.0009)
Age 5								
Ever breastfed	1 (0.00025)	-0.0010 (0.0070)	7,657 (94.2%)	-0.0062* (0.0035)	3,479 (96.2%)	-0.0026 (0.0059)	11,136 (94.8%)	(-0.0141, -0.0089)
Partial Breastfeeding								
> 4 weeks compared to never breastfed	3 (0.001)	-0.0010 (0.0080)	5,433 (95.9%)	-0.0087*** (0.0034)	3,520 (97.3%)	-0.0040 (0.0062)	8,953 (96.4%)	(-0.0162, -0.0082)
> 16 weeks compared to never breastfed	3 (0.0004)	-0.0146* (0.0087)	2,439 (66.6%)	-0.0062 (0.0077)	2,402 (66.4%)	-0.0104 (0.0082)	4,841 (66.5%)	(-0.0265, -0.0057)
Exclusive breastfeeding								
> 4 weeks compared to never breastfed	3 (0.0009)	-0.0091 (0.0081)	4,518 (97.3%)	-0.0065 (0.0040)	3,565 (98.6%)	-0.0080 (0.0063)	8,083 (97.9%)	(-0.0203, -0.0043)
> 16 weeks compared to never breastfed	3 (0.01)	-0.0198** (0.0099)	1,883 (97.87%)	0.0025 (0.0105)	3,540 (97.9%)	-0.0052 (0.0103)	5,423 (97.9%)	(-0.0253, -0.0149)
Age 7								
Ever breastfed	2 (0.0004)	-0.0216*** (0.0080)	6,684 (89.4%)	-0.0097*** (0.0038)	3,033 (93.9%)	-0.0179*** (0.0067)	9,717 (90.8%)	(-0.0311, -0.0047)
Partial Breastfeeding								
> 4 weeks compared to never breastfed	3 (0.00035)	-0.0267*** (0.0092)	4,955 (94.5%)	-0.0115*** (0.0029)	3,078 (95.3%)	-0.0209*** (0.0068)	8,033 (94.8%)	(-0.0342, -0.0075)
> 16 weeks compared to never breastfed	3 (0.0004)	-0.0283*** (0.0098)	2,531 (74.2%)	-0.0200*** (0.0071)	2,330 (72.1%)	-0.0243*** (0.0085)	4,861 (73.2%)	(-0.0411, -0.0076)
Exclusive breastfeeding								
> 4 weeks compared to never breastfed	3 (0.001)	-0.0245** (0.0095)	4,279 (99.3%)	-0.0085 (0.0067)	3,231 (100%)	-0.0176** (0.0083)	7,510 (99.6%)	(-0.0338, -0.0013)
> 16 weeks compared to never breastfed	3 (0.01)	-0.0272** (0.0112)	1,762 (98.2%)	-0.0233*** (0.0087)	3,186 (98.6%)	-0.0247*** (0.0096)	4,948 (98.4%)	(-0.0435, 0.0059)

Source: Millennium Cohort Study. Notes: * p < 0.1, ** p < 0.05, *** p < 0.01. [€]bootstrap standard error (500 repetitions). ^Standard errors assume propensity score is known.

Table A-17: Propensity Score Matching with Binary Outcome Overweight

Treatment	# NN (calliper)	ATT (s.e.^)	ATT sample size (com. support)	ATU (s.e.)	ATU sample size (com. support)	ATE (s.e.$^{\epsilon}$ ^)	ATE sample size (com. support)	ATE 95% CI
Age 3								
Ever breastfed	2 (0.00025)	-0.0190 (0.0140)	6,322 (81.5%)	-0.0133* (0.0074)	3,161 (91.7%)	-0.0171 (0.0118)	9,483 (84.7%)	(-0.0402, 0.0061)
Partial Breastfeeding								
> 4 weeks compared to never breastfed	3 (0.001)	-0.0175 (0.0160)	5,116 (94.8%)	-0.0298*** (0.0081)	3,329 (96.6%)	-0.0284** (0.0129)	8,445 (95.5%)	(-0.0537, 0.0032)
> 16 weeks compared to never breastfed	3 (0.0005)	-0.0433*** (0.0173)	2,820 (80.5%)	-0.0397*** (0.0063)	5,543 (79.8%)	-0.0415*** (0.0151)	5,543 (79.8%)	(-0.0710, 0.0120)
Exclusive breastfeeding								
> 4 weeks compared to never breastfed	3 (0.1)	-0.0378** (0.0164)	4,384 (98.8%)	-0.0296*** (0.0091)	3,439 (99.8%)	-0.0342** (0.0132)	7,823 (99.2%)	(-0.0601, 0.0083)
> 16 weeks compared to never breastfed	5 (0.01)	-0.0666*** (0.0206)	1,822 (98.8%)	-0.0575*** (0.0198)	3,361 (97.5%)	-0.0607*** (0.0201)	5,183 (98.0%)	(-0.1001, -0.0213)
Age 5								
Ever breastfed	2 (0.0005)	-0.03600*** (0.0132)	6,726 (82.8%)	-0.0217*** (0.0053)	3,270 (90.4%)	-0.0313*** (0.0106)	9,996 (85.12%)	(-0.0521, 0.0105)
Partial Breastfeeding								
> 4 weeks compared to never breastfed	3 (0.001)	-0.0343** (0.0150)	5,433 (96.0%)	-0.0256*** (0.0092)	3,520 (97.3%)	0.0308** (0.0127)	8,953 (96.4%)	(0.0558, 0.0059)
> 16 weeks compared to never breastfed	3 (0.0003)	-0.0542*** (0.0164)	2,710 (74.0%)	-0.0468*** (0.0134)	2,684 (74.2%)	-0.0505*** (0.0149)	5,394 (74.1%)	(-0.0798, 0.0212)
Exclusive breastfeeding								
> 4 weeks compared to never breastfed	3 (0.0025)	-0.0532*** (0.0148)	4,363 (94.0%)	-0.0339*** (0.0087)	3,466 (95.8%)	-0.0446*** (0.0121)	7,829 (94.8%)	(-0.0683, 0.0210)
> 16 weeks compared to never breastfed	5 (0.01)	-0.0656*** (0.0192)	1,883 (97.9%)	-0.0519*** (0.0194)	3,540 (97.9%)	-0.0566*** (0.0193)	5,423 (97.9%)	(-0.0946, 0.0187)
Age 7								
Ever breastfed	2 (0.0004)	-0.0382*** (0.0138)	6,684 (89.4%)	-0.0213*** (0.0064)	3,033 (93.9%)	-0.0329*** (0.0115)	9,717 (90.8%)	(-0.0555, -0.0104)
Partial Breastfeeding								
> 4 weeks compared to never breastfed	3 (0.00025)	-0.0312** (0.0148)	4,171 (79.6%)	-0.0320*** (0.0089)	2,696 (83.9%)	-0.0315** (0.0125)	6,867 (81.0%)	(-0.0560, -0.0070)
> 16 weeks compared to never breastfed	3 (0.0004)	-0.0328* (0.0182)	3,360 (98.5%)	-0.0326*** (0.0112)	3,174 (98.2%)	-0.0327** (0.0148)	6,534 (98.4%)	(-0.0618, 0.0036)
Exclusive breastfeeding								
> 4 weeks compared to never breastfed	3 (0.1)	-0.0523*** (0.0163)	4,279 (99.3%)	-0.0270*** (0.0089)	3,230 (>99.9%)	-0.0414*** (0.0131)	7,509 (99.6%)	(-0.0671, 0.0158)
> 16 weeks compared to never breastfed	5 (0.01)	-0.0729*** (0.0205)	1,762 (98.2%)	-0.0140*** (0.0050)	3,186 (98.6%)	-0.0350*** (0.0105)	4,948 (98.4%)	(-0.0752, -0.0052)

Source: Millennium Cohort Study. Notes: * p < 0.1, ** p < 0.05, *** p < 0.01. $^{\epsilon}$bootstrap standard error (500 repetitions). ^Standard errors assume propensity score is known.

Table A-18: First Stage of 2SLS Estimating Breastfeeding in Three Year Olds

	BMI				
	(1)	(2)	(3)	(4)	(5)
Delivered by Caesarean	-0.0362**	-0.0441***	-0.0511***	-0.0565***	-0.0462**
	(0.0113)	(0.0129)	(0.0142)	(0.0141)	(0.0158)
Age	-0.000565	-0.000715	-0.000357	-0.000618	-0.000417
	(0.000380)	(0.000428)	(0.000462)	(0.000461)	(0.000494)
Sex	0.0100	0.0102	0.00604	0.00808	-0.0138
	(0.00804)	(0.00906)	(0.00986)	(0.00978)	(0.0108)
Black	0.299***	0.360***	0.394***	0.332***	0.369***
	(0.0272)	(0.0289)	(0.0323)	(0.0365)	(0.0444)
Asian	0.206***	0.253***	0.275***	0.224***	0.256***
	(0.0160)	(0.0181)	(0.0205)	(0.0207)	(0.0237)
Other	0.203***	0.234***	0.248***	0.225***	0.234***
	(0.0229)	(0.0256)	(0.0284)	(0.0287)	(0.0341)
high education	0.0806***	0.1000***	0.133***	0.108***	0.175***
	(0.0132)	(0.0149)	(0.0165)	(0.0162)	(0.0189)
low education	-0.0894***	-0.118***	-0.135***	-0.122***	-0.0980***
	(0.0120)	(0.0136)	(0.0150)	(0.0147)	(0.0164)
high SES	0.0464***	0.0669***	0.0926***	0.0793***	0.132***
	(0.0123)	(0.0137)	(0.0150)	(0.0150)	(0.0177)
low SES	-0.0937***	-0.107***	-0.119***	-0.106***	-0.0952***
	(0.0101)	(0.0115)	(0.0127)	(0.0124)	(0.0138)
live with both natural parents	0.0938***	0.0885***	0.0770***	0.0806***	0.0530**
	(0.0136)	(0.0155)	(0.0166)	(0.0164)	(0.0172)
mother married	0.0120	0.0232	0.0221	0.0253*	0.0354*
	(0.0106)	(0.0120)	(0.0131)	(0.0129)	(0.0143)
home owners	0.0402***	0.0387**	0.0220	0.0385**	0.00427
	(0.0117)	(0.0134)	(0.0146)	(0.0144)	(0.0156)
private renters	0.0628***	0.0708***	0.0697***	0.0720***	0.0417*
	(0.0161)	(0.0183)	(0.0197)	(0.0196)	(0.0212)
birth weight	-0.00239	-0.00000455	0.0100	-0.00202	0.00547
	(0.00831)	(0.00936)	(0.0103)	(0.0102)	(0.0114)
hospital stay (log)	0.0403***	0.0309***	0.0211*	0.0283**	0.0131
	(0.00771)	(0.00869)	(0.00957)	(0.00941)	(0.0106)
planned pregnancy	0.0293**	0.0346***	0.0297**	0.0328**	0.0165
	(0.00909)	(0.0103)	(0.0112)	(0.0111)	(0.0122)
Premature	-0.0216	-0.0279	-0.0696**	-0.0492*	-0.0687**
	(0.0183)	(0.0207)	(0.0230)	(0.0227)	(0.0247)
mother obese	-0.00659	-0.0344*	-0.0840***	-0.0468*	-0.0956***
	(0.0150)	(0.0173)	(0.0194)	(0.0189)	(0.0211)
mother age at birth	0.00385***	0.00792***	0.0103***	0.00827***	0.0106***
	(0.000825)	(0.000929)	(0.00101)	(0.00100)	(0.00109)
smoker 1st trimester	-0.0267*	-0.0602***	-0.111***	-0.0647***	-0.0975***
	(0.0105)	(0.0121)	(0.0133)	(0.0129)	(0.0141)
smoker 2nd trimester	-0.125***	-0.143***	-0.133***	-0.124***	-0.118***
	(0.0266)	(0.0297)	(0.0311)	(0.0309)	(0.0319)
smoker 3rd trimester	-0.130***	-0.161***	-0.184***	-0.160***	-0.149***
	(0.0172)	(0.0195)	(0.0208)	(0.0205)	(0.0211)
alcohol during pregnancy	-0.000441	-0.000650	0.00394	-0.000200	0.00197
	(0.00407)	(0.00460)	(0.00471)	(0.00481)	(0.00527)
mother in care at 16 years	-0.0158	-0.0428	-0.0386	-0.0441	0.0218
	(0.0425)	(0.0491)	(0.0524)	(0.0521)	(0.0524)
maternal longstanding illness	0.0172	0.00363	-0.00844	-0.00464	-0.0303*
	(0.00982)	(0.0112)	(0.0123)	(0.0121)	(0.0135)
Constant	0.551***	0.390***	0.184*	0.356***	0.0921
	(0.0753)	(0.0852)	(0.0925)	(0.0917)	(0.0991)
N	11200	8845	6949	7885	5290

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Stage one of IV model estimating breastfeeding treatments; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks. Instrument is a binary variable indicating delivery by Caesarean section.

Table A-19: First Stage of 2SLS Estimating Breastfeeding in Five Year Olds

	BMI				
	(1)	(2)	(3)	(4)	(5
Delivered by Caesarean	-0.0441***	-0.0546***	-0.0655***	-0.0667***	-0.0589***
	(0.0111)	(0.0126)	(0.0139)	(0.0138)	(0.0155)
Age	0.000362	0.000390	0.000304	0.000266	-0.000127
	(0.000301)	(0.000337)	(0.000366)	(0.000364)	(0.000402)
Sex	0.000500	-0.000144	0.000473	-0.00177	-0.0119
	(0.00782)	(0.00880)	(0.00959)	(0.00951)	(0.0106)
Black	0.287***	0.350***	0.388***	0.316***	0.337***
	(0.0258)	(0.0274)	(0.0301)	(0.0340)	(0.0409)
Asian	0.211***	0.261***	0.284***	0.228***	0.257***
	(0.0155)	(0.0176)	(0.0200)	(0.0203)	(0.0234)
Other	0.213***	0.252***	0.264***	0.242***	0.263***
	(0.0223)	(0.0246)	(0.0274)	(0.0277)	(0.0325)
high education	0.0754***	0.0929***	0.132***	0.102***	0.165***
	(0.0129)	(0.0145)	(0.0162)	(0.0158)	(0.0186)
low education	-0.0955***	-0.126***	-0.134***	-0.130***	-0.106***
	(0.0117)	(0.0133)	(0.0147)	(0.0143)	(0.0161)
high SES	0.0483***	0.0718***	0.0975***	0.0867***	0.147***
	(0.0120)	(0.0134)	(0.0147)	(0.0146)	(0.0175)
low SES	-0.0949***	-0.104***	-0.117***	-0.100***	-0.0907***
	(0.00982)	(0.0112)	(0.0124)	(0.0121)	(0.0136)
live with both natural parents	0.0910***	0.0820***	0.0725***	0.0743***	0.0513**
	(0.0131)	(0.0148)	(0.0159)	(0.0157)	(0.0165)
mother married	0.0269**	0.0385***	0.0377**	0.0392**	0.0485***
	(0.0103)	(0.0116)	(0.0127)	(0.0125)	(0.0139)
home owners	0.0440***	0.0443***	0.0288*	0.0446**	0.00550
	(0.0113)	(0.0129)	(0.0140)	(0.0139)	(0.0151)
private renters	0.0616***	0.0667***	0.0712***	0.0699***	0.0462*
	(0.0154)	(0.0175)	(0.0188)	(0.0187)	(0.0201)
birth weight	0.00344	0.00574	0.0185	0.00558	0.00901
	(0.00803)	(0.00904)	(0.00997)	(0.00987)	(0.0110)
hospital stay (log)	0.0473***	0.0363***	0.0290**	0.0350***	0.0238*
	(0.00748)	(0.00842)	(0.00929)	(0.00912)	(0.0103)
planned pregnancy	0.0225*	0.0278**	0.0208	0.0244*	0.00818
	(0.00884)	(0.00996)	(0.0109)	(0.0108)	(0.0119)
Premature	-0.0193	-0.0234	-0.0599**	-0.0451*	-0.0588*
	(0.0175)	(0.0198)	(0.0221)	(0.0218)	(0.0237)
mother obese	-0.00798	-0.0356*	-0.0861***	-0.0494**	-0.0990***
	(0.0151)	(0.0175)	(0.0197)	(0.0191)	(0.0214)
mother age at birth	0.00348***	0.00767***	0.00997***	0.00782***	0.00985***
	(0.000805)	(0.000905)	(0.000982)	(0.000975)	(0.00106)
smoker 1st trimester	-0.0277**	-0.0602***	-0.108***	-0.0659***	-0.0952***
	(0.0102)	(0.0117)	(0.0128)	(0.0125)	(0.0136)
smoker 2nd trimester	-0.116***	-0.142***	-0.121***	-0.131***	-0.118***
	(0.0259)	(0.0292)	(0.0304)	(0.0304)	(0.0312)
smoker 3rd trimester	-0.130***	-0.152***	-0.166***	-0.155***	-0.126***
	(0.0165)	(0.0185)	(0.0196)	(0.0195)	(0.0200)
alcohol during pregnancy	0.000721	0.000586	0.00273	0.000809	-0.000343
	(0.00395)	(0.00442)	(0.00457)	(0.00464)	(0.00511)
mother in care at 16 years	0.00577	-0.0141	-0.0510	-0.0231	0.0263
	(0.0432)	(0.0494)	(0.0548)	(0.0529)	(0.0541)
maternal longstanding illness	0.0151	0.00611	-0.00651	-0.00238	-0.0297*
	(0.00959)	(0.0109)	(0.0120)	(0.0118)	(0.0132)
Constant	0.350***	0.157	0.0128	0.169	0.0573
	(0.0916)	(0.103)	(0.112)	(0.111)	(0.123)
N	11744	9283	7278	8259	5541

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Stage one of IV model estimating breastfeeding treatments; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks. Instrument is a binary variable indicating delivery by Caesarean section.

Table A-20: First Stage of 2SLS Estimating Breastfeeding in Seven Year Olds

	BMI				
	(1)	(2	(3)	(4)	(5)
Delivered by Caesarean	-0.0331**	-0.0422**	-0.0504***	-0.0504***	-0.0401*
	(0.0116)	(0.0132)	(0.0146)	(0.0145)	(0.0164)
Age	0.000536	0.000830*	0.000748	0.000623	0.0000680
	(0.000317)	(0.000356)	(0.000389)	(0.000385)	(0.000434)
Sex	0.00616	0.00205	0.00517	0.00260	-0.00973
	(0.00818)	(0.00923)	(0.0101)	(0.00998)	(0.0112)
Black	0.291***	0.351***	0.393***	0.325***	0.368***
	(0.0272)	(0.0290)	(0.0320)	(0.0363)	(0.0437)
Asian	0.200***	0.237***	0.252***	0.205***	0.232***
	(0.0164)	(0.0188)	(0.0212)	(0.0215)	(0.0248)
Other	0.187***	0.226***	0.247***	0.222***	0.259***
	(0.0238)	(0.0263)	(0.0287)	(0.0293)	(0.0340)
high education	0.0802***	0.0996***	0.137***	0.110***	0.168***
	(0.0134)	(0.0151)	(0.0169)	(0.0165)	(0.0195)
low education	-0.0872***	-0.116***	-0.127***	-0.117***	-0.0970***
	(0.0122)	(0.0139)	(0.0154)	(0.0150)	(0.0170)
high SES	0.0519***	0.0759***	0.104***	0.0898***	0.151***
	(0.0123)	(0.0138)	(0.0153)	(0.0151)	(0.0182)
low SES	-0.0937***	-0.0981***	-0.107***	-0.0971***	-0.0876***
	(0.0103)	(0.0118)	(0.0131)	(0.0127)	(0.0144)
live with both natural parents	0.0932***	0.0820***	0.0749***	0.0697***	0.0531**
	(0.0139)	(0.0158)	(0.0170)	(0.0167)	(0.0177)
mother married	0.0206	0.0320**	0.0336*	0.0338**	0.0426**
	(0.0107)	(0.0121)	(0.0133)	(0.0130)	(0.0146)
home owners	0.0438***	0.0460***	0.0294	0.0469**	-0.000703
	(0.0119)	(0.0137)	(0.0150)	(0.0148)	(0.0162)
private renters	0.0567***	0.0638***	0.0722***	0.0665***	0.0345
	(0.0163)	(0.0186)	(0.0200)	(0.0199)	(0.0216)
birth weight	-0.000744	0.00301	0.0151	0.00396	0.00993
	(0.00846)	(0.00954)	(0.0105)	(0.0104)	(0.0116)
hospital stay (log)	0.0428***	0.0330***	0.0252*	0.0299**	0.0172
	(0.00781)	(0.00881)	(0.00977)	(0.00957)	(0.0109)
planned pregnancy	0.0250**	0.0298**	0.0231*	0.0282*	0.0141
	(0.00923)	(0.0104)	(0.0114)	(0.0113)	(0.0126)
Premature	-0.0221	-0.0211	-0.0662**	-0.0487*	-0.0717**
	(0.0185)	(0.0210)	(0.0236)	(0.0233)	(0.0256)
mother obese	-0.00332	-0.0366	-0.0843***	-0.0491*	-0.0970***
	(0.0160)	(0.0187)	(0.0211)	(0.0204)	(0.0232)
mother age at birth	0.00372***	0.00826***	0.0104***	0.00838***	0.0106***
	(0.000843)	(0.000950)	(0.00104)	(0.00103)	(0.00113)
smoker 1st trimester	-0.0161	-0.0480***	-0.101***	-0.0547***	-0.0916***
	(0.0106)	(0.0122)	(0.0135)	(0.0131)	(0.0145)
smoker 2nd trimester	-0.124***	-0.161***	-0.148***	-0.147***	-0.132***
	(0.0269)	(0.0303)	(0.0318)	(0.0316)	(0.0327)
smoker 3rd trimester	-0.128***	-0.158***	-0.182***	-0.161***	-0.144***
	(0.0176)	(0.0199)	(0.0211)	(0.0209)	(0.0216)
alcohol during pregnancy	0.00191	0.000190	0.000762	0.000145	0.000182
	(0.00417)	(0.00474)	(0.00501)	(0.00499)	(0.00552)
mother in care at 16 years	-0.0515	-0.0397	-0.0631	-0.0517	0.0221
	(0.0483)	(0.0541)	(0.0589)	(0.0575)	(0.0580)
maternal longstanding illness	0.0149	0.00520	-0.00756	-0.00206	-0.0281*
	(0.01000)	(0.0114)	(0.0126)	(0.0124)	(0.0140)
Constant	0.248	-0.0645	-0.198	-0.00888	-0.0290
	(0.128)	(0.144)	(0.157)	(0.155)	(0.175)
N	10707	8474	6643	7542	5026

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Stage one of IV model estimating breastfeeding treatments; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks. Instrument is a binary variable indicating delivery by Caesarean section.

Table A-21: Second Stage of 2SLS Estimating BMI in Three Year Olds

	BMI				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	-0.916	-0.492	-0.949	-0.186	-0.187
	(1.145)	(1.049)	(1.053)	(0.872)	(1.331)
Age	-0.00235	-0.00141	-0.000521	-0.00210	-0.0000727
	(0.00154)	(0.00171)	(0.00180)	(0.00170)	(0.00200)
Sex	0.187***	0.158***	0.149***	0.165***	0.152***
	(0.0315)	(0.0342)	(0.0380)	(0.0348)	(0.0460)
Black	0.472	0.376	0.595	0.238	0.113
	(0.355)	(0.390)	(0.430)	(0.317)	(0.522)
Asian	-0.310	-0.408	-0.298	-0.451*	-0.489
	(0.244)	(0.275)	(0.301)	(0.211)	(0.356)
Other	0.0686	0.0552	0.196	0.00606	-0.186
	(0.247)	(0.263)	(0.282)	(0.221)	(0.339)
high education	0.129	0.0995	0.209	0.0877	0.151
	(0.104)	(0.117)	(0.153)	(0.109)	(0.243)
low education	-0.0903	-0.0671	-0.134	-0.00899	-0.00603
	(0.112)	(0.135)	(0.154)	(0.119)	(0.147)
high SES	0.0804	0.0667	0.131	0.0642	0.0343
	(0.0698)	(0.0859)	(0.113)	(0.0869)	(0.189)
low SES	-0.00557	0.0436	-0.0217	0.0451	0.0380
	(0.113)	(0.119)	(0.134)	(0.102)	(0.137)
live with both natural parents	0.0436	-0.0283	-0.0162	-0.0452	-0.0486
	(0.118)	(0.107)	(0.102)	(0.0897)	(0.0962)
mother married	-0.0513	-0.0343	-0.0190	-0.0780	-0.0784
	(0.0411)	(0.0494)	(0.0549)	(0.0501)	(0.0732)
home owners	-0.0500	-0.0593	-0.0514	-0.0892	-0.102
	(0.0627)	(0.0628)	(0.0600)	(0.0603)	(0.0611)
private renters	-0.0782	-0.0659	0.00218	-0.0636	0.0155
	(0.0929)	(0.0989)	(0.105)	(0.0924)	(0.0991)
birth weight	0.604***	0.634***	0.642***	0.636***	0.628***
	(0.0305)	(0.0336)	(0.0409)	(0.0357)	(0.0451)
hospital stay (log)	0.0575	0.0281	0.00573	0.0276	0.0169
	(0.0409)	(0.0324)	(0.0320)	(0.0301)	(0.0361)
planned pregnancy	0.0300	0.0273	0.0169	0.0153	0.00360
	(0.0469)	(0.0513)	(0.0524)	(0.0478)	(0.0523)
Premature	0.369***	0.454***	0.310**	0.467***	0.347**
	(0.0715)	(0.0800)	(0.114)	(0.0905)	(0.132)
mother obese	0.463***	0.428***	0.394***	0.486***	0.537***
	(0.0560)	(0.0741)	(0.119)	(0.0799)	(0.157)
mother age at birth	0.00878	0.00569	0.0104	0.00441	0.00502
	(0.00509)	(0.00865)	(0.0112)	(0.00772)	(0.0144)
smoker 1st trimester	0.153**	0.147	0.0670	0.157*	0.144
	(0.0499)	(0.0776)	(0.129)	(0.0732)	(0.143)
smoker 2nd trimester	-0.0676	-0.0368	-0.0885	-0.0239	0.00311
	(0.174)	(0.184)	(0.184)	(0.153)	(0.201)
smoker 3rd trimester	0.142	0.181	0.103	0.259	0.271
	(0.163)	(0.184)	(0.210)	(0.158)	(0.216)
alcohol during pregnancy	-0.0163	-0.0139	-0.0174	-0.0200	-0.0113
	(0.0149)	(0.0165)	(0.0183)	(0.0168)	(0.0207)
mother in care at 16 years	-0.0292	-0.0747	-0.0545	-0.111	-0.0860
	(0.157)	(0.182)	(0.203)	(0.186)	(0.206)
maternal longstanding illness	-0.0193	-0.0423	-0.0527	-0.0455	-0.0577
	(0.0406)	(0.0403)	(0.0476)	(0.0426)	(0.0668)
Constant	15.27***	14.85***	14.76***	14.84***	14.50***
	(0.704)	(0.525)	(0.410)	(0.457)	(0.410)
N	11200	8845	6949	7885	5290

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Stage two of IV model estimating BMI varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks. Instrument is a binary variable indicating delivery by Caesarean section.

Table A-22: Second Stage of 2SLS Estimating BMI in Five Year Olds

	BMI				
	(1)	(2)	(3)	(4)	(5
Breastfeeding 'treatment'	-1.712	-1.863	-1.871*	-1.447	-2.626*
	(1.050)	(0.970)	(0.912)	(0.821)	(1.246)
Age	-0.000312	-0.000136	0.000139	-0.000150	-0.00134
	(0.00131)	(0.00146)	(0.00159)	(0.00145)	(0.00192)
Sex	-0.0192	-0.0366	-0.0617	-0.0386	-0.100
	(0.0327)	(0.0370)	(0.0412)	(0.0376)	(0.0523)
Black	1.137***	1.316***	1.463***	1.098***	1.486**
	(0.318)	(0.356)	(0.374)	(0.292)	(0.464)
Asian	0.00887	0.0971	0.101	-0.136	0.289
	(0.233)	(0.265)	(0.274)	(0.206)	(0.343)
Other	0.284	0.425	0.510	0.252	0.447
	(0.241)	(0.264)	(0.265)	(0.226)	(0.361)
high education	0.129	0.184	0.255	0.175	0.407
	(0.0948)	(0.108)	(0.138)	(0.104)	(0.223)
low education	-0.135	-0.210	-0.217	-0.139	-0.279
	(0.112)	(0.135)	(0.139)	(0.121)	(0.154)
high SES	0.129	0.169	0.209	0.157	0.400*
	(0.0715)	(0.0899)	(0.110)	(0.0920)	(0.201)
low SES	-0.0512	-0.0632	-0.103	-0.0324	-0.113
	(0.108)	(0.111)	(0.119)	(0.0953)	(0.130)
live with both natural parents	0.139	0.114	0.0846	0.0919	0.0758
	(0.109)	(0.100)	(0.0945)	(0.0863)	(0.100)
mother married	-0.0155	0.0284	0.0533	-0.0151	0.0542
	(0.0514)	(0.0616)	(0.0646)	(0.0592)	(0.0900)
home owners	0.00144	0.00667	-0.0111	-0.0168	-0.0560
	(0.0663)	(0.0693)	(0.0659)	(0.0660)	(0.0719)
private renters	-0.0507	0.0151	0.0676	0.00370	0.0660
	(0.0912)	(0.0979)	(0.104)	(0.0931)	(0.111)
birth weight	0.641***	0.670***	0.691***	0.656***	0.693***
	(0.0337)	(0.0385)	(0.0462)	(0.0394)	(0.0535)
hospital stay (log)	0.142**	0.117**	0.0803*	0.109**	0.116**
	(0.0444)	(0.0362)	(0.0357)	(0.0338)	(0.0431)
planned pregnancy	0.0165	0.0571	0.0560	0.0374	0.0621
	(0.0435)	(0.0494)	(0.0501)	(0.0469)	(0.0572)
Premature	0.362***	0.381***	0.259*	0.388***	0.160
	(0.0765)	(0.0868)	(0.110)	(0.0949)	(0.135)
mother obese	0.718***	0.607***	0.544***	0.608***	0.488**
	(0.0642)	(0.0834)	(0.119)	(0.0881)	(0.165)
mother age at birth	0.00706	0.0127	0.0164	0.0104	0.0246
	(0.00472)	(0.00800)	(0.00964)	(0.00717)	(0.0129)
smoker 1st trimester	0.200***	0.147	0.0650	0.164*	0.0217
	(0.0522)	(0.0773)	(0.114)	(0.0742)	(0.137)
smoker 2nd trimester	-0.0401	-0.0936	0.0143	-0.0392	-0.118
	(0.162)	(0.184)	(0.171)	(0.162)	(0.209)
smoker 3rd trimester	0.105	0.0465	0.0606	0.129	0.0802
	(0.154)	(0.168)	(0.175)	(0.150)	(0.185)
alcohol during pregnancy	-0.0147	-0.0124	-0.0108	-0.0159	-0.0174
	(0.0165)	(0.0186)	(0.0198)	(0.0184)	(0.0243)
mother in care at 16 years	-0.127	-0.246	-0.346	-0.244	-0.234
	(0.180)	(0.208)	(0.240)	(0.210)	(0.259)
maternal longstanding illness	0.0574	0.0504	0.0233	0.0256	-0.0234
	(0.0427)	(0.0461)	(0.0519)	(0.0469)	(0.0733)
Constant	14.81***	14.47***	14.11***	14.33***	14.33***
	(0.547)	(0.466)	(0.483)	(0.468)	(0.592)
N	11744	9283	7278	8259	5541

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Stage two of IV model estimating BMI varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks. Instrument is a binary variable indicating delivery by Caesarean section.

Table A-23: Second Stage of 2SLS Estimating BMI in Seven Year Olds

	BMI				
	(1)	(2	(3)	(4)	(5)
Breastfeeding 'treatment'	-2.672	-2.692	-3.014	-2.582	-4.328
	(1.992)	(1.762)	(1.702)	(1.579)	(2.765)
Age	0.0113***	0.0125***	0.0134***	0.0121***	0.0112***
	(0.00207)	(0.00245)	(0.00258)	(0.00230)	(0.00293)
Sex	-0.151**	-0.193***	-0.198**	-0.199***	-0.315***
	(0.0483)	(0.0522)	(0.0602)	(0.0551)	(0.0802)
Black	1.669**	1.932**	2.190**	1.826***	2.490*
	(0.599)	(0.637)	(0.693)	(0.551)	(1.062)
Asian	0.396	0.482	0.590	0.290	0.842
	(0.412)	(0.434)	(0.449)	(0.349)	(0.669)
Other	0.578	0.704	0.865	0.605	1.026
	(0.396)	(0.425)	(0.451)	(0.388)	(0.752)
high education	0.178	0.251	0.418	0.290	0.733
	(0.176)	(0.193)	(0.250)	(0.194)	(0.480)
low education	-0.174	-0.249	-0.325	-0.223	-0.355
	(0.189)	(0.221)	(0.238)	(0.204)	(0.295)
high SES	0.138	0.203	0.247	0.227	0.584
	(0.126)	(0.155)	(0.199)	(0.165)	(0.435)
low SES	-0.118	-0.117	-0.216	-0.108	-0.262
	(0.196)	(0.185)	(0.198)	(0.169)	(0.261)
live with both natural parents	0.215	0.166	0.148	0.165	0.162
	(0.201)	(0.169)	(0.161)	(0.142)	(0.188)
mother married	-0.0666	-0.00998	0.0483	-0.0363	0.0882
	(0.0736)	(0.0886)	(0.0969)	(0.0895)	(0.154)
home owners	0.0274	0.0331	0.00255	0.0202	-0.0762
	(0.110)	(0.112)	(0.101)	(0.109)	(0.109)
private renters	0.105	0.204	0.352*	0.221	0.347*
	(0.146)	(0.154)	(0.170)	(0.151)	(0.173)
birth weight	0.692***	0.758***	0.794***	0.730***	0.792***
	(0.0484)	(0.0541)	(0.0672)	(0.0576)	(0.0836)
hospital stay (log)	0.176*	0.128*	0.0693	0.117*	0.0987
	(0.0751)	(0.0557)	(0.0525)	(0.0512)	(0.0651)
planned pregnancy	0.00604	0.0417	0.0532	0.0369	0.0695
	(0.0721)	(0.0786)	(0.0778)	(0.0763)	(0.0936)
Premature	0.371**	0.484***	0.314	0.375*	0.149
	(0.116)	(0.125)	(0.179)	(0.151)	(0.264)
mother obese	1.257***	1.097***	0.978***	1.079***	0.899**
	(0.0923)	(0.127)	(0.196)	(0.140)	(0.320)
mother age at birth	0.0224**	0.0328*	0.0399*	0.0303*	0.0519
	(0.00843)	(0.0150)	(0.0182)	(0.0139)	(0.0298)
smoker 1st trimester	0.312***	0.211	0.0612	0.194	-0.0636
	(0.0695)	(0.111)	(0.191)	(0.114)	(0.274)
smoker 2nd trimester	0.0566	-0.00878	0.150	0.0458	-0.0141
	(0.291)	(0.331)	(0.312)	(0.290)	(0.425)
smoker 3rd trimester	0.106	0.0790	0.0107	0.112	-0.0351
	(0.276)	(0.302)	(0.337)	(0.281)	(0.427)
alcohol during pregnancy	-0.0415	-0.0489	-0.0594*	-0.0578*	-0.0745*
	(0.0241)	(0.0267)	(0.0295)	(0.0275)	(0.0373)
mother in care at 16 years	-0.473	-0.493	-0.742*	-0.553	-0.483
	(0.295)	(0.313)	(0.363)	(0.327)	(0.397)
maternal longstanding illness	0.0960	0.0855	0.0436	0.0470	-0.0564
	(0.0639)	(0.0649)	(0.0753)	(0.0682)	(0.122)
Constant	10.69***	9.565***	8.910***	9.773***	9.535***
	(0.909)	(0.812)	(0.964)	(0.854)	(1.179)
N	10707	8474	6643	7542	5026

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Stage two of IV model estimating BMI varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks. Instrument is a binary variable indicating delivery by Caesarean section.

Table A-24: Roy Model (Age 3 Years)

	BMI (outcome equation)				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	0.101	0.0477	-0.419	-0.000959	-0.587*
	(0.191)	(0.259)	(0.260)	(0.291)	(0.235)
Age	-0.00177	-0.00102	-0.000330	-0.00199	-0.000233
	(0.00136)	(0.00154)	(0.00172)	(0.00162)	(0.00194)
Sex	0.177***	0.153***	0.146***	0.164***	0.147***
	(0.0287)	(0.0324)	(0.0367)	(0.0342)	(0.0426)
Black	0.170	0.183	0.387*	0.177	0.261
	(0.112)	(0.139)	(0.157)	(0.160)	(0.195)
Asian	-0.521***	-0.545***	-0.444***	-0.493***	-0.385***
	(0.0694)	(0.0922)	(0.105)	(0.0977)	(0.111)
Other	-0.138	-0.0716	0.0654	-0.0359	-0.0917
	(0.0904)	(0.110)	(0.124)	(0.120)	(0.145)
high education	0.0473	0.0461	0.139*	0.0678	0.221**
	(0.0496)	(0.0589)	(0.0704)	(0.0644)	(0.0848)
low education	0.00149	-0.00244	-0.0617	0.0139	-0.0457
	(0.0461)	(0.0577)	(0.0661)	(0.0624)	(0.0685)
high SES	0.0330	0.0304	0.0814	0.0495	0.0872
	(0.0447)	(0.0518)	(0.0609)	(0.0571)	(0.0762)
low SES	0.0897*	0.101*	0.0412	0.0647	-0.0000109
	(0.0401)	(0.0494)	(0.0563)	(0.0529)	(0.0586)
live with both natural parents	-0.0512	-0.0756	-0.0564	-0.0599	-0.0278
	(0.0517)	(0.0596)	(0.0647)	(0.0617)	(0.0685)
mother married	-0.0633	-0.0467	-0.0306	-0.0828	-0.0642
	(0.0378)	(0.0432)	(0.0490)	(0.0455)	(0.0567)
home owners	-0.0906*	-0.0801	-0.0631	-0.0962	-0.101
	(0.0424)	(0.0487)	(0.0544)	(0.0515)	(0.0613)
private renters	-0.142*	-0.104	-0.0347	-0.0768	0.0320
	(0.0586)	(0.0677)	(0.0756)	(0.0714)	(0.0836)
birth weight	0.606***	0.634***	0.636***	0.636***	0.631***
	(0.0296)	(0.0333)	(0.0385)	(0.0357)	(0.0445)
hospital stay (log)	0.0286	0.0192	0.00348	0.0257	0.0159
	(0.0246)	(0.0275)	(0.0310)	(0.0289)	(0.0361)
planned pregnancy	0.000704	0.00891	0.00151	0.00925	0.0101
	(0.0328)	(0.0376)	(0.0422)	(0.0397)	(0.0480)
Premature	0.391***	0.469***	0.346***	0.477***	0.319**
	(0.0652)	(0.0741)	(0.0873)	(0.0806)	(0.0981)
mother obese	0.474***	0.449***	0.442***	0.495***	0.496***
	(0.0534)	(0.0624)	(0.0755)	(0.0673)	(0.0856)
mother age at birth	0.00513	0.00158	0.00517	0.00294	0.00916
	(0.00300)	(0.00384)	(0.00455)	(0.00417)	(0.00490)
smoker 1st trimester	0.181***	0.180***	0.127*	0.169***	0.104
	(0.0377)	(0.0458)	(0.0573)	(0.0488)	(0.0601)
smoker 2nd trimester	0.0600	0.0403	-0.0175	-0.000864	-0.0445
	(0.0978)	(0.112)	(0.121)	(0.114)	(0.128)
smoker 3rd trimester	0.275***	0.268***	0.202*	0.289***	0.211*
	(0.0663)	(0.0812)	(0.0910)	(0.0855)	(0.0900)
alcohol during pregnancy	-0.0158	-0.0134	-0.0193	-0.0199	-0.0107
	(0.0145)	(0.0164)	(0.0175)	(0.0168)	(0.0207)
mother in care at 16 years	-0.0132	-0.0515	-0.0339	-0.103	-0.0776
	(0.152)	(0.175)	(0.195)	(0.182)	(0.206)
maternal longstanding illness	-0.0360	-0.0439	-0.0479	-0.0445	-0.0701
	(0.0351)	(0.0400)	(0.0457)	(0.0423)	(0.0534)
Constant	14.69***	14.63***	14.66***	14.77***	14.54***
	(0.289)	(0.321)	(0.347)	(0.337)	(0.389)

	Breastfeeding (treatment equation)				
Caesarean Section	-0.117**	-0.137**	-0.169***	-0.178***	-0.165**
	(0.0382)	(0.0430)	(0.0499)	(0.0455)	(0.0599)
Age	-0.00178	-0.00229	-0.000957	-0.00191	-0.00151
	(0.00124)	(0.00141)	(0.00165)	(0.00148)	(0.00198)
Sex	0.0351	0.0359	0.0233	0.0266	-0.0489
	(0.0268)	(0.0301)	(0.0350)	(0.0314)	(0.0417)
Black	1.249***	1.484***	1.635***	1.245***	1.429***
	(0.126)	(0.133)	(0.147)	(0.147)	(0.180)
Asian	0.680***	0.787***	0.854***	0.656***	0.823***
	(0.0571)	(0.0624)	(0.0705)	(0.0667)	(0.0820)
Other	0.757***	0.864***	0.954***	0.788***	0.887***
	(0.0888)	(0.0975)	(0.111)	(0.103)	(0.131)
high education	0.339***	0.365***	0.396***	0.357***	0.423***
	(0.0454)	(0.0493)	(0.0553)	(0.0512)	(0.0654)
low education	-0.254***	-0.330***	-0.406***	-0.334***	-0.367***
	(0.0384)	(0.0430)	(0.0495)	(0.0447)	(0.0595)
high SES	0.256***	0.307***	0.342***	0.320***	0.357***
	(0.0459)	(0.0491)	(0.0538)	(0.0508)	(0.0624)
low SES	-0.274***	-0.304***	-0.366***	-0.293***	-0.343***
	(0.0325)	(0.0365)	(0.0422)	(0.0380)	(0.0503)
live with both natural parents	0.276***	0.288***	0.334***	0.263***	0.327***
	(0.0429)	(0.0505)	(0.0625)	(0.0528)	(0.0778)
mother married	0.0320	0.0563	0.0460	0.0636	0.109*
	(0.0346)	(0.0388)	(0.0451)	(0.0405)	(0.0537)
home owners	0.0943*	0.0943*	0.0740	0.0970*	0.0538
	(0.0376)	(0.0430)	(0.0509)	(0.0451)	(0.0614)
private renters	0.180***	0.220***	0.271***	0.223***	0.221*
	(0.0517)	(0.0596)	(0.0706)	(0.0622)	(0.0870)
birth weight	-0.0105	-0.00558	0.0285	-0.0106	0.0160
	(0.0276)	(0.0311)	(0.0367)	(0.0328)	(0.0439)
hospital stay (log)	0.129***	0.0950**	0.0638	0.0865**	0.0423
	(0.0258)	(0.0290)	(0.0340)	(0.0304)	(0.0410)
planned pregnancy	0.0938**	0.108**	0.0983*	0.0994**	0.0580
	(0.0299)	(0.0335)	(0.0388)	(0.0349)	(0.0460)
Premature	-0.0797	-0.0976	-0.249**	-0.161*	-0.273**
	(0.0601)	(0.0685)	(0.0830)	(0.0727)	(0.0993)
mother obese	-0.0269	-0.110	-0.278***	-0.139*	-0.372***
	(0.0488)	(0.0561)	(0.0684)	(0.0592)	(0.0855)
mother age at birth	0.0118***	0.0247***	0.0358***	0.0255***	0.0435***
	(0.00270)	(0.00306)	(0.00359)	(0.00319)	(0.00428)
smoker 1st trimester	-0.0787*	-0.167***	-0.347***	-0.183***	-0.356***
	(0.0335)	(0.0384)	(0.0457)	(0.0400)	(0.0550)
smoker 2nd trimester	-0.335***	-0.415***	-0.456***	-0.371***	-0.583***
	(0.0826)	(0.0980)	(0.119)	(0.100)	(0.159)
smoker 3rd trimester	-0.341***	-0.454***	-0.650***	-0.474***	-0.740***
	(0.0532)	(0.0634)	(0.0805)	(0.0664)	(0.104)
alcohol during pregnancy	-0.000308	-0.00110	0.0148	0.000321	0.00926
	(0.0129)	(0.0151)	(0.0169)	(0.0155)	(0.0230)
mother in care at 16 years	-0.0328	-0.120	-0.131	-0.128	0.139
	(0.132)	(0.162)	(0.210)	(0.171)	(0.232)
maternal longstanding illness	0.0515	0.0136	-0.0241	-0.0119	-0.119*
	(0.0326)	(0.0371)	(0.0435)	(0.0389)	(0.0530)
Constant	0.116	-0.375	-1.204***	-0.480	-1.615***
	(0.247)	(0.281)	(0.331)	(0.294)	(0.395)
ath(ρ) constant	-0.0632	-0.0438	0.123	-0.0243	0.162
	(0.0749)	(0.102)	(0.101)	(0.115)	(0.0900)
ln(σ) constant	0.408***	0.410***	0.416***	0.406***	0.425***
	(0.00710)	(0.00785)	(0.0103)	(0.00809)	(0.0116)
N	11200	8845	6949	7885	5290

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Roy model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks. Outcome equation estimating BMI and treatment equation estimating breastfeeding treatment.

Table A-25: Roy Model (Age 5 Years)

	BMI (outcome equation)				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	-0.0443	-0.291	-0.607*	-0.242	-0.693**
	(0.200)	(0.296)	(0.252)	(0.261)	(0.229)
Age	-0.000885	-0.000716	-0.000205	-0.000434	-0.00101
	(0.00116)	(0.00130)	(0.00146)	(0.00136)	(0.00167)
Sex	-0.0196	-0.0361	-0.0621	-0.0365	-0.0775
	(0.0301)	(0.0337)	(0.0381)	(0.0355)	(0.0438)
Black	0.661***	0.770***	0.976***	0.717***	0.833***
	(0.115)	(0.147)	(0.154)	(0.151)	(0.186)
Asian	-0.346***	-0.316**	-0.260*	-0.415***	-0.215
	(0.0733)	(0.103)	(0.107)	(0.0969)	(0.114)
Other	-0.0686	0.0311	0.181	-0.0388	-0.0579
	(0.0956)	(0.120)	(0.127)	(0.121)	(0.147)
high education	0.00531	0.0405	0.0899	0.0534	0.0899
	(0.0518)	(0.0619)	(0.0724)	(0.0646)	(0.0858)
low education	0.0256	-0.00954	-0.0454	0.0182	-0.0723
	(0.0490)	(0.0633)	(0.0677)	(0.0634)	(0.0710)
high SES	0.0480	0.0550	0.0851	0.0523	0.115
	(0.0472)	(0.0554)	(0.0637)	(0.0592)	(0.0799)
low SES	0.107*	0.1000	0.0455	0.0884	0.0617
	(0.0423)	(0.0529)	(0.0574)	(0.0523)	(0.0600)
live with both natural parents	-0.0115	-0.0134	-0.00612	0.00417	-0.0215
	(0.0535)	(0.0616)	(0.0657)	(0.0617)	(0.0692)
mother married	-0.0602	-0.0323	0.00528	-0.0629	-0.0407
	(0.0400)	(0.0460)	(0.0514)	(0.0478)	(0.0586)
home owners	-0.0721	-0.0633	-0.0480	-0.0700	-0.0647
	(0.0445)	(0.0512)	(0.0563)	(0.0533)	(0.0626)
private renters	-0.154*	-0.0899	-0.0229	-0.0796	-0.0222
	(0.0604)	(0.0697)	(0.0768)	(0.0719)	(0.0838)
birth weight	0.635***	0.660***	0.667***	0.649***	0.674***
	(0.0309)	(0.0346)	(0.0399)	(0.0369)	(0.0455)
hospital stay (log)	0.0871***	0.0871**	0.0703*	0.0922**	0.107**
	(0.0262)	(0.0289)	(0.0324)	(0.0302)	(0.0373)
planned pregnancy	-0.0201	0.0145	0.0306	0.00847	0.0472
	(0.0343)	(0.0390)	(0.0435)	(0.0406)	(0.0493)
Premature	0.397***	0.420***	0.337***	0.445***	0.275**
	(0.0676)	(0.0762)	(0.0891)	(0.0825)	(0.0991)
mother obese	0.738***	0.671***	0.661***	0.675***	0.691***
	(0.0581)	(0.0680)	(0.0815)	(0.0726)	(0.0917)
mother age at birth	0.00177	0.00128	0.00433	0.00150	0.00623
	(0.00315)	(0.00406)	(0.00457)	(0.00410)	(0.00490)
smoker 1st trimester	0.248***	0.244***	0.203***	0.245***	0.208***
	(0.0395)	(0.0482)	(0.0578)	(0.0497)	(0.0607)
smoker 2nd trimester	0.152	0.130	0.168	0.120	0.111
	(0.102)	(0.119)	(0.125)	(0.118)	(0.132)
smoker 3rd trimester	0.324***	0.288***	0.274**	0.318***	0.327***
	(0.0687)	(0.0841)	(0.0888)	(0.0835)	(0.0877)
alcohol during pregnancy	-0.0157	-0.0129	-0.0138	-0.0165	-0.0157
	(0.0152)	(0.0169)	(0.0182)	(0.0173)	(0.0212)
mother in care at 16 years	-0.138	-0.225	-0.283	-0.217	-0.286
	(0.166)	(0.189)	(0.218)	(0.198)	(0.224)
maternal longstanding illness	0.0337	0.0417	0.0320	0.0299	0.0355
	(0.0370)	(0.0418)	(0.0477)	(0.0442)	(0.0552)
Constant	14.19***	14.18***	14.05***	14.09***	14.16***
	(0.360)	(0.396)	(0.445)	(0.417)	(0.508)

	Breastfeeding (treatment equation)				
Caesarean Section	-0.146***	-0.180***	-0.231***	-0.217***	-0.226***
	(0.0378)	(0.0427)	(0.0493)	(0.0451)	(0.0590)
Age	0.00123	0.00138	0.00109	0.000897	-0.000711
	(0.00101)	(0.00113)	(0.00131)	(0.00118)	(0.00156)
Sex	0.00592	0.00327	0.00632	-0.00316	-0.0470
	(0.0263)	(0.0295)	(0.0342)	(0.0308)	(0.0407)
Black	1.154***	1.391***	1.577***	1.161***	1.299***
	(0.115)	(0.121)	(0.133)	(0.133)	(0.163)
Asian	0.698***	0.811***	0.878***	0.665***	0.829***
	(0.0563)	(0.0614)	(0.0694)	(0.0659)	(0.0813)
Other	0.791***	0.922***	0.997***	0.849***	0.998***
	(0.0872)	(0.0948)	(0.108)	(0.100)	(0.125)
high education	0.330***	0.351***	0.397***	0.345***	0.393***
	(0.0446)	(0.0485)	(0.0546)	(0.0504)	(0.0643)
low education	-0.268***	-0.349***	-0.403***	-0.354***	-0.391***
	(0.0377)	(0.0421)	(0.0486)	(0.0439)	(0.0581)
high SES	0.272***	0.332***	0.363***	0.354***	0.406***
	(0.0454)	(0.0485)	(0.0533)	(0.0503)	(0.0618)
low SES	-0.279***	-0.297***	-0.359***	-0.278***	-0.324***
	(0.0319)	(0.0359)	(0.0415)	(0.0375)	(0.0497)
live with both natural parents	0.266***	0.269***	0.319***	0.246***	0.314***
	(0.0413)	(0.0485)	(0.0598)	(0.0508)	(0.0735)
mother married	0.0833*	0.109**	0.101*	0.110**	0.148**
	(0.0337)	(0.0377)	(0.0437)	(0.0394)	(0.0520)
home owners	0.105**	0.107*	0.0893	0.114**	0.0621
	(0.0367)	(0.0419)	(0.0493)	(0.0439)	(0.0592)
private renters	0.172***	0.201***	0.273***	0.214***	0.246**
	(0.0495)	(0.0570)	(0.0673)	(0.0594)	(0.0816)
birth weight	0.00755	0.0120	0.0606	0.0120	0.0369
	(0.0269)	(0.0302)	(0.0357)	(0.0319)	(0.0426)
hospital stay (log)	0.154***	0.115***	0.0957**	0.110***	0.0893*
	(0.0252)	(0.0284)	(0.0333)	(0.0297)	(0.0400)
planned pregnancy	0.0722*	0.0871**	0.0697	0.0745*	0.0304
	(0.0293)	(0.0328)	(0.0380)	(0.0342)	(0.0448)
Premature	-0.0684	-0.0810	-0.214**	-0.151*	-0.233*
	(0.0582)	(0.0662)	(0.0804)	(0.0706)	(0.0958)
mother obese	-0.0318	-0.113*	-0.280***	-0.147*	-0.384***
	(0.0495)	(0.0569)	(0.0698)	(0.0603)	(0.0880)
mother age at birth	0.0104***	0.0237***	0.0348***	0.0240***	0.0406***
	(0.00265)	(0.00300)	(0.00351)	(0.00313)	(0.00418)
smoker 1st trimester	-0.0821*	-0.170***	-0.333***	-0.187***	-0.343***
	(0.0327)	(0.0373)	(0.0444)	(0.0389)	(0.0533)
smoker 2nd trimester	-0.309***	-0.416***	-0.405***	-0.398***	-0.578***
	(0.0809)	(0.0973)	(0.117)	(0.100)	(0.157)
smoker 3rd trimester	-0.340***	-0.429***	-0.581***	-0.461***	-0.567***
	(0.0513)	(0.0603)	(0.0752)	(0.0634)	(0.0921)
alcohol during pregnancy	0.00324	0.00352	0.0108	0.00401	0.000219
	(0.0125)	(0.0145)	(0.0165)	(0.0150)	(0.0227)
mother in care at 16 years	0.0399	-0.0197	-0.250	-0.0564	0.115
	(0.136)	(0.165)	(0.237)	(0.177)	(0.244)
maternal longstanding illness	0.0440	0.0230	-0.0143	-0.00429	-0.106*
	(0.0320)	(0.0364)	(0.0427)	(0.0382)	(0.0520)
Constant	-0.538	-1.156***	-1.777***	-1.077**	-1.699***
	(0.305)	(0.343)	(0.400)	(0.359)	(0.477)
ath(ρ) constant	-0.0165	0.0636	0.155	0.0418	0.158
	(0.0724)	(0.109)	(0.0924)	(0.0967)	(0.0824)
ln(σ) constant	0.481***	0.476***	0.478***	0.469***	0.478***
	(0.00655)	(0.00811)	(0.0106)	(0.00806)	(0.0111)
N	11744	9283	7278	8259	5541

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Roy model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks. Outcome equation estimating BMI and treatment equation estimating breastfeeding treatment.

Table A-26: Roy Model (Age 7 Years)

	BMI (outcome equation)				
	(1)	(2)	(3)	(4)	(5)
Breastfeeding 'treatment'	0.0104	-0.197	-0.629	-0.196	-0.960**
	(0.230)	(0.307)	(0.341)	(0.318)	(0.303)
Age	0.00995***	0.0105***	0.0117***	0.0107***	0.0111***
	(0.00162)	(0.00182)	(0.00204)	(0.00191)	(0.00238)
Sex	-0.167***	-0.198***	-0.211***	-0.206***	-0.283***
	(0.0419)	(0.0467)	(0.0528)	(0.0495)	(0.0617)
Black	0.890***	1.061***	1.255***	1.051***	1.246***
	(0.155)	(0.182)	(0.214)	(0.208)	(0.265)
Asian	-0.144	-0.114	-0.0145	-0.206	0.0521
	(0.0959)	(0.120)	(0.141)	(0.125)	(0.153)
Other	0.0783	0.139	0.280	0.0722	0.154
	(0.129)	(0.150)	(0.172)	(0.161)	(0.203)
high education	-0.0343	0.00559	0.0967	0.0308	0.171
	(0.0709)	(0.0823)	(0.0995)	(0.0885)	(0.118)
low education	0.0616	0.0433	-0.0171	0.0595	-0.0246
	(0.0656)	(0.0790)	(0.0916)	(0.0832)	(0.0978)
high SES	-0.00142	0.0130	-0.00250	0.0124	0.0750
	(0.0643)	(0.0735)	(0.0873)	(0.0802)	(0.110)
low SES	0.134*	0.127	0.0389	0.124	0.0331
	(0.0569)	(0.0669)	(0.0774)	(0.0702)	(0.0833)
live with both natural parents	-0.0337	-0.0368	-0.0295	0.00152	-0.0143
	(0.0742)	(0.0835)	(0.0921)	(0.0858)	(0.0985)
mother married	-0.122*	-0.0896	-0.0318	-0.117	-0.0553
	(0.0550)	(0.0621)	(0.0703)	(0.0655)	(0.0814)
home owners	-0.0891	-0.0809	-0.0673	-0.0903	-0.0704
	(0.0619)	(0.0707)	(0.0789)	(0.0748)	(0.0888)
private renters	-0.0469	0.0452	0.179	0.0632	0.232
	(0.0844)	(0.0959)	(0.107)	(0.101)	(0.119)
birth weight	0.694***	0.750***	0.757***	0.719***	0.757***
	(0.0433)	(0.0482)	(0.0551)	(0.0515)	(0.0640)
hospital stay (log)	0.0898*	0.0795*	0.0482	0.0833*	0.0853
	(0.0359)	(0.0397)	(0.0447)	(0.0419)	(0.0522)
planned pregnancy	-0.0601	-0.0319	-0.00126	-0.0303	0.0222
	(0.0475)	(0.0536)	(0.0602)	(0.0566)	(0.0694)
Premature	0.433***	0.540***	0.473***	0.495***	0.391**
	(0.0950)	(0.106)	(0.125)	(0.117)	(0.142)
mother obese	1.274***	1.198***	1.191***	1.207***	1.239***
	(0.0819)	(0.0953)	(0.114)	(0.102)	(0.131)
mother age at birth	0.0131**	0.0129*	0.0158*	0.0110	0.0167*
	(0.00437)	(0.00537)	(0.00640)	(0.00567)	(0.00696)
smoker 1st trimester	0.358***	0.333***	0.305***	0.328***	0.249**
	(0.0545)	(0.0636)	(0.0786)	(0.0673)	(0.0845)
smoker 2nd trimester	0.389**	0.392*	0.500**	0.397*	0.429*
	(0.140)	(0.161)	(0.173)	(0.163)	(0.184)
smoker 3rd trimester	0.453***	0.476***	0.449***	0.500***	0.454***
	(0.0950)	(0.112)	(0.127)	(0.116)	(0.127)
alcohol during pregnancy	-0.0465*	-0.0487*	-0.0603*	-0.0576*	-0.0738*
	(0.0213)	(0.0240)	(0.0262)	(0.0247)	(0.0303)
mother in care at 16 years	-0.336	-0.395	-0.593	-0.429	-0.557
	(0.248)	(0.274)	(0.308)	(0.285)	(0.319)
maternal longstanding illness	0.0574	0.0735	0.0621	0.0534	0.0389
	(0.0513)	(0.0577)	(0.0657)	(0.0613)	(0.0772)
Constant	9.959***	9.656***	9.292***	9.712***	9.527***
	(0.654)	(0.725)	(0.821)	(0.768)	(0.959)

	Breastfeeding (treatment equation)				
Caesarean Section	-0.107**	-0.134**	-0.174***	-0.162***	-0.152*
	(0.0395)	(0.0445)	(0.0515)	(0.0470)	(0.0613)
Age	0.00168	0.00259*	0.00241	0.00187	-0.000182
	(0.00107)	(0.00119)	(0.00137)	(0.00124)	(0.00165)
Sex	0.0232	0.00930	0.0236	0.00923	-0.0334
	(0.0276)	(0.0309)	(0.0358)	(0.0322)	(0.0426)
Black	1.226***	1.444***	1.625***	1.219***	1.388***
	(0.127)	(0.133)	(0.146)	(0.147)	(0.177)
Asian	0.648***	0.726***	0.777***	0.595***	0.762***
	(0.0587)	(0.0643)	(0.0726)	(0.0688)	(0.0847)
Other	0.701***	0.835***	0.942***	0.783***	0.983***
	(0.0927)	(0.101)	(0.113)	(0.105)	(0.130)
high education	0.342***	0.369***	0.413***	0.366***	0.409***
	(0.0463)	(0.0503)	(0.0566)	(0.0523)	(0.0668)
low education	-0.245***	-0.318***	-0.377***	-0.316***	-0.353***
	(0.0393)	(0.0439)	(0.0507)	(0.0458)	(0.0606)
high SES	0.284***	0.346***	0.382***	0.362***	0.415***
	(0.0467)	(0.0499)	(0.0549)	(0.0517)	(0.0636)
low SES	-0.275***	-0.278***	-0.324***	-0.267***	-0.314***
	(0.0334)	(0.0376)	(0.0436)	(0.0392)	(0.0518)
live with both natural parents	0.273***	0.267***	0.322***	0.228***	0.315***
	(0.0439)	(0.0515)	(0.0632)	(0.0537)	(0.0775)
mother married	0.0636	0.0892*	0.0890	0.0937*	0.127*
	(0.0352)	(0.0393)	(0.0455)	(0.0410)	(0.0541)
home owners	0.103**	0.111*	0.0910	0.119*	0.0317
	(0.0386)	(0.0442)	(0.0522)	(0.0463)	(0.0624)
private renters	0.158**	0.191**	0.275***	0.203**	0.192*
	(0.0525)	(0.0604)	(0.0710)	(0.0629)	(0.0869)
birth weight	-0.00127	0.00805	0.0510	0.0117	0.0444
	(0.0284)	(0.0318)	(0.0373)	(0.0334)	(0.0444)
hospital stay (log)	0.137***	0.102***	0.0814*	0.0917**	0.0668
	(0.0264)	(0.0296)	(0.0347)	(0.0310)	(0.0417)
planned pregnancy	0.0798**	0.0932**	0.0784*	0.0863*	0.0529
	(0.0306)	(0.0342)	(0.0396)	(0.0357)	(0.0468)
Premature	-0.0747	-0.0716	-0.237**	-0.157*	-0.290**
	(0.0617)	(0.0700)	(0.0854)	(0.0749)	(0.103)
mother obese	-0.0184	-0.115	-0.264***	-0.144*	-0.353***
	(0.0527)	(0.0605)	(0.0733)	(0.0639)	(0.0912)
mother age at birth	0.0110***	0.0254***	0.0360***	0.0255***	0.0433***
	(0.00279)	(0.00314)	(0.00368)	(0.00328)	(0.00439)
smoker 1st trimester	-0.0478	-0.133***	-0.303***	-0.153***	-0.317***
	(0.0344)	(0.0391)	(0.0464)	(0.0408)	(0.0555)
smoker 2nd trimester	-0.326***	-0.472***	-0.512***	-0.447***	-0.689***
	(0.0839)	(0.101)	(0.124)	(0.104)	(0.168)
smoker 3rd trimester	-0.336***	-0.445***	-0.639***	-0.473***	-0.662***
	(0.0548)	(0.0646)	(0.0813)	(0.0678)	(0.101)
alcohol during pregnancy	0.00693	0.00189	0.00245	0.00158	0.00144
	(0.0133)	(0.0158)	(0.0186)	(0.0163)	(0.0242)
mother in care at 16 years	-0.120	-0.0982	-0.303	-0.147	0.0890
	(0.151)	(0.179)	(0.253)	(0.192)	(0.259)
maternal longstanding illness	0.0459	0.0213	-0.0146	-0.00204	-0.0960
	(0.0335)	(0.0380)	(0.0445)	(0.0398)	(0.0540)
Constant	-0.843*	-1.809***	-2.423***	-1.591**	-1.929**
	(0.428)	(0.479)	(0.555)	(0.500)	(0.664)
ath(ρ)	-0.0357	0.00360	0.103	0.000155	0.171*
constant	(0.0622)	(0.0847)	(0.0939)	(0.0878)	(0.0815)
ln(σ)	0.764***	0.756***	0.756***	0.756***	0.770***
constant	(0.00693)	(0.00768)	(0.00982)	(0.00814)	(0.0117)
N	10707	8474	6643	7542	5026

Source: Millennium Cohort Study. Notes: Standard errors in parentheses. * p < 0.1, ** p < 0.05, *** p < 0.01. Roy model varying by breastfeeding treatment; these binary treatments are (1) ever breastfed, (2) partially breastfed for four weeks, (3) partially breastfed for sixteen weeks, (4) exclusively breastfed for four weeks, (5) exclusively breastfed for sixteen weeks. Outcome equation estimating BMI and treatment equation estimating breastfeeding treatment.

B. Appendix: Childhood Adiposity and Underlying Family Lifestyle

Table B-1: Summary Statistics for Final Sample

Variable	N	Mean	Std. Dev.	Min	Max
		Independent Variables			
Age	8462	42.11617	2.075311	35	53
Sex	8462	0.501891	0.500026	0	1
High SES	8462	0.394588	0.488791	0	1
Low SES	8462	0.404278	0.490781	0	1
Maternal educ.	8462	2.945876	1.106289	1	5
Single parent – 9 months	8462	0.102222	0.302958	0	1
Single parent – age 3	8462	0.12113	0.326297	0	1
Single parent – age 5	8462	0.142401	0.349482	0	1
Single parent – age 7	8462	0.166509	0.372559	0	1
		Outcome Measures: Wave 1			
Child weight (kg)	8323	8.835099	1.253649	3.06	19.2
Pre-preg. weight status	7704	0.353193	0.645535	0	2
Paternal weight status	6784	0.706073	0.671211	0	2
Smoking status (preg)	8060	0.396154	0.712577	0	2
Planned pregnancy	8445	0.618828	0.485704	0	1
Breastfeeding status	8453	0.742458	0.437306	0	1
		Outcome Measures: Wave 2			
Child weight status	7906	0.295978	0.56275	0	2
Maternal weight status	7168	0.526228	0.717447	0	2
Paternal weight status	5394	0.773637	0.697617	0	2
Maternal smoking status	8416	0.246911	0.43124	0	1
Screen time	8417	0.15136	0.358421	0	1
Regular meals	8417	0.929547	0.255924	0	1
		Outcome Measures: Wave 3			
Child weight status	8392	0.266921	0.54563	0	2
Maternal weight status	7126	0.551221	0.73095	0	2
Paternal weight status	5674	0.82552	0.702199	0	2
Maternal smoking status	8440	0.23128	0.421676	0	1
Screen time	8438	0.133563	0.340202	0	1
Regular meals	8440	0.943483	0.23093	0	1
Sport	8440	1.043128	1.119493	0	5
Playground/park	8435	0.607706	0.488291	0	1
		Outcome Measures: Wave 4			
Child weight status	8372	0.254539	0.545458	0	2
Maternal weight status	7282	0.619061	0.762505	0	2
Paternal weight status	5884	0.872366	0.711139	0	2
Maternal smoking status	8438	0.224105	0.417016	0	1
Screen time	8441	0.140623	0.347653	0	1
Regular breakfast	8435	0.947244	0.22356	0	1
Sport	8441	1.579315	1.325432	0	5
Playground/park	8437	0.498281	0.500027	0	1
Unhealthy snacks	6995	0.407291	0.491365	0	1

Source: Millennium Cohort Study. Notes: Summary statistics for samples used in final model in Chapter III.

Table B-2: Mplus Output for Lagged Influence on Child Weight Status

	Estimate	S.E.	Est./S.E.	Two-Tailed P-Value
L1 BY				
SMOKE1	-0.733	0.104	-7.044	0.000
PPWTCAT1	-8.346	0.307	-27.227	0.000
BFDUM1	1.061	0.063	16.827	0.000
PLPREG1	0.710	0.078	9.135	0.000
FWTCAT1	-1.376	0.102	-13.467	0.000
WEIGHT1	-0.051	0.007	-7.738	0.000
WTCAT2	-1.335	0.088	-15.220	0.000
L2 BY				
CSMOKE2	-0.736	0.098	-7.499	0.000
MWTCAT2	-11.343	0.350	-32.451	0.000
FWTCAT2	-1.119	0.079	-14.088	0.000
REGMEAL2	0.566	0.089	6.372	0.000
HRSTV2	-0.842	0.074	-11.366	0.000
WTCAT3	-1.633	0.084	-19.454	0.000
L3 BY				
CSMOKE3	-0.657	0.087	-7.558	0.000
MWTCAT3	-11.343	0.350	-32.451	0.000
FWTCAT3	-1.119	0.079	-14.088	0.000
REGMEAL3	0.612	0.084	7.309	0.000
HRSTV3	-0.517	0.065	-7.971	0.000
SPORT3	0.635	0.049	12.875	0.000
PLAY3	0.139	0.053	2.614	0.009
WTCAT4	-1.557	0.076	-20.589	0.000
L4 BY				
CSMOKE4	-0.584	0.076	-7.665	0.000
MWTCAT4	-11.343	0.350	-32.451	0.000
FWTCAT4	-1.119	0.079	-14.088	0.000
HRSTV4	-0.392	0.056	-7.040	0.000
SPORT4	0.511	0.041	12.336	0.000
PLAY4	0.160	0.046	3.460	0.001
SNACK4	-0.263	0.051	-5.192	0.000
BFAST4	0.501	0.075	6.700	0.000
L2 ON				
L1	1.129	0.003	364.102	0.000
L3 ON				
L2	1.129	0.003	364.102	0.000
L4 ON				
L3	1.129	0.003	364.102	0.000
L1 ON				
SESHIGH1	0.030	0.008	3.822	0.000
SESLOW1	-0.066	0.008	-7.884	0.000
SINGLE1	-0.042	0.009	-4.560	0.000
MEDUC1	0.016	0.003	4.771	0.000
L2 ON				
SINGLE2	-0.002	0.005	-0.511	0.609
L3 ON				
SINGLE3	-0.004	0.003	-1.088	0.277
L4 ON				
SINGLE4	-0.008	0.004	-2.094	0.036
WEIGHT1 ON				
SEX1	0.066	0.003	25.267	0.000
AGE1	0.004	0.001	5.525	0.000
ASIAN1	-0.077	0.007	-11.132	0.000
BLACK1	-0.010	0.012	-0.816	0.415
OTHER1	-0.028	0.009	-3.332	0.001
WTCAT2 ON				
ASIAN1	-0.262	0.083	-3.147	0.002
BLACK1	0.191	0.113	1.684	0.092
OTHER1	-0.008	0.092	-0.090	0.928
WTCAT3 ON				
ASIAN1	-0.090	0.080	-1.127	0.260
BLACK1	0.358	0.103	3.475	0.001
OTHER1	-0.037	0.097	-0.385	0.700
WTCAT4 ON				
ASIAN1	0.097	0.076	1.273	0.203
BLACK1	0.347	0.100	3.453	0.001
OTHER1	0.063	0.097	0.651	0.515
L2 WITH				
L1	0.000	0.000	999.000	999.000

L3 WITH				
L2	0.000	0.000	999.000	999.000
L1	0.000	0.000	999.000	999.000
L4 WITH				
L3	0.000	0.000	999.000	999.000
L2	0.000	0.000	999.000	999.000
L1	0.000	0.000	999.000	999.000
Intercepts				
WEIGHT1	-0.169	0.027	-6.291	0.000
Thresholds				
SMOKE1$1	0.626	0.018	34.853	0.000
SMOKE1$2	1.102	0.020	55.179	0.000
BFDUM1$1	-0.181	0.020	-9.294	0.000
BFDUM1$2	0.248	0.020	12.534	0.000
BFDUM1$3	0.862	0.021	40.471	0.000
BFDUM1$4	1.876	0.031	60.657	0.000
PLPREG1$1	-0.289	0.017	-17.454	0.000
FWTCAT1$1	-0.271	0.024	-11.181	0.000
FWTCAT1$2	1.178	0.027	43.472	0.000
PPWTCAT1$1	0.977	0.114	8.564	0.000
PPWTCAT1$2	2.313	0.123	18.763	0.000
REGMEAL2$1	-1.473	0.022	-65.863	0.000
CSMOKE2$1	0.678	0.018	36.877	0.000
MWTCAT2$1	0.362	0.187	1.935	0.053
MWTCAT2$2	2.918	0.198	14.767	0.000
FWTCAT2$1	-0.456	0.024	-18.737	0.000
FWTCAT2$2	0.929	0.025	36.602	0.000
WTCAT2$1	0.691	0.024	29.043	0.000
WTCAT2$2	1.640	0.030	55.349	0.000
HRSTV2$1	1.033	0.021	49.530	0.000
CSMOKE3$1	0.730	0.019	39.059	0.000
SPORT3$1	-0.232	0.017	-13.433	0.000
SPORT3$2	0.576	0.018	31.767	0.000
SPORT3$3	1.255	0.021	58.516	0.000
SPORT3$4	1.907	0.030	64.186	0.000
SPORT3$5	2.415	0.045	54.078	0.000
PLAY3$1	-0.269	0.014	-19.014	0.000
MWTCAT3$1	0.362	0.187	1.935	0.053
MWTCAT3$2	2.918	0.198	14.767	0.000
FWTCAT3$1	-0.456	0.024	-18.737	0.000
FWTCAT3$2	0.929	0.025	36.602	0.000
WTCAT3$1	0.813	0.029	27.935	0.000
WTCAT3$2	1.735	0.034	50.523	0.000
REGMEAL3$1	-1.592	0.025	-64.636	0.000
HRSTV3$1	1.107	0.019	57.662	0.000
CSMOKE4$1	0.755	0.019	40.595	0.000
HRSTV4$1	1.074	0.018	58.202	0.000
SPORT4$1	-0.660	0.018	-37.668	0.000
SPORT4$2	0.080	0.017	4.730	0.000
SPORT4$3	0.740	0.018	40.542	0.000
SPORT4$4	1.387	0.022	62.260	0.000
SPORT4$5	1.928	0.030	64.304	0.000
SNACK4$1	0.227	0.016	14.150	0.000
PLAY4$1	0.009	0.014	0.671	0.502
MWTCAT4$1	0.362	0.187	1.935	0.053
MWTCAT4$2	2.918	0.198	14.767	0.000
FWTCAT4$1	-0.456	0.024	-18.737	0.000
FWTCAT4$2	0.929	0.025	36.602	0.000
WTCAT4$1	0.891	0.031	28.929	0.000
WTCAT4$2	1.738	0.035	49.080	0.000
BFAST4$1	-1.625	0.025	-66.019	0.000
Residual Variances				
WEIGHT1	0.014	0.000	42.151	0.000
L1	0.050	0.000	999.000	999.000
L2	0.001	0.000	999.000	999.000
L3	0.001	0.000	999.000	999.000
L4	0.001	0.000	999.000	999.000

Table B-3: Threshold Parameters for Lifestyle Outcome Measures

	Thresholds				
Parameter			**Initial Period**		
Maternal pre-pregnancy weight category	1.078*** (0.079)	2.434*** (0.092)	-	-	-
Father's Weight Category	-0.257*** (0.020)	1.193*** (0.024)	-	-	-
Mother's Smoking Behaviour whilst pregnant	0.634*** (0.016)	1.110*** (0.019)	-	-	-
Planned pregnancy	-0.297*** (0.015)	-	-	-	-
Breastfeeding behaviour	-0.193*** (0.017)	0.0237*** (0.017)	0.851*** (0.018)	1.865*** (0.029)	-
			Lifestyle Aged 3		
Weight category	0.705*** (0.020)	1.654*** (0.027)	-	-	-
Maternal weight category	0.537*** (0.127)	3.181*** (0.151)	-	-	-
Paternal weight category	-0.442*** (0.020)	0.945*** (0.021)	-	-	-
Mother is a smoker	0.686*** (0.016)	-	-	-	-
More than three hours of TV/computer per day	1.072*** (0.018)	-	-	-	-
Regular meals	-1.479*** (0.022)	-	-	-	-
			Lifestyle Aged 5		
Weight category	0.833*** (0.023)	1.756*** (0.030)	-	-	-
Maternal weight category	0.537*** (0.127)	3.181*** (0.151)	-	-	-
Paternal weight category	-0.442*** (0.020)	0.945*** (0.021)	-	-	-
Mother is a smoker	0.738*** (0.017)	-	-	-	-
More than three hours of TV/computer per day	1.114*** (0.018)	-	-	-	-
Regular meals	-1.600*** (0.024)	-	-	-	-
Times per week plays sport	-0.240*** (0.015)	0.567*** (0.016)	1.245*** (0.020)	1.898*** (0.029)	2.406*** (0.044)
Goes to playground or park at least once a week	-0.271*** (0.014)	-	-	-	-
			Lifestyle Aged 7		
Weight category	0.916*** (0.024)	1.764*** (0.030)	-	-	-
Maternal weight category	0.537*** (0.127)	3.181*** (0.151)	-	-	-
Paternal weight category	-0.442*** (0.020)	0.945*** (0.021)	-	-	-
Mother is a smoker	0.764*** (0.017)	-	-	-	-
More than three hours of TV/computer per day	1.081*** (0.018)	-	-	-	-
Eats breakfast everyday	-1.634*** (0.024)	-	-	-	-
Times per week plays sport	-0.668*** (0.016)	0.072*** (0.015)	0.732*** (0.017)	1.378*** (0.021)	1.920*** (0.029)
Goes to playground or park at least once a week	0.007 (0.014)	-	-	-	-
Unhealthy snacks between meals	0.231*** (0.015)	-	-	-	-

Notes: * p < 0.1, ** p < 0.05, *** p < 0.01.

Figure B-1: Kernel Densities of Lifestyle by Ethnicity

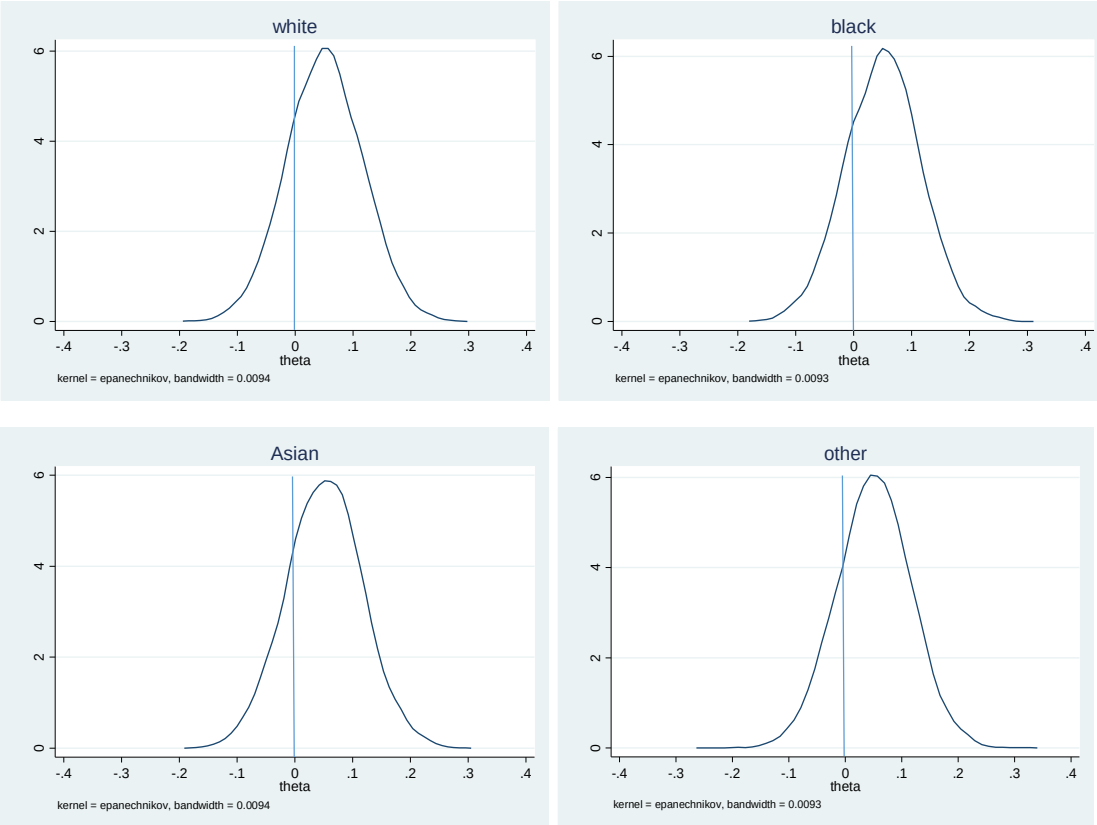


Figure B-2: Kernel Densities of Lifestyle by Family Structure

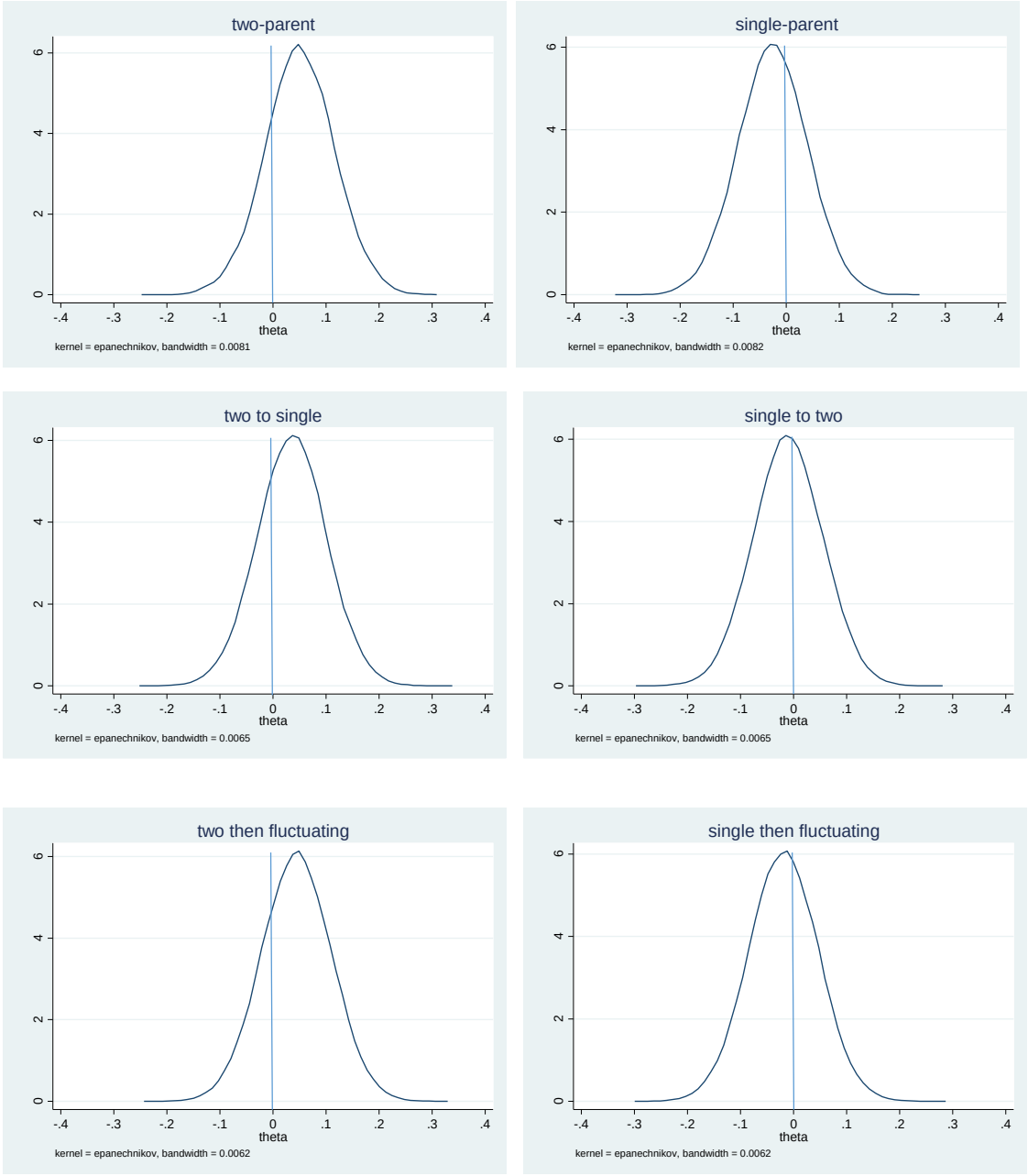


Figure B-3: Kernel Densities of Lifestyle by Advantaged and Disadvantaged

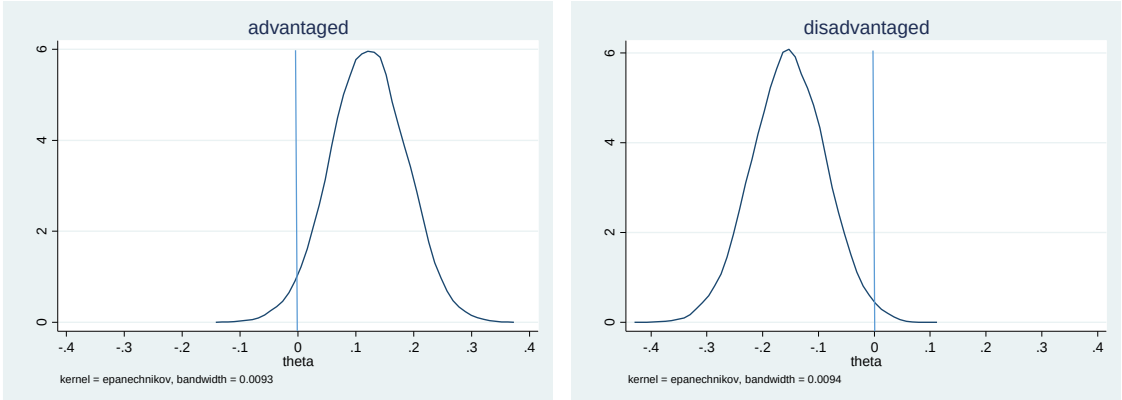


Figure B-4: Kernel Densities of Lifestyle by Parental Weight and SES

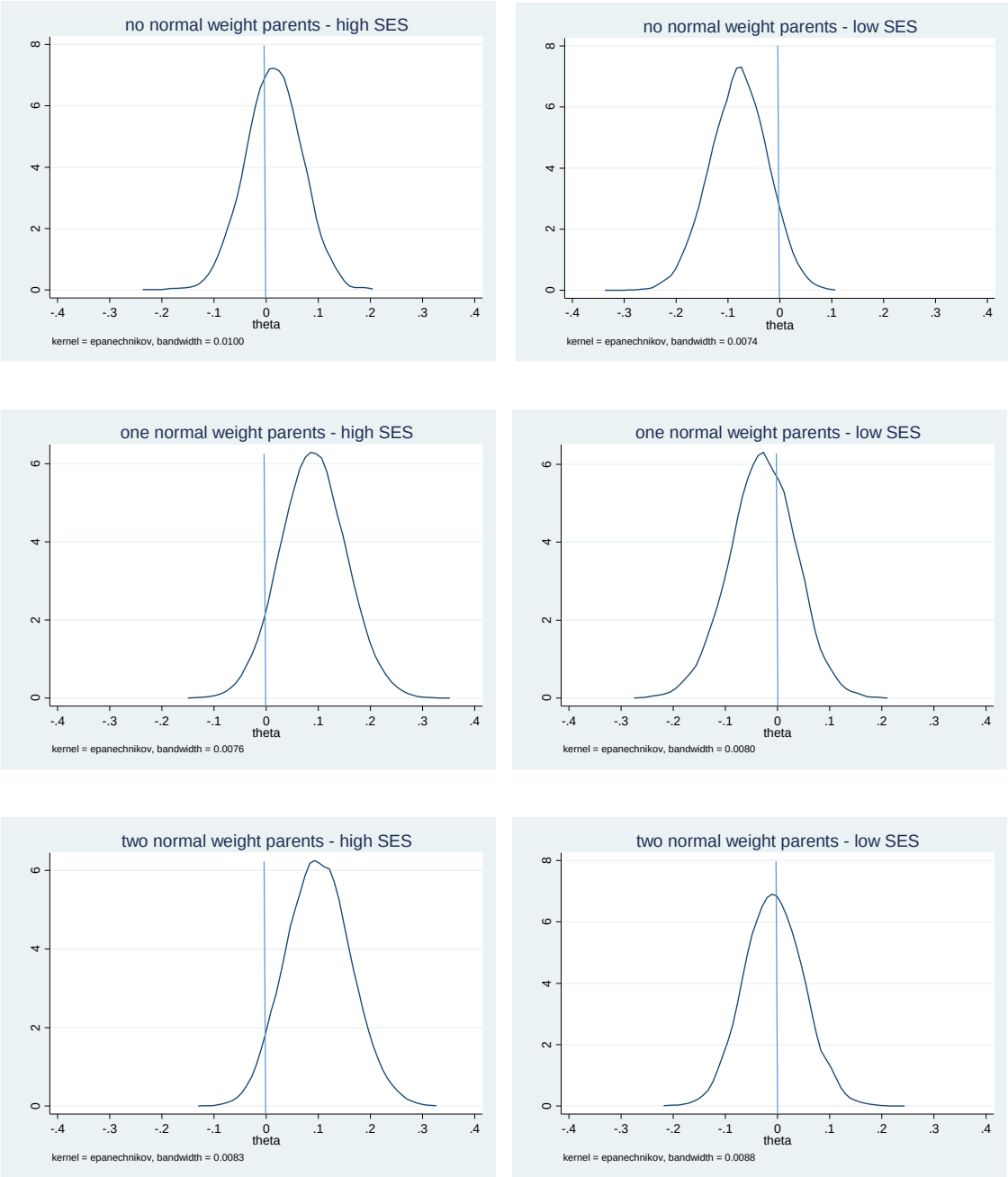


Figure B-5: Kernel Densities of Lifestyle by Lifestyle during Pregnancy

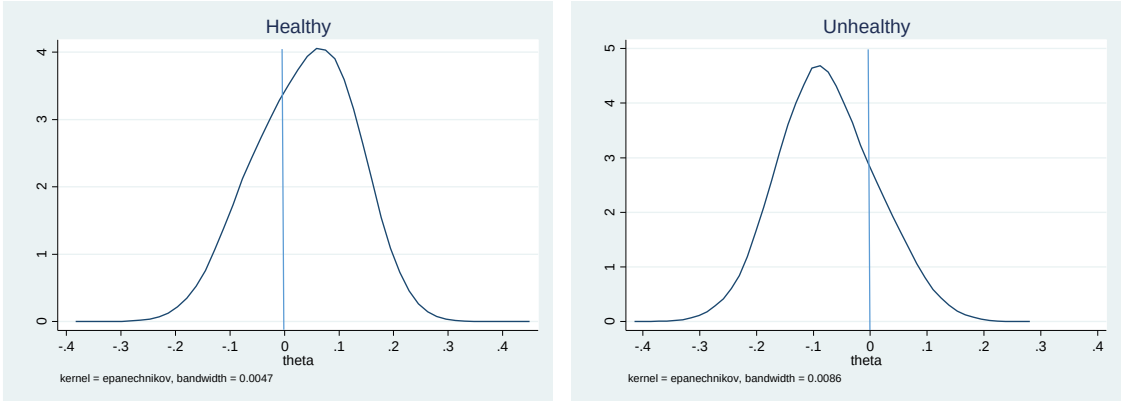


Figure B-6: Kernel Densities of Lifestyle by Diet

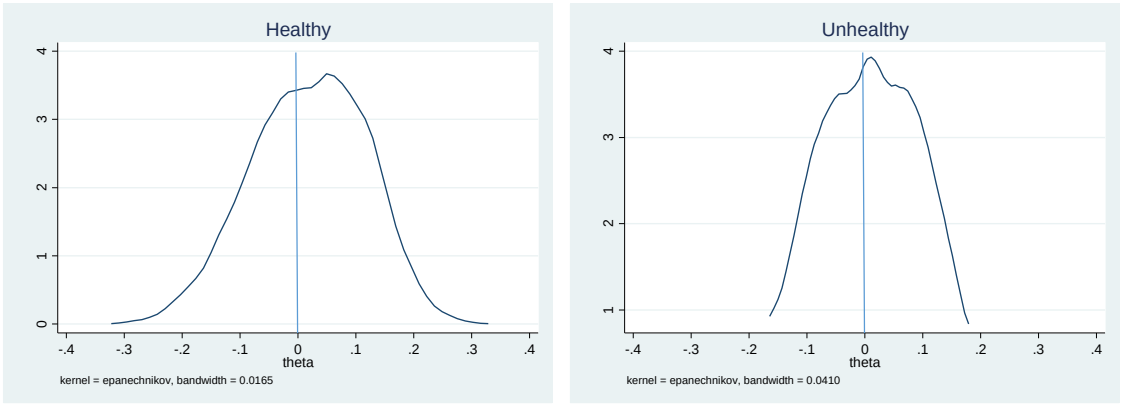
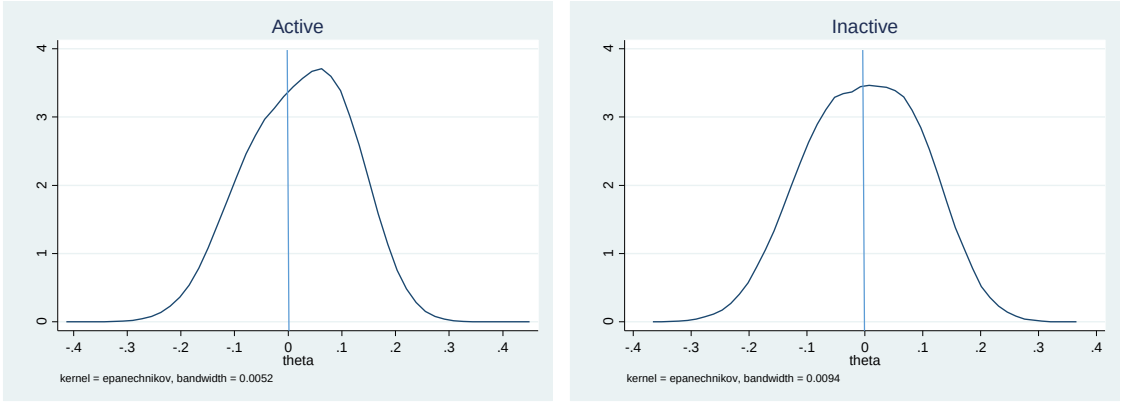


Figure B-7: Kernel Densities of Lifestyle by Physical Activity



C. Appendix: Childhood Adiposity, Family Lifestyle and Childhood Health

Table C-1: Summary Statistics for Final Model

Variable	N	Mean	Std. Dev.	Min	Max
		Independent Variables			
Age	9014	42.11676	2.07706	35	55
Sex	9014	0.509767	0.499932	0	1
High SES	9014	0.396755	0.489251	0	1
Low SES	9014	0.401611	0.490251	0	1
Maternal educ.	9014	2.947909	1.105146	1	5
Single parent – 9 months	9014	0.101755	0.302342	0	1
Single parent – age 3	9014	0.121179	0.326353	0	1
Single parent – age 5	9014	0.142258	0.349334	0	1
Single parent – age 7	9014	0.165986	0.372089	0	1
		Outcome Measures: Wave 1			
Child weight (kg)	8912	8.847002	1.26545	3.06	19.2
Pre-preg. weight status	8237	0.358626	0.650645	0	2
Paternal weight status	7270	0.707978	0.671844	0	2
Smoking status (preg)	8632	0.397243	0.713416	0	2
Planned pregnancy	8996	0.619264	0.485595	0	1
Breastfeeding status	9005	0.74304	0.436981	0	1
Hearing problems	7873	0.063381	0.243663	0	1
Birth weight	8999	3.482836	0.466491	2.5	5.87
Chest problems	9014	0.285178	0.451524	0	1
Gestational age	9010	279.3567	10.23994	218	296
Asthma	9014	0.061803	0.240811	0	1
Special care unit	9005	0.064295	0.245292	0	1
Feeding problems	9014	0.036199	0.186796	0	1
Growing problems	9014	0.012692	0.111947	0	1
		Outcome Measures: Wave 2			
Child weight status	8449	0.297432	0.563189	0	2
Maternal weight status	7658	0.53147	0.71905	0	2
Paternal weight status	5768	0.776526	0.697159	0	2
Maternal smoking status	9011	0.246477	0.430983	0	1
Screen time	9012	0.150577	0.357656	0	1
Regular meals	9012	0.928207	0.258159	0	1
Hearing problems	8960	0.044643	0.20653	0	1
Longstanding Illness	9011	0.151925	0.358969	0	1
Hospitalisation	9012	0.213271	0.499705	0	2
Headache/Stomach/Sickness	8806	0.017147	0.129828	0	1
Asthma	8967	0.150866	0.357938	0	1
Speech problems	9012	0.126609	0.332553	0	1
		Outcome Measures: Wave 3			
Child weight status	8980	0.269154	0.548206	0	2
Maternal weight status	7627	0.558411	0.733658	0	2
Paternal weight status	6088	0.829172	0.70067	0	2
Maternal smoking status	8991	0.231357	0.421723	0	1
Screen time	8989	0.133909	0.340573	0	1
Regular meals	8991	0.94324	0.231397	0	1

Sport	8991	1.050232	1.124594	0	5
Playground/park	8986	0.608214	0.488176	0	1
Hearing problems	8980	0.126399	0.332316	0	1
Longstanding Illness	8988	0.187604	0.390417	0	1
Hospitalisation	8991	0.135443	0.40227	0	2
Headache/Stomach/Sickness	8927	0.0237482	0.1522721	0	1
Medication	8988	0.090205	0.286491	0	1
Parent assessed health	8990	1.619232	0.808377	1	4
		Outcome Measures: Wave 4			
Child weight status	8966	0.256859	0.548328	0	2
Maternal weight status	7790	0.625161	0.765264	0	2
Paternal weight status	6303	0.872124	0.711127	0	2
Maternal smoking status	8988	0.223796	0.41681	0	1
Screen time	8990	0.140976	0.348016	0	1
Regular breakfast	8985	0.94752	0.223005	0	1
Sport	8991	1.585196	1.326849	0	5
Playground/park	8985	0.498063	0.500024	0	1
Unhealthy snacks	7500	0.4072	0.491345	0	1
Hearing problems	8971	0.062763	0.24255	0	1
Longstanding Illness	8986	0.178014	0.382546	0	1
Hospitalisation	8986	0.098638	0.335577	0	2
Headache/Stomach/Sickness	8917	0.031176	0.173804	0	1
Medication	8980	0.120527	0.325595	0	1
Parent assessed health	8990	1.491368	0.739907	1	4
Autism/Aspergers	8979	0.016619	0.127845	0	1
Measles	8972	0.06442	0.245512	0	1

Source: Millennium Cohort Study. Notes: Summary statistics for samples used in final model in Chapter IV.

Table C-2: Mplus Output for Lagged Influences on Child Weight Status

		Estimate	S.E.	Est./S.E.	Two-Tailed P-Value
L1	BY				
	SMOKE1	-0.734	0.100	-7.354	0.000
	PPWTCAT1	-8.392	0.303	-27.679	0.000
	BFDUM1	1.095	0.062	17.736	0.000
	PLPREG1	0.714	0.076	9.391	0.000
	FWTCAT1	-1.419	0.100	-14.150	0.000
	WEIGHT1	-0.055	0.006	-8.628	0.000
	WTCAT2	-1.337	0.084	-15.868	0.000
L2	BY				
	CSMOKE2	-0.660	0.099	-6.666	0.000
	MWTCAT2	-12.368	0.538	-22.978	0.000
	FWTCAT2	-1.434	0.102	-14.024	0.000
	WTCAT3	-1.642	0.080	-20.469	0.000
	REGMEAL2	0.639	0.089	7.160	0.000
	HRSTV2	-0.895	0.075	-11.928	0.000
L3	BY				
	CSMOKE3	-0.634	0.082	-7.691	0.000
	MWTCAT3	-12.274	0.551	-22.271	0.000
	FWTCAT3	-1.161	0.088	-13.245	0.000
	WTCAT4	-1.642	0.080	-20.469	0.000
	REGMEAL3	0.657	0.086	7.619	0.000
	HRSTV3	-0.489	0.053	-9.300	0.000
	SPORT3	0.582	0.043	13.694	0.000
	PLAY3	0.162	0.043	3.803	0.000
L4	BY				
	CSMOKE4	-0.634	0.082	-7.691	0.000
	MWTCAT4	-12.274	0.551	-22.271	0.000
	FWTCAT4	-1.161	0.088	-13.245	0.000
	HRSTV4	-0.489	0.053	-9.300	0.000
	SPORT4	0.582	0.043	13.694	0.000
	PLAY4	0.162	0.043	3.803	0.000
	SNACK4	-0.263	0.053	-5.011	0.000
	BFAST4	0.528	0.079	6.717	0.000
H1	BY				
	GEST1	0.091	0.020	4.575	0.000
	CHEST1	-0.653	0.096	-6.770	0.000
	ASTHMA1	-5.402	1.331	-4.057	0.000
	HEARING1	-0.761	0.155	-4.917	0.000
	BWEIGHT1	0.039	0.009	4.240	0.000
	SPCARE1	-1.090	0.205	-5.319	0.000
	FEEDPR1	-0.393	0.169	-2.328	0.020
	GROWPR1	-0.834	0.277	-3.015	0.003
	WTCAT2	-0.075	0.162	-0.465	0.642
H2	BY				
	LONGILL2	-2.134	0.185	-11.557	0.000
	HOSP2	-1.321	0.115	-11.456	0.000
	ASTHMA2	-3.005	0.403	-7.465	0.000
	HEARING2	-1.180	0.129	-9.185	0.000
	HEADA2	-0.684	0.128	-5.346	0.000
	CSMOKE2	-0.526	0.071	-7.419	0.000
	SPPROB2	-0.786	0.088	-8.892	0.000
	WTCAT3	-0.138	0.075	-1.832	0.067
H3	BY				
	LONGILL3	-4.956	0.277	-17.895	0.000
	MEDIC3	-3.337	0.173	-19.246	0.000
	HOSP3	-1.286	0.065	-19.659	0.000
	HEADA3	-0.950	0.081	-11.751	0.000
	HEARING3	-0.900	0.056	-16.145	0.000
	HEALTH3	-2.268	0.095	-23.862	0.000
	WTCAT4	-0.138	0.075	-1.832	0.067
H4	BY				
	LONGILL4	-4.956	0.277	-17.895	0.000
	MEDIC4	-3.337	0.173	-19.246	0.000
	HOSP4	-1.286	0.065	-19.659	0.000
	AUTASP4	-1.413	0.116	-12.142	0.000
	MEASLE4	-0.184	0.062	-2.977	0.003
	HEADA4	-0.950	0.081	-11.751	0.000
	HEARING4	-0.900	0.056	-16.145	0.000
	HEALTH4	-2.268	0.095	-23.862	0.000
U	BY				
	H1	1.000	0.000	999.000	999.000
	H2	1.000	0.000	999.000	999.000
	H3	1.000	0.000	999.000	999.000
	H4	1.000	0.000	999.000	999.000
L2	ON				
	L1	1.105	0.012	95.971	0.000

L3	ON				
L2		1.105	0.012	95.971	0.000
L4	ON				
L3		1.105	0.012	95.971	0.000
H2	ON				
H1		1.221	0.070	17.401	0.000
L1		0.160	0.027	5.857	0.000
H3	ON				
H2		0.812	0.046	17.540	0.000
L2		0.042	0.014	3.003	0.003
H4	ON				
H3		0.812	0.046	17.540	0.000
L3		0.042	0.014	3.003	0.003
L1	ON				
SESHIGH1		0.039	0.007	5.156	0.000
SESLOW1		-0.052	0.007	-7.561	0.000
SINGLE1		-0.043	0.009	-4.639	0.000
MEDUC1		0.023	0.003	7.814	0.000
L2	ON				
SINGLE2		0.008	0.007	1.173	0.241
L3	ON				
SINGLE3		-0.002	0.003	-0.646	0.518
L4	ON				
SINGLE4		-0.002	0.003	-0.646	0.518
H1	ON				
SESHIGH1		-0.003	0.012	-0.266	0.790
SESLOW1		-0.068	0.013	-5.375	0.000
WEIGHT1	ON				
SEX1		0.066	0.003	26.083	0.000
AGE1		0.004	0.001	5.812	0.000
ASIAN1		-0.073	0.007	-10.765	0.000
BLACK1		-0.011	0.012	-0.920	0.358
OTHER1		-0.028	0.008	-3.453	0.001
BWEIGHT1	ON				
SEX1		0.012	0.001	12.028	0.000
ASIAN1		-0.032	0.002	-16.141	0.000
BLACK1		-0.007	0.004	-1.875	0.061
OTHER1		-0.016	0.003	-5.420	0.000
WTCAT2	ON				
ASIAN1		-0.232	0.080	-2.920	0.004
BLACK1		0.161	0.110	1.464	0.143
OTHER1		-0.002	0.090	-0.020	0.984
WTCAT3	ON				
ASIAN1		0.006	0.070	0.080	0.936
BLACK1		0.318	0.092	3.458	0.001
OTHER1		0.000	0.087	0.005	0.996
WTCAT4	ON				
ASIAN1		0.006	0.070	0.080	0.936
BLACK1		0.318	0.092	3.458	0.001
OTHER1		0.000	0.087	0.005	0.996
L2	WITH				
L1		0.000	0.000	999.000	999.000
L3	WITH				
L2		0.000	0.000	999.000	999.000
L1		0.000	0.000	999.000	999.000
L4	WITH				
L3		0.000	0.000	999.000	999.000
L2		0.000	0.000	999.000	999.000
L1		0.000	0.000	999.000	999.000
H1	WITH				
L1		0.000	0.000	999.000	999.000
L2		0.000	0.000	999.000	999.000
L3		0.000	0.000	999.000	999.000
L4		0.000	0.000	999.000	999.000
H2	WITH				
L1		0.000	0.000	999.000	999.000
L2		0.000	0.000	999.000	999.000
L3		0.000	0.000	999.000	999.000
L4		0.000	0.000	999.000	999.000
H1		0.000	0.000	999.000	999.000

H3	WITH				
	L1	0.000	0.000	999.000	999.000
	L2	0.000	0.000	999.000	999.000
	L3	0.000	0.000	999.000	999.000
	L4	0.000	0.000	999.000	999.000
	H1	0.000	0.000	999.000	999.000
	H2	0.000	0.000	999.000	999.000
H4	WITH				
	L1	0.000	0.000	999.000	999.000
	L2	0.000	0.000	999.000	999.000
	L3	0.000	0.000	999.000	999.000
	L4	0.000	0.000	999.000	999.000
	H1	0.000	0.000	999.000	999.000
	H2	0.000	0.000	999.000	999.000
	H3	0.000	0.000	999.000	999.000
Intercepts					
	GEST1	2.406	0.001	1798.463	0.000
	BWEIGHT1	0.010	0.001	12.857	0.000
	WEIGHT1	-0.171	0.026	-6.532	0.000
Thresholds					
	CHEST1$1	0.594	0.016	37.326	0.000
	ASTHMA1$1	2.618	0.406	6.448	0.000
	HEARING1$1	1.572	0.027	58.719	0.000
	SPCARE1$1	1.601	0.030	52.623	0.000
	FEEDPR1$1	1.816	0.027	66.998	0.000
	GROWPR1$1	2.300	0.050	46.137	0.000
	SMOKE1$1	0.604	0.017	35.308	0.000
	SMOKE1$2	1.078	0.019	57.099	0.000
	BFDUM1$1	-0.135	0.018	-7.485	0.000
	BFDUM1$2	0.293	0.018	15.862	0.000
	BFDUM1$3	0.907	0.020	44.761	0.000
	BFDUM1$4	1.920	0.031	62.876	0.000
	PLPREG1$1	-0.268	0.016	-17.032	0.000
	FWTCAT1$1	-0.319	0.022	-14.481	0.000
	FWTCAT1$2	1.133	0.024	46.837	0.000
	PPWTCAT1$1	0.702	0.086	8.171	0.000
	PPWTCAT1$2	2.024	0.096	21.170	0.000
	LONGILL2$1	1.370	0.041	33.748	0.000
	HOSP2$1	1.086	0.025	43.437	0.000
	HOSP2$2	1.976	0.033	60.023	0.000
	ASTHMA2$1	1.633	0.136	12.012	0.000
	HEARING2$1	1.885	0.038	49.836	0.000
	HEADA2$1	2.206	0.042	52.669	0.000
	SPPROB2$1	1.211	0.022	56.150	0.000
	REGMEAL2$1	-1.443	0.021	-67.354	0.000
	CSMOKE2$1	0.688	0.019	35.588	0.000
	MWTCAT2$1	0.045	0.139	0.325	0.745
	MWTCAT2$2	2.496	0.158	15.838	0.000
	FWTCAT2$1	-0.435	0.025	-17.106	0.000
	FWTCAT2$2	0.967	0.026	36.842	0.000
	WTCAT2$1	0.649	0.022	29.873	0.000
	WTCAT2$2	1.604	0.028	57.603	0.000
	HRSTV2$1	1.007	0.019	52.314	0.000
	LONGILL3$1	2.032	0.088	23.180	0.000
	MEDIC3$1	2.106	0.057	36.881	0.000
	HOSP3$1	1.450	0.022	67.088	0.000
	HOSP3$2	2.406	0.029	83.532	0.000
	HEADA3$1	2.062	0.028	73.221	0.000
	HEARING3$1	1.405	0.019	75.623	0.000
	HEALTH3$1	0.352	0.025	13.975	0.000
	HEALTH3$2	1.588	0.030	52.734	0.000
	HEALTH3$3	2.601	0.040	65.522	0.000
	CSMOKE3$1	0.716	0.017	43.293	0.000
	SPORT3$1	-0.416	0.014	-29.962	0.000
	SPORT3$2	0.336	0.014	23.790	0.000
	SPORT3$3	0.984	0.016	63.272	0.000
	SPORT3$4	1.613	0.019	85.027	0.000
	SPORT3$5	2.126	0.025	83.669	0.000
	PLAY3$1	-0.121	0.011	-10.835	0.000
	MWTCAT3$1	-0.263	0.157	-1.673	0.094
	MWTCAT3$2	2.483	0.181	13.705	0.000
	FWTCAT3$1	-0.552	0.024	-23.046	0.000
	FWTCAT3$2	0.857	0.024	36.473	0.000
	WTCAT3$1	0.789	0.025	31.970	0.000
	WTCAT3$2	1.675	0.029	57.660	0.000
	REGMEAL3$1	-1.567	0.023	-67.899	0.000
	HRSTV3$1	1.070	0.015	73.632	0.000
	LONGILL4$1	2.032	0.088	23.180	0.000
	MEDIC4$1	2.106	0.057	36.881	0.000
	HOSP4$1	1.450	0.022	67.088	0.000
	HOSP4$2	2.406	0.029	83.532	0.000
	AUTASP4$1	2.458	0.057	42.964	0.000
	MEASLE4$1	1.526	0.021	73.349	0.000
	HEADA4$1	2.062	0.028	73.221	0.000
	HEARING4$1	1.405	0.019	75.623	0.000
	HEALTH4$1	0.352	0.025	13.975	0.000
	HEALTH4$2	1.588	0.030	52.734	0.000
	HEALTH4$3	2.601	0.040	65.522	0.000

CSMOKE4$1	0.716	0.017	43.293	0.000
HRSTV4$1	1.070	0.015	73.632	0.000
SPORT4$1	-0.416	0.014	-29.962	0.000
SPORT4$2	0.336	0.014	23.790	0.000
SPORT4$3	0.984	0.016	63.272	0.000
SPORT4$4	1.613	0.019	85.027	0.000
SPORT4$5	2.126	0.025	83.669	0.000
SNACK4$1	0.215	0.016	13.796	0.000
PLAY4$1	-0.121	0.011	-10.835	0.000
MWTCAT4$1	-0.263	0.157	-1.673	0.094
MWTCAT4$2	2.483	0.181	13.705	0.000
FWTCAT4$1	-0.552	0.024	-23.046	0.000
FWTCAT4$2	0.857	0.024	36.473	0.000
WTCAT4$1	0.789	0.025	31.970	0.000
WTCAT4$2	1.675	0.029	57.660	0.000
BFAST4$1	-1.605	0.023	-69.069	0.000
Variances				
U	0.002	0.002	1.013	0.311
Residual Variances				
GEST1	0.010	0.000	38.648	0.000
BWEIGHT1	0.002	0.000	42.717	0.000
WEIGHT1	0.014	0.000	42.386	0.000
L1	0.050	0.000	999.000	999.000
L2	0.001	0.000	999.000	999.000
L3	0.001	0.000	999.000	999.000
L4	0.001	0.000	999.000	999.000
H1	0.050	0.000	999.000	999.000
H2	0.050	0.000	999.000	999.000
H3	0.050	0.000	999.000	999.000
H4	0.050	0.000	999.000	999.000

Table C-3: Estimated Factor Loadings for Family Lifestyle

	Factor Loading λ (Equation (IV.4)) (Standard Error)			
Outcome Measure (dependent variable)	**Initial**	**Age Three Years**	**Age Five Years**	**Age Seven Years**
Weight (kg)	-0.054*** (0.006)	-	-	-
Weight Category	-	-1.161*** (0.076)	-1.441*** (0.075)	-1.441*** (0.075)
Maternal Weight Category¥	-8.383*** (0.303)	-12.276*** (0.532)	11.954*** (0.525)	11.954*** (0.525)
Father's Weight Category	-1.415*** (0.100)	-1.420*** (0.101)	-1.137*** (0.085)	-1.137*** (0.085)
Mother's Smoking Behaviour€	-0.736*** (0.100)	-0.655*** (0.098)	-0.622*** (0.080)	-0.622*** (0.080)
Planned Pregnancy	0.716*** (0.076)	-	-	-
Breastfeeding Behaviour	1.095*** (0.062)	-	-	-
Regular Meals	-	0.634*** (0.088)	0.646*** (0.085)	-
Over Three Hours TV per day	-	-0.887*** (0.074)	-0.479*** (0.051)	-0.479*** (0.051)
Sport	-	-	0.570*** (0.041)	0.570*** (0.041)
Playground/Park	-	-	0.158*** (0.042)	0.158*** (0.042)
Unhealthy Snacks	-	-	-	-0.257*** (0.051)
Regular Breakfast	-	-	-	0.516*** (0.077)

Notes: * p < 0.1, ** p < 0.05, *** p < 0.01, ¥pre-pregnancy weight category in initial conditions, €smoking behaviour during pregnancy in initial conditions.

Table C-4: Threshold Parameters for Family Lifestyle Outcome Measures

	Thresholds				
Parameter			**Initial Lifestyle**		
Maternal pre-pregnancy weight category	0.716*** (0.083)	2.037*** (0.093)	-	-	-
Father's Weight Category	-0.317*** (0.022)	1.135*** (0.024)	-	-	-
Mother's Smoking Behaviour whilst pregnant	0.605*** (0.017)	1.080*** (0.019)	-	-	-
Planned pregnancy	-0.269*** (0.016)	-	-	-	-
Breastfeeding behaviour	-0.136*** (0.018)	0.292*** (0.018)	0.905*** (0.020)	1.918*** (0.030)	-
			Lifestyle Aged 3		
Weight category$^{\Psi}$	0.651*** (0.021)	1.605*** (0.027)	-	-	-
Maternal weight category	0.066 (0.135)	2.519*** (0.154)	-	-	-
Paternal weight category	-0.432*** (0.025)	0.969*** (0.026)	-	-	-
Mother is a smoker$^{\Psi}$	0.689*** (0.019)	-	-	-	-
More than three hours of TV/computer per day	1.008*** (0.019)	-	-	-	-
Regular meals	-1.444*** (0.021)	-	-	-	-
			Lifestyle Aged 5		
Weight category$^{\Psi}$	0.792*** (0.024)	1.677*** (0.028)	-	-	-
Maternal weight category	-0.239 (0.151)	2.494*** (0.176)	-	-	-
Paternal weight category	-0.550*** (0.024)	0.860*** (0.023)	-	-	-
Mother is a smoker	0.718*** (0.016)	-	-	-	-
More than three hours of TV/computer per day	1.071*** (0.014)	-	-	-	-
Regular meals	-1.569*** (0.023)	-	-	-	-
Times per week plays sport	-0.417*** (0.014)	0.335*** (0.014)	0.983*** (0.015)	1.612*** (0.019)	2.125*** (0.025)
Goes to playground or park at least once a week	-0.122*** (0.011)	-	-	-	-
			Lifestyle Aged 7		
Weight category$^{\Psi}$	0.792*** (0.024)	1.677*** (0.028)	-	-	-
Maternal weight category	-0.239 (0.151)	2.494*** (0.176)	-	-	-
Paternal weight category	-0.550*** (0.024)	0.860*** (0.023)	-	-	-
Mother is a smoker	0.718*** (0.016)	-	-	-	-
More than three hours of TV/computer per day	1.071*** (0.014)	-	-	-	-
Times per week plays sport	-0.417*** (0.014)	0.335*** (0.014)	0.983*** (0.015)	1.612*** (0.019)	2.125*** (0.025)
Goes to playground or park at least once a week	-0.122*** (0.011)	-	-	-	-
Unhealthy snacks between meals	0.216*** (0.016)	-	-	-	-
Breakfast most days	-1.606*** (0.023)	-	-	-	-

Notes: * p < 0.1, ** p < 0.05, *** p < 0.01, $^{\Psi}$Thresholds apply to effects though both family lifestyle and child health.

Table C-5: Threshold Parameters for Child Health Outcome Measures

		Thresholds	
Parameter		**Initial Child Health**	
Asthma	2.605***	-	-
	(0.390)		
Hearing Problems	1.572***	-	-
	(0.027)		
Chest Infection	0.594***	-	-
	(0.016)		
Special Care Unit	1.601***	-	-
	(0.030)		
Feeding Problems	1.816***	-	-
	(0.027)		
Growing Problems	2.301***	-	-
	(0.050)		
		Health Aged 3	
Weight category[Ψ]	0.651***	1.605***	-
	(0.021)	(0.027)	
Asthma[¥]	1.630***	-	-
	(0.132)		
Mother is a smoker[Ψ]	0.689***	-	-
	(0.019)		
Longstanding Illness	1.371***	-	-
	(0.041)		
Hospitalised	1.087***	1.977***	-
	(0.025)	(0.033)	
Headaches/Sickness	2.207***	-	-
	(0.042)		
Hearing Problems	1.885***	-	-
	(0.038)		
Speech/Language Problems	1.211***	-	-
	(0.022)		
		Health Aged 5	
Weight category[Ψ]	0.792***	1.677***	-
	(0.024)	(0.028)	
Longstanding Illness	2.033***	-	-
	(0.087)		
Medication	2.108***	-	-
	(0.057)		
Hospitalised	1.450***	-	-
	(0.022)		
Headaches/Sickness	2.407***	-	-
	(0.029)		
Hearing Problems	1.405***	-	-
	(0.019)		
Parent Assessed Health	0.353***	1.589***	2.602***
	(0.025)	(0.030)	(0.040)
		Health Aged 7	
Weight category[Ψ]	0.792***	1.677***	-
	(0.024)	(0.028)	
Longstanding Illness	2.033***	-	-
	(0.087)		
Medication	2.108***	-	-
	(0.057)		
Hospitalised	1.450***	-	-
	(0.022)		
Headaches/Sickness	2.407***	-	-
	(0.029)		
Hearing Problems	1.405***	-	-
	(0.019)		
Parent Assessed Health	0.353***	1.589***	2.602***
	(0.025)	(0.030)	(0.040)
Weight category[Ψ]	0.792***	1.677***	-
	(0.024)	(0.028)	
Autism	2.458***	-	-
	(0.057)		
Measles	1.526***	-	-
	(0.021)		

Notes: * p < 0.1, ** p < 0.05, *** p < 0.01, [Ψ]Thresholds apply to effects though both family lifestyle and child health, [¥]Thresholds fixed at 5.

Figure C-1: Kernel Densities of Health by Socioeconomics Status

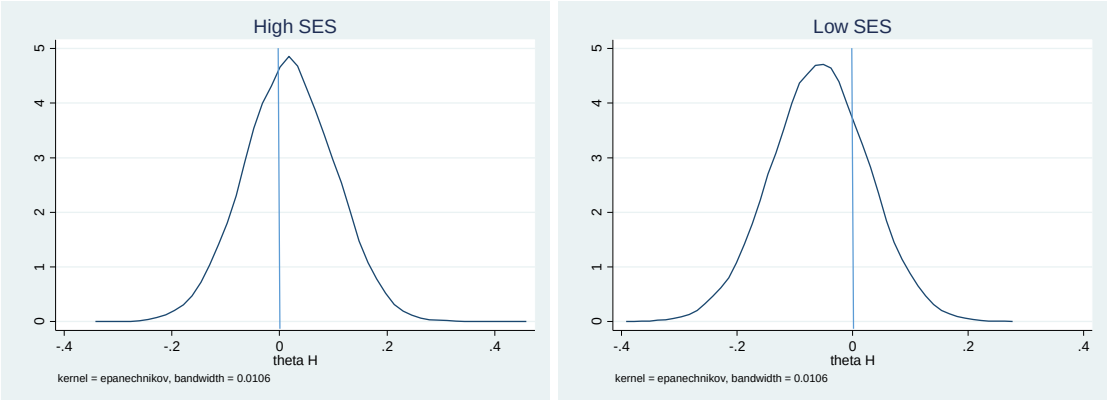


Figure C-2: Kernel Densities of Health by Birth Weight

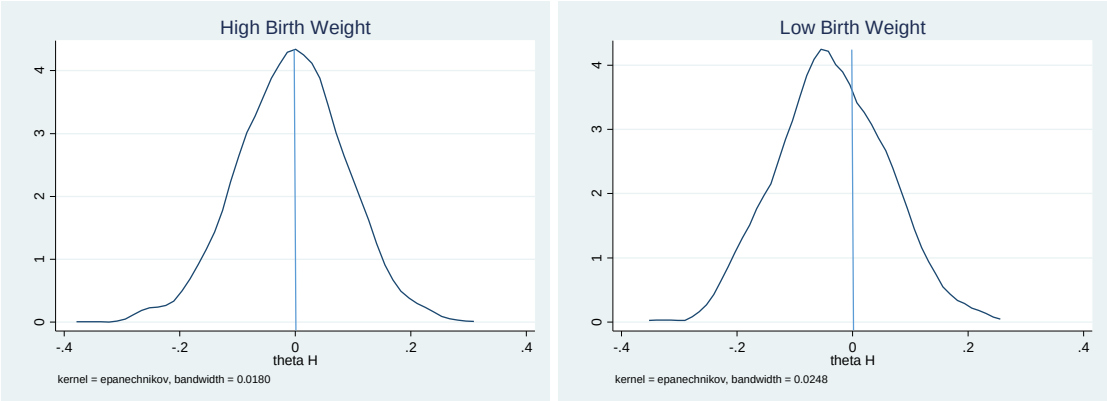


Figure C-3: Kernel Densities of Health by Diet

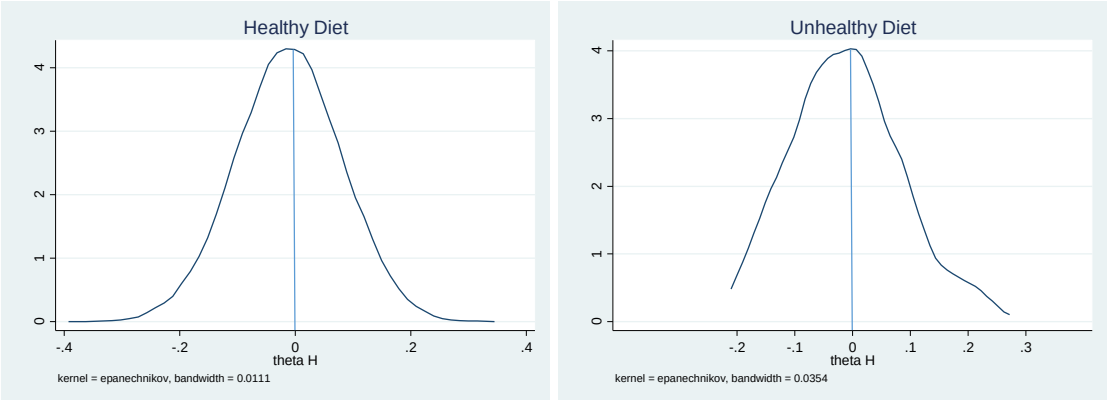


Figure C-4: Kernel Densities of Health by Physical Activity

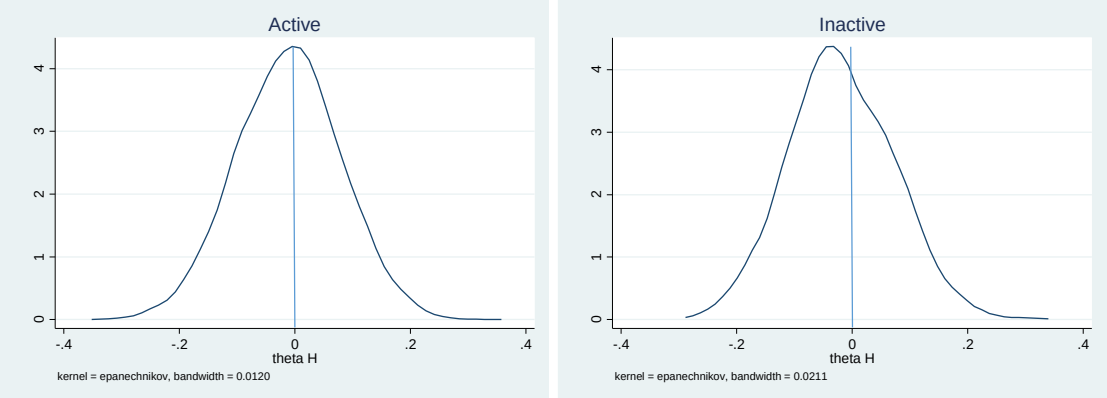


Figure C-5: Kernel Densities by Infant Feeding and Growing Characteristics