UNIVERSITY OF MEDICINE AND HEALTH SCIENCES

Royal College of Surgeons in Ireland

repository@rcsi.com

A social-ecological approach to examine body mass index during early childhood in Ireland

AUTHOR(S)

Samira Barbara Jabakhanji

CITATION

Jabakhanji, Samira Barbara (2020): A social-ecological approach to examine body mass index during early childhood in Ireland. Royal College of Surgeons in Ireland. Thesis. https://doi.org/10.25419/rcsi.13042145.v1

DOI

10.25419/rcsi.13042145.v1

LICENCE

CC BY-NC-SA 4.0

This work is made available under the above open licence by RCSI and has been printed from https://repository.rcsi.com. For more information please contact repository@rcsi.com

URL

https://repository.rcsi.com/articles/thesis/A_social-ecological_approach_to_examine_body_mass_index_during_early_childhood_in_Ireland/13042145/1

A social-ecological approach to examine body mass index during early childhood in Ireland

Samira Barbara Jabakhanji, BSc MSc
Department of Epidemiology and Public Health Medicine,
Royal College of Surgeons in Ireland

A thesis submitted to the Royal College of Surgeons in Ireland,
in fulfilment of the degree of Doctor of Philosophy,
in the Department of Epidemiology and Public Health Medicine,
Division of Population Health Sciences

Primary Supervisor: Dr. Regien Biesma
Co-Supervisors: Dr. Fiona Boland and Dr. Mark Ward

May 2019

Candidate Thesis Declaration

I declare that this thesis, which I submit to RCSI for examination in consideration of the award of a higher degree of Doctor of Philosophy, is my own personal effort. Where any of the content presented is the result of input or data from a related collaborative research programme this is duly acknowledged in the text such that it is possible to ascertain how much of the work is my own. I have not already obtained a degree in RCSI or elsewhere on the basis of this work. Furthermore, I took reasonable care to ensure that the work is original, and, to the best of my knowledge, does not breach copyright law, and has not been taken from other sources except where such work has been cited and acknowledged within the text.

Signed

Student Number 15147410

Date 7th May 2019

Table of Contents

LIST OF FIGURES **8**

LIST OF TABLES **10**

LIST OF ABBREVIATIONS **14**

SUMMARY **16**

PUBLICATIONS AND PRESENTATIONS ARISING FROM THIS THESIS **17**

ACKNOWLEDGEMENTS **18**

CHAPTER 1: INTRODUCTION TO OVERWEIGHT AND OBESITY IN THE EARLY CHILDHOOD YEARS **20**

1.1 Overview of childhood obesity **21**

1.2 Consequences and co-morbidities of childhood overweight and obesity **24**
1.2.1 Presence of overweight and obesity later in life 24
1.2.2 Early onset of ill-health 25
1.2.3 Higher risk of multiple morbidity 27
1.2.4 Early developmental disadvantages compared to lean peers. 28
1.2.5 Cost of childhood obesity 28

1.3 Research gaps **30**
1.3.1 Monitoring of childhood obesity 30
1.3.2 Inequalities in obesity prevalence and predictors of childhood obesity 31
1.3.3 Role of cohort studies in studying childhood obesity 33

1.4 Aims and objectives of this thesis **35**

1.5 Overview of chapters **36**

CHAPTER 2: EARLY CHILDHOOD OVERWEIGHT AND OBESITY IN THE LITERATURE **38**

2.1 Introduction and objectives of this chapter **39**

2.2 Conceptual models of childhood obesity **40**
2.2.1 The Social Ecological Model: Origin and use in childhood obesity 40
2.2.2 Extension of the Social Ecological Model based on evidence from the past 20 years 43

2.2.3 Literature review: Risk factors of childhood obesity 46

2.3 Early child growth and childhood obesity 50
2.3.1. Prenatal development 50
2.3.2 Infancy 55
2.3.3 Child growth after infancy 58
2.3.4 Key findings on early child growth and childhood obesity 68

2.4 Inequalities in childhood obesity 69
2.4.1 The socio-economic status of families with young children 69
2.4.2 Non-parental childcare environment 71
2.4.3 Characteristics of the neighbourhood families live in 72
2.4.4 Ethnic background 74
2.4.5 Theories on the development of inequalities in childhood obesity 76
2.4.6 Key findings on inequalities in childhood obesity until five years of age 82

2.5 Discussion 83
2.5.1 Presentation of the extended conceptual model 83
2.5.2 Hypotheses 85

CHAPTER 3: METHODOLOGY 88

3.1 Introduction and objectives of this chapter 89

3.2 The Growing Up in Ireland Longitudinal Study of Children – the infant cohort 91
3.2.1 Study design 91
3.2.2 Sampling frame 92
3.2.3 Sampling design 92
3.2.4 Panel design 93
3.2.5 Data collection 94
3.2.6 Sample size and response rates 102
3.2.7 Quality control during and after interviews 108
3.2.8 Ethical considerations and data protection 109

3.3 Methodology for secondary analysis of the Growing Up in Ireland infant cohort 111
3.3.1 Data retrieval 111
3.3.2 Outcome variable – body mass index 112
3.3.3 Explanatory variables 117
3.3.4 Data cleaning of explanatory variables and exploratory analysis 143
3.3.5 Epidemiological analysis and inferential statistics 145

3.4 Summary 145

CHAPTER 4: PREVALENCE OF OVERWEIGHT AND OBESITY IN YOUNG CHILDREN – AN ANALYSIS OF CHILD GROWTH CRITERIA AND DATA FROM THE GROWING UP IN IRELAND INFANT COHORT 148

Key points in this chapter 149

4.1 Introduction 150
4.1.1 Measurement of weight status in young children 150

4.1.2 Prevalence and trends of childhood overweight and obesity 153

4.2 Chapter aims and hypothesis 164

4.3 Methods 165
4.3.1 Literature search 165
4.3.2 Statistical analysis 165

4.4 Results 166
4.4.1 Definition of overweight and obesity based on various child growth criteria 166
4.4.2 Critical analysis 174
4.4.3 Prevalence of overweight and obesity in the Growing Up in Ireland infant cohort 184

4.5 Discussion 200
4.5.1 National and international child growth criteria 200
4.5.2 Need for comparable criteria and terminology 202
4.5.3 Prevalence of overweight and obesity in the Growing Up in Ireland infant cohort 204
4.5.4 Sex difference in the overweight and obesity prevalence 207
4.5.5 Conclusion 208

CHAPTER 5: CROSS-SECTIONAL ANALYSIS OF EARLY OVERWEIGHT AND OBESITY – INCLUSION OF THE SOCIAL ECOLOGICAL MODEL 210

Key points in this chapter 211

5.1 Introduction 212

5.2 Chapter aims and hypotheses 214

5.3 Methods 215
5.3.1 Variables 215
5.3.2 Statistical analysis 216

5.4 Results 220
5.4.1 Overview of variables and exploratory analysis 220
5.4.2 Distribution of OWOB based on population characteristics 232
5.4.3 Cross-sectional models 241

5.5 Summary 255

CHAPTER 6: INTEGRATION OF MULTI-DIMENSIONAL OBESITY RISK FACTORS INTO A MULTILEVEL MODEL OF CHILD BODY MASS INDEX 258

Key points in this chapter 259

6.1 Introduction 260

6.2 Chapter aims and hypothesis 262

6.3 Methods 263
6.3.1 Variables 263

6.3.2 Analysis 264
6.3.3 Sensitivity analysis 269

6.4 Results 270
6.4.1 Baseline model 270
6.4.2 Inclusion of sex 271
6.4.3 Inclusion of socio-economic status variables and ethnicity 272
6.4.4 Inclusion of birth and infancy factors, parental characteristics, neighbourhood variables and early lifestyle 276
6.4.5 Model 5 – Inclusion of child's lifestyle variables at three and five years of age 283
6.4.6 Cumulative changes in body mass index 292

6.5 Summary 297

CHAPTER 7: DISCUSSION 300

7.1 Introduction 301

7.2 Summary and discussion of findings 303
7.2.1 Prevalence of overweight and obesity in young children in Ireland 303
7.2.2 The obesogenic environment of young children in Ireland 307
7.2.3 The extended conceptual model 323

7.3 Possible actions needed to lower childhood overweight and obesity in Ireland 325
7.3.1 Policy recommendations 326
7.3.2 Recommendations for research 339

7.4 Strengths and limitations of the research 345
7.4.1 Strengths 345
7.4.2 Limitations 347
7.4.3 Alternative analysis options 350

7.5 Conclusion and verification of hypotheses 352

REFERENCES AND APPENDICES 356

References 357

Appendices 386
Appendix 1: Classification of body mass index in adult populations 386
Appendix 2: Details of the variable cleaning: Body mass index 387
Appendix 3: Additional details of cleaning the explanatory variables 389
Appendix 4: Differences between the NCD Risk Factor Collaboration study and the Global Burden of Disease study 396
Appendix 5: List of Irish data included in the NCD Risk Factor Collaboration study and the Global Burden of Disease study 397
Appendix 6: Distribution of body mass index according to World Health Organization percentiles 399

List of Figures

Figure 1: Projections of the joint overweight and obesity prevalence in adults and young children in Ireland **22**

Figure 2: Expanded version of the Davison and Birch ecological model of predictors of childhood overweight and obesity by Reed et al. (8) **41**

Figure 3: Complex web of potential determinants of overweight and obesity in children by Monasta et al. (1), colour-coded according to the levels of the Social Ecological Model **45**

Figure 4: Commission on Social Determinants of Health conceptual framework **77**

Figure 5: Extended Complex web of potential determinants of overweight and obesity in children by Monasta et al. (1), including colour-coding according to the levels of the Social Ecological Model and findings from the literature review of this thesis **84**

Figure 6: Sample size, response rates and attrition between waves in the Growing Up in Ireland infant cohort **103**

Figure 7: Estimates of the joint overweight and obesity prevalence in Ireland **155**

Figure 8: Estimates of the obesity prevalence in Ireland **155**

Figure 9: Overweight and obesity prevalence in the National Preschool Nutrition Survey **162**

Figure 10: Obesity prevalence in the National Preschool Nutrition Survey **162**

Figure 11: Published overweight and obesity prevalence in the Growing Up in Ireland infant cohort at nine months and three years of age **163**

Figure 12: Published overweight and obesity prevalence in the Growing Up in Ireland infant cohort at three and five years of age **163**

Figure 13: Correlation of body mass index (BMI) at the ages of nine months and three years **185**

Figure 14: Correlation of body mass index (BMI) at the ages of three and five years **186**

Figure 15: Correlation of body mass index (BMI) at the ages of nine months and five years **186**

Figure 16: Distribution of body mass index (BMI) in all children **193**

Figure 17: Distribution of body mass index (BMI) in children breastfed six months or longer only **194**

Figure 18: Weight status using the World Health Organization (WHO) growth standard classification **196**

Figure 19: Weight status using the World Health Organization (WHO) growth standard and growth reference **196**

Figure 20: Weight status using the International Obesity Task Force growth reference **197**

Figure 21: Prevalence of overweight (excluding obesity) across ages, based on equivalised household income quintiles **234**

Figure 22: Prevalence of obesity across ages, based on equivalised household income quintiles **234**

Figure 23: Prevalence of overweight (excluding obesity) across ages, based on children's birth-weight-for-gestational-age **237**

Figure 24: Prevalence of obesity across ages, based on children's birth-weight-for-gestational-age **237**

Figure 25: Prevalence of overweight (excluding obesity) across ages, based on household structure **240**

Figure 26: Prevalence of obesity across ages, based on household structure **240**

Figure 27: Body mass index of children with a high or low cumulative biological, psychosocial or material risk of overweight and obesity **293**

Figure 28: Verified extended Complex web of potential determinants of overweight and obesity in children, based on Monasta et al. (1), Reed et al. (8) and findings from this thesis **324**

List of Tables

Table 1: Caregiver characteristics that changed across waves, due to changes in the person who was the primary caregiver (PCG) or secondary caregiver (SCG) between waves **98**

Table 2: Questionnaires used in the Growing Up in Ireland infant cohort study **100**

Table 3: Reasons for non-response between waves in the Growing Up in Ireland infant cohort **105**

Table 4: Inclusion of risk factors of childhood overweight and obesity and associated population characteristics in this thesis **118**

Table 5: Conversion scale used in this thesis for food consumption labels assessed in the Primary Caregiver Main Questionnaire in wave 3 **121**

Table 6: Conversion scale used in this thesis for unstructured physical activity labels assessed in the Primary Caregiver Main Questionnaire in wave 3 **124**

Table 7: Distribution of ethnicity among secondary caregivers (SCGs) by the ethnicity of primary caregivers (PCGs) **137**

Table 8: Global prevalence of weight status in children aged zero to five years **154**

Table 9: Studies and surveys that collected weight and length/height data from children and adolescents in Ireland **158**

Table 10: Prevalence of overweight and obesity in Irish children since 1990 **161**

Table 11: Overview of studies used in developing child growth criteria applicable to overweight and obesity (OWOB) research **169**

Table 12: Means, standard deviation (SD) and ranges of weight, height and body mass index (BMI) in the Growing Up in Ireland infant cohort, after variable cleaning **184**

Table 13: Time trends in prevalence of weight categories in the Growing Up in Ireland infant cohort **188**

Table 14: Comparison of prevalence estimates of weight categories in the Growing Up in Ireland infant cohort **190**

Table 15: Sex-differences between prevalence rates of weight categories in the Growing Up in Ireland infant cohort **198**

Table 16: Thresholds of the World Health Organization (WHO) child growth criteria **203**

Table 17: Descriptive statistics of explanatory variables **222**

Table 18: Prevalence of body mass index (BMI) status across ages, based on equivalised household income quintiles **233**

Table 19: Prevalence of body mass index (BMI) status across ages, based on children's birth-weight-for-gestational-age **236**

Table 20: Prevalence of body mass index (BMI) status across ages, based on household structure **239**

Table 21: Logistic regression model showing odds ratios (OR) of overweight and obesity prevalence at nine months, three years and five years of age **246**

Table 22: Logistic regression model showing odds ratios (OR) of obesity prevalence at nine months, three years and five years of age **250**

Table 23: Multilevel Model 1, showing Body Mass Index (BMI) trajectories between nine months, three years and five years of age unadjusted **271**

Table 24: Multilevel Model 2, showing Body Mass Index (BMI) trajectories between nine months, three years and five years of age adjusted for sex **272**

Table 25: Multilevel Model 3, adjusted for sex, socio-economic status variables and ethnicity, showing Body Mass Index (BMI) trajectories between nine months, three years and five years of age **274**

Table 26: Multilevel Model 4, showing Body Mass Index (BMI) trajectories between nine months, three years and five years of age adjusted for demographics, birth and infancy factors, parental characteristics, neighbourhood variables and early lifestyle **280**

Table 27: Multilevel Model 5, showing Body Mass Index (BMI) trajectories between three years and five years of age adjusted for BMI at nine months, demographics, birth and infancy factors, parental characteristics, neighbourhood variables and early and later lifestyle **288**

Table 28: Summary of relationships between various overweight and obesity (OWOB) risk factors and 1) OWOB, 2) obesity and 3) body mass index (BMI) **295**

Table 29: Suggested protocol for reporting of childhood obesity research **341**

Table 30: Classification of body mass index in adult populations **386**

Table 31: Missing weight and length/height before and after the first step of data cleaning **387**

Table 32: Changes in weight and length/height that took place between waves **388**

Table 33: Descriptive statistics of body mass index before and after data cleaning **388**

Table 34: Full list and grouping of food items measured in wave 3 (at five years) **389**

Table 35: Proportion of five-year-old children meeting the food pyramid guidelines **390**

Table 36: Conversion scale used in this thesis to generate composite food scores based on the food consumption labels assessed in wave 3 **391**

Table 37: Distribution of the unstructured physical activity composite score **392**

Table 38: Proportion of children with short, normal and long sleep duration **392**

Table 39: Distribution of birth weight in gram after data cleaning **393**

Table 40: Distribution of main type of childcare used in each wave **394**

Table 41: Removal of observations from explanatory variables **394**

Table 42: Characteristics of the NCD Risk Factor Collaboration study and the Global Burden of Disease study **396**

Table 43: Data sources from Ireland included in recent estimates of overweight and obesity trends **397**

Table 44: Changes in the distribution of body mass index (BMI) in all children **399**

Table 45: Changes in the distribution of body mass index (BMI) in children breastfed six months or longer only **400**

List of abbreviations

AGA	Appropriate-for-gestational-age
AMF	Anonymised Microdata File
BMI	Body mass index
BWFGA	Birth-weight-for-gestational-age
CDC	Centers for Disease Control
CI	Confidence Interval
COSI	Childhood Obesity Surveillance Initiative
EU	European Union
GDM	Gestational diabetes mellitus
GP	General Practitioner
GUI	Growing Up in Ireland
GWG	Gestational weight gain
ICC	Intraclass correlation coefficient
IDEFICS	Identification and prevention of Dietary- and lifestyle-induced health EFfects In Children and infantS
IOTF	International Obesity Task Force
LGA	Large-for-gestational-age
MECC	Making Every Contact Count
NCHS	National Centre for Health Statistics
OWOB	Overweight and Obesity

PCG	Primary caregiver
PDL	Physical activity, diet and lifestyle (score)
RMF	Researcher Microdata File
SCG	Secondary caregiver
SD	Standard deviation
SEM	Social Ecological Model
SES	Socio-economic status
SGA	Small-for-gestational-age
STROBE	Strengthening the Reporting of Observational Studies
UK	United Kingdom
US	United States of America
WHO	World Health Organization

Summary

Background: Childhood overweight and obesity (OWOB) is associated with adult obesity (10) and ill-health (11); however the prevalence among young children in Ireland is unknown. For prevention programmes to target those children most in need, risk factors of OWOB and critical time points when these factors occur need to be identified. This thesis investigated in-depth the OWOB prevalence and risk factors among young children in Ireland.

Methods: A literature review was conducted to identify a suitable child growth criterion for young children in Ireland. Prevalence of OWOB was assessed through secondary analysis of the Growing Up in Ireland National Longitudinal Study of Children. Families in the nationally representative infant cohort were interviewed when the children were nine months (n=11,134), three years (n=9,793) and five years old (n=9,001). At all ages, standardised weight and length/height among other variables were collected from the children and their caregivers. Associations of explanatory variables with children's OWOB were analysed using binary logistic regression, and with longitudinal changes in body mass index (BMI) using multilevel regression models.

Results: Using the World Health Organization growth standard and growth reference to define OWOB (using the 1-z-score threshold), 40.4% of the nine-month-old children had OWOB, 45.7% of the three-year-olds and 31.5% of five-year-olds. At all ages, high birth-weight-for-gestational-age, late birth and rapid infant weight gain were strongly related to OWOB and elevated BMI. At specific ages, caregiver OWOB and education, household income, family structure, ethnic background, geographic region, screen use, sleep duration, antibiotic use and childcare were also associated with OWOB and longitudinal changes in BMI.

Discussion: Biological, material and psychosocial factors contribute to the high prevalence of early childhood OWOB seen in Ireland and risk factors should be targeted simultaneously to lower OWOB levels. Prevention should start as early as possible and be maintained throughout the early childhood.

Publications and presentations arising from this thesis

Peer-reviewed paper:

Jabakhanji SB, Boland F, Ward M, Biesma R. Body mass index changes in early childhood. Journal of Pediatrics. 2018;202:106-14.

Conference presentations:

A mixed-methods approach to examine risk factors for early childhood obesity. **Oral presentation** at the PhD Symposium of the Children's Research Network in Maynooth on 6th September 2016

Jabakhanji, SB, Boland, F, Ward, M, Biesma, R (2017). *Who grows most in Ireland? An analysis of weight status changes in early childhood*. **Poster presentation** on the European Congress of Obesity in Porto, Portugal on 17th-20th May 2017

Other:

Jabakhanji SB. Prevention and intervention of childhood obesity needed before school entry – longitudinal findings from Ireland. **Blog-post** on the SPHeRE blog, available from: www.sphereprogramme.ie/prevention-and-intervention-of-childhood-obesity-needed-before-school-entry-longitudinal-findings-from-ireland/ , 11th December 2018

Acknowledgements

I want to thank my supervisors *Dr. Regien Biesma*, *Dr. Fiona Boland* and *Dr. Mark Ward* for their invaluable guidance, patience and support, and for their unique contributions each of them brought to the team that helped me accomplish this thesis. Thank you for your time and the important lessons you taught me.

I would also like to thank the *Irish Research Council*, which funded this research under the Postgraduate Scholarship 2015.

I would like to acknowledge the work of the *Growing Up in Ireland study team*, in particular the *Economic & Social Research Institute* and *Trinity College Dublin*, and *all families and children* who kindly offered their time to participate in the Growing Up in Ireland study and therefore contributed to this thesis.

Furthermore, I would like to thank the Committee of the *Association for the Study of Obesity on the island of Ireland* for their inspiration and encouragement.

Many thanks also go to RCSI and everyone in the *School of Postgraduate Studies,* the *Division of Population Health Sciences,* and *Room 15 ½,* who welcomed me so kindly when I first arrived to Ireland and helped me at different occasions during my time in RCSI. In particular, I would like to acknowledge *Prof. Ronán Conroy* and *Dr. Finbarr Leacy* for their help with statistical queries. Acknowledgements also go to *Prof. Ronán Conroy* for beginner's lessons in understanding Irish culture.

On a personal note, I would like to thank my family and friends for their encouragement and endless belief in my abilities:

Mama, Papa, Karim und Faride, danke für eure offenen Arme und Ohren, die verrückten, gemeinsamen Stunden und ein zu Hause, in das ich immer wieder gerne zurückkehre. Ohne euch wäre ich sicherlich nicht so weit gekommen.

Liebe Omi, du warst mir stets ein großes Vorbild und begleitest mich im Herzen auf all meinen Reisen ï so auch auf dieser.

Jana, you have been one of my most loyal companions in the past few years and were one of my biggest sources of support along this PhD journey. I cannot thank you enough for your inspiration, thoughtfulness, generosity and patience, all the big and small favours along the way and your company in Myanmar.

Ronan, I would not have started this PhD without you and I would hardly have been able to finish it on my own. I am endlessly thankful for your loyalty, for making me laugh every day and for the many words of wisdom and support that helped me accomplish this. Your endless curiosity and hunger for new knowledge are truly inspirational and you do make me believe that anything is possible.

Many thanks also to *Laura* for all her support, generosity and invaluable friendship.

Norah, I could not have thought of a better two-person-PhD-support-group than ours. You helped me battle through this and I have no doubt that you will do an excellent job in reaching completion in no time! *Kate* (and *Hannah*), too were such good company, thanks for all the chats and walks and lunches! Many thanks also to the rest of the *SPHeRE 2015 cohort*, and in particular to *Marita*.

Ben, thank you for the many dinners and putting so much effort into making me feel at home in Dublin. To all *McArdles* including little *Clara*: It was so lovely to get to know you and I am deeply impressed by your eye-opening fight to equal women's rights. You really are making a difference!

Finally, I would like to thank *Mary*, *Kitty* and *Jan* for grounding me through their unique views on life which they kindly shared with me on a regular basis.

CHAPTER 1:

Introduction to overweight and obesity in the early childhood years

1.1 Overview of childhood obesity

Childhood overweight and obesity (OWOB) is a severe health problem in high-income countries, due to its high prevalence (12) and as it has been linked to significant increases in chronic diseases and associated mortality worldwide if carried into adulthood (11, 13, 14). Jointly, the co-morbidities of OWOB lead to a lowered life expectancy among overweight and obese individuals, and worldwide, more deaths occur due to OWOB than from the consequences of underweight and under-nutrition (13, 14). The latter remain a severe challenge for public health particularly in developing countries, where a double-burden of underweight and overweight is increasingly impacting the health of children (12). Globally, a minimum of 2.8 million (in 2016) (12) and up to 4 million individuals are estimated to have died from OWOB in 2015 (15). This marks an increase of up to 28% in the past quarter-century (15) and makes OWOB the fifth leading cause of death worldwide (14).

Nearly 14% of children and adolescents aged two to 19 years are estimated to have OWOB globally (15) and in some countries, age-standardised mean BMI in children aged five years and older is already exceeding that of adults (12). Levels have consistently been high in various high-income English-speaking countries for more than four decades (12) and many European countries, including Ireland (16), reported that more than 20% of five- to 17-year-olds were overweight or obese (17).

While few studies take the youngest age group into consideration, children at the ages of two to four years around the world were found to have higher OWOB levels than older children and adolescents in a recent study, suggesting an upward trend in OWOB prevalence if these younger children maintain their weight status (15). It is worrying to see that not only past trends of rising OWOB levels were seen in both children and adults, but that also future predictions suggest an increase in childhood obesity globally (18). This includes the youngest age group in Ireland in which increases are expected until 2030 (7), as shown in Figure 1.

Notably, a rise in childhood obesity is likely to contribute to increasing adult obesity levels and associated morbidity (10, 19, 20). Worldwide, adult obesity levels have

risen six-fold since 1975 and increases in prevalence were seen in almost all countries over the past decades (12, 15), with at least 1.9 billion adults (a prevalence of 39%) found to be overweight or obese from 2014 to 2016 (12, 13, 15, 21). The prevalence in Europe lies above the global average (22) and in Ireland, 39% of the population aged 15 years or older were overweight and an additional 23% obese in 2017 (23). This marks a dramatic increase in the last four decades (24) as can be seen in Figure 1. Also in adults, further increases are expected (9, 25) (Figure 1), possibly making Ireland the European country with the highest burden of OWOB by 2030 with levels of just under 90% (9).

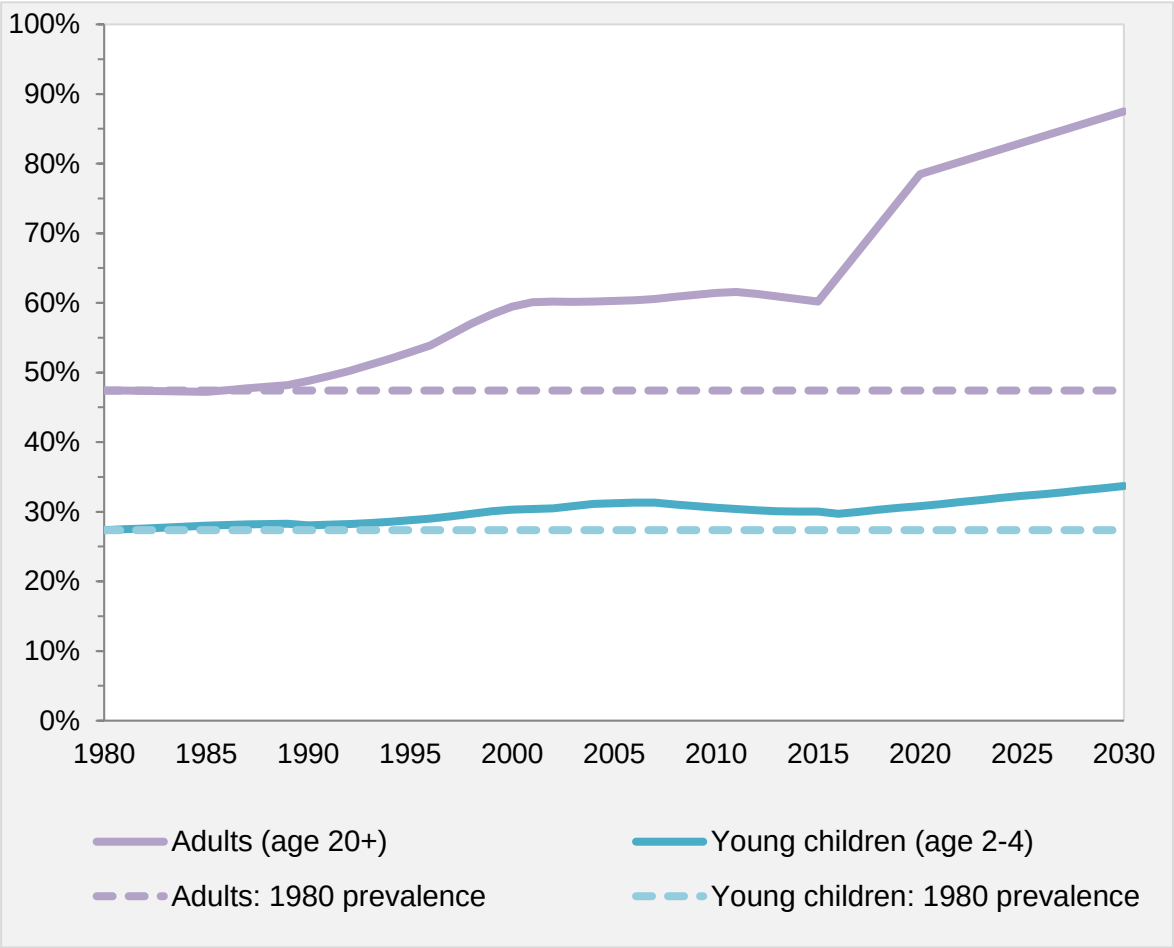

Figure 1: Projections of the joint overweight and obesity prevalence in adults and young children in Ireland. Data of young children and of adults until 2015 as well as projections in young children were retrieved from the Global Burden of Disease Study 2015 (GBD 2015) (5, 7). Projections of adults were retrieved from Keaver et al. (9)

Evidence is accumulating that pregnancy, infancy and preschool age are a critical period for the development of obesity (20, 26-29) and associated cardio-metabolic risk (30, 31), possibly explaining the higher incidence of OWOB that was seen in this age group compared to that in later childhood (32). Accordingly, prevention programmes that address obesity in children before the age of six years were found most cost-effective compared to obesity prevention at later ages (33, 34). At this early stage, many children can still be protected from becoming overweight and from moving from the overweight into the obese category, which is why effective prevention efforts are needed as early on in the lives of children as possible.

Policy-makers in Ireland put a strong emphasis on early intervention in order to gradually lower the number of Irish children with OWOB (35). While steady increases were seen in the body mass index (BMI) of children in Ireland from the late 1940s until 2005 (36, 37) estimates of obesity levels retrieved from recent studies are inconsistent; however the prevalence is likely to be high (38-41). For example, approximately one in five boys and one in four girls aged five years or older were found overweight or obese in 2014 (16). Particularly in children until the age of five, however, more research was demanded in a recent study as the evidence in young children in Ireland appears weak (16).

Accordingly, this thesis aimed to investigate childhood obesity and its main determinants from the age of zero to five years in Ireland, in order to fill this research gap. Section 1.3 of this chapter will describe the research needs in the area of early childhood obesity in Ireland more in detail, based on which aims and objectives for this thesis were delineated (Section 1.4). In advance of this and in order to provide a context for the topic of childhood obesity, the consequences of childhood obesity for the health and development of children and for the Irish health system will be presented in the following section.

1.2 Consequences and co-morbidities of childhood overweight and obesity

Childhood obesity has been linked to health risks and co-morbidities similar to those found in adults (11, 42), meaning that the early onset of OWOB increases the risk that adverse effects are experienced at a young age and put children into a disadvantaged position in their core period of development and growth, compared to children who maintain a healthy weight throughout childhood. These consequences may have irreversible and long-lasting impacts on children's (later) quality of life as well as on their health and well-being throughout the life-course (43, 44), as will be outlined in the following paragraphs.

1.2.1 Presence of overweight and obesity later in life

Child BMI has been found to strongly predict obesity in adult life, particularly when obesity tracks from childhood into adolescence. Up to two thirds of obese children remain obese in adult life (10, 19, 20) and the highest BMI scores and metabolic risk were seen in obese Irish adults who had been overweight before the age of five years (45). Notably, already during the infancy period, adiposity increases the risk of OWOB presence in the later childhood and adolescent years, which highlights the importance of children's early weight-related development for their lifetime risk of obesity (46). Obese parents can pass on the susceptibility to obesity (47, 48) and to associated co-morbidities to their offspring (49), which could create a viscous circle of passing on the risk of obesity from one generation to the next if early excess weight gains are not prevented.

Notably, if adults were overweight or obese during childhood or adolescence, their associated risk of adult mortality was found to be between 40% and 60% higher than among adults who had maintained a normal weight throughout these years (44). Furthermore, a higher risk of diabetes mellitus (type 2 diabetes), various cancers (43), hypertension (44) and coronary heart disease was seen among those adults who had had an elevated BMI in childhood (10, 44). These risks may partly be reversed if children move out of the OWOB categories before adulthood

(50); however as this is unlikely once children are obese, their health risks increase further when they grow into overweight or obese adults.

High BMI in adulthood is related to numerous diseases and health consequences, such as type 2 diabetes, hypertension, stroke, coronary artery disease, congestive heart failure (11, 51), asthma, pulmonary embolism and at least 13 different cancer types (11, 15, 52). Additionally, common consequences of OWOB are elevated rates of renal disorders, psychosomatic co-morbidities, digestive disorders, chronic back pain, osteoarthritis, gallbladder disease as well as chronic pain, psychosocial disparities and an overall reduced quality of life (11, 53, 54). The World Health Organization (WHO) classifies obesity as the fifth leading cause of death worldwide (14), predominantly due to chronic diseases (55), and in Ireland, it was the fifth leading cause of both mortality and disability in 2016 (56). Notably, more than one third of all deaths and morbidity related to a high BMI were seen in overweight individuals (which is the most prevalent group with excess weight worldwide) (15) while the risk of ill-health (45) and premature death appear to increase with increasing BMI and fat mass (57). This raises a need to target the prevention of overweight and of obesity jointly.

1.2.2 Early onset of ill-health

While many diseases related to OWOB only manifest themselves in adult lives, OWOB in children is also related to a poorer health-related quality of life (42, 58) and the earlier children start storing excess body fat, the earlier they were found to develop diseases or markers related to adult disease (42, 59). Two reviews of OWOB across the childhood and adolescence years found strong evidence for an increased risk of cardio-metabolic ill-health, particularly in the highest BMI ranges (42, 58). The findings from a European cohort study furthermore indicate that rapid increases in BMI in the ages from nine months to five years were strongly linked to metabolic risk in later childhood (30). Additionally, it has been shown that OWOB paired with physical inactivity in three- to ten-year-old children was highly linked to insulin resistance, which is a central component of the metabolic syndrome (60). The metabolic syndrome has individually been linked to childhood obesity in a

systematic review by Friend et al. (61). As a consequence, the risk of developing type 2 diabetes early in life is high among overweight and obese children (60).

While for a long time cardiovascular ill-health has been associated with middle-aged and elderly men in particular, studies now are pointing to an increasing prevalence among young adults and children (42) that may be explained in an increase of inflammatory processes among obese children (62). A European cohort study found that among two- to ten-year-old children, OWOB was associated with hypertension (63). Another systematic review identified an increased risk of arterial stiffness among five- to 24-year-olds who were obese, compared to their lean peers (59). This confirms that cardiovascular risk factors among obese individuals establish early in life, possibly leading to earlier onset of chronic disease and increasing the overall health risk (59). Some studies of obese children furthermore identified elevated levels of non-fatty liver disease, being another example of severe long-lasting health consequences (58).

An elevated occurrence of some autoimmune diseases also was observed among obese children. A high number of studies point to increased levels of asthma and asthma symptoms among obese children (42, 64). The development of OWOB had previously been considered as a consequence of asthma or as developing in parallel due to mutual causes, whereas one systematic review found strong evidence that obesity in children preceded asthma onset in adolescence (65). The results of a meta-analysis furthermore showed that children who had been classified as obese at various ages from one to twelve years were twice as likely to develop subsequent type 1 diabetes as children without obesity (66).

Orthopaedic symptoms and diseases may also result from early OWOB, such as flat foot in children, a degeneration of the disc found in adolescents and young adults, back pain, and the risk for atherosclerosis. This has important implications for the treatment and life-long progression of OWOB, as chronic symptoms experienced through these conditions may reduce the physical fitness of children (42) and accordingly the pursuit of physical activity throughout their lives (67).

The daily functioning of children with an elevated BMI may furthermore be impaired by higher levels of sleep apnoea and other sleep problems (42). A systematic

review showed that obese children and adolescents who suffered from obstructive sleep apnoea had significantly better sleep outcomes after weight loss, whereas conventional treatment (adenotonsillectomy) was less effective in improving this sleep condition in obese compared to normal-weight children (68).

In addition to physical ill-health, various studies indicate that obese children and adolescents tend to suffer from psychological disorders and symptoms of mental ill-health in different ways. Pulgaron (42) and Sanders et al. (58) reviewed the evidence from different countries and found a high risk for anxiety and depressive symptoms among children and adolescents with OWOB. Furthermore, obese children and adolescents were found at risk of different internalising and externalising behaviours and disorders, including a higher risk of Attention Deficit Hyperactivity Disorder, cigarette smoking, alcohol consumption and eating disorders, as well as increased levels of sadness, loneliness and nervousness, reduced social skills and a lack in self-esteem (42, 58, 69). In parts, these may arise due to the very common discrimination and stigmatisation of overweight or obese children based on their physical appearance (70). The mental health of overweight and obese children may therefore already be at risk at an early stage in life which is central to building relationships and developing social skills and self-esteem. As a result, the development of obesity may lead children to suffer from multi-faceted adverse consequences both for their mental and physical health, at a time that is most crucial for their later health and well-being.

1.2.3 Higher risk of multiple morbidity

Children in the highest weight categories and BMI ranges tend to suffer from more than one weight-related health impairment simultaneously. A systematic review found moderate evidence for higher rates of negative hospital outcomes among obese compared to non-obese children (71). This includes mortality of critically ill patients, after organ or stem cell transplant, and among cancer patients. Beyond that, obese children may catch infections more easily and they may need to stay hospitalised longer (71). A high level of ill-health in children may negatively affect

their weight-related lifestyles, for example the symptoms experienced from various diseases may act as a barrier to children for taking up physical exercise (67).

1.2.4 Early developmental disadvantages compared to lean peers.

Finally, overweight and obese children appear to develop more slowly in many ways than their lean peers, putting them at a disadvantage in school and sports which may continue on into their adult lives (67). A systematic review found that neurocognitive functioning overall was poorer among obese children than in non-obese. This included reduced motor skills, poorer executive functioning, lower levels of attention and poorer visuospatial performance among the obese children (67). A smaller study supports this finding (72).

1.2.5 Cost of childhood obesity

In addition to the co-morbidities and developmental disadvantages associated with OWOB that pose a threat to the individual health and well-being, the economic consequences of OWOB also put a burden on individuals, as well as on the healthcare system and society as a whole (16). The Organisation for Economic Co-operation and Development estimates that 3.4 million potentially productive life years were lost in the European Union (EU) due to chronic disease in 2013, including 24,000 in Ireland (73). The associated economic loss in the EU is estimated at €115 billion (73) and health systems struggle to meet the associated increased need for treatment, making the reduction of chronic disease one of the EU's main health targets (73). As mentioned above, the prevention of obesity has an important role in achieving this (55, 74, 75).

In Ireland, an estimate of between €1.3 million and €1.7 million in healthcare expenditures for children is attributable to childhood OWOB annually (16). This is in addition to healthcare expenditures for adult obesity which are much higher as most co-morbidities only develop in the adult ages. Adult healthcare spending in Ireland attributable to OWOB was projected to reach €5.4 billion in a twenty-year

period if OWOB levels increase to 85-89% as projected by Keaver et al. (9) by 2030. Combining childhood, adolescence and adult expenditures, the estimated average lifetime cost of childhood OWOB in Ireland based on 2015 expenditures exceeds €16,000 per child (16). Of these costs, 21% are related to regular health assessment and treatment. The remaining 79% are indirect costs not covered by the healthcare sector, but by employers, individuals and society (16). Societal costs related to productivity loss, such as due to absenteeism and premature death, as well as personal income losses are included in this amount. Furthermore, the estimate of €16,000 most likely underestimates the real lifetime cost significantly, as costs related to consequences of childhood OWOB on individual mental health and psychosocial well-being as well as on school performance and resulting career opportunities and outcomes were not accounted for in the study (16).

1.3 Research gaps

1.3.1 Monitoring of childhood obesity

The WHO identified obesity and related health problems as a major priority for prevention and intervention to tackle non-communicable diseases in the *Global Action Plan for the Prevention and Control of Noncommunicable Diseases* (74). Furthermore, it published specific aims and actions for Europe in the *European Food and Nutrition Action Plan 2015-2020*, which include the objectives of monitoring weight status on national level as well as pursuing the use and knowledge translation of existing anthropometric data (55). In Ireland, OWOB has been made a major public health priority under the *A Healthy Weight for Ireland: Obesity Policy and Action Plan 2016-2025*, with a specific focus on children and families. One of the actions promoted in this plan is the surveillance of OWOB in order to inform interventions and policy-making in the future (35).

Body mass index (BMI), which is a ratio of body weight (kg) over body height squared (m^2), in infancy and at the age of five years (76) as well as weight changes between these ages were found to predict BMI and weight gains later in childhood and adolescence (77, 78). As the persistence of OWOB from a young age into the later childhood, adolescent and adult years is high (10, 19, 20), population monitoring for young children is most crucial to control the *ó o b e s i t y e p i d e mi c ô* among children, adolescents and, ultimately, adults. Through early monitoring, population trends can be detected at an age where prevention and intervention are most effective and have the highest potential to prevent negative consequences for children's health and wider development (33, 34).

A recent study conducted by the WHO European Regional Office found that insufficient surveillance data are available for European countries in this youngest age group (up to the age of five years) (79), similar to findings from the United States (US) (32). A barrier to the study of OWOB prevalence in young children is the limited number of entry points to sample children and collect population-representative data (79). In addition, the WHO European Regional Office identified a need for the clarification and transparency of tools used to define OWOB among children up to the age of five years. No consensus on a standard definition for early

OWOB exists and various national and international child growth criteria are used by researchers across countries (79) and in Ireland (16, 38). No Irish child growth criteria are currently available (80); therefore it is not surprising that studies of the prevalence of OWOB in young children in Ireland, like in many other European countries, are limited (16, 79). An account of recent OWOB prevalence in young children is therefore needed that enables the identification of OWOB trends in Ireland over time, as well as comparisons with other countries. In order to facilitate this, consensus is needed as to which growth criterion should be used in infancy and subsequent childhood years. A more detailed background of existing growth criteria and a history of childhood obesity studies in Ireland are presented in Chapter 4 of this thesis.

1.3.2 Inequalities in obesity prevalence and predictors of childhood obesity

In addition to the overall obesity prevalence, childhood obesity has been outlined as a major public health issue in and across Europe due to significant disparities observed between different social groups that need to be addressed (35, 55). Recent findings show that the gaps in obesity prevalence between socio-economic status (SES) (81, 82) and between ethnic groups have widened among children (83). A report based on recurrent international health surveys estimated that socio-economic differences were responsible for 27% of adolescent obesity in European countries in 2014 (84), indicating that early socio-economic inequalities persist into later ages.

In Ireland, socio-economic differences were observed that impact the individual risk of obesity, and need to be addressed in the light of rising obesity numbers (35, 85) that are expected in Ireland (7, 9). While the *Obesity Policy and Action Plan* sets out to reduce socio-economic differences of childhood obesity in Ireland by 2025, baseline data assessed in the report and used for subsequent evaluations only include children from the age of six years and over (35), and socio-economic differences in OWOB levels in young children were not investigated. This is surprising as the first 1,000 days of children’s lives are of increasing concern and

led Irish policy-makers to make this period one of their core priorities in addressing OWOB in Ireland (35). More specifically, a healthy start with appropriate guidance on obesity prevention in the first 1,000 days is regarded as crucial to the life-course approach which Ireland is aiming to follow in order to reverse rising obesity trends. The life-course approach sets out that good health should be promoted continuously throughout life by addressing exposure to risk factors that impact individual health at different life stages and hence contribute to overall individual and population health (35). It furthermore stipulates that these exposures may impact the health of succeeding generations through social transmission (86). Consequently, the exposure of some young children to obesity risk factors is likely to contribute to inequalities in OWOB outcomes across their lifespan and may additionally lead to inequalities in succeeding generations, which may explain the observed increasing gaps in prevalence between population groups.

Accordingly, health indicators related to early childhood obesity need to be identified and defined so that they can provide entry points for sustainable prevention and intervention strategies on a policy-level in Ireland (35). From 2004 until 2014, the Irish healthcare system, including child health(care) services, was largely reorganised and underwent reforms that may pave the way for such strategies if established in an effective and timely manner (87). For example, subsequent to these reforms, primary care was made freely available to children under the age of six years in Ireland to make access to care more equitable. In general, however, the Irish health system is constrained by a long history of stagnation, as well as declining budgets (in particular since the recent economic recession in 2008) that co-exist with high childhood obesity levels and health inequalities (87). Therefore, strategies to promote healthy weight gains in children should build on available evidence, in order to use the limited resources effectively and prevent future costs to the Irish healthcare system.

Simultaneously, stressors found in the lives of young families in Ireland should be given consideration (88), in order to effectively tailor these strategies to the needs of families and increase the impact of healthy weight promotion in Ireland. The Social Ecological Model (SEM) provides a good framework for studying risk factors of childhood obesity as it includes numerous factors from different areas of

children's lives; that is their individual level of risk factors, the family environment they live in and the community environment (89). The *Complex web of potential determinants of overweight and obesity in children* by Monasta et al. (1) and the results of an additional comprehensive literature review furthermore added to the SEM based on more recent research findings. The extended framework was used to guide this thesis, as will be described in depth in Chapter 2. On the background of this framework, risk factors of early childhood obesity and population characteristics associated with inequalities in the obesity risk were identified that emerged in other studies and therefore appear relevant to the study of early childhood obesity in Ireland. For example, Chapter 2 presents prenatal factors and weight-related behaviours of children and their families that may put young children at an increased risk of OWOB. The theories underlying this thesis are that these risk factors and population characteristics lead to inequalities in early OWOB that occur through biological, material and psychosocial pathways, as will be explained in the context of the literature throughout Chapter 2 (86). Accordingly, a number of research hypotheses were formulated and presented at the end of Chapter 2.

1.3.3 Role of cohort studies in studying childhood obesity

Several countries have explored early childhood OWOB in birth cohort studies, such as Finland, Denmark, Norway, the United Kingdom (UK), the Netherlands, New Zealand and the US, in addition to a number of international cohorts (90, 91). However, this has not been examined in Ireland recently, where studies were only conducted in older children (92-96), regionally (40, 97) and in a small cross-sectional survey (n=371) (98).

Additionally, the Growing Up in Ireland (GUI) National Longitudinal Study of Children was initiated in Ireland and follows a nationally representative cohort of infants born in 2007 (99). The weights and lengths/heights of children in the GUI infant cohort were first assessed in 2008, when they were nine months old. Measurements were furthermore taken from the same sample of children when they were three years and five years old. Additionally, a large set of variables was collected from the cohort (99) which were identified as risk factors of early OWOB

in previous research (which will be presented in Chapter 2) and can be tested in relation to OWOB outcomes of young children in Ireland. The *HSE Framework for Action on Obesity 2008-2012* mentioned the linkage to the GUI infant cohort as one of their research priorities for the surveillance of childhood obesity (100). While childhood obesity was investigated in sub-sets of the GUI infant cohort data previously (discussed in Chapter 4), to date, no comprehensive study of the cohort has assessed OWOB prevalence and longitudinal associations of obesity risk factors across all ages of the infant cohort.

With three sequential measurements of weight and length/height now available for most children in the GUI infant cohort, the data offer a unique opportunity for longitudinal obesity research in young children in Ireland. As will be discussed more in detail in Chapters 2 and 3, a large proportion of studies that investigated OWOB in different countries are based on cross-sectional study designs. This raises a need for more longitudinal research that enables the identification of critical time points in young children's lives and allows for stronger conclusions than findings from cross-sectional studies. Thus, while cross-sectional studies of early childhood obesity are valuable to enable easy comparisons with past studies, longitudinal research is needed in the Irish context to inform health professionals, researchers and policy-makers about OWOB levels and associated risk factors among (young) children in Ireland. These findings could be effectively translated into both targeted and population-wide strategies in order to help prevent future OWOB on a national level in Ireland.

As the age group of up to five years has been identified as understudied both internationally and in the Irish context, the longitudinal data structure of the GUI infant cohort will be exploited in this thesis to investigate the BMI and weight status of children longitudinally.

1.4 Aims and objectives of this thesis

Main aim of this thesis

The overarching aim of this thesis is to investigate OWOB levels among infants and young children in Ireland and to identify how risk and protective factors of childhood obesity contribute to overweight, obesity as well as to different BMI trajectories in these children, in order to derive recommendations for future prevention and policy-making that aim at controlling childhood obesity and related consequences for population health in Ireland.

Based on the research gaps identified in Section 1.3, the following research objectives were specified for this thesis:

1. To investigate the prevalence of overweight and of obesity in a nationally representative cohort of children in Ireland at nine months, three years and five years of age, and to explore the impacts of the inconsistent use of child growth criteria used in studies of childhood obesity. This included the critical analysis of existing child growth criteria in order to identify criteria suitable for the study of young children in Ireland.

2. To identify differences in early childhood OWOB that exist between population groups in Ireland, as well as based on a number of risk factors observed on the *individual*, *family*, and *community, demographic and societal* level at the ages of nine months, three years and five years. This included the investigation of material, biological and psychosocial factors that may cause the development of OWOB in early childhood.

3. To identify critical time points at which factors on the *individual*, *family* and *community, demographic and societal* level appear to have the most important influence on weight developments within young children and potentially lead to inequalities in the risk of childhood OWOB in Ireland. This required investigating children's weight outcomes longitudinally.

1.5 Overview of chapters

In the following chapter, the research aims presented above will be refined. Five research hypotheses will be outlined in Chapter 2 (Section 2.5), based on the results of a comprehensive literature review that builds the background of this thesis (Sections 2.3 and 2.4). The SEM was used as a framework to guide this thesis, as will be explained in Chapter 2 (Section 2.1).

The methods used to investigate the hypotheses of this thesis are described in detail in Chapter 3 and the findings presented in separate Chapters 4, 5 and 6.

Chapter 4 reports on the prevalence of OWOB in children from the GUI infant cohort, and it includes a critical analysis of different child growth criteria that are available to study OWOB in young children. Hypothesis 1 that the prevalence of OWOB among young children in Ireland is high is explored in Chapter 4.

Chapter 5 corresponds to the second aim and shows the results of investigating risk factors and inequalities in relation to OWOB in the GUI infant cohort. Factors on various levels of the SEM and whether material, biological and psychosocial mechanisms may lead to inequalities in children's individual obesity risk are explored (Hypotheses 2, 3 and 4).

Chapter 6 shows the results of an advanced longitudinal analysis of BMI using the GUI infant cohort data that aims at the identification of critical time points early in children's lives in relation to risk factors and population characteristics, as specified in the third aim (Hypothesis 5).

Finally, Chapter 7 of this thesis discusses the findings from Chapters 4, 5 and 6 in relation to the children's mutual context of growing up as young children in Ireland at the height of the worldwide *obesity epidemic*. The hypotheses tested throughout this study will be verified or rejected, based on which a number of conclusions are drawn that help clarify the current situation of early childhood OWOB in Ireland. Findings from previous chapters are used to map out recommendations for policy-makers and for research. This includes the identification of strategies that might help prevent future increases in early childhood OWOB.

CHAPTER 2:

Early childhood overweight and obesity in the literature

2.1 Introduction and objectives of this chapter

To provide a context for the topic of childhood obesity, this chapter will present a comprehensive picture of the evidence that has been published in this field to date. Specifically, the growth of children from the gestational period through to their age of five years will be described (Section 2.3), in order to determine whether a child is growing healthily or whether their growth is characterised by excess weight gains. OWOB in the earliest periods of child development is related to a number of risk factors which will be discussed in the course of Section 2.3.

Following from this, inequalities that were observed in childhood obesity levels in and outside Ireland between various socio-economic and demographic groups will be described (Section 2.4). Throughout the chapter, theoretical underpinnings will be discussed that may explain the observed relationships between a number of factors and early childhood obesity. These theories will be summarised in Section 2.4.5.

Finally, findings and gaps identified in this background chapter will be discussed (Section 2.5.1), subsequent to which the research hypotheses (Section 2.5.2) will be presented.

2.2 Conceptual models of childhood obesity

The *Social Ecological Model* (SEM) and the *Complex web of potential determinants of overweight and obesity in children* will jointly be used as a framework to explore existing risk factors of early childhood obesity. The background and details of this framework will be described here.

2.2.1 The Social Ecological Model: Origin and use in childhood obesity

The conceptual framework of this thesis is based on Bronfenbrenner's ecological system theory, which evolved between the 1960s and 2005 and which led to the establishment of the SEM (101-103). According to Bronfenbrenner, the physical and psychosocial child development depends on the context or the environment the child grows up in; hence the ecological system theory has also been referred to as 'development in context' or 'human ecology theory'. The environment of children as well as interactions between the children and different environmental components are unique to each child and therefore the complexity of these factors needs to be studied together, rather than investigating each environmental component on an individual basis. The original model by Bronfenbrenner grouped environmental factors that influence child development into four dimensions, namely the microsystem, mesosystem, exosystem and macrosystem (101), whereas the number and naming of these dimensions varies in SEMs developed by other researchers since then (8, 89, 104) (e.g. the model shown in Figure 2). As shown below in Figure 2, the dimensions are represented by circles around the child, with the microsystem having the most direct influence on children, and less direct influence existing in subsequent outer circles.

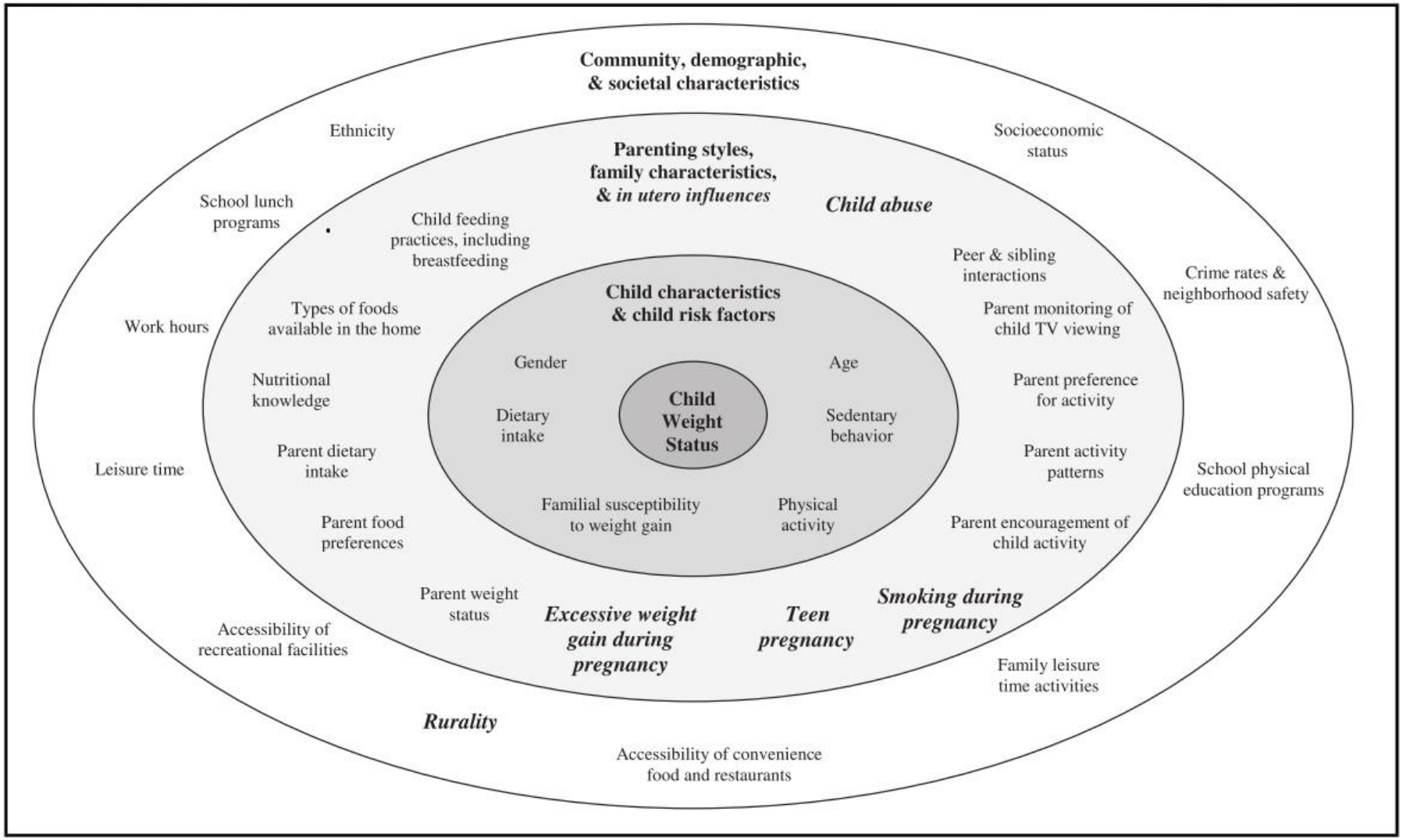

Figure 2: Expanded version of the Davison and Birch ecological model of predictors of childhood overweight and obesity by Reed et al. (8)

Later in his career, Bronfenbrenner added a fifth dimension, *time*, to the original model which he described as ‘chronosystem’ (102). According to Bronfenbrenner’s theory, relationships between child development and different systems constantly change over time. For example, the community gains an increasing influence over time as children start going to school. Also family dynamics may change over time, for instance parental divorce may influence child development most during a narrow time window prior to and after divorce, but have a weakened influence as time passes by. Additionally, the historical context influences these relationships, as policies and social norms change. For example, in past generations, few mothers pursued full-time employment and accordingly influenced the development of children differently from today (103). The SEM has been applied and extended in previous studies investigating complex child health outcomes, including studies of childhood obesity (89).

Use of the SEM in studies investigating childhood obesity, diet or the physical activity of children has been popular in the past and the interplay of factors on various levels of the SEM jointly explained weight-related behaviours and intervention outcomes in different studies (105-107). Moreover, the SEM has been applied to obesity research in older children in Ireland previously (108) and appears as a useful framework for studying childhood OWOB in the Irish family context.

Different interpretations of the ecological systems theory led to the development of a number of SEMs with a focus on childhood obesity that all follow the same principles, but vary in details. In 2001, Davison and Birch (89) developed the *Ecological model of predictors of childhood overweight*, which depicts dietary intake, physical activity and sedentary behaviour as risk factors of OWOB on the individual child level, and the child’s age, gender and familial susceptibility to weight gain as moderating variables that influence children’s risk factors and subsequent weight gain through the interaction with factors on more distant levels. The more distant levels include family variables and parenting practices on the middle level, and community, demographic and societal characteristics on the outer level (89). Ten years later, Reed et al. (8) added prenatal and other risk factors to this model, based on the findings from their research, offering the more

comprehensive *Expanded version of the Davison and Birch ecological model of predictors of childhood overweight* shown in Figure 2 (p. 41).

A number of other studies applied or developed similar models to investigate OWOB in children from an ecological perspective (109-112), for example Harrison et al. (104) developed the *Six-Cs* model that includes the six levels of cell, child, clan, community, country and culture. While the model is very relevant to obesity studies, factors captured under ‘country’ and ‘culture’ are understood to be relatively constant among young children born and growing up in Ireland. Children in Ireland are exposed to a mutual context of national policies, media coverage and norms that may be related to childhood obesity through their influence on food intake, physical activity and sedentary behaviours. Consequently, the in-depth study of the factors captured under ‘country’ and ‘cell’ as possible predictors of childhood obesity does not appear important for the identification of risk factors within this population. Therefore, the model proposed by Reed et al. (8) (Figure 2 on p. 41) was used as a framework for guidance in this thesis and will be referred to as ‘the SEM’ throughout the course of this study.

2.2.2 Extension of the Social Ecological Model based on evidence from the past 20 years

The SEM by Reed et al. (8), which will be used in this study, is particularly useful to the study of obesity in very young children and infants. The SEM displays various levels of influences that are related to child weight outcomes, with the understanding that the inner level (individual level) has the biggest influence on child weight, and decreasing strength of influence the more distant levels are from the child. However, a few limitations of this SEM need to be acknowledged that have implications for this study. First, the SEM does not specify the weight with which each factor or level contributes to the individual obesity risk of children, nor does the SEM explain pathways through which factors influence the obesity risk (beyond the broad idea that outer levels influence levels which are located closer to the centre of the SEM). Secondly, the field of obesity research has advanced significantly since the original model was developed and modified for the study of

childhood obesity by Davison and Birch (89). Thirdly, the model by Reed et al. (8) is not specific to young children and includes factors related to OWOB outcomes in the later childhood years. Therefore, in addition to this theory-driven framework to the study of obesity, evidence-based findings need to be incorporated in the design of novel studies in order to study inequalities in childhood obesity in a comprehensive way. While Reed et al. (8) have added to the original model incorporating the evidence retrieved through their research, more components may need to be added based on studies with alternative designs and focuses.

Particularly in a widely-studied field such as childhood obesity, systematic reviews and meta-analyses are useful tools to provide aggregate knowledge from studies that were independently assessed for their quality and are hence likely to provide valid and reliable evidence. Monasta et al. (1) have combined findings from numerous systematic reviews on early childhood obesity risk factors in their umbrella review. The authors identified and evaluated 22 systematic reviews that investigated OWOB in infants and young children from conception until the age of five years. Except one study from 1999, all systematic reviews were published between 2004 and 2008 (1) and can add to the original SEM of Davison and Birch (89). Based on the findings of the included systematic reviews, Monasta et al. (1) developed the *Complex web of potential determinants of overweight and obesity in children*, a model that explains how risk factors are related to childhood obesity, and how some factors may additionally act as mediators for other risk factors (1). As a result, this model addressed the limitations of the SEM.

In order for this study to investigate childhood obesity in a comprehensive theory- and evidence-driven way, the SEM by Reed et al. (8) and the *Complex web of potential determinants of overweight and obesity in children* by Monasta et al. (1) were merged (Figure 3). Both models include a variety of risk factors and population characteristics associated with childhood obesity in other studies, with some overlap between them. However, Monasta et al. (1) do not distinguish between factors that contribute to the obesogenic environment of children into various levels. Thus, risk factors displayed in the *Complex web of potential determinants of overweight and obesity in children* were colour-coded according to the levels of the SEM suggested by Reed et al. (8), as depicted in Figure 3.

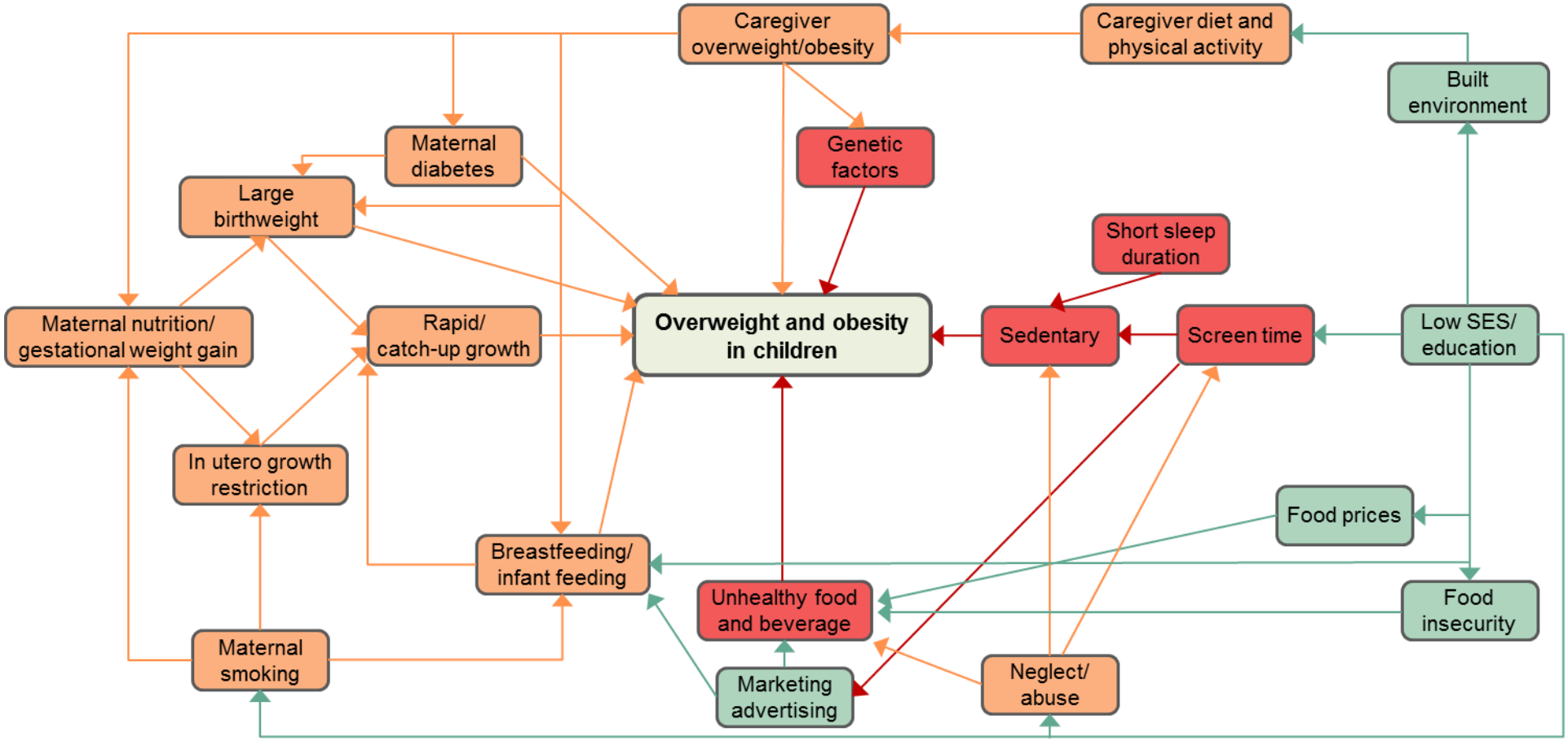

Figure 3: Complex web of potential determinants of overweight and obesity in children by Monasta et al. (1), (2), colour-coded according to the levels of the Social Ecological Model

Note: red colour = individual level; yellow colour = parenting style and family characteristics; blue colour = community, demographic and societal characteristics

SES = Socio-economic status

2.2.3 Literature review: Risk factors of childhood obesity

A literature review was conducted in order to identify literature that was published since the conceptualisation of Reed et al. (8) and the completion of the systematic reviews included by Monasta et al. (1), and to include factors that were excluded from the studies underlying both models.

Search strategy

A comprehensive literature review was conducted to identify potential risk and protective factors of (inequalities in) childhood obesity until the age of five years, based on literature that was published since the study of Monasta et al. (1). The main outcomes searched in the literature were childhood overweight, childhood obesity, or the combination of child OWOB, as well as BMI and other adiposity parameters. Additionally, the dietary quality or quantity and physical activity levels in children were included, due to their relevance to OWOB as prime drivers of energy intake and expenditure.

Literature was identified using the database *ó PubMed*. Due to the complex nature of childhood obesity and to identify a high number of potential risk and protective factors, the MeSH terms *ñ pediatric* and *ñ overweight* (which includes obesity) were searched. As childhood obesity is a widely studied topic, the age filters *ñ 0-23 months* and *ñ 5-19 years* were used to exclude literature that was not relevant for these age groups. Additionally, the *ó Cochrane Systematic Reviews of Reviews* was searched for the keywords *ñ child* and *ñ obese* (combined), in order to find important high quality research that may have not been listed on *PubMed*. Grey literature from experts was identified using the *ECOG Obesity eBook*, which consists of topical collections of literature in the field of childhood obesity. As all of these collections are in some way relevant to the topic of this thesis, no predefined search terms were used to filter results. The Boolean operators *ñ AND* and *ñ OR* were used to combine search or MeSH terms.

Titles were screened for relevance and abstracts of relevant articles reviewed to determine whether they fulfilled inclusion criteria. Inclusion criteria for all papers were English, French, German or Dutch language. Papers were included if they had been published between 2008 and 2018. However, due to the high number of studies published in the field, systematic reviews published within the last five years were prioritised. For each potential risk or protective factor, findings from individual observational studies were additionally included in the literature review only if these had been published after the latest systematic review that investigated this same risk or protective factor, or if findings from different studies were contradictory. Furthermore, studies explaining relationships between potential risk or protective factors and weight outcomes were included. Studies acknowledged within a systematic review or meta-analysis, and older studies that had been confirmed by more recent research, were not mentioned explicitly in this review, as the scope of available literature is too extensive to be mentioned in full. Therefore, this review should not be understood to cover all research falling within the inclusion criteria that was published since 2008. It is rather intended to provide a comprehensive up-to-date overview of established and emerging evidence on early OWOB risk factors, which needed to be considered for inclusion in the analysis of this thesis. Accordingly, this review should not be understood as an update to the systematic review of Monasta et al. (1) as different search criteria were used.

Overall, preference was given to systematic reviews, meta-analyses, longitudinal studies and in particular to those studies using large samples, in order to minimise the risk of bias. Individual cross-sectional studies, case-control studies, case series or other studies with small sample sizes were only included if no other information on the given variable was found.

Excluded from this review were intervention studies and studies focusing solely on children above the age of five years, on adolescents or on adults. Where there were similar studies identified combining different age groups, preference was given to those studies that focused on young children only.

Studies that fell within the inclusion criteria were used as a basis for cross-referencing; that means their reference lists were searched for additional literature. This was to identify more research findings, in particular those of relevant cohort

studies and researchers found in the initial searches. A number of specific study names or authors were separately entered into *PubMed* if any indication was found that additional research had been published which appeared relevant to this literature review and had not been identified in the initial searches.

Summary of study characteristics and quality of the evidence

A high proportion of the existing evidence was derived from observational studies, as the study of risk factors in experimental studies, such as randomised controlled trials, is less common. Partially, this rather one-sided study of childhood obesity risk factors can be explained by the fact that OWOB develops over a longer period of time and based on a high number of impact factors. In contrast, experimental studies are usually limited to a short period of time and can hardly rule out the noise of latent obesity risk factors. Moreover, ethical concerns may arise in experimental studies when a defined exposure variable is expected to increase the obesity risk in children, unless this exposure occurs naturally in some children and can be incorporated in quasi-experimental designs.

A significant proportion of the evidence underlying this literature review, including publications detailed in systematic reviews and meta-analyses, is based on cross-sectional studies. Data on obesity risk factors retrieved from these studies covered various countries, risk factors and sample sizes that fell within the inclusion and exclusion criteria of this literature review. Cross-sectional studies can be limited in assessing whether children's elevated BMI preceded or succeeded their exposures to risk factors and do not account for changes over time that may take place both in a child's individual weight status and their exposure to risk factors. In longitudinal analysis, both can be analysed in the same population over time, which offers opportunities to retrieve a fuller picture of what influences children's weight at what ages. A number of longitudinal studies (many birth cohorts) were also included in this literature review.

It should be noted that for some relationships that were observed in the literature review, mechanisms were not exactly clear. Occasionally, clarity only existed that

the OWOB risk was increased or lowered in the absence or presence of a defined risk factor, but how this factor altered the risk of OWOB was not always tested.

Findings of the literature review will be presented throughout Sections 2.3 and 2.4, outlining factors that are related to children's early weight gains and that may contribute to inequalities in OWOB established in the young childhood years, as well as potential pathways that may explain the relationships observed in the literature. Following from here, an examination of the growth of children until they reach the age of five years will be undertaken, in order to define OWOB and healthy growth. Biological and psychosocial factors related to early OWOB in the literature review will be outlined throughout this section (Section 2.3).

2.3 Early child growth and childhood obesity

The easiest way to determine an estimate of weight status is by calculating the individual BMI, which is based on measured height and weight (113):

BMI = (weight in kilograms) / (height in metres) ^{2}

BMI has been found to correlate with total body fat (113) and health risks (10) and is useful to assess the prevalence of childhood OWOB on population level across countries (114-116). The generic BMI cut-off values for adults (Appendix 1) are not applicable to children, who are supposed to have lower BMIs up to the age of five years due to their different physique. Children go through periods of growth throughout their development that imply changes in the relation of weight to length or height that occur naturally along with changes in age and that vary slightly between the sexes; thus weight, length/height, age and sex need to be examined in relation to each other (116). The following paragraphs will describe the growth of children throughout the foetal, infantile, toddlerhood and early childhood phase, based on which healthy and unhealthy weight ranges can be distinguished and the presence and prevalence of OWOB in young children be assessed. As this thesis investigates differences in (relative) weight, but not height gains, only a synapsis of length/height developments will be provided while the main focus is on weight.

2.3.1. Prenatal development

From conception until birth, children experience the fastest period of growth, gaining about 30% of their adult height (117). To assess whether adequate foetal growth took place, the length and weight of children at birth are measured in hospitals following standardised procedures and particularly birth weight is used as an early indicator of nutritional status (118). Low birth weight is commonly defined as 2500g or below and macrosomia, which is the medical term for high birth weight, is usually defined as equal or above 4000g, or alternatively as above 4500g (119); however different classifications exist (120). As children's birth weight and length is expected to increase with longer gestation (117), the assessment of

weight in relation to gestational age at birth can be used in addition to absolute birth weight to identify heavy or light children among those born pre-term or late (118). Additionally, while term infants are born with about 13% of body fat (121), much lower levels are stored in preterm infants at birth (117). The classification of new-born infants as small-for-gestational-age (SGA), appropriate-for-gestational-age (AGA) and large-for-gestational-age (LGA) thus provides a more accurate reflection of intrauterine growth (122) and foetal growth restrictions than absolute birth weight categories (123).

In addition to gestational age, variations in weight and length at birth depend on the uterine environment and nutrition, which in turn is influenced by the maternal diet and by maternal size (117). Birth records suggest that the risk for children to be born LGA is elevated if their mother had been overweight or obese prior pregnancy and is highest if she gained excessive weight during pregnancy (124). This has been confirmed by studies using absolute measurement of birth weight (124-126). Furthermore, term children born to mothers with high gestational weight gain (GWG) were found to have an increased proportion of fat to lean mass at birth (119). Maternal OWOB prior to becoming pregnant was additionally found to increase the risk of preterm birth (119, 126), whereas underweight women are at an increased risk to give birth to a child with small birth-weight-for-gestational-age (BWFGA) (125). Evidence is accumulating that an interaction may exist between GWG and pre-gestational weight, hence separate GWG recommendations exist for women of differing pre-gestational weight categories (119, 127).

Maternal diseases also were related to foetal weight gain. Pre-eclampsia, a condition characterised by high blood pressure during pregnancy, was related to lower BWFGA in the offspring of affected women (128). Additionally, a longitudinal study found that the ratio of fat and lean mass, adjusted for gestational age, was higher in foetuses of mothers with gestational diabetes mellitus (GDM) than of non-diabetic mothers, and that, foetuses of mothers with GDM were generally growing faster (129). GDM is a temporal insulin-resistance that occurs only during pregnancy, usually in the second half of pregnancy (130)[1]. Accordingly, women

[1] Insulin is a hormone produced by the pancreas and needed to break down glucose (sugar). Hyperglycaemia describes high rates of glucose levels in the blood, which occurs if not enough insulin is produced to lower blood glucose levels effectively (130).

suffering from GDM are at a higher risk to give birth to LGA children (124) or children with high birth weight (119). This relationship is understood to be based on a higher level of blood glucose, fat and amino acids transferred to the child through the blood chord, or higher production of anabolic hormones and other growth-promoting factors (119). A large Swedish birth cohort study found a positive association between pre-pregnancy BMI and risk of GDM, showing that severely obese pregnant women had the highest risk to suffer from GDM. Particularly, those obese pregnant women who had themselves been born SGA or LGA had an additionally elevated risk of GDM, whereas no association with their own birth weight and GDM could be shown in normal weight women (131). Additionally, the risk of GDM is higher in women with high GWG (119), showing how these factors can jointly contribute to high birth weights.

Nevertheless, maternal gestational disease should not be seen as a static precondition to elevated foetal weight gain as the treatment of GDM through a controlled diet or insulin therapy has the potential for lowering this risk (119). This highlights the importance of gestational behaviours in the prevention of childhood obesity, in addition to biological factors. Similar conclusions can be drawn from associations seen between gestational smoking and birth weight. Specifically, children of smoking mothers were found to suffer from nutrient deficiencies in the womb and after birth, which increased their risk of lower birth weight and BWFGA in a number of studies (128, 132, 133). As nutrient deficiencies and in utero exposures as well are held responsible for the subsequent development of early child OWOB, these relationships will be explored in the following sections.

Overweight and obesity susceptibility in the prenatal phase

All of the above factors that contribute to variations in weight and length at birth, as well as birth weight itself, were found to be associated with early OWOB in previous studies.

The earliest individual factor that may determine how children grow and that is already present in the unborn child is the individual genetic makeup (47, 134). A

large proportion of the human genome has not been fully understood and the functions of many genes (135), such as in relation to childhood obesity, are unknown (120, 136, 137). No sole gene that can be held responsible for rising obesity numbers has been identified (138), however, estimates indicate that genetics may be liable for between 40% to 70% of differences related to the individual obesity risk (136, 139). Mechanisms behind genes known to impact the obesity risk (120, 140-143) are not fully known, but most likely these genes act in congruence with environmental factors and other genes (143, 144). Genetic mechanisms are possibly linked to differences in the expenditure of energy, appetite and satiety regulation (135), food preference, intake and uptake, as well as growth differences among carriers of obesity-promoting gene variants (141, 142). Epigenetic mechanisms[2] are additionally likely to explain relationships, for instance the function of a gene may be temporarily or permanently disabled or weakened subsequent to exposure to early life factors (140, 142, 146).

Strong evidence has been found that a high birth weight is associated with an increased risk of childhood obesity at different ages (27, 120). Nonetheless, a systematic review by Ino (147) concluded that low birth weight is an independent risk factor of later childhood obesity. In addition to absolute birth weight, children born with a large BWFGA were found to weigh more during early childhood (148), primary school age (149) and as adolescents (150).

Maternal weight status at the onset of pregnancy has consistently been found to put children at a higher risk of OWOB (120, 151). In one meta-analysis, children's risk to develop OWOB was found to increase three-fold in the offspring of obese women (152) independent of other known risk factors of childhood obesity. In addition to pre-gestational measurements, maternal weight status measured later during the early childhood years was strongly associated with childhood obesity in numerous studies (153). Additionally, while less research has focused on the influence of paternal weight status on offspring obesity, Patro et al. (154) conclude that pre-pregnancy BMI by the mother and by the father are equally important in

[2] 'Epigenetics' is an overall term describing changes in the expression of genes which cannot be explained by changes in the DNA sequence. The predisposition to such changes, the 'phenotype', is heritable, but its activation can be triggered by events and conditions in the external environment (145).

the subsequent development of OWOB of their child, whereas a weaker contribution of paternal than of maternal weight status to children's OWOB was found in another study (155).

In addition to parental weight status, a high maternal GWG has been identified as an independent risk factor of OWOB in offspring (27, 120, 156), both during infancy and throughout all child and adolescence ages (48, 119). Children born to women with high GWG furthermore tend to have an early onset of OWOB and maintain their OWOB during childhood (119), even though GWG may have less influence on children's weight status than the maternal pre-gestational BMI according to one study (151). Additionally, first evidence is emerging suggesting diverging strengths of GWG effects on child weight per trimester of pregnancy (120), with a more severe association seen with GWG in the first and second trimester (48, 119). This suggests a direct relationship between GWG and childhood obesity which may be explained by an increased intrauterine transmission of fatty acids or glucose to the foetus (48). Moreover, a relationship appears to exist between GWG, maternal pre-gestational OWOB and childhood obesity that may in parts be mediated by maternal insulin resistance and blood glucose levels during pregnancy (119). Furthermore, it is likely that an intergenerational transmission of the OWOB risk from parents to their children takes place that can be explained by their shared genes (47, 86). Other studies additionally consider that a relationships between maternal weight status, GWG and offspring obesity may be either of genetic nature or lifestyle-related (48), or a combination of those with epigenetic mechanisms and a shared environment (157, 158).

The cumulative findings from the numerous studies presented above indicate that a number of risk factors appear to occur before or during gestation that may explain both differences in birth outcomes (birth weight and gestational age) as well as in the subsequent development of OWOB. Several of these risk factors appear to be interrelated and can potentially be explained by various biological mechanisms. While parents do not have full control over these risk factors and mechanisms, it should be noted that these mechanisms partly appear to be influenced through (pre-)gestational behaviours and healthcare and may therefore be modifiable if targeted early.

2.3.2 Infancy

Infants experience high weight, length and BMI gains; however growth is slower than in the prenatal period and does not evolve in a linear way. Shortly after birth, infantile growth increases steeply, before it slows down around the age of between 18 to 24 months (117, 121). Until 18 months of age, children gain about 15% of their adult height. Mainly the rate of growth depends on the nutrition and health status of children, as well as their thyroid function. Furthermore, length gains vary based on genetic predisposition (117).

Immediately after birth, children's weight tends to decline by 5 to 8%, due to the influences of a changing environment which new-born children need to adjust to, such as changes in mechanisms of nutrient and water uptake. After 14 days, infants are supposed to have grown back to their birth weight and should gain between 14g and 28g of weight daily until the 3rd month. By five months of age, birth weight should be doubled, by twelve months of age it should be tripled and by 24 months nearly quadrupled. Simultaneously, the proportion of body fat reaches its peak of 20-25% at around twelve months of age and falls slowly thereafter (121). Throughout infancy, boys tend to be both heavier and taller than girls (121).

In children born preterm or with a small BWFGA, catch-up growth towards mean weight is common within the infantile phase and similarly, children born with a large BWFGA may experience catch-down growth (113, 117). Growth patterns throughout infancy can additionally vary depending on whether children were breastfed or infant formula-fed (113). As BMI in the first two years of life can predispose infants to later OWOB (120), both infant weight gain and infant feeding practices will be discussed more in detail now.

Rapid infant weight gain

Strong evidence suggests that children with catch-up growth (159) or rapid weight gain (independent of birth outcomes) in the first year (27), or first two years of life, are more likely to have OWOB later in childhood (120, 160). For example, one

longitudinal study including nearly 45,000 children concluded that rapid weight gain in the first six months of life was associated with the highest prevalence of OWOB five and ten years later (160). Catch-up growth specifically has been related to the development of ‘catch-up fat’, which may be stored in the body due to changes in the regulation of adipose cells by insulin. It is possible that this event early in life programs cells to store glucose in the future more than cells of individuals who never experienced a shortage of nutrients or other reasons why catch-up growth or rapid weight gain in general may take place (161).

Importantly, inadequate weight gain or weight loss during infancy (referred to as ‘faltering growth’ or ‘weight faltering’) over a sustained period of time is associated with impairments of the physical development of children (117). Accordingly, parents regard weight gain in children as positive and tend not to be concerned about the risk of obesity at that age. As a result, little awareness exists for weight gain that may be too rapid (113). Rapid infant weight gain is common in children born SGA (159) or early (162). As maternal pre-pregnancy BMI was found to be indicative of pre-term births (119, 126), it also puts children at a greater risk of early rapid weight gain (162) and subsequent OWOB (120). Furthermore, children born to mothers with high GWG were found to have a higher risk of rapid growth in the first year of life (163). Additionally, rapid infant weight gain has been related to feeding practices (162).

Infant feeding

The rate of weight gain in infancy was found to depend on feeding practices (162, 164, 165), and associations with childhood obesity throughout the first five years of life were found on two dimensions: type and composition of foods fed to the infants, and timing and duration of feeding these foods to children before the child commences to follow the family diet.

With regards to the type of foods, evidence exists that children who were breastfed by their mother are protected from OWOB later in life, compared to children who received formula as a breastmilk substitute (27, 147). Importantly, this appears to

hold true even if other risk factors of childhood obesity are present, such as the exposure to GDM (166). Formula feeding, in turn, has been associated with rapid weight gain during infancy and in early childhood (162). Furthermore, there is limited evidence suggesting that exclusive breastfeeding is required to achieve a beneficial effect of breastfeeding on later child weight, as opposed to a combination of breastfeeding and bottle-feeding or breastfeeding and the feeding of solid foods (167). Exclusive breastfeeding implies that no foods or liquids other than breastmilk are given to the infant (168). Also the individual macronutrient composition of breastmilk may play a role in children's weight gain. In one study, caloric density and fat content of breastmilk were inversely associated with obesity risk and BMI in twelve-months-old infants, and particularly with the rate of weight gain between the ages of three to twelve months. In turn, higher BMIs were seen if the breastmilk contained more carbohydrates or protein (169).

An analysis of hormonal breastmilk components furthermore revealed potential explanations for the benefits seen in breastfed children. In contrast to infant formula, different hormones are present in breastmilk that appear to have a role in children's appetite regulation and metabolic processes (27). These hormones include grehlin (170) and leptin (171), which have a function in the stimulation of appetite, as well as obestatin, which is suspected to be involved in the regulation of appetite (120). A breastmilk component directly linked to growth from early life until adulthood is the insulin-like growth factor 1, and further associations have been suggested with peptin YY (27). Other breastmilk components include adiponectin and resistin, both of which are related to insulin sensitivity (120).

In addition to hormonal mechanisms of breastfeeding, breastfed infants may be better able to develop a sense of satiety-responsiveness as they determine amount and timing of food intake and as parents are less likely to intervene with this (172). However, it is speculated that the protective effect of breastfeeding seen in many studies may be mainly due to confounding factors (173), an example being beneficial effects of breastfeeding on the prevention of child diseases, which may themselves play a role in weight gain (167).

Furthermore, the timing and duration of feeding practices appeared as a risk factor for childhood obesity in the literature. A number of researchers disagree that any

form and length of exposure to breastfeeding is beneficial for children's weight gain and weight status in adult life (120, 167, 174). Their findings indicate that breastfeeding is only beneficial if pursued for a minimum period of four or six months, and that a prolongation to up to nine months, eleven months or 24 months would further increase benefits (27, 174). Also the WHO recommends a minimum duration of six months of exclusive breastfeeding for healthy child development and suggests that longer breastfeeding possibly maximises health benefits (175).

Related to the duration of breastfeeding or alternatively formula feeding is the timing of weaning, when solid food starts being given to a child in order to transition from infant feeding to the family diet. The timing of weaning has been discussed as a critical aspect that was related to later child weight in a meta-analysis (176); however numerous other studies concluded that this relationship has not been fully clarified (27, 120, 174, 177). Despite the conflicting evidence base, most studies conclude that solid food should not be given to an infant before the age of four months, whereas other studies set the minimum ideal age to be reached at three months, 15 weeks or 20 weeks (177).

In summary, various indicators on the level of family and parenting practices were related to childhood obesity in past research and strong links were particularly found in relation to factors occurring before and during pregnancy which were discussed in relation to birth outcomes. These factors may alter the early OWOB risk at least partly through mechanisms of rapid infant weight gain. Additionally, infant feeding practices appear relevant to the individual OWOB risk of young children, stressing the importance of biological pathways in the obesity prevention. Direct assessment of individual hormonal processes and the transmission of nutrients in utero discussed in this chapter is invasive and hence not feasible on population-level; however birth outcomes, infantile growth and infant feeding can be used as proxies to study these mechanisms.

2.3.3 Child growth after infancy

After the infantile period, height gains continue to decelerate until about the age of ten years, when a final spurt and peak height velocity is reached during puberty.

Height gains after infancy are determined by a combination of health and well-being, presence of growth hormones, thyroid function and genetics (117).

From the age of around two years, the weight gain of children is supposed to slow down to about 2kg per year. Weight gains become relatively smaller than height gains, leading to slow constant decreases in BMI score and in the proportion of fat to fat-free mass until the adiposity rebound is reached (121). The adiposity rebound marks the change from declining to inclining BMI which is a transition seen in the growth of all healthy children at what point their BMI is at its minimum. Traditionally, this takes place between the ages of five and seven years, although it has already been observed in younger children. Children with an early (before the age of five years or 61 months) or very early adiposity rebound (by 3.5 years or 43 months) appear to have an elevated risk of OWOB (178). Notably, higher occurrence of early adiposity rebound was found among children with at least one obese parent, indicating that a relationship between the two risk factors may exist (178). In contrast, breastfeeding was related to a later adiposity rebound which may mediate relationships seen between OWOB and infant feeding (173).

In the transition from infancy through to preschool-age, children start acquiring lifestyles that contribute to their individual OWOB risk and hence constitute the core components of the individual level of the SEM. In its simplest definition, weight gain equals energy expenditure subtracted from energy intake (13). The two major factors that influence weight gain according to this equation are the dietary in- and uptake of children, as well as their level of physical activity. These factors will therefore be explored more in detail below.

Contribution of children's lifestyle factors to the individual risk of overweight and obesity

Diet

Pearce and Langley-Evans (179) found that a high energy intake during the period of complementary feeding is associated with later OWOB. Nonetheless, in a time where OWOB levels were increasing among young children in many countries, a

simultaneous trend of decreasing energy intake was observed, based on which explanations for excess weight gain as simple as that by Pearce and Langley-Evans (179) might be questioned (173). To elaborate on this relationship more, the consumption of fat and sugar, which are generally associated with high energy intake, thus need to be discussed as potential obesity risk factors.

Even though the relationship between dietary fat intake and early obesity is unclear (173) it is possible that, against expectations, a diet high in fat may protect young children from OWOB (180), as was also implied by the association seen with breastmilk composition (169). Limited evidence suggests that reduced fat intake in early childhood may promote leptin resistance, a biological mechanism that possibly increases the OWOB risk. While many dietary patterns throughout the early child ages tend to be persistent (180), a high fat consumption at the age of ten months was not found to track into later childhood, at which point high fat consumption may in fact increase the OWOB risk. Accordingly, restrictions on children's early dietary fat intake do not appear necessary and a gradual decrease of fat intake is only recommended from the age of three years onwards (173).

Similarly, the relationship between carbohydrate intake and OWOB is not clear (120); however one study found that sugar intake in one- to two-year-old children may be related to increases in BMI (181). Furthermore, sugar-sweetened beverages (SSB) and overall sugar consumption have been related to increased weight, BMI, waist circumference (181) and an increased risk of OWOB in young children (27). Notably, two- to nine-year-old children have been found to commonly exceed levels of recommended sugar intake, particularly during weekend days (182). Another cause for concern is the quality of commercially available infant food in the UK; this was investigated in 2013 and was found to be too high in sugar content and energy density for children's needs (183).

Findings from a number of studies on protein intake point to a possible relationship between a high-protein diet during the first two years of life and subsequent OWOB (120, 179, 184), whereas the evidence after this age is limited (185). 'High' protein intake in young children (at one year of age) is defined as 15-20% of their total energy intakes retrieved through proteins (186) and may potentially increase the OWOB risk through the promotion of an early adiposity rebound (173). An

Australian cohort study investigated protein consumption in children at their ages of nine months, 18 months and five years and concluded that children's protein intake was two to three times higher than recommended and mainly retrieved through the consumption of animal products (186). Limited evidence suggests that early animal protein intake may have a stronger impact on later OWOB levels than the consumption of vegetable protein, possibly through children's endocrine responses to animal protein (185). Insufficient evidence exists to manifest a potential relationship between dairy intake in infancy and OWOB later in childhood (179). Rolland-Cachera et al. (173) postulate that a trend towards the use of low-fat milk alternatives to common full-fat milk has led to an increase of protein intake and a reduction in fat intake among infants that co-occurred with the rise in OWOB numbers across Europe in the 1960 to 1990s. The difference in low and full fat dairy products may explain why no clear relationship between dairy consumption and childhood obesity was seen (179).

Despite the overall understanding that an excess in energy intake, relative to energy expenditure, leads to elevated weight gain, studies provide insufficient evidence to derive clear recommendations in terms of what children particularly in the period of complementary feeding should or should not eat to avoid OWOB (27). A reason for the limited evidence on a potential association between individual food (groups) and childhood obesity could be their joint impact on children's metabolism and weight status. One study identified three main clusters of eating patterns among European children: one was characterised by a high consumption of fruit and vegetables, whole grain products and little consumption of processed food, a second pattern involved an overall balanced diet with only few foods standing out as preferred options, and the last pattern involved typical fast-food characteristics (187). A cluster characterised by a high consumption of vegetables and wholemeal was also identified in another study as having reduced levels of OWOB two years after children had been attributed to this cluster (188).

Potentially, this relationship between food clusters and OWOB does not only exist because of the contribution to the total caloric and macronutrient intake, but also due to influences on the metabolism and wider health of children. Children's low intake of vegetables has been linked to chronic inflammation, as measured by the

C-reactive protein. Chronic inflammation may additionally be promoted through the regular consumption of SSB, mayonnaise and cereals and is possibly lower in children eating fruit spreads or honey. Nonetheless, associations with child weight status in this regard are not clear (189).

Even though research in this area is novel and more research is needed (120), the relationship between metabolism and childhood obesity can be explained by variations in children's gut microbiome and possibly the timing of microbiotal build-up (120, 190-192). Biological mechanisms underlying this relationship have not been fully clarified, but initial evidence points towards changes taking place in hormones that signal satiety (193), increased usage and storage of indigestible carbohydrates as *triglycerides* (lipids) (194), and altered lipid processing of the liver (195). The consumption of sugar (and of fructose in particular), that is typical to the diet in Western countries, may have resulted in a global shift towards a microbiome with altered metabolic capacity (196). Additionally, previous studies found that the gut microbiota differ between infants and young children who were breastfed and those who were not (197, 198). Similarly, the gut microbiota composition differed according to the gestational age at birth, timing of weaning (197), exposure to antibiotics in infancy and parity (198), all of which were outlined as obesity risk factors earlier or will be discussed in the course of this section. Researchers furthermore assume that antibiotic exposures are responsible for a decrease in gut microbiota, which may explain increases in weight or weight status after antibiotic use (195, 199).

Physical activity

The World Health Organization (WHO) (200) recommends that children at the age of five years should engage in a minimum of 60 minutes of moderate and vigorous physical activity every day, pointing to additional benefits for health that can be taken from physical activities exceeding this recommendation. Nevertheless, only a small proportion of children from two to eleven years across Europe were shown to achieve recommended physical activity levels (201).

Increased physical activity levels from four to six years of age are strongly related to lower levels of OWOB later in childhood in a number of studies (202, 203), while no research has been identified that validly studies the relationship between OWOB and physical activity levels in the youngest children (120). In addition to the direct effect of increased energy expenditure on body weight, physical activity appears to lead to decreased food intake immediately after exercise (204). Accordingly, systematic reviews of intervention studies conclude that interventions should ideally combine dietary and physical activity components in order to effectively reduce obesity levels among children and adolescents (205). Physical activity may furthermore activate epigenetic mechanisms that protect children with a particular genetic make-up from OWOB (142). Barriers to physical activity mentioned by mothers of preschool-aged children in qualitative interviews suggest that time constraints, as well as parental safety concerns limit children's access to physical activity (206). Furthermore, maternal stress was associated with lower physical activity levels in children (207).

In order to assess the full scope of children's activity levels, their time spent with sedentary behaviours also needs to be studied, as particularly children with a low physical fitness tend to turn to sedentary activities more (67).

Sedentary behaviour

Evidence has been found for a relationship between television viewing (202), total screen time, or overall sedentary behaviour (208) in early childhood with later OWOB. Two- to five-year-old children are recommended to spend a maximum of one hour watching TV or engaging with other screen-based activities (209).

Theories to explain the mechanisms behind these relationships are numerous. Most obviously, time spent sedentary does not contribute to achieving physical activity recommendations (1). A possible relationship with TV use may be explained by the amount of TV commercials promoting sweets, snacks and fast food, relative to healthy foods. Evidence has shown that a high amount of screen time is related to higher exposure to advertisements, which in turn is related to food preferences and eating behaviour (27). However, studies controlling for exposure

to advertisement have shown that as well beyond the potential influence of advertisement, screen time increases the risk for children to develop OWOB (210). Reasons for this association could be that children are distracted while engaging in screen use and may therefore not correctly perceive and interpret feelings of hunger and satiety. Additionally, they may associate TV viewing with eating, or perceive a greater level of stress and eat more for this reason (210). This notion is supported by a study which found that children engaging much in TV viewing consumed more sugar and fat than other children, even if an objective measure of taste did not identify them as having a preference of these food groups. The conclusion of the researchers pointed to a passive consumption of fat and even more of sugar while children are watching TV (211). A review of studies conducted in two- to six-year-old children additionally revealed that children watching an hour of TV or more on a daily basis consumed less fruit and vegetables in most studies, and additionally their total energy intake was higher compared to children who watched less TV (212). Alternatively, screen use may result in a reduction of sleep (213), which is an additional risk factor of OWOB as discussed in the following section.

Sleeping hours

A lack of sleep has repeatedly been linked to increased body fat percentages (214) and OWOB levels in children (215, 216), particularly in young ages (27). Evidence-based guidelines by the National Sleep Foundation (216) recommend 14 to 17 hours per day in the first three months of life, twelve to 15 hours from age four to eleven months, eleven to 14 hours in one- and two-year-old children, and ten to 13 hours at the ages of three to five years (217). Taveras et al. (218) found that an average infant sleep duration of less than twelve hours per day increased the risk of OWOB at the age of three years. In a meta-analysis, a 9% decrease in the risk of OWOB was found per one hour increase in sleep duration across children of different ages (219).

A number of biological explanations as well as behavioural lifestyle patterns have been suggested to this association. A relationship between sleep and OWOB may

occur due to changes in hormone levels which regulate satiety and appetite, such as leptin and grehlin (219) that were already mentioned in relation to breastmilk previously. Lower levels of leptin and growth hormone (219), as well as increased levels of grehlin and cortisol were previously seen in sleep deprived children and are understood to lead to their weight changes (215, 219), although this could not be verified in one systematic review (220). These hormonal changes potentially alter energy expenditure and metabolism and lead to higher food and caloric intake (215, 219). Additionally, elevated cortisol levels have been found to increase visceral fat storage and lower levels of the thyroid-stimulating hormone found in individuals who sleep little may impact the metabolism and therefore the energy expenditure (215). A number of studies furthermore concluded that shorter sleep duration has negative consequences on glucose tolerance and can even lead to insulin resistance (214, 219, 220).

In addition to these biological mechanisms, a number of behaviours also were related to short sleep and may partly explain the association seen with childhood obesity. Strong evidence was found for an association between short sleep duration and increased sedentary behaviour, as well as unhealthy eating patterns (220). Other studies concluded that the longer children are awake, the more time they are likely to spend eating, snacking or possibly trying to fight their tiredness by increased food intake, while a lack of sleep may also decrease their overall (physical) activity level and thereby their energy expenditure (215, 218). These findings suggest that lifestyle factors should be examined together. Furthermore, they should be studied within the family context as early lifestyles are predominantly shaped by the social environment of young children (221).

The role of the social family environment

Weight-related lifestyles and lifestyle habits of parents tend to correlate with those of young children (221), which, to a large part, can be explained by the parental provision of lifestyle-related resources to children and parental role modelling (222, 223). As such, parental lifestyle is not a risk factor of children’s lifestyle, but rather an alternative measurement of family lifestyle. Family lifestyles are associated with

early childhood obesity and tend to persist from infancy throughout the early child years (224). In the study of early OWOB, it therefore appears sufficient to examine the weight-related lifestyle of either young children or their parents.

A factor that appears to influence the individual OWOB risk of children through psychosocial mechanisms on their weight-related lifestyle can in turn be found in the composition or structure of families. Parity was previously found protective of childhood obesity (120, 225) and there is limited evidence suggesting that lone parenthood may pose children at a higher risk of OWOB (27). Notably, the overall family lifestyle appears to differ depending of the number of adults and children that live together in one household, which may explain the relationship of family structure with early childhood obesity (224, 226). For example, families with two parents and more than one child were found to eat meals together on a more regular basis than smaller families (226). Children in two-parent households furthermore tend to watch TV less (226), possibly due to higher levels of parental supervision (1), and are less likely to have their own TV (226). Additionally, lone parents may be more socially isolated, partially due to their limited availability of time and money. Accordingly, they may have low levels of informal social support and be unaware of affordable formal support services that may benefit their children's weight-related lifestyles (223, 227). Single children, in turn, may eat out of boredom or loneliness and may spend more time pursuing sedentary activities with parents compared to children who play active games with siblings (223). Furthermore, it is possible that single children may be treated similar to adults and served bigger portions, potentially increasing children's energy intake (223).

Furthermore, a higher risk of OWOB was observed among children whose parents separated between the ages of two and five years, compared to children who grew up in a stable marital relationship or in a stable lone-parent household (228). Children often use eating as a coping mechanism to the psychosocial stress that they are likely to experience in unstable family structures, which may explain this association (223, 229). Particularly in young children, however, stress levels are difficult to measure, which is why such potential relationships between their stress and OWOB are difficult to assess and largely unknown (27). Similarly, there is no clear evidence that parental stress in general or stress related to parenting

specifically alters the OWOB risk directly (27, 230). However, a lower level of physical activity among young children may exist and parents appear to establish fewer rules for TV watching hours if they experience stress related to parenting, thus suggesting an indirect relationship through lifestyle adaptations (207, 230).

Higher levels of OWOB in young children were also observed in relation to parental mental disease (27). Similar to parental stress levels, maternal depressive symptoms were linked to increases in time that preschool children spent watching TV and limited evidence suggests that children had lower physical activity levels (231). Low maternal resilience to mental stressors was additionally related to an increased amount of TV time and a lower dietary quality among children (231). All together, these studies strengthen the assumption that family lifestyles are influenced by psychosocial mechanisms that increase the risk of early childhood obesity based on family structure and stress as well as resulting family dynamics.

Summary

The cumulative findings of children's early growth and lifestyles presented above suggest that while associations between factors related to birth and the infancy period may still be at stake, children's lives start getting more complex when individual factors appear to influence the OWOB risk. While OWOB is mainly understood to develop through a higher energy uptake than energy is expended, other lifestyle factors were also identified that contribute to the individual OWOB risk of young children. These factors are likely dependent on the family environment and should be studied mutually, as individual dietary factors may be insufficient to assess children's OWOB risk. Many factors identified in the literature were part of the original model by Monasta et al. (1); however additional factors were identified (e.g. children's sex, physical activity and psychosocial factors), that were merged with the model, as will be presented in Section 2.5.1.

2.3.4 Key findings on early child growth and childhood obesity

To summarise the evidence on early child growth and childhood obesity, the following main findings arose from the presented literature:

- ü Children's growth throughout the foetal, infantile and early childhood years goes through periods of accelerated and decelerated weight and height gains, depending on their age and sex
- ü A number of risk factors of early childhood obesity were identified on the individual child level of the SEM which relate to the behaviour of children, namely their nutrition, physical activity, sedentary behaviour and sleep
- ü Many risk factors of early childhood obesity were also identified on the level of family characteristics which influence the gestational period, birth outcomes and infant feeding, as well as children's weight-related lifestyles
- ü Not all relationships between risk factors and early OWOB have been fully clarified; however biological and psychosocial pathways are hypothesised to influence the obesity risk of young children

The literature presented above mainly relates to the inner two levels of the SEM. As Reed et al. (8) identified various risk factors of OWOB in the community, demographic and societal environment of children, these will be discussed in the following section.

2.4 Inequalities in childhood obesity

Socio-economic inequalities were found to affect the risk and prevalence of OWOB in Ireland (35, 85), including inequalities in obesity and related health behaviours that were seen among Irish school children (41, 94, 232). The report *ó Tackling health inequalities ï An all-Ireland approach* points out that inequalities particularly exist with regard to the accessibility of healthy diets and physical activity. Families with low incomes struggle to afford fresh healthy food, which is often not available at a low cost in shops situated in more deprived or rural neighbourhoods (85, 88, 233). Consequently, the diet of low-income families in Ireland tends to involve high amounts of energy-dense and processed foods high in fat and sugar (85, 88). This has important long-term implications for the nutritional habits of family members in low-income families who buy prepared meals and admit lower cooking skills in a qualitative study carried out in Ireland (88). As various indicators of SES have been related to early OWOB in past studies, each indicator, namely the occupational class of households, parental education and income, will be discussed more in detail below.

2.4.1 The socio-economic status of families with young children

Children of economically disadvantaged parents are more likely to develop OWOB and growing gaps of OWOB prevalence between low- and high-income groups seen in past decades may in fact reflect the widening of gaps between low and high incomes (234). Additionally, families with lower incomes tend to accumulate in low-income neighbourhoods. Within such neighbourhoods, a tendency towards lower availability of food outlets that sell a wide range of affordable healthy food, and lower availability of facilities which support physical activity, and higher availability of fast food restaurants exists (234, 235). However, even where healthy options are available these may not be accessible to low-income families due to a lack of affordability or perceived affordability (27). Processed food, particularly food high in fat and sugar, is often cheaper than micronutrient-dense food, such as fruit and vegetables, which have become comparably more expensive over time (236). Accordingly, a higher dietary quality and lower obesity levels were observed

among children in families with high income (236). These patterns have also been observed in Ireland, where families on social welfare benefits can often not afford recommended quantities of healthy food (85, 235). In turn, it is assumed that parents who go through financial hardship adopt child feeding and parenting practices explaining higher OWOB levels early in childhood (120). Low-income families may experience food and housing insecurity and adopt unhealthy behaviours to cope with these stressors (227, 237).

Another indicator of SES which some studies relate to childhood obesity is the occupational household class. In a systematic review of studies from the UK, the risk of childhood obesity was generally higher in lower social classes (237). Additionally, low social class is associated with high birth weight and early child growth, as well as higher rates of gestational smoking, lower breastfeeding, early age at onset of solid food, and children's dietary quality (238), all of which were discussed as potential risk factors of OWOB earlier in this chapter, and which may explain the relationship with childhood obesity.

A limited, but growing, amount of evidence relates lower educational levels of mothers (174, 234, 237, 239) and to some extent fathers (234) to childhood obesity, even though study results limited to children under the age of five were found to be mixed (234). One study found that disparities in OWOB based on maternal education only emerged at the age of four years and widened with growing age after this (29), which may explain the limited evidence in young children. Additionally, findings differ between countries (153); for example high parental education was related to OWOB among six-year-old children in Lithuania and Bulgaria, but not in the Czech Republic, Portugal and Sweden (240).

Despite the inconsistent findings, according to the systematic review by Barriuso et al. (234), maternal education is a better overall risk factor of childhood obesity than other socio-economic indicators. The relationship with OWOB is likely to be associated with healthier diets and physical activity levels found in children of higher-educated mothers (234, 236) as well as higher adherence to breastfeeding guidelines (241). Overall, the home environment of preschool-aged children was found more obesogenic with lower maternal educational levels (242), which may be due to a higher level of health-related knowledge, for example nutritional

knowledge or health literacy (227). Furthermore, parents with higher educational levels themselves tend to pursue healthier behaviours, which they often learned from their own parents, and which their children tend to imitate (227).

Irrespective of the SES indicator used, current research suggests that protective factors of childhood obesity are less accessible to lower SES groups than to families with a high SES. The same holds true for childcare services, where families with lower incomes may need to turn to cheaper facilities that potentially offer a lower quality of service and sub-optimal levels of physical activity and healthy dietary options (243). Simultaneously, families with high incomes may be able to provide parental care for children if one of the parents does not have to work. The relationship of childcare and obesity will be discussed as follows.

2.4.2 Non-parental childcare environment

The number of families with both parents in full-time employment has increased over the last decades and maternal employment has been associated with OWOB in preschool-aged children (174), indicating that non-parental childcare may influence the development of obesity among young children. Various types of childcare arrangements are available to families, such as formal childcare which is mostly centre-based, or informal childcare provided by non-professional childminders such as grandparents or neighbours (244). Infants attending any kind of childcare (compared to being minded by their parents) were found to have a slightly higher risk of obesity within the first two years of life (120). In another systematic review, informal childcare particularly was related to OWOB, and this relationship was stronger in children below the age of three years than in older preschool-aged children. While study findings differed, formal childcare attendance tended to protect children from OWOB in the later preschool years (243). In yet another study, only informal care arrangement involving grandparents and grandmothers specifically as the main carer were strongly related to increased levels of OWOB in infants and children up to the age of five years (174).

Moreover, it is suggested that the amount of time children spend in non-parental childcare every week may further increase the risk of OWOB (120, 243). The relationship between childcare attendance and OWOB may be mediated by a shorter breastfeeding duration and earlier weaning among children who attend childcare in general and informal childcare in particular (174). At the same time, formal childcare has the potential to contribute to a healthy diet and physical activity level through which the obesity risk of young children may be lowered (27), whereas grandparents may be more willing to provide 'treat foods' or inappropriate portion sizes to children (243). Overall, a lack of structure due to the exposure to different social environments as well as heterogeneous caregiving styles in parental and non-parental childcare may explain OWOB outcomes (243).

2.4.3 Characteristics of the neighbourhood families live in

Particularly within urban settings, green spaces, parks and playgrounds offer a unique opportunity for children to become active outdoors. Objectively measured proximity to larger parks has been related to increased prevalence of OWOB in children of various ages (111). Aggio et al. (245) found a link between greater distance to green spaces and preschoolers' time spent watching TV, which also has been observed among older girls in Ireland previously (108). The availability of green spaces in the immediate environment may therefore help reduce children's sedentary time and potentially increase their physical activity levels. This finding was reinforced in a large Japanese study which identified an increased incidence of childhood obesity in Japan following the Great East Japan Earthquake. The only explanation supported by this study was that children's access to outdoor play areas had been reduced dramatically, due to radiation contamination, numerous building sites and the use of former play areas for temporary housing (246).

Simultaneously, however, strong evidence was seen that identified a higher level of child OWOB among families living in rural regions (247), where higher access to green and play spaces would generally be expected. One theory behind the higher OWOB risk in rural regions is that a lack of transport and greater distances to food outlets result in greater car reliance (85). Furthermore, the distance to shops

selling healthy and affordable food may lead to an increased consumption of unhealthy food found in small local shops, or foods with a long shelf-life, which excludes fresh fruit and vegetables (85).

Additionally, the average SES of a neighbourhood has been linked to an increased individual OWOB risk among five- to six-year-old children (248), which is supported by other studies relating objectively measured deprivation status of the neighbouring environment to OWOB (237, 249). This trend was observed independent of other socio-economic characteristics of families. Adjusting for such characteristics, a high level of neighbourhood deprivation was associated with a 1.7-fold increase in the incidence of obesity among a large cohort of zero- to 14-year-old children that were followed over a period of eleven years (249). Similarly, an association with lower OWOB levels was found among neighbourhoods with a higher average income (111, 234). Potential mechanisms for this relationship were discussed in relation to family income in Section 2.4.1.

Parental perceptions of the neighbourhood

Apart from objectively measured neighbourhood characteristics, the parental perception of the environment seems to play an important role and may in fact have a higher impact on the risk of childhood obesity (111). Increased levels of child OWOB were found with increasing parental self-reported proximity of the family's main food outlet and the availability of fresh fruit and vegetables in this outlet (111). A Portuguese study furthermore found that levels of OWOB were lower if parents perceived the environment to have a low level of crime, be safe to walk or cycle during day and night, be comfortable and safe for pedestrians to walk, and offer intact footpaths (250). Other studies found higher TV watching hours and a lower participation in physical activity in children whose parents reported the environment not to be safe, which could explain the relationship between perceived neighbourhood safety and childhood obesity (206, 251).

2.4.4 Ethnic background

Inequalities in the risk of childhood obesity were observed by the age of five years based on children's ethnic background (174), in addition to the differences that generally exist between countries (239). In a German cohort study, the risk of high weight was elevated in six-month-old children with Turkish or Eastern European background. These children were found to have had a similar birth weight to other children, followed by rapid weight gain (252). Higher levels of OWOB and of rapid infant weight gain were as well found in two-year-old children of Turkish or Moroccan origin living in the Netherlands (253).

It needs to be noted that much of the evidence on ethnic differences in childhood obesity comes from American studies where the ethnic variety differs slightly to that in many European countries. In these studies, higher levels of OWOB tend to be seen among children of African-American or of Hispanic ethnicity (254), as well as among Mexican American and Native American, compared to white American children (236). The obesity levels of young children may be affected by different distributions of fat in the body, different metabolic functioning (including different insulin responses) (236) or varying hormonal levels (of leptin) that were found in children based on ethnicity (255). Furthermore, Hispanic and African-American children were found to have higher records of rapid infant weight gain, inadequate sleeping hours, lower exclusive breastfeeding rates, and earlier weaning (255), suggesting that biological pathways lead young children to develop OWOB.

Moreover, inequalities may occur due to psychosocial stress from the experience of discrimination within certain ethnic groups (236) or due to depression (256). Some ethnic groups in the US tend to live in poorer neighbourhoods relative to their income and have different incomes relative to their education level than observed in other ethnic groups (236). This may further increase psychosocial influences on the obesity risk through social exclusion (85), or aggravate the risk through material mechanisms (see Section 2.4.1 for details).

In terms of lifestyle, young children were more likely to have their own TV, and to consume sugar-sweetened beverages and fast food more frequently if their mothers were Hispanic or African-American, compared to white American (256).

Variations in food consumption patterns are also considered a likely explanation for ethnic differences in childhood obesity in other studies, just as culture-related beliefs and knowledge about the benefits and risks of certain foods, physical activity and obesity itself. This ties in with different beliefs of which body size is defined as ideal or as acceptable, for instance a preference for higher BMI has been seen in African-American compared to white populations (27, 236). Overall, it appears that differences in the risk of childhood OWOB between ethnic groups may operate through various pathways. Therefore, it was suggested that interventions tailored for ethnic groups, such as increasing their health knowledge, may be needed pre-conceptionally, throughout pregnancy and during the infancy period (257), need to account for potential language barriers and need to consider the possibility that parents may not be aware of existing health services (227).

2.4.5 Theories on the development of inequalities in childhood obesity

A number of relationships with early OWOB in children on the community, demographic and societal level of the SEM, in addition to the parental influences and factors related directly to children identified earlier (Section 2.3), were discussed in the literature review. An overall higher OWOB prevalence among children growing up in families of lower SES was seen, suggesting a strong socio-economic pattern. These differences in prevalence were related to financial barriers to physical activity and access to healthy nutrition, or to neighbourhoods which provided (perceived) access to either. Many possible pathways to OWOB furthermore exist that explain the inequalities that were observed based on the ethnic background of families. Childcare service may have potential to either lower (formal childcare) or increase (informal childcare) the OWOB risk of young children and accordingly inequalities between them.

A number of theories were developed that aim at explaining inequalities in health outcomes. In 2008, the Commission on Social Determinants of Health (4) adopted a framework by Solar and Irwin (6) that acknowledges a number of health determinants which were observed in relation to early childhood obesity throughout the literature that was reviewed for this chapter. The framework is depicted in Figure 4. More specifically, the determinants of health included in this framework, which are referred to as *ó s t r u c t u r a l* by the Commission on Social *ô* Determinants of Health, are the social position, education, occupation, income, gender and race/ethnicity of individuals (86). Most of these *structural determinants* are included on the outer level of the SEM and were discussed in this chapter (Sections 2.4.1 to 2.4.4), while the *structural determinant* of gender is an individual-level factor of the SEM.

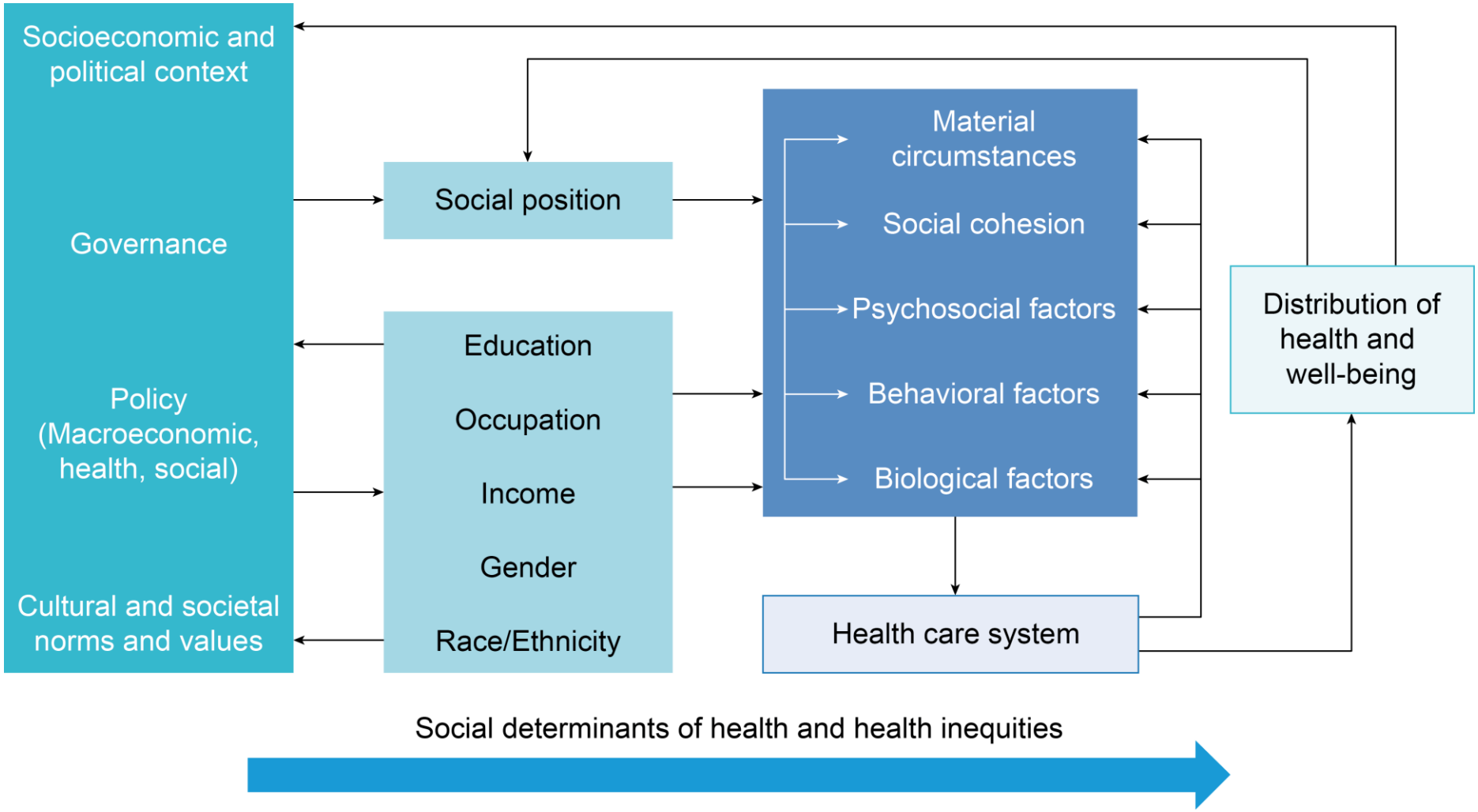

Figure 4: Commission on Social Determinants of Health conceptual framework

Retrieved from the Commission on Social Determinants of Health (4), amended from Solar and Irwin (6)

As has been described previously, the literature review revealed that many of the factors on the community, demographic and societal level may impact the risk of early childhood OWOB through changes in the family's weight-related behaviour. However, mechanisms were also seen in relation to biological processes, the psychosocial stress of families or the affordability of resources that may alter the obesity risk among families in various population groups. The *Commission on Social Determinants of Health conceptual framework,* which visualises how a number of health determinants translate into differential outcomes in health and well-being through these mechanisms, calls these mechanisms *intermediate determinant*. Importantly, the Commission on Social Determinants of Health is based on the idea of a life-course perspective that has been adopted in health theory and is also core to the Irish *Obesity Policy and Action Plan* (35, 86).

According to the life-course perspective, determinants of health that occur among adults are likely to translate into health inequalities among their children which themselves translate into children's later inequalities through latent, pathway or cumulative effects (258). Latent effects on health occur through events or exposures that impact health outcomes independent of later events or exposures, for example effects of breastfeeding or in utero exposures may impact children's health throughout their childhood, independent of their later nutrition. In turn, early nutritional and physical activity habits may have pathway effects on children's health; that means they are likely to influence the children's later nutrition and physical activity habits and affect adult health through the behaviours learned in childhood and pursued in adulthood (or alternatively through the choice to change these behaviours). Cumulative effects refer to effects on health from long-term exposure, such as growing up in a deprived neighbourhood (bid). Moreover, the life-course perspective on the development of health stipulates that negative effects on health tend to cluster and accumulate over time (259), meaning that exposure to risk factors of OWOB early in children's lives can potentially set children onto a path of recurrent exposures to risk factors. In Chapter 1 (Section 1.2), it was argued that early childhood obesity is likely to translate into later obesity and associated ill-health, developmental disadvantage and economic cost. Therefore, identifying latent, pathway and cumulative causes of early childhood OWOB is crucial to prevent health inequalities in the Irish population.

As indications for an intergenerational transmission of obesity through latent, pathway and cumulative effects were seen in the literature that forms the background to this thesis, the *Commission on Social Determinants of Health conceptual framework* was adopted as the theoretical background to this study. Previous research has shown that the use of theory is important both to refine existing theories applicable to health research through its application and verification in various research settings, as well as to improve current health-related practice (260).

According to the *Commission on Social Determinants of Health conceptual framework,* a mediating factor for health outcomes is the healthcare system. As this is universal to children in Ireland, the influence of this factor should be the same across children as is the political context for children depicted in the framework. For this reason, while the *Commission on Social Determinants of Health conceptual framework* refers to health inequities (that is, different opportunities to maintain a healthy weight), it is more appropriate to talk of health inequalities in the Irish context of childhood obesity where different outcomes are expected between population groups despite equal opportunities.

In the next section, a brief description of *intermediate determinants of health* that appear most relevant to the development of early childhood obesity will be provided. Material circumstances, psychosocial factors, behaviours and biological factors were the most common theories derived from the literature review and are accordingly expected to explain how the prevalence and potential inequalities of childhood OWOB evolve in Ireland. In order to explore the causes of inequalities in complex health outcomes, such as OWOB, Arcaya et al. (258) suggest that all four theories need to be taken into consideration.

Material circumstances

The material circumstances of individuals refer to their ability to afford health-related resources, such as healthy food, as well as living conditions and the overall affluence of the neighbourhood that they live in. Individuals who experience financial hardship or live in disadvantaged neighbourhoods are likely to experience

negative health consequences not only due to their exclusion from one particular health-promoting resource, but their tendency to have lower access to a number of resources that lead to an accumulation of risk factors for their health. For example, the infrastructure of poorer neighbourhoods is likely to offer a reduced range of health-promoting services and facilities, but low-income families most likely cannot afford moving to more affluent areas that offer more. Consequently, low-income families are more likely to adopt a range of behaviours that may negatively influence their health (86), including their own and their children's weight status. The distribution of health in a population is understood to reflect the population's distribution of resources. In populations where health and resources are distributed unequally, an improvement in the health of disadvantaged groups can be reached through the provision of resources, such as through improved living conditions. Nevertheless, it is difficult to close gaps in resources and related health outcomes as improvements in disadvantaged groups frequently occur in congruence with improvements in advantaged groups, therefore only shifting the distribution to better average health with remaining inequalities (261).

Psychosocial factors

These factors refer to psychosocial stressors, which include the social support and quality of relationships, stress related to living circumstances, as well the individual ability to cope with stress. While stress may lead to changes in neuroendocrine processes that ultimately affect individual health, the perceived positioning in society, social exclusion and a lack of social support may as well lower the capability of individuals to achieve better health (86). Accordingly, it is possible that dynamics in the family environment cause parents and children to experience prolonged stress and adopt behaviours that promote excess weight gain. Additionally, a lack of social support experienced by young children as well as weak family relationships and a lack of structure may cause children to adopt unhealthy behaviours, for example to use eating as coping mechanism. This may be increasingly promoted by the time constraints of lone and working parents, which lower the opportunities for family members to interact and to respond to children's emotions (229). Particularly in the early ages, responsive parenting and

positive attachment in the family appear crucial to children's physical development and a lack of these experiences may increase the likelihood to negative health outcomes (227).

Biological factors

While biological factors are tied to behavioural factors according to the Commission on Social Determinants of Health, unique to the domain of biological factors is the individual genetic makeup (86).

Genetics and epigenetic factors were found to influence the way children grow in the literature reviewed for this study. Beyond the individual genetic make-up, however, which is hard to test and (almost) impossible to change[3], differences in growth also seem to occur based on the sex and ethnicity of a child. Moreover, a central role in the weight gain of children was attributed to in utero exposures, children's birth outcomes and early feeding practices (27, 119, 174). While associations between parental and child weight at a later age may be explained by a shared environment, parental role modelling and the pursuit of a similar lifestyle across families, such assumptions cannot be made before children transition to a normal family diet. Hence, the maternal weight status, weight gain and overall nutrition during the pregnancy, all of which are associated with early childhood OWOB, is very likely to cause biological mechanisms that influence the child in utero and lead to varying birth outcomes (158). Additionally, the observed protective effect of breastfeeding on early OWOB in children most likely occurs based on biological factors that differ between children who were breastfed and those who were formula fed. For example, differences were observed in the microbiome of children of both groups that may explain this relationship. The microbiome, in turn, may also be altered by frequent antibiotic exposures, which could increase the risk of children to become overweight or obese (198).

[3] The possibility of genome editing exists and has recently been performed in embryos using CRISPR/Cas9; however this entails serious ethical concerns and a high risk of adverse health effects, which is why no ethical approval was granted to the researcher for this procedure (262).

Behaviours

The main groups of health-related behaviours outlined by the Commission on Social Determinants of Health are nutritional behaviours, physical activity levels and the consumption of tobacco and alcohol. Healthy behaviours tend to be observed more in higher socio-economic groups; however the adoption of behaviours as well may vary based on psychosocial factors, and some behaviours in fact are linked to health outcomes mostly through biological processes (86). Therefore, with respect to this thesis, they are not regarded as an individual mechanism that may act on children's weight gains, but should be investigated in congruence with material, psychosocial and biological factors. Furthermore, the behaviour of children and their parents need to be studied together for instance gestational behaviours and infant feeding are guided by parental decisions.

2.4.6 Key findings on inequalities in childhood obesity until five years of age

A number of findings were collated from the literature explored in Section 2.4, the main points of which are as follows:

- Inequalities in the distribution of OWOB among children in and outside Ireland exist, such as based on ethnicity and SES
- Community, demographic and societal characteristics appear to influence the individual risk of early childhood obesity
- Theories that explain why the relationships between risk factors and early childhood obesity may exist mainly surround those of material circumstances, biological factors and psychosocial stress which are hypothesised to either influence the weight-related behaviours of children and their families, or the obesity risk of children directly.

2.5 Discussion

2.5.1 Presentation of the extended conceptual model

Based on the cumulative findings of the literature review, the merged SEM and *Complex web of potential determinants of overweight and obesity in children* were extended, as depicted in Figure 5. The various factors added to the model were colour-coded according to different levels of the SEM, and arrows were added to the model that indicate how these factors are assumed to be related to childhood obesity. For example, no direct relationship between parental stress and childhood obesity could be seen in the literature, but parental stress may contribute to increased time children spend watching TV, as well as it could influence their diet, both of which is related to childhood obesity. As can be seen from the colour-coding, factors that are on the outer circle of the SEM, which includes community, demographic and societal factors (blue), tend to influence each other or factors on the more inner circles. Factors on the parenting and family level (orange) also influence each other, or they influence factors on the individual child level or the risk of childhood obesity directly. Individual child level factors (red) influence each other or the risk of childhood obesity. However, no relationships were seen in the opposite direction; that means no factors from the inner levels of the SEM appeared to influence factors on the outer levels. Therefore, the findings from the literature review confirm the usefulness of the SEM to give structure to studies investigating early childhood obesity. The finalised model in Figure 5 will be applied to this thesis to test the theories outlined above (Section 2.4.5), which were developed into a number of research hypotheses that are presented in the following section.

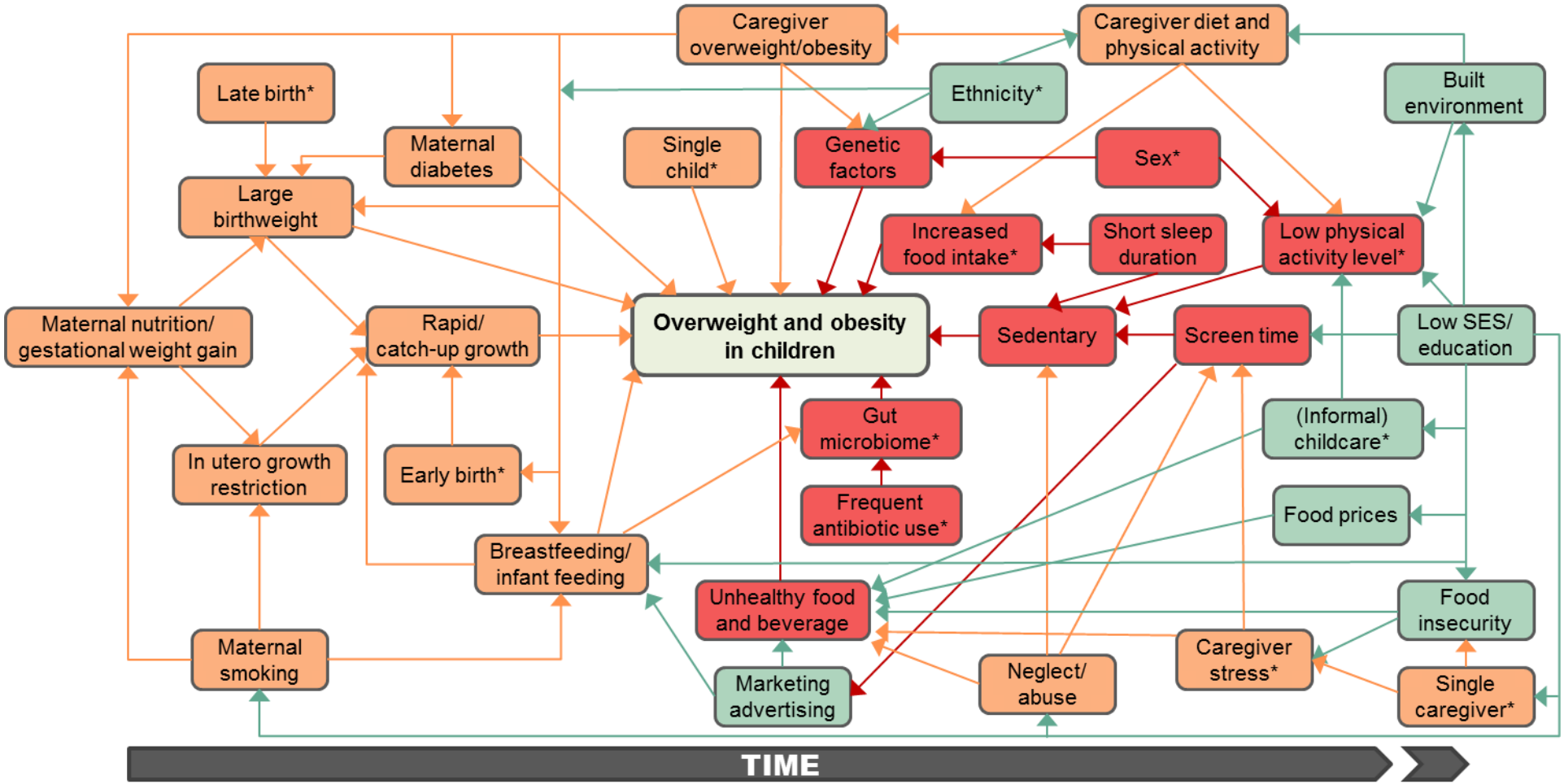

Figure 5: Extended Complex web of potential determinants of overweight and obesity in children by Monasta et al. (1), including colour-coding according to the levels of the Social Ecological Model and findings from the literature review of this thesis

Note: red colour = individual level; orange colour = parenting style and family characteristics; blue colour = community, demographic and societal characteristics

* These factors were not part of the original model but were integrated based on evidence from the literature

SES = Socio-economic status

2.5.2 Hypotheses

The research gaps presented in Chapter 1 and the background presented in this chapter motivated and informed the design of this thesis. In the following section, the specific hypotheses of this thesis will be presented that seek to fill the mentioned research gaps and reach the aims and objectives of this thesis.

Hypothesis 1: Ireland has a high prevalence of early childhood overweight and obesity

It is hypothesised that the prevalence of OWOB in children up to the age of five years in Ireland is high and may be increasing over time. At least one in five children is expected to be overweight or obese, similarly to levels in older children, but the prevalence in the youngest age group may be higher.

Hypothesis 2: Material circumstances increase the risk of early childhood overweight and obesity

It is furthermore hypothesised that the risk of developing childhood OWOB in the early years is not the same across all population groups. Socio-economic inequalities are expected to be seen between children that are likely to explain variations in the OWOB risk through material circumstances of the families that children grow up in. Higher OWOB levels are expected among children in low-income families, in children whose parents have lower educational levels and in those children who live in neighbourhoods that offer low levels of safety and few opportunities to play outside.

Hypothesis 3: Biological factors increase the risk of early childhood overweight and obesity

Moreover, biological factors are expected to influence a child's individual OWOB risk particularly early in their lives. Hence, it is hypothesised that elevated OWOB

levels will be seen in children with high BWFGA, children with rapid infant weight gain and children who were not breastfed or weaned early. Furthermore, after infancy, a number of lifestyle factors (high dietary intake, low physical activity levels, high amount of sedentary behaviour, antibiotic intake and short sleep duration) are expected to increase the risk of OWOB due to biological mechanisms.

Hypothesis 4: Psychosocial factors increase the risk of early overweight and obesity

In addition to material and biological factors, it is acknowledged that families are increasingly experiencing time pressure and stress due to a change in family dynamics over the past decades. Hence, psychosocial factors, namely family stress, living in a lone-parent household and spending time in non-parental childcare, are hypothesised to act as a force that may increase children's individual risk to develop OWOB, such as through impacting family lifestyles.

Hypothesis 5: Different risk factors of obesity lead to excess weight gains at various time points in children's early years

Lastly, it is hypothesised that material and biological factors, as well as psychosocial factors do not have a constant impact on young children's weight status, but influence changes in children's BMI differently over time. It is assumed that critical time points in the development of BMI vary for the different risk factors and mechanisms that ultimately contribute to the development of OWOB but lead to an elevation in relative weight gain before a change in weight status becomes visible. Specifically, biological factors during the prenatal and infantile period are hypothesised to lead to early BMI changes, whereas material and psychosocial factors are hypothesised to gain relevance after infancy.

CHAPTER 3:

Methodology

3.1 Introduction and objectives of this chapter

The previous chapter of this thesis outlined a number of research gaps, as well as aims and objectives for this thesis to investigate the prevalence and risk of OWOB in young children in Ireland longitudinally. Accordingly, this chapter will outline the research approach and methodology used in this study to fulfil these aims and objectives and fill gaps in the current state of knowledge in Ireland.

Secondary analysis of a longitudinal prospective study was conducted to achieve these aims. Specifically, the quantitative infant cohort data from the GUI National Longitudinal Study of Children was used following a critical literature analysis of child growth criteria. The GUI infant cohort includes the weights and heights of 11,000 children who were first studied at the age of nine months and again at three and five years. In addition to basic demographic information, a large number of variables were collected from the child and the child's caregivers, who were usually their mother and father, at each of the studied ages. These include variables relating to the development of children, their behaviour, social environment and physical environment, which make the data particularly suitable to this thesis. Moreover, the longitudinal nature of the GUI study and the large sample size offered major advantages to this thesis, which will be discussed throughout the course of this methodology chapter.

This chapter is divided into two main parts, first, the study design and methodology that was originally used to collect the GUI infant cohort data, and second, the description of the methodology to conduct the analysis of the GUI data that was specific to this thesis. The first part, described in Section 3.2, will describe the overall study design, sampling frame and data collection employed by the GUI study team during each wave of data collection. Furthermore, characteristics of the study population will be presented, along with considerations of attrition that took place between waves and the handling of non-random attrition by the GUI study team. This includes the use of weighting factors that were used to make the GUI infant cohort data nationally representative. Other efforts that were undertaken to improve the quality of the GUI data will be discussed, and ethical considerations by the GUI study team included at the end of this section.

Secondly, in Section 3.3, a closer look will be taken at variables collected as part of the GUI study and those variables relevant to the study of OWOB in young children used in this thesis.

Finally, Section 3.3 will describe the analyses undertaken to test the hypotheses and achieve the specific research aims and objectives of this thesis that were outlined in Chapter 2.

3.2 The Growing Up in Ireland Longitudinal Study of Children – the infant cohort

3.2.1 Study design

The GUI National Longitudinal Study of Children is a prospective cohort study based on a fixed panel design that follows children to study their development and well-being, as well as factors that may contribute to various areas of child development. Data collection began in 2008 with data being collected on 11,134 nine-month-old infants (99).

The GUI study followed on from The National Children's Strategy, which was published by the Department of Health and Children in 2000. The study commenced in 2006, led by the Economic and Social Research Institute and Trinity College Dublin (99). The objective of this study was to investigate child outcomes over time in the areas of 'physical health and development', 'social, emotional and behavioural well-being', and 'educational achievement and intellectual capacity' (263). Longitudinal data were collected from the infant cohort in 2008-2009 (wave 1), 2011 (wave 2) and 2013 (wave 3).

Previously, no study of this scope had been carried out in young children in Ireland. The GUI infant cohort data hence offer a unique opportunity to investigate the broad array of topics that are part of children's development and that were included in the interviews with the children and caregivers. In addition to demographic information and variables on the social, emotional, behavioural and cognitive development of children, also physical health data were collected in the interviews. These include standardised measurements of length or height and weight at the ages of nine months, three years and five years (99).

Due to the representative and longitudinal nature, as well as the age range, sample size and set of variables included in the GUI infant cohort (99), the data were chosen for investigation in this thesis. Following from here, the sampling frame, data collection and further study characteristics will be described that were used in the GUI infant cohort study.

3.2.2 Sampling frame

Children were sampled from the Child Benefit Register, which is maintained by the Department of Social Protection (formerly Department of Social and Family Affairs). Children were included in the sample if they were born between 1st December 2007 and 30th June 2008, during which time 41,185 births were registered on the Child Benefit Register (264).

The Child Benefit Register appeared to offer the most appropriate sampling frame as it provided comprehensive information about the age of children and other critical population characteristics. Child Benefits are paid to one parent or guardian of children and adolescents under the age of 16 years in Ireland. Due to this financial incentive, it is very likely that all infants are registered by their parents or guardians within the required period of six months from the date of birth, or alternatively from the start of residence in Ireland or the date that the child became a member of the family. A comparison of previous births by the GUI team showed that numbers from the Child Benefit Register were highly consistent with Vital Statistics from Ireland, and that only 3% of national births had not been recorded in the Child Benefit Register previously. Furthermore, the Department of Social Protection has an interest in avoiding duplication on the Register. Hence, the Child Benefit Register holds an up-to-date and comprehensive list of children who live in Ireland at any given time (264).

3.2.3 Sampling design

A systematic selection procedure, pre-stratified by a number of variables, and based on a random start and constant sampling fraction was used (265). In addition to the birth dates of registered children, the Child Benefit Register holds information about the marital status of recipients, county of residence, nationality and the number of children for whom the Child Benefit is claimed within one family or by one guardian. The sample included in the GUI infant cohort was pre-stratified based on these variables to represent overall population demographics, and required sample sizes for the different strata were calculated for each birth month

within the sampling period separately (264). The selected children are therefore a probability sample from the Child Benefit Register and, accordingly, from all Irish infants born in Ireland at the time of sampling. This offers major advantages over non-probability sampling as it removes the risk of sampling bias; that means all infants within the sampling frame had the same chance to be included in the study.

Furthermore, a pilot and dress rehearsal of the GUI study revealed that families in a number of population groups, including single parents and non-national families, were less likely than others to respond to the invitation to take part in the GUI study. Families in these groups were oversampled, in order to reach representative numbers of families in these groups in the final sample (264).

3.2.4 Panel design

All children and their families who had completed the first interview were eligible to participate in subsequent waves of data collection if they were still resident in Ireland. As the study follows a pure fixed panel design, no children were included who did not participate in wave 1. However, a number of children from wave 1 did not participate in wave 2 and/or in wave 3 due to non-response or attrition (266). Families were excluded from wave 2, and not targeted for inclusion in wave 3, if they wished to be excluded following the first wave (‘hard refusal’) or if they could not be located in wave 2 as they had moved homes and not forwarded their new address. Families who refused to take part only in wave 2, but did not wish to be excluded from the study overall (‘soft refusal’), were contacted again for participation in wave 3. Additionally, families who had lived abroad in wave 2 but returned to an address in Ireland before the fieldwork of wave 3 commenced were included in the sample of eligible families in wave 3. Therefore, the final sample of children in wave 3 consists of children who had previously participated in waves 1 and 2, and those who only participated in wave 1 prior to collection of the wave 3 data (266).

3.2.5 Data collection

The GUI infant cohort data were collected by trained interviewers, using a number of questionnaires and tools to assess various domains of child development. Specific procedures and instruments used for data collection in each of the three waves are outlined as follows.

Instrument development

The questionnaires used to study various domains in the nine-month-old children were developed based on the scientific and policy expertise of the Scientific and Policy Advisory Committee, which included ten experts from different disciplines and including those that were familiar with the design and conduction of comparable longitudinal studies. Additionally, 69 experts were included in a two-round Delphi process (a systematic, interactive method for data aggregation in complex fields with multi-stakeholder involvement), more than 45 stakeholders were included in a Panel of Experts, and other stakeholders were consulted for advice, all of which contributed to the development of the questionnaires and additional tools used to collect data. Furthermore, questionnaires and additional tools were aligned with those used in other child cohort studies, in order to make the study outcomes comparable to those from other countries (99, 267).

The development of questionnaires used in waves 2 and 3 followed similar procedures, however no Delphi study took place (266, 268). Additionally, for wave 3, an expert panel was formed that focused on health and health policy (266). Details of the instrument development for each wave have been published elsewhere (266, 267, 269).

Pilot studies

The questionnaires of waves 1 and 2 were tested in a pre-pilot, pilot and a dress rehearsal phase before the respective wave of the GUI infant cohort data was

collected (263, 268). In wave 3, two pilot studies were conducted to test the questionnaires; a pilot took place in the household of families, and a second pilot was run in schools as a number of children had started going to school before the third wave of the GUI infant cohort data were collected, in which case teachers and principals were interviewed in addition to caregivers (266). Details of the pilot studies are published elsewhere (263, 268, 270, 271), and summaries are provided as follows.

Pre-pilot phase

In summary, the pre-pilot of wave 1 involved a convenience sample of 22 families with infants aged seven to eleven months who were sampled through staff of the Economic and Social Research Institute and Trinity College Dublin (99). In the pre-pilot of wave 2, 21 of these families were interviewed again (268). This was to test preliminary questionnaires and led to the removal of a number of questions for use in the final questionnaires, as well as the distinction of questions into those included in a main questionnaire administered by the interviewers, and those included in a sensitive questionnaire for self-completion by the families (99, 268). Additionally, in wave 2, direct cognitive assessments were tested for feasibility among study children in the home environment, and they were included in the later interviews based on the outcome in the pre-pilot (268). No pre-pilot was conducted for wave 3.

Pilot phase

In the pilot phase of wave 1, 359 families were sampled from the Child Benefit Register which included infants born between 10th April and 9th May 2007. An introductory letter, information sheet and opt-out form were posted to selected families. If the families did not wish to participate in the study, they were asked to fill in and return the opt-out form to the study team. Otherwise an interviewer visited the families in their homes for an interview approximately two weeks later, which was between the end of January and mid-February 2008. The main purpose of the

pilot was to receive feedback on procedures, protocols, instrumentation and measures. It led to the inclusion of a number of tools for the developmental assessment of the infant, as well as to decisions around the means of assessment in the questionnaires developed by GUI (99).

In the pilot phase of wave 2, families who had participated in the wave 1 pilot were sent and introductory letter and information sheet again, followed by an interview in the home. Interviews in the pilot phase of wave 2 were completed in 177 families, which amount to a response rate of 88%. Families provided feedback on the questionnaires, cognitive tests and other instruments used to collect data, as well as on overall procedures (268).

The household pilot of wave 3 was undertaken in October and November 2012 and 198 families were interviewed who had previously participated in the wave 1 pilot (a response rate of 90% since wave 2). A main focus of this pilot was the integration of educational, preschool and school variables in the interviews, as these had not been assessed in past interviews. Additionally, the household pilot led to the exclusion of a number of questions to caregivers and hence the reduction of the overall interview length. Other questions were identified where additional response categories were needed, and the final questionnaires were adjusted accordingly (266).

The school-based pilot study of wave 3 was carried out from November 2012 until February 2013. Primary caregivers (PCGs) who participated in the household pilot were asked for written consent to contact the school and teacher of children if applicable. A sub-set of 60 schools were approached for participation in the pilot study, 52 of which completed all questionnaires. Based on the school-based pilot, the school component was agreed for inclusion in the GUI infant cohort at wave 3 and questionnaires were adjusted (266). However, no information from the school-based interviews was included in this thesis as not all children had started attending school at the time of data collection. Therefore, additional details on the methodology used in school-based interviews will not be presented (266).

Dress rehearsal phase

Finally, the dress rehearsal included 413 children sampled from the Child Benefit Register that were to be nine months old between 12th May and 13th June 2008, when the dress rehearsal of wave 1 took place. The dress rehearsal was conducted to retrieve information about the time interviewers required per household visit, including their introduction and the signing of consent forms, the duration of interviews, as well as physical measurements (99). In wave 2, 237 of these families participated in wave 2 (a response rate of almost 88%). The dress rehearsal was used to refine the questionnaires and instruments in use, leading to the final interview design (268). No dress rehearsal took place in wave 3.

Interviews

The final questionnaires were used to record information from the PCG of each infant, who was usually their mother, and the secondary caregiver (SCG) of the infant, who was usually their father (99, 266, 268). Furthermore, questionnaires were sent out to home- or centre-based carers of the child if families had a regular childcare arrangement in place (waves 1 and 2) (265) and to schools, for children who had started attending school (wave 3) (266).

The PCG was specified as the “self-defined person who provides most care to the child [or] knows [the] child best” (265) and the SCG was “self-defined as resident spouse/partner of [the] PCG” (265). Characteristics of PCGs and SCGs in each wave are provided in Table 1. As the PCG or SCG may have been a spouse or guardian who was not the mother or the father of the child, even though this was only the case in a small proportion of the families, the terminology suggested by GUI will be used throughout this thesis. Furthermore, there was a possibility that roles would change in the household across waves, and that another person was the self-defined PCG or SCG in wave 1 than in subsequent waves, which is most accurately reflected in this terminology (99).

Table 1: Caregiver characteristics that changed across waves, due to changes in the person who was the primary caregiver (PCG) or secondary caregiver (SCG) between waves

Characteristics of the PCG	**N in wave 1 (%)**	**N in wave 2 (%)**	**N in wave 3 (%)**
Total	11,134 (100%)	9,793 (100%)	9,001 (100%)
Parent	11,127 (99.94%)	9,783 (99.90%)	8,985 (99.82%)
Adoptive parent/relative	7 (0.06%)	10 (0.10%)	16 (0.18%)
Male	38 (0.34%)	159 (1.62%)	190 (2.11%)
Female	11,096 (99.66%)	9,634 (98.38%)	8,811 (97.89%)
PCG was the PCG in previous wave	N/A	9,638 (98.42%)	8,840 (98.21%)
PCG was the SCG in previous wave	N/A	144 (1.47%)	140 (1.56%)
PCG not interviewed in previous wave	N/A	11 (0.11%)	21 (0.23%)
Characteristics of the SCG			
Total	9,775 (100%)	8,576 (100%)	7,908 (100%)
Parent	9,760 (99.85%)	8,520 (99.36%)	7,791 (98.51%)
Adoptive parent/relative	6 (0.06%)	9 (0.10%)	13 (0.17%)
PCG's partner	9 (0.09%)	45 (0.52%)	94 (1.19%)
Other non-relative	0	1 (0.01%)	11 (0.14%)
Male	9,797 (99.61%)	8,423 (98.23%)	7,732 (97.77%)
Female	38 (0.39%)	152 (1.77%)	176 (2.23%)
SCG was the SCG in previous wave	N/A	8,232 (95.99%)	7,538 (95.32%)
SCG was the PCG in previous wave	N/A	137 (1.40%)	127 (1.41%)
SCG not interviewed in previous wave	N/A	206 (2.10%)	243 (2.70%)

Table 2 provides an overview of the various questionnaires that were used in each wave (272). For both the PCG and SCG, interviews were split into a main questionnaire for assessment by the interviewer using the Computer Assisted Personal Interview system, and a supplementary questionnaire that contained questions on sensitive topics and was self-administered by the respective caregiver using the computer system (99, 266, 268) (Table 2). The interviews furthermore included physical measurements of the length/height and weight of both infants and adults (99, 266, 268) as well as a number of cognitive and fine and gross motor tests with the study child during the second and third wave (266, 268). Blaise software was used for the conduction and encryption of interviews (267).

Among the infants included in the GUI infant cohort, 398 were non-singleton children (twins or triplets) and in 54 of their families, both twins were included as study children in the cohort. In this case, separate child-specific interviews (Table 2) were conducted with the caregivers for each child, in addition to one mutual questionnaire that was used for overall household and caregiver information and linked to each of the children included in the interview (99).

Table 2: Questionnaires used in the Growing Up in Ireland infant cohort study

Wave	Respondent/ assessment	Questionnaires
1	PCG in the home	• Primary Caregiver Main Questionnaire • Primary Caregiver Supplementary Questionnaire • Primary Caregiver Twin Questionnaire
	SCG in the home	• Secondary Caregiver Main Questionnaire • Secondary Caregiver Supplementary Questionnaire • Secondary Caregiver Twin Supplement
	Postal for parent (SCG)	• Non-resident Parent Questionnaire
	Postal for childminder	• Home Based Carer Questionnaire • Centre Based Carer Questionnaire
2	PCG in the home	• Primary Caregiver Main Questionnaire • Primary Caregiver Supplementary Questionnaire The Primary Caregiver Twin Supplement
	SCG in the home	• Secondary Caregiver Main Questionnaire • Secondary Caregiver Supplementary Questionnaire • Secondary Caregiver Twin Supplement
	Postal for parent (SCG)	• Non Resident Parent Questionnaire • Secondary Caregiver Supplementary Questionnaire
	Postal for childminder	• Home-Based Carer Questionnaire
3	PCG in the home	• Primary Caregiver Main Questionnaire • Primary Caregiver Supplementary Questionnaire • Primary Caregiver Twin Supplement
	SCG in the home	• Secondary Caregiver Main Questionnaire • Secondary Caregiver Supplementary Questionnaire • Secondary Caregiver Twin Supplement
	Postal for parent (SCG)	• Parent Living Elsewhere Questionnaire
	Postal for school	• Principal Questionnaire • Teacher on Pupil Questionnaire • Teacher on Self Questionnaire

Note: The list was taken from The Economic and Social Research Institute (272). The questionnaires involved additional measurement items and scales not provided online due to copyrights, which are listed in the Growing Up in Ireland summary guides (99, 266, 268).

PCG = primary caregiver

SCG = secondary caregiver

All questionnaires and other documentation were available in English and Irish, as well as in Chinese, French, Latvian, Lithuanian, Polish and Romanian, in order to prevent the exclusion of families due to language barriers (99). Information sheets were furthermore available in Braille and large font, and translators were available to households if required. Caregivers with disabilities were included and aided in the interview process as much as possible, for example deaf caregivers self-completed the main questionnaire with pen and paper, or the interviewer offered to read out and fill in the sensitive questionnaire for caregivers with vision problems (267).

Interviewers belonged to the Economic and Social Research Institute's national panel of interviewers and were trained for the fieldwork of the GUI infant cohort during a minimum period of four days. This was to familiarise the interviewers with the study, questionnaires and other tools and documentation, including ethical issues that may arise from this work and the incident report system that was in place for interviewers and operated on a 24/7-basis. The training also involved instructions on length, height and weight measurements both in infants and their caregivers (99, 266, 268). For example, prior to the data collection of wave 1, a public health nurse provided practical guidance on use of the weighting scales and measuring mats, which were recommended to be placed on the floor, rather than on a table, when children were measured. Furthermore, a mother and her nine-month-old child participated in this training session, as the interviewers needed to involve the PCG and ask them to hold the infant in a defined way, in order to take the measurements (99). Where possible, the same interviewer was employed to conduct the wave 2 interview who had interviewed the family in wave 1 (268). Also in wave 3, an effort was made to send the same interviewer to a family as in previous waves (266).

The first wave of data collection took place between September 2008 and April 2009, the second wave from December 2010 to July 2011, and the third wave from March until September 2013. In each wave, families were contacted by GUI and an introductory letter and information leaflet sent out to them before the interviewers established a first contact and visited the households to organise an appointment for the interview. Face-to-face interviews took place in the homes of the families

upon signing of a consent form by the parent(s) or guardian(s) of the study child. Caregivers could opt out of the study at any time before, during or after the interview had taken place. At the end of the interview, the PCG was asked to notify the GUI team of their new address if the family moved homes. Furthermore, the interviewer recorded contact details of two people outside the household who would be asked to establish contact with the family for the next wave of data collection, in case the family moved between the interview waves and did not notify GUI. The PCG received a copy of this tracing document. As a third means to re-contact families who moved to another address between interviews, the PCG was asked to provide their written consent which allowed GUI to trace the family through the Child Benefit Register (99, 266, 268). Additionally, written consent and contact details were requested from the PCG to contact the childminder in waves 1 and 2 (99, 268) or the schools in wave 3 (266) and interview the child's childminder or teacher about the study child (99, 266, 268). However, as previously mentioned, data collected from carers, teachers and principals are not described here further as this information was not included in this thesis.

3.2.6 Sample size and response rates

Children were selected from the Child Benefit Register as to be nine months old at the time of their first interview, and PCGs of a total of 11,134 infants agreed to take part in the study and completed the interview. The families of 9,793 children were interviewed in the month after the child's third birthday and 9,001 when the children were five years old. A total of 8,712 families completed the interviews in all three waves (78.2%). An overview of sample sizes and response rates can be seen in Figure 6.

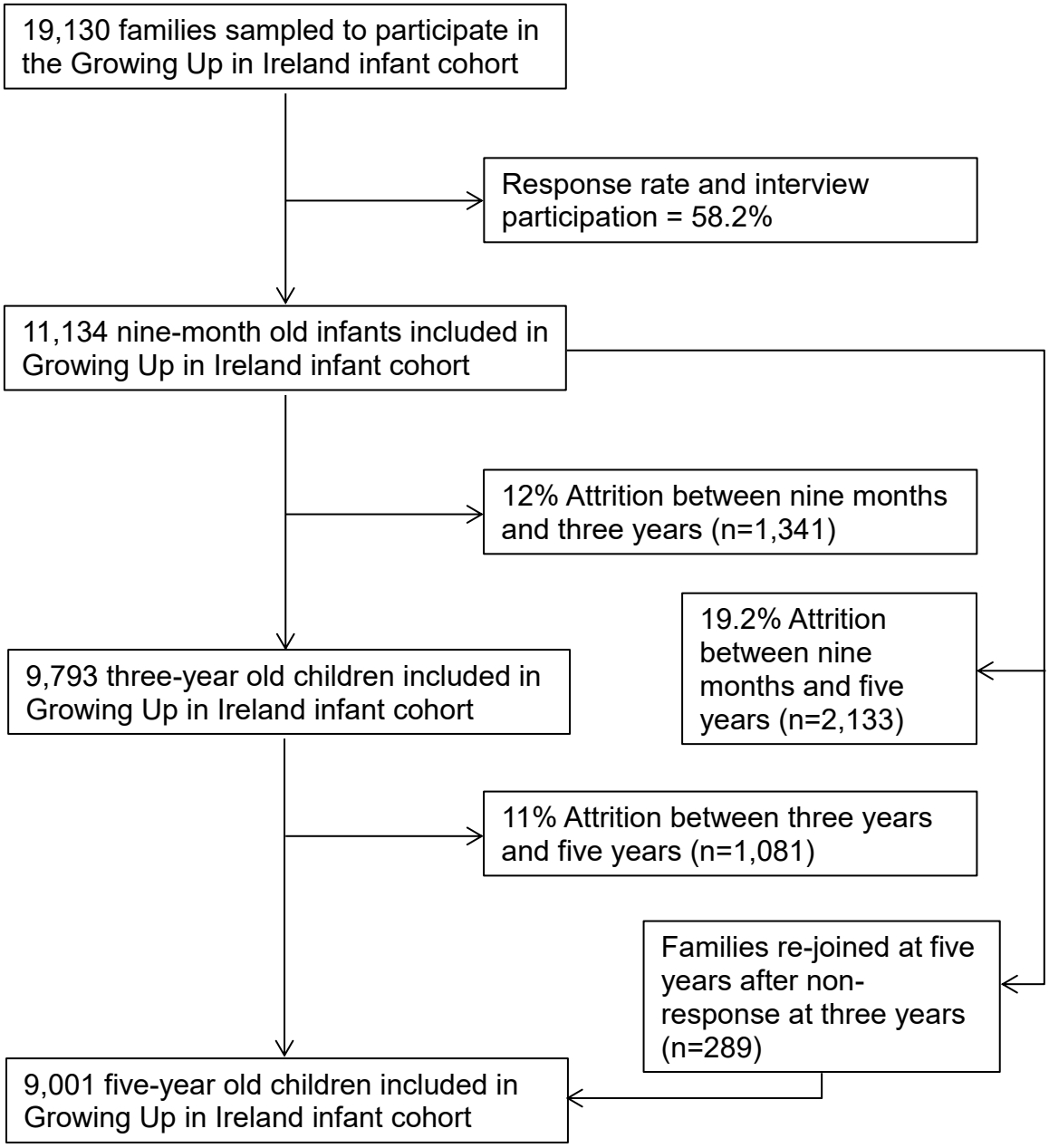

Figure 6: Sample size, response rates and attrition between waves in the Growing Up in Ireland infant cohort

Of all families sampled from the Child Benefit Register, 58.2% (n=11,134) completed the first wave, and 41.8% were excluded for the reasons provided in Table 3. If those families were not taken into account that could not be located or where the child was deceased, a response rate of 64.3% among eligible families was reached (264).

Of the 11,134 families who were interviewed in wave 1, 3.8% were identified as ineligible to participate in wave 2 because the family had moved abroad or the child was deceased (Table 3). In total, 88% (n=9,793) of families were interviewed in wave 2 if the complete wave-1 sample was taken into consideration, and 91.4% of wave-1 families who were eligible for interviews in wave 2. Families in the refusal category (4.4%) were split into ‘hard refusals’, which led to the exclusion of families from the study in future waves, and ‘soft refusals’, which included families who were willing to participate in the study but were ‘too busy’ (e.g. due to the birth of another child during the period of data collection) or otherwise did not complete an interview in wave 2 (e.g. because the families continuously broke appointments). Families with ‘soft refusal’ were kept in the cohort for possible inclusion in wave 3. Despite the loss of some children between waves 1 and 2, the complete sample of the GUI infant cohort in wave 2 still had a high resemblance with the entire population of three-year-old children living in Ireland at that time, indicated by a number of population characteristics captured in the 2011 Census of Population (268).

Furthermore, of the total wave-1 sample, 10,360 families were eligible for an interview in wave 3 and hence approached for interviews. This included all families who had participated in wave 2 and still lived in Ireland in wave 3. Additionally, the eligible wave-3 sample consisted of families who had participated in wave 1, lived outside Ireland or could not be located or contacted in wave 2, but had moved back to Ireland or otherwise informed the GUI study team about their new address since wave 2. Families who were excluded from wave 2 due to ‘soft refusal’ as well were included in the sample of eligible families in wave 3 (266). Of the 10,360 eligible families, 86.9% (n=9,001) completed the interviews and were included in the final wave 3 sample. Compared to the final wave-2 sample (n=9,793), the response rate in wave 3 was 91% and compared to the complete wave 1 sample (n=11,134), it was 80.8%, respectively. Overall, the response rate in wave 3 was higher in families that had completed wave 2 (91%) compared to all eligible families (86.9%) (266).

Table 3: Reasons for non-response between waves in the Growing Up in Ireland infant cohort. Families included in wave 1 were n=11,134, in wave 2 n=9,793 and in wave 3 n=9,001.

Reason for non-response	Non-response in wave 1: % of original sample	Non-response in wave 2: % of wave 1 sample	Non-response in wave 3: % of wave 1 sample
Refusal to take part or complete the interview	23.6%	4.4%	7.5%
No contact established/ address was unknown *	17.1%	3.1%	4.5%
Ineligible (family moved abroad/ child deceased)	0.1%	3.8%	7.0%
Other reason	1.0%	0.7%	0.3%

* Families did not establish contact despite repeated call backs from the study team; address was unknown because the family moved since their registration with the Child Benefit Register and provided no forwarding address; or the address could not be located, was derelict, demolished or vacant

Attrition

Attrition in studies rarely occurs purely on a random basis, but usually varies depending on a number of population characteristics, as well as based on the respondent's satisfaction with the interviewer and the overall interview process (268).

Both between waves 1 and 2 and between waves 2 and 3, higher attrition[4] was observed in families with lower educational levels, lower social classes and in single caregiver families. Furthermore, eligible families were significantly more

[4] The GUI study team conducted binary logistic regression analysis among families from wave 1 who were eligible for an interview in wave 2, and among families from wave 2 who were eligible for an interview in wave 3, to investigate trends in attrition. Details of logistic regression analysis are discussed in Section 3.3.5 of this chapter.

likely to complete an interview in wave 2 if the same interviewer contacted them that had visited their home in wave 1. No significant differences in attrition were seen based on the duration of the wave 1 interview, based on the PCG's self-rated health, between families in rural or urban regions, or between PCGs who were born in Ireland and those that had moved to Ireland at various stages in their lives. Most of the observed differences in attrition were explained by varying rates of refusal or because no contact could be established with families for the second interview (268). Despite minor differences, such as a higher attrition in smokers and in low-income families in wave 3, the general trend of lower response rates in lower SES groups was consistent between waves (266). Differences in attrition based on population characteristics were addressed by statistically reweighting the data during each wave, which is presented as follows.

Weighting

The completed wave-1 sample was statistically weighted on the basis of the external population estimates to ensure that it was wholly representative of all children aged one year or less in Ireland. Information on population characteristics was retrieved through the Central Statistics Office based on the 2006 Census of Population, and the Child Benefit Register, based on the data of all 73,662 births that had been registered for the calendar year 2008. The GROSS programme developed by the Economic and Social Research Institute in 1996 was used, which is based on a minimum information loss algorithm using a regression framework and has been the standard for surveys in Ireland since the time of development. The weighting factor adjusts the sample to the distribution of demographic variables in the population while maintaining the original sample size (4).

Based on the Census of Population, information on the family structure, maternal age, maternal Principal Economic Status, paternal Principal Economic Status, family social class, maternal education, household tenure, region and gender was included in the weights. Based on the Child Benefit Register, information was additionally taken on the maternal marital status, maternal nationality and maternal residency status. Based on these variables, 99 features were defined and their

representation in the original sample of the GUI infant cohort statistically reweighted to match the distribution in the total population. An exact breakdown of these features in the general population and the GUI infant cohort can be found in Quail et al. (264).

As significant differences were observed in the attrition of families based on a number of population characteristics, the sample of three-year-old children included in wave 2 of the GUI infant cohort needed to be statistically reweighted to match the population characteristics of infants in Ireland who were born in the same period of time as the GUI infant cohort, and to match population characteristics of those children aged three years who continued to reside in Ireland or had moved to Ireland since wave 1. Due to the fixed panel design of the study, the latter were not included in the sample in subsequent waves. The GROSS programme was used again for weighting the wave-2 data (268).

The weighting factor generated in wave 2 is a combination of (1) the wave-1 weight, which accounts for population differences due to the design and response in wave 1, (2) overall demographic changes that took place in the Irish population over time and were known from the 2011 Census of Population, and (3) additional population differences introduced to the sample due to the variations in attrition outlined above. The weighting factor in wave 2 hence was constructed on the basis of the same 99 features identified along eleven variables for the wave-1 weight. Additionally, the following variables that were measured in the GUI questionnaires in wave 1 were included in the wave-2 weight: whether or not the child was breastfed, the length of time a family had lived in the local area, rural or urban location of the household, equivalised income quintile of the household, ethnic background of the PCG, hours worked by the PCG, PCG depression status, PCG smoking status and PCG BMI (268).

Similarly, new weighting factors were needed in wave 3, as the attrition between waves 2 and 3 was not random, and as some children from wave 1 did not participate in wave 2 but re-joined the study in wave 3. Weighting factors used in waves 1 and 2 needed to be readjusted to the sample in wave 3 to match the population of five-year-old children living in Ireland in 2013; that means to make it representative of the population of infants born at the same time as the GUI infant

cohort that was still resident in Ireland in wave 3 (estimated n=69,300) (266), taking into consideration those children who moved to Ireland since the original infant cohort was sampled (266).

Two weighting factors were added to the study in wave 3 using the GROSS programme: the five-year weight ‘a’ that is applicable to all children who participated in waves 1 and 3 (n=9,001), and weight ‘b’ that is applicable to children who participated in all three waves (n=8,712). The weighting factors generated in wave 3 are a combination of (1) the wave-1 weight, which accounts for population differences due to the design and response in wave 1, (2) the wave-2 weight, which accounts for variations in attrition since wave 1 and demographic changes in the Irish population known from the 2011 Census of Population, and (3) additional population differences introduced to the sample due to the variations in attrition between waves 2 and 3 (266).

Differences between the sample characteristics for each wave prior and after weighting are small, and details have been published elsewhere (4)(266, 268).

3.2.7 Quality control during and after interviews

In addition to considerations in the study design, sampling and reweighting of the GUI infant cohort data to make them representative on a population level, a number of mechanisms during and after data collection were used to achieve a high quality of the GUI infant cohort data.

Reponses to interview questions, test results and measurements were recorded digitally using Blaise software (267). Most questions were closed questions and hence only accepted answers in predefined ranges and categories. Furthermore, the programme conducted cross-variable consistency checks and automatically coded the answers retrieved to closed questions during the interview. A few open questions were additionally included in the interview and answers to these questions were manually coded into categories after the interview. While these newly-coded categories were included in the GUI infant cohort dataset, the original

verbatim responses were removed to protect personal data of respondents. Data were later checked for errors and edited if needed (99).

Furthermore, the acquisition of data from the second and third wave of data collection allowed for consistency checks of longitudinal data collected from the same family. A number of key variables, such as marital status (268) or educational attainment of caregivers (266), were checked across waves and errors removed. However, the study team notes that not all inconsistencies could be removed from the dataset as it was not always possible to judge which of the data entries was correct (266, 268).

3.2.8 Ethical considerations and data protection

Ethical approval for each wave was granted to GUI by an independent Research Ethics Committee through the Department of Health and Children, which reviewed all materials and procedures that were included in the study (99, 266, 268).

The study was carried out under consideration and adherence to principles outlined in the following guidelines and Acts of the Irish legislation:

- the Children First Guidelines outlined 1999 by the Department of Health and Children
- the Children First: National Guidance for the Protection and Welfare of Children 2011 by the Department of Children and Youth Affairs
- the Data Protection Act 1988
- the Data Protection Act 2003
- the Statistics Act 1993 (99, 266, 268)

This involved the development of information sheets handed to all potential participants and a consent form that needed to be signed by a parent or guardian before data were collected. At any time during the study, participants were allowed to withdraw from the study or access their personal data held by GUI (267, 269). Furthermore, numerous mechanisms were in place to protect the confidentiality of the families, such as the coding of family data and password and encryption

mechanisms that were in place on all laptops that stored GUI data. To protect children during the interview process, interviewers were not allowed to be left alone with the study child and only were allowed minimum physical contact, such as during anthropometric measurements (267, 269).

Interviewers were appointed ‘Officers of Statistics’ under the Statistics Act 1993 to ensure the confidentiality of information retrieved through the families. Furthermore, all staff and interviewers involved in the GUI study were security vetted by An Garda Siochana. An incident report system was in place during the data collection of each wave that operated on a 24/7- basis and could be used by interviewers, in addition to a child welfare and protection protocol that was developed by the GUI study team (99, 266, 268).

In order to protect the anonymity of families, names, dates of birth and open text responses were removed from the dataset. Furthermore, the dataset was available in two files: the Anonymised (AMF) and Researcher (RMF) Microdata Files. The AMF had all other data removed or grouped into fewer categories where there was a possibility to identify families based on very specific or rare characteristics. For numerical ranges, bottom or top coding was used to group outliers together with more frequent values, or continuous scores were grouped into categories all together. Similarly, for categorical variables, different responses that were rarely mentioned in the interviews were grouped into a category named ‘other’ for use in the AMF. Lastly, most information captured in the sensitive questionnaire was removed from the AMF (266, 268).

For use in this thesis, the extended data available in the RMF were considered most suitable to study OWOB, as well as continuous BMI and a number of variables of the children and their families that were specified in less detail in the AMF. For example, the ethnic background of children and measurements of height and weight, both among children and their caregivers, were provided in detail in the RMF, including any potential outliers.

3.3 Methodology for secondary analysis of the Growing Up in Ireland infant cohort

This section focuses on the use and modifications to the RMF of the GUI infant cohort data in this thesis. Following on from the retrieval of the data, a number of variables from the GUI infant cohort datasets were chosen for inclusion in this thesis. Data were taken from all three waves (i.e. when the children were nine months, three years and five years old). In Sections 3.3.2 and 3.3.3, the measurement of various variables as well as specifications of their use on this thesis will be described.

Following from the description of variables and data cleaning, exploratory analysis for all variables will be described in Section 3.3.4. In Section 3.3.5, more advanced statistical analysis used in this thesis will be described.

3.3.1 Data retrieval

Researcher Microdata File application

The application form for acquiring the RMF was completed and submitted to the National Longitudinal Study of Children in Ireland Project Office at the Department of Children and Youth Affairs several months before the data were obtained. Approval of the application was required from the Project Office and from the Director General of the Central Statistics Office (273). Access to the dataset was granted to Officers of Statistics only, appointment of which was subject to the completion of a training session by the Central Statistics Office. Details of the application are set out in the RMF application form, which is available online: https://www.dcya.gov.ie/documents/research/20180815_GUINLSCIRMFAppForm.doc .

3.3.2 Outcome variable – body mass index

BMI can be analysed in various ways, for example it can be categorised based on the use of child growth criteria, or it can be used as a continuous variable, either in the form of raw BMI scores, standard deviation (SD) or z-scores, or as BMI centiles, all of which were considered for this study. As explained in Chapter 2, BMI is not subject to constant growth during childhood, neither is it expected to be constant over time, unlike that of adults. For instance, according to those BMI scores provided for reference in the WHO growth standard and WHO growth reference, the average BMI in girls lies at 16.7 kg/m^2 at nine months of age and at 15.4 and 15.3 kg/m^2 at three and five years, respectively. For the same ages, boys have an average BMI of 17.2, 15.6 and 15.2 kg/m^2 (274, 275).

This implies that it is useful to compare children's growth curves to the distribution of BMI in a reference population, such as that of the WHO growth standard, and position a child's BMI within that distribution. Most commonly, a child's individual BMI would be approximately in the middle of the BMI distribution observed in the reference population, and while the range of sex-specific BMI scores in this distribution may change over time, an individual child would be expected to have a BMI that continuously lies close to the mean or median recorded within this distribution. As BMI tends to be normally distributed, the mean and median of the BMI distribution are interchangeable (274, 275). If a child exceeds a defined sex- and age-specific threshold within the distribution in the growth reference population (that is, the population underlying the respective child growth criterion), the child is considered to be overweight or obese. Because mean BMI values within the reference population differ between the sexes and ages, a standardisation of the reference BMI distribution can be useful when children of different sexes or ages shall be compared. In its standardised form, BMI has the same value range for every age and sex, but the same values correspond to different raw BMI scores in different age groups and sexes.

Continuous use of body mass index data

Two standard conversions to standardise BMI scores are the transformation of raw BMI values into BMI percentiles, or into BMI SD or z-scores, the latter of which are interchangeable. Conversions of BMI can be performed using any underlying BMI distribution, for example that of the population under investigation at a given time point, or that of a comparable reference population which is available in the form of growth references or growth standards.

Percentiles are, as the word indicates, a hundred parts of information given on a population (or sample). In a distribution of BMI, the 50th centile describes the median BMI value, meaning that half of the population will have a BMI below and the other half will have a BMI at or above this value. The 85th centile represents the BMI value that includes only the top 15% of the population based on their BMI above this value (80).

Z-scores are based on the SD of a particular value from the mean. In a normal distribution, 68% of children have BMI scores that fall within a SD of between -1 to 1, 95% have a BMI within -2 to 2 SD, and 99.7% within -3 to 3 SD. Therefore, the closer the BMI z-score of a child is to 0, the closer that means it is to the average of all children in the same sex and age group. Weight, length/height and BMI measurements outside the range of -5 to 5 SD are considered invalid by the WHO as these deviate more from the mean of the respective anthropometric measurements than what is considered realistic variations (275-277).

The standardised nature of BMI z-scores and percentiles offers the advantage that BMI scores can be compared easily between ages and sexes; however the use of standardised BMI also bears a number of disadvantages. First, the use of z-scores or percentiles relies on data from an underlying reference population. Chapter 1 showed that no consensus exists as to which child growth criterion is the most suitable to assess children's weight status, and z-score and percentile distributions of BMI are subject to the same limitations. Secondly, Berkey and Colditz (278) criticise the complexity added to the interpretation of findings, identifying raw BMI scores as the much more meaningful measurement unit of BMI change. Thirdly, various studies indicate that the use of BMI z-scores may lead to imprecise

estimations of change based on explanatory variables, particularly in the higher BMI ranges of severely obese children and especially among young children (278-280). While these studies investigated z-scores underlying the Centers for Disease Control (CDC) growth charts, which are based on the US population (278-280), these limitations appear applicable to other growth criteria including those used in this thesis.

Accordingly, as this thesis seeks to investigate children's BMI and particularly the higher BMI ranges, and after careful consideration of all options, it was decided that raw BMI presents the more suitable continuous outcome variable and therefore was used in this study. As BMI in the GUI infant cohort was assessed at the same ages of children in each wave, raw BMI scores between children are comparable with appropriate analysis. Despite this preference, standardised scores of BMI were used in sensitivity analysis, and also to group children's BMI into weight categories. Both are described in the following sections.

Standardisation of body mass index in the Growing Up in Ireland data

In the GUI infant cohort, trained interviewers took standardised weight and length/height measurements of the children in each round of interviews. This offers major advantages over parental or children's self-reports of weight and length/height, the latter of which are more likely to be biased, due to measurement errors or social desirability of having a 'normal' BMI (12).Weight was assessed using the Class III SECA 835 portable electronic weight scale (99, 266, 268), which measures weight graduated by 20g up to 20kg and by 50g thereafter (267). Length was measured with the SECA 210 measuring mat when the infants were nine months old (99) and height was measured with a Leicester measuring stick at the following ages (266, 268). Both length and height were recorded to the nearest millimetre (267). Raw BMI was calculated dividing the weight in kilograms by the square root of length/height in metres.

To standardise BMI, the raw BMI scores were converted into BMI-for-age z-scores based on the WHO criteria, namely the WHO growth standard at nine months and

three years of age and the WHO growth reference at five years. This conversion was based on the *zanthro* work package, which is an add-on to Stata developed by Vidmar et al. (281) and Vidmar et al. (282), some of whom were also involved in the development of child growth criteria. The *zanthro* package allows users to transform anthropometric measurements of children, such as their weight-for-age, length/height-for-age and BMI-for-age, into z-scores based on various child growth criteria included in the package (282).

There are a number of benefits in using the *zanthro* package in Stata. First, it provides a built-in option to exclude observations that are outside the range of -5 and 5 SD from the mean of the anthropometric measure used.

A second advantage of the *zanthro* package is its main function, the conversion of anthropometric measures into age- and sex-specific z-scores. The values retrieved from anthropometric measurements in the dataset are matched with the reference z-scores, based on the specified growth criterion, and converted into a continuous variable ranging from -5 to 5.

Lastly, the conversion of anthropometric data with *zanthro* does not only specify z-scores adjusted for the age and sex of the child, but additionally offers the option to adjust z-scores for gestational age at birth.

Accordingly, in addition to BMI and age at BMI measurement, the children's gestational age (in weeks) and sex were used to standardise BMI in each wave using the *zanthro* package. For each wave, three variables were generated specifying *BMI-for-age z-scores*, *weight-for-age z-scores* and *length/height-for-age z-scores* based on WHO criteria.

Categorical use of body mass index data

Sex- and age-specific cut-off points for weight categories were available for young children from the WHO growth standard (applicable to ages zero to below five years) (277) and WHO 2007 child growth reference (age five years and older) (114). For both WHO growth criteria, thresholds of weight categories are based on

SD. In addition to the WHO criteria, the International Obesity Task Force (IOTF) growth reference (age two years and older) (283) was used as an alternative criterion. The IOTF growth reference categorises BMI based on defined sex- and age-specific thresholds which correspond to the adult raw BMI sores of 25 and 30 kg/m^2; that is thresholds for overweight and obesity, respectively (Appendix 1).

Chapter 4 will provide more detail about these child growth criteria.

Data cleaning

The cleaning of the outcome variables followed a two-step procedure.

First, all weight, length/height and BMI observations were plotted for each wave individually, in order to identify potentially invalid values and outliers that needed to be removed from the dataset. Furthermore, all weight, length/height and BMI observations outside the range of -5 to 5 SD of WHO criteria were identified using the *zanthro* package and removed (see Appendix 2 for details).

In a second step, changes in weight and changes in length/height within the same child over time were investigated which led to further exclusion of observations. With approximately two years lying between each measurement of a child, both weight and length/height were expected to increase from one wave to the next. To examine changes across waves, three variables were generated that indicated the weight gain between waves 1 and 2, 2 and 3, and 1 and 3, and three variables of length/height gains respectively. Where decreases or only marginal increases were seen based on these variables, all weight or length/height and BMI data for both concerned waves were removed from the dataset as it was unclear during which of the waves measurement or reporting errors had taken place. ‘Marginal increases’ were defined based on outlier analysis and skewness (Appendix 2).

Details of values that were excluded from the dataset at each wave are provided in Appendix 2. Overall, relatively few observations were excluded from the dataset and little or no changes in mean BMI at each wave were seen (Appendix 2).

3.3.3 Explanatory variables

A number of explanatory variables from the GUI infant cohort were included in this thesis to test the second to fifth hypothesis that were outlined in Chapter 2. The choice of variables was primarily driven by the SEM, the *Complex web of potential determinants of overweight and obesity in children*, and the literature review in Chapter 2, which will be referred to for more detail on each of the respective variables presented below. The full questionnaires can be accessed on the GUI website (284). Detailed information on item development and tools used in the three infant cohort waves is provided elsewhere (266, 267, 269).

Table 4 summarises the various explanatory variables that were included on different levels of the SEM and that were investigated in relation to the outcome variables (discussed in Section 3.3.2).

Table 4: Inclusion of risk factors of childhood overweight and obesity and associated population characteristics in this thesis. Population characteristics and risk factors are structured according to the levels of the Social Ecological Model (SEM) based on Reed et al. (8)

Level of the SEM	Population characteristics and risk factors of childhood overweight and obesity
Individual level	Genetic factors: ü sex Lifestyle of the child: ü diet: number of food recommendations met ü frequency of unstructured physical activity ü frequency of sports club use ü daily screen time ü antibiotic courses in the past twelve months ü daily sleep duration
Parenting styles & family characteristics	Parental characteristics: ü primary caregiver weight status ü secondary caregiver weight status Birth outcomes and early growth: ü gestational age ü birth-weight-for-gestational-age ü rapid infant weight gain Infant feeding: ü breastfeeding initiation and duration ü timing of introduction to solid foods Family structure: ü household structure ü primary caregiver stress
Community, demographic & societal characteristics	Socio-economic status of families: ü household class ü equivalised household income ü highest education of caregivers Ethnic background: ü ethnic background of caregiver(s) Characteristics of the neighbourhood families live in: ü region Parental perceptions of the neighbourhood: ü neighbourhood perceived as safe ü neighbourhood perceived to offer green and play spaces Non-parental childcare environment ü formal or informal childcare arrangements

Explanatory variables on the child level

In the following section, various explanatory variables on the individual child level of the SEM will be described that were measured in the GUI infant cohort and were included in this thesis. This includes the development of composite scores that were generated to assess the children's dietary intake and physical activity, in addition to other mainly behavioural factors.

Sex

The sex of the children was recorded as 'boy' or 'girl' in wave 1. There were no missing data.

Diet composite score

The children's diet was assessed in waves 2 and 3. In wave 2, 14 food items were measured using an adapted version of the Sallis Amherst Questionnaire based on a 24-hour recall by the PCG (269). This excluded essential food groups (such as main foods rich in carbohydrates and protein rich foods other than dairy) and the PCG was asked to indicate whether the item was consumed 'Not at all', 'Once' or 'More than once' on that day. No indication was provided whether the food recall referred to a weekend or week day, which limits the validity of the data (285), in addition to the fact that food intake in 24 hours may not represent a child's usual consumption (286). In wave 3, based on the Food Frequency Questionnaire developed by the School of Public Health, Physiotherapy and Population Science in University College Dublin (266), 53 food items were measured that represent a wide range of foods and food groups. The PCG was asked to indicate whether the child ate each item 'Never', 'Less than once per month', 'At least once per month', 'At least once per week', 'Most days', 'Daily', '2-3 times per day', '4-5 times per day' or '6 times per day or more often'. Parental assessment in this age group is recommended (285). While food frequency questionnaires (as opposed to food

quantity questionnaires) may be inaccurate on the individual level (285), they can capture overall consumption patterns of children on population level (286). Alternative approaches such as food weighing and prospective food records (285) were not feasible in GUI due to its broad scope and scale (266). Given the various measures used and the lack of information in waves 1 and 2, for this thesis it was decided to use the dietary data from wave 3 as a proxy for overall family diet that would most likely determine the child's earlier diet as well. Dietary patterns were previously found to track through children's early ages (180, 287).

As a part of this thesis, a food composite score was generated based on the 53 food items measured in wave 3. Other studies used food composite scores to assess the dietary quality of children in a simplified way (287-289). Cluster analysis or principal component analysis are commonly used to group individual food items into consumption patterns (*a-posteriori* approach). These data-driven techniques frequently split diets into an 'unhealthy pattern' characterised by a high proportion of processed food, fast food and items typical to a western diet, and a predominantly 'healthy pattern' that is in compliance with food guidelines (180, 287, 290, 291). Accordingly, no benefit of using clustering techniques over hypothesis-driven (*a-priori*) approaches was seen (289). The 53 items measured in the GUI infant cohort were therefore grouped into food groups *a-priori* based on similarities in their (macro-) nutrient profile to generate a food composite score. A full list of the 53 food items is provided in Appendix 3.1.

Food items were grouped together according to the food groups listed in the 2016 food pyramid, which is applicable to children aged five years and older (292). Similarly, the Healthy Eating Index was previously used as a composite diet quality index in the US, Greece and Portugal (288, 293). The measurement scale used by GUI was converted into consumption frequencies per month; that means a numerical value was given to each of the nine original categories, as can be seen in Table 5. This was necessary to sum the consumption frequencies of different food items in a group. A similar approach was previously taken in a study by Lioret et al. (287), in which the scale was converted into frequencies per week. Consumption frequency quartiles were considered as an alternative to quantify the

consumption per food group as has been done in a previous study (293), however this is less precise when food items are grouped together.

Table 5: Conversion scale used in this thesis for food consumption labels assessed in the Primary Caregiver Main Questionnaire in wave 3

Original label in the Growing Up in Ireland study	Approximate frequency of consumption per month
Never	0
Less than once per month	0.5
At least once per month	2
At least once per week	10
Most days	20
Once daily	30
2-3 times per day	75
4-5 times per day	135
6 times per day or more	165

The food pyramid recommendations suggest that children should eat five to seven servings of fruit and vegetables per day, three to five servings of foods rich in carbohydrates, such as grains or potatoes but excluding sugar, three servings of dairy products, two servings of protein, with limited or no amounts of processed meats, and two small servings of spreads and oils. No minimum recommended consumption of sweet and salty snacks, including fizzy drinks, exists, and therefore these items should be consumed less than every day, according to the food pyramid guidelines (292). Furthermore, items from various of these food groups were added into a seventh food group ‘processed food’ which is not mentioned in the food pyramid; however it was identified in a number of previous studies and appears relevant to the study of childhood OWOB (187, 287).

Food groups, as well as individual food items were tested for collinearity. No correlation was seen between either the seven food groups or the 53 food items ($-0.5 < r < 0.5$). Descriptive statistics of the individual food groups revealed that children in Ireland tend to over-consume, rather than under-consume food groups

in relation to food pyramid recommendations. Between 0% and 4% of children consumed less than what was recommended in each of the seven food groups, as opposed to between 8% and 93% of children who consumed more than what was recommended in the respective food groups. Further detail is provided in Appendix 3.1. For fat intakes, this is in line with findings from the Irish National Preschool Nutrition Survey 2011-2012, in which 25% of 500 preschool-aged children exceeded fat intake recommendations. Additionally, a high consumption of sweet and salty snacks was observed, similar to the GUI infant cohort. However, children in this survey commonly did not consume the recommended minimum amount of fruit and vegetables (294), whereas recommendations were met in the GUI infant cohort and in young children from different European countries in another study (295). Similar to other European cohorts, it is possible that the PCG in GUI over-reported the intake of fruit and vegetables due to social desirability bias (296).

Based on the food pyramid recommendations, the score of each food group, that means the total estimated consumption frequency of food items in that food group per month, was categorised as contributing to a high or low quality diet. Two approaches were taken into consideration for this. First, the ‘Physical activity, diet and lifestyle’ (PDL) scoring system suggested by Manios et al. (297) applies a maximum score of four to children if they are meeting a food recommendation, a score of zero for the lowest dietary quality, and scores of one to three based on how much children deviate from the dietary recommendation. These standardised scores of all food groups can be added and a continuous scale generated to indicate overall dietary quality. However, the PDL score appeared unsuitable for this study as, once food groups are merged into one continuous scale, it is unclear whether scores in the middle range occur due to over- or under-consumption of certain food groups. Over-consumption, in the PDL scoring system, is valued higher than under-consumption (297) which makes the interpretation and translation of findings into prevention strategies for OWOB nearly impossible.

Therefore, a second, simplified approach was used to combine the food groups, similar to the Mediterranean Diet Score that was used in young children in a European cohort study (286). A continuous scale was generated with values ranging from zero to seven that indicate how many of the seven food

recommendations a child is meeting or almost meeting. It was decided to include children in this who slightly over- or under-consumed food items because PCGs in GUI only self-reported the frequency, but not portion sizes, of food items. Therefore, it was not possible to differentiate whether children precisely met the recommendations or deviated slightly. In contrast, on population level, this can be delineated from a very high or very low consumption of food groups. While the risk of noise cannot be eliminated from the continuous composite score entirely, this approach appeared most applicable to generate an aggregate measure of the food frequency questionnaire that would reflect the dietary quality of children on a population level without adding much complexity to the analysis. Detailed information on the scoring is provided in Appendix 3.1.

Physical activity

Sports club membership

Data on physical activity levels of children were only collected in wave 3 of the GUI infant cohort and are split between organised sports and unstructured active play. Organised sports use was assessed by asking PCGs how often the child attends a sports club, grouped into ‘Never’, ‘Twice a month’, ‘Regularly, one hour per week’, ‘Regularly, two hours per week’ and ‘Regularly, more than two hours per week’.

This variable was adopted in this thesis without modifications, as all values appeared valid and only a low potential for social desirability bias was seen. Bias or measurement errors were not expected much as sports clubs tend to be used on a paid membership basis.

Unstructured physical activity composite score

To assess unstructured physical activity levels of children, PCGs were asked whether their child engaged in a number of activities categorised as ‘Climbing on trees, climbing frames, wall bars, etc.’, ‘Playing with a ball’, ‘Playing chasing’, ‘Riding a bike, tricycle or scooter’ and ‘Skating’. Similar to the aggregation of food

items, the development of a composite score appeared reasonable. Most composite scores in previous studies of young children tend to focus on physical fitness and motor skill development, rather than physical activity levels (298, 299), or they integrate physical activity levels with other lifestyle items such as diet and screen time (297, 300). Therefore, similar to the a-priori approach used for the dietary quality score, the frequencies of unstructured physical activity were added together and a continuous composite score generated to study individual items in synergy. A similar approach was used by Hinkley et al. (301).

For each unstructured physical activity item, the PCG was asked to indicate whether the child pursues the activity ‘Never’, ‘Less than once per week’, ‘Once or twice per week’, ‘Three to six times per week’, or ‘Every day'. These frequencies were converted into events per week, similar to the conversion of food consumption in monthly frequencies for the food composite score. That means numeric values were given to each of the original categories as can be seen in Table 6.

Table 6: Conversion scale used in this thesis for unstructured physical activity labels assessed in the Primary Caregiver Main Questionnaire in wave 3

Original label in the Growing Up in Ireland study	**Approximate frequency of the activity per week**
Never	0
Less than once per week	0.5
1-2 times per week	1.5
3-6 times per week	4.5
every day	7
Don’t know	Missing

A composite score was generated by calculating the average frequency per week of the five different activities and rounding it to the nearest full number, thereby retrieving a continuous variable that ranged from zero to seven. However, few children were grouped in the values zero (n=52) and seven (n=140), which is why children scoring zero on this composite variable were grouped together with those

children scoring one, and children originally scoring seven were grouped together with those scoring six. Details of this can be seen in Appendix 3.2.

It was considered to reduce the complexity of the data further by integrating the frequency of sports club use (presented above) in the composite score. However, the potential for measurement error or bias appeared much higher in the assessment of various unstructured physical activities, compared to sports club use, which is why the two variables were kept separate. In some children, error was expected if children were minded by someone other than the PCG, and it is possible that some PCGs over-reported the frequency of unstructured physical activity due to social desirability bias. The number of daily step counts using pedometer data would have been preferable to assess physical activity levels more objectively; however this was not feasible as the administrative burden and cost of this would have been beyond scope for the GUI infant cohort study that aimed to collect data from a multitude of domains of child development.

Screen time

Screen time was recorded in waves 2 and 3 of the GUI infant cohort. In wave 2, the PCG was asked how many minutes the child spent watching TV on an average day. In wave 3, a wider definition related to ‘screen time’ was used and the PCG was asked how much time the child spends with screen-based devices. As few children used no screens at all, a category ‘none to less than one hour’ per day was used to indicate little use. Furthermore, children were grouped as using screens ‘one to less than two hours’ daily if they were within or just above recommendations, or ‘two to less than three hours’ and ‘three or more hours’ which indicates that they are regularly exceeding recommendations (209).

It is possible that the total amount of screen time may be underestimated for some children due to social desirability bias introduced by the PCG, or if children use screens when minded by somebody other than the PCG. However, this study seeks to assess an approximate level of screen time, in other words, to distinguish between low, normal and high use and to investigate trends in screen time use on the population level, rather than in the individual child. While minor levels of bias

can occur in the lower categories that need to be acknowledged when interpreting findings, the variable is understood to reflect overall population trends.

Antibiotic intake

In waves 2 and 3, the PCG indicated how many courses of antibiotics the child received in the twelve months prior to the interview. This was used as a continuous variable in the analysis as no thresholds are known that link a defined dose to OWOB outcomes. Children with equal or more than 20 courses of antibiotics were grouped as having received 20 courses in the past twelve months (n=12 in wave 2, n=14 in wave 3). The retrospective assessment of a twelve-month period may induce some level of recall bias; however the PCG is expected to remember approximate levels because General Practitioners (GPs) and pharmacists tend to clearly label medicines as antibiotics. While this limitation needs to be acknowledged, the exact level of error cannot be assessed and adjusted for in the study as medical records were not available.

Sleep

The child's sleep duration, including daytime sleep, was recorded from the PCG in each wave. For this thesis, the GUI data were used to group children's sleep at the respective ages as below recommendations, within recommendations, or above the recommended hours of daily sleep, based on recommendations by the National Sleep Foundation (217). Less than 200 children in GUI had a recorded sleep duration that fell in the lowest category at three years of age and in the lowest and highest category at five years, therefore cut-off values were altered for use in the analyses to achieve meaningful and statistically relevant results: For nine-months-old children, the original categories according to the sleep recommendations were kept. At three years, children were grouped into sleeping 'less than eleven', 'eleven to under 13', or '13 or more' hours per day. At their age of five years, children were grouped into sleep categories of 'less than eleven', 'eleven to under twelve', and 'twelve or more hours per day'. The differences in counts in each category are detailed in Appendix 3.3.

Parent-reported sleeping times of infants may not always be accurate, compared to more sophisticated tools such as wrist actigraphy. However, population-level studies use parental reports as a standard and observed associations with OWOB in young children regardless of the assessment tool used (219).

Explanatory variables on the family and parenting level

Various explanatory variables from the GUI infant cohort on the family and parenting level of the SEM were included in this thesis, which are described as follows.

Caregiver weight status

The weight of the PCG and SCG, if available, was assessed during each interview using the Class III medically approved SECA 761 flat mechanical weight scale. Self-reported weight was assessed at each wave, in addition to measured weight, and both were compared to test for measurement or reporting errors if the measured weight appeared extremely high or low.

Height was measured in wave 1 using a standard Leicester portable height stick and measurements were fed forward to subsequent waves. Height was only assessed in waves 2 or 3 if a new person participated in the PCG or SCG interview or if the information from previous interviews appeared faulty during the data cleaning of previous waves by the GUI study team. For example, validity was tested using the self-reported height which the caregivers were asked to provide during the interview. If large discrepancies between measured and self-reported height were observed, height was measured again in the following wave if the same adult was available for an interview (266). Among those PCGs who were still the PCG later in the study, 1,143 had their height re-assessed in wave 2 and 153 in wave 3. Of those SCGs who were still the SCG in later waves, 1,089 had their height reassessed and changed in wave 2, and 377 in wave 3. Furthermore, 68

new height measurements were adopted that had been measured in initial PCGs who participated in the study as the SCG in a later wave, and vice versa.

For PCGs and SCGs, BMI was calculated and classified as normal or underweight if their BMI was below 25, those with a BMI of between 25 and below 30 were classified as overweight and those with a BMI of 30 or higher were classified as obese (94, 302) in line with international cut-offs (Appendix 1).

A separate 'No SCG interview' category was created for the weight status of the SCG, to avoid exclusion of children from the analysis where data were missing because no such interview had taken place (n=1,143 in wave 1). Furthermore, observations from 1,359 SCGs (wave 1) were missing because only one caregiver lived in the household. The majority of single caregivers have never worked, are 16 to 24 years old of Irish white origin, and were never married (data not shown). Accordingly, a category 'Lone caregiver household' was added to the variable that indicates the SCG's weight status, in order to control for potential confounding and avoid loss of data in children in lone caregiver households. Furthermore, to address the potential for confounding, sensitivity analysis with the sub-sample of two-caregiver households was run, which will be described in Chapter 6.

Gestational age

The gestational age of children at birth was collected from the PCG in the first GUI interview and recorded in weeks. It was classified as 'very early' (before 33 weeks), 'somewhat early' (after 33 to 36 weeks), 'on time' (after 37 to 41 weeks) and 'late birth' (after 42 weeks or later) (303). Most children were born after between 38 and 42 weeks and no clear outliers were seen despite lower counts in the higher and lower gestational ages (less than 25 observations were seen in each week of gestation from 23 to 31 weeks, as well as after 45 and 46 weeks).

No bias was expected as most parents remember well the duration of the gestational period, or whether their child was born prematurely, on term or late (304). In particular nine months after birth, no recall bias is expected (304), and no risk of any other bias or measurement error seen.

Birth-weight-for-gestational-age

Birth weight was self-reported by the PCG in wave 1 (see Appendix 3.4 for descriptive statistics). The recall of birth weight nine months after birth was evaluated as very reliable in a comparable British cohort (305).

The variable BWFGA was generated, using thresholds available from the German Perinatal Survey 1995-2000. These are based on a dataset with standardised anthropometric data and gestational ages of 2.3 million singleton births in Germany that were retrieved from hospitals in 1995 to 2000 (306). Anthropometric measurements were taken by doctors, nurses or midwives after birth and documented for use in the dataset of the German Perinatal Survey. Means and SD specific for the sexes and gestational ages were derived from the dataset and percentiles of BWFGA calculated (307) and smoothed using polynomial regression (306). The dataset of the German Perinatal Survey 1995-2000 underlying the German classification of BWFGA offers the advantage of a high sample size that can hardly be reached within such a short time frame in Ireland or other western European countries due the annual number of births in these countries (on average, 600,000 singleton births were included in each of the first three years of the German Perinatal Survey (307), which is more than eight times as many births as those registered in Ireland in 2007 (264)). The advantage of using hospital data from one single country is that standardised measurement practices are expected to reduce the rate of measurement error and high internal validity can be expected within the data. Furthermore, while birth weight is known to vary between ethnic groups, it is fairly stable across Caucasian children, which is why the dataset from Germany is understood to offer sufficient external validity to births around the same time period in Ireland.

Separate percentile values were available for boys and girls, and values were adjusted for gestational age in weekly intervals, to categorise the continuous birth weight variable as SGA, AGA or LGA. Like in other studies, SGA was defined as BWFGA < 10th percentile and LGA as BWFGA > 90th percentile (118, 149).

Rapid infant weight gain

A variable to measure rapid infant weight gain was generated based on the weight of children at birth and at the age of nine months (both assessed in wave 1). Ong et al. (308) suggest that rapid infant weight gain can be defined as a gain in weight that is more than 0.67 z-scores above the mean weight gain, a definition that has been adopted in many studies previously (309). Z-scores of weight at birth and at nine months were calculated as:

ά ύ Ώ Ώ ώ ύ Ώ Ώ ζ ά Ώ ώ ύ Ώ Ώ ΤΎ Ό ύ Ώ Ώ

where ά ύ Ώ Ώ is the z-score of a child's weight at a given age, ύ Ώ Ώ is their crude weight in kilogram, ά Ώ ώ ύ Ώ Ώ is the mean weight in kilogram of all children who participated in wave 1 of the GUI infant cohort, and ΎΌ ύ Ώ Ώ is the SD of weight in kilogram in the cohort at wave 1. Based on the mean and SD retrieved from the cohort, at birth, this corresponded to:

ά ύ Ώ Ώ ύ Ώ ζ σ φ υ σ ρ ω π π υ ρ χ

Furthermore, the individual z-score at nine months of age was calculated as:

ά ύ Ώ Ώ ύ Ώ ζ ω φ π υ ρ φ σ ρ φ ω

Following from this, the z-score of weight gain between birth and nine months was calculated:

ά ύ Ώ Ώ ά ύ Ώ Ώ ζ ά ύ Ώ Ώ

, where ά ύ Ώ Ώ describes the z-score of weight gain, that means how much weight a child gained proportionally to others. ά ύ Ώ Ώ, as well as ά ύ Ώ Ώ and ά ύ Ώ Ώ all were distributed normally.

Finally, the binary variable 'rapid weight gain' was calculated as:

ύ Ώ Ώ ά ύ Ώ Ώ χ

, where ύ Ὼ῎ΏὯΏ ῴ῎Ὼ ὲ defines rapid weight gain, depending on the child’s individual z-score of weight gain.

Breastfeeding initiation and duration

The PCG was asked whether the study child has ever been breastfed, and, if applicable, how long the child had been breastfed, measured in days. Additionally, the PCG was asked whether and how long the study child had been exclusively breastfed. However, the understanding of exclusive breastfeeding is likely to differ between parents and may not conform to clinical definitions (168). Particularly in Ireland, where breastfeeding rates have traditionally been low and still are much below those in other countries (310), no clear distinction between exclusive and non-exclusive breastfeeding may be made by caregivers. Therefore, overall (non-exclusive) breastfeeding onset and duration appeared as the more reliable variable and was used in this study.

Children were grouped as those not breastfed at all, those breastfed much shorter than recommended (below three months), those breastfed between three and less than six months, and those breastfed six months or longer. The latest category is in line with recommendations by the WHO (175) and includes 1,040 infants who were still breastfed when the wave 1 data were collected.

Timing of introduction to solid foods

In addition to information on breastfeeding, the PCG was asked in wave 1 at what age in days the study child had eaten solid food for the first time. For use in this study, the continuous variable was split into three groups: ‘before the age of four months’, ‘between the ages of four to below six months’, and ‘at the age of six months or later’. The threshold of four months was chosen as this is the earliest recommended introduction to solid food (267) and the threshold of six months corresponds to the minimum duration of six months of exclusive breastfeeding that

is recommended for infants by the WHO (175). The latter category includes 70 infants who had not been introduced to solid food yet at the age of nine months.

While there is a risk that the exact age in days may not always be remembered correctly by the PCG, the impact of possible recall bias was expected to be low when the ages were grouped in a three-fold category.

Household structure

Family structure was grouped into two binomial variables, one expressing whether the child had siblings at each wave, and the second expressing whether the household was a single- or two-caregiver household. Both variables were combined to one composite measure of family structure, categorising families as ‘one child with one caregiver’, ‘one child with two caregivers’, ‘two or more children with one caregiver’ and ‘two or more children with two caregivers’. Valid values were observed for all families and no potential reason for bias was seen. Cross-variable consistency checks were conducted by GUI to maximise validity.

Primary caregiver stress

In each wave, a sub-set of the Parental Stress Scale by Berry and Jones (311) was used. Topics assessed on the Parental Stress Scale include Parental Rewards, Parental Stressors, Lack of Control, and Parental Satisfaction. A total of 18 items are measured, all of which are rated on a five-point Likert scale which ranges from ‘strongly disagree’ to ‘strongly agree’. In wave 1, all sub-scales were assessed in the PCG interview and retained for analysis in this study, while only a subset of items were assessed in subsequent waves. As a result, only the Parental Stressors sub-scale (six items) was analysed in waves 2 and 3 in this study, which means that the wave 1 score ranges from 18 to 90, compared to a range from 6 to 30 in waves 2 and 3.

Overall, the scale was evaluated to have satisfactory internal reliability and test-retest-reliability, as well as satisfactory construct *(convergent)* validity levels. This

means that test results were found reproducible in the same parents in studies prior GUI, and that parents of children with developmental or behavioural problems were distinguishable from those of typically developing children, based on scores retrieved from the scale (311). With regards to the scales used in GUI, however, it should be noted that the assessment of PCG stress was part of the main questionnaire in wave 1 and part of the sensitive questionnaires in wave 2 and 3. As a number of PCGs did not fill in the sensitive questionnaires in wave 2 (n=87) and wave 3 (n=148), the proportion of missing values for PCG stress is higher in these waves. Furthermore, it was assumed that caregivers were more reluctant to answer the interview questions honestly in the main interview, compared to the self-administered sensitive interview, which may limit the reliability of the wave 1 score. Even though a GUI report from the dress rehearsals found a moderate correlation between scores in waves 1 and 2 (r=0.58) (269) the findings across waves should be compared and interpreted with caution. Nevertheless, acknowledging these limitations, PCG stress was included in the analysis to test psychosocial pathways of childhood obesity as no more reliable data were available that were indicative of self-perceived stress.

Explanatory variables on the community, demographic and societal level

Additional variables were included in this thesis that were measured in the GUI infant cohort on the community, demographic and societal level of the SEM. These are described as follows.

Household class

In each wave, the GUI study team assessed social class based on the occupation of the PCG and, if applicable, the SCG. This referred to the current occupation or, if caregivers were unemployed or retired, their previous occupation. Occupations were grouped in seven ranked categories according to the International Standard Classification of Occupations 1988. In families where both a PCG and SCG lived in

the household, the higher ranking occupational category was used as a household proxy for social class, according to the dominance criterion procedure. Based on this classification, families were assigned to the following social class groups: ‘Professional workers’, ‘Managerial and technical’, ‘Non-manual’, ‘Skilled manual’, ‘Semi-skilled’, ‘Unskilled’, ‘All other gainfully occupied and unknown’, and ‘Unclassified’, the latter of which referred to families in which all caregivers had never been economically active.

While the social class of families was likely to correlate with the equivalised household income or the educational achievement of caregivers, it appeared reasonable to take alternative measures of SES into consideration in the analysis of this thesis, rather than to choose a random SES scheme. This was advocated by Braveman et al. (312), and is related to the idea that different SES indicators may influence the risk of childhood obesity via different pathways; for example education may influence childhood obesity through health literacy, income through the affordability of health resources and social class through lifestyles and shared value systems (313).

Equivalised household income

During each of the GUI interviews, the PCG was asked to indicate the total household income, including social welfare payments. The total household income was then equivalised to the household size and composition to make the income of smaller and larger families comparable. Different weights were applied for household members, namely the PCG was weighted as one adult, each additional adult and adolescent over 13 years who lived in the household was given the weight of 0.66 adults, and children aged 13 years or less were given the weight of 0.33 adults. The weighted number of household members was summed and the total disposable income in the household divided by the summed number of adult equivalents. The total disposable income was the sum of all forms of gross income in the household with income tax and social insurance contributions deducted.

The equivalised household income was then divided into ranked quintiles of income. In this thesis, differences in OWOB or BMI outcomes were primarily

expected in the lowest, compared to the highest income groups, whereas no such trend may be seen in the middle range of income. To test this, the analysis of quintiles offers better comparability of the lowest and highest income group than the continuous variable. Furthermore, a number of outliers were seen at the high end of the income distribution which appeared valid but may have confounded the analysis (see Appendix 3.5 for details). To test whether material circumstances may explain children's susceptibility to OWOB, the ordinal variable of equivalised income quintiles was therefore used in this study. Moreover, to prevent the loss of data, a category 'missing' was added as 8% of families had no income reported.

While the risk of reporting bias cannot be fully avoided, this was estimated to be small as no incentive was seen for PCGs to over- or under-report the family's income, given the possibility of non-reporting that 8% of PCGs availed of. However, it needs to be acknowledged that the cost of living differs across geographic areas of Ireland, for example discrepancies in housing expenses exist between Dublin and other cities, as well as between cities and rural areas. Unfortunately, no information was available that indicated the proportion of the income that families spent on living expenses; hence the predictive validity of the disposable income to assess the discretionary budget of families could not be tested. Nevertheless, the weighting of income to household size and composition accounts for much of the variation in expenses that would be expected between families, which makes this variable suitable to investigate the material theory.

Highest education of caregivers

The highest level of educational attainment of the PCG and the SCG, if applicable, was recorded in each wave. Various educational levels were grouped into the four categories of 'primary or lower secondary', 'higher secondary', 'post-secondary' and 'tertiary' education. For use in this thesis, the highest educational attainment of the PCG and SCG were combined to a variable that indicated the highest level of education in the household of either the PCG or SCG. Thus, where no SCG interview took place and in lone caregiver households, the highest educational level of the PCG was used. The educational attainment of caregivers was

combined in order to reduce the complexity of the analysis, but also because it was assumed that the caregiver with the higher education would influence the weight-related behaviour not only of the study child, but as well of other household members including the other caregiver. Similar to the assumption that the highest occupational category in the household impacts the lifestyle of the entire family, also the highest level of education in the household is expected to influence the health literacy (and hence the weight-related behaviour) of families (313). While a more refined grouping of educational attainment categories (in eleven to 13 categories) was considered, no advantage of this over the four-fold categorisation was expected, particularly certain categories had very few values. Sensitivity analysis with more categories confirmed this notion.

Ethnic background

The ethnicity of both the PCG and the SCG, if applicable, was collected in each wave. Most ethnic minorities were grouped to protect their anonymity, which split the caregivers into ‘Irish’ and six other groups, as can be seen in Table 7.

While the ethnicity of parents may influence the child’s growth and weight gain through biological pathways, also cultural value systems and traditions inherent to ethnic groups may influence the weight-related behaviours of families, which is why GUI reported ethnicity for each caregiver separately, rather than for children directly. For this study, analysis was initially conducted using the separate PCG and SCG ethnicities; however this appeared of little use to test biological pathways. When the association with childhood obesity is studied both biological and cultural implications of ethnicity should be considered (236). Furthermore, as can be seen in Table 7, most SCGs belonged to the same ethnic group as the PCG of the child (a correlation of r=0.62). The discriminate validity between PCG and SCG ethnicity was low.

Table 7: Distribution of ethnicity among secondary caregivers (SCGs) by the ethnicity of primary caregivers (PCGs)

PCG ethnicity	N SCG ethnicity (%)							
	Irish	**Any other white**	**African**	**Any other black**	**Chinese**	**Any other Asian**	**Other incl. mixed**	**Total**
Irish	6,313 (92.80%)	426 (34.83%)	20 (8.93%)	6 (25.00%)	4 (11.76%)	25 (11.01%)	18 (38.30%)	6,812 (79.38%)
Any other white	414 (6.09%)	785 (64.19%)	15 (6.70%)	0	2 (5.88%)	8 (3.52%)	9 (19.15%)	1,233 (14.37%)
African	7 (0.10%)	2 (0.16%)	179 (79.91%)	1 (4.17%)	0	1 (0.44%)	2 (4.26%)	192 (2.24%)
Any other Black	3 (0.04%)	1 (0.08%)	5 (2.23%)	17 (70.83%)	0	1 (0.44%)	1 (2.13%)	28 (0.33%)
Chinese	16 (0.24%)	2 (0.16%)	0	0	27 (79.41%)	0	0	45 (0.52%)
Any other Asian	32 (0.47%)	2 (0.16%)	2 (0.89%)	0	1 (2.94%)	188 (82.82%)	1 (2.13%)	226 (2.63%)
Other incl. mixed	18 (0.26%)	5 (0.41%)	3 (1.34%)	0	0	4 (1.76%)	16 (34.04%)	46 (0.54%)
Total	6,803 (100%)	1,223 (100%)	224 (100%)	24 (100%)	34 (100%)	227 (100%)	47 (100%)	8,582 (100%)

Note: The orange fields indicate that the PCG and SCG belong to the same ethnic group.

N = Number

For these reasons, the ethnicity of the PCG and the SCG, if applicable, were combined to assess the ethnic background of the child. The ethnic background from data collected in wave 1 was carried forward, as only seven PCGs (0.06%) and 15 SCGs (0.15%) were not the biological parents of the study child in wave 1. A different person could take on the PCG or SCG roles in later interviews, and a larger proportion of PCGs (0.18%) and SCGs (1.5%) were not the biological parent of the child by the time the wave 3 data were collected, which is why the wave 1 data offered the best opportunity to test biological pathways and was used throughout the analysis. Where only one caregiver lived in the household, the ethnic background of the caregiver was used as this appeared the dominant influence on children, for example in terms of child feeding. Additionally, a separate category was used for children with two caregivers but where only the ethnicity of one was known from the interviews.

Region

In each interview wave, the PCG was asked to describe whether the family lived in the open country, a village, towns of various sizes, major cities or Dublin county. The responses were grouped as ‘rural’ for families who lived in the open country, a village, or a town with less than 10,000 inhabitants. Otherwise, they were grouped in the category ‘urban’.

Parental perceptions of the neighbourhood

In each wave, the PCG was asked to agree or disagree to a number of statements about their neighbourhood that were measured on a four-point Likert scale ranging from ‘strongly agree’ to ‘strongly disagree’. Two statements that were assessed in every wave were included in this thesis, first, “This is a safe neighbourhood”, and second, “There are good parks, playgrounds and play spaces”.

The first item was chosen to assess how the overall quality of the neighbourhood and community was perceived by the PCG, which may have implications on the psychosocial stress of families. The perceived level of neighbourhood safety may not reflect the real level of safety, based on standardised measures such as road traffic intensity and crime rates. However, the perception of caregivers was expected to determine their behaviour and stress levels more than a measured level of safety that may be miss-perceived by caregivers.

The second item was used to measure the family’s perceived availability of facilities to pursue outdoor activities, such as sports and active play. Again, this variable may not reflect the real availability of green spaces and outdoor play areas, but the perceived availability is expected to influence the willingness of families to use available facilities. Some caregivers may perceive the availability as insufficient due to the proximity to their family home, or because they don’t find the green space sufficiently clean or family-friendly. Such factors cannot be taken into account when the geographic locations of family homes and green and play spaces are compared. Overall, the perceived availability appeared as a useful construct to measure promoters of healthy family lifestyles in the neighbourhood.

Childcare arrangements

In each wave of data collection, PCGs were asked whether their child is regularly minded by someone other than the PCG or SCG outside regular school and preschool hours for more than eight hours per week. If their child fell within this definition of being in a childcare arrangement, the PCG was asked to specify whether the child was minded by ‘a relative in the relative’s home’, ‘a relative in the child’s home’, a non-relative in their home’, ‘a non-relative in the child’s home’, ‘in a childcare institution’ (e.g. a crèche), or in any other arrangement classified as ‘other’. Furthermore, for five-year-old children, ‘after school service care within school’ was added as a possible answer. Responses are detailed in Appendix 3.6.

To integrate these variables into this thesis, a three-fold composite measure of childcare was created for each wave, splitting children into those in no care arrangement, those in formal care, that means ‘in a childcare institution’ or ‘after school service care’ within a school, and those in informal childcare. The latter constitutes all other forms of childcare including the ‘other’ category.

The measurement of this variable has a number of limitations that needed to be addressed in this study. First, the assessed type of childcare arrangement referred to the main arrangement in place, whereas theoretically a child could have been in formal and informal care regularly. This limitation remains in the study as most children were expected to be minded by relatives or neighbours occasionally, and the benefit of controlling the study for this did not justify the added complexity.

Secondly, the childcare arrangement in place was specified as ‘regularly’ for more than eight hours per week. As a regular arrangement could have started shortly before the GUI data were collected or soon after children were born and maintained throughout their early childhood, the age at onset of childcare was considered for inclusion in the analysis. However, as many children tended to start childcare at the same ages (Appendix 3.6), no need was seen to include this.

Lastly, a regular childcare arrangement was defined to be used ‘eight hours or more’, which can possibly refer to one or up to seven days a week. While the exact number of weekly hours was assessed in the GUI interviews, no benefit was seen to include this as many children were not in childcare. Hence, a continuous

variable would have been highly skewed (if children not in care were coded as ‘zero’ hours) or would have had a high number of missing cases for children not in childcare. Alternatively, the use of a binary or ordinal variable with different cut-off values appeared similarly arbitrary like the eight-hour threshold suggested by GUI. Moreover, a higher threshold (for a binary variable) may have shifted children into the ‘no childcare’ category who in fact were in a childcare arrangement. Even during eight hours per week, children were perceived likely to eat meals and pursue sedentary or physical activities that varied from habits in the family home. Thus, the original threshold of eight hours was maintained for use in this study.

Additional data which were excluded from this thesis

In addition to the data described throughout this chapter, a number of variables and qualitative data were considered for inclusion in this thesis, but excluded from the analysis. These data and reasons for their exclusion are described as follows.

Explanatory variables

A number of additional variables were considered for inclusion, based on the evidence in the literature presented in Chapter 2. Most of these variables refer to the pregnancy period, whereas the data collection in GUI started when infants were nine months old. Accordingly, variables were excluded from the analysis either because no data were available from GUI or because measurement errors or confounding of these self-reported data were likely, as described below.

While both small and large birth weight or BWFGA are commonly and strongly related to OWOB outcomes in children (see Section 2.3.1), BMI-for-age at birth was considered as an alternative indicator of birth size. Unfortunately, more than half of the records of length at birth were implausible or missing. Consequently, BMI at birth could not be studied.

Inclusion of caregiver depression status was considered, in addition to caregiver stress; however there was moderate correlation between these variables in wave 3. As caregiver stress and mental health are expected to influence children’s

weight outcomes through similar mechanisms (see Section 2.3.3 for details), depression status was excluded from the analysis.

Furthermore, a number of gestational variables were excluded, mainly because each of these variables individually is strongly linked to birth weight, which was included as an independent variable in this thesis, and their inclusion would have confounded the analysis. Additional reasons specific to the individual variables are detailed as follows.

The weight status of caregivers at the onset of pregnancy was considered for inclusion; however the GUI study did not assess BMI in caregivers retrospectively. Instead, caregiver weight status when children were nine months old was used as the earliest indication of weight status.

Maternal gestational weight gain was also considered for inclusion in the analysis, but was excluded for two reasons. First, gestational weight gain was self-reported by caregivers and had a high proportion of missing values (14%) and an unrealistic data range (between zero and 66kg). Multiple imputation of missing values was considered. However, the nature of the variable itself is prone to reporting bias and the accuracy of values could not be assessed, which would have limited the reliability of imputed values. Secondly, internationally adopted recommendations by the Institute of Medicine define healthy gestational weight gain relative to women's weight status at the onset of pregnancy (48, 127). This was impossible in the GUI infant cohort as maternal weight status before pregnancy is unknown.

Furthermore, after consideration GDM was not included in the analysis. The children in the GUI infant cohort were born in 2007/2008, at which time only women evaluated to have a high-risk pregnancy were screened for GDM in Ireland (314). Therefore, GDM was likely to be undiagnosed and hence underrepresented in GUI. Furthermore, this means that most women reporting GDM must have had additional risk factors which categorised them to have a high-risk pregnancy. These risk factors may have confounded the analysis; however as families in GUI were not asked whether the pregnancy with the study child had been a high-risk pregnancy, the possibility of unmeasured confounding could not be tested. Furthermore, pre-eclampsia was excluded from the analysis for similar reasons.

Lastly, gestational smoking and alcohol consumption were measured in the GUI infant cohort, but excluded from this study after careful consideration. Both variables were understood not only to measure consumption patterns during pregnancy, but most likely to indicate the families' overall health literacy and lifestyle. The risk of residual confounding appeared high, especially as health literacy was not directly measured in GUI and this association could hence not be tested. Additionally, the risk of reporting bias was high.

On the level of societal, demographic and community factors, no data were collected by GUI that indicated how caregivers perceived the food environment in their neighbourhood. A request to the GUI study team revealed that no small area population statistics were available for the infant cohort (315), hence the measured proximity to (healthy) food outlets could not be used as a proxy. However, to a point this information may be captured in the regional classification, with higher availability and choice of nearby food outlets expected in urban regions.

Alternative variables to assess the family's SES, such as those based on poverty scales, were considered. For example, families were asked about their ability to make ends meet, and about their ability to afford certain basic and luxury goods. The SES indicators chosen for inclusion in this study (as described above) appeared most instructive as they are frequently assessed and studied in other surveys nationally and internationally. They hence have the highest potential for comparison. Furthermore, possible high-risk groups in the Irish population defined by alternative poverty scales are believed to be difficult to reach through interventions and policies if these poverty scales are compared to less complex or less sensitive indicators such as occupation and education.

Qualitative data

Initially, a total of 122 qualitative interviews were also received from GUI through an application to the Irish Qualitative Data Archive. Interviews had been conducted with a sub-sample of families from the quantitative cohort that represented the quantitative cohort as well as the Irish population of infants below one year of age, based on a number of socio-demographic characteristics. Interviews took place in

2009, when the infants were nine to eleven months old. Interview questions touched upon the areas of 1) Perception of the baby, 2) Baby's routine and habits, 3) Parent and child relationship, 4) Service utilisation, 5) Transition to parenthood, 6) Perception of being a parent, 7) Decision-making and social support, 8) Community and neighbourhood, 9) Work/life balance, 10) Childcare, and 11) Current and future concerns and aspirations for [the] baby (316). The interviews were planned to be analysed thematically, performing a text search and coding the interviews inductively based on occurring topics and statements related to health, healthcare utilisation, health promotion and weight. However, after preliminary inductive coding of 15 interviews, it became clear that little weight-related information was gathered through the interview questions. In fact, the main related topic surrounded issues of breastfeeding and public health support during the first days after the child was born, but these findings had already been published by Growing Up in Ireland NLSCI (317). Furthermore, no linkage of the qualitative data to quantitative records of the GUI infant cohort for sub-sample analysis is possible (318), hence the qualitative analysis of the GUI infant cohort excluded from this thesis.

3.3.4 Data cleaning of explanatory variables and exploratory analysis

As with any large dataset, a high amount of data cleaning was undertaken, in addition to the cleaning that the GUI study team conducted (Section 3.2.7).

This involved the removal of outliers, invalid and extreme data values. All continuous variables were tested for outliers using boxplots. Outliers were recorded and assessed based on comparable value ranges found in other studies published in relevant fields. Outliers which are likely to be related to reporting errors were deleted from the dataset and recorded. Invalid values within each variable were identified by exploring the value range of possible categories. Values which can clearly be attributed to a missing values or other category were recoded into the appropriate category. Extreme data values seen in the continuous variables were removed or the variable was categorised so that individual

observations could not confound the analysis. Detailed numbers and reasons for the exclusion of observations from the dataset can be seen in Appendix 3.7.

Missing value analysis

Missing values were investigated for patterns of non-reporting to assess whether they were missing at random. Variables relating to the SCG were examined separately. Categories were generated for values that were missing (1) because the SCG was not interviewed, or (2) because the child lived in a household with only one caregiver.

Multiple imputation was considered for replacing missing values in the dataset, in order to include as many observations as possible and prevent loss of information and statistical power. However, as the proportion of missing values in each variable was small (<10%), this was not necessary (319). Instead, a ‘missing’ category was created for variables where the exclusion of missing values would significantly reduce the sample size. The percentage of missing values was 0-2% for most variables and 2-3% in breastfeeding duration, rapid infant weight gain and onset of solid foods. Higher levels of missing values were found among caregiver BMI (4-5%) and the equivalised household income (almost 8%). Details for each explanatory variable are presented in Chapter 5.

Exploratory analysis

Means and SD for continuous variables and frequencies and proportions for categorical variables at each wave were reported (details can be seen in Chapter 5). Additional analysis was conducted to investigate trends if deemed appropriate.

Continuous data were tested for normality by plotting the data and comparing means, medians and inter-quartile ranges. For continuous scales which evidenced to show some skew, a median and inter-quartile is presented (Chapter 5).

Relationships among variables were tabulated and investigated using a variety of suitable methods (bar charts and line graphs, Spearman and Pearson's correlation, chi-squared tests and t-tests). P-values < 0.2 were used as a threshold to highlight potential univariate associations between the primary outcomes with all other variables. Collinearity was investigated using the threshold of -0.8 > r > 0.8. The highest Spearman correlations of between r= -0.53 and r= -0.61 were seen between household class and equivalised income quintile, and between household class and caregiver education in each wave (results not shown).

McNemar's test was used to assess the significance of changes over time and p<0.05 deemed significant. Bonferroni-corrections were used to adjust p-values for multiple comparisons.

3.3.5 Epidemiological analysis and inferential statistics

Various analysis approaches were chosen to test the research hypotheses and reach the aims and objectives of this thesis (described in Section 1.4). Initially descriptive statistics were used to describe the participants and prevalence (Chapter 4), secondly cross-sectional analysis at nine months, three years and five years of age was conducted (Chapter 5) and finally, a longitudinal analysis was undertaken, looking at repeated measures over time (Chapter 6). Details of the analysis and sensitivity analyses will be presented in the respective results chapters. All data cleaning and statistical analysis, including the analysis described in Chapters 4, 5 and 6, was conducted using Stata version 13 (320). The *Strengthening the Reporting of Observational Studies* (STROBE) guidelines were used for reporting of the methods and statistical results (321).

3.4 Summary

This chapter provided the methodological background to the GUI infant cohort data that were used for secondary analysis in this thesis to pursue the research aims detailed in Chapter 1. The GUI National Longitudinal Study of Children provides

longitudinal BMI data of approximately 11,000 children at their ages of nine months, three years and five years. A number of variables on the individual child level, family and parenting level, and the community, demographic and societal level were also assessed at the respective ages and were adopted and modified for use in this thesis to test the study hypotheses. Despite a number of limitations seen in the study design and data, the representative nature and large sample size are clear benefits of using the dataset and hold promise to reach the aim of this thesis, namely to investigate OWOB in early childhood in Ireland.

CHAPTER 4:

Prevalence of overweight and obesity in young children – an analysis of child growth criteria and data from the Growing Up in Ireland infant cohort

Key points in this chapter

Aims of this chapter

- To critically analyse and identify child growth criteria suitable for young children
- To describe OWOB prevalence in children at their ages of nine months, three years and five years in a young cohort in Ireland

Knowledge generation

- Different child growth criteria will be identified and compared based on existing literature. Recommendations for use in children in Ireland will be discussed.
- Longitudinal data from a nationally representative population sample in Ireland will be used to determine OWOB prevalence among 11,000 young children at their ages of nine months, three years and five years. Estimates of OWOB prevalence in young children in Ireland will be presented for boys and girls, separately, and differences based on varying child growth criteria explored

4.1 Introduction

While in the past, undernutrition posed a major risk towards population health, today it is obesity that causes multi-morbidity and pre-mature death in adults and children alike (14) (for more details see Chapter 1). However, consistent and up-to-date surveillance studies on country level in Europe are missing in the age group of zero to five years (12, 79). In order to control what has recently been called the 'obesity epidemic', prevalence needs to be monitored, changes identified and trends examined in relation to both risk factors exposures and the implementation of intervention strategies (55, 75, 79). While it is suggested that OWOB levels in this young age group are high and therefore need to be tackled in most European countries, the situation in Ireland, as in many other countries, remains unclear (79). It is hypothesised that the OWOB prevalence among young children in Ireland reaches similarly worrying levels like those in other countries and age groups. However, this needs to be tested using recent surveillance data that are representative of all children in Ireland.

4.1.1 Measurement of weight status in young children

The most common way to determine a healthy weight status is by calculating the individual BMI, which was already introduced in the earlier chapters. National or international population-level data can be used to study a child's weight, height or BMI in relation to other children of the same sex and age, thereby matching it to children with a similar growth profile in the reference population. Thresholds for children to fall into a healthy weight category are usually expressed in z-score deviations from the mean, or based on a defined threshold percentile within the child growth criterion, depending on which criterion is being used (322). A distinction in the use of child growth criteria exists between BMI measurements used for individual health assessments in clinical practice, and those used for population monitoring. Criteria used in clinical practice need to identify individual children who are at an increased risk of ill-health because of their excess weight or adiposity and require interventions. Varying growth patterns were observed across countries, which is why criteria used in clinical practice need to be population-

specific in order to be relevant for respective health risks (37, 323). In addition to this, for population monitoring, child growth criteria need to be used consistently over time and should be comparable across countries, which is why consensus is needed as to which criterion to use (79).

While there are other means of measuring OWOB in children, such as waist and hip circumference, or percentages of body fat and fat-free mass, a number of arguments promote the use of BMI for population monitoring over those. Clear advantages of BMI over other methods are its simplicity and cost-effectiveness. Weighing scales and height sticks are widely available for use in households, or measures can be taken by healthcare providers and in pharmacies, sports institutions or schools. In contrast, assessments of fat mass or percentage of body fat (measuring skinfold thickness or using computerised tomography or dual-energy x-ray absorptiometry) are very complex and expensive and therefore not applicable on population level (324). Additionally, available cheap means to assess body composition, such as measurements of waist circumference or waist-to-height ratio, were not superior to BMI in identifying a high percentage of body fat or a high health risk in children and adolescents (115, 325-328).

Furthermore, while measurement error using BMI is possible, this can be limited by using standardised equipment, weighing infants naked and children in light clothing, and removing children's shoes to assess height (113, 116, 117). Therefore, standardisation of BMI measurement is relatively easy to achieve, compared to other anthropometric measurements, such as waist circumference, where correct location of the waist can only be ensured through repeated measurements (329).

Another advantage of BMI is the high level of evidence available that relates BMI in children to later weight status and health outcomes. A synthesis of this was presented in Chapter 1. Other cost-effective and simple tools, such as bioelectrical impedance analysis, have not yet found much application on population level compared to BMI (330) and may not reveal accurate estimates of weight status in the highest BMI ranges (331), which makes them less useful for population monitoring. This includes the wide applicability of BMI to children from different countries. Despite small differences that may have implications for the clinical

assessment of individual children (37, 323), BMI was found to be distributed similarly across healthy children from different geographic regions (114). In turn, larger discrepancies were found between the percentile values of children in different countries based on other anthropometric measurements, such as skinfold thickness, waist circumference, waist-to-hip-ratio and body fat mass (332).

Lastly, the use of BMI to assess children's weight status is preferential to many other methods due to a high level of understanding of weight and height measurements among parents (333). Therefore, the majority of studies that assess child weight status use BMI, which provides a good basis for cross-country and longitudinal comparisons (10, 115, 322).

Taking into consideration all of the above arguments, the use of BMI to determine child weight status has been evaluated as a good tool to estimate approximate levels of body fat (115, 116, 322, 323) and identify health risk (10, 322, 334). Nonetheless, the availability of various child growth criteria adds complexity to the assessment of OWOB levels based on BMI in children, and a lack of consensus exists as to which criterion is the most appropriate. While the sex- and age-specific growth reference suggested by the IOTF seems to have been the most widely used criterion for many years, now criteria with alternative cut-off values are increasingly used. For example, the two criteria introduced by the WHO jointly cover a bigger age span than the IOTF growth reference. Other popular growth criteria are that of the CDC, which is derived from a reference population in the US, and the UK90, which is based on a British reference population. More country-specific criteria based on regional or national assessments of children and adolescents are in use (322), such as the Kromeyer-Hausschild percentiles in Germany (335), those of the Italian Society for Pediatric Endocrinology and Diabetology (336), or national criteria in France, Belgium, the Netherlands (164) and Brazil (63). Caution is needed when results from different studies are interpreted, as particularly in young children, classifications of weight status as well as trends between boys and girls can vary widely depending on which child growth criterion is used (12, 79, 239, 322, 337). This raises the question of how obesity and OWOB are and should be defined in this age group. No single Gold Standard appears to exist, which makes prevalences across countries difficult to compare

(79) and which raises a need to study estimates of OWOB in young children using different growth criteria.

To provide a context in which to place the Irish prevalence of childhood OWOB, a summary of international and Irish surveillance data is presented as follows.

4.1.2 Prevalence and trends of childhood overweight and obesity

Two major international studies published global and country-specific estimates of the recent OWOB prevalence and trends over the last quarter-century (see Appendix 4 for details). Both studies conducted pooled analyses of numerous transnational, national and sub-national studies and surveys from up to 200 countries that provided representative data on BMI or OWOB. This includes several studies from Ireland with data from adults as well as children and adolescents (12, 15) (see Appendix 5 for details). However, to date, no database seems to exist which summarises all data for Ireland, Europe, or globally in the youngest age group of children from birth until five years of age (79). While the Global Burden of Disease Study offers prevalence estimates for children starting from the age of two years in 195 countries, including Ireland, actual data underlying these estimates in Ireland are based on only one dataset studying four to 16-year-olds and dating back to the years of 2001 to 2002 (Appendix 5). Irish estimates below four years hence were generated based on age-specific trends observed in other countries. The NCD Risk Factor Collaboration study furthermore provides prevalence levels only starting from the age of five years (Appendix 5). One global study in children from birth until the age of five years was identified, however it only provides limited data for developed countries including Europe but excluding Ireland (18). Nevertheless, findings of these and smaller studies will be summarised in the following paragraphs, for example trends of the OWOB prevalence based on the Global Burden of Disease Study 2015 (5) as well as OWOB projections are shown in Figures 7 and 8 (p. 155).

International and European prevalence and trends over time

In the age group of up to five years, OWOB was found to have increased globally, from 4.2% in 1990 to 6.7% in 2010, and a faster increase was seen after 2000 than in previous decades. Additionally, more children were at risk of overweight (Table 8). Estimates were *higher* using the IOTF growth reference based on which one in six young children had OWOB in 2015, as can be seen in Figures 7 and 8.

The prevalence of OWOB in young children in developed countries was estimated to lie much above the global average in 2010 (Table 8). The validity of these estimates, however, is limited as data were available for less than two thirds of developed countries in 2010 (18) and still only for approximately two thirds of European countries in 2017 (79). Furthermore, a large proportion (one third) of weight-related data had been collected mainly to study undernutrition, which is why little detail was provided on OWOB levels in some of the studies (79).

Table 8: Global prevalence of weight status in children aged zero to five years. Prevalence was estimated using the World Health Organization growth standard.

Countries/regions	% at risk of overweight	% overweight or obese
Global	14.3%	6.7%
Developed countries	21.4%	11.7%

Source: de Onis et al. (18)

While the age group until five years appears understudied and data often out of date (18, 79), findings in older children support the notion that the prevalence of OWOB is high. From 1975 to 2016, obesity levels from the age of five to 19 years increased from 0.8% to 6.7% globally (using the WHO child growth reference), and an additional 11.6% of children and adolescents were estimated to be overweight in 2016 (12). Unlike the difference between criteria in young children, slightly *lower* levels were found in two- to 19-year-old children and adolescents when the IOTF definition was used (15) (Figures 7 and 8). As can be seen in Figures 7 and 8, also for Irish children, adolescents and adults, prevalence appears much above the global average, which suggests high OWOB levels also in the youngest children.

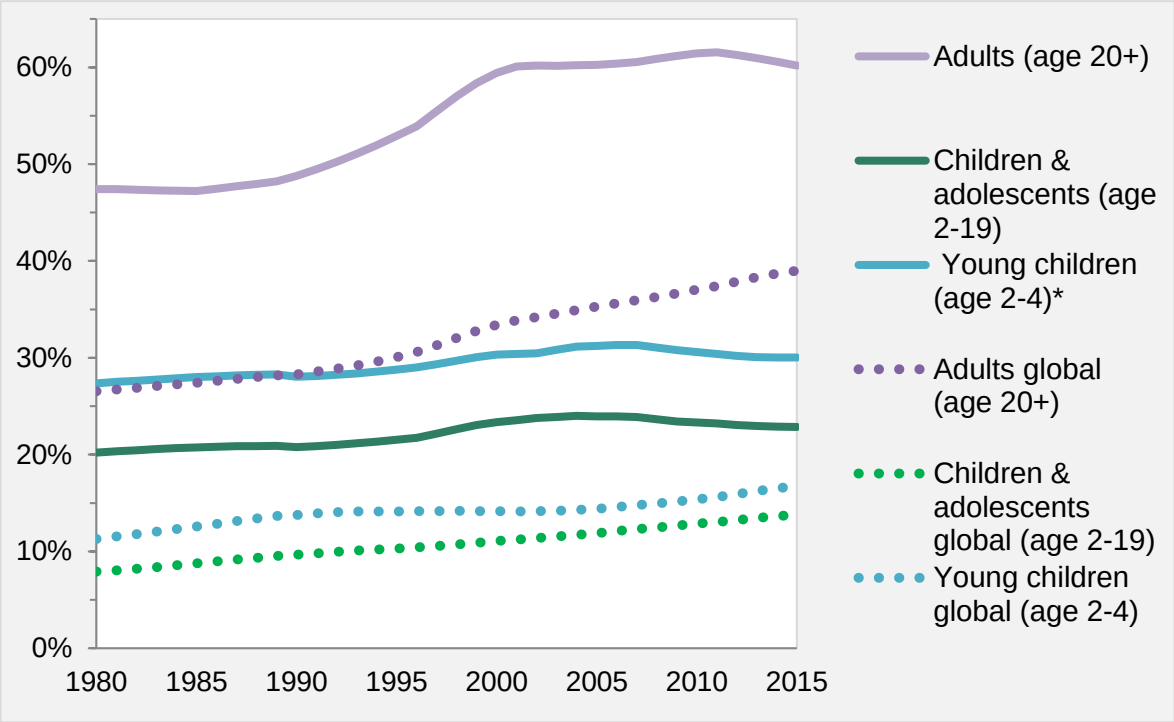

Figure 7: Estimates of the joint overweight and obesity prevalence in Ireland.

Note: Global estimates are provided for comparison. Prevalence in children is based on criteria by the International Obesity Task Force. Data were retrieved from the Global Burden of Disease Study (5). *Estimates not based on real observations

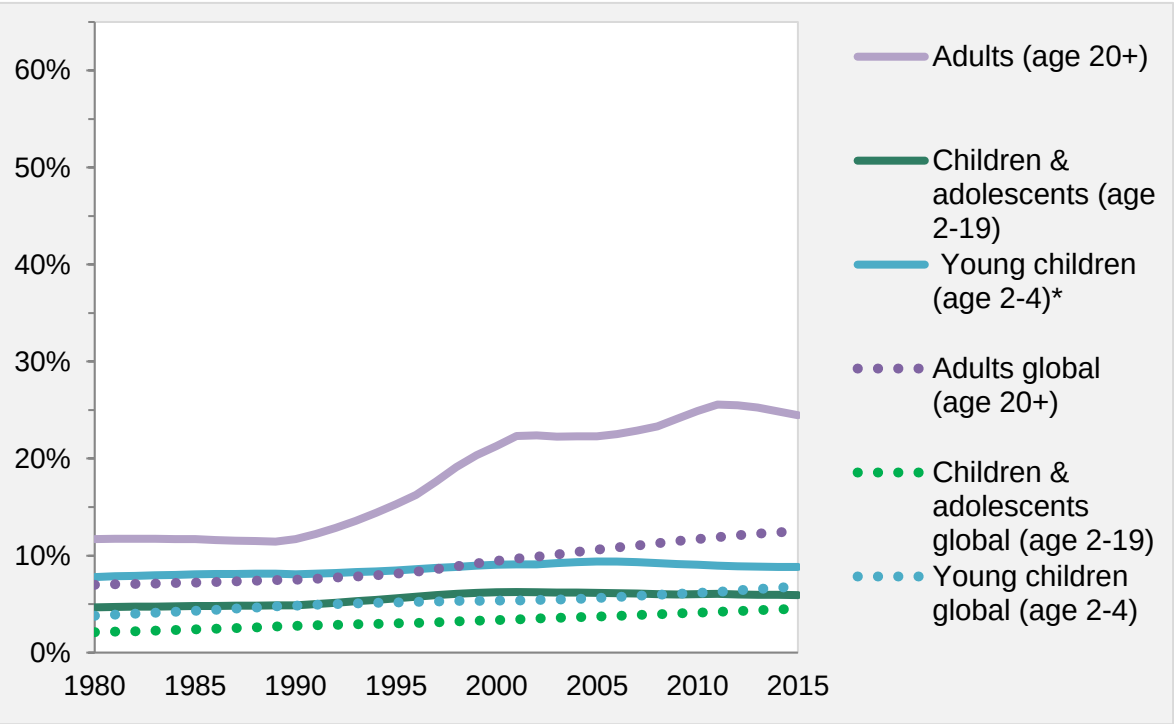

Figure 8: Estimates of the obesity prevalence in Ireland.

Note: Global estimates are provided for comparison. Prevalence in children is based on criteria by the International Obesity Task Force. Data were retrieved from the Global Burden of Disease Study (5). *Estimates not based on real observations

In their review of European OWOB prevalence in young children, Jones et al. (79) identified large differences in the measurement of OWOB between and within countries, making comparisons between geographic regions and over time a challenge. Consistently measured and hence comparable data were found more in higher age groups, such as those collected in repeated survey waves and cohort studies. For example, the *Childhood Obesity Surveillance Initiative* (COSI) collects cross-sectional and longitudinal surveillance data from school children in fifteen European countries, including Ireland. Since 2007, new cohorts of six-year-old children are sampled bi-annually and followed longitudinally (41, 338). The *Identification and prevention of Dietary- and lifestyle-induced health EFfects In Children and infantS* (IDEFICS) study furthermore collected data from children aged two to eleven years living in eight European countries (excluding Ireland), which also commenced in 2007 (239). Both cohort studies follow standardised measurement procedures to assess weight status longitudinally in representative and large samples of children, thereby reducing the risk of bias (239, 338).

Results from IDEFICS and the COSI showed that significant differences in the OWOB prevalence exist between European countries, with higher levels in southern than in northern Europe (239, 338), opposite adult geographic trends (22). Furthermore, over a two-year period, it could be shown that the prevalence of OWOB among studied children decreased in a few countries that had started off with high BMI values while countries with low mean BMI scores at baseline appeared to catch up and follow an increasing prevalence trend, whereas changes in Ireland were inconclusive (338).

Prevalence and trends in Ireland

An overview of studies and surveys conducted among children and adolescents in Ireland is provided in Table 9. While no national longitudinal collection of anthropometric data took place in Ireland until 2007, a number of nationally representative surveys with large sample sizes reported weight and height across past decades, many of which were subject to aggregate analysis in reviews previously.

In 1948, shortly after the Second World War had ended and when Europeans were commonly suffering from malnutrition, the National Nutrition Survey reported data for nearly 15,000 children between four and 14 years. Furthermore, during the 1970s, approximately 3,500 children of the same age span were additionally measured. In 2002, 17,500 children and adolescents took part in the North South Survey of Children's Oral Health (36), height and weight data of which were also included in the Global Burden of Disease study (Appendix 5).

A comparison of the trends visible within these data found that height, weight and BMI had overall increased in boys and girls of all ages from four to 14 years (36). This is an expectable trend considering that post-war Ireland had experienced high economic gains, in particular during the era of the Celtic Tiger in the 1990s until mid-2000s. While the country as a whole prospered, an increase in height among children most likely reflects better nutrition and healthcare, better housing conditions and lower levels of pollution, compared to 1948. Similar trends were seen in other countries that experienced a comparably positive economic development (36).

However, particularly after the 1970s, weight increased more rapidly than it had before, and it was increasing relatively more than height. Consequently, as BMI is a ratio of weight and height, mean BMI increased from 17 kg/m² in 1948 to 21 kg/m² in 2002 in boys and from 18 kg/m² to 22 kg/m² in girls, which corresponds to findings of international studies around that time (36). The two smaller Irish National Nutrition Survey (n=148) and National Children's Food Survey (n=596) furthermore indicated that increases in mean BMI between 1990 and 2003/2004 were more drastic than those before 1990 (36, 37).

More studies were conducted in Ireland after the millennium (Table 9) and Keane et al. (38) summarised and evaluated the data available for four- to 13-year-old children between the years 2002 and 2012 in a systematic review. Combining the studies included in the systematic review, no time trends were found in the prevalence of overweight. However, acknowledging the variety and limitations of studies, the authors conclude that there may have been a decrease in the prevalence of obesity from 7% to 4%, comparing nationally representative studies that were undertaken prior 2008 to those conducted in or after 2008 (38).

Table 9: Studies and surveys that collected weight and length/height data from children and adolescents in Ireland

Data collection	Data source	Represen-tation	Age range	N	Design
1948	National Nutrition Survey (36)	National	4-14	14,835	Cross-sectional
1970s	Measurements for an Irish growth reference (36)	National	4-14	3,509	Cross-sectional
1990	Irish National Nutrition Survey (37)	National	8-12	148	Cross-sectional
1998	HBSC (339)	National	13, 15	826	Cross-sectional
2001-2002	North/South Survey of Children's Oral Health (36, 340, 341)	National	4-16	17,518	Cross-sectional
2001-2002	McMaster et al. (38, 342)	Regional	4.2-7.9	328	Retrospective cohort
2003	Switch Off-Get Active intervention (38, 343)	Regional	9-11	312	Baseline data from an intervention study
2003-2004	National Children Food Survey (37, 344)	National	5-12	596	Cross-sectional
2004-2006	Health Service Executive West surveillance (345)	Regional	6	N.A.	Cross-sectional
2005-2006	National Teens Food Survey (344)	National	13-17	440	Cross-sectional
2004-2007	Senior infants school health check (38, 346)	Regional	6-6.9	3,482	Cross-sectional
2007	Barron et al., 2009 (38, 347)	Regional	6-9	301	Cross-sectional
2007	ROLO study (345, 348)	Regional	5	570	Observations from an intervention study
2007-2008	GUI child cohort (38, 349)	National	9-9.9	8,136	Baseline data from a prospective cohort

Data collection	Data source	Represen-tation	Age range	N	Design
2007-2008	Lifeways Cross-Generational Cohort Study (38, 350)	Regional	5-7	529	Cross-sectional analysis from a prospective cohort
2008	COSI Round 1 (38, 344)	National	7-7.9	2,420	Baseline data from a prospective cohort
2008	Study on pedometer step count (38, 351)	Regional	6-9	301	Cross-sectional
2008-2009	Fitzgerald, 2010 (38)	Regional	9-12	204	Cross-sectional
2009	Observations from the Health Service Executive Meath (38)	Regional	11-13	1,468	Cross-sectional
2009	Healthy Schools Programme (38, 97)	Regional	4-12	537	Baseline data from a prospective cohort
2010	Bizzy Break intervention study (344, 352)	Community	7-12	90	Baseline data from an intervention study
2010	COSI Round 2 (38, 344)	National	6-9	1,022	Baseline data from a prospective cohort
2010-2011	National Preschool Nutrition Survey (98)	National	2-4	371	Cross-sectional
2007-2012	GUI child cohort (353)	National	9-13	8570	Longitudinal analysis of a prospective cohort

Data collection	Data source	Represen-tation	Age range	N	Design
2008-2013	GUI infant cohort (2, 3, 238)	National	0.7-5	11,134	Cross-sectional analysis of a prospective cohort
2012-2013	COSI Round 3 (38, 344)	National	7-7.7	1,087	Baseline data from a prospective cohort
2012-2013	Cork Children's Lifestyle Study (38, 354)	Regional	8-11	1,068	Cross-sectional
2015	Active Classroom Study (344, 355)	Community	8-11	244	Baseline data from an intervention study
2008-2015	COSI Rounds 1-4 (41, 356)	National	6-13	9,911	Cross-sectional and longitudinal analysis of four cohorts
2013-2016	Project Spraoi (344, 357)	Community	5-11	903	Baseline data of an intervention study
2017-2018	National Children's Food Survey II (358)	National	5-12	N.A.	Cross-sectional
2008-2018	Cork BASELINE Birth Cohort Study (40, 359)	Regional	0-5	2,137	Prospective cohort

N = Number

N.A. = Not available because the number of observations was not published at the time of writing

HBSC = Health Behaviour in School-aged Children

COSI = Childhood Obesity Surveillance Initiative

GUI = Growing Up in Ireland study

Based on the various studies, estimates of childhood OWOB prevalence in Ireland since 1990 are presented in Table 10. Prevalence is only reported using the IOTF growth reference as this was available in many studies, and aggregate findings are reported where available. As can be seen, prevalence of overweight and of obesity increased until shortly after the millennium, whereas it appeared to remain more constant thereafter. The possibility that the prevalence of childhood OWOB combined may have decreased in Ireland in the last decade, as suggested by Keane et al. (38), is further indicated by Irish longitudinal data from the COSI in children aged six to eleven years (41, 345) (Table 10).

Table 10: Prevalence of overweight and obesity in Irish children since 1990

Year(s)	Age(s)	Overweight	Obesity	Source
1990	8-12 years	10%	2%	Irish National Nutrition Survey (37)
2003-2004	8-12 years	15%	7%	National Children's Food Survey (37)
2002-2012	4-13 years	17-21%	7-9%	Keane et al.* (38)
2015	6-7	12%	5%	COSI Round 4 (356)
2015	8-9	16%	4%	COSI Round 4 (356)
2015	10-11	16%	4%	COSI Round 4 (356)

* Systematic review of studies between 2002 and 2012. Only those studies were included that the authors evaluated to have a high quality.
COSI = Childhood Obesity Surveillance Initiative

Notably, while the ages of four and five years were sometimes included in national or sub-national studies in Ireland, the ages from birth until four years have only been studied based on a small sample (n=371) from a cross-sectional survey conducted in 2011 to 2012 (98), in the sub-sample of a regional prospective cohort study (n=1,189) from 2008 to 2013 (40) and in the infant cohort of the GUI study (n=11,134) that was conducted in 2008 to 2013 (Table 9). The findings from the National Preschool Nutrition Survey are summarised in Figures 9 and 10. Notably, applying the UK/WHO growth criteria, the prevalence of both overweight and of obesity was higher than in Figures 9 and 10 at all ages. Thus, while prevalence levels appeared lower at four years compared to two and three years, irrespective

of the definition used, almost one in three of the two- and three-year olds had OWOB according to the UK90/WHO classification and less than one in four if the IOTF classification was used (98).

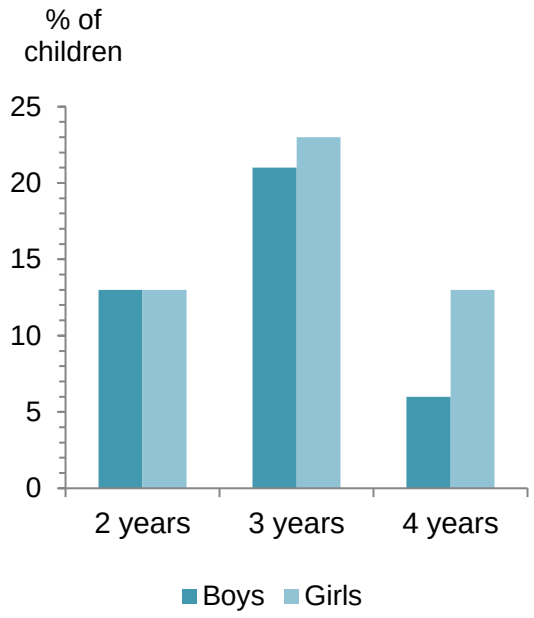

Figure 9: Overweight and obesity prevalence in the National Preschool Nutrition Survey.

Prevalence estimated using the International Obesity Task Force growth reference

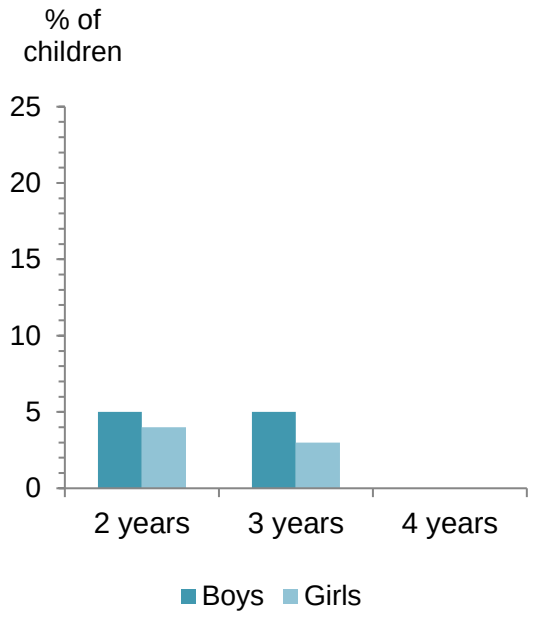

Figure 10: Obesity prevalence in the National Preschool Nutrition Survey.

Prevalence estimated using the International Obesity Task Force growth reference

Longitudinal findings from the GUI infant cohort confirm the prevalence observed in the National Preschool Nutrition Survey at the age of three years based on the IOTF growth reference and indicate a decrease in overweight, but no change in obesity thereafter (3) (Figure 12). However, another study using the GUI infant cohort data indicates that the prevalence from the age of nine months to three years may be higher, applying clinical cut-off criteria from the WHO growth standard, as pictured in Figure 11 (2). As can be seen in Figures 11 and 12, the obesity estimates in particular vary significantly in the three-year-old children from GUI, depending on the definition in use. No more details of the underlying methodology were available and no other in-depth analysis, such as research published in peer-reviewed journals, was available at the time of writing, to investigate these nationally representative data based on all three measurements over time.

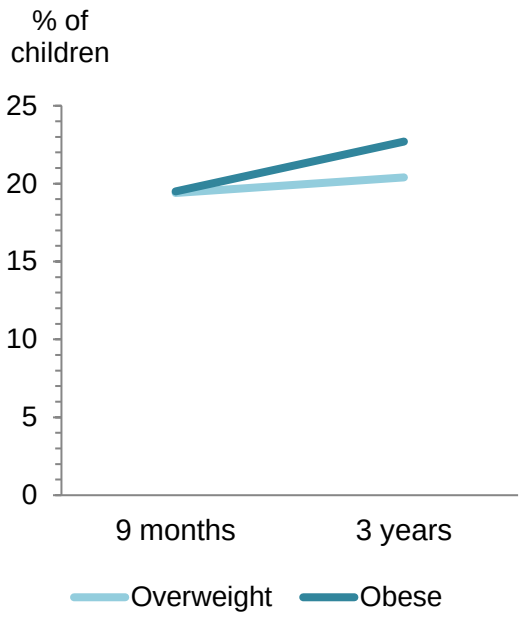

Figure 11: Published overweight and obesity prevalence in the Growing Up in Ireland infant cohort at nine months and three years of age.

Prevalence estimated using the World Health Organization growth standard clinical criteria (2)

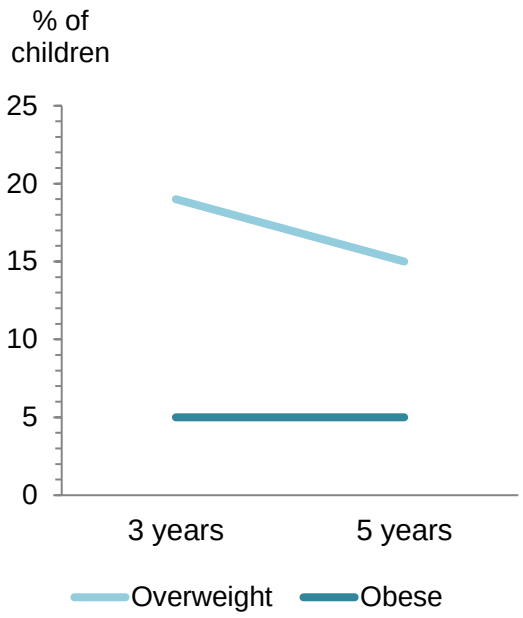

Figure 12: Published overweight and obesity prevalence in the Growing Up in Ireland infant cohort at three and five years of age.

Prevalence estimated using the International Obesity Task Force growth reference (3)

Findings from the Cork BASELINE study published to date only include two-year-old children and prevalence levels are not nationally representative (40, 359). As little is known about the OWOB prevalence in young children in Ireland, this is an important research gap, especially given that data were collected as part of the GUI study that could fill this gap.

In summary, while the trend in OWOB among older children has been extensively studied in Ireland, little data exist in children from their birth until the age of five years. The fact that many children carry excess weight around the age of six years implies that elevated weight gains must have taken place in these children in their younger ages. In fact, children in the youngest age group may have a higher prevalence of OWOB than older children (5), which is supported by the limited evidence available for young children in Ireland. Given that an increase in OWOB in this age group in Ireland was projected until 2030 (7), the current prevalence of OWOB needs to be studied and trends monitored carefully.

4.2 Chapter aims and hypothesis

Following on from the previous section, the identified challenge in childhood obesity research and accordingly the aim of this chapter are two-fold;

- ü First, it needs to be considered how best to measure prevalence of OWOB in the youngest age group below six years, in order to enable comparisons between countries and within Ireland over time
- ü Secondly, in order to monitor childhood obesity as a critical population health determinant in Ireland, the prevalence of OWOB in this understudied age group needs to be measured and compared over time

Therefore, this chapter critically analyses various child growth criteria to define childhood obesity in the age group of up to five years based on BMI. The origin of these criteria is presented and their current use in research is discussed. This includes advantages and disadvantages related to the use of individual criteria, as well as differences in OWOB estimates that were observed between the various criteria in earlier studies.

Based on this research, a subset of suitable growth criteria was defined that appear applicable to young children in the Irish population and that have the potential to improve the comparability between national and cross-national studies. Prevalence of OWOB in young children in Ireland was assessed based on this subset of criteria and differences in OWOB estimates between these criteria investigated, based on population-representative data from the GUI infant cohort, which was described in Chapter 3. Prevalence of girls and boys at the ages of nine months, three years and five years was compared between the different criteria, and recommendations on future use of child growth criteria in this age group were derived from the combined findings in the literature and in the GUI data.

4.3 Methods

4.3.1 Literature search

The literature was searched for use of child growth criteria (based on BMI or other means of measuring child weight status). Classification systems for BMI that are applicable to young children specifically were extensively reviewed. Details of the literature review were outlined in Chapter 2 (Section 2.2.3).

4.3.2 Statistical analysis

To investigate the prevalence of overweight and of obesity in young children in Ireland (Hypothesis 1), the GUI infant cohort data from all waves were used.

Initially, descriptive analysis of weight, length/height and BMI was conducted, as described in detail in Chapter 3 (Section 3.3.2).

Furthermore, based on the literature search, child growth criteria were selected and BMI in the GUI infant cohort was grouped into sex-and age-specific weight categories. Crude prevalence of under- and normal weight (combined), risk of overweight, overweight, and obesity in the GUI infant population were identified by investigating proportions of these weight categories among children at each age (i.e. at nine months, three years and five years, separately). Differences in proportions were assessed between various criteria at three and five years of age, overall and for boys and girls. Also, changes in each weight category and criterion over time were investigated. An adjustment for multiple comparisons was used (Bonferroni correction) in order to lower the risk of type I errors. The differences in proportions, 95% confidence intervals (CI) and test statistics are reported. P-values < 0.05 were deemed significant.

4.4 Results

In the following paragraphs, assessment of weight status in children based on the use of BMI is investigated. Different child growth criteria that classify BMI are explored, their advantages and disadvantages presented and the best child growth criterion for population monitoring of children up to five years of age selected.

4.4.1 Definition of overweight and obesity based on various child growth criteria

Individual child growth criteria are discussed below and details can additionally be seen in Table 11 (pp.169-173).

International Obesity Task Force growth reference

The growth reference published by the IOTF is based on nearly 193,000 children, adolescents and young adults aged zero to 25 years and surveyed in Brazil, the Netherlands, Hong Kong, Great Britain, Singapore and the US. Unlike most growth references, the IOTF provides cut-off values for the ages and sexes based on what translates to adult BMIs of 16, 17, 18.5, 25 and 30 kg/m^2 (322). The IOTF growth reference was developed mainly because existing child growth criteria at the time were limited to national data and criticised for their arbitrary cut-off values (283). In 2012, the IOTF growth reference was extended to combine growth criteria used for thinness and OWOB (360) and it is now internationally acknowledged and used throughout research (322). However, it should be noted that the weight status of children below the age of two years cannot be assessed based on these reference values, which is the main criticism related to this growth reference (322).

World Health Organization child growth standard

In 2006, the WHO published a child growth standard applicable to children aged zero to five years that is based on nearly 27,000 measurements of children from Brazil, Ghana, India, Norway, Oman and the US (322). Longitudinal and cross-sectional data were taken from the Multicentre Growth Reference Study and collected between 1997 and 2003. Cut-off values are based on SD from the mean height, weight or BMI per age and sex. Children are classified as at risk of overweight if their BMI exceeds 1SD, as overweight is their BMI is > 2SD and obese if their BMI is >3SD. Furthermore, a specific growth line of the WHO child growth standard is available for preterm infants (born before 32 weeks of gestation) (361). In clinical practice, the 85th and 95th percentile are displayed on growth charts and used to define overweight and obesity, respectively (361). It should be noted that this growth standard is not used to display average growth in a population, but the optimal healthy growth in children, which is the main difference to the growth references described in this chapter. That means that only children were included in the development of this standard whose mothers were of good health and followed healthy behaviours during the pregnancy, and pursued healthy feeding practices in the children's first year of life (361, 362).

World Health Organization 2007 growth reference

The WHO 2007 growth reference combines the child growth standard of children aged 18 months and older with data from the US National Centre for Health Statistics (NCHS) 1977. The latter include data collected in the 1960s and 1970s in the US among one- to 24-year-old children, adolescents and adults of European ancestry (322). The NCHS data had previously been used in the NCHS/WHO international growth reference that was published in 1991 and only provided BMI references for children aged nine years and older (114).The 2007 growth reference is used for children from five to 19 years and defines weight categories based on SD. Children > 1SD above the mean are considered overweight and >2SD obese. Additionally, percentile curves display the 85th centile to define overweight and the 95th centile to define obesity in the clinical assessment of BMI (114, 322).

2000 Centers for Disease Control growth charts

The 2000 CDC growth charts are a growth reference based on data from the US, and are accordingly mainly applied to two- to 20-year-old children and adolescents within the US (322). Data were taken from five National Health Examination and National Health and Nutrition Examination Surveys, in addition to a limited number of birth records that provided information on the weight, length and head circumference of children born between 1963 and 1995. Cut-off values are based on centiles, where overweight is defined above the 85th centile and BMIs above the 95th centile are considered obese. Sometimes the 90th and 97th centile are additionally used in clinical assessments (322). Until the age of two years, the CDC recognises the WHO growth standard as preferable reference (362).

UK90 growth charts

The UK90 growth charts are suggested for assessing child and adolescent weight status in the UK in a clinically meaningful way (322). This growth reference is based on weight and length/height measurements collected from eleven surveys in the UK between 1978 and 1990 and replaces the Tanner-Whitehouse standards developed in the 1960s (363). From the age of two weeks until four years, the revised UK90/WHO growth charts have been adopted which combine data from the pre-term children in the UK90 growth charts with those from term children that were used to construct the WHO growth standard. These provide separate charts for preterm children born before the 32nd gestational week and those born later (80, 361). On the various UK90 and UK90/WHO growth charts, the 91st and 98th centiles are used to identify overweight and obesity, respectively, in clinical practice. For population monitoring, overweight is defined at the 85th centile and obesity at the 95th centile (322).

Table 11: Overview of studies used in developing child growth criteria applicable to overweight and obesity (OWOB) research

Child growth criteria	Age range co-vered	Population the criteria were developed from	Sample size included in develop-ment	Type of data collection	Date of data collec-tion	Sex- and age-specific cut-off points	Coun-tries of use	Notes
Inter-national Obesity Task Force growth reference (283, 322, 360)	2-18 years	Children, adolescents and young adults aged 0 to 25 years were sampled from nationally representative surveys in Brazil (Second national anthropometric survey, 1989), the Netherlands (Third nationwide growth survey, 1980), Hong Kong (National growth survey, 1993), Great Britain (Data pooled from five national growth surveys, 1978-1993), Singapore (School health service survey, 1993) and the United States (Data pooled from four national surveys, 1963-1980). Data from Singapore are included for ages 6-18 only. The *lambda, mu, sigma* method was used to construct smoothed growth curves. Sex-specific centile curves passing through a BMI of 16, 17, 18.5, 25 and 30 at 18 years, respectively, were used to define cut-off values in the earlier ages	192,727 children, adolescents and young adults	Cross-sectional surveys	1963-1993	Cut-off values for the ages and sexes based on what translates to adult BMIs of 16, 17, 18.5, 25 and 30 kg/m^2	Inter-national	Extended in 2012 to combine growth criteria used for thinness and OWOB
WHO child growth standard (277, 322, 364)	0-5 years	0- 71-months old children from Brazil, Ghana, India, Norway, Oman and the United States taken from the Multicentre Growth Reference Study Longitudinal component: included were healthy, single birth, term	8,406 children (1,737 in the longitudinal study)/ 27,000 measure-ments	Combined longitudinal component (birth until 24 months) and cross-sectional component	1997-2003	Children are grouped as at risk of overweight if BMI > 1SD, as overweight if their BMI is >2SD and as	Inter-national	These growth standards are not used to display average growth in a population, but the optimal healthy growth in children of this population expected under ideal environmental conditions

Child growth criteria	Age range co-vered	Population the criteria were developed from	Sample size included in develop-ment	Type of data collection	Date of data collec-tion	Sex- and age-specific cut-off points	Coun-tries of use	Notes
		children predominantly or exclusively breastfed for at least 4 months and breastfed for at least 12 months if they had a birth weight ≥2500g, or breastfed for at least 3 months if their birth weight was <2500g; complementary food was introduced by age 6 months; mothers had to be healthy and non-smoking pre- and post-partum. Children were included only if no other constraints to growth were known (e.g. socio-economic/environmental risk factors) Cross-sectional component: similar criteria but only three months breastfeeding required	(19,900 in the longitudinal study)	(18-71 months)		obese if their BMI is >3SD. Other cut-offs based on SD are used to define underweight, stunting and wasting. The 85th and 95th centile are used to display overweight and obesity on clinical growth charts		Children with excess adiposity or underweight (weight for length/height <-3SD or >3SD in the longitudinal sample and <-3SD or >2SD in the cross-sectional sample) were excluded from the total sample used to construct the growth standard, because the distribution of children from the cross-sectional sample was highly skewed to the right A Box-Cox power exponential method with 4 parameters and cubic splines was used to construct smoothed growth curves
WHO 2007 growth reference (114, 322)	5-19 years	Combines the child growth standard of children aged 18 months and above with data from the National Centre for Health Statistics 1977 of 0-24-year old children, adolescents and young adults of European ancestry in the United States. Data from the National Centre for Health Statistics were based on the Health Examination Survey Cycle II in 6-11-year-old children and Cycle III in 12-17-year-old children, and on the Health and Nutrition	30,907 observa-tions	Cross-sectional data	1997-2003; 1960 and 1970s	Children >1SD from the mean are considered overweight and those >2SD from the mean are considered obese. The 85th and 95th centile are used to display overweight and obesity on	Inter-national	Box-Cox power exponential method with curve smoothing and cubic splines was used to develop growth curves with smooth transitions between the WHO child growth standard data and the National Centre for Health Statistics data

Child growth criteria	Age range co-vered	Population the criteria were developed from	Sample size included in develop-ment	Type of data collection	Date of data collec-tion	Sex- and age-specific cut-off points	Coun-tries of use	Notes
		Examination Survey Cycle I (age 0-24 years).				clinical growth charts		
2000 Centers for Disease Control growth charts (322, 362, 365).	2-20 years (0-20 years avai-lable but not in use)	Data from 5 National Health Examination (NHES) and National Health and Nutrition Examination Surveys (NHANES), in addition to a limited number of birth records that provided information on the weight, length and head circumference of children born between 1963 and 1995. Surveys: NHES II (1963-1965), NHES III (1966-1970), NHANES I (1971-1974), NHANES II (1976-1980) & NHANES III (1988-1994); birth records: United States Vital Statistics (1968-1980, 1985-1994), State of Wisconsin Vital Statistics (1989-1994), State of Missouri Vital Statistics (1989-1994), Fels Longitudinal Study (1960-1994) & Pediatric Nutrition Surveillance System (!975-1995); Children born with a weight <1500g (all data sources) and children aged ≥6 years in the NHANES III were excluded. Survey data were statistically weighted to achieve population representativeness. Growth curves were smoothed using parametric and non-parametric regression, and curves were transformed using *lambda, mu, sigma* estimation procedures	4,697 observa-tions and children	Cross-sectional data	1963-1995	Overweight is defined above the 85th centile. BMIs above the 95th centile are considered obese, and sometimes the 90th and 97th centile are additionally used in clinical assessments. Additionally, sex- and age-specific means and SD are provided for use in research.	Mainly United States	Until the age of 2 years, the Centers for Disease Control recognise the WHO growth standards as preferable reference, using the 2SD to define Cut-off points below the 3rd and above the 97th percentile should be interpreted with caution as the methodology used is understood to offer limited precision outside this range Due to the representation of various racial and ethnic groups in the population of the United States, the criteria can be applied to ethnically diverse groups of children

Child growth criteria	Age range co-vered	Population the criteria were developed from	Sample size included in develop-ment	Type of data collection	Date of data collec-tion	Sex- and age-specific cut-off points	Coun-tries of use	Notes
UK90 growth charts (80, 322, 363, 366)	0-23 years	Weight measurements collected from 11 surveys in the United Kingdom: the Human Measurements Anthropometry and Growth Research Group's children's growth studies (4 studies in 1978-1987) and adult body dimensions studies (2 studies in 1984-1987), the Tayside Growth Study (1989-1990), the National Study of Health and Growth (1989-1990), the Department of Health survey of the statures and weights of British adults (late 1980s), the Cambridge Infant Growth Study (1984-1988), and the Whittington birth data study (1987-1988). Data were weighted based on geographic region and age. The *lambda, mu, sigma* method, using a Box-Cox power transformation to normalise the data, and penalised likelihood were used to fit growth curves to the data	30,535 children, adolescents and young adults from birth (earliest: 33rd week of gestation) until the age of 23 years	Cross-sectional survey data	1978-1990	Overweight is defined at the 85th centile for population monitoring, whereas only children crossing the 91st centile are treated as overweight in clinical practice. Obesity is accordingly defined at and above the 95th centile for population monitoring and the 98th centile for clinical assessment.	United King-dom and Ireland used after the age of 4 years in Ireland until new child growth criteria have been implem-ented there	Data from pre-term children are included and were used to develop separate growth charts for children born before the 32nd week which can be used until the age of 2 years Suggested for assessing child and adolescent weight status after the age of four years in the United Kingdom in a clinically meaningful way
UK90/WHO growth charts (80, 322, 361, 367)	2 weeks - 4 years	Combination of data from pre-term children included in the UK90 growth charts, and term children included in the WHO growth standard		Cross-sectional and longitudinal data	1978-2003	The 85th centile is used to define overweight for population monitoring, while the 91st centile is used in the clinical assessment of overweight. The	United King-dom and Ireland The growth charts for pre-term and term	Separate growth charts are available for children born before the 32nd gestational week which can be used until 2 years of age, and for children who were born on time or no earlier than the 32nd gestational week

Child growth criteria	Age range co-vered	Population the criteria were developed from	Sample size included in develop-ment	Type of data collection	Date of data collec-tion	Sex- and age-specific cut-off points	Coun-tries of use	Notes
						95th and 98th centile are used for obesity, respectively.	babies are recom-mended for use in Ireland	The birth weight standard of the WHO is not adopted. Growth charts only start being used 2 weeks after gestation as within the 2 weeks after birth, weight as a proportion of birth weight offers better assessment of nutritional status Growth charts display the optimal healthy growth in children that is expected under ideal environmental conditions and following feeding guidelines, as it is based on the WHO growth standard The charts are considered to be applicable to various ethnic groups as the data in the WHO growth standard were collected from an international sample

WHO = World Health Organization

SD = standard deviation

4.4.2 Critical analysis

The child growth criteria presented in the previous section differ significantly from each other, hence it is essential to report which criteria were used in studies to make childhood obesity research comparable. The following paragraphs focus more on the frequency and specifics of use of these criteria, and shed light on the known advantages and disadvantages with regards to the use of each of these criteria.

Child growth criteria across countries

Use in research

The predominance of the major growth criteria in scientific publications was assessed. No consistent recommendations across journals appeared to exist. One journal that focuses solely on childhood obesity (Pediatric Obesity) asks authors submitting their research to the journal to provide estimates of weight status both using the WHO growth standard or WHO growth reference, and the IOTF growth reference (368). This approach has also been suggested by the European Childhood Obesity Group (369). To the best of the author's knowledge, other journals in the area of childhood obesity do not request use of fixed criteria, and accordingly, studies published in those journals use a mix of child growth criteria.

In addition, a number of surveillance studies were looked at to examine which child growth criteria were used. The COSI that investigates obesity in European school children and includes 7,903 children from Ireland (first class students in Rounds 1-4) (41) quantifies obesity numbers using both WHO and IOTF growth references (338), in line with requirements set by Pediatric Obesity. Another surveillance study, the *Health Behaviour in School-aged Children* study, surveys 11-, 13- and 15-year-old children and adolescents in more than 30 countries in Europe and North America every four years since 2002. Obesity levels based on this study are only published based on the IOTF growth reference (370). Similarly, prevalence of OWOB in official GUI publications is only based on IOTF criterion (3). Of the two global surveillance studies that were described earlier in this chapter (Section

4.1.2), one studied OWOB in children and adolescents from the age of five to 19 years using the WHO growth reference (12) and the other covering the ages of two to 19 years using the IOTF growth reference (15). Overall, use of the WHO child growth standard is advised in children below the age of five years, while the IOTF growth reference and the CDC growth charts are frequently used in older children (302, 322).

Growth reference vs. growth standard

The child growth criteria presented in Table 11 differ in various ways based on how, when and where the underlying datasets were collected and one important distinction between the criteria is their use as a growth reference or a growth standard. While growth references display the actual growth measured in a population, growth standards present the distribution of sex- and age-specific BMI in a population growing up under optimal environmental condition. Among the identified major child growth criteria, only the WHO criterion applicable to children from birth to five years was designed for use as a growth standard. A major advantage of the WHO growth standard is that the underlying data were collected from healthy children only, defined by their health status as well as gestational behaviours of the mother and early feeding practices (277). Therefore, BMI has been measured in children under standardised, ideal conditions in their early lives, which makes the WHO growth standard a useful guideline for determining the range of healthy BMI among infants and young children.

In fact, both the WHO growth standard and growth reference have been shown to identify children at risk of weight-related ill-health. In one study, a set of cardiometabolic risk factors was investigated in relation to OWOB estimates based on the WHO growth criteria (using the growth reference classification), the IOTF growth reference and the criteria of the Italian Society for Pediatric Endocrinology and Diabetology. The sex- and age-specific estimates of overweight and of obesity based on the IOTF and Italian criterion approximated each other and the specificity of IOTF criteria in identifying high cardiometabolic risk was found to be higher than that of the WHO criteria. However, a higher sensitivity was found in the WHO reference values, which led the authors to conclude that the WHO criteria appeared more useful in the clinical setting and for use in epidemiological studies (336). Furthermore, in a population of two- to 19-year-old children and adolescents

with cardiometabolic diseases or disease markers, more children were identified as obese using the WHO criteria than when the CDC growth charts were used. Considering the association between cardiometabolic ill-health and OWOB, the identification of children with an increased risk based on their weight status is crucial, which is why the WHO criteria were also favoured in this study (371). Furthermore, in a study of six- to 13-year-old children in Brazil, the Brazilian child growth reference and the CDC growth charts proved to identify more children with high blood pressure as overweight or obese than when the IOTF growth reference was used (63). Overall, the availability of studies relating various child growth criteria to disease or disease risk was low. Therefore, studies were looked at that compared various criteria, either for clinical purposes or to monitor population trends, without investigating their ability to identify other health parameters.

Difference in prevalence

In 2011, a study compared the WHO growth standard to national growth references used in Belgium, France, the Netherlands, and the UK and to the CDC growth charts (164). Growth curves particularly in infants diverged between the WHO growth standard and national references used by paediatricians in the mentioned countries. In all countries, mean weight of the children included was below that of the WHO growth standard until the age of three months, but exceeded the WHO growth standard from six to 18 months. Furthermore, in France, mean BMI of the French growth reference was similarly lower than that of the WHO growth reference, exceeded the WHO growth standard values at six months and was still higher by the age of five years (164). Consequently, more children tend to be identified as overweight or obese using the WHO growth standard than when these national references are used. Previous studies suggest that higher OWOB levels may have been identified by the WHO growth standard because of the removal of outliers, often referred to as ‘trimming’, that was part of its methodological construction (337, 372). In fact, if similar trimming procedures were applied to the CDC growth charts data, prevalence of BMI above the 95th percentile were comparable in two- to five-year-old children using either the WHO growth standard or the CDC growth charts (372). However, for developing the WHO growth standard, outliers were only removed in the cross-sectional dataset, but not in the longitudinal component of the data available in infants from birth until

the age of two years. As large differences between the WHO growth standard and other criteria were also seen below the age of two years, a real difference appears to exist between the criteria beyond this methodological explanation (337).

In fact, what may explain part of the differences between various criteria may be related to infant feeding practices. National growth curves in Belgium resembled the WHO standards much more if only data from infants were studied who had been fed based on healthy feeding guidelines, than when all children were included that contributed to the construction of the Belgian growth curves (164). Similarly, a smaller Canadian study compared the growth curves of 73 infants until their age of 18 months against CDC growth charts and the WHO growth standard. The authors identified the WHO growth standard as superior because only the WHO growth curves resembled the weight-for-height development of those infants that had followed early feeding recommendations existing in Canada (165).

A number of studies assessed various child growth criteria with the aim of introducing early intervention to children with OWOB. In this regard, a study based on nearly 144,000 two- to five-year-old children concluded that the WHO growth standard was preferential to the CDC growth charts. In that study, the WHO growth standard identified children's obesity status earlier than obesity was identified using the CDC growth charts, therefore allowing for earlier intervention in children with weight problems. Furthermore, the study showed that obese children were identified early irrespective of whether the 95th percentile displayed on WHO growth charts or the threshold of 2 SD of this growth standard were used to define obesity (337).

In turn, the CDC growth charts were evaluated as superior to the IOTF growth reference in young children in a Canadian and a smaller Italian study (n=258) because more children were identified as obese using the CDC growth charts (373, 374), a trend that appears to continue as children grow older (375, 376). The same was seen in a Spanish study that compared growth curves in children from two to 24 years of age (n=3,534) (376). These studies concluded that particularly in young children, use of the CDC criterion would be preferential because any indication of weight problems needed to be acknowledged in clinical settings in order to intervene early (373, 374, 376). This was illustrated by an example from France. In

a comparison of the French growth reference and the WHO growth standard, physicians came to very different conclusions about how infants were growing and how they should accordingly intervene. A third of the exclusively breastfed children in this study were growing normally according to the WHO growth standard, but had gained too much weight according to the French growth reference from birth to their age of two months. In conclusion, the physicians involved in this study recommended restrictive feeding for the children based on the French growth reference, whereas they did not recommend this if the same children were assessed using the WHO growth standard (164). This may have important implications for children's later energy intake as they are supposed to learn how to interpret and respond to signals of satiety and hunger through breastfeeding (172).

Additionally, Edwards et al. (373) raised the concern that an underestimation of the obesity prevalence in surveillance studies may lead to insufficient resources being dedicated to obesity prevention. For this reason, in Europe, where countries are affected more by the consequences of OWOB than undernutrition (5, 12), the risk of an underestimation of underweight prevalence using the WHO growth standard (377) may be less critical on population level than the possible underestimation of OWOB levels using alternative child growth criteria. However, it should also be noted that it is not clear whether use of the WHO growth standard even leads to an underestimation of underweight in young children (378).

Timing of data collection underlying the child growth criteria

In addition to the health characteristics and feeding practices of children in the reference population underlying a child growth criterion, potential reason for differences between OWOB estimates based on various criteria may also be the time when data were collected. The French growth reference is based on data from children born in the 1950s, shortly after World War II, which may not adequately reflect child growth in the 21st century. While the birth weight and length of 77,000 infants born after 2003 were similar to those in the French growth reference, weight and length after birth and until the age of two years was significantly higher than those set out in the reference (379). In fact, data from children born in France in the early 1990s deviated less from the WHO growth standard values than the data

that had been collected to generate the French growth reference (164). Differences in OWOB estimates across child growth criteria were also related to the different timing of data collection underlying the criteria in a Swedish study (380). The authors of the Swedish study pointed to the fact that the increase in childhood obesity in the 1970s and 1980s was considerably rapid, which means that data collected with only a few years' difference could have resulted in very different child growth criteria (380). For this reason, a set of more recent data was excluded from the pool of studies that were used to construct the CDC growth charts (363). Additionally, the WHO Multicentre Growth Reference Study Group (277) found discrepancies between growth data collected from 1929 to 1975 for the NCHS/WHO child growth reference, and data collected in the 1990s that formed the basis for the WHO child growth standard. Beyond the differences between breastfed and non-breastfed children, the study group also related these discrepancies to differences in the nutritional components of infant formula that were marketed to families over time (277).

Differences in the time of data collection are also observed when the main child growth criteria presented in this study are compared. The data underlying the CDC growth charts and the IOTF growth reference both were collected between 1963 and the mid-1990s. The data used to construct the UK90 growth charts were collected from 1978 until 1990 and hence overlap with the second half of the CDC and IOTF data collection. The WHO growth standard is based on data from 1997-2003 and the WHO growth reference is based on a combination from these same data and earlier studies (1960s to 1970s). Therefore, the WHO growth standard and growth reference are based on most recent data, with a much longer period of data collection underlying the WHO growth reference. In contrast, most recent data underlying the CDC and IOTF criteria are seven and ten years older, and the earliest data points integrated into their construction are almost 35 years older than the earliest in the WHO growth standard. On the contrary, that means that the CDC, UK90 and IOTF growth criteria have been extensively used in prior research (302), whereas fewer studies have reported OWOB according to the current WHO criteria which were only published in 2006 and 2007.

Other advantages and disadvantages of various child growth criteria

A number of other arguments support use of the various child growth criteria which are related to the nature of how the criteria were constructed. A major advantage of the IOTF growth reference is the number of observations included in the study. Data from nearly 200,000 children were included (283), which is much higher than in any of the other child growth criteria reviewed.

Additionally, both the IOTF growth reference and the two WHO criteria are based on samples from various countries, making them particularly useful for international comparisons. Prior research confirmed that the WHO growth standard is applicable across ethnic groups other than those included in the study design (361), including children from Chile, Thailand and Kenya (381). Due to the representation of various racial and ethnic groups in the US, however, the CDC growth charts were also recommended for use in ethnically diverse groups of children (365).

Another major advantage of the WHO growth standard over other child growth criteria is the longitudinal component of the data underlying the growth standard (380). Along with the strict inclusion criteria that were subject to the development of the WHO growth standard, also its longitudinal nature has led other countries to adopt the WHO growth standard in parts. Until the age of two years, the CDC recognises the WHO growth standard as preferable to the CDC growth charts (362), and also the UK90 growth charts adopted the WHO growth standard for use in all term children from the age of two weeks until four years (361).

Lastly, an advantage of the UK90/WHO charts is their availability of separate growth charts used in preterm children until their age of two years (80).

For the mentioned reasons, use of the WHO growth standard as the sole reference or in addition to national growth criteria may be a better reference to monitor the growth of young children on population level (but with a high clinical relevance) than sole use of national reference data. However, the strengths of the various other criteria should not be ignored and, where feasible, a combination of criteria should be considered in order to provide comprehensive and comparable estimates of OWOB.

Choice of child growth criteria for application in the GUI cohort

In summary, based on the reviewed literature, the international child growth criteria published by the IOTF and WHO appear to be suitable tools to classify children's BMI within and across countries. On a population level, BMI has been shown to identify children at risk of later obesity and weight-related ill-health in a meaningful way. Therefore, use of IOTF and WHO criteria should be promoted also in national cohort studies. For application in this cohort study, the WHO growth criteria appear more useful than the IOTF growth reference, simply because the latter only applies to children aged two years and older, whereas the GUI study measured BMI as early as at the age of nine months. Additionally, the critical analysis of child growth criteria revealed a slight preference for the WHO criteria over use of the IOTF growth reference. Nevertheless, it needs to be acknowledged that a considerable proportion of findings on OWOB levels in two- to five-year-old children, both in the past as well as in recent studies, are based on the IOTF criterion. Thus, to enable comparisons with those studies, application of the IOTF growth reference at the ages of three and five years, in addition to WHO criteria, is also recommendable for this study.

In addition, the UK90+WHO criterion has been adopted in clinical practice in Ireland to assess individual child BMI against growth curves (80). While a number of studies used the UK90 or UK90+WHO references to assess BMI prevalence on population level, this does not appear to offer a huge advantage to the GUI infant cohort data for a number of reasons. First, the UK90+WHO references use the exact same sex- and age-specific reference values from birth until the age of four years as the WHO growth standards. Therefore, differences between the UK90+WHO and WHO child growth criteria would only be possibly seen when the children in the GUI infant cohort are five years old. Secondly, use of UK90+WHO growth criteria is most popular in the UK and Ireland, which means that findings based on these criteria would not be comparable against surveillance data from most other countries. As it is a primary aim of this study and of current health reports to make surveillance data, particularly among young children, more comparable across countries (79), use of the UK90+WHO criteria does not appear to provide an added benefit beyond an assessment of population estimates at national level. Thirdly, new Irish growth charts are underway to replace the

UK90+WHO growth charts currently in use in Ireland. Growth charts applicable to children aged four years and older, based on a combination of the UK90+WHO criteria and Irish reference data collected in the 1980s, were planned for implementation in 2017 (80). At the time of writing, the new growth charts have not been implemented. Nonetheless, estimates of OWOB based on the UK90+WHO child growth criteria would likely be outdated in the near future, which is why population level data in Ireland, including this study, should better be monitored using international criteria.

Lastly, one further limitation must be noted that concerns the definitions of weight categories used in the WHO growth standard and in the WHO growth reference. Based on the WHO growth standard, children with an age- and sex-specific BMI > 1SD above the reference median are understood to be ‘at risk of overweight’, whereas exceeding 1SD in the WHO growth reference refers to actual overweight. Furthermore, age- and sex-specific BMI > 2SD in the growth standard marks children’s overweight, whereas children exceeding 2SD of the growth reference are classified as obese. For children to be classified as obese in the growth standard, they need to cross 3SD, which in turn refers to severe obesity based on the growth reference. In practice, this means that a child classified as overweight at the age of 59 months, based on the growth standard, would likely be classified as obese one month later, because the growth reference applies from the age of five years (60 months) onwards. As children in the GUI cohort turned five years shortly before the last interview took place, they had just crossed the threshold for application of the growth reference. Notably, growth data underlying the WHO growth reference from the ages of 60 to 71 months were smoothed based on anthropometric data from the children that had contributed to the development of the WHO growth standard (114). Accordingly, the clinical thresholds of overweight (85th percentile) and obesity (95th percentile), which are occasionally also used in research, are consistent across all ages (336, 337).

Therefore, it appears feasible to investigate children’s BMI at all ages using *ó> 1SD Ò + ôSóD 2 S D ôÒ* and *3ó> 3SDô* as thresholds, in order to track changes in BMI deviations from the median between the ages in a coherent manner, in addition to examining OWOB prevalence at nine months and three years based on the WHO growth standards and prevalence at five years using the WHO growth reference.

Furthermore, percentile lines of BMI according to the WHO growth standard and WHO growth reference were looked at across ages for comparison of the two WHO criteria. Percentile lines were provided for all children with valid BMI data in a first step, and for children who were breastfed for six months or longer in a separate graph. The latter was provided because the WHO growth standard is based on data from families who adhered to a number of infant feeding guidelines. As adherence to breastfeeding guidelines in Ireland is low (310), it appeared reasonable to investigate whether the distribution of BMI differed significantly among children who were breastfed for six months or longer, compared to the distribution of the entire cohort.

Accordingly, prevalence and trends of OWOB in the GUI infant cohort were investigated using 1) a combination of the WHO growth standard and WHO growth reference based on the official SD thresholds of the respective child growth criterion, 2) an extended use of the WHO growth standard, using the WHO growth reference percentiles but setting thresholds according to those defined in the WHO growth standard throughout all ages, and 3) the IOTF growth reference which is applicable to children in the GUI cohort only at their ages of three and five years.

Chapter 3 described in detail how the GUI infant cohort data were collected. It furthermore specified the proportion of children with no weight or height data at each age and reasons that led to the exclusion of invalid BMI and outliers. After the data cleaning, prevalence of OWOB was investigated in 10,810 children (49% girls) when they were nine months old, 9,422 (49.5% girls) at the age of three years and 8,781 (49.4% girls) at five years. Before prevalence for each age is presented, the following section shows how weight, length/height and BMI were distributed and correlated across the ages.

4.4.3 Prevalence of overweight and obesity in the Growing Up in Ireland infant cohort

Descriptive statistics

After the exclusion of missing values, outliers and invalid values, 10,810 children (49% girls) remained in the cohort that had a valid BMI reported at nine months, 9,422 (49.5% girls) at three years and 8,781 (49.4% girls) at five years of age. Of those, 8,119 children had valid BMI measurements reported for all waves (Table 12). Additionally, after the cleaning, consistent BMI ranges of between 11 and 27 kg/m^2 across the ages and sexes were seen. As shown in Table 12, no major differences existed between weight, height and BMI at five years of age when only those children were included who had anthropometric measurements reported in each of the three waves, compared to all five-year-old children. Weight, length/ height and BMI were distributed normally after the exclusion of invalid values.

Table 12: Means, standard deviation (SD) and ranges of weight, height and body mass index (BMI) in the Growing Up in Ireland infant cohort, after variable cleaning

Age	Variable	N	*% missing in wave**	Mean	SD	Range
9 months	Weight (kg)	10926	1.9	9.7	1.2	5.2 – 15.4
	Height (cm)	10844	2.6	72.9	3.1	58.5 – 83.0
	BMI (kg/m²)	10810	2.9	18.2	1.7	11.9 – 26.8
3 years	Weight (kg)	9489	3.1	15.6	2.0	9.8 – 27.1
	Height (cm)	9542	2.6	96.2	3.9	80.8 – 115.0
	BMI (kg/m²)	9422	3.8	16.8	1.5	10.6 – 25.0
5 years	Weight (kg)	8839	1.8	20.2	3.0	12.1 – 38.0
	Height (cm)	8867	1.5	111.3	4.7	91.3 –129.4
	BMI (kg/m²)	8781	2.4	16.3	1.6	11.3 – 26.5
5 years (only in children with the respective variable available at all ages*)	Weight (kg)	8291	7.9	20.2	2.9	12.1 – 38.0
	Height (cm)	8256	8.3	111.3	4.7	94.1 –129.0
	BMI (kg/m²)	8119	9.8	16.2	1.6	11.3 – 26.5

* Missing values include missing measurements as well as observations excluded from the analysis.

Correlation of anthropometric data over time

Weight, height and BMI measurements of children were plotted to test the collinearity between waves. Figures 13 to 15 display the plots of BMI in the same children at three years in relation to their nine-month BMI, at five years in relation to three years, and at five years in relation to nine months. As can be seen from the correlation coefficients displayed under the plots, BMI measurements correlated over time, as expected, with the highest correlation seen between three and five years of age.

The correlation coefficients for weight were 0.71 between nine months and three years of age, 0.81 between three and five years, and 0.57 between nine months and five years. For height, the coefficients were 0.62, 0.85 and 0.58 respectively; hence, anthropometric data correlated most from three to five years.

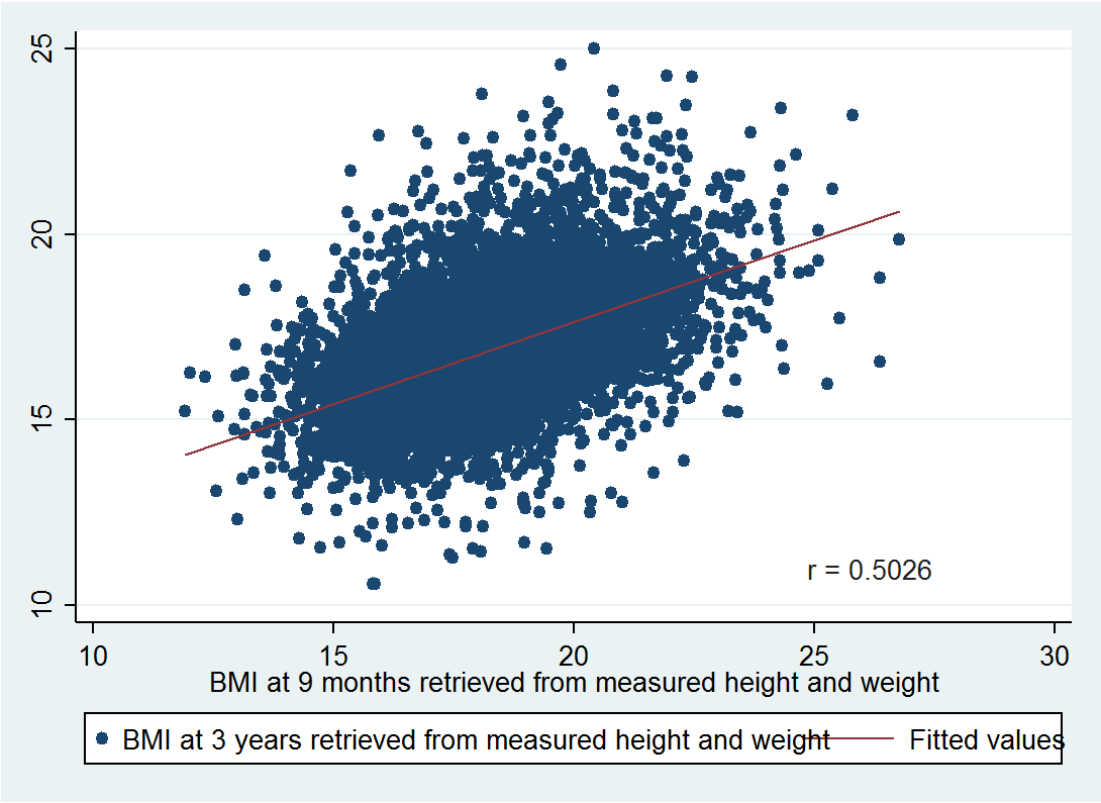

Figure 13: Correlation of body mass index (BMI) at the ages of nine months and three years

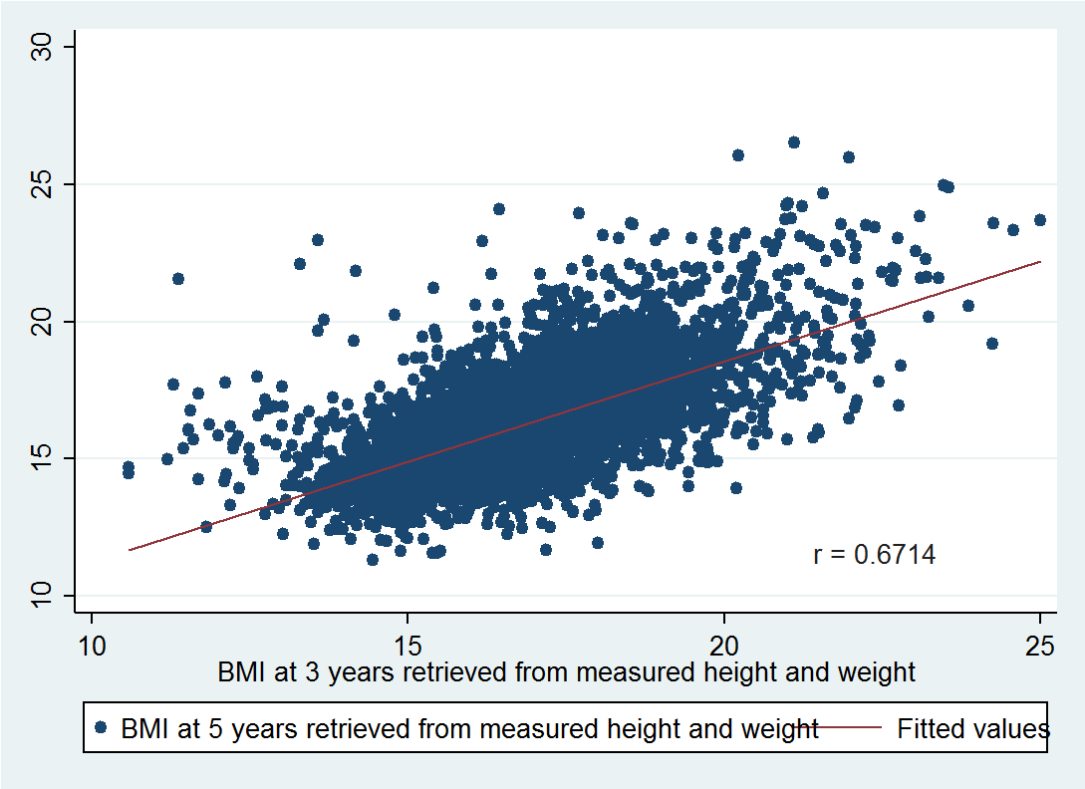

Figure 14: Correlation of body mass index (BMI) at the ages of three and five years

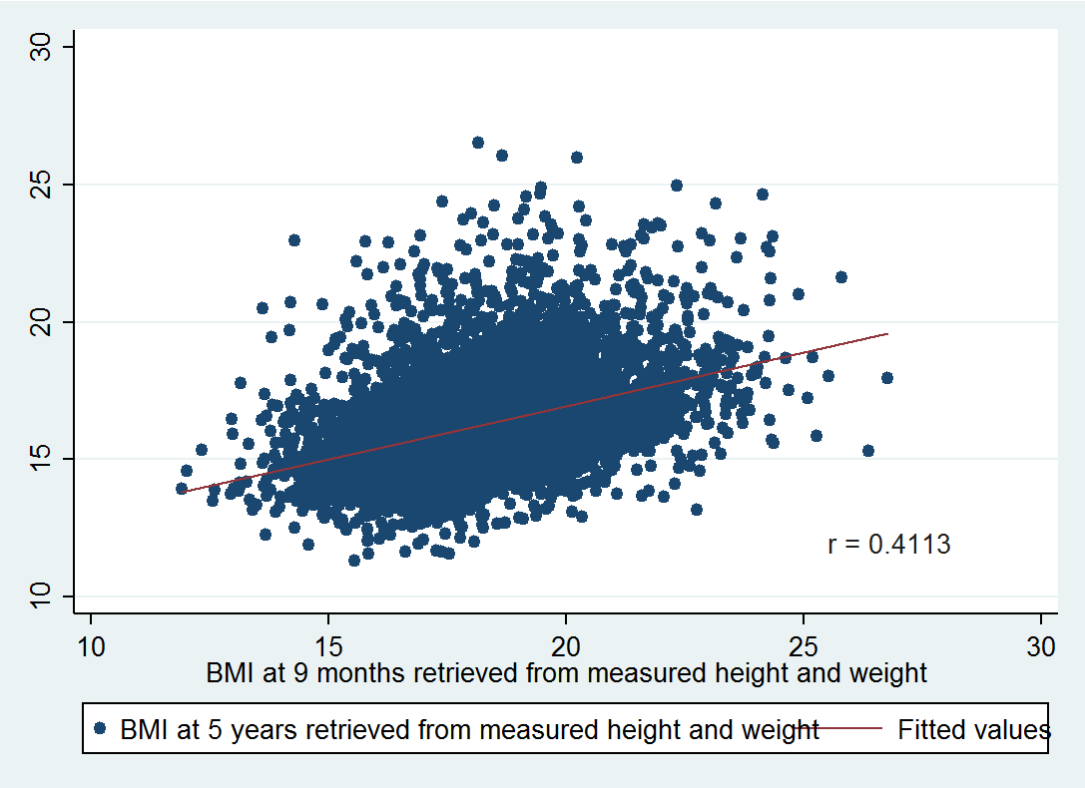

Figure 15: Correlation of body mass index (BMI) at the ages of nine months and five years

Differences across child growth criteria and over time

Tables 13 and 14 present the prevalences of under- and normal weight, risk of overweight, overweight and obesity. As can be seen from the data, the prevalence of overweight at the age of nine months was 9.4% and prevalence of obesity was 2%. Using the WHO growth standard classification, this increased significantly to almost 11% of overweight and 2.7% of obesity at three years of age. Depending on which WHO criterion was applied when the children were five years old, prevalence of both overweight and obesity decreased significantly (to 5.8% and 1.9% using the WHO growth standard classification) or increased significantly (to 23.8% and 7.7% using the WHO growth reference) (Table 13). From the age of nine months to five years, the prevalence of all weight groups differed significantly if WHO growth standard estimates were used at nine months and WHO growth reference estimates at five years. When the WHO growth standard classification was used both at nine months and five years of age, differences were significant for all weight groups except the obesity category (Table 13). The decrease from 2% to 1.9% in obesity from the start until the end of the study period was not significant (p=0.99), even though significant changes had been seen from nine months to three years (p=0.02) and from three to five years (p=0.01).

Furthermore, at the ages of three and five years, prevalence was assessed using the IOTF growth reference. At three years, 18.3% of children were overweight and 5.2% obese, which decreased to 15.1% and 4.8% at five years, respectively. Only the decrease in overweight (p=0.01) was significant (Table 13).

As the use of various child growth criteria led to different estimates of prevalence at the ages of three and five years, differences between the respective estimates were compared. Both at the ages of three and five years, prevalence of overweight and of obesity was significantly lower using the WHO growth standard compared to the IOTF growth reference (Table 14). Furthermore, at five years, prevalence of overweight and of obesity was higher when the WHO growth reference was used, than when the WHO growth standard or IOTF growth reference was applied. The WHO growth standard and the WHO growth reference led to identical estimates of under- and normal weight prevalence, but significantly higher levels were found using the IOTF growth reference both at three and five years (Table 14).

Table 13: Time trends in prevalence of weight categories in the Growing Up in Ireland infant cohort. Prevalence was estimated at nine months (n=10,810), three years (n=9,422) and five years of age (n=8,781) using the World Health Organization growth standard (WHO-S), International Obesity Task Force growth reference (IOTF) and World Health Organization growth reference (WHO-R) to classify body mass index. Differences in proportions, 95% Confidence Intervals (CI) and Bonferroni adjusted p-values are presented.

Criterion	Child age	Under- and normal weight	Risk of overweight	Overweight	Obesity
WHO-S	**9 months % (95% CI)** **N**	**59.6%** (58.7% to 60.5%) n=6,444	**29.0%** (28.1% to 29.8%) n=3,132	**9.4%** (8.8% to 9.9%) n=1,015	**2.0%** (1.8% to 2.3%) n=219
	3 years % (95% CI) **N**	**54.4%** (53.4% to 55.4%) n=5,124	**32.1%** (31.1% to 33.0%) n=3,021	**10.9%** (10.2% to 11.5%) n=1,024	**2.7%** (2.4% to 3.0%) n=253
	5 years % (95% CI) **N**	**68.5%** (67.6% to 69.5%) n=6,017	**23.8%** (22.9% to 24.7%) n=2,092	**5.8%** (5.3% to 6.2%) n=505	**1.9%** (1.6% to 2.2%) n=167
	Difference in proportions (95% CI) **Adjusted p-values**	*9 months v 3 years* *5.2% (3.8% to 6.6%)* *p=0.01* *3 years v 5 years* *-14.1% (-15.5% to -12.7%)* *p=0.01* *9 months v 5 years* *-8.9% (-10.2% to -7.6%)* *p=0.01*	*9 months v 3 years* *-3.1% (-4.4% to -1.8%)* *p=0.01* *3 years v 5 years* *8.3% (7.0% to 9.6%)* *p=0.01* *9 months v 5 years* *5.2% (3.9% to 6.4%)* *p=0.01*	*9 months v 3 years* *-1.4% (-2.2% to -0.6%)* *p=0.02* *3 years v 5 years* *5.0% (4.2% to 5.8%)* *p=0.01* *9 months v 5 years* *3.6% (2.9% to 4.3%)* *p=0.01*	*9 months v 3 years* *-0.7% (-1.1% to -0.3%)* *p=0.02* *3 years v 5 years* *0.8% (0.4% to 0.1%)* *p=0.01* *9 months v 5 years* *0.1% (-0.3% to 0.2%)* *p=0.99*

Criterion	Child age	Under- and normal weight	Risk of overweight	Overweight	Obesity
WHO-S + WHO-R	**9 months % (95% CI) N**	**59.6%** (58.7% to 60.5%) n=6,444	**29.0%** (28.1% to 29.8%) n=3,132	**9.4%** (8.8% to 9.9%) n=1,015	**2.0%** (1.8% to 2.3%) n=219
	3 years % (95% CI) N	**54.4%** (53.4% to 55.4%) n=5,124	**32.1%** (31.1% to 33.0%) n=3,021	**10.9%** (10.2% to 11.5%) n=1,024	**2.7%** (2.4% to 3.0%) n=253
	5 years % (95% CI) N	**68.5%** (67.6% to 69.5%) n=6,017	N/A	**23.8%** (22.9% to 24.7%) n=2,092	**7.7%** (7.1% to 8.2%) n=672
	Difference in proportions (95% CI) Adjusted p-values	*9 months v 3 years* *5.2% (3.8% to 6.6%)* *p=0.01* *3 years v 5 years* *14.1% (-15.5% to -12.7%)* *p=0.01* *9 months v 5 years* *-8.9% (-10.2% to -7.6%)* *p=0.01*		*9 months v 3 years* *-1.4% (-2.2% to -0.6%)* *p=0.02* *3 years v 5 years* *-13.0% (-14.1% to -11.9%)* *p=0.01* *9 months v 5 years* *-14.4% (%-15.4 to -13.4%)* *p=0.01*	*9 months v 3 years* *-0.7% (-1.1% to -0.3%)* *p=0.02* *3 years v 5 years* *-5.0% (-5.6% to -4.4%)* *p=0.01* *9 months v 5 years* *-5.7% (-6.3% to -5.1%)* *p=0.01*
IOTF	**3 years % (95% CI) N**	**76.5%** (75.7% to 77.4%) n=7,212	N/A	**18.3%** (17.5% to 19.1%) n=1,724	**5.2%** (4.7% to 5.6%) n=486
	5 years % (95% CI) N	**80.2%** (79.4% to 81.0%) n=7,042	N/A	**15.1%** (14.3% to 15.8%) n=1,322	**4.7%** (4.3% to 5.2%) n=417
	Difference in proportions (95% CI) Adjusted p-values	*3 years v 5 years* *-3.7% (-4.9% to -2.5%)* *p=0.01*	N/A	*3 years v 5 years* *3.2% (2.1% to 4.3%)* *p=0.01*	*3 years v 5 years* *0.4% (-0.2% to 10.3%)* *p=0.99*

Table 14: Comparison of prevalence estimates of weight categories in the Growing Up in Ireland infant cohort. Prevalence was estimated at nine months (n=10,810), three years (n=9,422) and five years of age (n=8,781) using the World Health Organization growth standard (WHO-S), International Obesity Task Force growth reference (IOTF) and World Health Organization growth reference (WHO-R) to classify body mass index. Differences in proportions, 95% Confidence Intervals (CI) and Bonferroni adjusted p-values are presented.

Child Age	Criterion	Under- and normal weight	Risk of overweight	Overweight	Obesity
9 months **% (95% CI)** **N**	**WHO-S**	**59.6%** (58.7% to 60.5%) n=6,444	**29.0%** (28.1% to 29.8%) n=3,132	**9.4%** (8.8% to 9.9%) n=1,015	**2.0%** (1.8% to 2.3%) n=219
3 years **% (95% CI)** **N**	**WHO-S**	**54.4%** (53.4% to 55.4%) n=5,124	**32.1%** (31.1% to 33.0%) n=3,021	**10.9%** (10.2% to 11.5%) n=1,024	**2.7%** (2.4% to 3.0%) n=253
	IOTF	**76.5%** (75.7% to 77.4%) n=7212	N/A	**18.3%** (17.5% to 19.1%) n=1724	**5.2%** (4.7% to 5.6%) n=486
	Difference in proportions (95% CI) ***Adjusted p-values***	*22.1% (20.8% to 23.4%)* *p<0.01*	N/A	*7.5% (6.5% to 8.4%)* *p<0.01*	*2.5% (1.9% to 3.1%)* *p<0.01*
5 years **% (95% CI)** **N**	**WHO-S**	**68.5%** (67.6% to 69.5%) n=6,017	**23.8%** (22.9% to 24.7%) n=2,092	**5.8%** (5.3% to 6.2%) n=505	**1.9%** (1.6% to 2.2%) n=167
	IOTF	**80.2%** (79.4% to 81.0%) n=7,042	N/A	**15.1%** (14.3% to 15.8%) n=1,322	**4.7%** (4.3% to 5.2%) n=417
	WHO-R	**68.5%** (67.6% to 69.5%) n=6,017	N/A	**23.8%** (22.9% to 24.7%) n=2,092	**7.7%** (7.1% to 8.2%) n=672
	Difference in proportions (95% CI)	*WHO-S v IOTF* *11.7% (10.4% to 13.0%)*	N/A	*WHO-S v IOTF* *9.3% (8.4% to 10.2%)*	*WHO-S v IOTF* *2.9% (2.4% to 3.4%)*

Child Age	Criterion	Under- and normal weight	Risk of overweight	Overweight	Obesity
	Adjusted p-values	*p<0.01* *WHO-S v WHO-R* *0% (-1.4% to 1.4%)* *p=0.99* *IOTF v WHO-R* *11.7% (10.4% to 13.0%)* *p<0.01*		*p<0.01* *WHO-S v WHO-R* *18% (17.0% to 19.0%)* *p<0.01* *IOTF v WHO-R* *8.7% (7.5% to 9.9%)* *p<0.01*	*p<0.01* *WHO-S v WHO-R* *5.8% (5.2% to 6.4%)* *p<0.01* *IOTF v WHO-R* *2.9% (2.2% to 3.6%)* *p<0.01*

Differences in estimates diverged strongly depending on which WHO criterion was applied, hence a percentile curve was furthermore produced which shows how the BMI of children in the GUI infant cohort was distributed relative to the respective WHO reference values at each age, that is the WHO growth standard at the ages of nine months and three years, and the WHO growth reference at the age of five years (Figure 16). The grey area describes the expected distribution of BMI values based on the WHO percentile groups which are displayed on the x-axis. As shown in Figure 16, the majority of children in the GUI infant cohort lie in the 25th until 95th percentile and at all ages more children had BMI scores in the 75th percentile or higher than in the reference populations which the WHO criteria are based on. The number of children less than the 50th WHO percentiles decreased significantly ($p<0.05$) between nine months and three years, while the number in the 75th to 84th, 90th to 94th and 99.9th percentile or higher increased significantly ($p<0.05$) (Appendix 6). From three years to five years, decreases in the 90th percentile and higher were significant ($p<0.05$) (Appendix 6).

Furthermore, Figure 17 provides the same distribution only in children who were breastfed six months or longer. This was investigated because the WHO growth standard was developed based on data from breastfed infants, whereas adherence to breastfeeding guidelines is relatively low in Ireland. Minor deviations can be seen in the BMI distribution when the children were nine months old and had just been weaned or were still breastfed at the time their BMI was measured. At the later ages, the children's BMI was distributed similarly to BMI scores of the entire cohort, and at all ages, the frequency of children in the 75th percentile and higher was still exceeding the expected distribution based on the respective WHO criteria.

Exact figures for the respective ages and percentiles, as well as an indication of significant changes in each percentile group between the ages, are provided in Appendix 6.

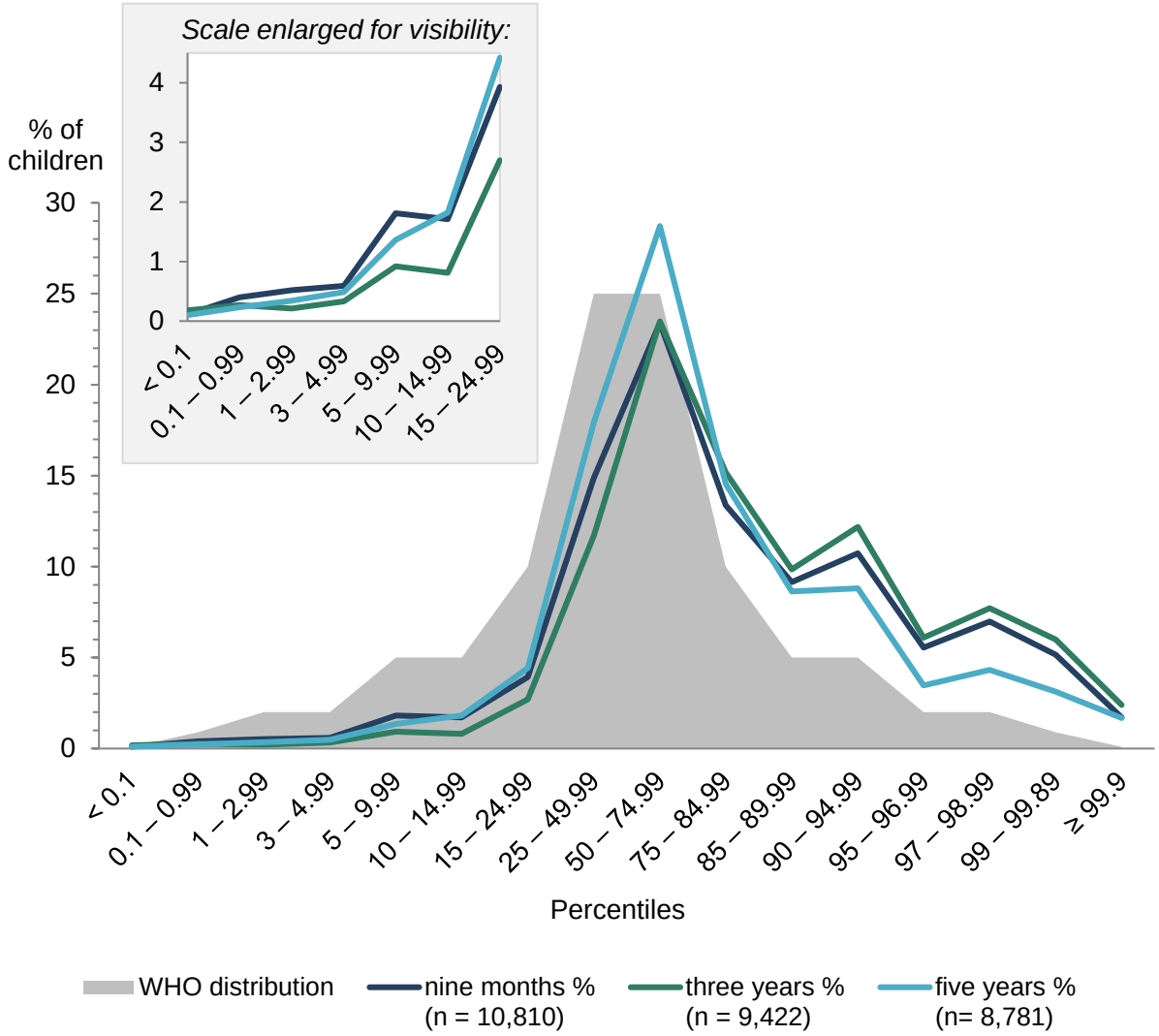

Figure 16: Distribution of body mass index (BMI) in all children. BMI in the Growing Up in Ireland infant cohort was compared to the age- and sex-adjusted BMI percentiles defined by the World Health Organization (WHO) growth standard and growth reference.

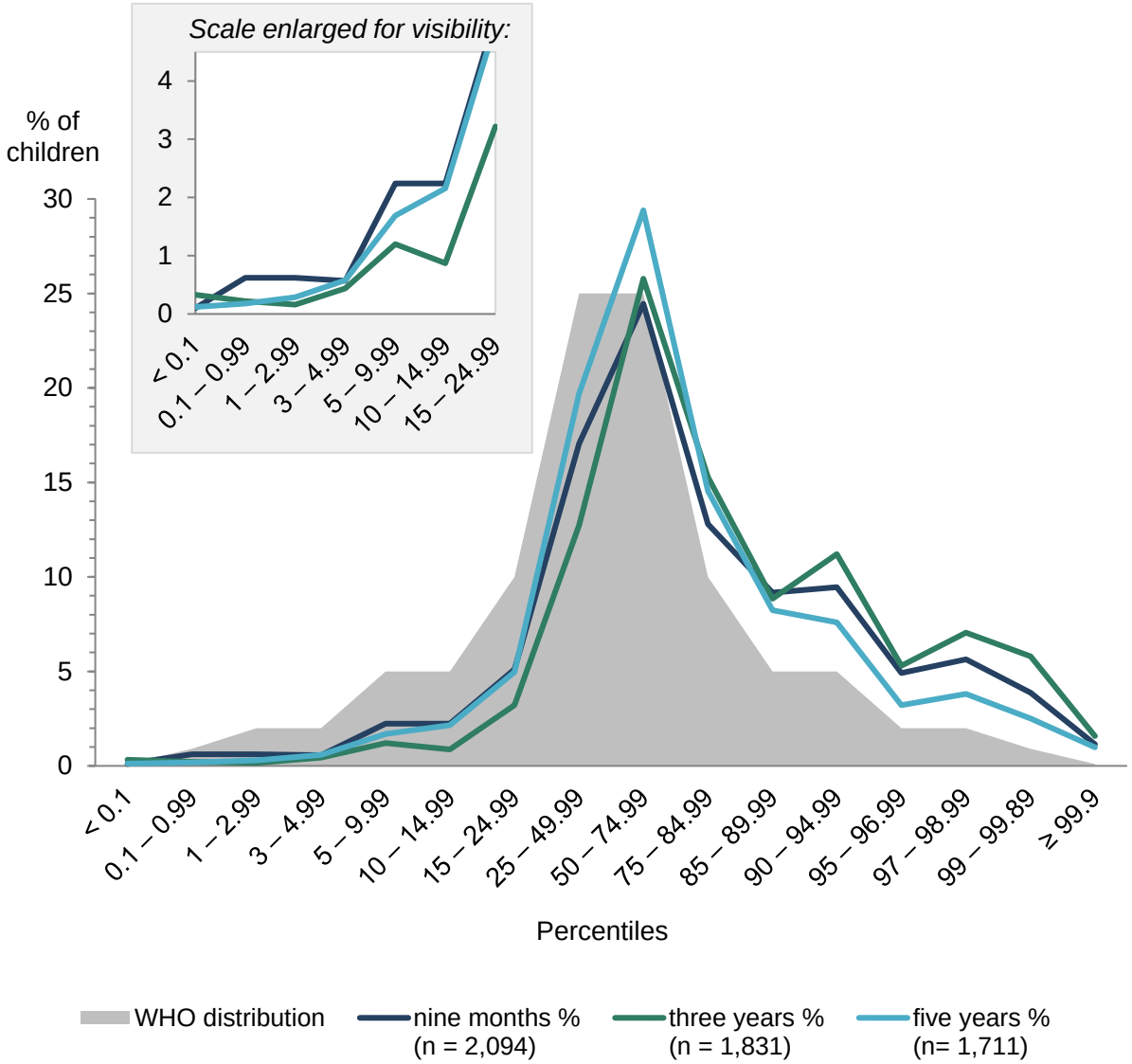

Figure 17: Distribution of body mass index (BMI) in children breastfed six months or longer only. BMI in the Growing Up in Ireland infant cohort was compared to the age- and sex-adjusted BMI percentiles defined by the World Health Organization (WHO) growth standard and growth reference.

Sex differences

Differences of prevalence between boys and girls can furthermore be seen in Figures 18-20. Significantly more girls than boys were under- or normal weight when they were three (51% of boys vs. 58% of girls) or five years old (66% vs. 72%), based on the WHO growth standard or growth reference, but significantly more boys were under- or normal weight if the IOTF growth reference was applied at five years (82% vs. 80%). Prevalence of overweight and of obesity were higher among boys than among girls using the different WHO criteria (Figures 18 and 19). At the ages of nine months and three years, the differences in overweight prevalence were significant, and at five years, overweight was significantly different using the WHO growth reference, only. However, at five years, obesity was significantly different irrespective of the WHO growth criterion used. Also, prevalence of risk of overweight was higher in boys using the WHO growth standard classification when the children were five years old (Table 15). At three years of age, the same sex trend was seen only for overweight using the IOTF growth reference although this was not significant (Table 15). At the age of five years, this trend was reversed and significantly more girls were overweight using the IOTF growth reference (Figure 20).

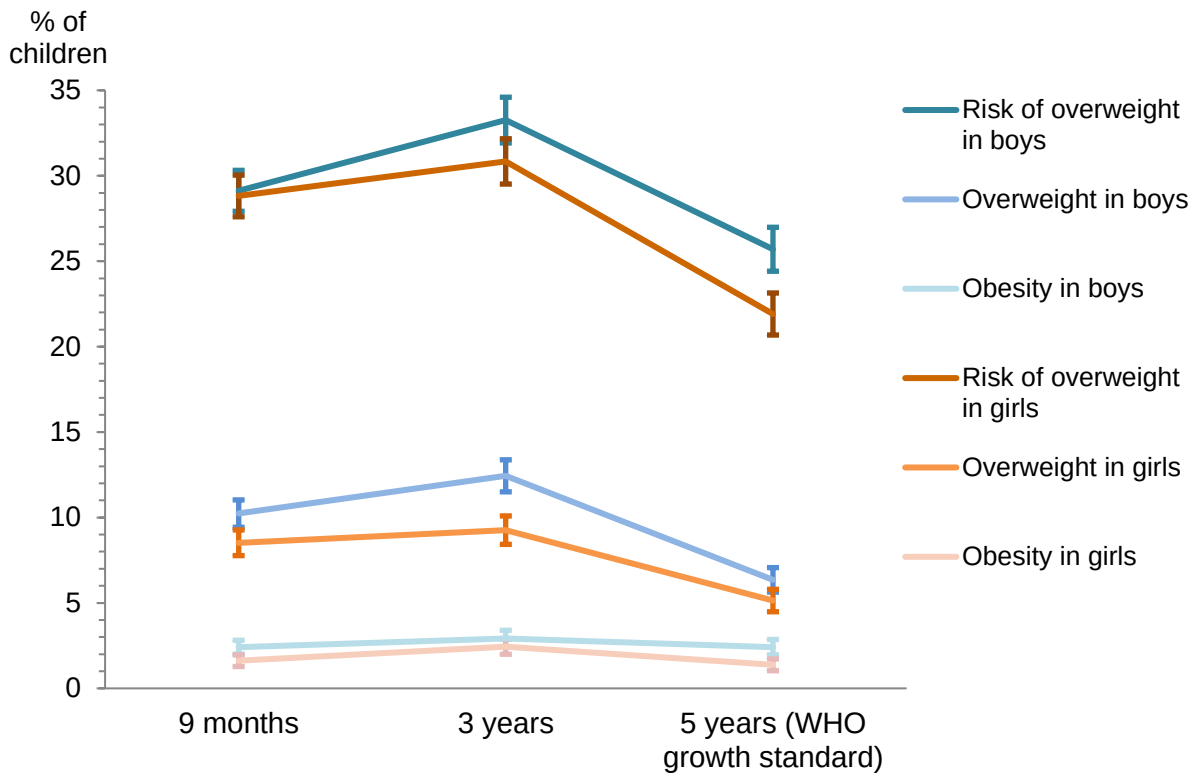

Figure 18: Weight status using the World Health Organization (WHO) growth standard classification. Prevalence of risk of overweight, overweight and obesity was estimated at nine months, three years and five years of age in boys and girls

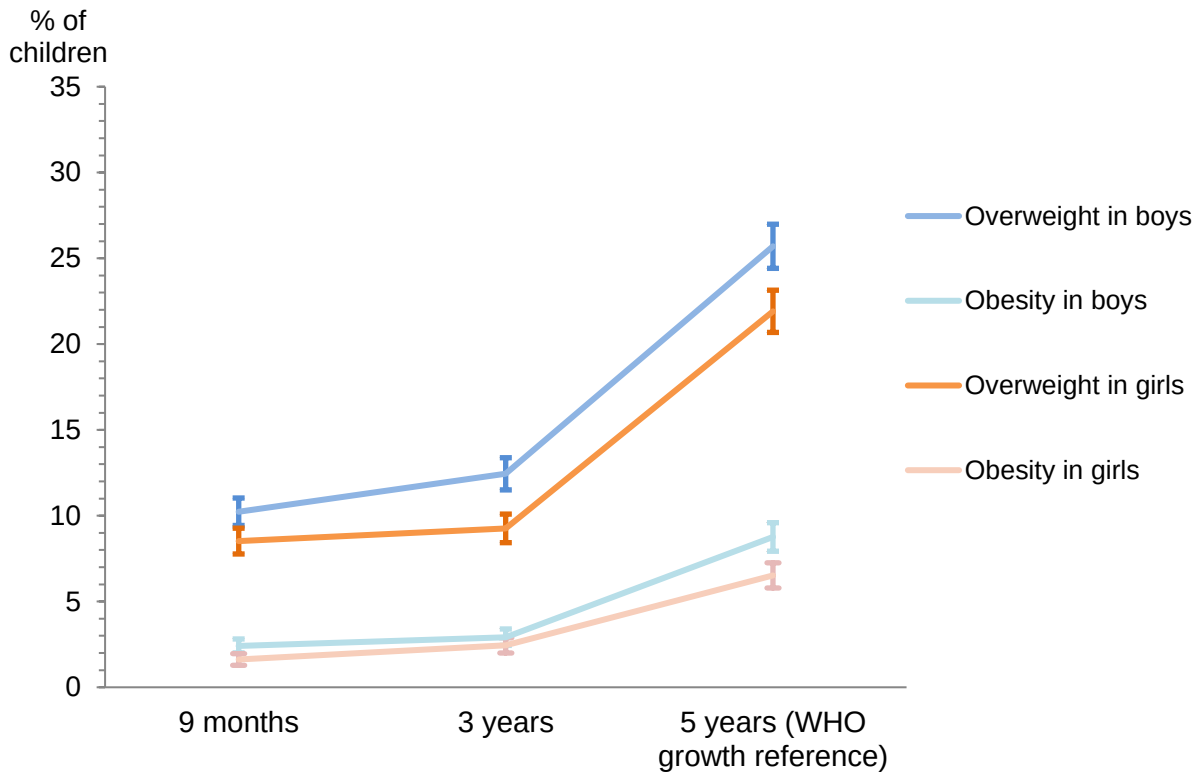

Figure 19: Weight status using the World Health Organization (WHO) growth standard and growth reference. Prevalence of overweight and obesity was estimated at nine months, three years and five years of age in boys and girls

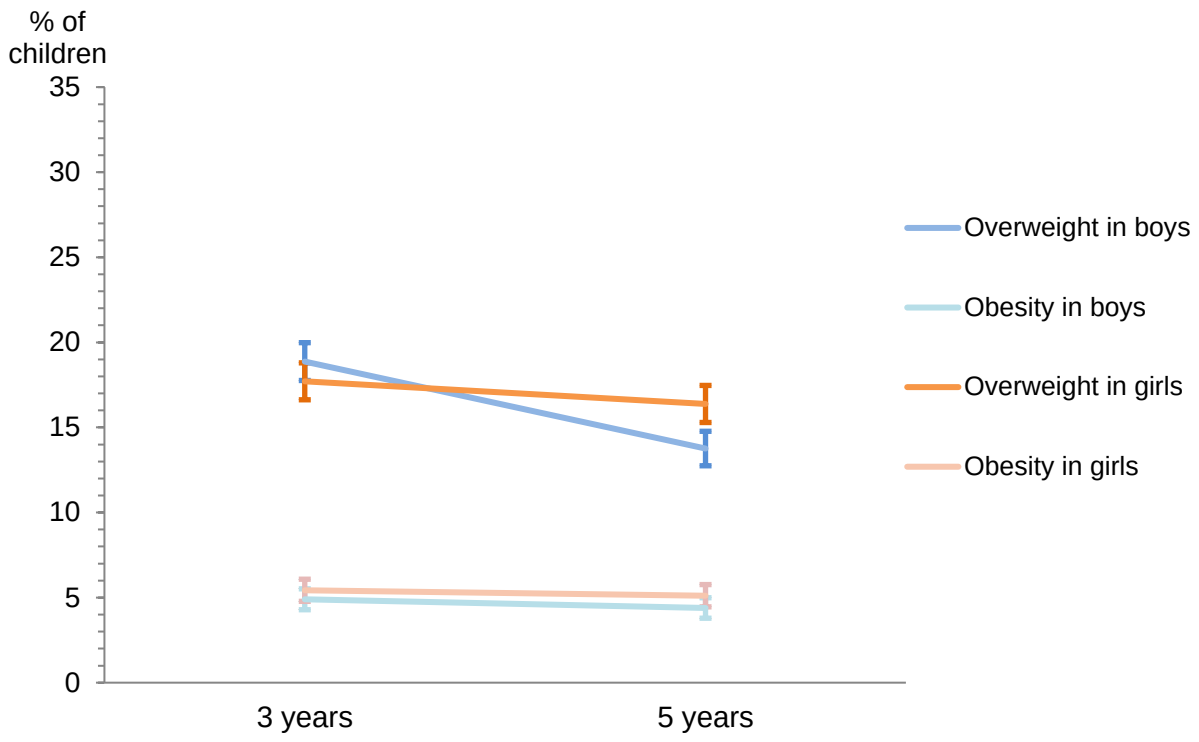

Figure 20: Weight status using the International Obesity Task Force growth reference. Prevalence of overweight and obesity was estimated at three years and five years of age in boys and girls

Table 15: Sex-differences in prevalence of weight categories in the Growing Up in Ireland infant cohort. Prevalence was estimated at nine months (n=10,810), three years (n=9,422) and five years of age (n=8,781), using the World Health Organization growth standard (WHO-S), International Obesity Task Force growth reference (IOTF) and World Health Organization growth reference (WHO-R) to classify body mass index. Differences in proportions, 95% Confidence Intervals (CI) and Bonferroni adjusted p-values are presented.

Child age		Under- and normal weight	Risk of overweight	Overweight	Obesity
9 months % (95% CI) N	**Boys WHO-S**	**58.2%** (56.9% to 59.5%) n=3,212	**29.1%** (27.9% to 30.3%) n=1,606	**10.2%** (9.4% to 11.0%) 564	**2.4%** (2.0% to 2.8%) 133
	Girls WHO-S	**61.0%** (59.7% to 62.4%) n=3,232	**28.8%** (27.6% to 30.3%) n=1,526	**8.5%** (7.8% to 9.3%) n=451	**1.6%** (1.3% to 2.3%) n=86
	Difference in proportions (95% CI) Adjusted p-value	*-2.8% (-4.6% to -1.0) p=0.06*	*0.3% (-1.4% to 2.0%) p=0.99*	*1.7% (0.6% to 2.8%) p=0.04*	*0.8% (0.3% to 1.3%) p=0.06*
3 years % (95% CI) N	**Boys WHO-S**	**51.4%** (50.0% to 52.8%) n=2,445	**33.3%** (31.9% to 34.6%) n=1,583	**12.4%** (11.5% to 13.4%) n=592	**2.9%** (2.4% to 3.4%) n=139
	Girls WHO-S	**57.5%** (56.0% to 58.9%) n=2,679	**30.8%** (29.5% to 32.3%) n=1,438	**9.3%** (8.4% to 10.1%) n=432	**2.4%** (2.0% to 2.9%) n=114
	Difference in proportions (95% CI) Adjusted p-value	*-6.1% (-8.1% to -4.1%) p=0.02*	*2.5% (0.6% to 4.3%) p=0.18*	*3.1% (1.8% to 4.3%) p=0.02*	*0.5% (-0.1% to 1.1%) p=0.99*
	Boys IOTF	**76.2%** (75.0% to 77.4%) n=3,628	N/A	**18.9%** (17.8% to 20.2%) n=898	**4.9%** (4.3% to 5.5%) n=233
	Girls IOTF	**76.9%** (75.6% to 78.1%) n=3,584	N/A	**17.7%** (16.6% to 18.8%) n=826	**5.4%** (4.8% to 6.1%) n=253
	Difference in proportions (95% CI)	*-0.7% (-2.4% to 1.0%) p=0.99*	N/A	*1.2% (-0.4% to 2.8%) p=0.99*	*-0.5% (-1.4% to 0.4%) p=0.99*

Child age		Under- and normal weight	Risk of overweight	Overweight	Obesity
	Adjusted p-values				
5 years **% (95% CI)** **N**	**Boys WHO-S**	**65.6%** (64.1% to 66.9%) n=2,910	**25.7%** (24.4% to 27.0%) n=1,141	**6.4%** (5.6% to 7.1%) n=282	**2.4%** (2.0% to 2.9%) n=107
	Girls WHO-S	**71.6%** (70.2% to 72.9%) n=3,107	**21.9%** (20.7% to 23.1%) n=951	**5.1%** (4.5% to 5.8%) n=223	**1.4%** (1.0% to 1.7%) n=60
	Difference in proportions (95% CI) ***Adjusted p-values***	*-6.1% (-8.0% to -4.2%)* *p=0.02*	*3.8% (2.0% to 5.6%)* *p=0.02*	*1.3% (0.3% to 2.3%)* *p=0.14*	*1.0% (0.4% to 1.6%)* *p=0.02*
	Boys IOTF	**81.8%** (80.7% to 83.0%) n=3,634		**13.8%** (12.7% to 14.8%) n=611	**4.4%** (3.8% to 5.0%) n=195
	Girls IOTF	**78.5%** (77.3% to 79.7%) n=3,408		**16.4%** (15.3% to 17.5%) n=711	**5.1%** (4.5% to 5.8%) n=222
	Difference in proportions (95% CI) ***Adjusted p-values***	*3.4% (1.7% to 5.1%)* *p=0.02*	N/A	*-2.6% (-4.1% to -1.1%)* *p=0.02*	*-0.7% (-1.6% to 0.2%)* *p=0.99*
	Boys WHO-R	**65.5%** (64.1% to 66.9%) n=2,910	N/A	**25.7%** (24.4% to 27.0%) n=1,141	**8.8%** (7.9% to 9.6%) n=389
	Girls WHO-R	**71.6%** (70.2% to 72.9%) n=3,107	N/A	**21.9%** (20.7% to 23.1%) n=951	**6.5%** (5.8% to 7.3%) n=283
	Difference in proportions (95% CI) ***Adjusted p-values***	*-6.1% (-8.0% to -4.2%)* *p=0.02*	N/A	*3.8% (2.0% to 5.6%)* *p=0.02*	*2.3% (1.2% to 3.4%)* *p=0.02*

4.5 Discussion

4.5.1 National and international child growth criteria

Child growth criteria were identified in this chapter that are used on a national or international level. While national growth criteria may be more suitable for the clinical assessment of individual children, these criteria are not particularly useful with regards to epidemiological surveillance of children on country-level or in cross-country comparisons. Mainly, use of various criteria leads to limitations in terms of the comparability of studies internationally (79). In order to monitor population trends across ages, time, and countries, the use of international, as opposed to national criteria, on population data is therefore recommendable (116, 327). Widely used international child growth criteria are the WHO growth standard, the WHO growth reference and the IOTF growth reference, whereas the CDC growth charts are a national growth criterion from the US occasionally applied to studies in other countries. On national level, the UK90/WHO growth charts are applicable to children in Ireland in clinical settings, however, these are currently subject to review and adjustment based on Irish data (80). The revised child growth criteria are expected for implementation in Ireland in the near future.

Previous studies identified discrepancies between OWOB estimates depending on which (international) child growth criterion was used to assess prevalence. Consequently, consensus is needed as to which criterion should be used in young children and, once established, this standard should be adhered to in the research field. An analysis of the literature revealed that use of the WHO growth standard for infants and young children (below five years) and the WHO growth reference in children aged five to 19 years is preferential. Namely, the BMI distribution in the WHO criteria was found to resemble the growth curves of healthy children most, the criteria were able to identify children who were at an increased cardiometabolic risk, and they identified OWOB earlier than other criteria (165, 336, 337).

The major critique of using the WHO criteria is their potential to underestimate the prevalence of underweight, compared to other growth criteria (377). While this argument has important implications for clinical application, particularly in countries

where undernutrition is a common health risk, the WHO growth standard identified young children with malnourishment and risk of undernutrition most accurately in one study (378). Furthermore, it needs to be acknowledged that the burden of childhood OWOB on individual health and on health systems appears much higher in Ireland and across Europe than that of underweight. Therefore, the WHO criteria are expected to classify the weight status of more children correctly than other criteria, even if a small proportion of underweight children may not be identified. For this thesis in particular, this criticism of the WHO criteria was not relevant as the investigation of underweight was not part of the study aim.

Additionally, it needs to be acknowledged that criteria published by the IOTF, which apply to children from the age of two years, were established before the WHO growth standard and reference were in place, and therefore use of the IOTF growth reference appears popular among researchers. Studies conducted prior 2006/2007 had no alternative option than to use the IOTF criteria or national references, which is reflected in the use of criteria among studies published until 2011 (302). However, even though use of the IOTF growth reference is still popular, a number of studies point to the fact that child growth criteria based on data collected in the 1970s or earlier may be outdated (164, 380).

In the light of missing consensus, a number of more recent studies have therefore moved to providing OWOB estimates using both IOTF and WHO criteria (239, 338, 382). This was recommended as a useful solution to making studies that monitor OWOB prevalence comparable both across time and countries (369). In fact, two large reviews of global childhood OWOB trends that were recently published are mainly distinguishable by whether studies were included that assessed weight status based on the IOTF (5) or the WHO growth reference (12). If prevalence according to both criteria were reported in future studies, there would be no need for this distinction and valuable research resources could be saved. Accordingly, this approach as well was supported and applied in this study.

4.5.2 Need for comparable criteria and terminology

A lack of consistency appears to exist in how the WHO growth standard and WHO growth reference are applied. The WHO supplies sex- and age-specific growth charts based on both criteria, displaying growth curves at the median, 85th percentile and 95th percentile, among others that are commonly used to assess OWOB in clinical practice (322) (Table 16). In fact, in a limited number of studies, the 85th and 95th percentile have as well been used a cut-off points to define OWOB in the research field (336, 337, 383). While it may not be incorrect to use the same cut-off criteria in research, the WHO provides thresholds based on SD for use in research specifically, as shown in Table 16. The proximity of 1SD to the 85th percentile and of 2SD to the 95th and 97th percentile lines may lead researchers to conclude incorrectly that both can be used interchangeably.

Furthermore, not all studies adhere to the different interpretation of SD thresholds between the WHO growth standard and the WHO growth reference. For example, commonly 1SD was used as the threshold to define overweight in children below the age of five years (239, 382) whereas 2SD would be correct, as shown in Table 16. As both WHO growth criteria follow on from different study designs and partly different populations, it appears reasonable that these are kept separate by the WHO. However, especially as no difference between the two WHO criteria exists for the clinical assessment, instructions on the WHO website may need to clarify the different meaning of SD thresholds in a more transparent way, so that use of the criteria and cut-off points might be confused less in future studies.

Despite the preference of WHO growth criteria over national or IOTF criteria, this difference in interpretation of epidemiological thresholds was found to offer a major disadvantage when children were monitored longitudinally who span across ages that require use of both the WHO growth standard and the WHO growth reference (114, 277). This is the case in the GUI infant cohort, where children fall into the categorisation based on the WHO growth standard when they are nine months and three years old, but into that of the WHO growth reference during the third BMI measurement at their age of five years. According to the thresholds seen in Table 16, a child that would be classified as overweight at the age of four years and eleven months would be classified as obese at the age of five years and zero

months if they stayed on the same percentile of the respective BMI distribution of the WHO growth standard and the WHO growth reference. In terms of percentiles, the transition from the WHO growth standard to the WHO growth reference has been excellently smoothed (277), but unfortunately this is not the case for definitions of OWOB based on SD.

Table 16: Thresholds of the World Health Organization (WHO) child growth criteria (114, 322, 361)

Thresholds	**WHO growth standard** (0-59 months)	**WHO growth reference** (5-19 years)
For clinical assessment		
85th percentile	Overweight	Overweight
95th percentile	Obesity	Obesity
For epidemiological studies		
1SD	Risk of overweight	Overweight
2SD	Overweight	Obesity
3SD	Obesity	Severe obesity

In order to prevent such difficulties, Rolland-Cachera et al. (116) propose the implementation of a more uniform terminology across child growth criteria, which would define all children currently categorised as at risk of overweight in the WHO growth standard, and as overweight in the WHO and IOTF growth references, as ‘grade 1 overweight’. Furthermore, children currently classified as overweight based on the WHO growth standard and those classified as obese based on the two growth references would be caught under a common category ‘grade 2 overweight’ or ‘obesity’ (116). Similarly, the former classification of children as ‘at risk of overweight’ and ‘overweight’ using the CDC criteria was changed to classifying children in the same percentile groups as ‘overweight’ and ‘obese’, respectively, because the original terms were not believed to reflect the actual health risk associated with children’s BMI (384). In terms of the WHO growth standard, however, it was noted that the current definitions may in fact be preferential in the communication between healthcare providers, parents and children, as well as in the media, to avoid stigmatisation (116). As a consequence

of the suggested adjustment in the terminology of OWOB for research purposes, SD in the WHO growth standard and growth reference would become comparable across ages and correct use of the WHO criteria may be encouraged in studies involving children that span across early and later childhood and adolescence.

4.5.3 Prevalence of overweight and obesity in the Growing Up in Ireland infant cohort

The WHO criteria and the IOTF growth reference were used to estimate the prevalence of OWOB in the GUI infant cohort. While only the WHO growth standard was available to assess prevalence when the children were nine months old, the IOTF growth reference was used as an alternative criterion at the ages of three and five years. At five years of age, both the thresholds of the WHO growth reference and WHO growth standard were applied as children had just crossed the age when BMI assessment should be shifted from the former to the latter.

Large discrepancies were found between prevalence estimates of overweight and of obesity depending on which child growth criteria were used. When the children were nine months old, 29% were at risk of overweight, 9.4% overweight and 2% obese. The WHO growth standard identified 32.1% of children as at risk of overweight at three years and 23.8% at five years. Additionally, 10.9% of the three-year-olds and 5.8% of the five-year-olds were overweight, and 2.7% and 1.9% of the children were obese at the respective ages. Hence, between the ages of three and five years, prevalence tended to decrease if the WHO growth standard classification was used, or if percentiles based on the applicable WHO criteria for the respective ages were compared; however it increased dramatically if the WHO growth reference was used at five years. When the WHO growth reference was applied to the five-year-old children, estimates of both overweight (23.8%) and obesity (7.7%) were highest, compared to the other criteria. The IOTF growth reference overall found higher levels of overweight and of obesity than the WHO growth standard, but lower levels than the WHO growth reference, which has also been observed in other studies (239). Based on the IOTF growth criteria, 18.3% of three-year-olds and 15.1% of five-year-olds were overweight and 5.2% and 4.8%

were obese, respectively. Generally, trends over time in the GUI infant cohort appeared more stable over the study period using the IOTF growth reference than using WHO criteria, which is in line with other studies (239, 338).

Both for overweight and obesity, these estimates lie below those that were published in the Global Burden of Disease Study (using the IOTF growth criteria), data of which were not based on real observations but calculated from prevalences seen in other age groups and other countries (5). In contrast, this thesis confirms the prevalence of OWOB observed in the small sample of three-year-old children assessed in the National Preschool Nutrition Survey, even though the obesity prevalence was slightly lower in that survey. Prevalence of OWOB in that survey was furthermore lower at the ages of two and four years (98). A lower prevalence of OWOB (14%) was also seen in an urban, regional (non-representative) sample of two-year-old children in Ireland (40). Additionally, the children in the GUI infant cohort were born around the same time as the children examined in Round 4 of the COSI, who were six to seven years old in 2015. When both cohorts are compared, the prevalence of obesity is the same, however less children (12%) were overweight in the COSI (356) and the sex gap was much bigger, even though sex trends were similar (41). The declining overweight trend seen between the ages of three and five years in the GUI data may therefore have continued in Irish children that were born approximately a decade before the writing of this thesis, but more so in boys. In contrast, since the beginning of the millennium, a small but continuous decline in the obesity, but not the overweight prevalence, was seen based on national data from primary school children. The overweight prevalence in the same study remained relatively stable over time (38).

A comparison to other countries revealed that the prevalence of OWOB appears to vary between countries within Europe (239). The prevalence of obesity identified in this study using the IOTF growth reference approximates the global average in two- to four-year-olds in 2011 to 2013; however the prevalence of overweight is much above the global average of about 10% (5). In turn, a very similar prevalence of overweight and of obesity to the global average was previously seen in young children in Belgium, Cyprus and Italy, even if findings are based on cross-sectional studies and slightly older (385). In France, prevalence of overweight among two-

year-old children, using the IOTF criteria, was much lower than in Ireland (between 5% and 6%) from 2004 to 2013 (379). Much lower levels than in our study were also identified in infants and five-year-old children in Denmark. A pooled analysis of Danish infants and children revealed that the overweight prevalence in infants from 1998 until 2010 was between 1.2 and 7.3%, and obesity prevalence between 0 and 1.2% using the WHO growth standard. Overweight and obesity combined was prevalent in less than 7% of boys and 5% of girls who were eight to ten months old and studied at the same time as the GUI infant cohort (386). Again, this is much lower than the estimates found in this study. Lower levels of OWOB as well were seen in preschool-aged children from the Netherlands, using the IOTF growth reference and the WHO growth standard, and in two-to five-year old children from Norway, using the IOTF growth reference (79).

Similar OWOB levels in a comparable study period were seen in representative studies from Portugal (three- to four-year-old children) using the IOTF criteria, and in Turkey (zero- to four-year-old children) using the WHO growth standard (79). In turn, higher levels of OWOB were found using IOTF and WHO criteria in Greek preschool children that were studied in 2009 and 2010 (382). The differences seen in Greece between OWOB estimates using the IOTF growth reference and the WHO growth reference classification compare to the differences found in this study (382). Also, slightly higher obesity levels (16%) were found among two- to five-year-old children in Massachusetts between 2001 and 2006, using the WHO growth standard (337).

In a number of previous studies, discrepancies between estimates of overweight and/or obesity were declining over time, and more agreement between WHO growth criteria and the IOTF growth reference was seen as children grew older in many countries (239, 337), however not in all (387). In this thesis, irrespective of using the WHO growth reference or WHO growth standard classification when children were five years old, gaps between either of these criteria with the IOTF estimates of overweight and of obesity remained rather stable over time. A potential reason that was considered to explain this is the fact that other studies used the WHO growth reference classification also in children below the age of five years, or the 85th and 95th centile of the WHO growth standard or growth reference

instead of SD. In this thesis, overweight based on the WHO growth reference classification would have been 32% at the age of three years, and prevalence of obesity nearly 11%. In fact, the gap between the WHO growth reference and the IOTF growth reference decreases until the age of five years if this unofficial classification system is applied to the GUI data, whereas this finding was not confirmed by trends seen in Greek preschool children (382). An additional reason may be that children in this cohort were only followed until their age of five years, whereas prevalence estimates based on the WHO and IOTF criteria diverged less from around the age of eight years in a previous study (239). It is possible that the gap between different estimates of OWOB may narrow down also in the GUI cohort as children grow older.

Many studies identified higher agreement between estimates of overweight (including obesity) following various definitions of the different child growth criteria, compared to a lower agreement identified between estimates of obesity (336, 373, 374, 376). Considering that the use of child growth criteria varies considerably based on preferences of researchers and age groups included in studies, more agreement in the field of childhood obesity research may therefore be reached studying trends of OWOB combined. Particularly in the investigation of risk factors for the development of evidence-based interventions, and in the evaluation of such interventions, OWOB estimates may be more comprehensive. Nevertheless, obesity should also continue to be measured for overall population monitoring and clinical assessment of child weight due to its important health implications.

4.5.4 Sex difference in the overweight and obesity prevalence

Notably, sex trends of obesity were reversed at three and five years of age, as were overweight trends when the children were five years old between the criteria. In the GUI infant cohort, the WHO growth standard classification consistently identified a higher number of boys than girls as overweight and as obese at all ages (not statistically significant in the obesity category at nine months and three years), while the IOTF growth reference identified a significantly higher number of girls than boys as overweight at five years. In turn, similar to the WHO growth

standard, more boys were identified as overweight using the IOTF growth reference at three years, but this difference was not significant. The sex trend observed using the WHO growth reference and the WHO growth standard classification were comparable. Longitudinal data from eight European countries in the IDEFICS cohort also found higher OWOB levels among boys than girls using WHO growth criteria and higher levels among girls using IOTF criteria from the age of two to seven years (239). Similarly, sex trends of obesity in Sweden were reversed between the IOTF and the WHO growth reference (380). In Greece, however, the only significant sex trends for OWOB using either the IOTF or the WHO growth reference identified a higher prevalence in boys than in girls (382).

4.5.5 Conclusion

The prevalence of OWOB in young children in Ireland appears high compared to many European countries (79, 379, 385, 386), with a peak prevalence of up to 46% (WHO growth reference classification) observed at the age of three years. Estimates need to be interpreted with caution as large variations were seen depending on the child growth criterion in use. In order to promote future decreases in the OWOB prevalence among young children in Ireland, factors that contribute to the development of OWOB and the maintenance of a healthy weight need to be investigated. Use of the WHO child growth standard and child growth criteria in combination, but using unique SD thresholds, is recommended to investigate risk factors. Therefore, in the remainder of this thesis, OWOB will refer to a BMI > 1SD and obesity to a BMI > 2SD. Accordingly, the next chapter of this thesis presents results from cross-sectional analysis of various risk factors of OWOB in the GUI infant cohort.

CHAPTER 5:

Cross-sectional analysis of early overweight and obesity – inclusion of the Social Ecological Model

Key points in this chapter

Aims of this chapter

- To investigate differences in the risk of childhood OWOB in relation to risk factors and between population groups in Ireland at the ages of nine months, three years and five years
- To test the role of material circumstances, biological risk factors and psychosocial factors in the development of OWOB in young children while applying a suitable child growth criterion

Knowledge generation

- Using a combination of the WHO growth standard and WHO growth reference, risk of OWOB (combined) and risk of obesity in young children in Ireland will be quantified for different population groups and material, biological and psychosocial risk factors are linked to childhood OWOB

5.1 Introduction

A high prevalence of OWOB until the age of five years in Ireland was identified in Chapter 4, with the highest level seen in three-year-old children. Large variations in the OWOB prevalence were observed over time, and differences exist between the prevalence in young children in Ireland and those in other countries (Chapter 4). In order to detect how such differences may arise, different paths need to be explored which may explain why children put on excess weight. A number of theories were described in Chapter 2 which explain how inequalities in health emerge, and possibly the development of OWOB. One theory focuses on the possibility that material circumstances impact the family's ability to afford and access resources and services that support the maintenance of a healthy child weight. This includes resources in the physical environment of children, such as safe outdoor areas to play active games, as well as health knowledge, such as parental cooking skills and nutritional knowledge. Secondly, biological risk factors are suspected to play a role in the development of OWOB, for example through (epi-)genetic mechanisms and early feeding practices. Thirdly, families may be more likely to adopt unhealthy behaviours if they are suffering from psychosocial strain, which may as well affect the retention of a healthy weight in young children.

The environment of children until the age of five years mainly constitutes of the family they live in, and, to a lesser extent, the community (e.g. childcare services), social policy (e.g. through its influence on healthcare) as well as socio-economic factors that impact on the family as a whole. The SEM has divided the obesogenic environment of children into these various levels and the *Complex web of potential determinants of overweight and obesity in children* has further helped researchers explain how various child, family or community, demographic and societal characteristics may be related to childhood obesity. Both frameworks have been combined and adopted to guide this thesis, as explained in Chapter 2 (Section 2.2). While an extensive array of literature was furthermore identified in Chapter 2 that supports the links between many child, family and community, demographic and societal characteristics with early childhood obesity, little is known about the risk factors of young Irish children specifically.

A number of studies in Ireland have assessed risk factors of childhood obesity using nationally representative data in the past, of which very few focused on children at their age of three years (2, 238) and most on school-aged children (92-94, 232, 346, 349, 388, 389). Children below the age of three years and between three and six years therefore appear understudied in Ireland. However, international research revealed that the first 1,000 days of children's lives appear crucial in the development of OWOB, and that weight gains before school entry have important implications for the later obesity risk, as shown in Chapter 1 (Section 1.3). Furthermore, one of the Irish studies used clinical (WHO growth standard), rather than epidemiological criteria to define OWOB (2) and all other studies made use of the IOTF growth reference. The IOTF growth reference is not applicable to children below the age of two years, and the critical analysis of various child growth criteria presented in Chapter 4 identified use of the WHO growth standard and WHO growth reference as preferential to investigate children's weight status.

No population-level study is known that provides a recent comparison of risk factors that are related to the development of OWOB at different ages across the early childhood years, using a uniform and suitable definition of OWOB and adjusting for the same risk factors across ages. In order to tackle the high OWOB levels in Ireland, population groups who are at an increased risk of childhood obesity, as well as material, biological and psychosocial risk factors that contribute to this risk, need to be identified. Only upon the identification of families who are at risk of having young children with OWOB, prevention and intervention strategies can be developed that target those families who are most in need.

5.2 Chapter aims and hypotheses

A research gap has been identified in Ireland, where many young children have OWOB as early as at the age of nine months, but where factors explaining this high prevalence at that age and in the later preschool years have not been studied in detail. Therefore, the aim of this chapter is to identify characteristics of children, their families, and the wider community they live in that increase the likelihood of OWOB when they are nine months, three years and five years old. Three hypotheses were tested that may explain how early childhood OWOB develops in Ireland. First, it was hypothesised that low familial socio-economic conditions lead children to an elevated risk of developing OWOB due to material circumstances. This relationship may be explained by the family's ability to afford certain goods and services that help them promote a healthy child weight. Secondly, biological risk factors were hypothesised to predispose children to develop OWOB, for example by influencing their metabolism and growth through the gestational development and early nutrition. Thirdly, psychosocial factors were hypothesised to promote excess weight gain in children, for example psychosocial stressors in the child's environment may lead to the adoption of unhealthy feeding practices and increased screen use. These hypotheses were tested using data from the GUI infant cohort, which was described in detail in Chapter 3.

5.3 Methods

The GUI infant cohort data were analysed using a mix of simple and multiple logistic regression models to investigate potential risk factors and population characteristics associated with OWOB in children until the age of five years.

5.3.1 Variables

Outcome variables

Weight status at the ages of nine months, three years and five years in the GUI infant cohort was assessed based on BMI, using the WHO growth standard and the WHO growth reference which has been described in Chapter 4. Based on this categorisation of BMI in relation to the WHO growth criteria, two binary outcome variables were investigated at the ages of nine months, three years and five years; first, children with OWOB (BMI > 1SD) were compared to children with under- or normal weight, and secondly, obesity (BMI > 2SD) was used as an alternative outcome variable (compared to children in all other weight groups), on the sex-and age-specific distribution of the applicable WHO criterion. This is the classification system used in the WHO growth reference, while the WHO growth standard suggests use of alternative thresholds. However, for practical reasons, the above thresholds have been adopted in this thesis as one common definition to classify weight categories across the ages, as has been suggested by Rolland-Cachera et al. (116) (see Chapter 4 for further detail).

Use of overweight as the outcome variable was favoured over the use of obesity as outlined in Chapter 4. Nevertheless, obesity was used as an alternative outcome variable for two reasons. First, the threshold of 2SD includes children with the highest BMI scores, only, which may be associated with other risk factors than the wider inclusion criteria of BMI > 1SD. Secondly, as shown in Chapter 4, the WHO originally used the threshold of 2SD to define overweight in children under the age of five years, and it was expected that the study of weight status according to either threshold may reveal different important findings. Lastly, use of BMI > 3SD as a

third outcome variable was considered, because this defines obesity in the original classification of the WHO growth standard for children under the age of five years. However, only 2% of all BMI observations across the studied ages fall into this category. Therefore, use of this threshold to define obesity would have reduced the statistical power of the analysis.

Explanatory variables

Differences in OWOB prevalence and obesity prevalence were investigated based on a number of child, family as well as community, demographic and societal characteristics. This was to test whether material circumstances, biological factors or psychosocial and associated behavioural factors could play a role in the development of obesity in young children in Ireland. In Chapter 2, ethnic background and various SES indicators were identified as main demographic population characteristics based on which inequalities in childhood obesity exist; hence, they were included as explanatory variables in this study. Furthermore, as detailed in Chapter 2, consensus appeared to exist that parental weight status is related to children's obesity levels, as well as biological infant characteristics, such as BWFGA, and initiation and duration of breastfeeding. These factors were also incorporated in the analysis. Other variables, such as behavioural factors and aspects of the social and physical environment of children, were additionally included, based on the SEM and the *Complex web of potential determinants of overweight and obesity in children*, and based on the evidence presented in Chapter 2. An overview of all explanatory variables investigated in this Chapter and their measurement can be found in Chapter 3 (Section 3.3.3, Table 4).

5.3.2 Statistical analysis

Initially, descriptive statistics, including means and SD, and frequencies and percentages, as described in Chapter 3 (Section 3.3.3), were performed.

Logistic regression analysis

Initially, simple logistic regression analysis was used to investigate relationships between the risk of OWOB in the GUI infant cohort and material, biological or psychosocial theories at the different ages. Associations between weight status and equivalised household income were investigated at the ages of nine months, three years and five years, as household income seemed the most logical indicator to test the material theory. Additionally, associations between weight status and BWFGA were investigated at all three ages, to test whether biological factors are associated with differences in OWOB, and furthermore between household structure and weight status, to test the possibility of psychosocial pathways to OWOB. However, bivariate analysis does not account for potential confounding that may exist due to relationships between explanatory variables and hence multivariable analyses approaches were also explored, following simple linear regression analysis.

Logistic regression models, which investigate the probability of a binary outcome variable, based on one or more potential explanatory variables, were used. Cross-sectional multiple logistic regression analysis was conducted at the ages of nine months, three years and five years, to adjust for multiple OWOB risk factors simultaneously, i.e. to assess the association between variables, if all other explanatory variables in the model were held constant. Separate logistic regression models were run for each age and both outcome variables (OWOB and obesity). All explanatory variables outlined above were integrated into the analysis, in order to identify which factors and population characteristics were associated with varying odds of OWOB or obesity at the different ages. A number of lifestyle variables were only measured at the ages of three and/or five years, which is why they were only included in the respective models. Furthermore, at the ages of three and five years, previous weight status (ó*under- and normal weight*ô, ó*overweight*ô, or ó*obese*ô) was included as an explanatory variable in order to isolate the association of other variables with previous weight status from associations between explanatory variables and weight status at the investigated age. Sensitivity analysis was run for all models without adjusting for previous weight status, and without adjusting for rapid infant weight gain, as these variables may have mediated other

relationships and hence masked their association in the models (390). Weighting factors developed for each age as part of the GUI study were included in the models.

A general limitation of cross-sectional analysis that was mentioned in Chapter 2 is that no causal inferences are possible when outcome and explanatory variables are measured simultaneously, as it is unknown whether explanatory variables preceded the outcome. While this limitation needs to be acknowledged, a few explanatory variables in this thesis, namely birth characteristics, infant weight gain and infant feeding practices, were present before BMI was assessed first at the age of nine months. Additionally, as multiple waves of data are available to this study, the analysis of waves 2 and 3 could be adjusted for the outcome variable in the previous wave. For example, if an association between breastfeeding duration and OWOB at the age of five years was seen, this association could be argued to be mediated by the child's earlier weight status when the model was not adjusted for previous weight status. However, by conducting a longitudinal analysis, adjusting the five-year analysis for OWOB at three years, weight status at three years is held constant and the association between breastfeeding duration and OWOB at five years is tested. Hence, the longitudinal analysis can shed more light on the relationships between explanatory variables and OWOB.

While a sophisticated longitudinal analysis is possible and was conducted using the GUI infant cohort data (results will be presented in Chapter 6), there are relatively few longitudinal studies in young children available. In the past, cross-sectional designs have widely been used across countries to investigate risk factors of childhood obesity and therefore offer a higher level of comparability between studies. Novel knowledge based on longitudinal studies is needed, but simultaneously new findings need to be bridged to past research in order to compare trends over time and between countries, and to build on past research, regardless of the analysis approach chosen. For example, in Ireland, the COSI data of children aged six years and older consists of both cross-sectional and longitudinal elements, as new cohorts of six-year-old children are sampled and children within each cohort reassessed every two years (41). While children in the COSI cohorts are followed until an older age than the children in the GUI infant

cohort (41), a comparison of the five-year data from GUI and the six- to seven-year data from the COSI appears feasible due to the small age gap between them and offers opportunities to test the external validity of both studies.

If the results of the cross-sectional analysis of the GUI data are comparable to those found in the COSI (and other studies) it would suggest that the longitudinal findings in this study may be relevant to other countries. Furthermore, if similar cross-sectional findings occur in both studies, a longitudinal analysis of the GUI infant cohort can help explain or validate pathways that may lead to these cross-sectional associations in both cohorts. The interpretability of findings in the COSI children (and many other studies) is otherwise limited as they were not studied during infancy. Therefore, while a longitudinal analysis is preferable over several cross-sectional analyses, cross-sectional analysis was also conducted in this thesis for comparison to other studies.

5.4 Results

5.4.1 Overview of variables and exploratory analysis

The data cleaning and exploratory analysis led to the final set of variables that was integrated in the analyses of this thesis, details of which are presented in Table 17.

There was almost an even split of boys and girls and those living in a rural or urban area. On average, children met about half of the food recommendations and received antibiotics more than once in the year preceding the three- and the five-year data collection. More than half of the children participated in organised sports and children tended to pursue all of the different unstructured physical activities about half of the days. Most three- and five-year-old children spent between one and two hours per day using screens, however 20% at three years and 13% at five years spent more than three hours using screens. At all ages, most children had a normal daily sleep duration, however no direct comparison is possible as sleep recommendations vary by ages and altered categories were used in this study (Table 17).

Most children (82%) were born on time and 11% late. Furthermore, 77% of children were born AGA, with an almost even split of SGA and LGA-born children. One in four children had a rapid gain in weight between birth and the age of nine months. While more than half of the children were breastfed, only one fifth of children were breastfed for the recommended length of six months or longer. The most common age for children to be introduced to solid food was between four and six months (55%), however significant proportions of children were introduced to solid foods earlier (15%) or later (30%) (Table 17). Among those caregivers who had their BMI reported, over 50% of PCGs were normal or underweight in wave 1; however only 30% of SCGs were. In both caregivers, proportions decreased marginally across waves.

The majority of children were Irish white (67%), were in the ‘other’ group (10%), which constituted mainly of mixed background children, or could not be grouped because a SCG lived in the house but no interview had taken place and hence only incomplete data were available (10%). In all waves, most study children lived in

families with two caregivers and at least one other child; however the proportion increased from 55% in wave 1 to 80% in wave 3 as more children were born into single-child families. The proportion of single caregiver families was relatively stable over time. In all waves, nearly one third of children lived in families that belonged to the managerial and technical household class, with relatively even splits of families in the professional, non-manual and skilled-manual class and fewer families in the lower classes. The mean equivalised annual income of families decreased from more than EUR 21,000 in wave 1 to above EUR 18,000 in waves 2 and 3 (Table 17). Sub-sample analysis with families that participated in all waves revealed that this decrease was not due to attrition (results not shown) and it is likely that this occurred due to the economic recession that hit Ireland just after the wave 1-data were collected (391). More details on household income can be found in Appendix 3.5. Similarly, as can be seen in Table 17, the proportion of caregivers with third level education increased over time (from 44% to 48%) but sub-sample analysis revealed that this difference was not attributable to attrition (results not shown). Rather, a number of PCGs (n=368) but mostly SCGs (n=793) acquired third level education between the first two waves, or had no data recorded in wave 1 (n=131).

Most PCGs agreed that their neighbourhood was safe in waves 1 and 2 (54% and 52%) and most strongly agreed to this in wave 3 (58%). Similarly, most PCGs agreed that the neighbourhood offered green and play spaces throughout waves (38% to 43%) but the proportion of PCGs who strongly agreed to this increased in wave 3. Most children were not in a childcare arrangement throughout waves (49% to 61%). Of children who were in childcare regularly, more were in informal childcare arrangements in waves 1 and 3 and more in formal childcare in wave 2 (Table 17).

Table 17: Descriptive statistics of explanatory variables: ranges, means and SD (SD) are provided for continuous variables, and median and interquartile ranges for skewed continuous variables. Frequencies and proportions are provided for categorical variables.

Variable	Wave	N [a]	Missing in wave (%) [b]	Range	Mean (SD)	Categories/ Interquartile range	Frequencies (%) [c]
Sex	1	11,134	0			Girls Boys	5,455 (48.99%) 5,679 (51.01%)
	2	9,793	0			Girls Boys	4,826 (49.28%) 4,967 (50.72%)
	3	9,001	0			Girls Boys	4,433 (49.25%) 4,568 (50.75%)
Number of food recommendations	3	8,998	3 (0.03%)	1 - 6	3.26 (1.82)		
Number of antibiotic courses	2	9,774	19 (0.19%)	0 - 20	1.46 (1.93)	1st quartile: 0 Median: 1 3rd quartile: 2	
	3	8,986	15 (0.17%)	0 - 20	1.19 (1.82)	1st quartile: 0 Median: 1 3rd quartile: 2	
Unstructured physical activity	3	8,920	81 (0.90%	1 - 6	3.80 (1.47)		
Sports club use	3	8,986	15 (0.17%)			Never twice/months 1 hour/week 2 hours/week >2 hours/week	4,391 (48.86%) 193 (2.15%) 2,614 (29.09%) 1.160 (12.91%) 628 (6.99%)
Daily screen time	2	9,783	10 (0.10%)			0 to <1 hour 1 to <2 hours 2 to < 3 hours 3 or more	1,397 (14.28%) 3,627 (37.07%) 2,807 (28.69%) 1,952 (19.95%)
	3	8,999	2 (0.02%)			0 to <1 hour	241 (2.68%)

Variable	Wave	N [a]	Missing in wave (%) [b]	Range	Mean (SD)	Categories/ Interquartile range	Frequencies (%) [c]
						1 to <2 hours	5,078 (56.43%)
						2 to <3 hours	2,505 (27.84%)
						3 or more	1,175 (13.06%)
Daily sleep duration	1	11,095	39 (0.27%)			<12 hours	2,103 (18.95%)
						12 to <15 hours	7,668 (69.11%)
						≥15 hours	1,324 (11.93%)
	2	9,766	27 (0.27%)			<11 hours	1,154 (11.82%)
						11 to <13 hours	7,285 (74.60%)
						≥13hours	1,327 (13.59%)
	3	8,974	27 (0.30%)			<11 hours	1,767 (19.96%)
						11 to <12 hours	5,118 (57.03%)
						≥12 hours	2,089 (23.28%)
Primary caregiver weight status	1	10,595	539 (4.84%)			Normal or underweight	5,710 (51.28%)
						Overweight	3,158 (28.36%)
						Obese	1,727 (15.51%)
						Missing	539 (4.84%)
	2	9,274	519 (5.30%)			Normal or underweight	4,726 (48.26%)
						Overweight	2,893 (29.54%)
						Obese	1,655 (16.90%)
						Missing	519 (5.30%)
	3	8,418	583 (6.48%)			Normal or underweight	4,193 (46.58%)
						Overweight	2,571 (28.56%)
						Obese	1,654 (18.38%)
						Missing	583 (6.48%)
SCG weight status	1	10,769	365 (3.28%)			Normal or underweight	2,531 (22.73%)
						Overweight	4,093 (36.76%)
						Obese	1,643 (14.76%)
						No SCG interview [d]	1,143 (10.27%)
						No SCG [e]	1,359 (12.21%)

Variable	Wave	N [a]	Missing in wave (%) [b]	Range	Mean (SD)	Categories/ Interquartile range	Frequencies (%) [c]
						Missing	365 (3.28%)
	2	9,263	530 (5.41%)			Normal or underweight	1,925 (19.66%)
						Overweight	3,581 (36.57%)
						Obese	1,541 (15.74%)
						No SCG interview [d]	999 (10.20%)
						No SCG [e]	1,217 (12.43%)
						Missing	530 (5.41%)
	3	8,230	771 (8.57%)			Normal or underweight	1,712 (19.02%)
						Overweight	2,996 (33.29%)
						Obese	1,273 (14.14%)
						No SCG interview [d]	1,157 (12.86%)
						No SCG [e]	1,092 (12.13%)
						Missing	771 (8.56%)
Gestational age	1	11,094	40 (0.36%)			Very early	148 (1.33%)
						Somewhat early	589 (5.31%)
						On time	9,097 (82.00%)
						late	1,260 (11.36%)
	2	9,759	34 (0.35%)			Very early	130 (1.33%)
						Somewhat early	493 (5.05%)
						On time	8,036 (82.34%)
						late	1,100 (11.27%)
	3	8,975	26 (0.29%)			Very early	118 (1.31%)
						Somewhat early	447 (4.98%)
						On time	7,390 (82.34%)
						late	1,020 (11.36%)
Birth-weight-for-gestational-age	1	10,954	180 (1.62%)			Small	1,070 (9.61%)
						Appropriate	8,508 (77.41%)
						Large	1,376 (12.36%)
						Missing	180 (1.62%)

Variable	Wave	N [a]	Missing in wave (%) [b]	Range	Mean (SD)	Categories/ Interquartile range	Frequencies (%) [c]
	2	9,634	159 (1.62%)			Small Appropriate Large Missing	914 (9.33%) 7,487 (76.45%) 1,233 (12.59%) 159 (1.62%)
	3	8,863	138 (1.53%)			Small Appropriate Large Missing	819 (9.10%) 6,901 (76.67%) 1,143 (12.70%) 138 (1.53%)
Rapid infant weight gain	1	10,749	344 (3.09%)			Yes No Missing	2,658 (24.24) 8,091 (72.67%) 344 (3.09%)
	2	9,508	285 (2.91%)			Yes No Missing	2,321 (23.70%) 7,187 (73.39%) 285 (2.91%)
	3	8,749	252 (2.80%)			Yes No Missing	2,119 (23.54%) 6,630 (73.66%) 252 (2.80%)
Breastfeeding initiation and duration	1	10,881	253 (2.27%)			Not breastfed < 3 months 3 to < 6 months > 6 months	4,621 (42.47%) 2,860 (26.28%) 1,247 (11.46%) 2,153 (19.79%)
	2	9,569	224 (2.34%)			Not breastfed < 3 months 3 to < 6 months > 6 months	4,021 (42.02%) 2,536 (26.50%) 1,113 (11.63%) 1,899 (19.85%)
	3	8,799	202 (2.24%)			Not breastfed < 3 months 3 to < 6 months > 6 months	3,657 (41.56%) 2,362 (26.84%) 1,032 (11.73%) 1,748 (19.87%)

Variable	Wave	N [a]	Missing in wave (%) [b]	Range	Mean (SD)	Categories/ Interquartile range	Frequencies (%) [c]
Timing of introduction to solid foods	1	10,845	289 (2.60%)			< 4 months	1,621 (14.95%)
						4 to 6 months	5,986 (55.20%)
						> 6 months	3,238 (29.86%)
	2	9,547	246 (2.51%)			< 4 months	1,423 (14.91%)
						4 to 6 months	5,278 (55.28%)
						> 6 months	2,846 (29.81%)
	3	8,777	224 (2.49%)			< 4 months	1,293 (14.73%)
						4 to 6 months	4,822 (54.94%)
						> 6 months	2,662 (30.33%)
Household structure	1	11,134	0			1 caregiver, 1 child	590 (5.30%)
						1 caregiver, > 1 child	769 (6.91%)
						2 caregivers, 1 child	3,694 (33.18%)
						2 caregivers, > 1 child	6,081 (54.62%)
	2	9,793	0			1 caregiver, 1 child	526 (5.37%)
						1 caregiver, > 1 child	691 (7.06%)
						2 caregivers, 1 child	1,483 (15.14%)
						2 caregivers, > 1 child	7,093 (72.43%)
	3	9,001	0			1 caregiver, 1 child	412 (4.58%)
						1 caregiver, > 1 child	680 (7.55%)
						2 caregivers, 1 child	711 (7.90%)
						2 caregivers, > 1 child	7,198 (79.97%)
Primary caregiver stress	1	11,033	1 (0.01%)	18 - 77	32.11 (6.82)		
	2	9,637	156 (1.59%)	6 - 30	12.35 (4.17)		
	3	8,843	158 (1.76%)	6 - 30	11.75 (4.07)		
Household class	1	11,134	0			Professional	2,040 (18.32%)
						Managerial	3,386 (30.41%)
						Non-manual	1,828 (16.42%)
						Skilled manual	1,543 (13.86%)
						Semi-skilled	831 (7.46%)

Variable	Wave	N [a]	Missing in wave (%) [b]	Range	Mean (SD)	Categories/ Interquartile range	Frequencies (%) [c]
						Unskilled	189 (1.70%)
						Other	56 (0.50%)
						Unclassified	1,261 (11.33%)
	2	9,793	0			Professional	1,749 (17.86%)
						Managerial	3,105 (31.71%)
						Non-manual	1,639 (16.74%)
						Skilled manual	1,382 (14.11%)
						Semi-skilled	807 (8.24%)
						Unskilled	148 (1.51%)
						Other	76 (0.78%)
						Unclassified	887 (9.06%)
	3	9,001	0			Professional	1,604 (17.82%)
						Managerial	2,974 (33.04%)
						Non-manual	1,492 (16.58%)
						Skilled manual	1,217 (13.52%)
						Semi-skilled	756 (8.40%)
						Unskilled	106 (1.18%)
						Other	88 (0.98%)
						Unclassified	764 (8.49%)
Equivalised household income	1	10,270	864 (7.76%)	Not shown	21.464 (13,230)	Lowest quintile	2,219 (19.93%)
						2nd quintile	1,925 (17.29%)
						3rd quintile	1,970 (17.69%)
						4th quintile	2,204 (19.80%)
						Highest quintile	1,952 (17.53%)
						Missing	864 (7.76%)
	2	9,260	533 (5.44%)	Not shown	18,334 (10,682)	Lowest quintile	1,813 (18.51%)
						2nd quintile	1,761 (17.98%)
						3rd quintile	1,848 (18.87%)
						4th quintile	1,836 (18.75%)

Variable	Wave	N [a]	Missing in wave (%) [b]	Range	Mean (SD)	Categories/ Interquartile range	Frequencies (%) [c]
						Highest quintile	2,002 (20.44%)
						Missing	533 (5.44%)
	3	8,556	445 (4.94%)	Not shown	18,017 (10,799)	Lowest quintile	1,594 (17.71%)
						2nd quintile	1,617 (17.96%)
						3rd quintile	1,687 (18.74%)
						4th quintile	1,772 (19.69%)
						Highest quintile	1,886 (20.95%)
						Missing	445 (4.94%)
Highest education of caregiver(s)	1	11,124	10 (0.09%)			Lower secondary	969 (8.71%)
						Higher secondary	1,658 (14.90%)
						Post-secondary	3,602 (32.37%)
						Third level	4,898 (44.02%)
	2	9,784	9 (0.09%)			Lower secondary	616 (6.30%)
						Higher secondary	1,020 (10.43%)
						Post-secondary	3,453 (35.29%)
						Third level	4,695 (47.99%)
	3	8,990	11 (0.12%)			Lower secondary	449 (4.99%)
						Higher secondary	738 (8.21%)
						Post-secondary	3,503 (38.97%)
						Third level	4,300 (47.83%)
Ethnic background	1	11,083	51 (0.46%)			Irish	7,423 (66.98%)
						Other white	914 (8.25%)
						African	265 (2.39%)
						Other black	35 (0.32%)
						Chinese	29 (0.26%)
						Other Asian	196 (1.77%)
						Other, including mixed	1,078 (9.73%)
						No SCG interview [d]	1,143 (10.31%)
	2	9,756	37 (0.38%)			Irish	6,736 (69.04%)

Variable	Wave	N [a]	Missing in wave (%) [b]	Range	Mean (SD)	Categories/ Interquartile range	Frequencies (%) [c]
						Other white	708 (7.26%)
						African	214 (2.19%)
						Other black	23 (0.24%)
						Chinese	11 (0.11%)
						Other Asian	157 (1.61%)
						Other, including mixed	937 (9.60%)
						No SCG interview [d]	970 (9.94%)
	3	8,966	35 (0.39%)			Irish	6,296 (70.22%)
						Other white	588 (6.56%)
						African	192 (2.14%)
						Other black	23 (0.26%)
						Chinese	10 (0.11%)
						Other Asian	139 (1.55%)
						Other, including mixed	869 (9.69%)
						No SCG interview [d]	849 (9.47%)
Region	1	11,087	47 (0.42%)			Urban	4,942 (44.57%)
						Rural	6,145 (55.43%)
	2	9,745	48 (0.49%)			Urban	4,195 (43.05%)
						Rural	5,550 (56.95%)
	3	8,972	29 (0.32%)			Urban	3,576 (39.86%)
						Rural	5,396 (60.14%)
Neighbourhood perceived as safe	1	11,090	44 (0.40%)			Strongly agree	2,346 (21.15%)
						Agree	5,975 (53.88%)
						Disagree	2,164 (19.51%)
						Strongly disagree	605 (5.46%)
	2	9,758	35 (0.36%)			Strongly agree	2,601 (26.66%)
						Agree	5,102 (52.29%)
						Disagree	1,684 (17.26%)
						Strongly disagree	371 (3.80%)

Variable	Wave	N [a]	Missing in wave (%) [b]	Range	Mean (SD)	Categories/ Interquartile range	Frequencies (%) [c]
	3	8,988	13 (0.14%)			Strongly agree Agree Disagree Strongly disagree	5,234 (58.23%) 3,399 (37.82%) 281 (3.13%) 74 (0.82%)
Neighbourhood perceived to offer green and play spaces	1	11,107	27 (0.25%)			Strongly agree Agree Disagree Strongly disagree	2,527 (22.75%) 4,791 (43.13%) 2,249 (20.25%) 1,540 (13.87%)
	2	9,774	19 (0.19%)			Strongly agree Agree Disagree Strongly disagree	2,617 (26.78%) 4,260 (43.59%) 1,753 (17.94%) 1,144 (11.70%)
	3	8,987	14 (0.16%)			Strongly agree Agree Disagree Strongly disagree	3,020 (33.60%) 3,448 (38.37%) 1,713 (19.06%) 806 (8.97%)
Childcare arrangement	1	11,134	0			Not in childcare Informal childcare Formal childcare	6,714 (60.30%) 3,198 (28.72%) 1,222 (10.98%)
	2	9,791	2 (0.02%)			Not in childcare Informal childcare Formal childcare	4,785 (48.87%) 2,030 (20.73%) 2,976 (30.40%)
	3	8,988	13 (0.14%)			Not in childcare Informal childcare Formal childcare	5,496 (61.15%) 2,477 (27.56%) 1,015 (11.29%)

N = Number

SCG = Secondary caregiver

BMI = Body mass index

Not shown = no data are provided to protect the confidentiality of families

[a] This indicates the total number of families for whom valid data were available; that means missing or otherwise invalid data are excluded even if a ‘missing’ category was used.

[b] This is in relation to the number of completed interviews in each wave, that means for information collected from the primary caregiver 100% corresponds to n=11,134 in wave 1, n=9,793 in wave 2 and n=9,001 in wave 3, and for information collected from the SCG, 100% corresponds to n=8,632 in wave 1, n=7,577 in wave 2 and n=6,751 in wave 3.

[c] Percentages refer to the total number of children in all categories listed, that means for variables with a ‘missing’ category, children in this category are included in the stated proportions, whereas for variables with no such category, percentages refer to the sum of valid values, only.

[d] A SCG existed but was not interviewed in this wave.

[e] This is a lone caregiver household and hence no SCG was available for inclusion in this study.

5.4.2 Distribution of OWOB based on population characteristics

Significant differences in the OWOB prevalence were found between children based on the equivalised income of the household they lived in, based on the children's BWFGA, and based on the household structure in the bivariate analysis. Results will be presented as follows.

Income

Prevalence of under- and normal weight and prevalence of obesity differed across equivalised household income groups, as can be seen in Table 18 and Figures 21 and 22. At nine months and three years of age, the prevalence of overweight appeared rather stable across income groups, whereas a small inverse trend was visible at the age of five years (Figure 21); however this was not significant (Table 18). A similar inverse trend was seen in the prevalence of obesity across income groups at all ages studied (Figure 22), and most differences in prevalence were significant (Table 18).

At the age of nine months, obesity levels were significantly lower in the 3rd, 4th and highest income quintile than in the lowest quintile. At the age of three years, significantly higher levels of under- and normal weight were seen in the 3rd than in the lowest quintile and the reverse trend was seen in obesity levels, with lower levels in the 3rd, 4th and highest quintile than in the lowest quintile, and significantly lower obesity levels in the highest compared to the 2nd quintile. Lastly, at the age of five years, under- and normal weight also was significantly more prevalent in 4th quintile than in the lowest income quintile, and significantly more prevalent in the highest compared to the lowest, 2nd and 3rd quintile. Obesity trends were reversed; they were significantly lower in the 4th compared to the lowest quintile, and significantly lower in the highest, compare to the lowest, 2nd and 3rd quintile (Table 18). No trends in overweight levels across income groups were seen at any of the ages.

Table 18: Prevalence of body mass index (BMI) status across ages, based on equivalised household income quintiles. Proportions and 95% Confidence Intervals (CI) are shown.

BMI category	Lowest quintile	2nd quintile	3rd quintile	4th quintile	Highest quintile	Missing
BMI status at 9 months (n=10,810)						
Under- or normal weight **% (95% CI)** **N**	56.82 (54.71 ; 58.93) n=1,208	60.01 (57.79 ; 62.23) n=1,124	60.18 (57.99 ; 62.37) n=1,153	60.62 (58.56 ; 62.68) n=1,304	59.82 (57.63 ; 62.01) n=1,148	61.45 (58.13 ; 64.77) n=507
Overweight **% (95% CI)** **N**	29.68 (27.74 ; 31.62) n=631	27.66 (25.63 ; 29.69) n=518	29.33 (26.90 ; 31.37) n=562	28.82 (26.91 ; 30.73) n=620	29.96 (27.91 ; 32.01) n=575	27.39 (24.35 ; 30.43) n=226
Obesity **% (95% CI)** **N**	13.5 (12.05 ; 14.95) n=287	12.33 (10.84 ; 13.82) n=231	10.49 (7.76 ; 11.86) n=201	10.55 (9.25 ; 11.85) n=227	10,21 (8.86 ; 11.56) n=196	11.15 (9.00 ; 13.30) n=92
BMI status at 3 years (n=9,422)						
Under- or normal weight **% (95% CI)** **N**	51.40 (49.04 ; 53.76) n=883	52.75 (50.36 ; 55.14) n=883	56.30 (54.00 ; 58.60) n=1,005	56.06 (53.76 ; 58.36) n=999	55.60 (53.40 ; 57.80) n=1,087	52.56 (48.22 ; 56.90) n=267
Overweight **% (95% CI)** **N**	31.90 (29.70 ; 34.10) n=548	32.26 (30.02 ; 34.50) n=540	31.20 (29.05 ; 33.35) n=557	31.76 (29.60 ; 33.92) n=566	32.99 (30.91 ; 35.07) n=645	32.48 (28.41 ; 36.55) n=165
Obesity **% (95% CI)** **N**	16.7 (14.94 ; 18.46) n=287	14.99 (13.28 ; 16.70) n=251	12.49 (10.96 ; 14.02) n=223	12.18 (10.66 ; 13.70) n=217	11.40 (9.99 ; 12.81) n=223	14.96 (11.86 ; 18.06) n=76
BMI status at 5 years (n=8,871)						
Under- or normal weight **% (95% CI)** **N**	65.19 (62.81 ; 67.57) n=1,002	66.69 (64.36 ; 69.02) n=1,051	67.36 (65.09 ; 69.63) n=1,102	70.45 (68.31 ; 72.59) n=1,230	71.85 (69.81 ; 73.89) n=1,335	69.39 (65.02 ; 73.76) n=297
Overweight **% (95% CI)** **N**	24.98 (22.82 ; 27.14) n=384	24.56 (22.43 ; 26.69) n=387	24.27 (22.19 ; 26.36) n=397	23.08 (21.10 ; 25.06) n=403	22.87 (20.96 ; 24.78) n=425	22.43 (18.48 ; 26.38) n=96
Obesity **% (95% CI)** **N**	9.82 (8.33 ; 11.31) n=151	8.76 (7.36 ; 10.16) n=138	8.37 (7.03 ; 9.71) n=137	6.47 (5.32 ; 7.62) n=113	5.27 (4.25 ; 6.29) n=98	8.18 (5.58 ; 10.78) n=35

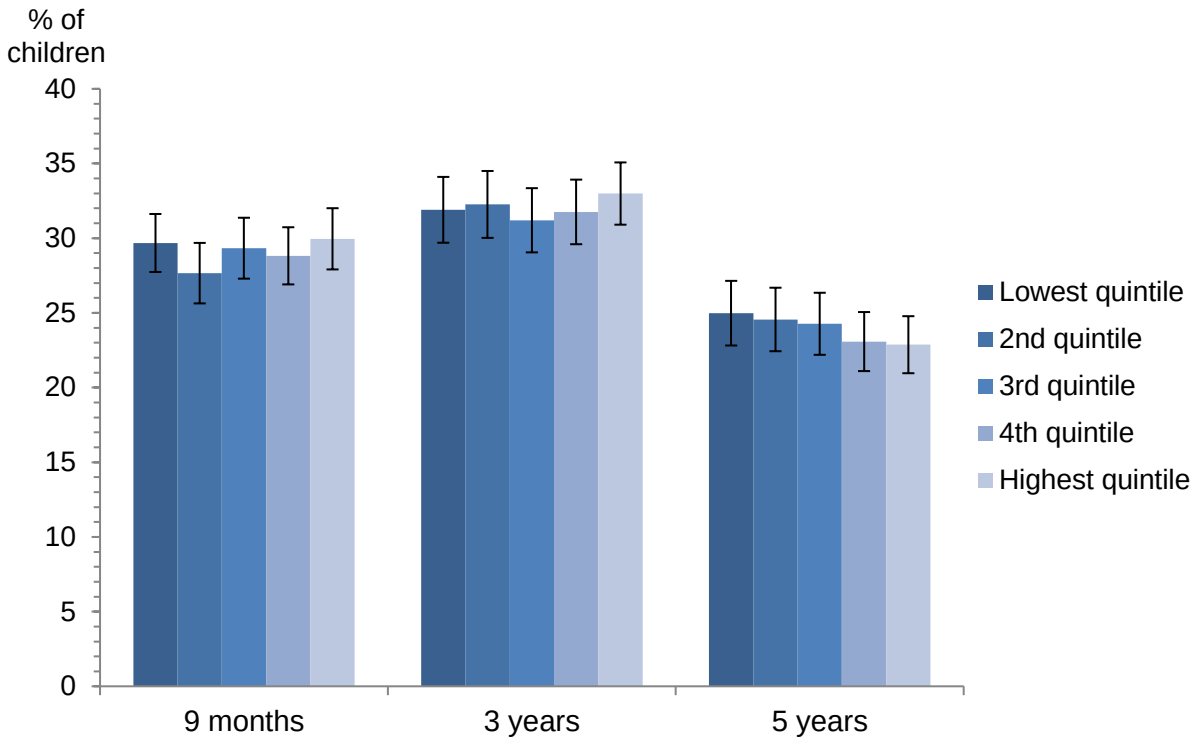

Figure 21: Prevalence of overweight (excluding obesity) across ages, based on equivalised household income quintiles

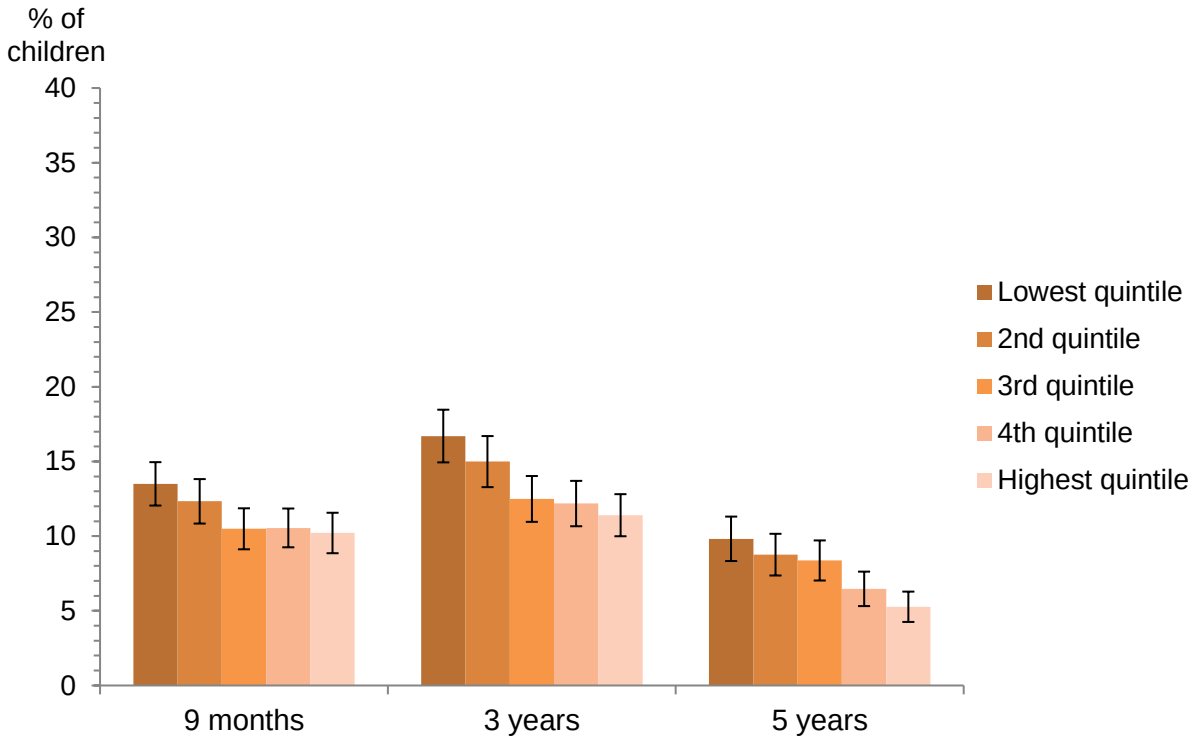

Figure 22: Prevalence of obesity across ages, based on equivalised household income quintiles

Birth-weight-for-gestational-age

As can be seen in Figure 23, children who were born SGA had much lower levels of overweight at each age than children born AGA or LGA. Children born LGA had the highest overweight levels at each age (Figure 23). A similar trend based on BWFGA was seen in the obesity prevalence at each age. However, differences between children born SGA and AGA appear smaller than those between AGA and LGA born children (Figure 24), whereas differences across BWFGA groups appeared rather similar in the overweight prevalence (Figure 23).

Table 19 presents estimates of under- and normal weight, overweight and obesity at the ages of nine months, three years and five years, based on the three BWFGA groups. At all ages, children born SGA had significantly higher levels of under- and normal weight, and lower levels of overweight than children born AGA and LGA. Additionally, at the ages of nine months and three years, their prevalence of obesity was significantly lower than that of children born AGA, whereas this difference was not significant at the age of five years (Table 19), confirming what was seen in the graphs (Figures 23 and 24). Compared to children born LGA, the prevalence of obesity was significantly lower in children born SGA at all ages. Additionally, children born LGA had significantly lower levels of under- and normal weight than those born AGA, significantly higher prevalence of obesity at all ages and significantly higher prevalence of overweight at all ages except at five years (Table 19).

Table 19: Prevalence of body mass index (BMI) status across ages, based on children's birth-weight-for-gestational-age. Proportions and 95% Confidence Intervals (CI) are shown.

BMI category	Small-for-gestational-age	Appropriate-for-gestational-age	Large-for-gestational-age	Missing
BMI status at 9 months (n=10,810)				
Under- or normal weight **% (95% CI)** **N**	70.57 (67.80 ; 73.34) n=736	59.78 (58.72 ; 60.84) n=4,958	49.70 (47.02 ; 52.38) n=665	62.96 (60.37 ; 65.55) n=85
Overweight **% (95% CI)** **N**	21.48 (18.99 ; 23.97) n=224	29.05 (28.07 ; 30.03) n=2,409	34.83 (32.28 ; 37.38) n=466	24.44 (22.14 ; 26.74) n=33
Obesity **% (95% CI)** **N**	7.96 (6.32 ; 9.60) n=83	11.13 (10.50 ; 11.86) n=927	15.47 (13.53 ; 17.41) n=207	12.59 (10.81 ; 14.37) n=17
BMI status at 3 years (n=9,422)				
Under- or normal weight **% (95% CI)** **N**	67.99 (64.91 ; 71.07) n=601	54.73 (53.58 ; 55.88) n=3,956	41.88 (39.08 ; 44.68) n=498	57.02 (54.21 ; 59.83) n=69
Overweight **% (95% CI)** **N**	23.76 (20.95 ; 26.57) n=210	32.33 (31.25 ; 33.41) n=2,337	37.17 (34.42 ; 39.92) n=442	26.45 (23.94 ; 28.96) n=32
Obesity **% (95% CI)** **N**	8.26 (6.45 ; 10.07) n=73	12.93 (12.16 ; 13.70) n=935	20.94 (18.63 ; 23.25) n=249	16.53 (14.42 ; 18.64) n=20
BMI status at 5 years (n=8,871)				
Under- or normal weight **% (95% CI)** **N**	78.32 (75.46 ; 81.18) n=625	69.51 (68.41 ; 70.61) n=4,693	55.79 (52.88 ; 58.70) n=626	66.97 (64.22 ; 69.72) n=73
Overweight **% (95% CI)** **N**	16.67 (14.08 ; 19.26) n=133	23.43 (22.42 ; 24.44) n=1,582	31.46 (28.74 ; 34.18) n=353	22.02 (19.60 ; 24.44) n=24
Obesity **% (95% CI)** **N**	5.01 (3.50 ; 6.52) n=40	7.06 (6.45 ; 7.67) n=477	12.75 (10.80 ; 14.70) n=143	11.01 (9.18 ; 12.84) n=12

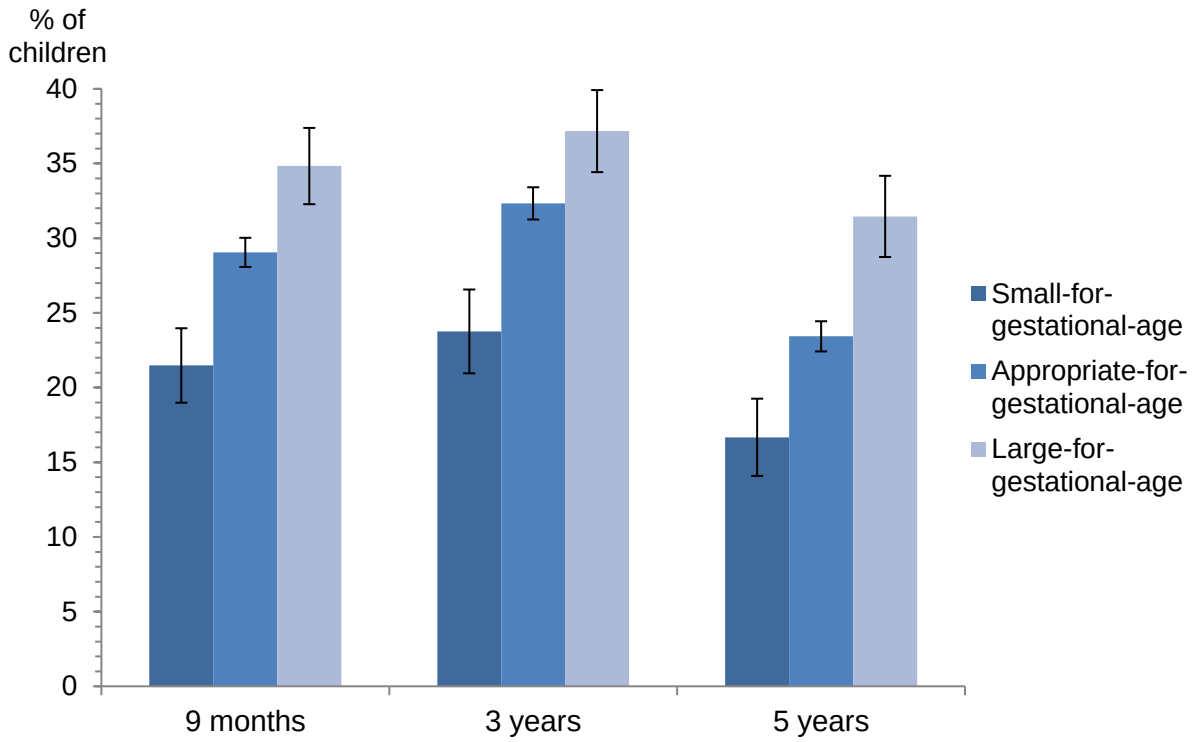

Figure 23: Prevalence of overweight (excluding obesity) across ages, based on children's birth-weight-for-gestational-age

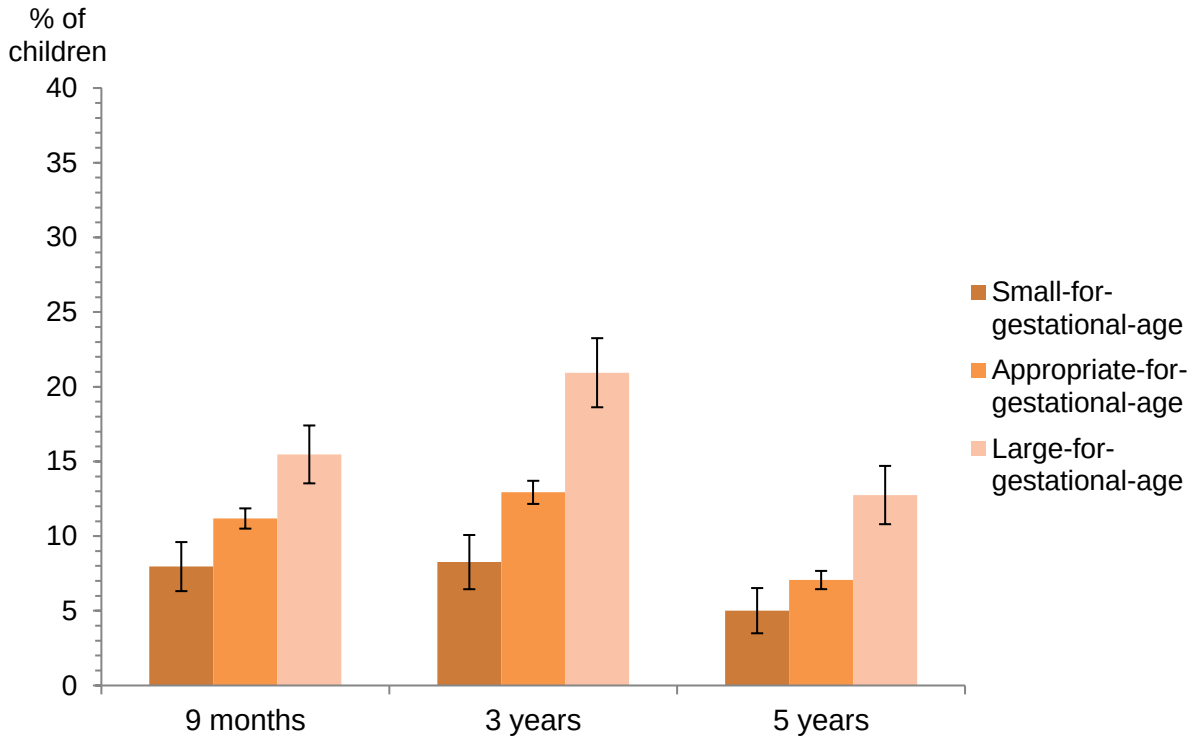

Figure 24: Prevalence of obesity across ages, based on children's birth-weight-for-gestational-age

Household structure

Prevalence of under- and normal weight, overweight and obesity was assessed at the ages of nine months, three years and five years, based on the household structure of children's families. This was grouped into families with a lone caregiver and only the study child, families with a lone caregiver and two or more children, families with two caregivers and the study child, and families with two caregivers and two or more children living in the household. Children with two caregivers seemed to have slightly lower levels of overweight than children with lone caregivers at the age of nine months and at five years, irrespective of whether other children lived in the household (Figure 25). Furthermore, at nine months of age, children with a lone caregiver and one or more other children in the household seemed to have the highest obesity prevalence. At the age of three years, prevalence of obesity was high in lone caregiver households irrespective of the number of children. Finally, at the age of five years, obesity levels seemed similar in all households except those with two caregivers and two or more children, in which lower obesity numbers were observed (Figure 26).

At the age of nine months, prevalence of under- and normal weight was significantly lower in families with a lone caregiver and two or more children, compared to families with two caregivers, irrespective of the number of children. In turn, prevalence of obesity was higher in children with a lone caregiver and no other child in the household, compared to those children who lived with two caregivers and with or without children. No significant differences were seen in the prevalence of overweight. At the age of three years, a similar trend was seen like at nine months, however none of the differences was significant. When the children were five years old, their prevalence of under- and normal weight was significantly lower if they lived together with one caregiver and no other children in the household, compared to those children who lived in households with two caregivers and at least one other child. Children's obesity levels were significantly higher in households with two caregivers and only the study child than in those with two caregivers and two or more children in the household (Table 20).

Table 20: Prevalence of body mass index (BMI) status across ages, based on household structure. Proportions and 95% Confidence Intervals (CI) are shown.

BMI category	Lone caregiver, one child	Lone caregiver, two or more children	Two caregivers, one child	Two caregivers, two or more children
BMI status at 9 months (n=10,810)				
Under- or normal weight **% (95% CI)** **N**	57.24 (53.19 ; 61.29) n=328	52.54 (48.91 ; 56.17) n=382	60.84 (59.25 ; 62.43) n=2,198	59.96 (58.71 ; 61.21) n=3,537
Overweight **% (95% CI)** **N**	31.24 (27.45 ; 35.03) n=179	31.77 (28.39 ; 35.15) n=132	28.86 (27.38 ; 30.34) n=1,042	28.48 (27.33 ; 29.63) n=1,680
Obesity **% (95% CI)** **N**	11.52 (8.9 ; 14.13) n=66	15.68 (13.04 ; 18.32) n=114	10.30 (9.31 ; 11.29) n=372	11.56 (10.74 ; 12.38) n=682
BMI status at 3 years (n=9,422)				
Under- or normal weight **% (95% CI)** **N**	51.38 (47.03 ; 55.73) n=260	51.22 (47.39 ; 55.05) n=335	55.18 (52.58 ; 57.78) n=777	54.74 (53.56 ; 55.92) n=3,752
Overweight **% (95% CI)** **N**	33.79 (29.67 ; 37.91) n=171	32.11 (28.53 ; 35.69) n=210	31.96 (29.52 ; 34.40) n=450	31.95 (30.85 ; 33.05) n=2,190
Obesity **% (95% CI)** **N**	14.82 (11.72 ; 17.92) n=75	16.67 (13.81 ; 19.53) n=109	12.86 (11.11 ; 14.61) n=181	13.31 (12.51 ; 14.11) n=912
BMI status at 5 years (n=8,871)				
Under- or normal weight **% (95% CI)** **N**	64.53 (59.88 ; 69.18) n=262	64.3 (60.61 ; 67.99) n=416	67.78 (64.28 ; 71.28) n=465	69.21 (68.13 ; 70.29) n=4,874
Overweight **% (95% CI)** **N**	26.35 (22.06 ; 30.64) n=107	25.97 (22.59 ; 29.35) n=168	22.16 (19.05 ; 25.27) n=152	23.64 (22.65 ; 24.63) n=1,665
Obesity **% (95% CI)** **N**	9.11 (6.31 ; 11.91) n=37	9.74 (7.46 ; 12.02) n=63	10.06 (7.81 ; 12.31) n=69	7.14 (6.54 ; 7.74) n=503

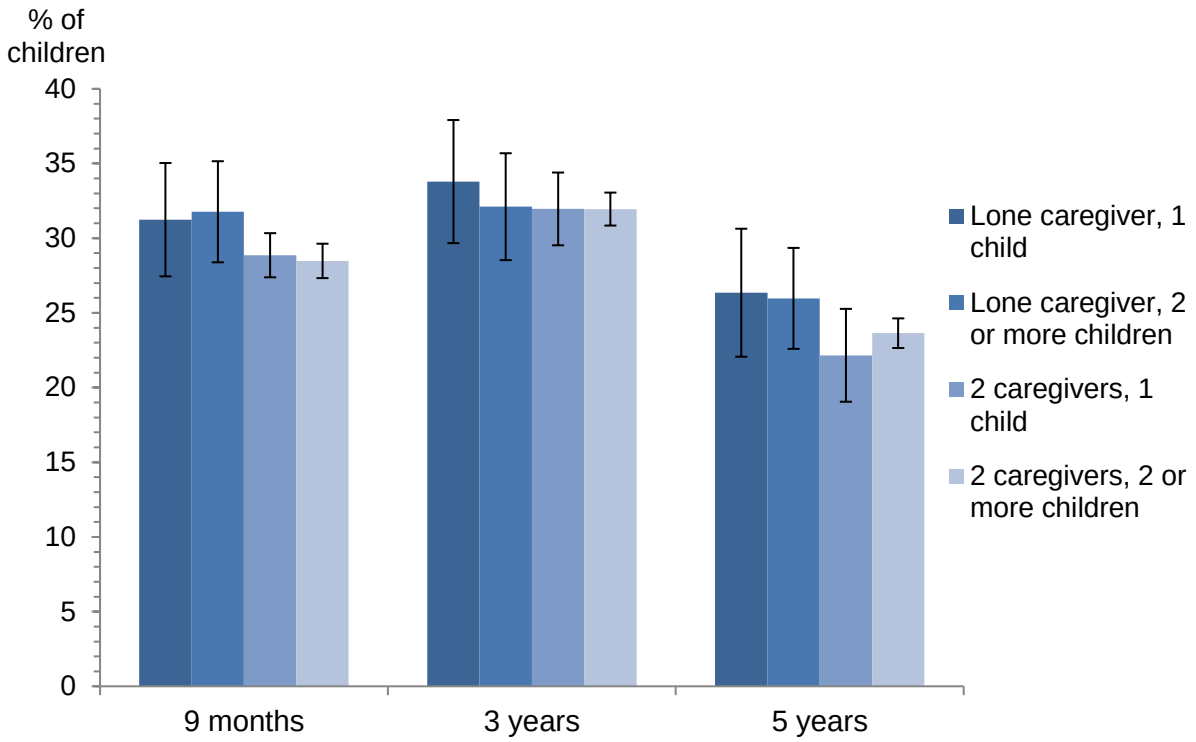

Figure 25: Prevalence of overweight (excluding obesity) across ages, based on household structure

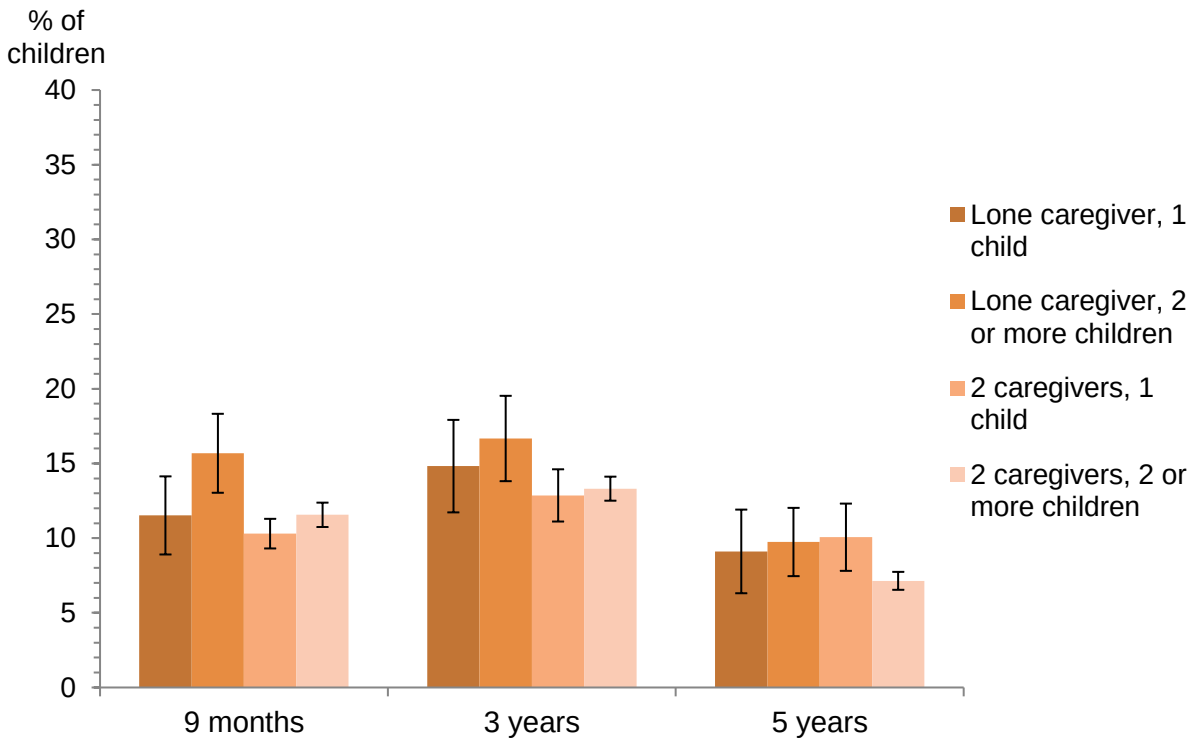

Figure 26: Prevalence of obesity across ages, based on household structure

5.4.3 Cross-sectional models

Indications for biological, material and psychosocial associations with OWOB were seen in the univariate analysis (Section 5.4.2), and more explanatory variables were included in the cross-sectional analysis to investigate Hypotheses 2-4. Table 21 presents the odds ratios of OWOB (BMI ≥ 1SD) at the ages of nine months, three years and five years individually, based on the various explanatory variables, and Table 22 shows associations between obesity (BMI ≥ 2SD) and explanatory variables, accordingly. All models were adjusted for the same explanatory variables, except for a number of lifestyle variables that were not applicable to children at the younger ages. Furthermore, at the ages of three and five years, the models were adjusted for previous weight status of children. Overall, more risk and protective factors were identified with increasing age, both in the OWOB and in the obesity models. The main findings are presented as follows, according to the levels of the SEM.

Individual level

On the individual level, girls were significantly associated with higher odds of OWOB at the age of nine months. Daily sleep duration was not significant and other variables on the individual level were not assessed at this age. Furthermore, no association with obesity was observed.

In contrast, at the age of three years, girls were associated with lower odds of OWOB. Furthermore, previous overweight or obesity status was associated with significantly higher odds of both OWOB and obesity, compared to children who had under- or normal weight when they were nine months old. Notably, the risk of obesity at three years was nearly 42 times higher if children had been obese at the age of nine months. Children were furthermore significantly more likely to have OWOB at three years if they slept little, compared to children with normal sleep, and they were significantly less likely to be obese if they watched no TV or less than one hour daily, compared to children who watched TV between one and less than two hours daily.

Also at the age of five years, previous overweight or obesity status, compared to under- or normal weight, was associated with significantly higher odds of OWOB, and of obesity. The highest odds ratio was seen between children who were obese at three years, who had a nearly 39 times higher chance of being obese at five years than children who were under- or normal weight at three years. Furthermore, short sleep was significantly associated with higher odds of OWOB, and long sleep with lower odds of obesity at five years, compared to normal sleeping hours. Additionally, the odds of obesity were significantly higher in children who spent three hours or more per day using screens, and it was significantly lower if they spent no screen time or less than one hour, compared to children who had between one and less than two hours of screen time daily. Lastly, the more antibiotic doses children had taken in the twelve months preceding the interview, the higher the odds of obesity at the age of five years. No associations with dietary intake or physical activity were seen for either OWOB or obesity.

Family level

Most risk and protective factors identified on this level are related to birth outcomes and early infant feeding. At the age of nine months, children born late had significantly higher odds of OWOB and of obesity compared to term children, whereas the risk of OWOB and of obesity were significantly lower in children born very early or early. Furthermore, children born LGA had significantly higher odds of OWOB and of obesity than AGA children, and those born SGA had significantly lower odds of both. Rapid infant weight gain was associated with significantly higher risk of OWOB and obesity than non-rapid infant weight gain. The risk of OWOB was additionally significantly higher in children whose PCG was obese, compared to under- or normal weight PCGs, and in those children who were in informal childcare arrangements at nine months, compared to those not in a permanent care arrangement. The risk of obesity was significantly lower in single children with one caregiver, compared to children who lived in a household with two caregivers and at least one other child.

At the age of three years, similar relationships were observed like at nine months, in addition to associations seen with the caregiver weight status. Significantly increased odds of OWOB and obesity were as well seen in children born LGA and in those with rapid infant weight gain, and significantly lower odds were seen in children born SGA and those born early, compared to the reference groups. Very early birth and late birth, however, were only significantly associated with lower and higher odds of OWOB, respectively, but they were not associated with obesity. Additionally, weight status of the PCG and SCG were significantly associated with OWOB and obesity at three years. Compared to children with an under- or normal weight PCG, those with an overweight PCG had higher odds of obesity, and those with an obese PCG had higher odds of OWOB and of obesity. In SCGs, compared to under- and normal weight, overweight status and obesity status both were related to higher odds of OWOB and of obesity in children. Household type also was related to children's weight status. Single children with one caregiver had significantly higher odds of OWOB, and children in households with one caregiver and two or more children had significantly higher odds of obesity, compared to children who lived in households with two caregivers and two or more children. Lastly, at three years of age, children were significantly more likely to have OWOB if they were in an informal childcare arrangement, compared to children who were not permanently in childcare.

Risk and protective factors seen on the family level at the ages of nine months and three years were partly also associated with weight status at the age of five years. Children born LGA and those with rapid infant weight gain were still at a significantly increased risk of OWOB and obesity when they were five years old. Children born SGA, very early or early still had significantly lower odds of OWOB, but not of obesity. Additionally, the odds of OWOB were significantly higher if children were introduced to solid food before the age of four months, compared to children who started eating solid food between the ages of four and six months. The odds of obesity were significantly lower if children were breastfed for six months or longer, compared to children who were not breastfed at all. PCG and SCG weight status were significantly related to children's weight status also at the age of five years. PCG overweight and obesity status both were significantly associated with higher odds of OWOB and obesity in children, compared to

children with an under- or normal weight PCG. Obesity in the SCG was associated with significantly higher odds of OWOB and of obesity, whereas overweight was only associated with higher OWOB odds, compared to under- or normal weight status of the SCG. Lastly, children with a lone caregiver had significantly higher odds of OWOB irrespective of whether other children lived in the household or not, compared to children who lived in a household with two caregivers and two or more children.

Community, demographic and societal level

A number of children had altered odds of OWOB or obesity depending on a number of community, demographic and societal characteristics. At the age of nine months, children who lived in families not grouped into a social class had significantly higher odds of OWOB and of obesity, compared to children whose families were in the managerial and technical class. Furthermore, compared to white children with Irish ethnic background, children with non-Irish white background had significantly lower odds of obesity and children with Asian (excluding Chinese) background had significantly lower odds of OWOB at the age of nine months.

When the children were three years old, children with non-Irish white background had significantly lower odds of OWOB, compared to those with Irish white background. Children in the ‘other’ ethnic background group, which includes children with mixed ethnic background, were significantly less likely to be obese at three years.

At the age of five years, children with African or with Asian, non-Chinese ethnic background were significantly more likely to have OWOB or obesity than children with Irish white background, and children with other black background (excluding African) were significantly less likely to have OWOB. A significantly higher risk of obesity also was seen in children with Chinese ethnic background, however the small sample size of children in this group and the resulting large measurement error need to be noted as they likely affected the precision of this estimate.

Furthermore, when the children were five years old, caregiver education was associated with OWOB. Compared to third level education, children whose caregivers had post-secondary education had significantly higher odds of OWOB. Additionally, the odds of OWOB were significantly lower if children's families were in the lowest income quintile, compared to the highest. Lastly, neighbourhood characteristics were associated with OWOB and obesity at five years. Children were significantly more likely to have OWOB if their PCG disagreed that they were living in a safe neighbourhood, compared to children whose PCG perceived the neighbourhood as safe ('agree'). Furthermore, compared to PCGs who perceived the neighbourhood as safe, children with PCGs who agreed strongly that the neighbourhood was safe had significantly higher odds of obesity. Lower odds of OWOB were seen in children if their PCG perceived the neighbourhood to offer insufficient green and play spaces ('disagree'), compare to PCGs who found the availability sufficient ('agree'). Also, children were significantly less likely to have OWOB if they lived in urban, compared to rural regions.

Table 21: Logistic regression model showing odds ratios (OR) of overweight and obesity prevalence at nine months, three years and five years of age. Overweight and obesity were defined as a body mass index (BMI) ≥ 1 standard deviation (SD) based on the World Health Organization growth reference. Point estimates and 95% confidence interval (CI) are shown.

	Overweight and obesity (BMI[b])		
Model components	**OR (95% CI) at age nine months**	**OR (95% CI) at age three years**	**OR (95% CI) at age five years**
Number	10,048	8,521	7,601
Constant	**0.37 (0.28 ; 0.48)*** **	**0.32 (0.23 ; 0.43)*** **	**0.05 (0.03 ; 0.08)*** **
BMI status at previous measurement (under- or normal weight) [a]			
Overweight (BMI ≥ +1SD < +2SD) [b]	N.A.	**3.24 (2.86 ; 3.67)*** **	**7.08 (6.03 ; 8.31)*** **
Obese (BMI ≥ +2SD) [b]	N.A.	**8.38 (6.66 ; 10.55)*** **	**41.61 (32.72; 52.91)*** **
Sex (boys)[a]			
Girls	**1.21 (1.10 ; 1.35)*** **	**0.87 (0.78 ; 0.98)* **	0.89 (0.77 ; 8.31)
Household class (managerial and technical) [a]			
Professional	1.02 (0.88 ; 1.17)	0.87 (0.74 ; 1.03)	1.10 (0.89 ; 1.35)
Non-manual	1.03 (0.88 ; 1.21)	1.05 (0.88 ; 1.24)	0.84 (0.67 ; 1.05)
Skilled manual	1.04 (0.87 ; 1.25)	0.96 (0.78 ; 1.18)	1.04 (0.80 ; 1.34)
Semi-skilled	0.94 (0.74 ; 1.19)	0.95 (0.73 ; 1.24)	1.18 (0.86 ; 1.62)
Unskilled	1.31 (0.83 ; 2.05)	0.93 (0.54 ; 1.58)	0.99 (0.54 ; 1.79)
Other	0.67 (0.18 ; 2.42)	1.24 (0.58 ; 2.66)	1.69 (0.78 ; 3.65)
Unclassified	**1.39 (1.04 ; 1.86)**	0.93 (0.68 ; 1.27)	1.04 (0.69 ; 1.56)
Ethnic background (Irish white)[a]			
Any other white background	0.82 (0.65 ; 1.04)	**0.69 (0.53 ; 0.91)** **	1.42 (0.98 ; 2.06)
African	0.74 (0.51 ; 1.09)	1.33 (0.82 ; 2.15)	**1.76 (1.04 ; 2.98)* **
Any other Black background	1.22 (0.49 ; 3.04)	1.06 (0.25 ; 4.49)	**0.26 (0.07 ; 0.96)* **
Chinese	0.95 (0.38 ; 2.38)	2.35 (0.50 ; 11.01)	0.77 (0.10 ; 5.68)
Any other Asian background	**0.51 (0.33 ; 0.78)** **	1.21 (0.76 ; 1.93)	**2.38 (1.33 ; 4.28)** **
Other, including mixed background	0.94 (0.79 ; 1.13)	0.86 (0.71 ; 1.05)	0.97 (0.75 ; 1.25)
No secondary caregiver data	1.04 (0.85 ; 1.27)	1.17 (0.95 ; 1.43)	0.84 (0.65 ; 1.09)
Equivalised household income (highest quintile)[a]			
Lowest quintile	1.14 (0.92 ; 1.41)	1.12 (0.90 ; 1.41)	**0.71 (0.53 ; 0.96)* **
2nd quintile	1.03 (0.85 ; 1.25)	1.20 (0.97 ; 1.49)	0.93 (0.71 ; 1.21)
3rd quintile	1.05 (0.88 ; 1.26)	0.92 (0.76 ; 1.12)	1.01 (0.79 ; 1.29)
4th quintile	1.02 (0.87 ; 1.20)	0.97 (0.82 ; 1.15)	0.90 (0.72 ; 1.11)
Missing	0.98 (0.77 ; 1.25)	1.10 (0.82 ; 1.46)	**0.65 (0.45 ; 0.94)* **

	Overweight and obesity (BMI[b]		
Model components	**OR (95% CI) at age nine months**	**OR (95% CI) at age three years**	**OR (95% CI) at age five years**
Highest education of caregivers (ref. third level)			
Lower secondary education or less	0.92 (0.75 ; 1.14)	1.08 (0.82 ; 1.43)	1.52 (1.00 ; 2.32)
Higher secondary education	0.99 (0.84 ; 1.15)	1.01 (0.81 ; 1.25)	1.31 (0.98 ; 1.75)
Post-secondary education	0.97 (0.85 ; 1.10)	0.92 (0.79 ; 1.06)	**1.22 (1.01 ; 1.47)***
Region (rural)[a]			
Urban	0.99 (0.88 ; 1.10)	0.90 (0.80 ; 1.02)	**0.77 (0.66 ; 090)*****
Household structure (Two caregivers, two or more children)[a]			
Lone caregiver, one child	0.89 (0.66 ; 1.20)	**1.45 (1.06 ; 2.00)***	**1.66 (1.09 ; 2.54)***
Lone caregiver, two or more children	1.12 (0.82 ; 1.54)	1.14 (0.81 ; 1.60)	**1.96 (1.31 ; 2.92)****
Two caregivers, one child	0.96 (0.85 ; 1.08)	0.98 (0.83 ; 1.15)	1.04 (0.78 ; 1.38)
Neighbourhood perceived to offer green and play spaces (agree) [a]			
Strongly agree	1.02 (0.88 ; 1.17)	1.08 (0.93 ; 1.26)	0.97 (0.81 ; 1.17)
Disagree	1.01 (0.88 ; 1.17)	0.98 (0.84 ; 1.14)	**0.81 (0.66 ; 0.99)***
Strongly disagree	0.95 (0.81 ; 1.12)	0.92 (0.76 ; 1.11)	1.24 (0.95 ; 1.61)
Neighbourhood perceived as safe (agree) [a]			
Strongly agree	1.01 (0.88 ; 1.16)	1.01 (0.87 ; 1.17)	1.03 (0.87 ; 1.21)
Disagree	0.94 (0.82 ; 1.08)	1.02 (0.88 ; 1.19)	**1.54 (1.01 ; 2.34)***
Strongly disagree	0.79 (0.62 ; 1.02)	1.07 (0.79 ; 1.45)	1.17 (0.41 ; 3.36)
Gestational age at birth (on time)[a]			
Very early	**0.11 (0.06 ; 0.19)*****	**0.39 (0.23 ; 0.65)*****	**0.45 (0.24 ; 0.85)***
Somewhat early	**0.26 (0.20 ; 0.34)*****	**0.60 (0.45 ; 0.79)*****	**0.64 (0.43 ; 0.95)***
Late	**1.32 (1.13 ; 1.56)****	**1.22 (1.02 ; 1.46)***	1.21 (0.98 ; 1.49)
Birth-weight-for-gestational-age (appropriate)[a]			
Small-for-gestational-age	**0.28 (0.23 ; 0.35)*****	**0.47 (0.38 ; 0.58)*****	**0.64 (0.48 ; 0.87)****
Large-for-gestational-age	**2.10 (1.81 ; 2.44)*****	**1.63 (1.37 ; 1.93)*****	**1.61 (1.31 ; 1.96)*****
Missing	**0.20 (0.13 ; 0.32)*****	0.63 (0.38 ; 1.03)	0.69 (0.40 ; 1.20)
Breastfeeding duration (not breastfed)[a]			
Less than three months	0.95 (0.83 ; 1.08)	0.99 (0.86 ; 1.14)	1.10 (0.92 ; 1.31)
Three to under six months	1.06 (0.89 ; 1.26)	1.09 (0.90 ; 1.32)	0.92 (0.72 ; 1.16)
Six months or more	0.88 (0.75 ; 1.03)	1.03 (0.87 ; 1.23)	0.94 (0.75 ; 1.17)
Introduction of solid food (between age four and under six months)[a]			
Before age four months	1.08 (0.93 ; 1.26)	0.91 (0.77 ; 1.07)	**1.39 (1.13 ; 1.70)****

	Overweight and obesity (BMI[b]		
Model components	**OR (95% CI) at age nine months**	**OR (95% CI) at age three years**	**OR (95% CI) at age five years**
After age six months or later	0.98 (0.87 ; 1.11)	0.97 (0.85 ; 1/11)	1.10 (0.92 ; 1.30)
Rapid infant weight gain (no rapid weight gain)[a]			
Rapid weight gain	**7.37 (6.41 ; 8.49)*** **	**2.01 (1.71 ; 2.36)*** **	**1.87 (1.55 ; 2.24)*** **
Missing			
BMI status of the primary caregiver (normal/ underweight)[a]			
Overweight	1.08 (0.96 ; 1.22)	1.14 (1.00 ; 1.30)	**1.64 (1.39 ; 1.93)*** **
Obese	**1.23 (1.06 ; 1.42)** **	**1.55 (1.31 ; 1.83)*** **	**2.01 (1.64 ; 2.46)*** **
Missing	1.33 (0.99 ; 1.79)	**1.49 (1.13 ; 1.95)** **	**2.30 (1.59 ; 3.31)*** **
BMI status of the secondary caregiver (normal/ underweight)[a]			
Overweight	0.99 (0.87 ; 1.13)	**1.23 (1.05 ; 1.44)* **	**1.32 (1.07 ; 1.62)* **
Obese	1.16 (0.98 ; 1.37)	**1.74 (1.44 ; 2.11)*** **	**2.00 (1.57 ; 2.56)*** **
No secondary caregiver interview	omitted	1.01 (0.79 ; 1.29)	**1.70 (1.28 ; 2.26)*** **
Missing	0.92 (0.66 ; 1.29)	1.33 (1.00 ; 1.76)	**1.71 (1.20 ; 2.46)** **
Primary caregiver stress			
Continuous score	1.00 (0.99 ; 1.01)	1.01 (0.99 ; 1.02)	1.01 (0.99 ; 1.03)
Childcare (no childcare arrangement)[a]			
Informal childcare	**1.15 (1.01 ; 1.30)* **	**1.20 (1.02 ; 1.40))* **	1.00 (0.83 ; 1.19)
Formal childcare	1.15 (0.97 ; 1.37)	1.05 (0.91 ; 1.21)	1.03 (0.80 ; 1.33)
Daily sleep duration (normal sleep)[a]			
Short sleep	0.95 (0.83 ; 1.09)	**1.26 (1.07 ; 1.49)** **	**1.23 (1.02 ; 1.48)* **
Long sleep	0.95 (0.81 ; 1.11)	0.94 (0.78 ; 1.11)	0.91 (0.75 ; 1.09)
Food recommendations met	N.A.	N.A.	1.01 (0.93 ; 1.09)
Antibiotic doses in the past 12 months	N.A.	1.02 (1.00 ; 1.06)	1.03 (0.99 ; 1.07)
Frequency of unstructured physical activity	N.A.	N.A.	1.02 (0.97 ; 1.07)
Sports club use (never)[a]			
Twice a month	N.A.	N.A.	0.90 (0.52 ; 1.53)
One hour per week	N.A.	N.A.	1.09 (0.91 ; 1.30)
Two hours per week	N.A.	N.A.	1.17 (0.94 ; 1.45)
More than two hours per week	N.A.	N.A.	1.08 (0.82 ; 1.41)
Missing	N.A.	N.A.	2.99 (0.71 ; 12.64)
Daily screen time (one to less than two hours)[a]			
None to less than one hour	N.A.	1.01 (0.85 ; 1.20)	0.70 (0.44 ; 1.10)
Two to less than three hours	N.A.	1.01 (0.88 ; 1.16)	0.98 (0.83 ; 1.15)
Three hours or more	N.A.	1.13 (0.96 ; 1.33)	1.19 (0.95 ; 1.51)

a = reference group

b = The 1SD threshold is used to define overweight in children based on the WHO growth reference. In children below five years of age, this threshold corresponds to the category of ‘risk of overweight’ based on the WHO growth standard. Additionally, the 2SD threshold is used to define obesity based on the WHO growth reference, but this threshold defines overweight (including obesity) in the WHO growth standard. For this analysis, the WHO growth reference classification was used to define overweight and obesity for consistency across ages.

N.A. = not applicable, no information was recorded for this variable at this age

* P<0.05

** P<0.01

*** P<0.001

Table 22: Logistic regression model showing odds ratios (OR) of obesity prevalence at nine months, three years and five years of age. Obesity was defined as a body mass index (BMI) ≥ 2 standard deviation (SD) based on the World Health Organization growth reference. Point estimates and 95% Confidence Intervals (CI) are shown.

	Obesity (BMI ≥ +2SD)		
Model components	**OR (95% CI) at age nine months**	**OR (95% CI) at age three years**	**OR (95% CI) at age five years**
Number	10,048	8,521	7,601
Constant	**0.06 (0.04 ; 0.09)*** **	**0.04 (0.02 ; 0.06)*** **	**0.002 (0.001 ; 0.005)*** **
BMI status at previous measurement (under- or normal weight) [a]			
Overweight (BMI ≥ +1SD < +2SD) [b]	N.A.	**2.72 (2.25 ; 3.28)*** **	**5.70 (3.92 ; 8.30)*** **
Obese (BMI ≥ +2SD) [b]	N.A.	**6.97 (5.52 ; 8.82)*** **	**38.69 (26.60; 56.26)*** **
Sex (boys)[a]			
Girls	1.14 (0.97 ; 1.34)	0.90 (0.77 ; 1.06)	0.86 (0.68 ; 1.10)
Household class (managerial and technical) [a]			
Professional	0.93 (0.74 ; 1.17)	0.97 (0.76 ; 1.23)	1.33 (0.93 ; 1.91)
Non-manual	0.84 (0.66 ; 1.08)	0.89 (0.69 ; 1.15)	1.20 (0.84 ; 1.71)
Skilled manual	0.83 (0.62 ; 1.10)	1.14 (0.86 ; 1.51)	1.38 (0.91 ; 2.08)
Semi-skilled	0.85 (0.58 ; 1.23)	0.90 (0.63 ; 1.26)	1.17 (0.73 ; 1.87)
Unskilled	0.96 (0.51 ; 1.80)	1.10 (0.57 ; 2.10)	1.51 (0.57 ; 4.03)
Other	1.32 (0.30 ; 5.78)	0.94 (0.35 ; 2.50)	1.33 (0.23 ; 7.86)
Unclassified	1.19 (0.77 ; 1.84)	1.07 (0.68 ; 1.67)	1.60 (0.89 ; 2.88)
Ethnic background (Irish white)[a]			
Any other white background	**0.64 (0.42 ; 0.97)***	1.03 (0.69 ; 1.55)	0.88 (0.40 ; 1.93)
African	1.18 (0.69 ; 2.01)	0.91 (0.47 ; 1.73)	**3.39 (1.69 ; 6.80)** **
Any other Black background	1.20 (0.29 ; 4.94)	0.43 (0.13 ; 1.47)	Omitted
Chinese	0.74 (0.10 ; 5.24)	1.09 (0.06 ; 18.81)	**8.86 (1.45 ; 54.26)***
Any other Asian background	0.84 (0.42 ; 1.66)	1.28 (0.65 ; 2.49)	**4.08 (1.74 ; 9.57)** **
Other, including mixed background	0.79 (0.59 ; 1.06)	**0.70 (0.51 ; 0.96)***	1.09 (0.68 ; 1.74)
No secondary caregiver data	0.87 (0.63 ; 1.19)	1.04 (0.78 ; 1.39)	0.65 (0.42 ; 1.01)
Equivalised household income (highest quintile)[a]			
Lowest quintile	1.25 (0.91 ; 1.72)	1.35 (0.98 ; 1.85)	1.03 (0.63 ; 1.67)
2nd quintile	1.21 (0.90 ; 1.63)	1.13 (0.84 ; 1.52)	1.16 (0.74 ; 1.80)
3rd quintile	1.00 (0.76 ; 1.33)	1.01 (0.77 ; 1.33)	1.19 (0.80 ; 1.77)
4th quintile	1.04 (0.81 ; 1.34)	1.06 (0.82 ; 1.36)	1.23 (0.85 ; 1.78)
Missing	1.26 (0.87 ; 1.81)	0.91 (0.60 ; 1.37)	1.30 (0.74 ; 2.31)

	Obesity (BMI ≥ +2 SD)		
Model components	**OR (95% CI) at age nine months**	**OR (95% CI) at age three years**	**OR (95% CI) at age five years**
Highest education of caregivers (ref. third level)			
Lower secondary education or less	0.96 (0.69 ; 1.33)	1.13 (0.79 ; 1.61)	1.26 (0.70 ; 2.30)
Higher secondary education	1.00 (0.78 ; 1.28)	0.94 (0.70 ; 1.26)	1.19 (0.78 ; 1.83)
Post-secondary education	0.87 (0.71 ; 1.06	0.92 (0.75 ; 1.13)	1.07 (0.78 ; 1.46)
Region (rural)[a]			
Urban	0.94 (0.79 ; 1.11)	0.87 (0.73 ; 1.04)	1.22 (0.95 ; 1.56)
Household structure (Two caregivers, two or more children)[a]			
Lone caregiver, one child	**0.65 (0.42 ; 0.98)***	1.43 (0.92 ; 2.24)	1.45 (0.75 ; 2.82)
Lone caregiver, two or more children	0.92 (0.57 ; 1.47)	1.29 (0.81 ; 2.05)	1.44 (0.78 ; 2.63)
Two caregivers, one child	0.88 (0.73 ; 1.06)	0.99 (0.78 ; 1.26)	1.33 (0.88 ; 2.01)
Neighbourhood perceived to offer green and play spaces (agree) [a]			
Strongly agree	0.99 (0.79 ; 1.24)	0.99 (0.80 ; 1.23)	0.91 (0.67 ; 1.22)
Disagree	1.11 (0.90 ; 1.37)	1.05 (0.84 ; 1.31)	0.90 (0.66 ; 1.24)
Strongly disagree	0.90 (0.70 ; 1.15)	1.08 (0.83 ; 1.41)	0.87 (0.56 ; 1.34)
Neighbourhood perceived as safe (agree) [a]			
Strongly agree	1.17 (0.94 ; 1.46)	1.03 (0.83 ; 1.27)	**1.40 (1.06 ; 1.83)***
Disagree	1.04 (0.84 ; 1.28)	1.05 (0.84 ; 1.30)	0.97 (0.47 ; 2.00)
Strongly disagree	0.76 (0.51 ; 1.13)	1.23 (0.82 ; 1.82)	3.02 (0.88 ; 10.33)
Gestational age at birth (on time)[a]			
Very early	**0.08 (0.02 ; 0.30)*****	0.45 (0.17 ; 1.16)	0.33 (0.10 ; 1.15)
Somewhat early	**0.24 (0.16 ; 0.36)*****	**0.60 (0.39 ; 0.90)***	0.71 (0.40 ; 1.28)
Late	**1.34 (1.05 ; 1.71)***	1.17 (0.92 ; 1.49)	1.20 (0.86 ; 1.67)
Birth-weight-for-gestational-age (appropriate)[a]			
Small-for-gestational-age	**0.31 (0.23 ; 0.42)*****	**0.64 (0.45 ; 0.90)***	0.91 (0.54 ; 1.52)
Large-for-gestational-age	**3.07 (2.45 ; 3.84)*****	**1.84 (1.49 ; 2.28)*****	**1.74 (1.29 ; 2.34)*****
Missing	**0.36 (0.19 ; 0.66)****	0.97 (0.49 ; 1.92)	0.60 (0.22 ; 1.64)
Breastfeeding duration (not breastfed)[a]			
Less than three months	0.92 (0.76 ; 1.12)	0.82 (0.67 ; 1.01)	0.82 (0.62 ; 1.10)
Three to under six months	0.90 (0.67 ; 1.21)	0.89 (0.67 ; 1.19)	0.95 (0.62 ; 1.46)
Six months or more	0.79 (0.62 ; 1.02)	1.02 (0.79 ; 1.33)	**0.57 (0.39 ; 0.85)****
Introduction of solid food (between age four and under six months)[a]			
Before age four months	1.16 (0.96 ; 1.45)	0.94 (0.75 ; 1.17)	0.84 (0.60 ; 1.18)

	Obesity (BMI ≥ +2 SD)		
Model components	**OR (95% CI) at age nine months**	**OR (95% CI) at age three years**	**OR (95% CI) at age five years**
After age six months or later	0.99 (0.82 ; 1.21	0.91 (0.75 ; 1.11)	1.11 (0.85 ; 1.45)
Rapid infant weight gain (no rapid weight gain)[a]			
Rapid weight gain	**11.20 (9.33 ; 13.46)*** **	**2.14 (1.75 ; 2.62)*** **	**1.42 (1.08 ; 1.86)* **
Missing			
BMI status of the primary caregiver (normal/ underweight)[a]			
Overweight	1.09 (0.91 ; 1.32)	**1.35 (1.12 ; 1.63)** **	**1.49 (1.12 ; 1.99)** **
Obese	1.23 (0.98 ; 1.54)	**2.02 (1.62 ; 2.53)*** **	**2.43 (1.80 ; 3.26)*** **
Missing	0.93 (0.60 ; 1.44)	**1.74 (1.23 ; 2.46)** **	**1.96 (1.11 ; 3.47)* **
BMI status of the secondary caregiver (normal/ underweight)[a]			
Overweight	0.83 (0.67 ; 1.03)	**1.31 (1.03 ; 1.66)* **	1.45 (0.98 ; 2.15)
Obese	0.89 (0.69 ; 1.16)	**1.86 (1.42 ; 2.44)*** **	**2.67 (1.76 ; 4.05)*** **
No secondary caregiver interview	Omitted	**1.32 (1.03 ; 2.02)* **	**3.04 (1.05 ; 2.86)* **
Missing	0.86 (0.52 ; 1.42)	1.32 (0.88 ; 1.99)	**3.04 (1.70 ; 5.42)*** **
Primary caregiver stress			
Continuous score	0.99 (0.98 ; 1.01)	1.01 (0.99 ; 1.03)	1.00 (0.97 ; 1.03)
Childcare (no childcare arrangement)[a]			
Informal childcare	1.10 (0.91 ; 1.33)	1.05 (0.84 ; 1.31)	1.27 (0.95 ; 1.69)
Formal childcare	1.00 (0.75 ; 1.34)	0.97 (0.79 ; 1.19)	1.24 (0.83 ; 1.85)
Daily sleep duration (normal sleep)[a]			
Short sleep	1.14 (0.94 ; 1.40)	1.20 (0.94 ; 1.54)	1.13 (0.84 ; 1.51)
Long sleep	0.79 (0.60 ; 1.03)	1.01 (0.79 ; 1.29)	**0.66 (0.49 ; 0.90)** **
Food recommendations met	N.A.	N.A.	1.03 (0.91 ; 1.16)
Antibiotic doses in the past 12 months	N.A.	1.03 (0.98 ; 1.07)	**1.10 (1.04 ; 1.16)** **
Frequency of unstructured physical activity	N.A.	N.A.	1.06 (0.98 ; 1.16)
Sports club use (never)[a]			
Twice a month	N.A.	N.A.	0.97 (0.40 ; 2.35)
One hour per week	N.A.	N.A.	1.04 (0.78 ; 1.38)
Two hours per week	N.A.	N.A.	1.15 (0.79 ; 1.68)
More than two hours per week	N.A.	N.A.	1.03 (0.66 ; 1.61)
Missing	N.A.	N.A.	Omitted
Daily screen time (one to less than two hours)[a]			
None to less than one hour	N.A.	0.89 (0.69 ; 1.15)	**0.35 (0.14 ; 0.83)* **
Two to less than three hours	N.A.	0.98 (0.80 ; 1.19)	1.25 (0.95 ; 1.64)
Three hours or more	N.A.	1.21 (0.97 ; 1.51)	**1.89 (1.35 ; 2.64)*** **

a = reference group

b = The +1SD threshold is used to define overweight in children based on the WHO growth reference. In children below five years of age, this threshold corresponds to the category of ‘risk of overweight’ based on the WHO growth standard. Additionally, the +2SD threshold is used to define obesity based on the WHO growth reference, but this threshold defines overweight (including obesity) in the WHO growth standard. For this analysis, the WHO growth reference classification was used to define overweight and obesity for consistency across ages.

N.A. = not applicable, no information was recorded for this variable at this age

* P<0.05

** P<0.01

*** P<0.001

By the age of five years, a large number of variables were associated with OWOB or obesity in the GUI infant cohort, and a large proportion of children were in those categories with the highest OWOB risk, as shown in Section 5.4.1. Accordingly, it is likely that more than one risk factor is present in some children, putting these children at the highest risk to gain excess weight. For example, by the age of five years, children who lived in rural regions, whose families were in the lowest income quintile and whose caregivers had less then third level education (n=768) had a cumulative OWOB prevalence of 36.9% (95% CI: 33.5 ; 40.5) and obesity prevalence of 8.7% (95% CI: 6.8 ; 11.0). In comparison, 23.7% (95% CI: 20.8 ; 26.8) of children had OWOB in families in urban regions in the highest income quintile where at least one caregiver had third level education (n=712), of which 5% (95% CI: 3.6 ; 6.7) had obesity (results not shown).

Furthermore, children in lone caregiver households who attended informal childcare (n=293) had an OWOB prevalence of 37.9% (95% CI: 32.5 ; 43.6), compared to children with two caregivers in formal childcare (n=877), of which 28.3% (95% CI: 25.4 ; 31.4) had OWOB. Similarly the obesity prevalence was 8.9% (95% CI: 6.1 ; 12.7) and 5.6% (95% CI:4.2 ; 7.3) in both groups, respectively, indicating the importance of the psychosocial environment of children by the time they reach the age of five years (results not shown).

An elevated OWOB prevalence due to an accumulation of biological factors was also seen, namely 27.8% (95% CI: 25.6 ; 30.1) in children born AGA who slept twelve or more hours (n=1,521), compared to 50.48% (95% CI: 43.7 ; 57.2) in children who were born LGA and slept less than 11 hours daily when they were five years old (n=208). In the latter group, the obesity prevalence was particularly high (21.6%, 95% CI:16.6 ; 27.7, compared to 6.1%, 95% CI: 5.0 ; 7.4, results not shown).

5.5 Summary

Findings from this chapter showed that inequalities in the risk of OWOB exist at the ages of nine months, three years and five years, and that these inequalities are likely to be mediated through biological, material and psychosocial pathways. While a more thorough discussion will be provided in Chapter 7, a synthesis of findings from this chapter will be provided as follows.

Both psychosocial and biological associations with OWOB already seemed to be present when the children were nine months old. OWOB levels differed based on the ethnic background, BWFGA, gestational age and rapid infant weight gain of children. All of these associations are indicative of biological causes of OWOB in this cohort. Also informal childcare attendance was associated with OWOB at nine months, suggesting psychosocial pathways to inequalities of OWOB at this young age, such as through the exposure to opposing health messages and caregiving styles. While minor differences in income were seen in the bivariate analysis with weight status, this disappeared in the adjusted multivariable logistic regression models and only a weak indication of material mechanisms at this age was seen through an elevated OWOB risk among children that did not belong to any social class.

Looking at OWOB prevalence when the children were three years old, similar patterns to the nine-months data emerged. Relationships between OWOB and various factors that support the biological theory remained and additional relationships, such as with caregiver weight status, were seen. Also the relationship between psychosocial factors and OWOB was strenthened, as indicated by the variations in OWOB risk based on household structure.

Also at the age of five years, biological and psychosocial pathways to OWOB appear to remain. Additionally, cues for material mechanisms were found that may explain how childhood obesity evolves. Fewer infants were obese if they lived in households with high equivalised household income or if at least one of their caregivers had third level education, with inverse trends seen for both SES variables. Furthermore, various neighbourhood characteristics were associated with OWOB, all of which confirms the material theory.

Taken together, the findings of this chapter support all theories (outlined in Section 2.4.5) and accordingly the second, third and fourth hypothesis of this thesis (Section 2.4.6). However, different associations were seen at different ages, suggesting that OWOB evolves through these theories at different time points in the lives of young children. Furthermore, children's previous weight status was most predictive of their subsequent weight status. Accordingly, weight should be investigated longitudinally, in order to account for changes that may take place both in children's BMI as well as the variables that may explain their weight gains. Additionally, given the multiple statistical testing conducted, it should be noted that the change of committing a Type I error (the likelihood of coming about a significant result by chance) is increased and no causal inferences are possible based on cross-sectional studies. Therefore, while this chapter provided a good overview of the different risk factors and mechanisms that may lead children to put on excess weight, the longitudinal study of the GUI infant cohort enables a more comprehensive investigation of early childhood obesity. The longitudinal analysis of the GUI data can hence build on the findings from this chapter and deepen the understanding of when various pathways may start impacting children's weight gains.

More relationships were identified with OWOB than with obesity in the cross-sectional analysis in this chapter. This, together with the fact that increasingly more factors were related to children's weight status with increasing age, suggests that most children exposed to obesity risk factors gradually gain excess weight over the first five years of life. Ideally, excess weight gains should be identified in children before they translate into differences in weight status. Small changes in weight gain based on various risk factors should accordingly be examined in the longitudinal analysis of the GUI infant cohort, rather than differences in the prevalence of weight status.

Lastly, only a few risk and protective factors emerged on the individual level, compared to the level of community, demographic and societal characteristics. However, most factors impacting weight status were seen on the level of family and parenting characteristics. The latter includes variables related to the gestational period, birth outcomes and infant feeding, which were dominant factors

related to the odds of OWOB, and of obesity separately. Also a nationally representative study in nine-year-old children in Ireland found that most inequalities in childhood obesity were related to the parental level (94). This leads to the conclusion that prevention of early childhood OWOB should not only target individual child behaviour, but the obesogenic environment they grow up in, including factors related to family characteristics and parenting practices.

CHAPTER 6:

Integration of multi-dimensional obesity risk factors into a multilevel model of child body mass index

Key points in this chapter

Aims of this chapter

- To investigate longitudinally critical time-points in relation to obesity risk factors and population characteristics associated with OWOB, and to measure the significance of each factor on population level in the age group up to five years
- To test whether the role of material, psychosocial and biological mechanisms on BMI developments may differ across the ages of young children in Ireland

Knowledge generation

- Obesity risk factors related to BMI changes over the course of children's first five years of life will be identified
- Critical ages within the early childhood years will be identified during which material, psychosocial and biological pathways may impact on BMI changes and predispose children to the onset of OWOB

6.1 Introduction

In Chapter 4 of this thesis, it was shown that the OWOB prevalence among young children in Ireland is high. Differences in the risk of OWOB were furthermore found in Chapter 5 which suggest that biological, material and psychosocial factors may contribute to the development of OWOB and hence create inequalities between young children in Ireland. These were found on different levels of the SEM, indicating that OWOB is a multifaceted health problem that requires prevention on various levels. However, only limited conclusions can be draw from the findings to this point, leaving a number of questions open that need to be addressed by more sophisticated analyses of children's weight gains in relation to risk factors of childhood obesity.

One of the limitations pointed out in the previous chapter is the binary classification of children as those with OWOB and those without. The classification of children's BMI into healthy and unhealthy ranges is important to identify groups of children who are at an increased risk of overweight or of obesity, and this was studied using logistic regression models as described in Chapter 5 (Section 5.3). This analysis identified risk factors that should be targeted in prevention strategies. However, in the analysis of OWOB as outcome variable, children with the highest BMI values were grouped together with those that just about fell into the category of overweight, but were very close to the defined cut-off value of normal weight in the respective age group. This analysis may therefore have failed to identify potential differences in risk factors for slightly overweight and for severely obese children. Furthermore, the classification of children's weight status depended on the use of a child growth criterion. Consequently, the analysis shared the limitations of the criterion in use, which are discussed in Chapter 4.

Furthermore, the cross-sectional analyses conducted in Chapter 5 and in many other studies do not follow the development of BMI in the same children over time. This is a major barrier to identifying critical time points in children's lives which could otherwise be used for the timely delivery of prevention programmes.

A more sophisticated analysis technique was therefore needed which accounts for the longitudinal structure of the data on the one hand and which is independent of weight status classification systems on the other hand.

As a result, multilevel linear regression analysis (mixed model) was used to study BMI (as a continuous outcome variable) longitudinally, following the children in the GUI infant cohort over the entire study period. Explanatory variables were included as potential risk factors of BMI changes over time. Using this analysis technique, a distinction can be made between, first, associations seen between BMI and obesity risk factors, and secondly, correlation that may exist between measurements that were conducted in the same child over time. This approach enabled the identification of small inequalities or small changes in BMI on population level based on potential risk factors, which appeared useful to test the fifth hypothesis of this thesis using the GUI infant cohort data.

Therefore, this chapter will shed light on the relationships identified in the previous chapter in a more advanced longitudinal analysis of children's BMI, based on the inclusion of obesity risk factors in multilevel models of BMI changes.

6.2 Chapter aims and hypothesis

This chapter aims to build on the findings from Chapter 5, namely to investigate OWOB risk factors and population characteristics that were associated with OWOB in relation to children's longitudinal BMI changes from the age of nine months to three and five years. This can help identify critical time points in order to direct prevention strategies at those children most in need before they start gaining excess weight. In Chapter 2, it was hypothesised that biological, psychosocial and material theories explain how OWOB evolves in young children in Ireland. Chapter 5 has already shown that these theories can explain inequalities that were observed in the OWOB risk of young children in Ireland. The following chapter accordingly aims to investigate these theories more in depth and identify potential differences in timing when these biological, psychosocial or material mechanisms start impacting children's excess weight gains. Risk factors will be investigated in the GUI infant cohort according to the different levels of the SEM and the *Complex web of potential determinants of overweight and obesity in children*, similar to Chapter 5. However, children will be followed longitudinally, that means the correlation of BMI within the same child can be studied separate to BMI changes that occur between children due to various risk factors.

6.3 Methods

In order to study the development of BMI in children longitudinally, data from the nationally representative GUI infant cohort were analysed using multilevel regression. Details on the methodology used by GUI can be found in Chapter 3. The following paragraphs will explain how this analysis technique was used to study BMI trajectories in the GUI infant cohort.

6.3.1 Variables

Outcome variables

The outcome variable was children's BMI which was calculated based on standardised height and weight measurements at the ages of nine months, three years and five years. Different to the analysis in Chapter 5, raw BMI scores were used continuously, which enables the analysis of children's BMI independent of child growth criteria and differentiates between mildly overweight and severely obese children. This has been described more in Chapter 3 (Section 3.3.5). As has been elaborated also in Chapter 3 (Section 3.3.2), use of BMI z-scores was considered, in addition to BMI raw scores which were used in this study; however a number of limitations of this approach led to the exclusion of z-scores from the main study (278, 280). However, sensitivity analysis was run using BMI z-scores as outcome variable.

Explanatory variables

OWOB risk factors on various levels of the SEM were integrated into the analysis, based on the literature reviewed in Chapter 2. Their measurement was described in Chapter 3 of this thesis (Section 3.3.3, e.g. Table 4) and descriptive statistics were shown in Chapter 5. In order to produce a set of comparable and meaningfully interpretable coefficients, commensurability of continuous variables needs to be assured, meaning that all continuous explanatory variables need to be

based on a standardised scale (392). Therefore, the scales of continuously measured explanatory variables in this analysis were standardised by means centring, which means that the mean value is given a value of zero and all other values of that variable can be interpreted as distances to the mean.

6.3.2 Analysis

There are two main approaches to fitting growth curves and trajectories: within the structural equation modelling framework, incorporating the observed repeated measures as multiple indicators on one or more latent factors, or within the multilevel framework (393). The longitudinal GUI infant cohort data can be viewed as having a two-level hierarchical structure and therefore multilevel modelling was used. (393)

Suppose you observe n data points $y_1, \ldots, y_n$ and that you want to explain them using n values for each of p explanatory variables $x_{11}, \ldots, x_{1p}, x_{21}, \ldots, x_{2p}, \ldots, x_{n1}, \ldots, x_{np}$. The x values may be either regression-type continuous variables or categorical variables. The standard general linear model is written as follows:

$$y_i = b_1 x_{i1} + \ldots + b_p x_{ip} + \varepsilon_i$$

Where $b_1, \ldots, b_p$ are unknown fixed-effects parameters to be estimated and $\varepsilon_1, \ldots, \varepsilon_n$ are unknown independent and identically distributed normal random variables with mean 0 and variance σ^2.

For simplicity the model can be written as follows:

$$y = Xb + \varepsilon$$

The mixed model extends the general linear model by allowing a more flexible specification of the covariance matrix of ε. In other words, it allows for both correlation and heterogeneous variances. The mixed model is written as follows:

ώ؎ ۨ ۨ ط ۨ ط

where X*ƀ* and ˧ are the same as in the general linear model, **Z** is the known design matrix and *ɔ* is the vector of unknown random-effects parameters. As for **X**, the matrix **Z** can contain either continuous or categorical variables. The name mixed model comes from the fact that the model contains both fixed-effects parameters, *ƀ*, and random-effects parameters, *ɔ* (394).

Multilevel modelling, which is also known as hierarchical linear modelling or random effects modelling, enables the investigation of data with a hierarchical structure; that is observations that are nested within groups. A typical example involves observations of children who are nested in schools. In this example, children (cases) in the same school (group) are expected to share a certain level of the same characteristics that are not shared by children from other schools; hence, these similarities need to be accounted for when the variability of an outcome in children is studied. This can be achieved by combining two equations that estimate the individual child-level effects and the group-level effects (392). Multilevel models can also be applied to data with repeated measurements on the same individuals. The children's age, or the timing of measurements, can be used as a level of data within each child, thereby accounting for residual variances that may occur within children over time, in addition to variation between children. Additionally, multilevel modelling can handle unbalanced data with different numbers of repeated measures per child, which is important as not all children in the GUI infant cohort have three BMI measurements reported (see Section 3.2.6 for details on attrition and Section 3.3.2 for missing BMI values). Accordingly, children could be included in the models if they had BMI data available at only one or two time-points, thus preventing unnecessary loss of data.

In the GUI infant cohort, multilevel models were used that describe the change of BMI, where level 1 describes the within-child variance, that is how a child's BMI changed over time, and level 2 describes the between-child differences in BMI over time. The BMI measurements nested within a child (level 1) are more likely to be correlated than BMI measurements between children (level 2). BMI data were available at three time-points, when the infants were nine months, three years and

five years old. Children were also included in the multilevel models if they had a valid BMI measurement available for only one or two of these time-points.

Explanatory variables were included as fixed effects in the multilevel models, in order to explain why the BMI (change) of some children differs from that of other children. However, these variables may not have the same relationship with BMI at each age. As indicated in Chapter 2, the BMI of young children does not develop linearly due to different rates in weight and length/height gains over time. Data on children's BMI were available at three time-points (nine months, three years and five years) and the measure of time (i.e. age) was included in the analysis. Time trend analysis was included by fitting an interaction term between each of the explanatory variables and waves (time), where *time 2* refers to wave 2 (i.e. when children were three years old) and *time 3* refers to wave 3 (when children were five years old). The analysis therefore provided information about the children's initial BMI (intercept) and their BMI trajectory during the study period (slope). This model allows for the relationship between explanatory variables and BMI at various ages to be explored (i.e. changes in BMI from nine months to three years and to five years). A number of multilevel models were run, building up the analysis based on the literature and previous results of this thesis.

Tests for interactions were run in each model to exclude non-significant interaction terms; that means for interactions with time where no time-variant association between an explanatory variable and BMI was seen (that is where an independent variable may change the intercept of BMI, but not the slope). This was important to reduce the complexity of the models. While it is important to adjust for the analysis for as many known confounders as possible, models with a high complexity are likely to hide the relationships between some confounders and the outcome variable if these confounders are related to each other. The risk is particularly high among the interaction terms of independent variables with time as not all relationships of independent variables with BMI may be time-dependent. Therefore, separate tests for interactions were run in each model, and interaction terms with $p > 0.05$ were excluded from the final models presented in this chapter. Variables were also tested for multicollinearity from within the model, calculating the variance inflation factor of each explanatory variable and interaction term.

For the mixed models analysis, robust standard errors were used. These are standard error estimates that correct for dependence among the repeated observations. Furthermore, for interpretive purposes, variables in regression models were mean-centred.

The weighting factors developed by the GUI researchers for each wave have been incorporated in the analysis in order to adjust for external population characteristics, as has been described in detail in Chapter 3 (Section 3.2.6). The intraclass correlation, which describes the correlation of BMI measurements within children, was assessed for each model individually.

Specification of models

First, a basic model (Model 1) was established that did not adjust for any demographic information or obesity risk factors. This model was run in order to get an idea of the unadjusted development of BMI in this cohort. Secondly, Model 1 was used as the basis for four more multilevel models that were used to explore the data. In the analysis of these four models, BMI change over time was investigated according to various demographic characteristics and obesity risk factors.

Model 2 is an extension of Model 1, adding sex, as well as interaction terms with sex to the fixed effect of the model. This addition is based on the observation of different growth curves and trajectories that exist between the sexes at the different ages and that were therefore integrated in international growth references, as has been described in Chapter 4. The interaction terms with the time variables were included based on the assumption that a possible relationship between sex and BMI may not be constant over time, but change between different time points measured in the GUI infant cohort.

Chapter 5 showed that the OWOB prevalence differs between demographic groups, suggesting that material resources may play a role in the development of childhood obesity. Therefore, in Model 3, SES variables and the ethnic background

of children were added to the multilevel model, in addition to sex. Interaction terms were included for all variables with significant tests for interactions ($p<0.05$).

Model 4 includes those explanatory variables that were associated with BMI in the previous multilevel models, either based on the baseline measure or based on an interaction term between the respective explanatory variable and time. Additional explanatory variables were included in this model that provided information related to the child's birth and early life, parental characteristics, neighbourhood variables and early lifestyle (see Table 4 in Section 3.3.3 for a list of all variables). This was to further test theories of biological and psychosocial mechanisms that may explain why some children put on excess weight. A number of lifestyle variables from the child were only recorded in waves 2 and 3 of the GUI data. These variables were excluded from Model 4 for two reasons. First, at nine months of age, children cannot be understood to follow a 'lifestyle' based on family food choices and physical activity. At this age, children start transitioning from infancy to toddlerhood, which involves frequent changes with regards to their weaning, eating, physical activity and play routines. It appears unreasonable to use their momentary lifestyle at the time of the family interview as a proxy for overall infancy lifestyle while they undergo so many changes. Secondly, beyond this theoretical consideration, the analysis of lifestyle variables in the wave 1 data was also not possible computationally. As the GUI team had only started recording food item consumption, physical activity and screen time use when the children were three years old, all observations from the first wave were excluded from the model when these lifestyle variables were included in the model.

In order to adjust for lifestyle variables that were not included in Model 4, Model 5 was run including all measured explanatory variables but analysing the outcome of BMI changes only based on the three-year- and five-year-measurements. This allowed the integration of lifestyles that had not been established at the children's age of nine months. Explanatory variables that were only measured at wave 1, such as birth characteristics and infant feeding, were carried forward and analysed in relation to later BMI as the literature has shown a long-lasting relationship with early-life events (Chapter 2). In turn, variables measured later in life cannot automatically be understood to be similarly associated with earlier BMI changes.

Nevertheless, lifestyle variables referring to the nutrition and physical activity of children were considered to reflect on overall family lifestyle, even if they had only been measured in the GUI data when the children were five years old. As children are expected to be well integrated in most family lifestyles by their age of three years, lifestyle variables measured in wave 3 were as well used as a proxy for lifestyle at the age of three years in Model 5. Lastly, the analysis was adjusted for BMI at nine months, in order to identify relationships between later BMI and lifestyle that are independent of earlier BMI developments. Therefore, Model 5 includes explanatory variables and BMI measured in wave 1, and additionally children's lifestyle variables measured in waves 2 and 3, to investigate BMI changes in relation to only two time points (waves 2 and 3).

6.3.3 Sensitivity analysis

Additionally, for Models 3, 4 and 5, four models each were run for sensitivity analysis. For each of these models, sensitivity analysis was conducted including only singleton births (Model 3a: n=10,335; Model 4a: n=10,008; Model 5a: n=7,798), excluding families in which the SCG existed but had not completed the interview (Model 3b: n=9,715; Model 4b: n=9,394; Model 5b: n=7,293), or including only families with two caregivers living in the household and completed PCG and SCG interviews (Model 3c: n=8,293; Model 4c: n=8, 024; Model 5c: n=6,470). Additionally, one model was run including only families in which both the person who participated in the interviews as the SCG and the person who was the PCG did not change throughout the study period, (Model 3d: n=10,306; Model 4d: n=9,973; Model 5d: n=7,729).

As rapid early weight gains may mediate the association between infant feeding and BMI in children (168), all models were additionally run without the inclusion of rapid infant weight gain as an exploratory variable. All models were additionally run using BMI z-scores as alternative outcome variable.

6.4 Results

The original sample interviewed in the GUI infant cohort included the families of 11,134 infants at baseline. After the exclusion of missing values, outliers and invalid values, 10,810 had a valid BMI reported at nine months of age, 9,422 at three years and 8,781 at five years of age. Furthermore, a number of children were excluded from the analysis because their PCG changed over time, resulting in 10,763 children who were included in the baseline model presented below. Children were included that may not have had a valid BMI recorded at nine months, but only at a later age, or vice versa. Fewer children were included in the extended models due to missing or invalid values in the explanatory variables which the respective models were adjusted for.

6.4.1 Baseline model

To start with, a baseline (unadjusted) multilevel model with mixed effects (Model 1, n=10,763) was set up, as depicted in Table 23. The intraclass correlation coefficient (ICC) was 0.52 (95% CI: 0.52 ; 0.52), implying that 52% of the variance in this model was explained by within-child differences.

Statistically significant differences in BMI were seen across the ages. Children had an average BMI of 18.17 (95% CI: 18.13 ; 18.2) at their age of nine months, which significantly decreased to 16.83 kg/m^2 at the age of three years and 16.25 kg/m^2 at five years.

In order to minimise the error term and the variation explained by random effects, Model 1 was extended and explanatory variables were added to the model gradually, as shown in the following sections.

Table 23: Multilevel Model 1, showing Body Mass Index (BMI) trajectories between nine months, three years and five years of age unadjusted. Point estimates and 95% Confidence Intervals (CI) are shown.

Model components	**BMI ß-coefficient (95% CI) at age 9 months**
Number (observations)	28268
Number (individuals)	10763
Constant	**18.17** (18.13 ; 18.20)***
Log-pseudolikelihood	-49906.78
Wald Chi-Square	8129.89
Change in BMI since the age of 9 months	
Until 3 years	**-1.34** (-1.38 ; -1.30)***
Until 5 years	**-1.92** (-1.96 ; -1.88)***
Random effect components	
Residual SD	1.01 (0.95 ; 1.08)
Measurement (time) 2	0.24 (0.16 ; 0.36)
Measurement (time) 3	0.56 (0.45 ; 0.70)
Random intercept SD	1.39 (1.33 ; 1.46)
Intraclass correlation coefficient id	0.52 (0.52 ; 0.52)

SD = standard deviation

*** p<0.001

6.4.2 Inclusion of sex

Model 1 was extended by adding sex and interactions of sex with the time variables (i.e. age) in the fixed part of the multilevel model. As can be seen in Table 24, the newly generated Model 2 differs slightly from Model 1. Compared to Model 1, the error term of the random effect in Model 2 was only reduced marginally and the ICC of Model 2 remained constant (Table 24).

Mean BMI is now displayed for boys, who appear to have a BMI of 18.37 (95% CI: 18.33 ; 18.42) at the age of nine months, 16.95 at three years and 16.27 at five years. Girls, in contrast, have a significantly lower BMI at nine months of age, which is still lower at three years despite an increased weight gain taking place between these two time points. By the age of five years, the BMI of girls and boys is almost the same, as girls continued to gain significantly more weight relatively to boys.

Table 24: Multilevel Model 2, showing Body Mass Index (BMI) trajectories between nine months, three years and five years of age adjusted for sex. Point estimates and 95% Confidence Intervals (CI) are shown.

Model components	**BMI ß-coefficient (95% CI) at age 9 months**
Number (observations)	28,268
Number (individuals)	10,763
Constant	**18.37** (18.33 ; 18.42)***
Log-pseudolikelihood	-49791.60
Wald Chi-Square	8411.91
Time (9 months) [a]	
3 years	**-1.42** (-1.47 ; -1.36)***
5 years	**-2.10** (-2.16 ; -2.04)***
Sex (boys)[a]	
Girls	**-0.42** (-0.49 ; -0.35)***
Girls * time 2	**0.15** (0.07 ; 0.23)***
Girls * time 3	**0.38** (0.29 ; 0.46)***
Random effect components	
Residual SD	1.00 (0.93 ; 1.07)
Measurement (time) 2	0.25 (0.17 ; 0.37)
Measurement (time) 3	0.56 (0.45 ; 0.71)
Random intercept SD	1.38 (1.31 ; 1.44)
Intraclass correlation coefficient id	0.52 (0.51 ; 0.53)

a = reference group

SD = standard deviation

*** p<0.001

6.4.3 Inclusion of socio-economic status variables and ethnicity

In addition to sex which was included in the multilevel model in the previous section, Model 3 also controls for a number of SES variables and ethnicity, as well as their interaction terms with time, as can be seen in Table 25. The fixed effect of the model was significantly contributing to differences in BMI changes (p<0.001, results not shown). While Model 3 identified additional population groups that follow elevated or lower mean BMI trajectories, the ICC remained at 52% (95% CI: 0.52 ; 0.52). However, the error term of the random effect was reduced, implying that Model 3 may be a better fit than the previous models.

In Irish white boys, whose families belong to the managerial and technical household class, lie in the highest income quintile and where at least one caregiver has third level education, BMI at nine months of age was on average 18.39 (95% CI: 18.31 ; 18.47). At three years of age, their average BMI had decreased to 16.95 kg/m^2 and at five years to 16.14 kg/m^2. Similar associations between sex and BMI over time were seen in Model 3 like in Model 2.

Additional associations were seen between BMI and ethnic background, household income and education of caregivers. At nine months of age, non-Irish white children had a significantly lower BMI than Irish white children, and this difference was not found to change as the children grew older. A similar trend was seen in children of other, including mixed background. Children from Asian, but not Chinese background had the lowest average BMI at the age of nine months. However, their relative weight gain increased significantly compared to that of Irish white children, which reversed the difference seen between these groups at the age of five years slightly. At five years of age, also African children had a BMI that was significantly higher than that of Irish white children, even though no significant difference between these groups was seen at earlier ages. Chinese children at five years of age had a very high BMI; however the wide 95% CI of this coefficient indicates that further analysis may be required to confirm this association.

Compared to children living in families in the highest income quintile, those in the lowest quintile had a slightly higher average BMI across all ages. Additionally, compared to children with at least one caregiver who had acquired third level education, children whose caregivers had other post-secondary education or only lower secondary education had a higher BMI at five years of age.

Sensitivity analysis

A few differences were seen between the original Model 3 and the sensitivity models. A number of variables were no longer significant in some of the models used for sensitivity analysis, most likely due to decreasing sample size. In particular, income was no longer associated with BMI in Models 3b-d. Furthermore, Asian ethnicity at the age of three years and Chinese ethnicity at the age of five years were no longer significantly associated with BMI (Models 3b and 3c). However, lower secondary education of the caregivers at three years ($\beta = 0.18$; 95% CI: <0.01 ; 0.35), and higher secondary education at five years ($\beta = 0.17$; 95% CI: 0.01 ; 0.32) were significantly related to higher BMI scores, compared to caregivers with third level education (Model d).

Table 25: Multilevel Model 3, showing Body Mass Index (BMI) trajectories between nine months, three years and five years of age, adjusted for sex, socio-economic status variables and ethnicity. Point estimates and 95% Confidence Intervals (CI) are shown.

	Main effects	**Interaction terms with time [b]**	
Model components	**BMI ß-coefficient (95% CI) at age 9 months**	**BMI ß-coefficient (95% CI) at age 3 years**	**BMI ß-coefficient (95% CI) at age 5 years**
Number (observations)	28,149		
Number (individuals)	10,717		
Constant	**18.39** (18.31 ; 18.47)***		
Log-pseudolikelihood	-49495.77		
Wald Chi-Square	9888.29		
Time (9 months) [a]			
3 years	N.A.	**-1.44** (-1.51 ; -1.37)***	N.A.
5 years	N.A.	N.A.	**-2.25** (-2.33 ; -2.18)***
Sex (boys)[a]			
Girls	**-0.42** (-0.49 ; -0.35)***	**0.15** (0.07 ; 0.22)***	**0.36** (0.28 ; 0.45)***
Household class (managerial and technical) [a]			
Professional	-0.04 (-0.10 ; 0.03)	N.A.	N.A.
Non-manual	0.01 (-0.06 ; 0.07)	N.A.	N.A.
Skilled manual	0.01 (-0.06 ; 0.09)	N.A.	N.A.
Semi-skilled	0.01 (-0.08 ; 0.10)	N.A.	N.A.
Unskilled	0.17 (-0.04 ; 0.38)	N.A.	N.A.
Other	-0.01 (-0.28 ; 0.28)	N.A.	N.A.
Unclassified	0.07 (-0.03 ; 0.17)	N.A.	N.A.
Ethnic background (Irish white)[a]			
Any other white background	**-0.33** (-0.46 ; -0.20)***	-0.07 (-0.24 ; 0.10)	0.06 (-0.10 ; 0.23)
African	0.08 (-0.19 ; 0.35)	0.14 (-0.20 ; 0.48)	**0.51** (0.15 ; 0.87)**
Any other Black background	0.05 (-0.67 ; 0.77)	0.51 (-0.37 ; 1.40)	0.50 (-0.21 ; 1.21)
Chinese	-0.32 (-1.10 ; 0.47)	0.11 (-1.15 ; 1.36)	**2.44** (0.07 ; 4.81)*
Any other Asian background	**-0.78** (-1.04 ; -0.53)***	**0.38** (0.06 ; 0.69)*	**0.83** (0.46 ; 1.19)***
Other, including mixed background	**-0.20** (-0.31 ; -0.09)***	-0.01 (-0.12 ; 0.11)	0.03 (-0.09 ; 0.16)
No secondary caregiver data	-0.11 (-0.23 ; 0.01)	0.09 (-0.04 ; 0.23)	0.13 (-0.02 ; 0.28)
Equivalised household income (highest quintile)[a]			
Lowest quintile	**0.10** (0.02 ; 0.18)*	N.A.	N.A.
2nd quintile	0.04 (-0.03 ; 0.12)	N.A.	N.A.
3rd quintile	0.06 (-0.01 ; 0.12)	N.A.	N.A.

	Main effects	Interaction terms with time [b]	
Model components	**BMI ß-coefficient (95% CI) at age 9 months**	**BMI ß-coefficient (95% CI) at age 3 years**	**BMI ß-coefficient (95% CI) at age 5 years**
4th quintile	0.01 (-0.05 ; 0.07)	N.A.	N.A.
Missing	0.01 (-0.09 ; 0.10)	N.A.	N.A.
Highest education of caregivers (third level) [a]			
Lower secondary education or less	-0.02 (-0.15 ; 0.11)	0.15 (-0.01 ; 0.32)	**0.37** (0.16 ; 0.58)***
Higher secondary education	0.07 (-0.03 ; 0.17)	-0.07 (-0.19 ; 0.06)	0.13 (-0.02 ; 0.28)
Post-secondary education	-0.03 (-0.10 ; 0.05)	0.05 (-0.04 ; 0.13)	**0.22** (0.13 ; 0.31)***
Random effect components			
Residual SD	0.99 (0.93 ; 1.06)		
Measurement (time) 2	0.25 (0.17 ; 0.37)		
Measurement (time) 3	0.54 (0.44 ; 0.68)		
Random intercept SD	1.35 (1.29 ; 1.42)		
Intraclass correlation coefficient id	0.52 (0.52 ; 0.52)		

a = reference group

b = β-coefficients in the columns under 'Interaction terms with time' display the change in BMI differences between groups (categorical variables) or linear changes with increasing score (continuous variable) from nine months to three years and from nine months to five years of age, respectively

N.A. = not applicable, no interaction with time was included in the analysis

SD = standard deviation

* p<0.05

** p<0.01

*** p<0.001

6.4.4 Inclusion of birth and infancy factors, parental characteristics, neighbourhood variables and early lifestyle

Building on Model 3 that was presented in the previous section, a number of additional explanatory variables were added to the multilevel Model 4. These variables include information on the development of children prior birth and during infancy, parental characteristics, parental perceptions of the neighbourhood and children's lifestyles measured in wave 1. Household social class was removed from the model, as no associations had been seen in Model 3. The results of Model 4 are shown in Table 26. The estimate of the ICC was 0.45 (95% CI: 0.44 ; 0.47) and the error terms were smaller than in the previous models. The fixed part of the model was significantly associated with BMI ($p<0.001$).

Based on Model 4, mean BMI of the reference groups taken together was 18.03 (95% CI: 17.9 ; 18.16) at the age of nine months, 16.48 at three years and 15.47 at five years. Sex was associated with BMI at nine months and five years of age, but the β-coefficients were smaller than in the previous models. Furthermore, by the age of five years, the trend had fully reversed, meaning that girls had a higher BMI than boys at that age.

Socio-economic status and ethnic background

Among ethnic groups, the same trend was seen in non-Irish white, African and Asian (excluding Chinese) children in Model 4 as had been seen in Model 3. However, the discrepancy between Irish and non-Irish white children was reduced. Furthermore, the difference between Irish white and Asian (excluding Chinese) children at their age of nine months was lower in Model 4, while the other coefficients remained stable. The associations in Model 3 seen between BMI changes and Chinese background, as well as with other or mixed ethnicity, were not significant in Model 4. Differences seen in the nine-months-old children that had no SCG data available in Model 4 were outbalanced at the later ages.

Differences seen between BMI and household income did not differ between Models 3 and 4. Also the association with caregiver education seen in Model 3 was significant in Model 4. However, children whose caregivers had only lower secondary education or less already had significantly higher BMI scores than those whose caregivers had third level education at the age of three years. This association had not been significant in Model 3.

Moreover, a number of other variables included in Model 4 were related to BMI.

Region, household structure, and the perceived neighbourhood

Children who lived in urban regions had a lower average BMI than those from rural regions, and this difference did not change significantly as children grew older. Differences were also seen based on the household structure.

Compared to children with two caregivers and at least one sibling living in the household, single children with two caregivers had a lower average BMI at nine months of age. However, by five years, their BMI was significantly higher. Additionally, at five years, single children as well as children with one or more siblings living in lone caregiver households had a higher BMI than those in families with two caregivers and one or more siblings. The highest BMI was seen in single children with one caregiver, which was already elevated at the age of three years. No associations with BMI were seen depending on the caregiver perception of the level of safety or the availability of play and green spaces in the neighbourhood.

Birth and infancy factors

The largest deviations in BMI were seen in relation to birth and early life factors (Table 26). At nine months of age, compared to term infants, children born very early (β=-2.00; 95% CI: -2.3 ; -1.69), and somewhat early (β=-1.09 ; 95% CI: -1.23 ; -0.94) had a significantly lower average BMI. However, those born late had a 0.14 kg/m^2 (95% CI: 0.04 ; 0.24) higher BMI. At the ages of three and five years, very

early and early born children had a relative increase in BMI compared to term children, while late children stayed on their higher trajectory. By five years, very early born children and early born children had reduced the differences to term children seen at nine months of age. Furthermore, compared to children born AGA, those born SGA had a significantly lower BMI at nine months, but reduced that gap until the age of five years. Children born LGA had a constantly higher BMI, but a smaller weight gain than AGA children until three years. Children who were breastfed for six months or more, compared to those never breastfed, had a significantly lower BMI at nine months of age; however a relatively higher (but not significant) weight gain brought them closer to the BMI trajectory of non-breastfed children at three and five years. No significant relationship was seen with BMI and the timing of solid food onset. Rapid infant weight gain was significantly associated with BMI. When the children were nine months old, those with rapid infant weight gain had a 1.9 kg/m^2 (95% CI: 1.81; 1.98) higher average BMI than those without rapid weight gain. BMI trajectories of children with rapid weight gain decelerated from nine months onwards, but their BMI scores remained higher overall.

Caregiver weight status and stress

Furthermore, the BMI of children differed at the ages of three and five years based on the weight status of their caregivers. In PCGs, compared to normal or underweight, overweight was related to increases in BMI trajectories from three to five years and obesity was associated with a constantly increasing BMI trajectory in children. Among SCGs, compared to normal or underweight, overweight and obesity status both were associated with increasing BMI trajectories from nine months to five years. At five years, the gap had risen to 0.3 kg/m^2 (95% CI: 0.20; 0.41) if the SCG was overweight and to 0.62 kg/m^2 (95% CI: 0.48; 0.76) if the SCG was obese. There were small associations seen between PCG stress scores at nine months and three years of age which evened out by five years.

Sleep and childcare

On average, children who were in informal childcare arrangements had a slightly higher BMI at all ages than children who did not attend childcare regularly. Additionally, children with short sleep duration had a significantly lower BMI than those with normal sleep at nine months, but a higher relative weight gain until three and five years, therefore exceeding the BMI trajectory of normal sleepers.

Sensitivity analysis

Sensitivity analysis did not alter the overall findings (results not shown). There were, however, a few minor differences noted compared to the original model, most likely due to much smaller sample sizes. In particular, the association between BMI and income (Models 4b-d), and with urban region (Model 4c) was no longer significant. Late birth (Models 4b and 4c) and PCG stress level (Models 4a-d) were no longer significantly associated with BMI at nine months. At three years, very early birth (Model 4c), Asian ethnicity (Models 4b and 4c) and lower secondary education (Models 4b and 4c), and finally, at five years, very early birth (Model 4c), LGA births (Models 4b-d) and lower secondary education or less (Model 4b), were no longer significantly associated with BMI.

Table 26: Multilevel Model 4, showing Body Mass Index (BMI) trajectories between nine months, three years and five years of age adjusted for demographics, birth and infancy factors, parental characteristics, neighbourhood variables and early lifestyle. Point estimates and 95% Confidence Intervals (CI) are shown.

	Main effects	**Interaction terms with time [b]**	
Model components	**BMI ß-coefficient (95% CI) at age 9 months**	**BMI ß-coefficient (95% CI) at age 3 years**	**BMI ß-coefficient (95% CI) at age 5 years**
Number (observations)	26,867		
Number (individuals)	10,377		
Constant	**18.03 (17.90 ; 18.16)*** **		
Log-pseudolikelihood			
Wald Chi-Square			
Time (9 months) [a]			
3 years	N.A.	**-1.55** (-1.70 ; -1.40)***	N.A.
5 years	N.A.	N.A.	**-2.56** (-2.73 ; -2.40)***
Sex (boys)[a]			
Girls	**-0.19** (-0.26 ; -0.13)***	0.04 (-0.03 ; 0.12)	**0.25** (0.17 ; 0.33)***
Ethnic background (Irish white)[a]			
Any other white background	**-0.22** (-0.34 ; -0.09)***	-0.08 (-0.26 ; 0.10)	0.01 (-0.17 ; 0.19)
African	-0.01 (-0.27 ; 0.25)	0.16 (-0.21 ; 0.53)	**0.5** (0.11 ; 0.88)*
Any other Black background	0.17 (-0.48 ; 0.82)	-0.25 (-0.76 ; 0.26)	-0.4 (-0.97 ; 0.16)
Chinese	-0.37 (-1.09 ; 0.35)	1.13 (-0.11 ; 2.38)	2.53 (-0.02 ; 5.08)
Any other Asian background	**-0.52** (-0.75 ; -0.29)***	**0.36** (0.02 ; 0.70)*	**0.81** (0.42 ; 1.2)***
Other, including mixed background	-0.08 (-0.19 ; 0.02)	-0.04 (-0.16 ; 0.08)	0.01 (-0.12 ; 0.13)
No secondary caregiver data	**-0.33** (-0.49 ; -0.16)***	**0.33** (0.14 ; 0.51)***	**0.32** (0.14 ; 0.49)***
Equivalised household income (highest quintile)[a]			
Lowest quintile	**0.10** (0.02 ; 0.18)*	N.A.	N.A.
2nd quintile	0.05 (-0.03 ; 0.12)	N.A.	N.A.
3rd quintile	0.05 (-0.01 ; 0.12)	N.A.	N.A.
4th quintile	0.02 (-0.04 ; 0.08)	N.A.	N.A.
Missing	0.01 (-0.10 ; 0.10)	N.A.	N.A.
Highest education of caregivers (third level) [a]			
Lower secondary education or less	-0.05 (-0.17 ; 0.07)	**0.19** (0.01 ; 0.38)*	**0.34 (**0.13 ; 0.55**)****
Higher secondary education	0.05 (-0.04 ; 0.14)	-0.04 (-0.17 ; 0.08)	0.09 (-0.06 ; 0.25)
Post-secondary education	-0.07 (-0.15 ; 0.01)	0.07 (-0.02 ; 0.16)	**0.21** (0.11 ; 0.30)***
Region (rural)[a]			
Urban	**-0.05** (-0.10 ; -0.01)*	N.A.	N.A.

	Main effects	Interaction terms with time [b]	
Model components	**BMI ß-coefficient (95% CI) at age 9 months**	**BMI ß-coefficient (95% CI) at age 3 years**	**BMI ß-coefficient (95% CI) at age 5 years**
Household structure (two caregivers, two or more children)[a]			
Lone caregiver, one child	-0.08 (-0.24 ; 0.09)	**0.29** (0.08 ; 0.5)**	**0.49** (0.24 ; 0.73)***
Lone caregiver, two or more children	0.11 (-0.06 ; 0.27)	0.03 (-0.19 ; 0.25)	**0.29** (0.06 ; 0.51)*
Two caregivers, one child	**-0.08** (-0.15 ; -0.01)*	0.9 (-0.01 ; 019)	**0.26** (0.12 ; 0.41)***
Neighbourhood perceived as safe (agree) [a]			
Strongly agree	0.02 (-0.03 ; 0.06)	N.A.	N.A.
Disagree	0.01 (-0.05 ; 0.06)	N.A.	N.A.
Strongly disagree	-0.01 (-0.12 ; 0.10)	N.A.	N.A.
Neighbourhood perceived to offer green and play spaces (agree) [a]			
Strongly agree	-0.01 (-0.06 ; 0.04)	N.A.	N.A.
Disagree	0.02 (-0.03 ; 0.07)	N.A.	N.A.
Strongly disagree	-0.01 (-0.07 ; 0.06)	N.A.	N.A.
Gestational age at birth (on time)[a]			
Very early	**-2.00** (-2.30 ; -1.69)***	**0.61** (0.27 ; 0.95)***	**0.69** (0.29 ; 1.08)**
Somewhat early	**-1.09** (-1.23 ; -0.94)***	**0.42** (0.24 ; 0.6)***	**0.37** (0.19 ; 0.55)***
Late	**0.14** (0.04 ; 0.24)**	0.02 (-0.10 ; 0.13)	0.02 (-0.08 ; 0.18)
Birth-weight-for-gestational-age (appropriate)[a]			
Small-for-gestational age	**-1.01** (-1.12 ; -0.90)***	**0.29** (0.16 ; 0.43)***	**0.39** (0.24 ; 0.54)***
Large-for-gestational-age	**0.78** (0.68 ; 0.88)***	**-0.14** (-0.26 ; -0.01)*	**-0.14** (-0.27 ; -0.01)*
Missing	-0.25 (-0.75 ; 0.24)	0.06 (-0.41 ; 0.54)	-0.22 (-0.55 ; 0.11)
Breastfeeding duration (not breastfed)[a]			
Less than three months	-0.02 (-0.10 ; 0.07)	-0.05 (-0.15 ; 0.04)	-0.02 (-0.13 ; 0.08)
Three to under six months	-0.04 (-0.15 ; 0.07)	0.01 (-0.13 ; 0.13)	-0.01 (-0.14 ; 0.14
Six months or more	**-0.13** (-0.23 ; -0.04)**	0.1 (-0.01 ; 0.21)	0.06 (-0.05 ; 0.18)
Introduction of solid food (between age four and under six months)[a]			
Before age four months	0.05 (-0.02 ; 0.12)	N.A.	N.A.
After age six months or later	-0.04 (-0.09 ; 0.02)	N.A.	N.A.
Rapid infant weight gain (no rapid weight gain)[a]			
Rapid weight gain	**1.90** (1.81 ; 1.98)***	**-0.83** (-0.92 ; -0.73)***	**-0.98** (-1.09 ; -0.87)***
Missing	**0.67** (0.3 ; 1.04)***	-0.14 (-0.44 ; 0.16)	omitted
BMI status of the primary caregiver (normal/ underweight)[a]			
Overweight	0.03 (-0.04 ; 0.11)	0.05 (-0.03 ; 0.14)	**0.24** (0.15 ; 0.34)***

	Main effects	Interaction terms with time [b]	
Model components	**BMI ß-coefficient (95% CI) at age 9 months**	**BMI ß-coefficient (95% CI) at age 3 years**	**BMI ß-coefficient (95% CI) at age 5 years**
Obese	0.03 (-0.07 ; 0.12)	**0.29** (0.17 ; 0.42)***	**0.51** (0.38 ; 0.64)***
Missing	0.09 (-0.09 ; 0.27)	0.22 (-0.01 ; 0.44)	**0.31** (0.08 ; 0.54)**
BMI status of the secondary caregiver (normal/ underweight)[a]			
Overweight	-0.03 (-0.11 ; 0.05)	**0.16** (0.06 ; 0.26)**	**0.30** (0.20 ; 0.41)***
Obese	-0.02 (-0.12 ; 0.09)	**0.35** (0.22 ; 0.48)***	**0.62** (0.48 ; 0.76)***
No secondary caregiver interview	**0.25** (0.13 ; 0.37)***	**-0.19** (-0.34 ; -0.04)**	omitted
Missing	-0.07 (-0.27 ; 0.14)	**0.32** (0.07 ; 0.57)*	**0.25** (0.01 ; 0.49)*
Primary caregiver stress			
Continuous score	**-0.01** (-0.01 ; -0.01)*	**0.01** (0.01 ; 0.02)*	0.01 **(**-0.01 ; 0.02)
Childcare (no childcare arrangement)[a]			
Informal childcare	**0.06** (0.01 ; 0.11)*	N.A.	N.A.
Formal childcare	0.01 (-0.05 ; 0.05)	N.A.	N.A.
Daily sleep duration (short sleep)[a]			
Normal sleep	**-0.11** (-0.19 ; -0.02)*	**0.14** (0.03 ; 0.26)*	**0.20** (0.08 ; 0.32)**
Long sleep	-0.06 (-0.15 ; 0.03)	-0.04 (-0.17 ; 0.09)	-0.06 (-0.18 ; 0.05)
Random effect components			
Residual SD	0.85 (0.79 ; 0.91)		
Measurement (time) 2	0.36 (0.28 ; 0.47)		
Measurement (time) 3	0.59 (0.48 ; 0.72)		
Random intercept SD	1.00 (0.95 ; 1.06)		
Intraclass correlation coefficient id	0.45 (0.44 ; 0.47)		

a = reference group

b = β-coefficients in the columns under 'Interaction terms with time' display the change in BMI differences between groups (categorical variables) or linear changes with increasing score (continuous variable) from nine months to three years and from nine months to five years of age, respectively

N.A. = not applicable, no interaction with time was included in the analysis

SD = standard deviation

* p<0.05

** p<0.01

*** p<0.001

6.4.5 Model 5 – Inclusion of child's lifestyle v years of age

Table 27 presents the results of the multilevel Model 5 that assessed BMI only at the ages of three and five years, and that includes lifestyle variables that were not measured at nine months, in addition to the explanatory variables included in the previous model (Model 4).

In Model 5, 64% (95% CI: 0.62 ; 0.66) of the variance of BMI was explained by the random part of the model. The error terms differed from the previous models, however the random part of Model 5 only describes the change between two, rather than three time points studied. The fixed part of the model was significantly associated with BMI ($p<0.001$).

The mean BMI of three-year-old children in the reference population of this model was 16.62 (95% CI: 16.49 ; 16.75). At five years of age, it was significantly lowered to 15.56. At three years of age, the BMI of children was 0.39 kg/m^2 (95% CI: 0.37 ; 0.41) higher for every 1 kg/m^2 increase in the child's BMI at their age of nine months. At five years of age, a negative association was seen with the child's nine months' BMI, namely by 0.05 kg/m^2 (95% CI: -0.07 ; -0.03) for every 1 kg/m^2 increase in BMI at nine months. Like in Model 4, also in this model girls significantly exceeded the BMI trajectories of boys by the time they were five years old, even though they had still been on a lower trajectory at three years of age.

Socio-economic status and ethnic background

Similarities with Model 4 existed in this model based on the ethnic background of children. At three years of age, children with a non-Irish white background had a significantly lower average BMI than those of Irish white background, and this difference was kept by the time they were five years old. A similar trend was seen in children in the other and mixed background group. At the age of five years, furthermore, elevated BMI scores were seen in children with African, Chinese and other Asian ethnic background. These children significantly exceeded the average

BMI of Irish white children by far, even though no differences in BMI had been seen at the age of three years.

Also with regards to the highest educational level of the child's caregivers, trends were similar between Models 4 and 5. Compared to caregivers with third level education, children whose caregivers had lower secondary education or less had a higher BMI at the age of three years that increased until they were five years old. Also in those children whose caregivers had higher secondary or post-secondary education, significantly higher BMI scores were observed at the age of five years. However, unlike the findings from Models 3 and 4, no association was seen between the equivalised household income of families and children's BMI.

Region, household structure, and the perceived neighbourhood

An overall trend towards higher average BMI scores in children living in rural regions was seen in Model 5, just like in Model 4. Also trends based on the household structure were seen in Model 5. Children had an elevated average BMI at the age of five years if they did not live in a two-caregiver household with at least one sibling. However, both at three and at five years the deviations between the reference group and other household structures were smaller than those that had been observed in Model 4.

In contrast to Model 4, a small but significant relationship was seen between BMI and the perceived neighbourhood. Children were found to have a higher BMI overall if their PCG disagreed to the question whether their neighbourhood offered enough green and play spaces, compared to those PCGs who agreed. No association existed with the level of perceived neighbourhood safety.

Birth and infancy factors

In contrast to Model 4, BMI differences found in some of the variables in this group did not vary over time in Model 5. Children who were born very early or early had a

significantly lower average BMI than term children, overall. The BMI of children born later was significantly higher. Children born SGA had a significantly lower average BMI than AGA born children overall, and those born LGA had a significantly higher BMI. While the differences between groups in both variables appear high, they are much smaller than those found in Model 4. The same was found for rapid infant weight gain, which led children to have a significantly higher BMI in Model 5 overall, compared to children without rapid infant weight gain, but this deviation was much smaller than the one seen at the age of nine months in Model 4.

In contrast to Model 4, breastfeeding in Model 5 was not associated with BMI changes but the onset of solid food in the infant's diet was. Children who started eating solid food only after the age of six months or later had a significantly lower average BMI in Model 5 overall than those children who started eating solid food when they were between four and six months old.

Caregiver weight status and stress

Associations seen between caregiver BMI status and child BMI were similar in Models 4 and 5, despite differences in the missing categories. In PCGs, overweight led children to have a significantly higher BMI than those with a normal or underweight PCG only at the age of five years. Obesity, however, was associated with an elevated and increasing BMI trajectory seen from three to five years. Among SCGs, both overweight and obesity were related to a higher BMI when the children were three years old and to an increasing BMI until the age of five years, compared to children whose SCG was normal or underweight. No association was seen between PCG stress score and child BMI in Model 5.

Sleep and childcare

Like in Model 4, children who were in informal, compared to no childcare arrangements, had a slightly but significantly increased BMI in Model 5. Sleep

duration as well was associated with BMI in both Models 4 and 5. However, while in Model 4 a significantly increasing BMI trajectory had been seen in short sleepers, compared to children with normal sleep duration, Model 5 showed that children with very long sleeping hours appeared to have a lower BMI overall.

Later lifestyle

Differences in BMI were seen based on lifestyle variables measured in children when they were three and five years old. At the age of five years, a small but significant increase in BMI was seen with every additional course of antibiotics that a child had taken throughout the past 12 months. Additionally, the more unstructured physical activity of the child had been reported by the PCG, the higher the child's BMI was at five years of age. Furthermore, compared to children who did not participate in organised sports when they were five years old, those that joined a sports club twice a month (n=169) had a lower BMI overall, whereas no association was seen between more frequent use of sports club and child BMI. The largest deviation in BMI among lifestyle factors was seen based on screen use. Compared to children who were reported to use screens between one and less than two hours per day, those children who used screens never or less than an hour had significantly lower average BMI scores when they were five years old (β = -0.29 ; 95% CI: -0.45 ; -0.13). No association was seen between the number of food pyramid recommendations met by the children and their BMI.

Sensitivity analysis

Sensitivity analysis did not change the overall findings (results not shown). However, a few minor differences were noted compared to the original model, most likely due to smaller sample sizes in variables. Overall, urban region (Model 5b), neighbourhood availability of play and green spaces (Models 5b-d), childcare (Models 5b-d) and onset of solid food in the child's diet (Models 5c and 5d) were no longer significant in the sensitivity analysis while relationships had been seen in

the original Model 5. Furthermore, sex (Model 5c), household type (Model 5c), non-Irish white ethnic background (Model 5c), and caregiver education (Model 5b) were no longer significant at the age of three years. At five years, living in a household with one caregiver and no siblings (Model 5c), lower secondary education or less (Models 5b-d) and higher secondary education (Models 5b and 5c) were no longer significantly associated with BMI.

At three years of age, furthermore, Chinese children had a significantly higher BMI in Models 5b (β = 0.93, 95% CI: 0.01 ; 1.85) and 5c (β = 0.97, 95% CI: 0.06 ; 1.88), but therefore this association disappeared in both models at five years of age, at which point it had been seen in the original Model 5. Lastly, in Model 5b, children who spent more than two hours per week in a sports club had a higher BMI overall than those who never did (β = 0.13, 95% CI: 0.02 ; 0.24), and those children using screens three or more hours per day at the age of five years had a higher BMI compared to those who used screens between one and less than two hours (β = 0.13, 95% CI: 0.01 ; 0.25).

Table 27: Multilevel Model 5, showing Body Mass Index (BMI) trajectories between three years and five years of age adjusted for BMI at nine months, demographics, birth and infancy factors, parental characteristics, neighbourhood variables and early and later lifestyle. Point estimates and 95% Confidence Intervals (CI) are shown.

	Main effects	**Interaction terms with time [b]**
Model components	**BMI ß-coefficient (95% CI) at age 3 years**	**BMI ß-coefficient (95% CI) at age 5 years**
Number (observations)	15,422	
Number (individuals)	8,085	
Constant	**16.62** (16.49 ; 16.75)***	
Log-pseudolikelihood	-23904.03	
Wald Chi-Square	5163.95	
Time (3 years) [a]		
5 years	N.A.	**-1.06** (-1.15 ; -0.96)***
BMI at the age of nine months		
Continuous score	**0.39** (0.37 ; 0.41)***	**-0.05** (-0.07 ; -0.03)***
Sex (boys)[a]		
Girls	**-0.09** (-0.15 ; -0.03)**	**0.23** (0.17 ; 0.29)***
Ethnic background (Irish white)[a]		
Any other white background	**-0.20** (-0.34 ; -0.06)**	0.10 (-0.04 ; 0.25)
African	0.07(-0.21 ; 0.36)	**0.37** (0.12 ; 0.63)**
Any other Black background	-0.12 (-0.59 ; 0.35)	0.03 (-0.46 ; 0.53)
Chinese	0.81 (-0.14 ; 1.75)	**1.01** (0.06 ; 1.95)*
Any other Asian background	0.13 (-0.20 ; 0.46)	**0.49** (0.15 ; 0.83)**
Other, including mixed background	**-0.11** (-0.20 ; -0.02)*	0.03 (-0.06 ; 0.12)
No secondary caregiver data	0.01 (-0.11 ; 0.12)	-0.01 (-0.13 ; 0.11)
Equivalised household income (highest quintile)[a]		
Lowest quintile	0.03 (-0.05 ; 0.12)	N.A.
2nd quintile	0.03 (-0.05 ; 0.11)	N.A.
3rd quintile	0.03 (-0.04 ; 0.09)	N.A.
4th quintile	0.02 (-0.04 ; 0.09)	N.A.
Missing	0.05 (-0.05 ; 0.15)	N.A.
Highest education of caregivers (third level) [a]		
Lower secondary education or less	**0.19** (0.03 ; 0.35)*	**0.21** (0.02 ; 0.40)*
Higher secondary education	-0.02 (-0.12 ; 0.09)	**0.14** (0.02 ; 0.26)*
Post-secondary education	0.01 (-0.05 ; 0.08)	**0.09** (0.03 ; 0.16)**

	Main effects	Interaction terms with time [b]
Model components	**BMI ß-coefficient (95% CI) at age 3 years**	**BMI ß-coefficient (95% CI) at age 5 years**
Region (rural)[a]		
Urban	**-0.06** (-0.11 ; -0.01)*	N.A.
Household structure (two caregivers, two or more children)[a]		
Lone caregiver, one child	**0.15** (0.01 ; 0.30)*	**0.19** (0.01 ; 0.36)*
Lone caregiver, two or more children	0.08 (-0.06 ; 0.21)	**0.19** (0.03 ; 0.35)*
Two caregivers, one child	0.01 (-0.07 ; 0.08)	**0.14** (0.03 ; 0.25)*
Neighbourhood perceived as safe (agree) [a]		
Strongly agree	-0.01 (-0.06 ; 0.04)	N.A.
Disagree	-0.01 (-0.07 ; 0.05)	N.A.
Strongly disagree	0.10 (-0.04 ; 0.24)	N.A.
Neighbourhood perceived to offer green and play spaces (agree) [a]		
Strongly agree	0.01 (-0.04 ; 0.06)	N.A.
Disagree	**0.06** (0.01 ; 0.11)*	N.A.
Strongly disagree	0.02 (-0.05 ; 0.09)	N.A.
Gestational age at birth (on time)[a]		
Very early	**-0.60** (-0.87 ; -0.33)***	N.A.
Somewhat early	**-0.25** (-0.39 ; -0.12)***	N.A.
Late	**0.10** (0.01 ; 0.19)*	N.A.
Birth-weight-for-gestational-age (appropriate)[a]		
Small-for-gestational age	**-0.32** (-0.41 ; -0.23)***	N.A.
Large-for-gestational-age	**0.36** (0.27 ; 0.44)***	N.A.
Missing	0.07 (-0.19 ; 0.32)	N.A.
Breastfeeding duration (not breastfed)[a]		
Less than three months	-0.05 (-0.12 ; 0.02)	N.A.
Three to under six months	-0.01 (-0.10 ; 0.08)	N.A.
Six months or more	0.01 (-0.07 ; 0.10)	N.A.
Introduction of solid food (between age four and under six months)[a]		
Before age four months	-0.03 (-0.11 ; 0.05)	N.A.
After age six months or later	**-0.07** (-0.13 ; -0.01)*	N.A.
Rapid infant weight gain (no rapid weight gain)[a]		
Rapid weight gain	**0.28** (0.20 ; 0.36)***	N.A.

	Main effects	Interaction terms with time [b]
Model components	**BMI ß-coefficient (95% CI) at age 3 years**	**BMI ß-coefficient (95% CI) at age 5 years**
Missing	omitted	N.A.
BMI status of the primary caregiver (normal/ underweight)[a]		
Overweight	0.04 (-0.02 ; 0.10)	**0.16** (0.09 ; 0.23)***
Obese	**0.21** (0.13 ; 0.30)***	**0.21** (0.11; 0.31)***
Missing	**0.21** (0.08 ; 0.34)**	0.10 (-0.06 ; 0.26)
BMI status of the secondary caregiver (normal/ underweight)[a]		
Overweight	**0.08** (0.01 ; 0.15)*	**0.14** (0.06 ; 0.23)**
Obese	**0.21** (0.12 ; 0.30)***	**0.30** (0.19 ; 0.40)***
No secondary caregiver interview	0.04 (-0.08 ; 0.15)	**0.18** (0.04 ; 0.31)*
Missing	0.13 (-0.01 ; 0.27)	0.08 (-0.09 ; 0.25)
Primary caregiver stress		
Continuous score	0.01 (-0.01 ; 0.01)	N.A.
Childcare (no childcare arrangement)[a]		
Informal childcare	**0.06** (<0.01 ; 0.11)*	N.A.
Formal childcare	0.02 (-0.03 ; 0.08)	N.A.
Daily sleep duration (short sleep)[a]		
Normal sleep	0.02 (-0.03 ; 0.08)	N.A.
Long sleep	**-0.08** (-0.13 ; -0.02)**	N.A.
Food recommendations met	0.01 (-0.02 ; 0.04)	N.A.
Antibiotic doses in the past 12 months	0.01 (-0.01 ; 0.02)	**0.03** (0.01 ; 0.05)*
Frequency of unstructured physical activity	-0.01 (-0.03 ; 0.01)	**0.04** (0.01 ; 0.06)**
Sports club use (never)[a]		
Twice a month	**-0.23** (-0.41 ; -0.06)**	N.A.
One hour per week	0.04 (-0.02 ; 0.10)	N.A.
Two hours per week	0.07 (-0.01 ; 0.16)	N.A.
More than two hours per week	0.09 (-0.02 ; 0.19)	N.A.
Missing	0.14 (-0.72 ; 1.00)	N.A.
Daily screen time (one to less than two hours)[a]		
None to less than one hour	-0.01 (-0.08 ; 0.06)	**-0.29** (-0.45 ; -0.13)***
Two to less than three hours	0.01 (-0.05 ; 0.07)	0.06 (-0.03 ; 0.15)
Three hours or more	0.06 (-0.02 ; 0.14)	0.09 (-0.02 ; 0.21)
Random effect components		
Residual SD	0.22 (0.13 ; 0.38)	

	Main effects	Interaction terms with time [b]
Model components	**BMI ß-coefficient (95% CI) at age 3 years**	**BMI ß-coefficient (95% CI) at age 5 years**
Measurement (time) 3 Random intercept SD Intraclass correlation coefficient id	0.97 (0.76 ; 1.23) 1.22 (1.09 ; 1.38) 0.64 (0.62 ; 0.66)	

a = reference group

b = β-coefficients in the columns under 'Interaction terms with time' display the change in BMI differences between groups (categorical variables) or linear changes with increasing score (continuous variable) from nine months to three years and from nine months to five years of age, respectively

N.A. = not applicable, no interaction with time was included in the analysis

SD = standard deviation

* $p<0.05$

** $p<0.01$

*** $p<0.001$

6.4.6 Cumulative changes in body mass index

Similar to findings in Chapter 5, there was a high chance that several material, psychosocial and biological risk factors were present in some children. Accordingly, a number of risk and protective factors were grouped together and BMI changes in these groups across the ages are shown in Figure 27. Specifically, children who lived in rural regions, whose families were in the lowest income quintile and whose caregivers had less then third level education (n=768) were grouped together, based on their high material risk of OWOB seen in Chapter 5 (Section 5.5). At the age of three years, their BMI was above the1-SD thresholds of the WHO growth standard for boys and girls, which were used to classify OWOB in this thesis (details were presented in Chapter 4). At nine months and five years of age, the BMI of children with a high material risk was (almost) below all respective OWOB thresholds. However, their BMI was higher than in children with material protective factors, namely children who lived in urban regions, were in the highest income quintile and where at least one caregiver had third level education. Children in the latter group (n=712) had particularly low BMIs at the age of five years, compared to OWOB thresholds (Figure 27).

Figure 27 also shows the average BMI of children with a high psychosocial risk, namely those in lone caregiver households who attended informal childcare (n=293). Their average BMI was similar to that of children with a high material risk at all ages. Similarly, the BMI changes of children with a low psychosocial risk (i.e. children with two caregivers in formal childcare, n=877) were comparable to those of children with a low material risk (Figure 27).

Additionally, children were grouped as having a high biological risk if they were born LGA and slept little (n=208). While their average BMI was comparable to that of children with a higher material or psychosocial risk at the age of nine months, it was much higher at the ages of three and five years. Moreover, it deviated much from the BMI trajectory of children with a low biological risk (i.e. children born AGA who slept much, n=1,521). This deviation increased over time. Furthermore, both at the ages of three and five years, the average BMI of children in the high

biological risk group was higher than the OWOB cut-offs, whereas children in the low-risk group were constantly below them (Figure 27).

Overall, children in material, psychosocial and biological high- and low-risk groups appear to have similar BMIs at the age of nine months, after which age deviations between children exposed to several risk factors or exposed to several protective factors of OWOB appear to increase (Figure 27).

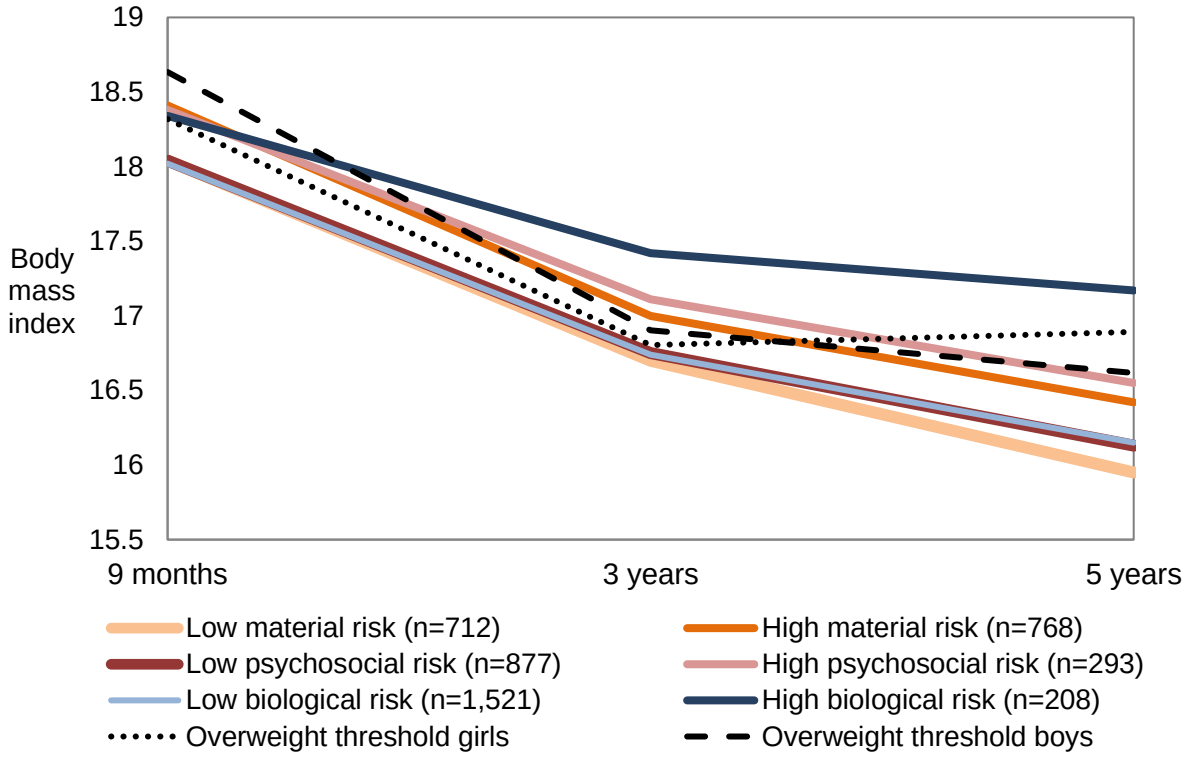

Figure 27: Body mass index of children with a high or low cumulative biological, psychosocial or material risk of overweight and obesity

Note:

Low material risk = urban region + highest income quintile + third level education

High material risk = rural region + lowest income quintile + below third level education

Low psychosocial risk = Two-caregiver household +formal childcare

High psychosocial risk = Lone-caregiver household + informal childcare

Low biological risk = appropriate-for-gestational-age + long sleep

High biological risk = large-for-gestational-age + short sleep

Overweight thresholds are based on the 1-standard deviation threshold of the World Health Organization growth standard (9 months and 3 years) and growth reference (5 years).

To integrate the findings of this and the previous chapter, Table 28 provides an overview of factors that were investigated in this thesis and their relationships with OWOB, with obesity and with BMI at the ages of nine months, three years and five years. Both chapters support the material, psychosocial and biological theories that were presented in Chapter 2 (Section 2.4.5) to explain inequalities in the OWOB prevalence among young children in Ireland, as can be seen in Table 28. Furthermore, there was high agreement between the results in Chapters 5 and 6 (Table 28).

Table 28: Summary of relationships between various overweight and obesity (OWOB) risk factors and 1) OWOB, 2) obesity and 3) body mass index (BMI). Relationships at nine months (9 m), three years (3 y) and five years (5 y) of age are based on analysis undertaken in Chapters 5 and 5 of this thesis.

Level of the SEM	Population characteristics and risk factors of childhood OWOB	Pathway	OWOB			Obesity			BMI		
			9 m	3 y	5 y	9 m	3 y	5 y	9 m	3 y	5 y
Individual level	Sex	Biological	)	)	-	-	-	-	)	)	( ))
	Diet	Biological	N/A	-	-	N/A	-	-	N/A	-	-
	Physical activity	Biological	N/A	-	-	N/A	-	f	N/A	-	( ))
	Daily screen time	Biological	N/A	-	-	N/A	)	)	N/A	-	)
	Antibiotic use	Biological	N/A	-	-	N/A	-	)	N/A	-	)
	Daily sleep duration	Biological	-	)	)	-	-	)	)	)	)
Parenting styles & family characteristics	Primary caregiver weight status	Biological	)	)	)	-	)	)	-	)	)
	Secondary caregiver weight status	Biological	-	)	)	-	)	)	-	)	)
	Gestational age	Biological	)	)	)	)	)	-	)	)	)
	Birth-weight-for-gestational-age	Biological	)	)	)	)	)	)	)	)	)
	Rapid infant weight gain	Biological	)	)	)	)	)	)	)	)	)
	Breastfeeding initiation and duration	Biological	-	-	-	-	-	)	( ))	-	-
	Timing of introduction to solid foods	Biological	-	-	)	-	-	-	-	( ))	( ))
	Household structure	Psychosocial	-	)	)	( ))	-	-	-	)	)
	Primary caregiver stress	Psychosocial	-	-	-	-	-	-	-	-	-

Level of the SEM	Population characteristics and risk factors of childhood OWOB	Pathway	OWOB			Obesity			BMI		
			9 m	3 y	5 y	9 m	3 y	5 y	9 m	3 y	5 y
Community, demographic & societal characteristics	Household class	Material	)	-	-	)	-	-	-	-	-
	Equivalised household income	Material	-	-	)	-	-	-	)	)	)
	Highest education of caregivers	Material	-	-	)	-	-	-	-	)	)
	Ethnic background of caregiver(s)	Biological	)	)	)	)	)	)	)	)	)
	Region	Material	-	-	)	-	-	-	)	)	)
	Neighbourhood perceived as safe	Material	-	-	-	-	-	)	-	-	-
	Neighbourhood perceived to offer green and play spaces	Material	-	-	)	-	-	-	-	( ))	( ))
	Childcare arrangements	Psychosocial	)	)	-	-	-	-	)	)	)

N/A = This variable was not available at the respective age

SEM = Social Ecological Model

) An association was seen in this thesis, as discussed in Chapters 5 and 6

( )) Only an inconclusive association was seen

6.5 Summary

In this longitudinal study, changes in BMI in relation to OWOB risk factors were investigated that spanned across the first five years of children's lives in Ireland. As children gained in height over time, mean BMI decreased from 18 kg/m^2 to 17 kg/m^2 and 16 kg/m^2 from nine months to three and five years of age, respectively. In all models, BMI decreased over time and appeared to depend on random deviations emerging between children (45% in Model 4 to 64% in Model 5) and on fixed effects introduced by a number of explanatory variables ($p < 0.001$).

Having investigated several obesity risk factors in this cohort, factors occurring at birth or during the infancy period had the largest impacts on BMI measured at nine months, three years and five years of age. Biological pathways to OWOB that influence children's weight gains through risk factors on the parental level thus appear to exist at a very early age and associations persist throughout the early childhood years. Differences in BMI from the age of three years additionally were seen based on ethnicity and the weight status of the caregiver. Additionally, smaller variations in BMI existed based on other risk factors on various levels of the SEM. Slight variations between the BMI of girls and boys were seen which appear normal according to growth criteria (395). Only small associations existed between children's lifestyles, the most influential being screen use after the age of nine months. No or inconclusive longitudinal associations were seen between BMI and dietary intake, as well as between BMI and physical activity.

Longitudinal BMI changes also differed based on socio-demographic group membership from the age of three years onward, indicating that material factors contribute to the development of OWOB later than biological factors. Similarly, associations observed between family structure and BMI changes from the age of three years furthermore confirm that psychosocial risk factors evolve between the ages of nine months and three years.

As expected, a high number of risk factors of OWOB or obesity identified in Chapter 5 were also related to differences in BMI changes. In particular, relationships seen between explanatory variables and OWOB in Chapter 5 were similar to those seen with BMI in this chapter, indicating that studies of early

childhood obesity should apply the threshold of 1 SD to classify OWOB, using the WHO child growth standard. The following chapter will discuss these findings more in relation to the evidence retrieved through other studies, and by integrating these findings with those from the two previous results chapters.

CHAPTER 7:

Discussion

7.1 Introduction

This thesis used longitudinal cohort data from infants and young children up to the age of five years in Ireland to investigate OWOB levels and changes in BMI over time, in relation to risk and protective factors of childhood obesity. The findings of this study addressed a number of research gaps that were identified in relation to OWOB in young children in Ireland and adds to the current body of knowledge in the wider field of obesity research.

In Chapter 1 of this thesis, the issue of OWOB and the implications of its rising prevalence on health worldwide was outlined. In particular, children are increasingly affected by the consequences of OWOB, which are likely to affect children's health and well-being throughout life. Chapters 1 and 2 specifically shed light on the youngest children up to the age of five years, who proportionally appear understudied in many countries, including Ireland. Chapter 2 furthermore presented the conceptual framework that guided the research in this thesis, based on the SEM, Monasta et al. (1) and the current state of obesity.

The nationally representative GUI infant cohort was presented in Chapter 3, due to the suitability of this dataset to study OWOB longitudinally and fill the research gaps identified in Ireland. The methodology used by the GUI study team as well as details of the secondary analysis conducted in this study were described, which includes the choice of variables and quantitative analysis techniques that were employed in this thesis.

In Chapter 4, a number of child growth criteria were critically assessed and the WHO growth standard, WHO growth reference and IOTF growth reference were applied to the GUI infant cohort data to assess the prevalence of weight categories over time, in the entire cohort, and stratified by sex. There were large differences in the estimated prevalence of OWOB, depending on the criteria used. Overall, the prevalence of OWOB appeared high, but declined with increasing age. This was discussed in detail in Chapter 4 (Section 4.4.3), and therefore only a synopsis of findings as well as their implications for the field of childhood obesity will be presented in the course of this discussion chapter.

In Chapter 5, risk factors for young children in relation to OWOB or obesity were analysed cross-sectionally, based on the SEM and the *Complex web of potential risk factors for childhood obesity* using the GUI data. This was to test biological, material and psychosocial pathways of childhood obesity.

Following this (Chapter 6), the BMI of children in the GUI infant cohort was studied longitudinally, using multilevel modelling, to account for changes in children's BMI over time while adjusting for important obesity risk factors to further test these theories.

A number of potential risk factors associated with OWOB, obesity or BMI in young children in Ireland were identified in Chapters 5 and 6. Strong associations were seen based on factors that occurred at birth and in the infantile period and support that biological pathways explain a high proportion of OWOB in young children in Ireland. As children grow older, socio-economic grouping, parental characteristics and to some extent children's lifestyles play an increasing role in children's weight-related outcomes, suggesting that material and psychosocial pathways are increasingly more important for children's healthy weight development after infancy, while biological associations prevail. These findings will be discussed in more detail in this chapter. Importantly, the distribution of children in population groups that were at an increased risk of developing OWOB implies that children may likely belong to more than one high-risk group at the same time, hence increasing their risk of OWOB beyond the individual odds seen for single variables.

In this chapter, the results of previous chapters will be discussed in depth, followed by recommendations that can help guide policy-makers and researchers with the aim of lowering the prevalence and associated adverse consequences of childhood OWOB in Ireland. The discussion will take into account findings from previous studies, as well as evidence-based interventions that effectively lowered OWOB or the BMI of young children in prior research. Additionally, the Irish policy-context will be examined.

7.2 Summary and discussion of findings

7.2.1 Prevalence of overweight and obesity in young children in Ireland

In the GUI infant cohort, huge discrepancies were found between the prevalence of overweight and of obesity depending on which child growth criteria were used. As children increased in height over time, mean BMI decreased from 18 kg/m^2 at nine months to 17 kg/m^2 and 16 kg/m^2 to three and five years of age, respectively. This is higher than the corresponding mean BMIs on WHO growth charts (395). To enable better comparability of prevalence estimates across the ages, the classification system of the WHO growth reference was used at all ages studied, in addition to the WHO growth standard (all ages) and the IOTF growth reference at three and five years of age. At nine months of age, 9% of children were overweight using the WHO growth standard and 29% using the WHO growth reference. Furthermore, the prevalence of overweight ranged from 11% to 32% at three years and from 6% to 24% at five years of age based on the respective criterion. Additionally, 2% of children were classified as obese at nine months using the WHO growth standard and 9% using the WHO growth reference classification. Obesity ranged from 3 to 11% at three years and 2 to 6% at five years of age, respectively.

While the WHO growth standard and WHO growth reference appeared most useful to study the OWOB prevalence in the GUI infant cohort (165, 336, 337), estimates using the IOTF growth reference in the three- and five-year-old children were provided for comparison with other research (239, 338, 382). Prevalence of overweight and of obesity using the IOTF growth reference was higher than the WHO growth standard estimates, but lower than the WHO growth reference estimates. Additionally, the prevalence of OWOB and obesity appeared more stable from three to five years of age when the IOTF growth reference was used. These findings match results in other studies (239, 338).

Accounting for the differences between various criteria, the prevalence of OWOB in this thesis was below the levels that were expected in Irish two- to four-year-old children by a global database (5), whereas it confirms findings from the smaller National Preschool Nutrition Survey that was conducted in young Irish children

around the same time the GUI infant cohort was collected (98). As up to 46% of three-year-olds in the nationally representative GUI cohort were overweight or obese, prevention efforts are clearly needed before children reach school age, in order to lower the prevalence of childhood OWOB in Ireland over time.

Changes in prevalence over time

For OWOB combined, prevalence was highest at the age of three years and declined thereafter, irrespective of the child growth criterion that was applied to the GUI data. However, using the IOTF growth reference, only a decline in overweight (but not obesity) was seen between three and five years of age, whereas both the overweight and the obesity prevalence declined if either the WHO growth reference classification or growth standard classification were used consistently across these ages. This time trend is consistent with contemporaneous findings from the smaller National Preschool Nutrition Survey (98).

No older data from young Irish children are available for comparison that may indicate whether this trend constitutes a time trend in prevalence or an age-typical pattern. In many other countries, based on large cross-sectional and longitudinal studies, peak OWOB prevalence occurred at an earlier or later age within the early childhood years (79, 337, 382), suggesting that variability across the ages is normal but not following a clear pattern. The low rate of breastfeeding women in Ireland was considered a possible reason for the early peak in OWOB prevalence; however only marginal differences were seen between the BMI distribution in children breastfed for six months or longer and the distribution in the entire cohort.

As a result, the fact that fewer children in the GUI infant cohort were overweight or obese at five years of age, compared to previous years, possibly supports studies which find that OWOB levels in Ireland have stabilised or may in fact be decreasing (38). Additionally, findings from Round 4 of the COSI imply that overweight levels may have further declined in Ireland two years after the GUI data were collected, whereas, similar to findings in this thesis, no change in obesity was seen in the COSI (using the IOTF growth reference) (356). Children included in Round 4 of the

COSI were born only a year after the GUI infant cohort (41), so both studies are comparable. Jointly, the findings of this thesis and the COSI contradict projections that OWOB among the youngest children in Ireland may increase until 2030 (5, 7). It is possible that the adoption of the *HSE Framework for Action on Obesity 2008-2012* (100) and the implementation of child-specific strategies outlined in this Framework (100) may have contributed to a declining trend in OWOB during the GUI study period. These strategies included a number of national health promotion and awareness campaigns as well as the regulation of food marketing to children (100) and educational initiatives in preschools (396) that may have influenced the lifestyles of families and children's weight gains post 2011. Future follow-up of the GUI infant cohort and younger children is warranted to confirm a potential decline in childhood OWOB in Ireland and, if applicable, examine the causes.

At three and five years of age, the strongest association was seen for preceding weight status and childhood OWOB and obesity in the cross-sectional analysis, adjusted for a large number of risk factors. In the longitudinal analysis, 45% of the variation in BMI was explained by the correlation of all BMI measurements within the same children, confirming that the tracking of BMI in this cohort was common. Compared to under- or normal weight children, those who were overweight at nine months or three years of age were more likely to have OWOB or obesity in the subsequent measurement, and those who were obese had the highest risk of subsequent OWOB or obesity. Specifically, obese nine-month-olds were more than eight times as likely to have OWOB and seven times as likely to be obese at three years of age, compared to under- or normal weight children. Notably, obese three-year-olds were nearly 42 and 39 times as likely to have OWOB or obesity, respectively, when they were five years old. Between three and five years, a larger proportion of the variation in BMI (64%) than from nine months to five years was explained by the correlation of BMI measurements within children. This suggests that while persistence of weight status from nine months to three years is high, proportionally many more overweight or obese children appear to return to normal weight by the age of three years than between the ages of three and five years.

Frequent changes in BMI also were seen in other studies until children turned two to three years old, after which age BMI or weight status largely determined

subsequent BMI and weight status (28, 77, 397-399) and led to decreases in incidence of OWOB over time (32). While the prevalence of OWOB and obesity in the GUI infant cohort was higher at the ages of nine months and three years than at five years, the plasticity of children's BMI appears highest in the youngest ages. Accordingly, healthy weight promotion may be most effectively introduced to families before children reach the age of three years. However, at the age of four to six months, children with a high BMI were already at an elevated risk of severe obesity in mid-childhood in two other cohorts (400), suggesting that prevention should start as early as possible. Despite decreases in OWOB prevalence seen in the GUI infant cohort, mean BMI still corresponded to the sex- and age-specific 75th BMI percentile on WHO growth charts (395) at the end of this study, indicating that the average BMI in Ireland is higher than what would be expected in a healthy population of zero- to five-year-old children. Hence, the early plasticity seen in this thesis should not be understood as a mechanism by which heavy infants will automatically grow out of their excess weight states, but high BMIs or weight gains before the age of three years should be detected and intervened with early to reduce its persistence in this modifiable yet susceptible age group.

Sex differences in body mass index and weight status

In the longitudinal analysis of BMI, adjusted for various explanatory variables, girls were below the BMI trajectory of boys from nine months to three years, before they crossed the trajectory of boys and reached a similar BMI at five years of age. A trend towards lower BMI in girls appears normal at the ages of nine months and three years, according to the WHO growth standard, and differences would indeed be expected to disappear, or even reverse, by the age of five years (395). This was also previously seen in BMI trajectories of Greek children (382).

In line with other published research (239, 380, 401), the WHO growth standard and WHO growth reference classification identified a higher number of boys than girls as overweight and as obese across the ages (not statistically significant in the obesity category at nine months and three years using the WHO growth standard),

while the IOTF growth reference identified a significantly higher number of girls than boys as overweight at five years.

Notably, when the WHO growth reference classification was used to define weight status in the cross-sectional analysis (adjusted for explanatory variables), girls were more likely than boys to have OWOB at the age of nine months, whereas this trend was reversed at three years and no sex difference in weight status could be observed when the children were five years old. Similar differences were seen in previous research (382) (Section 4.5). Additionally, the unadjusted differences in BMI trajectories seen between boys and girls over time became smaller as more variables were included in the multilevel models, but remained significant in all analyses, suggesting that these variables do not fully explain sex trends. A study by Li et al. (402) found that boys were more likely to follow BMI trajectories predisposing them to onset of overweight between two and twelve years of age, whereas other researchers did not observe this trend (403). Overall, it appears difficult to identify clear sex trends in children until the age of five years in Ireland and a longer study period may be needed to confirm the findings by Li et al. (402) in the Irish context. However, the contradicting findings in this thesis and other studies stress that correct indication of child growth criteria is of utmost importance when OWOB levels among young children are reported, as the choice of criterion is likely to affect observed sex trends. Furthermore, young children may benefit more from interventions targeted at common OWOB risk factors on a population level, than from sex-specific strategies. The various risk factors investigated in the cross-sectional and longitudinal analysis of this thesis will be discussed from here.

7.2.2 The obesogenic environment of young children in Ireland

Role of birth and infancy factors on changes in body mass index

Factors occurring during birth and infancy had the largest impact on BMI changes in the GUI infant cohort. Firstly, during all measurements the BMI of children who were born very early, early or SGA all remained much below that of term children or those born AGA, respectively, despite catch-up growth. Secondly, one in eight

children were born LGA and persisted on a high BMI trajectory until the age of five years, despite catch-down growth. Children with rapid weight gain from birth to nine months of age (25% of children) had the highest BMI throughout the study period, even though their weight gain decelerated between the age of nine months to five years. Although the effect sizes of these associations became smaller when additional variables were introduced to the models, they still remained in three- and five-year-old children even when adjusting for their nine-month BMI. Previous studies confirm this trend in children born with a small birth weight or prematurely (403-405) and in those born LGA (402, 403, 406). A longitudinal investigation of weight and fat mass trajectories by Koontz et al. (407) showed that rapid weight gain in the first eight months of life resulted in a five times increased risk of developing childhood obesity in later life, which is reflected in the trends observed in this thesis. Children in the GUI infant cohort were only 2.1 times as likely to be obese at the age of three years and 1.4 times at five years. The differences in coefficients are likely due to the small sample size in the study by Koontz et al. (407); however the direction of their findings was confirmed in this thesis. Overall, the cross-sectional analysis in this thesis showed that associations both with OWOB and obesity existed with each of these factors at all ages, confirming that these risk factors occurring during birth and infancy lead to meaningful differences in BMI between young children and should be targeted through prenatal intervention.

It remains to be seen whether children in this cohort maintain their high and low BMIs when they grow older, or whether they catch up and down fully, thus meeting or exceeding the BMI trajectory of term children born AGA and without rapid infant weight gain. Higher BMI trajectories previously observed in children with rapid weight gain (168) and those born LGA (28, 406) tracked into later childhood and early adulthood in various birth cohort studies (28, 168, 309, 406). A study by Taal et al. (159) furthermore suggests that children in different BWFGA groups may be split into those who reach population mean BMI in early childhood, and those who fail to do so. For example, this may differ depending on maternal gestational behaviours and characteristics (408). Importantly, these different growth patterns were associated with variations in cardio-metabolic risk during mid-childhood (398). It may therefore be relevant to investigate sub-groups of various gestational ages,

BWFGA and rapid infant weight gain in future studies to see whether separate BMI trajectories exist within these groups and identify factors that lead children to catch up or down to the age- and sex-specific norm.

Additionally, children who started consuming solid food after they were six months old (30% of the cohort), compared to those children who started between the ages of four to six months, had a slightly lower BMI at the ages of three and five years when the analysis was adjusted for various lifestyle variables. Furthermore, the odds of OWOB, but not obesity, increased significantly at the age of five years in children who were weaned before the age of four months. Also in other studies, early weaning increased the OWOB risk in children aged three to eleven years (176), but not before that age (184). However, while previous studies saw higher OWOB levels in children weaned before the age of four months, they did not see the protective effect of weaning after six months or later which was observed in this thesis (176). Even though the effect size of later weaning on children's BMI appeared small in this thesis, it implies that between the various recommendations on what constitutes the ideal timing (177), a later introduction of solid food after six months should be favoured.

This ties in with recommendations suggesting that children should be breastfed six months or longer, which were partly confirmed in this study. Children who were breastfed for six months or longer (19%), compared to those who were never breastfed, had a slightly lower BMI at nine months and did not fully catch up to the BMI trajectories of non-breastfed children by their age of five years. While the difference in BMI between these two groups was marginal at the age of five years, in the cross-sectional analysis the risk of obesity, but not of OWOB, was 43% lower (OR: 0.57; SD: 0.39-0.85) in five-year-old children breastfed six months or longer. The mixed findings on a potential association between breastfeeding and child weight in this study reflect outcomes of previous research. A protective effect of breastfeeding on early BMI trajectories has been shown in various studies, but findings differ as to how long-lasting this effect is (153, 402, 405, 409) and the possibility of confounding has not been fully ruled out (167, 173). A protective effect of breastfeeding on the obesity risk was also seen in older children in Ireland (92). Therefore, as the association between infant feeding and children's

subsequent BMI or OWOB was inconsistent across the analyses carried out in this study, longer breastfeeding duration may protect some children from moving into the highest weight category while this association may not prevail in others.

It is possible that latent patterns may exist between breastfeeding, weaning and other variables that might explain why only weak relationships were seen in this study. In previous studies, breastfeeding partly explained SES differences in children's BMI trajectories (78) and lowered the obesity risk particularly in high-risk children that had been exposed to GDM (166, 409). The gestational period and SES of families temporally precede the infancy period and associations with infant feeding are possibly smaller in magnitude, because the models include potential mediating factors (e.g. education). The residual association between infant feeding and BMI indicates what is not explained by the other variables. Additionally, no information was collected on the composition of infant foods in this study. A high proportion of children in the GUI study received infant formula, and variations in its protein content may explain that no consistent association with BMI was observed (184, 410). Moreover, breastfeeding was found beneficial for children's gut microbiome previously (411), effects of which may be lowered after antibiotic treatment (198). In fact, antibiotic intake was associated with an elevated BMI in this study, as will be discussed later together with other lifestyle factors.

These findings taken together support the theory that biological links between early childhood obesity and various risk factors exist, such as through microbiotal, hormonal and epigenetic mechanisms (119, 146, 161) as well as appetite and satiety regulation (120, 135). Even if these mechanisms may not be associated with weight gain in all children, the promotion of healthy foetal and infantile weight gain along with appropriate infant feeding practices may contribute to lowering the obesity risk of many children through prevention efforts pursued in the year preceding and subsequent to birth, when many risk factors are amenable (119).

Associations between the weight status of children and their caregivers

Major differences in BMI and children's weight status were seen based on the caregivers' weight status. A higher OWOB risk was seen across children's ages if the PCG was obese. Differences in BMI and in the obesity risk of children only emerged after the age of nine months, when children with overweight or obese PCGs or SCGs continuously gained more weight relatively to other children. If their PCG or SCG was obese, children's BMI at five years was highest and children were twice as likely to be overweight than children with an under- or normal weight PCG or SCG, and 2.4 (PCG) to 2.7 (SCG) times as likely to be obese (at the age of five years, based on the cross-sectional analysis). This supports previous studies that strongly linked a high parental weight status or BMI to children's increasing BMI trajectories, with an aggravating influence as the children grow older (153, 402, 405) that leads to an elevated obesity risk in children's adult life (20). Also in this thesis, BMI deviations between children with normal or underweight and those with obese caregivers increased over time, suggesting that children of overweight or obese caregivers may stay on their upward trajectories and have a high risk to be obese in their later childhood years. Notably, in Ireland, this affects proportionally many children, namely 47% (PCG) and 70% (SCG) of five-year-olds whose caregivers' BMI was available for this study, which is why this risk factor should be a major target for childhood obesity.

Prevention programmes that target the weight status and weight-related lifestyle of young adults (including parents and expectant couples) early may have long-lasting benefits for a healthy weight gain in a large proportion of children. As parental lifestyles correlate with those of their young children (221), prevention programmes should target their shared environment and behaviours. This may benefit both the weight status of parents and children (157, 158).

Changes in body mass index differ in ethnic background groups

Variations in BMI over time were also observed in different ethnic groups. Children from Asian and African descent (each constituting 2% of this cohort) were partly

found to gain weight quicker over time, relative to their height, compared to Irish white children. While the small number of children in most ethnic background groups needs to be acknowledged, findings were similar to those in other studies.

The highest relative weight gain was seen in children from Asian (excluding Chinese) descent, who had a particularly low BMI and a very low risk of OWOB at nine months, but crossed the trajectory of Irish white children before the age of five years. The difference between Asian and Irish white children at nine months became smaller when the analysis was adjusted for more explanatory variables, which could be due to the fact that Asian infants tend to be born smaller (412). However, the subsequent steep increase in relative weight gain among children with Asian background remained after the inclusion of additional variables, indicating that birth characteristics did not explain differences after infancy. Specifically, children with Asian background had a 2.4 times higher risk of OWOB and a 4.1 times higher risk of obesity in the fully adjusted models, compared to white Irish children. While it is possible that Asian children may follow different growth patterns than those with European ethnic background, due to their genetic makeup or other biological factors, cultural and environmental risk factors that were not measured in this study should also be considered. Perhaps, interventions should be directed at children with Asian descent to limit their rapid weight gain in the first five years of life and prevent sudden increases of fat mass and the risk of related cardio-metabolic ill-health (30). Children of Chinese ethnicity also had a very high BMI when they were five years old, however the sample size of this group appears too small to allow for significant conclusions. Future studies should sample more Chinese children to examine whether similar growth patterns exist in this group.

Children with an African background had the highest BMI of all ethnic groups when they were five years old in most analyses in this thesis, but only appeared to start gaining excess weight between the ages of three and five years. This is in line with prior research that found the ages of two to four years most relevant in the development of different BMI trajectories based on ethnicity (413), and that black children were more likely than children in other ethnic groups to follow trajectories predisposing them to overweight (402). In the cross-sectional analysis in this

thesis, an elevated risk of OWOB and of obesity was seen among five-year-old African children. In previous studies, African American children reached their peak BMI in infancy (414) and their adiposity rebound (415) earlier than white children, the latter of which is associated with an increased risk of OWOB (178). A possible reason for the BMI differences observed in this thesis may hence be that more African than white children experience an increase in BMI, following on from an early adiposity rebound, before the age of five years (415), which would suggest a biological cause to their OWOB onset. Nevertheless, the difference between the five-year BMI of African and Irish white background children reduced in the fully adjusted longitudinal analysis of this study that included lifestyle factors, indicating that lifestyle behaviours explain a high proportion of the elevated OWOB risk seen in African children. In previous research, elevated BMI trajectories among young African children were explained by lifestyle factors (255, 256, 404). These factors should therefore be targeted through interventions tailored for children with African background. Lifestyle factors did not explain BMI differences in other ethnic groups, suggesting that several parallel mechanisms may lead to these disparities.

Children of non-Irish white ethnicity (8%) had a consistently lower BMI than Irish white children that led their trajectory to lie below the majority of children at all ages, even when other explanatory variables were included in the analysis. While non-Irish white children were less likely to be obese at the age of nine months or to have OWOB at three years, the OWOB risk identified in the cross-sectional models did not differ at the age of five years. Reasons for this difference may be early biological factors, as it was already present at the age of nine months before children start acquiring family lifestyles. Additionally, mothers from other countries (416) seemed to recognise early OWOB more than Irish-born mothers and hence Irish-born mothers may be less likely to seek advice on children's weight management.

A similar trend to BMI changes in non-Irish white children was seen among children with 'other' or mixed ethnic background in some, but not all models. The sample size of non-African black children was too small to identify meaningful BMI differences; however both for mixed background and non-African black children, no clear patterns were expected due to the variety of ethnicities potentially included in

these groups. A review of recent literature suggests that children from white and non-white backgrounds in Western countries follow different BMI trajectories (417), which was partly confirmed in this thesis. Generally, young white children appear to have relatively lower weight gain (399, 404, 406); however the comparability of those studies with this thesis is limited as their study populations vary. Hence, this thesis contributes to identifying specific population groups that may require tailored interventions to lower inequalities in OWOB in Ireland.

Inequalities in the obesity risk based on socio-economic status

Significant socio-economic differences were also observed in the BMI trajectories of children, likely contributing to inequalities in childhood obesity in Ireland.

The strongest socio-economic determinant of BMI trajectories in this study appeared to be the highest educational level of caregivers in the household. Compared to families in which at least one caregiver had acquired third level education, other post-secondary education and lower secondary education or less were consistently related to higher BMI trajectories when children were five years old. BMI scores were highest in households with the lowest educational level, and this difference was already visible in children's BMI by the age of three years. In previous studies, high parental education has been associated with lower BMI trajectories (404, 406), but differences in this relationship were found between countries (153). In this thesis, the educational gradient remained when the analysis was adjusted for additional variables, including lifestyle.

Similar to previously reported findings (78), BMI trends in caregiver education in our study only became visible after infancy. A potential reason for this could be latent influences of SES on birth outcomes, the latter of which were strongly associated with BMI in this study, which may hide early associations of BMI with SES. This could not be tested as socio-economic data for this study were collected only after birth. Additionally, Ruijsbroek et al. (241) reported higher adherence to breastfeeding guidelines among higher educated women. However, as one of the associations (with 'lower secondary education or less') disappeared in some of the

sensitivity analyses and was not seen in the cross-sectional analysis, it appears more likely that BMI changes among children in lower educational groups only start emerging from the age of three to five years, but that trajectories at this age have not widened enough to translate into significant differences in the OWOB prevalence. One reason for this may be that children start adopting family lifestyles at which point a high level of parental health knowledge may be required to promote healthy weight gains (227). Additionally, it was noted in another study that a lower level of awareness appears to exist in lower educational groups about what constitutes a healthy weight in three- and five-year-old children (416) and the environments of these families tend to be more obesogenic (242). However, awareness was not assessed in this thesis. As inequalities in the obesity prevalence based on SES appear to increase worldwide (81, 82), early differences in BMI should be taken seriously and sustainable interventions delivered to families. Based on this, children in the age group of three to five years may benefit from educational interventions delivered before this age which take advantage of the early weight-related plasticity identified in this study.

While associations with caregiver education only were seen at the ages of three and five years, children in the lowest income quintile, compared to the highest, followed a slightly elevated, but not aggravating BMI trajectory at all ages. However, this association disappeared when lifestyle variables were integrated in the analysis and in most sensitivity analyses, indicating that families on the lowest income may pursue different lifestyles to others. For example, they may have less access to affordable healthy food (27, 236) and less opportunities to pursue physical activity, as they may not be able to afford a house with a garden or to enrol their children in sports clubs. In turn, this would be expected to lead children to spend more time using screens or pursuing other sedentary behaviours, and families to adopt unhealthy behaviours to cope with potential food and housing insecurity (227, 237). Surprisingly, a lower OWOB risk was seen in the lowest income quintile when children were five years old, which may have occurred because the analysis was adjusted for several other variables. No relationship was seen with either OWOB or obesity and income at any of the other ages, indicating that income may not explain much of the OWOB prevalence in Ireland or that the relationship may be fully explained by differences in lifestyle and living conditions.

Additionally, children in families who could not be grouped into a social class were more likely to have OWOB or obesity at the age of nine months than children in the managerial and technical class. However, this had no long-term implication on their OWOB or obesity risk, neither in the cross-sectional nor in the longitudinal analysis. As previous studies observed a social class gradient in early childhood (413, 418), it is possible that this relationship was fully explained through the inclusion of other variables in the models.

Despite the small association that was partly seen between income and BMI, it should be noted that more children in this cohort lived in families with lower than third level education than in families in the lowest income quintile, suggesting that interventions delivered to parents with lower educational levels may reach out to more children who are at risk of gaining excess weight. Additionally, in a systematic review of SES and adiposity, high parental education was more consistently associated with lower OWOB levels in children aged five years and older, compared to income or occupation as alternative indicators of SES (419). These findings indicate that material pathways likely explain inequalities in the OWOB prevalence between children. To reduce inequalities in OWOB, resources increasing the health knowledge and awareness of OWOB among parents may be needed as much as financial incentives that facilitate behavioural changes.

Obesity risk factors in the psychosocial environment of children

Large variations in BMI were associated with family structure. Single children partly had a slightly lower BMI than children with at least one sibling at the age of nine months. However, at five years of age, children who did not have at least one sibling or who lived in lone-caregiver families followed elevated BMI trajectories, with the highest BMI seen in single children with one caregiver, confirming previous literature (404, 420). These differences translated into a higher OWOB risk among children in lone-caregiver households, suggesting that psychosocial pathways may impact on the risk of early OWOB. When lifestyle variables were introduced to the multilevel model, the differences between children’s BMI based on family structure narrowed down. This suggests that variations in lifestyle between one- and two-

caregiver families as well as between families with one or more children may differ and partly explain why some children put on excess weight, as has been suggested in previous studies (224, 226). For example, bigger families were found to eat more meals together (226) and watch less TV, whereas single children may tend to be more sedentary than children who can play active games with their siblings (223). In turn, lone caregivers are more likely to lack social support in and outside of the household (223, 227) and may have to accommodate their children's needs with a tighter budget and timely schedule than coupled caregivers (223, 227). It is therefore not surprising that lifestyles between families with different household structures differ.

The inclusion of PCG stress in this study retrieved inconclusive results, implying that this may not be a standalone risk factor for high BMI trajectories in Ireland. It needs to be acknowledged that the inclusion of household structure in the analysis may have captured the level of stress that may lead families to adjust their weight-related routines, to cope with the lack of support from a second caregiver, e.g. by compromising on time-consuming and expensive activities. In contrast, other (temporary) sources of stress included in the stress score may not lead families to lifestyle adaptations that might impact on children's weight gains.

By the age of five years, 20% of children in the GUI infant cohort lived in household structures that were associated with an elevated BMI (as detailed above). Hence, differences in household structure are likely to contribute to inequalities in the obesity prevalence in Ireland, which adds to the limited findings in previous studies (27). Particularly families depending on a lone caregiver may require additional support before their children turn three years old, in order to remove these inequalities. Furthermore, single children may benefit from enrolment in childcare institutions that promote active play with other children at this core time of developing lifestyle routines and confidence in physical activity.

The wider obesogenic environment of children outside the family home

More than half of the children in this study lived in rural regions and were following a slightly higher BMI trajectory throughout the ages than children in urban regions. This is in line with other studies that reported similar findings in childhood (421) and until late in adolescence (406). Although the difference between these two groups in the GUI cohort was small, it translated into a higher OWOB risk among children in rural regions at the age of five years. A likely reason for this are larger distances to food outlets, childcare and recreational facilities in rural regions, which require families to rely on their car or public transport more, the latter of which is often underdeveloped in rural areas. Families may accordingly walk less or consume the limited range of (unhealthier) affordable food options that are typically available in small, local shops (85). Consequently, local shops and community services (including childcare) may need to be subsidised to offer families living in rural regions in Ireland the same healthy choices that are available in cities. While the difference in BMI between children in rural and urban regions was small, the impact of this difference on population-level should not be underestimated, due the high proportion of children who live in rural regions.

Additionally, BMI changes were investigated in relation to the degree of safety that the PCG perceived in the neighbourhood, as well as the perceived level of green and play spaces that were available in the neighbourhood. In the various longitudinal models assessing BMI changes as well as in the cross-sectional analysis investigating weight status, only inconclusive results were seen. Lower perceived neighbourhood safety was previously related to OWOB in a cross-sectional Portuguese study (250). However, no relationships between children's BMI trajectories and perceived or objectively measured neighbourhood characteristics other than rural region were seen in a Canadian cohort (421) and a US cohort (251), despite marginally lower levels of physical activity and higher screen time use among children in neighbourhoods perceived as unsafe (251). Additional efforts may therefore be needed to deliver interventions in rural settings in Ireland generally, while a focus on individual neighbourhood characteristics may not contribute to lowering OWOB levels in young children in Ireland. However, it

should be considered that the perception of the neighbourhood may vary between families and that associations with objectively measured characteristics may differ.

Moreover, nearly a third of children were in informal childcare arrangements and had a slightly elevated BMI, compared to those who were not in a permanent care arrangement, a difference that did not change over time. The elevated BMI led children in informal care arrangements to be at a higher risk of OWOB at the ages of nine months and three years, which is in line with prior research (174, 243), indicating that the influence of the psychosocial environment of children on their weight extends beyond the immediate family.

Notably, children in formal care arrangements had similar BMIs to those who were not in permanent care, which is most likely due to the fact that formal childcare institutions are better regulated and employ trained carers who may encourage physical activity and healthy diets more than untrained informal carers. Many informal carers were relatives of the child and may want to spoil children by allowing them treat foods and additional screen time, as has been seen in previous research (243, 396). Furthermore, the higher OWOB risk of children in non-parental childcare does not appear to be attributable to shorter breastfeeding duration and earlier weaning (243), as the latter variables were included in the analysis of this thesis. Additionally, even though this mechanism was observed in older children in Ireland previously (422), it would be expected to be seen among children in both formal and informal childcare settings. Considering the high proportion of families that avail of informal childcare arrangements in Ireland, the educational role of informal carers may need to be emphasised more, and parents and informal caregivers should seek the dialogue about weight-related behaviours of children to protect them from obesity.

Early weight-related lifestyles of young children

A number of associations were found between children’s lifestyle factors and their BMI trajectories, the largest of which was seen between daily duration of screen use and BMI at the age of five years. Children who used screens less than an hour

a day or not at all had almost 0.3kg/m^2 lower BMI scores than those who used screens between one and two hours a day, based on what the PCG reported. This is in line with prior research (423, 424). Accordingly, children who used screens less than an hour or never were much less likely to be obese, whereas children who used screens three hours or more had a much higher obesity risk, which was also observed in older Irish children (388). The sedentary nature of screen-based activities and the resulting reduction in energy expenditure deliver a plausible explanation for this relationship. Additionally, screen time may trigger children's excess weight gain through the influence of food advertising on food consumption (27), through the release of stress hormones (210) or through a reduction in sleep (213), and it is possible that these mechanisms act in congruence, hence explaining the strong association seen in this study. While no association was seen at the age of three years, this may likely be due to measurement errors, as parents were only asked about the time children spent watching TV, which is unlikely to give a full account of the totality of screen use. Notably, high screen use at the age of two years tracked into adolescence in a Canadian study and was related to an unhealthy diet, high BMI and reduced school engagement at age 13, which stresses the long-lasting impacts early lifestyles can have on children's weight and weight-related behaviour that may reach into other areas of children's development (425). An early focus on minimising the amount of screen time may therefore help children adopt healthy lifestyles in later life and provide an important opportunity for children's development beyond a healthy BMI.

Furthermore, children who slept little had a slightly lower BMI than children with normal sleep at nine months of age; however this association reversed and short sleepers exceeded the BMI of normal sleepers at three years, remaining on a slightly increasing trajectory thereafter. When the analysis was adjusted for other lifestyle factors, this association disappeared and instead children with the longest sleep were found to have significantly lower BMI scores overall. Accordingly, a higher OWOB risk at the ages of three and five years was seen in short sleepers, whereas the risk of obesity in long sleepers at the age of five years was even below that of normal sleepers. In another study, the longitudinal follow-up of children's sleep habits from the age of six months to seven years confirmed that BMI scores were higher among seven-year-old children with chronically shorter

sleep (216), implying that the trend seen in this thesis continues as children grow older. Thus, the importance of children's adequate sleeping hours may need to be communicated more to parents. Importantly, while short sleeping hours should clearly be avoided, longer than average sleeping hours appear beneficial for a healthy weight development and should be promoted by parents.

A small positive association between antibiotic intake and BMI as well as with obesity were observed when the children were five years old, suggesting that metabolic processes, such as due to an alteration of the gut microbiome (198, 411), may influence children's early weight gain. A similar small relationship between increasing number of antibiotic courses and BMI, overweight and obesity was consistently seen in systematically reviewed cohort data previously (120, 199). The relationship was independent of common childhood infections (426) and was not observed for non-antibiotic medication (427), which implies that not the treated disease but the antibiotic treatment itself is likely to trigger weight gain in children. Similar to the findings in this study, differences across the ages were seen in a British cohort study (427), which may be due to other factors impacting the gut microbiome of young children (197, 198), and a cohort study is recently investigating this relationship in depth (428). Unfortunately, no data were available to test the relationship between antibiotic intake and BMI in the GUI infant cohort at the age of nine months. Nevertheless, the findings from this study stress that healthcare professionals should prescribe antibiotics to children cautiously, and when antibiotic treatment is required, they should consider the choice of antibiotics (426) and the simultaneous prescription of probiotic supplements (429).

Surprisingly, no clear relationship was seen between BMI and either the diet or physical activity of children. Small relationships existed between BMI and both unstructured and structured physical activity at the age of five years; however both coefficients were very small and the findings inconclusive. Similarly, no conclusions can be drawn from the food composite score used in this thesis, which was not related to BMI or weight status in the analysis. Alternative models that replaced the food composite score with individual food groups also did not identify associations with BMI, OWOB or obesity. High BMI trajectories among young children were associated with the consumption of fast food and sugared drinks in previous

studies while lower trajectories were seen if children consumed a high amount of vegetables and wholemeal cereals or participated in organised sports (188, 404, 423). However, inconclusive relationships between dietary behaviours and BMI or weight status as well as no relationship with physical activity were seen in preschool-aged children across seven European countries (430, 431).

In terms of children's dietary intake and physical activity, it is likely that the variables used in the GUI study may not be representative of daily dietary intake and physical activity and that a different, more detailed set of variables should preferably be used to assess these variables in the future. The present physical activity scores are based on a number of different unstructured and playful physical activities, and on sports club use, both of which were self-reported by the PCG. Children in this cohort had just started attending school before the physical activity data were collected, which is why physical education in school was not integrated in the analysis. In the future, a more comprehensive composite score or even the use of a pedometer to measure activity levels should be considered to better capture physical activity in young children. Additionally, based on the underlying food frequency questionnaire, an accurate assessment of children's diet was impossible. Self-reports of food are often subject to bias as these may be unintentionally incomplete, or they may be adjusted by caregivers based on their perception of foods which are more or less socially desirable (296, 432), possibly masking or reversing relationships between diet and childhood obesity (433). In future studies, a combination of food diaries with food quantity questionnaires appears preferential, in order to assess portion sizes and an accurate account of the dietary composition.

Beyond the potential of measurement error, a number of studies suggest that the effect of early diet and physical activity on weight status may only become visible at a later age, bearing the risk that parents and carers of young children may underestimate the importance of lifestyle behaviours (202, 203, 430). Notably, dietary patterns were found to change little from the ages of two to five years previously (287) and high continuation of physical activity and dietary habits from childhood into adulthood was seen in a broad systematic review (434). Together with the findings of sleep duration, screen time and antibiotic intake, in relation to

BMI in this thesis, this indicates that a healthy lifestyle should be pursued early in children's lives in order to prevent them from developing OWOB. As it is likely that parental and children's lifestyles overlap after the infancy period (221), families need to be tackled jointly.

7.2.3 The extended conceptual model

The findings presented in this thesis add to the literature on early childhood obesity, and to the SEM by Reed et al. (8) and the *Complex web of potential determinants of overweight and obesity in children* by Monasta et al. (1) in particular. The findings confirm that the extended conceptual model presented in Chapter 2 (Section 2.5.1) is a useful guide to studies of childhood obesity, with a few small amendments that are shown in Figure 28 and discussed below.

On the individual level, as suggested in Chapter 2 (Section 2.5.1), antibiotic use was added to the model, due to the association with OWOB at the age of five years seen in this thesis. Associations of OWOB with other individual-level factors (gut microbiome, physical activity level, increased food intake and sex) were not seen in this thesis; however these factors are suspected to lie on the pathway to OWOB and should therefore be investigated in future studies (Figure 28).

On the level of parenting style and family characteristics, gestational age (early and late birth) and household structure (lone caregiver and living with siblings) were added to the final model, as these factors were associated with OWOB at all ages studied. However, Chapter 2 (Sections 2.3.3 and 2.5.1) suggested that children in lone-caregiver households may be at a higher risk of OWOB partly due to increased caregiver stress. No meaningful relationship with caregiver stress was seen in this thesis. In fact, based on the literature presented in Section 7.2.2, it is suggested that a lack of social support may better explain the relationship with OWOB. This should be explored more in future research (Figure 28).

On the level of community, demographic and societal characteristics, ethnic background and informal childcare use were added to the model (Figure 28).

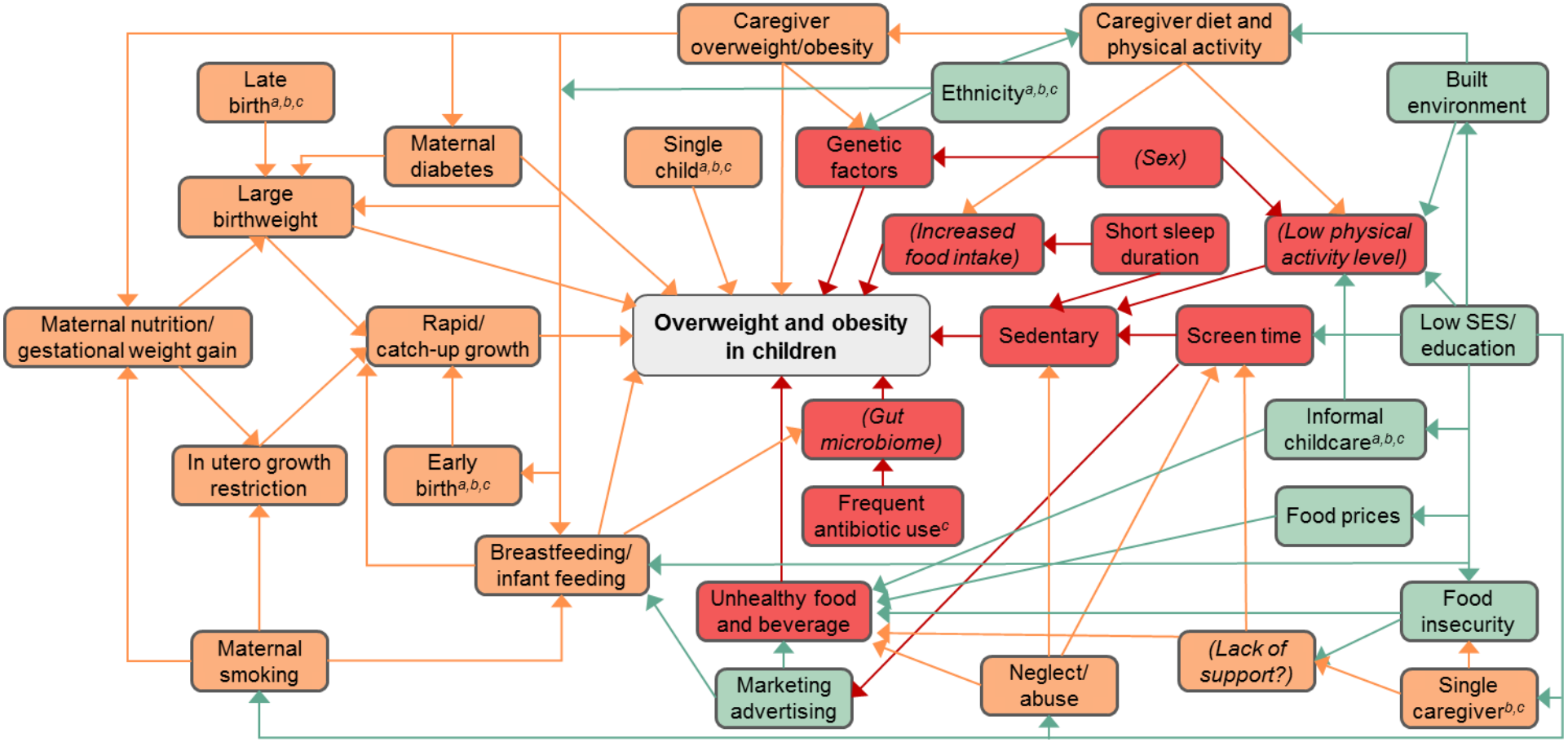

Figure 28: Verified extended Complex web of potential determinants of overweight and obesity in children, based on Monasta et al. (1), Reed et al. (8) and findings from this thesis in children aged zero to five years

Note:

red colour = individual level; orange colour = parenting style and family characteristics; blue colour = community, demographic and societal characteristics; factors in brackets & italics = factors need to be investigated in future studies

Factors added to the model based on the findings of this thesis: [a] = association at 9 months, [b] = association at 3 years, [c] = association at 5 years

SES = Socio-economic status

7.3 Possible actions needed to lower childhood overweight and obesity in Ireland

Based on the findings of this thesis, a targeted approach to lowering OWOB in young children could help to reduce inequalities between children from various population groups or exposed to a variety of risk factors. Effective prevention, intervention programmes and policy-making are required that aim at controlling childhood obesity and its consequences for population health in Ireland. The cumulative evidence of this and other studies from Ireland indicates that there is a possibility to halt, and possibly reverse, the increasing trend of childhood OWOB over time; however the multi-faceted nature of OWOB needs to be acknowledged and targeted appropriately. The results of this thesis can be used as a guide to inform strategies aiming to reach decreases in prevalence of and inequalities in OWOB. Specifically:

- Prevention and interventions are needed before school entry, with a strong focus on the 'first 1000 days' of children's lives
- Prevention and interventions are needed that extend across various areas of the obesogenic environment young children grow up in and tackle defined risk factors and groups identified in this study jointly. This is because many risk factors exist that only affect a small proportion of children or only lead to marginal changes in BMI if studied in isolation
- Prevention and interventions for different risk factors and groups are needed at different time points throughout the development of young children. Biological risk factors should be targeted before birth, a multitude of material and psychosocial factors targeted within the first three years of children's lives and healthy lifestyles continuously promoted thereafter.

In the following section, a number of recommendations are described that were derived from the findings of this thesis outlined previously (Section 7.2). Recommendations for future research in the area of childhood obesity that arise from this study will be discussed below. Furthermore, recommendations for policy-makers will take into consideration the policy framework in Ireland that may be supportive or act as a barrier to pursuing these goals.

7.3.1 Policy recommendations

Health in All Policies

To effectively lower early childhood OWOB levels, interventions that operate through changes at policy level are needed (435) and Health in All Policies as well as whole-of-government approaches are increasingly in more demand and utilised (436), including in Ireland (437). It is argued that sectors other than the food industry need to be approached and that the promotion of healthy child weight should be integrated at various policy levels as single interventions are rarely effective (436). Based on the findings in this thesis, policy sectors involved in transport and local planning and childcare and preschool services may need to develop strategies, in addition to the healthcare sector, that may potentially help to lower (inequalities in) OWOB levels. Policy-led intervention programmes that target various levels of children's obesogenic environments and promote the collaboration of stakeholders within and across these levels have potential to decrease childhood obesity both in urban and rural regions (438). However, effective interventions that stretch across different areas of early child development rarely occur (435).

Moreover, there is a need to develop both more scalable interventions (439) and interventions tailored to minority groups (440) and specific settings (441), as well-planned multi-component interventions to lower OWOB levels in young children are likely to fail if delivered at scale only (441). Hence, various hierarchical levels of policy interventions, for example a combination of national and local strategies, are required to encourage behaviour change and address childhood obesity on a population-level. National strategies in this regard have the potential to effectively reach out to the whole of society, change behavioural norms and provide resources for sustainable implementation, whereas tailored policies are needed to target the physical environment of children and local needs (442). For example, the EU School Fruit Scheme provides funding to EU countries for running national programmes to increase the consumption of fruit and vegetables in preschool settings. EU countries that avail of this funding are required to match this funding and deliver these programmes on a national level. However, it is up to preschools,

on a local level, to implement the programmes effectively, which may involve the cooperation with local fruit and vegetable suppliers or canteen services (442).

In Ireland, the *Obesity Policy and Action Plan 2016-2025* aims to reduce inequalities between the highest and lowest socioeconomic groups by 10% through a combination of national actions and targeted policies, such as in children and disadvantaged groups. To achieve this, two levels are proposed, *ô t -d p w n ô* measures where different sectors play key roles in shaping the obesity prevention environment and *ó b o t-ut* approaches which empower individuals, families and communities to make healthier choices and inform relevant sectors of future strategies (35).

To respond to the multi-faceted nature of OWOB risk factors identified in this study, Irish policy-makers should take various factors into consideration that contribute to the obesogenic environment of young children and their families. In Ireland, changes in the physical environment may be needed that require policy involvement particularly in rural regions. This may involve an increase of healthy foods in shops and a reduction in availability of cheap takeaway restaurants, as has been initiated through policies in the UK (442). Furthermore, regulation of media content may help lower children's exposure to marketing through screen use. However, both the physical environment and media environment of families are difficult to control and thus appear unsuitable for trials; as a result, evidence-based interventions that might be applied to the Irish context are missing (440, 442).

As psychosocial risk factors of early childhood OWOB were observed in this study, this highlights the need for policy-makers to take account of contextual factors, such as the employment situation of parents which largely determines family routines (440). For example, to promote healthy infant feeding, better regulation of maternity leave and more flexible working arrangements may be needed In Ireland where these appear to influence feeding practices (443), while improved working conditions during pregnancy may help lower the risk of preterm delivery (444). Such changes will require the joint effort from various policy sectors and are not feasible through individual bottom-up approaches. However, given the strong focus on early intervention and its potential benefit on the life-cycle of obesity and overall

population health (35), this should be made a core priority in Ireland. Furthermore, this may have the potential to reduce early (socioeconomic) inequalities, as a prolonged period of sustained income during maternity leave may relax the financial situation of families otherwise struggling to afford high-quality childcare services.

The Healthy Ireland Strategy (445) sets out to include early prevention and intervention, as well as health promotion across policy sectors by 2025. To date, this has resulted in a voluntary code of practice that limits the marketing of food high in fat, sugar or salt in non-broadcasting media including retail environments (446). Furthermore, a ban of food marketing on children's TV channels exists in Ireland, however this appears relatively ineffective as children continued to be exposed to a high level of advertising after the ban came into effect (447). Hence, these policies are unlikely to contribute to a reduction of the association between screen use and OWOB seen in this thesis, unless implemented more effectively, e.g. by banning food marketing from all TV channels and media other than TV.

To encourage Ireland in the pursuit of more inclusive weight-related policies, more efforts may be required to translate health research into policies. In Ireland, a lack of prioritisation among policy-makers was identified as a potential barrier for the promotion of childhood obesity prevention strategies (437). Better priority-setting that considers the integration of obesity prevention across sectors may be reached through (economic) evaluations (437), for example using microsimulation models to investigate cost-effectiveness of interventions (16, 448). The recent cost-of-illness study on childhood obesity in Ireland can help guide evaluations (16).

Breaking the life cycle of obesity: Establishment and use of early points of contact

This study showed that prevention efforts are required before children start attending school, whereas internationally and in Ireland, most healthy weight promotion is delivered in the school setting and thus targets older children (205, 449). While it is relatively easy to reach out to children through schools, points of

contact before school entry are more scattered, with few that are regularly accessed by all families. The aggravating relationship between caregiver and child BMI over the first five years of life that was seen in this study confirms the notion that parents pass on their weight status to the offspring. If the development of high birth weight and rapid infant weight gain could be reduced through effective intervention during pregnancy (450), and risk factors found during infancy and early childhood be targeted through continued prevention and intervention subsequently (451), many young children may not move into the overweight or obesity category before school entry. Consequently, fewer children could carry excess weight into adolescence and adulthood, and fewer adults would ultimately pass on an increased obesity risk to the next generation. To fully exploit this early opportunity to lower OWOB throughout the life-cycle, contact points in Ireland need to be identified and prevention strategies effectively incorporated in them that can support all families in protecting young children from excess weight gains. Some potential options are discussed next.

Childcare and preschool services

One such point of contact that was frequently availed of by families in this study is the early childcare setting. Formal childcare institutions were previously found useful to give children access to a healthy diet and sufficient physical activity and may have the potential to reduce prevalence of and inequalities in childhood obesity in Ireland (452-454). Importantly, previous interventions delivered through the childcare setting that only focused on the diet of children had little to no impact on child weight outcomes, whereas combined lifestyle interventions appeared more effective that included the home and community environments children live in (452), both of which were associated with OWOB in this thesis. Additionally, interventions tend to require expert delivery to be effective (452); hence the resources required for effective prevention of obesity through childcare settings should not be underestimated.

Moreover, as was seen in this study, not all children regularly attend childcare, and many families make use of informal care arrangements that are not subject to state

regulation. As the cost of crèches in Ireland is high, families with a low income or who have more than one young child requiring childcare may tend to use informal childcare arrangements more, which in fact was associated with higher OWOB levels and a slightly higher BMI in this study. Hence, policies solely addressing the care of children in formal childcare institutions may help decrease the total incidence of OWOB in young children but may contribute to inequalities between those children attending and those not attending these institutions. Ideally, formal childcare should be substituted and made accessible to all children; however this may not be feasible from a cost-perspective.

Preschool may therefore be more suitable to reach out to children across income groups and social strata. For example, the provision of healthful food (such as the provision of free fruit under the EU School Fruit Scheme) as well as nutrition education can effectively contribute to reducing inequalities in childhood obesity (442). Similarly, the inclusion of physical activity in preschool curricula is a cost-effective strategy to increase exercise (455) and the reduction of screen time could also be addressed in preschools (442). Nevertheless, it needs to be acknowledged that only three hours per weekday within the academic year in preschool are free of charge for three- to five-year-old children in Ireland (456), which limits the opportunity for behavioural interventions during preschool hours. It is rather programmes delivered through preschool that involve home-based components, such as nutrition education and skills provided to children and their parents through the preschool setting, which hold promise as a sustainable and cost-effective intervention (442), as behaviours learned or pursued in preschool can effectively be reinforced in the home environment and vice versa (457). A positive example is the European ToyBox intervention study that achieved an improvement in weight-related lifestyles both among children attending preschool and their families over a nine-month period. Delivery of this intervention was at a relatively low cost per child and evaluated likely to be cost-effective, in addition to a high feasibility and acceptability among families across social classes (442).

In Ireland, nutrition guidelines for preschool services exist (458) and preschools can participate in the SMART START (459) training programme which educates preschool staff to deliver appropriate care to children and promote healthy weight-

related behaviours in preschools. Additionally, educational materials are available to preschool staff through a tailored online hub (460) and the *Get Ireland Active!* website (461). *Get Ireland Active!* also sets out the development of National Guidelines on Physical Activity for children aged zero to five years through the Health Service Executive by 2018 and the promotion and inclusion of physical activity in childcare and preschool services by 2020 (462). As of October 2018, these plans have not been implemented; however the *Kids Active Physical Activity Resource* was developed in 2017 to increase children's fundamental movement skills and physical activity in Irish preschools (463).

Numerous scalable resources thus are available to or planned for implementation in preschools in Ireland; however children do not start attending preschool before the age of three years (456), which may be too late as sole point to deliver prevention strategies, given the high prevalence of OWOB before this age. The adaptation and implementation of successful programmes to (informal) childcare settings should therefore be explored to target the youngest children. Educational materials and interventions delivered in preschool should furthermore be provided to families for home use. While the initiatives in preschool should be continued, an evaluation of their (cost-) effectiveness may help focus this width of resources.

Healthcare providers and community services

A promising point of contact that is part of the policy agenda in Ireland lately (35) can be found in the delivery of primary healthcare and health services (105) as they can reach out to families before children start attending (pre-) school and already during or before pregnancy. While a deep debate about the Irish healthcare system is beyond the scope of this thesis, it should be mentioned that the efficiency of the system gained much attention publicly in the past decade. The system was long criticised for its resistance to change and reforms that were not (fully) implemented, as well as budget cuts owing to the economic disruption of Ireland due to the recession from 2008 to 2010 (87). Since the GUI infant cohort data were collected, the role of health services in raising awareness and managing

OWOB in young children has been acknowledged more (464, 465), providing an opportunity to lower OWOB levels through various levels of healthcare provision.

Ireland announced to take a life-course perspective on obesity (35) and various policies, frameworks and health information resources emerged in the decade after the GUI infant cohort was born that aim at lowering obesity levels in the youngest children. It is now time for health policy-makers to connect these initiatives, reinforce their implementation and evaluate their outcomes, in order to ensure continuation of care from preconception through pregnancy and the first five years of life. It is furthermore of utmost importance to identify and remove bottlenecks, such as staff shortages and time-constraints that presented barriers to effective healthy weight promotion in Ireland previously, as well as to close loopholes that may exclude individual population groups from the benefits of these policies.

Already family planning services and antenatal service providers should promote a healthy lifestyle, to target parental weight and children's birth outcomes before and during pregnancy. High GWG, particularly in the first two trimesters of pregnancy (48, 119) is related to both high birth weight (119, 124) and rapid infant weight gain (163), which were associated with the highest BMI in this study. Dietary, exercise or combined interventions delivered to pregnant women effectively reduced excessive GWG in various randomised controlled trials (450), implying that gestational behaviours can be targeted through effective healthy weight promotion. In addition to the promotion of general advice on healthy gestational lifestyles, the implementation of interventions in diabetic women through antenatal services should be promoted. Similar to GWG, GDM is associated with high birth weight (129); however excess foetal growth can be slowed down and the risk of macrosomia and neonatal adiposity lowered through a controlled diet, physical activity or insulin therapy in diabetic women (119, 450).

Furthermore, resources for effective weight management should be extended to family planning services which offer an additional opportunity to decrease the risk of future childhood obesity before pregnancy. Prior research identified an elevated risk of large BWFGA and lower breastfeeding onset and duration if mothers were obese before pregnancy (466). Furthermore, a reduction in pre-gestational BMI among overweight or obese women combined with limiting their GWG can

effectively lower the risk of GDM (119). Additionally, a high maternal pre-pregnancy BMI was found to be indicative of pre-term births (119, 126) and nutrient deficiencies were found to occur in children of smoking women in the womb and after birth (128, 132, 133), both bearing a high risk for rapid infant weight gain (162). As adult OWOB and smoking are highly prevalent in the Irish population (467) these factors should be targeted through counselling before the onset of pregnancy to eliminate their impact on foetal and infantile weight gain. While only one third of women accessed preconception care in Ireland until recently, Healthy Ireland intends to make early health counselling available to more women (467), which could open doors for novel prevention strategies that may reduce the demand for later intervention.

Without better and more equitable access to preconception care, however, the delivery of interventions and weight promotion through primary care holds potential to further increase socioeconomic inequalities in the risk of childhood obesity. The current split in public and private health service provision leads to varying degrees of availability and quality of healthcare in Ireland, where economically disadvantaged young adults may not be able to afford the same level of care as more affluent individuals and families. While the Irish government expressed their intentions to thrive for universal healthcare provision in Ireland, it is questionable whether these structural changes can be achieved in the near future (87).

Additionally, healthcare providers require appropriate training and evidence-based resources to facilitate effective weight counselling prior to and during pregnancy. Pregnant women in Ireland previously reported a lack of information on healthy GWG, nutrition (468) and physical activity levels (469) during antenatal care, including that they were not weighed (468). As a result, many women stopped exercising when they were pregnant (469). Disengagement of antenatal service providers, such as due to a lack in time and knowledge, can hence act as a barrier to healthy gestational lifestyles (470). To address this, guidelines on healthy gestational behaviours are provided to healthcare professionals in Ireland (471). Additionally, since 2015, the *Brief Intervention for Healthy Weight Management Before, During and After Pregnancy* sets out that GPs should measure women's weight and height and recommend daily moderate physical activity during

antenatal health checks; that means at the start of pregnancy and in the 24th to 28th gestational week (472, 473). A multitude of dietetic services are furthermore available to diabetic pregnant women in Ireland (467) and effective screening for GDM increased since the GUI infant cohort was born (314). However, access to dietetic services in non-diabetic pregnant women is limited, raising a need for additional weight management services (467). Moreover, as most guidelines are relatively young, Irish policy-makers should assess their use and effectiveness in daily practice and try and remove barriers such as GPs' time-constraints.

In addition to intervening in the (pre-) gestational period per se, antenatal services and maternal wards can function as contact points for interventions initiated during pregnancy that stretch into the children's early childhood years and provide continued guidance to families, including those in rural regions (474). A qualitative study among a subset of families from the GUI infant cohort revealed that the first days postpartum were critical for women to initiate and gain confidence in infant feeding practices. However, the women indicated that a lack of support existed in some Irish hospitals, and that care was not provided in a timely manner due to staff shortages, leading many women not to breastfeed their infants (317). The recent National Maternity Strategy sets out to improve these services; however it acknowledges that cultural changes in Ireland are also needed (467), which may impact on the uptake and effectiveness of this strategy if delivered in isolation.

In other countries, combined pre- and postnatal interventions delivered through nurses across the 'first 1000 days' of children's lives effectively lowered the BMI of two-year-old children (475) and the OWOB prevalence up until school age (451, 476). Nutritional or responsive feeding strategies appear particularly effective (477). One successful intervention guided mothers to identify cues of hunger better, provide alternatives to food for calming children and promote better sleep and activity routines (476). Furthermore, trials based on a modification of standard infant formula to contain less or different forms of protein resulted in a lower child BMI up until the age of six years (478, 479). Policy-makers in Ireland should review these interventions and consider their integration in Irish antenatal services, in order to target children's weight developments before the age of three years, and particularly rapid weight gains in the year succeeding birth.

As Irish women perceived home visits by public health nurses after hospital discharge positive and helpful (317), early OWOB prevention may reach out to a broad spectrum of young mothers and yield a high level of acceptability among them if embedded in these home visits. Since 2016, *The Nurture Programme* delivers online training to public health nurses and practice nurses that includes information on breastfeeding, weaning, physical activity and sleep needs of infants and young children, as well as culturally appropriate advice tailored to minority groups (480, 481). Information materials on infant feeding (471) and the *Nutrition Reference Pack for Infants 0-12 months*, were also developed for use by area medical officers, public health nurses and practice nurses and are annually updated by health authorities (482). As a result, existing health service structures for infants in Ireland appear to be well exploited. However, their timely initiation after birth and continuation of care should be monitored to maximise beneficial effects on children's weight gain (317).

Beyond the home visits to guide families in Ireland through the first period of child growth, GPs have recently been recognised for their role in the monitoring and promotion of children's healthy weight gains. In 2016, the *Making Every Contact Count (MECC)* framework was established that seeks to integrate health prevention and intervention in routine primary care (464), such as through growth monitoring of children during vaccination appointments (35). Under this framework, training of brief interventions on nutrition and physical activity is provided to health professionals and healthcare support staff and is planned to be integrated in medical university curricula (464). Furthermore, an obesity algorithm for children is available to GPs for guidance in weight management (483) and, if required, GPs are responsible for referrals to appropriate specialist services and health information (464). For weight management, *MECC* aims to liaise with community services to account for the limited capacities and resources available in primary care (464). The inequalities seen in this thesis may be reduced through community strategies by increasing social capital (484) and due to their feasibility in rural settings (485). With the involvement and reinforcement through the primary care sector, these strategies have further potential to lower OWOB levels in young children (486).

The implementation of *MECC* holds promise to lower OWOB levels, as shown by a small trial that introduced a brief lifestyle intervention to Irish families at the 13-month immunisation appointment (487). The intervention effectively improved children's weight-related behaviours, including their daily screen time, which was associated with OWOB in this thesis, and the intervention was acceptable to parents (487). As socio-economic differences in the obesity risk existed in this thesis, it needs to be considered that families may only make full use of health services if they are freely accessible and incentivised (442). Furthermore, children with lone caregivers may particularly face barriers to accessing separate services for weight monitoring or counselling due to the required time commitment and travel (486). The context of a wider health examination or vaccination appointment under *MECC*, which is provided freely to young children in Ireland, hence has potential to reach out to all families and reduce inequalities (465). A few years after the implementation of this relatively young framework, the impact of *MECC* on weight outcomes in young Irish children should now be evaluated, such as through weight surveillance and the investigation of facilitators and barriers for reaching the framework's full potential to benefit child health outcomes.

Currently, routine growth monitoring in young children in Ireland only takes place at the ages of two and five years (35, 465). The findings of this thesis show that weight gains in infancy and across the first three years of children's lives have important implications for their later weight status. Hence, weight and height should be monitored more regularly than set out in the current GP contract (465), indicating that more frequent healthcare contacts may be required to lower obesity levels than delivered under the original *MECC* umbrella.

Furthermore, as Irish healthcare providers hesitate to raise the topic of childhood obesity with parents (333), additional efforts may be needed to achieve full uptake of *MECC*. Parents commonly perceive their overweight or obese young children to have a healthy weight or to be underweight (488). Consequently, parental engagement in changing lifestyle routines is low (489) and children grow up with a distorted idea of the ideal body image leaning towards a heavier norm (416, 490). A lack in communication and professional involvement might contribute to the low degree of parental awareness about childhood obesity and child weight

management (416, 491) that was also seen in Ireland (389, 416). A non-stigmatising communication guide for health professionals and families is already available in Ireland that aims to improve their dialogue about childhood obesity (483, 492). The use of visual materials such as body image scales may further aid the unbiased discussion of the sensitive childhood obesity topic and increase the recognition of OWOB by parents (493) and should be considered in Ireland.

Public awareness campaigns through social media

The increasing gap of OWOB levels across educational groups highlights the need to include educational components in Ireland's health policy-making, to raise parental awareness of what constitutes a healthy lifestyle for children and to make healthy lifestyle choices the norm. Interventions delivered during occasional consultations with health professionals should be reinforced through continuous health promotion outside the clinical setting that can reach families across SES groups, geographic locations and irrespective of time constraints.

Given the high level of screen use in the GUI infant cohort, new media and the growing familiarity with those offer opportunities for eHealth and mHealth prevention and intervention (494). However previous interventions were largely ineffective in improving weight-related outcomes, the dietary quality or physical activity of children (494). Additionally, active video games that aim at improving children's nutritional knowledge or increasing their physical activity were evaluated as unsuitable as sole intervention for the prevention or treatment of obesity (495).

A more promising and cost-effective tool to increase the health knowledge among parents of young children may be the use of health communications which could positively influence parental awareness and health choices in a sustainable manner in Ireland (416). For example, campaigns with celebrity involvement, such as the involvement of Jamie Oliver in the *#AdEnough* campaign to stop fast food marketing to children (496), hold promise to raise awareness for healthy behaviours through positive role modelling (442). In Ireland, the media campaign *Let ôs take on childhood obesity* aims to tackle small *one small* weight-related everyday habits of families, such as the regular consumption of

‘treat foods’, inappropriate portion sizes, screen time and sleep habits, and provides parents with practical solutions to change these behaviours (497). Moreover, this campaign promotes use of these strategies among carers other than a child’s parents (497), which appears useful given that informal childcare was associated with OWOB in this thesis. Notably, a real risk of these campaigns is the creation of stigma that children with OWOB frequently need to live with (70); hence it is important that campaigns are delivered in a neutral, non-stigmatising manner (498). In order to prevent adverse effects, such as eating disorders and reduced well-being among children, healthy lifestyles should be encouraged without specifically referring to or criticising children’s weight (499).

Additionally, internet platforms are a promising way to deliver nutrition education to children (500), even though parents need to be targeted before children become literate. As part of *The Nurture Programme*, *mychild.ie* has been developed, which is an online resource with health-related information for expectant couples and parents of young children and links to community services (481). A collection of books, booklets and recipes is also promoted to parents of young children online to encourage them towards healthy nutrition and physical activity levels (501).

Given that social media are increasingly being used for communications and knowledge generation, they provide a useful platform that may likely deliver healthy-weight promoting messages in a cost-effective way. However, similar to the marketing of food to children (447), where feasible, governments may need to control the advertisement of information resources, in order to ensure that families with various levels of literacy and media use have equal access to information. Evaluation of campaigns and information platforms is crucial to identify their effectiveness and potential for social inequalities, misguidance and stigma.

7.3.2 Recommendations for research

Assessment and reporting of overweight and obesity prevalence in young children

The variation seen in OWOB estimates based on different child growth criteria and cut-off values within them confirms that careful consideration is required as to which child growth criterion to apply, and that a transparent description of the methodology used to assess OWOB prevalence in young children is of utmost importance. Prevalence trends can easily be misinterpreted as different clinical and surveillance cut-off values for child growth criteria tend to be used interchangeably (336, 337, 383), and terminologies to describe BMI categories are frequently confused (239, 382). To increase the transparency of future studies, use of a common terminology and more comparable cut-off criteria are suggested to define BMI categories, such as a distinction between ‘grade 1 overweight’ and ‘grade 2 overweight’ or ‘obesity’ suggested by Rolland-Cachera et al. (116).

The WHO growth reference classification (defining OWOB as > 1SD), applied to the data underlying the WHO growth standard, appears most useful to estimate prevalence of OWOB in young children and to enable the comparison with children aged five years and older. The IOTF growth reference is less useful as it tends to underestimate the real scope of OWOB prevalence (327, 376) and the WHO growth standard classification is not suitable as OWOB estimates are even lower. In the cross-sectional analysis of this study, more relationships with risk factors were detected when the threshold of 1SD was used to define OWOB compared to 2SD, and there was a higher level of agreement between the longitudinal analysis and the cross-sectional analysis that used 1SD as a threshold. Hence, OWOB risk factors on population level may be identified earlier and more accurately using the WHO growth reference classification and, specifically, its overweight threshold. Accordingly, this should be used to monitor OWOB in young children and to assess the effectiveness of interventions and policies in Ireland and internationally.

However, even though the WHO growth reference classification is favoured for population-level data of young children in this study, it is difficult to determine its clinical relevance without assessing the adiposity status of individual children, for

example through direct measurements of children's body fat using dual-energy x-ray absorptiometry (502). Moreover, while diseases related to OWOB typically evolve in the later childhood years, adolescence or adulthood (30, 42), markers for these diseases, such as insulin resistance (60), hypertension (63) and arterial stiffness can already be present by the age of five years (59). A comparison of the sensitivity and specificity of existing child growth criteria to predict these outcomes could provide the evidence needed to reach consensus as to which criterion is most applicable to young children. The additional effort required to test this appears relatively small, once detailed anthropometric and medical data are collected to investigate childhood OWOB. Ultimately, this consensus-building process could further improve the effectiveness of BMI as a cheap and simple tool to assess adiposity levels in young children.

Additionally, considering the inconsistent and partially non-transparent use of criteria across studies of childhood obesity, a standard or protocol for reporting future studies is suggested as its use could make findings more comparable over time, age groups and across countries. The detailed reporting based on a protocol offers potential to increase the replicability of studies and the adoption of more coherent study designs over time and may hence contribute to a standardised assessment and reporting system of OWOB prevalence in the future. As a result, the translation of research into policy may become easier. Specifically, to maximise the quality of reporting, such a protocol should be embedded in and used in addition to other reporting guidelines, such as the STROBE checklist for observational studies (321), and could contain the information shown in Table 29.

Table 29: Suggested protocol for reporting of childhood obesity research

Category	Details
Anthropometric measurements	Nature of the anthropometric data used *(such as body mass index, body weight, body fat percentage)*
	Measurement instruments and details of procedures *(such as regulations around clothing)*
	Indication of mechanisms in place to standardise measurements, if applicable
	Indication of mechanisms in place to lower the rate of bias and error, in cases were measurement is not standardised *(such as self-reported data)*
Child growth criteria	Name of the child growth criterion or criteria applied to classify weight status
	Mentioning of an identifiable scientific source that describes the criterion or criteria and the respective threshold values in use to classify weight status
	Scale on which the thresholds were measured *(such as z-scores/SD, centiles, or crude scores)*
	Location of threshold values for body mass index categories on the scale used (such as 1 z-score or 85th percentile)
	Indication whether the overweight category includes or excludes obesity, and whether the 'normal' or healthy weight category includes or excludes underweight children
	Distinction of the child growth criterion or criteria as mainly intended for 'clinical' or 'research/surveillance' purposes
Relationship to other criteria	Rationale for why the respective child growth criterion or criteria and threshold values were chosen
	Indication whether findings based on an alternative child growth criterion or criteria are available in supplementary documents or can be requested from the authors

Researchers should consider how best to place their study in the existing pool of knowledge of OWOB studies and dedicate more awareness to making their research transparent and comparable within this pool. For example, to connect future findings to past research, the parallel use of WHO and IOTF criteria appears more useful than choosing between them. The additional use of a national criterion may be reasonable in countries where national data have traditionally been assessed through this.

Future research of overweight and obesity in young children in Ireland

The longitudinal data collected by GUI offer a unique opportunity to study OWOB levels in young children from 2008 to 2013; however newer data need to be compared to the findings in this thesis to investigate early OWOB in the future. A decade after the first wave of GUI infant cohort data was collected, it would be interesting to see whether policies and interventions implemented since then were able to lower OWOB levels in the ages from birth to five years. A national monitoring and surveillance system was recommended for implementation as an effective tool to monitor this progress (436), and the collection of data from a new infant cohort in the near future is worthwhile. The GUI study design appears suitable for this undertaking, given that the instruments for data collection have already been developed and tested in pilot studies. Nevertheless, it needs to be acknowledged that additional resources would be required to sample, collect and maintain the data of a third cohort, and to follow up with families over time.

Alternatively, other studies have investigated the BMI of children at various ages using anthropometric data from medical records (503, 504), and this is also underway in Ireland for children aged two and five years (465). As part of the periodic growth monitoring at these ages, GPs should deliver a dataset to the Health Service Executive containing height and weight data and the sex and age of each child (465). At the date of this writing, there is no indication of how this dataset is going to be used and whether the data will also be connected to hospital records. Following the principles of the *MECC* and considering the strong association with birth factors seen in this study, measured weight and length data

should additionally be collected from birth certificates and linked to later measurements from GP visits. Furthermore, an additional growth assessment during infancy appears reasonable to identify children with rapid infant weight gain.

Advantages of using health record data are the relative ease of integrating growth assessments in GP consultations, and the representativeness of a dataset that can be expected to include standardised anthropometric panel and longitudinal data of most Irish children. Thus, children in population groups that are difficult to reach could be included (e.g. children living in rural regions who had slightly higher BMI scores in this study), but who tend to be under-represented in health surveys (504). On the contrary, particularly factors at the community, demographic and societal level are unlikely to be assessed through medical records, as a high administrative burden and ethical concerns around storing such data may arise. To investigate risk factors of childhood OWOB in future generations, a subset of children could be sampled from GP visits for more detailed data collection.

Even though data are from regional population samples or do not include all ages, OWOB levels and trends identified in this thesis should also be compared to the five-year data from the National Children's Food Survey II (358) and data from the Cork BASELINE Birth Cohort Study, which were not available at the time of writing (40, 359). The latter includes data collected during pregnancy and more detailed biological data (359), which may help identify factors that lead preterm or small children to catch up and post-term or late children to catch down to mean BMI.

For a number of explanatory variables included in this study, no or inconclusive relationships with OWOB or BMI were seen, for many of which contradicting or inconsistent findings as well appeared in the literature. These factors should be studied more in depth in future research to clarify relationships and understand mechanisms behind them. For example, a few weak relationships of OWOB were seen with infant feeding or later nutrition. As more evidence is accumulating that indicates an association between high protein intake in infancy (184, 410) or in the subsequent years (505) with increases in BMI or OWOB, future studies should collect dietary data that enable a thorough assessment of this relationship. Additionally, objectively measured neighbourhood characteristics are suggested for

future study, such as the measured distance to recreational facilities and (un)healthy food outlets, as these may impact children's weight-related lifestyles.

Future studies may furthermore help explain the identified differences between ethnic groups by investigating their obesogenic behaviours and isolating potentially modifiable differences between them. This could help tailoring interventions to ethnic and cultural groups (257). Studies with larger sample sizes in the different ethnic groups may bring more light into the question of the true relationship between ethnicity, lifestyle and BMI trajectories among children.

The extended conceptual model, based on findings from this thesis (Figure 28 presented in Section 7.2.3), should be used for guidance in future studies. This model highlights areas that require further investigation, as well as factors that future studies should control for to comprehensively study OWOB risk factors.

Assessment of children's lifestyle

Future studies need to incorporate more sophisticated tools when investigating lifestyle variables in young children, particularly diet and physical activity, which are more complex to measure than sleep and screen time. Physical activity levels may be assessed measuring step counts by using a pedometer, as this is relatively easy and cheap and has high accuracy if worn by children throughout the entire study period. For example, in GUI, interviewers visited the family homes once before the interview took place, to inform families and arrange an interview date, and pedometers could have been handed and explained to families at this first visit or otherwise sent via post.

To assess dietary intake of children, portion sizes should be indicated and ideally, food should be weighed. Mobile phone apps may be helpful to record food diaries, such as through the use of barcode scanners that hold nutrient information about food items. Additionally, a number of validated tools for dietary assessment were published over the course of this thesis. The Healthy Kids dietary and lifestyle assessment tool was shown to validly predict micronutrient consumption and was

associated with BMI in children aged two to five years (432). While primarily designed as a screening tool to assess children's early OWOB risk, it appears useful for data collection in observational studies where no pedometer or food weighing is possible. More tools, including those for young children, were published on https://www.nutritools.org/, and future studies should avail of this evidence-based guide to assess dietary intake (506). Technical innovations furthermore enable the more efficient data collection through online questionnaires and physical activity and fitness parameters can be assessed through the use of a mobile research unit (507).

7.4 Strengths and limitations of the research

7.4.1 Strengths

To the best of this researcher's knowledge, a study examining preschool-aged children longitudinally for this length of time has never been done in the Republic of Ireland previously. A major strength of this study is the representative nature of the GUI infant cohort on a national level. With 11,000 infants sampled through the Irish Child Benefit Register, the initial sample equated to almost one seventh of all births in Ireland in 2007 (267). Previous studies in Ireland that included young children were limited to regional urban settings (40, 97, 342, 348) or small sample sizes (98). The reliability of the data was increased by means of the random sampling technique, as well as based on the intentional over-sampling of defined population groups and the use of weighting factors to address the possibility of response bias. Furthermore, several mechanisms were in place to reduce attrition between waves, and to limit the number of invalid or missing values. Generally, attrition in the cohort was low and little information was missing. Standardised height and weight measurements of almost 8,900 children remained in the last wave. Additionally, a number of mechanisms were in place to increase the quality of the data, and to protect the confidentiality of the children and their families.

Accordingly, the GUI infant cohort offers a sample size that is understood to provide a comprehensive picture of longitudinal changes in weight status and BMI

among preschool-aged children in Ireland from 2008 to 2013. In this secondary analysis of the GUI infant cohort data, prevalence of OWOB including trends over time and between sexes was compared using various child growth criteria. The WHO growth standard, WHO growth reference and IOTF growth reference were applied to the data and their estimates for different weight categories compared. This allows for the comparison of findings in this study to data from other countries, as well as across ages. Additionally, a number of studies (similar to this study) have identified reversed sex trends in prevalence of overweight and of obesity (239, 380, 401). This study contributes to clarifying differences between available child growth criteria used for national and international studies, as well as it assessed benefits and disadvantages implied in using the various criteria.

The use of various categorical and continuous outcome variables to assess children's weight gains throughout this thesis is another strength. Childhood obesity is associated with negative (health) consequences, which is why OWOB categories were examined; however this bears potential for relationships to be masked as children close to the lower and upper threshold value of a weight category were grouped and analysed mutually. Thus, the additional investigation of continuous BMI scores increased the quality of this study. Furthermore, tracking changes in the same children over time allows for stronger conclusions than cross-sectional investigations of weight status. The combined approach of cross-sectional and longitudinal analyses adds value to this study as it increases the comparability of findings to other national and international studies.

Additionally, weight and height were measured by trained researchers on standardised scales, both in children and their caregivers. It is known that self-reported data on weight and height only offer limited accuracy, but are often used in studies as their collection is easier, cheaper and less time-consuming than measuring each child and parent in a standardised way (508, 509). However, no such limitations apply to this study.

Data of the study child and both caregivers were studied, while most other research includes maternal and child data only. A number of variables were assessed at multiple time points and time-variant variables could be included due to the flexible longitudinal analysis approach chosen. The choice of explanatory

variables was theory-driven and additional tests (e.g. for collinearity) were applied to further increase the validity of the study.

7.4.2 Limitations

A few limitations to this study exist: Firstly, standardised anthropometric measurements to assess BMI were only available at the children's ages of nine months, three years and five years, while length at birth is self-reported, with a high proportion (> 50%) of implausible or missing records. Consequently, BMI at birth could not be studied. Furthermore, only limited analysis of prevalence using the IOTF growth reference was possible, as no criteria are available for use in children below the age of two years. While this limitation is bound to the nature of how the IOTF growth reference was constructed and cannot be addressed any better in this thesis, use of the IOTF growth reference at the ages of three and five years was perceived useful, due to the extensive use of this criterion in prior research.

Secondly, only a few studies were identified that investigated the sensitivity and specificity of various child growth criteria to detect cardio-metabolic risk factors and link defined cut-off values to health outcomes in children. Hence, definitions of weight status used in this study need to be interpreted with caution. This limitation applies to all studies that assess weight status in young children based on BMI.

Thirdly, despite the usefulness of BMI to assess body weight status on population level (508) and in infants (414), it needs to be acknowledged that BMI does not provide accurate estimates of body composition or fat mass for individual children that may be achieved by using more sophisticated tools (510). For example, body fat mass trajectories in infancy were related to childhood obesity in former research (407). However other available means to assess the weight status of young children were either subject to the same limitations (115, 325-328) or they were not applicable to population-level data (324).

Fourthly, a number of limitations exist with regards to explanatory variables. A restricted amount of data is available from the children's SCGs because no SCG

was available in the household (12%), or because the SCG did not participate in the interviews (10%). This limitation was addressed by conducting sensitivity analyses. Additionally, a relatively small number of children were born early or very early, but the benefits of separating these two groups were perceived to justify this decision, as differences in growth patterns between the two groups were expected.

Furthermore, typical to observational population-level studies is the reliance on self-reported data that bear the risk of measurement error. In this thesis, birth weight, gestational age at birth, breastfeeding duration, onset of solid food and variables related to the child's lifestyle are self-reported. There is potential for inaccuracies due to recall bias, although this is expected to be low nine months after birth. Some lifestyle variables may furthermore be biased due to social desirability bias, and because dietary items were measured in consumption quantities, rather than portion sizes. More objective tools exist to assess dietary intake, physical activity and sleep; however these are difficult to integrate in a population study of the scale and scope that are covered by GUI. Accordingly, aggregate scores of dietary intake and physical activity that were developed in this thesis are subject to the same limitations as the collected variables. More validated tools to collect dietary data in young children have been developed since the initiation of this thesis, as discussed in Section 7.3.2. These tools may enable more accurate dietary assessment in future studies. A high level of reporting bias is also possible for children's screen time. Particularly in children who spend time in non-parental childcare, the risk of under-reporting is high. However, objective measurement of either screen time or, more broadly, time spent sedentary was not feasible in the GUI infant cohort.

In general, it needs to be considered that reporting bias may be socially patterned and that misreporting may occur in self-reported data. The latter implies that standard deviations and value ranges of continuous variables, as well as the proportion of children in categories viewed as 'unfavourable' (e.g. high amount of screen time, high sugar consumption), may in reality be higher than observed in this study. Accordingly, the effect size of a number of relationships seen in this thesis may deviate from the real relationships. However, for most of these variables, the strength of relationships with weight outcomes would in fact be

expected to be underestimated, based on such biases, whereas the direction and significance of relationships should not be altered due to potential measurement errors.

Additionally, a number of lifestyle variables were only reported at three and five years of age, which is why these variables could not be included in all analyses without losing the nine-months observations. However, while the antibiotic intake of infants could have been recorded in wave 1 of the GUI study, most other lifestyle variables are not applicable at nine months of age. Inaccuracies may have occurred because the PCG was only asked to provide information on the time the child spent watching television or DVDs in wave 2, whereas this was extended to all types of screen use in wave 3. There is a possibility that such inaccuracies may explain why stronger associations with OWOB were seen in wave 3 than in wave 2; however this would not alter the overall findings in this thesis. Also, no retrospective data are available to test whether caregiver weight status or SES variables changed from conception until the child was nine months old, even though this is unlikely. Factors preceding birth could possibly have impacted birth outcomes and should be explored in relation to BMI in future work.

Several other variables (GDM, GWG and nutrition, gestational smoking, advertising, abuse, food insecurity, food prices, caregiver lifestyle, food quantities, gut microbiome, genome) of the conceptual framework used in this thesis (Section 2.5.1) were not tested as they were not recorded in GUI or due to a high risk of bias or measurement error. These variables were mainly expected to lie on the pathway of relationships between other explanatory variables and OWOB. Accordingly, they may have confounded the analysis and increased the complexity of models. However, these variables are recommended for investigations, including pathway analysis, in future studies.

Another limitation is that no linkage of the GUI surveys with small area population statistics or qualitative data is possible (315, 318). This may have improved the quality of this study based on detailed information about the physical environment and wider circumstances of families.

Response bias was seen between waves, based on a number of population characteristics. This was addressed by over-sampling population groups expected to refuse participation, and by the weighting factors developed in each wave of data collection.

Lastly, no causal relationship between BMI or weight status and explanatory variables can be established on the basis of this study.

7.4.3 Alternative analysis options

The longitudinal analysis approach chosen in this thesis was multilevel modelling, which offered a number of advantages. However, other methods were considered, outlined below.

One option was cluster analysis. This aims to group ‘objects’ together, based on their individual characteristics, and thus creates groups or ‘clusters’ of objects which are relatively homogenous within each group but distinct from other groups (511). Two types of cluster analysis were considered in order to identify common co-occurrences of risk and protective factors of OWOB within the GUI infant cohort, and to assess the basic demographics of these risk factor clusters: k-means clustering, which is a type of partition clustering (512), and hierarchical cluster analysis (HCA). Explanatory variables can be integrated into the analysis as determinants of dissimilarity according to which the population shall be clustered, and dissimilarity can be measured using the Euclidean distance between observations (511). However, cluster analysis has been criticised for being too subjective (as the ‘correct’ number of clusters is determined by the researcher) and often produce clusters with little meaning to health outcomes (513).

It also needs to be acknowledged that growth trajectories can be fitted using structural equation modelling, in addition to the multilevel modelling used in this thesis (393). The aim of this thesis was to study various risk factors of childhood obesity known from international literature in the Irish context, in order to identify a set of risk factors that should be targeted through interventions and policies to reduce the OWOB prevalence in Ireland. Specifically, the association of each

potential risk factor with BMI and small changes in mean BMI in relation to each risk factor were of interest. In contrast, structural equation modelling tends to be useful to identify broad growth patterns that are characterised by large differences in BMI (changes), depending on latent patterns in the data. Furthermore, multilevel modelling enabled to study the BMI of children with minimal loss of data, either because children did not participate in all three waves of GUI or because they had missing data for one or more variables in some of the waves.

Using multilevel modelling, the relationship between explanatory variables and BMI at the different ages was explored, accounting for residual variances that may occur within children over time. This longitudinal analysis approach complemented the cross-sectional analysis of the GUI infant cohort well and strengthened the validity of cross-sectional findings.

7.5 Conclusion and verification of hypotheses

Hypothesis 1: Ireland has a high prevalence of early childhood overweight and obesity

In Chapter 4, the hypothesis was that OWOB levels in young children in Ireland are high compared to those in many other countries. This was confirmed using the GUI infant cohort data (79, 379, 385, 386) , with a peak prevalence of up to 46% (WHO growth reference classification) seen at the age of three years. Particularly from the age of three until five years, persistence of weight status was high, which raises a need for early obesity prevention long before children in Ireland reach school age. Simultaneously, while the prevalence of overweight and of obesity among young children in Ireland remains high, it decreased over the study period. Future studies are needed to study time trends, such as through continuous investigation of the GUI infant cohort into older ages and monitoring of BMI in young children born after 2007, and verify the possibility of declining OWOB levels in the youngest children in Ireland. Important research implications furthermore arise from the inconsistent use of child growth criteria identified in this study.

Hypothesis 2: Material circumstances increase the risk of early childhood overweight and obesity

Chapters 5 and 6 investigated the hypothesis that the risk of developing OWOB in the early childhood years may depend on material circumstances of the families that children grow up in. This theory was confirmed by a strong inverse relationship that was observed between the educational level of caregivers and the risk of childhood OWOB. Notably, the inequalities seen between educational groups widened as the children got older, indicating that a potential lack of knowledge around OWOB and associated behaviours should be addressed through public health policies, such as through the effective provision of health information resources. Additionally, a slightly higher OWOB risk was seen in the lowest income group, which implies that access to healthy-weight promoting resources may be restricted and needs to be improved in most deprived families. The same need was seen for families in rural regions whose children tended to have a slightly elevated

BMI. No need for interventions in specific social classes or targeting neighbourhood characteristics (other than rurality) was seen in this study.

Hypothesis 3: Biological factors increase the risk of early childhood overweight and obesity

In Chapters 5 and 6, it was hypothesised that biological factors influence the individual OWOB risk of young children particularly in the earliest years. Strong evidence for this was seen in the GUI infant cohort, in which a high BWFGA, high gestational age, rapid infant weight gain, and children's previous OWOB status were most dominantly related to OWOB and high BMI. Additionally, the weight status of caregivers was positively associated with that of children and this relationship led to increasing discrepancies between children's OWOB risk over time. Variations in OWOB risk as well existed in ethnic groups, some of which could be explained by differences in lifestyles. Among lifestyle factors, frequent antibiotic use, short daily sleep duration and a high amount of screen time all increased the OWOB risk. Contrary to the expectations based on previous research, only small or inconclusive associations of OWOB were seen with infant feeding, later diet and physical activity. This may be explained by measurement and reporting biases that should be methodologically addressed in future studies. Only marginal differences existed between the OWOB risk of boys and girls; thus no need is seen for sex-specific prevention strategies. Prevention should rather target the population of young children and their families as a whole to intervene with the life-cycle of obesity.

Hypothesis 4: Psychosocial factors increase the risk of early overweight and obesity

The fourth hypothesis focused on psychosocial factors that may explain why young children gain excess weight, which was confirmed in Chapters 5 and 6. Children had a higher OWOB risk if they lived in lone caregiver families, and if they had no siblings. This relationship aggravated over time, suggesting that supports to lone caregiver families and tailored strategies for families with single children are

required at an early age in Ireland. These include supports to strengthen children's resilience after parental separation and increase their opportunities for active play, as well as awareness-raising to reach out to isolated families. Additionally, efforts are needed that promote healthy lifestyles in children attending informal childcare, as these children were at a higher OWOB risk than children in formal or home care.

Hypothesis 5: Different risk factors of obesity lead to excess weight gains at various time points in children's early years

Finally, it was hypothesised that material circumstances, biological factors and psychosocial factors do not have a consistent impact on young children's weight status, but influence changes in children's BMI differently over time. Evidence to verify this hypothesis presented in Chapter 6. Many biological factors persisted already in infancy and remained strong across the children's first five years of life. This implies that prevention of childhood obesity should already start in young couples before pregnancy, in order to reduce the prevalence of risk factors. Nevertheless, prevention efforts need to be maintained across and beyond the 'first 1000 days' of children's lives during which a high plasticity of weight gains was seen. As children move out of infancy, their individual lifestyle factors gain increased importance and hence should be addressed. Material and psychosocial mechanisms also increasingly influence children's weight gains after infancy. Importantly, some inequalities in BMI based on material circumstances were already visible by the time children reached the age of three years that only developed into discrepancies in OWOB levels by five years, further stressing that prevention has to start early, before these differences in OWOB occur.

While many risk factors before the age of three years should be targeted jointly on a population level, the establishment of equal opportunities for healthy weight development in all population groups may additionally require tailored interventions. By the age of five years, most BMI trajectories appear to be defined; however growth patterns in some ethnic groups only emerge at this late age. Additionally, children may only later become exposed to time-variant risk factors, such as living in a lone caregiver household, raising the need for continuous growth monitoring, multi-faceted prevention and weight management services in

Ireland beyond the age of three years. The Irish healthcare sector holds particular promise to address various factors throughout the young childhood ages.

In conclusion, the approach taken in this study of early childhood OWOB in Ireland appeared useful to identify multiple risk factors in the environments of young children that are involved in the development of early childhood OWOB and contribute to the high prevalence seen in Ireland. Even though the prevalence of childhood OWOB may be declining in Ireland, social inequalities remain to be seen and disparities between children in different population groups became larger across the children's first five years of life in this thesis. From the age of three to five years, children with OWOB seem more likely to remain overweight or obese than before that age, and the higher level of plasticity in the earliest years may offer an opportunity to prevent later obesity. Where possible, prevention of childhood obesity should start before pregnancy, or as soon as possible during pregnancy when the contact with prenatal services is established. The multi-dimensionality of risk factors that were associated with children's excess weight gain needs to be acknowledged by policy-makers, researchers and healthcare providers alike and risk factors should be targeted simultaneously through various channels in the endeavour to lower OWOB levels in Ireland. Even if biological factors may partly explain inequalities in OWOB, it is likely that psychosocial and material factors contribute to some of these relationships, all of which should be given consideration when strategies to lower (inequalities in) early childhood OWOB are designed. The policy-sector has an important role to play in the development of (non-)obesogenic environments and a Health-in-All-Policies approach may be required to remove inequalities in childhood obesity. While the implementation of prevention programmes may demand significant investments initially, it has potential to prevent adverse lifelong consequences of childhood OWOB and associated costs to individuals, the health system and society in a large proportion of Irish children.

REFERENCES AND APPENDICES

References

1. Monasta L, Batty GD, Cattaneo A, Lutje V, Ronfani L, Van Lenthe FJ, et al. Early-life determinants of overweight and obesity: a review of systematic reviews. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2010;11(10):695-708.
2. Jabakhanji SB, Pavlova M, Groot W, Boland F, Biesma R. Social class variation, the effect of the economic recession and childhood obesity at 3 years of age in Ireland. European journal of public health. 2017;27(2):234-9.
3. Growing Up in Ireland NLSCI. Key findings: infant cohort (at 5 years) 2013.
4. Commission on Social Determinants of Health. Closing the gap in a generation: health equity through action on the social determinants of health. Final report of the Commission on Social Determinants of Health. Geneva, Switzerland: World Health Organization; 2008.
5. Global Burden of Disease Study 2015 (GBD 2015) Obesity and Overweight Prevalence 1980-2015 [Internet]. Institute for Health Metrics and Evaluation. 2017 [cited 2018 Jun 6]. Available from: http://ghdx.healthdata.org/record/global-burden-disease-study-2015-gbd-2015-obesity-and-overweight-prevalence-1980-2015.
6. Solar O, Irwin A. A conceptual framework for action on the social determinants of health. Discussion paper for the Commission on Social Determinants of Health. Geneva, Switzerland; 2007.
7. Health-related SDGs [Internet]. University of Washington. 2017 [cited 2018 Jun 7]. Available from: https://vizhub.healthdata.org/sdg/.
8. Reed DB, Patterson PJ, Wasserman N. Obesity in rural youth: looking beyond nutrition and physical activity. Journal of nutrition education and behavior. 2011;43(5):401-8.
9. Keaver L, Webber L, Dee A, Shiely F, Marsh T, Balanda K, et al. Application of the UK foresight obesity model in Ireland: the health and economic consequences of projected obesity trends in Ireland. PLoS One. 2013;8(11):e79827.
10. Simmonds M, Burch J, Llewellyn A, Griffiths C, Yang H, Owen C, et al. The use of measures of obesity in childhood for predicting obesity and the development of obesity-related diseases in adulthood: a systematic review and meta-analysis. Health technology assessment (Winchester, England). 2015;19(43):1-336.
11. Guh DP, Zhang W, Bansback N, Amarsi Z, Birmingham CL, Anis AH. The incidence of co-morbidities related to obesity and overweight: a systematic review and meta-analysis. BMC Public Health. 2009;9:88.
12. NCD Risk Factor Collaboration. Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults. The Lancet. 2017;390(10113):2627-42.
13. World Health Organization (WHO). Obesity and overweight 2016 [updated June 2016. Available from: http://www.who.int/mediacentre/factsheets/fs311/en/.
14. European Association for the Study of Obesity (EASO). Obesity Facts & Figures [updated 2013. Available from: http://easo.org/education-portal/obesity-facts-figures/.
15. G. B. D. Obesity Collaborators. Health Effects of Overweight and Obesity in 195 Countries over 25 Years. N Engl J Med. 2017;377(1):13-27.
16. Perry IJ, Millar SR, Balanda KP, Dee A, Bergin D, Carter L, et al. What are the estimated costs of childhood overweight and obesity on the island of Ireland? Cork, Ireland, : Safefood; 2017 November 2017.
17. Organisation for Economic Co-operation and Development. Obesity update. 2014 June.
18. de Onis M, Blossner M, Borghi E. Global prevalence and trends of overweight and obesity among preschool children. The American journal of clinical nutrition. 2010;92(5):1257-64.
19. Singh AS, Mulder C, Twisk JW, van Mechelen W, Chinapaw MJ. Tracking of childhood overweight into adulthood: a systematic review of the literature. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2008;9(5):474-88.

20. Brisbois TD, Farmer AP, McCargar LJ. Early markers of adult obesity: a review. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2012;13(4):347-67.
21. World Health Organization. Overweight and obesity - Adults aged 18+. Global Health Observatory (GHO) data2017.
22. Gallus S, Lugo A, Murisic B, Bosetti C, Boffetta P, La Vecchia C. Overweight and obesity in 16 European countries. European journal of nutrition. 2015;54(5):679-89.
23. Ipsos MRBI. Healthy Ireland survey 2017 - summary of findings. Dublin; 2017. Report No.: ISBN 978-1-4064-2965-7.
24. NCD Risk Factor Collaboration, editor Adult body-mass index. Evolution of BMI over time; 2017.
25. NCD Risk Factor Collaboration. Ireland. Country profile2017.
26. Hanson M, Godfrey KM, Lillycrop KA, Burdge GC, Gluckman PD. Developmental plasticity and developmental origins of non-communicable disease: theoretical considerations and epigenetic mechanisms. Progress in biophysics and molecular biology. 2011;106(1):272-80.
27. Zeiher J, Varnaccia G, Jordan S, Lange C. [What are the determinants of childhood obesity? : A literature review as part of the project "Nationwide Monitoring of Childhood Obesity Determinants"]. Bundesgesundheitsblatt, Gesundheitsforschung, Gesundheitsschutz. 2016;59(11):1465-75.
28. Geserick M, Vogel M, Gausche R, Lipek T, Spielau U, Keller E, et al. Acceleration of BMI in Early Childhood and Risk of Sustained Obesity. N Engl J Med. 2018;379(14):1303-12.
29. Howe LD, Tilling K, Galobardes B, Smith GD, Ness AR, Lawlor DA. Socioeconomic disparities in trajectories of adiposity across childhood. Int J Pediatr Obes. 2011;6(2-2):e144-53.
30. Bornhorst C, Tilling K, Russo P, Kourides Y, Michels N, Molnar D, et al. Associations between early body mass index trajectories and later metabolic risk factors in European children: the IDEFICS study. European journal of epidemiology. 2015.
31. Owen CG, Woolacott N, Bornhorst C. Associations between early body mass index trajectories and later metabolic risk factors in European children: the IDEFICS study. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2016;31(5):513-25.
32. Cheung PC, Cunningham SA, Narayan KM, Kramer MR. Childhood Obesity Incidence in the United States: A Systematic Review. Childhood obesity. 2016;12(1):1-11.
33. European Union. EU action plan on childhood obesity 2014-2020. 2014/02/24 ed2014.
34. Gardner DSL, Hosking J, Metcalf BS, Jeffery AN, Voss LD, Wilkin TJ. Contribution of Early Weight Gain to Childhood Overweight and Metabolic Health: A Longitudinal Study (EarlyBird 36). Pediatrics. 2009;123(1):e67-e73.
35. Department of Health. A healthy weight for Ireland: Obesity policy and action plan 2016 - 2025. In: Health Do, editor. Dublin: Stationary office; 2016.
36. Perry IJ, Whelton H, Harrington J, Cousins B. The heights and weights of Irish children from the post-war era to the Celtic tiger. Journal of epidemiology and community health. 2009;63(3):262-4.
37. O'Neill JL, McCarthy SN, Burke SJ, Hannon EM, Kiely M, Flynn A, et al. Prevalence of overweight and obesity in Irish school children, using four different definitions. European journal of clinical nutrition. 2007;61(6):743-51.
38. Keane E, Kearney PM, Perry IJ, Kelleher CC, Harrington JM. Trends and prevalence of overweight and obesity in primary school aged children in the Republic of Ireland from 2002-2012: a systematic review. BMC Public Health. 2014;14:974.
39. Growing Up in Ireland NLSCI. Key findings: infant cohort (at 3 years). 2011.
40. McCarthy EK, Chaoimh C, Murray DM, Hourihane JO, Kenny LC, Kiely M. Eating behaviour and weight status at 2 years of age: data from the Cork BASELINE Birth Cohort Study. European journal of clinical nutrition. 2015;69(12):1356-9.

41. Bel-Serrat S, Heinen MM, Murrin CM, Daly L, Mehegan J, Concannon M, et al. The Childhood Obesity Surveillance Initiative (COSI) in the Republic of Ireland: Findings from 2008, 2010, 2012 and 2015. Dublin: Health Service Executive; 2017.
42. Pulgaron ER. Childhood obesity: a review of increased risk for physical and psychological comorbidities. Clinical therapeutics. 2013;35(1):A18-32.
43. Llewellyn A, Simmonds M. Childhood obesity as a predictor of morbidity in adulthood: a systematic review and meta-analysis. 2016;17(1):56-67.
44. Park MH, Falconer C, Viner RM, Kinra S. The impact of childhood obesity on morbidity and mortality in adulthood: a systematic review. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2012;13(11):985-1000.
45. O'Connell J, Kieran P, Gorman K, Ahern T, Cawood TJ, O'Shea D. BMI > or = 50 kg/m2 is associated with a younger age of onset of overweight and a high prevalence of adverse metabolic profiles. Public health nutrition. 2010;13(7):1090-8.
46. Baird J, Fisher D, Lucas P, Kleijnen J, Roberts H, Law C. Being big or growing fast: systematic review of size and growth in infancy and later obesity. BMJ. 2005;331(7522):929.
47. Classen TJ, Thompson O. Genes and the intergenerational transmission of BMI and obesity. Economics & Human Biology. 2016;23:121-33.
48. Lau EY, Liu J, Archer E. Maternal weight gain in pregnancy and risk of obesity among offspring: a systematic review. 2014;2014:524939.
49. Eriksson JG, Sandboge S, Salonen MK, Kajantie E, Osmond C. Long-term consequences of maternal overweight in pregnancy on offspring later health: findings from the Helsinki Birth Cohort Study. Annals of medicine. 2014;46(6):434-8.
50. Juonala M, Magnussen CG, Berenson GS, Venn A, Burns TL, Sabin MA, et al. Childhood adiposity, adult adiposity, and cardiovascular risk factors. N Engl J Med. 2011;365(20):1876-85.
51. Singh GM, Danaei G, Farzadfar F, Stevens GA, Woodward M, Wormser D, et al. The age-specific quantitative effects of metabolic risk factors on cardiovascular diseases and diabetes: a pooled analysis. PLoS One. 2013;8(7):e65174.
52. Pearson-Stuttard J, Zhou B, Kontis V, Bentham J, Gunter MJ, Ezzati M. Retracted: Worldwide burden of cancer attributable to diabetes and high body-mass index: a comparative risk assessment. The Lancet Diabetes & Endocrinology. 2018;6(2):95-104.
53. Bluher M. Are metabolically healthy obese individuals really healthy? European journal of endocrinology. 2014;171(6):R209-19.
54. Fruehbeck G, Toplak H, Woodward E, Halford JCG, Yumuk V. Need for a paradigm shift in adult overweight and obesity management - an EASO position statement on a pressing public health, clinical and scientific challenge in Europe. Obesity facts. 2014;7(6):408-16.
55. World Health Organization Regional Office for Europe. European Food and Nutrition Action Plan 2015-2020. Copenhagen, Denmark; 2014 15-18 September 2014.
56. Institute for Health Metrics and Evaluation. Ireland. 2018 Jun 7.
57. Global BMIMC, Di Angelantonio E, Bhupathiraju Sh N, Wormser D, Gao P, Kaptoge S, et al. Body-mass index and all-cause mortality: individual-participant-data meta-analysis of 239 prospective studies in four continents. Lancet. 2016;388(10046):776-86.
58. Sanders RH, Han A, Baker JS, Cobley S. Childhood obesity and its physical and psychological co-morbidities: a systematic review of Australian children and adolescents. European journal of pediatrics. 2015;174(6):715-46.
59. Cote AT, Phillips AA, Harris KC, Sandor GG, Panagiotopoulos C, Devlin AM. Obesity and arterial stiffness in children: systematic review and meta-analysis. Arteriosclerosis, thrombosis, and vascular biology. 2015;35(4):1038-44.
60. Peplies J, Bornhorst C, Gunther K, Fraterman A, Russo P, Veidebaum T, et al. Longitudinal associations of lifestyle factors and weight status with insulin resistance (HOMA-IR) in preadolescent children: the large prospective cohort study IDEFICS. The international journal of behavioral nutrition and physical activity. 2016;13(1):97.

61. Friend A, Craig L, Turner S. The prevalence of metabolic syndrome in children: a systematic review of the literature. Metabolic syndrome and related disorders. 2013;11(2):71-80.
62. McEneny J, Blair S, Woodside JV, Murray L, Boreham C, Young IS. High-density lipoprotein subfractions display proatherogenic properties in overweight and obese children. Pediatr Res. 2013;74(3):279-83.
63. Moraes LI, Nicola TC, Jesus JS, Alves ER, Giovaninni NP, Marcato DG, et al. High blood pressure in children and its correlation with three definitions of obesity in childhood. Arquivos brasileiros de cardiologia. 2014;102(2):175-80.
64. Liu PC, Kieckhefer GM, Gau BS. A systematic review of the association between obesity and asthma in children. Journal of advanced nursing. 2013;69(7):1446-65.
65. Noal RB, Menezes AM, Macedo SE, Dumith SC. Childhood body mass index and risk of asthma in adolescence: a systematic review. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2011;12(2):93-104.
66. Verbeeten KC, Elks CE, Daneman D, Ong KK. Association between childhood obesity and subsequent Type 1 diabetes: a systematic review and meta-analysis. Diabetic medicine : a journal of the British Diabetic Association. 2011;28(1):10-8.
67. Liang J, Matheson BE, Kaye WH, Boutelle KN. Neurocognitive correlates of obesity and obesity-related behaviors in children and adolescents. International journal of obesity. 2014;38(4):494-506.
68. Andersen IG, Holm JC, Homoe P. Obstructive sleep apnea in obese children and adolescents, treatment methods and outcome of treatment - A systematic review. International journal of pediatric otorhinolaryngology. 2016;87:190-7.
69. Sikorski C, Luppa M, Luck T, Riedel-Heller SG. Weight stigma "gets under the skin"- evidence for an adapted psychological mediation framework: a systematic review. Obesity (Silver Spring, Md). 2015;23(2):266-76.
70. Guardabassi V, Mirisola A, Tomasetto C. How is weight stigma related to children's health-related quality of life? A model comparison approach. Quality of life research : an international journal of quality of life aspects of treatment, care and rehabilitation. 2018;27(1):173-83.
71. Bechard LJ, Rothpletz-Puglia P, Touger-Decker R, Duggan C, Mehta NM. Influence of obesity on clinical outcomes in hospitalized children: a systematic review. JAMA pediatrics. 2013;167(5):476-82.
72. Camargos AC, Mendonca VA, Andrade CA, Oliveira KS, Lacerda AC. Overweight and obese infants present lower cognitive and motor development scores than normal-weight peers. Research in developmental disabilities. 2016;59:410-6.
73. Organisation for Economic Co-operation and Development, European Union. Health at a Glance: Europe 2016 - State of health in the EU cycle. Paris; 2016 December 2016. Contract No.: W-01-16-980-EN-N
74. World Health Organization. Global Action Plan for the Prevention and Control of NCDs 2013-2020. Geneva, Switzerland; 2013.
75. Bray GA, Kim KK, Wilding JPH. Obesity: a chronic relapsing progressive disease process. A position statement of the World Obesity Federation. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2017.
76. Bornhorst C, Siani A, Tornaritis M, Molnar D, Lissner L, Regber S, et al. Potential selection effects when estimating associations between the infancy peak or adiposity rebound and later body mass index in children. International journal of obesity. 2017;41(4):518-26.
77. Ziyab AH, Karmaus W, Kurukulaaratchy RJ, Zhang H, Arshad SH. Developmental trajectories of Body Mass Index from infancy to 18 years of age: prenatal determinants and health consequences. Journal of epidemiology and community health. 2014;68(10):934-41.
78. Stuart B, Panico L. Early-childhood BMI trajectories: evidence from a prospective, nationally representative British cohort study. Nutrition & diabetes. 2016;6:e198.

79. Jones R, Jewell J, Saksena R, Ramos Salas X, Breda J. Overweight and Obesity in Children under 5 Years: Surveillance Opportunities and Challenges for the WHO European Region. Frontiers in Public Health. 2017;5(58).
80. Health Service Executive. Training programme for public health nruses and doctors - Growth monitoring module. In: National Growth Charts Implementation Group, editor. Dublin: Health Service Executive,; 2012.
81. Black M, Joseph V, Mott L, Maheswaran R. Increasing inequality in childhood obesity in primary schools in a northern English town. Public health. 2018;158:9-14.
82. Chung A, Backholer K, Wong E, Palermo C, Keating C, Peeters A. Trends in child and adolescent obesity prevalence in economically advanced countries according to socioeconomic position: a systematic review. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2016;17(3):276-95.
83. de Wilde JA, Meeuwsen RC, Middelkoop BJ. Growing ethnic disparities in prevalence of overweight and obesity in children 2–15 years in the Netherlands. European journal of public health. 2018:cky104-cky.
84. World Health Organization Regional Office for Europe, European Commission. Adolescent obesity and related behaviours: trends and inequalitites in the WHO European Region, 2002-2014. 2017.
85. Farrell C, McAvoy H, Wilde J, Combat Poverty Agency. Tackling health inequalities - an all-Ireland approach to social determinants. Dublin: Combat Poverty Agency/Institute of Public Health in Ireland; 2008. Contract No.: ISBN: 978-1-905485-63-5.
86. Solar O, Irwin A. A conceptual framework for action on the social determinants of health. Social Determinants of Health Discussion Paper 2 (Policy and Practice). Geneva, Switzerland; 2010 11/2010.
87. Staines A, Balanda KP, Barron S, Corcoran Y, Fahy L, Gallagher L, et al. Child Health Care in Ireland. The Journal of pediatrics. 2016;177S:S87-S106.
88. safefood. Food on a low income - Four households tell their story. 2011. Report No.: ISBN: 978-1-905767-19-9.
89. Davison KK, Birch LL. Childhood overweight: a contextual model and recommendations for future research. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2001;2(3):159-71.
90. Nybo Andersen A-M, Casas M. Birthcohorts.net.
91. Larsen PS, Kamper-Jorgensen M, Adamson A, Barros H, Bonde JP, Brescianini S, et al. Pregnancy and birth cohort resources in europe: a large opportunity for aetiological child health research. Paediatric and perinatal epidemiology. 2013;27(4):393-414.
92. McCrory C, Layte R. Breastfeeding and risk of overweight and obesity at nine-years of age. Soc Sci Med. 2012;75(2):323-30.
93. Shiely F, Ng H, Berkery EM, Murrin C, Kelleher C, Hayes K. The association between weight perception and BMI: report and measurement data from the growing up in Ireland cohort study of 9-year olds. Int J Obes. 2017;41.
94. Walsh B, Cullinan J. Decomposing socioeconomic inequalities in childhood obesity: evidence from Ireland. Economics and human biology. 2015;16:60-72.
95. Kelly GE, Murrin C, Viljoen K, O'Brien J, Kelleher C. Body mass index is associated with the maternal lines but height is heritable across family lines in the Lifeways Cross-Generation Cohort Study. BMJ open. 2014;4(12):e005732.
96. Li X, Kearney PM, Keane E, Harrington JM, Fitzgerald AP. levels and sociodemographic correlates of accelerometer-based physical activity in Irish children: a cross-sectional study. Journal of epidemiology and community health. 2017;71(6):521-7.
97. Hollywood E, Comiskey C, Begley T, Snel A, O'Sullivan K, Quirke M, et al. Measuring and modelling body mass index among a cohort of urban children living with disadvantage. Journal of advanced nursing. 2013;69(4):851-61.

98. Irish Universities Nutrition Alliance. National Pre-School Nutrition Survey - Summary Report. 2012 2018 Jun 14.
99. Quail A, Williams J, McCrory C, Murray A, Thornton M. A summary guide to wave 1 of the infant cohort (at 9 months) of Growing Up in Ireland. Dublin: The Economic and Social Research Institute, Office of the Minister for Children and Youth Affairs, University of Dublin Trinity College; 2011 4/2011.
100. Health Service Executive. HSE Framework for Action on Obesity 2008-2012. 2008.
101. Bronfenbrenner U. The ecology of human development: Experiment by nature and design. Cambridge: Harvard University Press; 1979.
102. Bronfenbrenner U. Making human beings human: Bioecological perspectives on human development. Thousand Oaks, California: Sage; 2005.
103. Bronfenbrenner U, Evans GW. Developmental science in the 21st Century: Emerging questions, theoretical models, research designs and empirical findings. Social Development. 2000;9(1).
104. Harrison K, Bost KK, McBride BA, et al. Towards Developmental Conceptualization of Contributors to Overweight and Obesity in Childhood: The Six-Cs Model. Child Development Perspectives. 20
105. Lindsay AC, Greaney ML, Wallington SF, Mesa T, Salas CF. A review of early influences on physical activity and sedentary behaviors of preschool-age children in high-income countries. Journal for specialists in pediatric nursing : JSPN. 2017;22(3).
106. Pardo BM, Bengoechea EG, Julian Clemente JA, Lanaspa EG. Empowering adolescents to be physically active: three-year results of the Sigue la Huella intervention. Preventive medicine. 2014;66:6-11.
107. Pfeiffer KA, Saunders RP, Brown WH, Dowda M, Addy CL, Pate RR. Study of Health and Activity in Preschool Environments (SHAPES): study protocol for a randomized trial evaluating a multi-component physical activity intervention in preschool children. BMC Public Health. 2013;13:728.
108. Garcia JM, Healy S, Rice D. The Individual, Social, and Environmental Correlates of Physical Activity and Screen Time in Irish Children: Growing Up in Ireland Study. Journal of physical activity & health. 2016;13(12):1285-93.
109. Perez-Escamilla R, Kac G. Childhood obesity prevention: a life-course framework. International journal of obesity supplements. 2013;3(Suppl 1):S3-s5.
110. Dwyer J, Needham L, Simpson JR, Heeney ES. Parents report intrapersonal, interpersonal, and environmental barriers to supporting healthy eating and physical activity among their preschoolers. Applied physiology, nutrition, and metabolism = Physiologie appliquee, nutrition et metabolisme. 2008;33(2):338-46.
111. Ohri-Vachaspati P, DeLia D, DeWeese RS, Crespo NC, Todd M, Yedidia MJ. The relative contribution of layers of the Social Ecological Model to childhood obesity. Public health nutrition. 2015;18(11):2055-66.
112. Kellou N, Sandalinas F, Copin N, Simon C. Prevention of unhealthy weight in children by promoting physical activity using a socio-ecological approach: what can we learn from intervention studies? Diabetes & metabolism. 2014;40(4):258-71.
113. Hall D, Elliman D. Health for all children. fourth edition (revised) ed. New York, United States: Oxford University Press Inc.; 2010.
114. de Onis M, Onyango AW, Borghi E, Siyam A, Nishida C, Siekmann J. Development of a WHO growth reference for school-aged children and adolescents. Bulletin of the World Health Organization. 2007;85(9):660-7.
115. Simmonds M, Llewellyn A, Owen CG, Woolacott N. Simple tests for the diagnosis of childhood obesity: a systematic review and meta-analysis. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2016;17(12):1301-15.
116. Rolland-Cachera MF, Akrout M, Peneau S. History and meaning of the body mass index. Interest of other anthropometric measurements. [cited 2018 Feb 2]. In: Childhood Obesity eBook

[Internet]. Brussels, Belgium: European Childhood Obesity Group ASBL, [cited 2018 Feb 2]. Available from: http://ebook.ecog-obesity.eu/chapter-growth-charts-body-composition/history-meaning-body-mass-index-interest-anthropometric-measurements/.
117. Lissauer T, Carroll W. Illustrated textbook of paediatrics. fifth edition ed: Elsevier Limited; 2018.
118. Gibson E, Nawab U. Growth Parameters in Neonates. Merck Manual Professional Version. Kenilworth, NJ, USA: Merck Sharp & Dohme Corp.; 2015.
119. Institute of Medicine. Weight gain during pregnancy: reexamining the guidelines. Washington (DC); 2009. Report No.: 9780309131131.
120. Woo Baidal JA, Locks LM, Cheng ER, Blake-Lamb TL, Perkins ME, Taveras EM. Risk Factors for Childhood Obesity in the First 1,000 Days: A Systematic Review. American journal of preventive medicine. 2016;50(6):761-79.
121. Graber EG. Physical Growth of Infants and Children. Merck Manual Provessional Version. Kenilworth, NJ, USA: Merck Sharp & Dohme Corp.; 2016.
122. Hutcheon JA, Jacobsen GW, Kramer MS, Martinussen M, Platt RW. Small Size at Birth or Abnormal Intrauterine Growth Trajectory: Which Matters More for Child Growth? American journal of epidemiology. 2016;183(12):1107-13.
123. Gelbaya TA, Nardo LG. Customised fetal growth chart: a systematic review. Journal of obstetrics and gynaecology : the journal of the Institute of Obstetrics and Gynaecology. 2005;25(5):445-50.
124. Kim SY, Sharma AJ, Sappenfield W, Wilson HG, Salihu HM. Association of maternal body mass index, excessive weight gain, and gestational diabetes mellitus with large-for-gestational-age births. Obstetrics and gynecology. 2014;123(4):737-44.
125. Yan J. Maternal pre-pregnancy BMI, gestational weight gain, and infant birth weight: A within-family analysis in the United States. Economics and human biology. 2015;18:1-12.
126. Lutsiv O, Mah J, Beyene J, McDonald SD. The effects of morbid obesity on maternal and neonatal health outcomes: a systematic review and meta-analyses. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2015;16(7):531-46.
127. Rasmussen KM, Abrams B, Bodnar LM, Butte NF, Catalano PM, Maria Siega-Riz A. Recommendations for weight gain during pregnancy in the context of the obesity epidemic. Obstetrics and gynecology. 2010;116(5):1191-5.
128. Spracklen CN, Ryckman KK, Harland K, Saftlas AF. Effects of smoking and preeclampsia on birth weight for gestational age. The journal of maternal-fetal & neonatal medicine : the official journal of the European Association of Perinatal Medicine, the Federation of Asia and Oceania Perinatal Societies, the International Society of Perinatal Obstet. 2015;28(6):679-84.
129. de Santis MS, Taricco E, Radaelli T, Spada E, Rigano S, Ferrazzi E, et al. Growth of fetal lean mass and fetal fat mass in gestational diabetes. Ultrasound in obstetrics & gynecology : the official journal of the International Society of Ultrasound in Obstetrics and Gynecology. 2010;36(3):328-37.
130. Diabetes Ireland. Gestational Diabetes [updated 2017. Available from: https://www.diabetes.ie/living-with-diabetes/diabetes-pregnancy/gestational-diabetes/.
131. Lagerros YT, Cnattingius S, Granath F, Hanson U, Wikstrom AK. From infancy to pregnancy: birth weight, body mass index, and the risk of gestational diabetes. European journal of epidemiology. 2012;27(10):799-805.
132. Rayfield S, Plugge E. Systematic review and meta-analysis of the association between maternal smoking in pregnancy and childhood overweight and obesity. Journal of epidemiology and community health. 2017;71(2):162-73.
133. Suzuki K, Sato M, Zheng W, Shinohara R, Yokomichi H, Yamagata Z. Effect of maternal smoking cessation before and during early pregnancy on fetal and childhood growth. Journal of epidemiology / Japan Epidemiological Association. 2014;24(1):60-6.

134. Silventoinen K, Rokholm B, Kaprio J, Sorensen TI. The genetic and environmental influences on childhood obesity: a systematic review of twin and adoption studies. International journal of obesity. 2010;34(1):29-40.
135. Lee YS. The role of leptin-melanocortin system and human weight regulation: lessons from experiments of nature. Annals of the Academy of Medicine, Singapore. 2009;38(1):34-11.
136. Razquin C, Marti A, Martinez JA. Evidences on three relevant obesogenes: MC4R, FTO and PPARgamma. Approaches for personalized nutrition. Molecular nutrition & food research. 2011;55(1):136-49.
137. Willer CJ, Speliotes EK, Loos RJ, Li S, Lindgren CM, Heid IM, et al. Six new loci associated with body mass index highlight a neuronal influence on body weight regulation. Nature genetics. 2009;41(1):25-34.
138. Martinez JA, Parra MD, Santos JL, Moreno-Aliaga MJ, Marti A, Martinez-Gonzalez MA. Genotype-dependent response to energy-restricted diets in obese subjects: towards personalized nutrition. Asia Pacific journal of clinical nutrition. 2008;17 Suppl 1:119-22.
139. Hebebrand J, Hinney A. Environmental and genetic risk factors in obesity. Child and adolescent psychiatric clinics of North America. 2009;18(1):83-94.
140. Bradfield JP, Taal HR, Timpson NJ, Scherag A, Lecoeur C, Warrington NM, et al. A genome-wide association meta-analysis identifies new childhood obesity loci. Nature genetics. 2012;44(5):526-31.
141. Qi Q, Downer MK, Kilpelainen TO, Taal HR, Barton SJ, Ntalla I, et al. Dietary Intake, FTO Genetic Variants, and Adiposity: A Combined Analysis of Over 16,000 Children and Adolescents. Diabetes. 2015;64(7):2467-76.
142. Loos RJ, Yeo GS. The bigger picture of FTO: the first GWAS-identified obesity gene. Nature reviews Endocrinology. 2014;10(1):51-61.
143. Bordoni L, Marchegiani F, Piangerelli M, Napolioni V, Gabbianelli R. Obesity-related genetic polymorphisms and adiposity indices in a young Italian population. IUBMB life. 2017.
144. Lazopoulou N, Gkioka E, Ntalla I, Pervanidou P, Magiakou AM, Roma-Giannikou E, et al. The combined effect of MC4R and FTO risk alleles on childhood obesity in Greece. Hormones (Athens, Greece). 2015;14(1):126-33.
145. Berger SL, Kouzarides T, Shiekhattar R, Shilatifard A. An operational definition of epigenetics. Genes & Development. 2009;23(7):781-3.
146. Demetriou CA, van Veldhoven K, Relton C, Stringhini S, Kyriacou K, Vineis P. Biological embedding of early-life exposures and disease risk in humans: a role for DNA methylation. European journal of clinical investigation. 2015;45(3):303-32.
147. Ino T. Maternal smoking during pregnancy and offspring obesity: meta-analysis. Pediatrics international : official journal of the Japan Pediatric Society. 2010;52(1):94-9.
148. McCormick DP, Sarpong K, Jordan L, Ray LA, Jain S. Infant obesity: are we ready to make this diagnosis? The Journal of pediatrics. 2010;157(1):15-9.
149. Sparano S, Ahrens W, De Henauw S, Marild S, Molnar D, Moreno LA, et al. Being macrosomic at birth is an independent predictor of overweight in children: results from the IDEFICS study. Maternal and child health journal. 2013;17(8):1373-81.
150. Farfel A, Derazne E, Tzur D, Linder N, Laron Z. Anthropometric indices of adolescents who at birth were full-term long and/or overweight for gestational age. The Israel Medical Association journal : IMAJ. 2012;14(2):93-5.
151. Leonard SA, Rasmussen KM, King JC, Abrams B. Trajectories of maternal weight from before pregnancy through postpartum and associations with childhood obesity. The American journal of clinical nutrition. 2017;106(5):1295-301.
152. Yu Z, Han S, Zhu J, Sun X, Ji C, Guo X. Pre-pregnancy body mass index in relation to infant birth weight and offspring overweight/obesity: a systematic review and meta-analysis. PLoS One. 2013;8(4):e61627.

153. Bornhorst C, Siani A, Russo P, Kourides Y, Sion I, Molnar D, et al. Early Life Factors and Inter-Country Heterogeneity in BMI Growth Trajectories of European Children: The IDEFICS Study. PLoS One. 2016;11(2):e0149268.
154. Patro B, Liber A, Zalewski B, Poston L, Szajewska H, Koletzko B. Maternal and paternal body mass index and offspring obesity: a systematic review. Annals of nutrition & metabolism. 2013;63(1-2):32-41.
155. Gaillard R, Steegers EA, Duijts L, Felix JF, Hofman A, Franco OH, et al. Childhood cardiometabolic outcomes of maternal obesity during pregnancy: the Generation R Study. Hypertension. 2014;63(4):683-91.
156. Mamun AA, Mannan M, Doi SA. Gestational weight gain in relation to offspring obesity over the life course: a systematic review and bias-adjusted meta-analysis. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2014;15(4):338-47.
157. O'Reilly JR, Reynolds RM. The risk of maternal obesity to the long-term health of the offspring. Clinical endocrinology. 2013;78(1):9-16.
158. Poston L. Maternal obesity, gestational weight gain and diet as determinants of offspring long term health. Best practice & research Clinical endocrinology & metabolism. 2012;26(5):627-39.
159. Taal HR, Vd Heijden AJ, Steegers EA, Hofman A, Jaddoe VW. Small and large size for gestational age at birth, infant growth, and childhood overweight. Obesity (Silver Spring, Md). 2013;21(6):1261-8.
160. Taveras EM, Rifas-Shiman SL, Sherry B, Oken E, Haines J, Kleinman K, et al. Crossing growth percentiles in infancy and risk of obesity in childhood. Archives of pediatrics & adolescent medicine. 2011;165(11):993-8.
161. Dulloo AG, Jacquet J, Seydoux J, Montani JP. The thrifty 'catch-up fat' phenotype: its impact on insulin sensitivity during growth trajectories to obesity and metabolic syndrome. International journal of obesity. 2006;30 Suppl 4:S23-35.
162. Karaolis-Danckert N, Buyken AE, Kulig M, Kroke A, Forster J, Kamin W, et al. How pre- and postnatal risk factors modify the effect of rapid weight gain in infancy and early childhood on subsequent fat mass development: results from the Multicenter Allergy Study 90. The American journal of clinical nutrition. 2008;87(5):1356-64.
163. Li N, Liu E, Guo J, Pan L, Li B, Wang P, et al. Maternal prepregnancy body mass index and gestational weight gain on offspring overweight in early infancy. PLoS One. 2013;8(10):e77809.
164. Rolland-Cachera MF, Peneau S. Assessment of growth: variations according to references and growth parameters used. The American journal of clinical nutrition. 2011;94(6 Suppl):1794S-8S.
165. van Dijk CE, Innis SM. Growth-curve standards and the assessment of early excess weight gain in infancy. Pediatrics. 2009;123(1):102-8.
166. Crume TL, Ogden L, Maligie M, Sheffield S, Bischoff KJ, McDuffie R, et al. Long-term impact of neonatal breastfeeding on childhood adiposity and fat distribution among children exposed to diabetes in utero. Diabetes care. 2011;34(3):641-5.
167. Lefebvre CM, John RM. The effect of breastfeeding on childhood overweight and obesity: a systematic review of the literature. Journal of the American Association of Nurse Practitioners. 2014;26(7):386-401.
168. Rzehak P, Oddy WH, Mearin ML, Grote V, Mori TA, Szajewska H, et al. Infant feeding and growth trajectory patterns in childhood and body composition in young adulthood. The American journal of clinical nutrition. 2017.
169. Prentice P, Ong KK, Schoemaker MH, van Tol EA, Vervoort J, Hughes IA, et al. Breast milk nutrient content and infancy growth. Acta paediatrica. 2016;105(6):641-7.
170. Nguo K, Walker KZ, Bonham MP, Huggins CE. Systematic review and meta-analysis of the effect of meal intake on postprandial appetite-related gastrointestinal hormones in obese children. International journal of obesity. 2016;40(4):555-63.

171. Savino F, Liguori SA, Fissore MF, Oggero R. Breast milk hormones and their protective effect on obesity. International journal of pediatric endocrinology. 2009;2009:327505.
172. Bartok CJ, Ventura AK. Mechanisms underlying the association between breastfeeding and obesity. Int J Pediatr Obes. 2009;4(4):196-204.
173. Rolland-Cachera MF, Akrout M, Peneau S. Nutrient Intakes in Early Life and Risk of Obesity. International journal of environmental research and public health. 2016;13(6).
174. Alberdi G, McNamara AE, Lindsay KL, Scully HA, Horan MH, Gibney ER, et al. The association between childcare and risk of childhood overweight and obesity in children aged 5 years and under: a systematic review. European journal of pediatrics. 2016;175(10):1277-94.
175. World Health Organization (WHO). Infant and young child feeding - model chapter for textbooks for medical students and allied health professionals. Geneva, Switzerland: WHO Press; 2009.
176. Wang J, Wu Y, Xiong G, Chao T, Jin Q, Liu R, et al. Introduction of complementary feeding before 4months of age increases the risk of childhood overweight or obesity: a meta-analysis of prospective cohort studies. Nutrition research (New York, NY). 2016;36(8):759-70.
177. Pearce J, Taylor MA, Langley-Evans SC. Timing of the introduction of complementary feeding and risk of childhood obesity: a systematic review. International journal of obesity. 2013;37(10):1295-306.
178. Hughes AR, Sherriff A, Ness AR, Reilly JJ. Timing of adiposity rebound and adiposity in adolescence. Pediatrics. 2014;134(5):e1354-61.
179. Pearce J, Langley-Evans SC. The types of food introduced during complementary feeding and risk of childhood obesity: a systematic review. International journal of obesity. 2013;37(4):477-85.
180. Lim GH, Toh JY, Aris IM, Chia AR, Han WM, Saw SM, et al. Dietary Pattern Trajectories from 6 to 12 Months of Age in a Multi-Ethnic Asian Cohort. Nutrients. 2016;8(6).
181. Perez-Morales E, Bacardi-Gascon M, Jimenez-Cruz A. Sugar-sweetened beverage intake before 6 years of age and weight or BMI status among older children; systematic review of prospective studies. Nutricion hospitalaria. 2013;28(1):47-51.
182. Svensson A, Larsson C, Eiben G, Lanfer A, Pala V, Hebestreit A, et al. European children's sugar intake on weekdays versus weekends: the IDEFICS study. European journal of clinical nutrition. 2014;68(7):822-8.
183. Garcia AL, Raza S, Parrett A, Wright CM. Nutritional content of infant commercial weaning foods in the UK. Arch Dis Child. 2013;98(10):793-7.
184. Patro-Golab B, Zalewski BM, Kolodziej M, Kouwenhoven S, Poston L, Godfrey KM, et al. Nutritional interventions or exposures in infants and children aged up to 3 years and their effects on subsequent risk of overweight, obesity and body fat: a systematic review of systematic reviews. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2016;17(12):1245-57.
185. Hornell A, Lagstrom H, Lande B, Thorsdottir I. Protein intake from 0 to 18 years of age and its relation to health: a systematic literature review for the 5th Nordic Nutrition Recommendations. Food & nutrition research. 2013;57.
186. Campbell KJ, Abbott G, Zheng M, McNaughton SA. Early Life Protein Intake: Food Sources, Correlates, and Tracking across the First 5 Years of Life. Journal of the Academy of Nutrition and Dietetics. 2017;117(8):1188-97 e1.
187. Greve B, Pigeot I, Huybrechts I, Pala V, Bornhorst C. A comparison of heuristic and model-based clustering methods for dietary pattern analysis. Public health nutrition. 2016;19(2):255-64.
188. Pala V, Lissner L, Hebestreit A, Lanfer A, Sieri S, Siani A, et al. Dietary patterns and longitudinal change in body mass in European children: a follow-up study on the IDEFICS multicenter cohort. European journal of clinical nutrition. 2013;67(10):1042-9.
189. Gonzalez-Gil EM, Santabarbara J, Russo P, Ahrens W, Claessens M, Lissner L, et al. Food intake and inflammation in European children: the IDEFICS study. European journal of nutrition. 2016;55(8):2459-68.

190. Dogra S, Sakwinska O, Soh SE, Ngom-Bru C, Bruck WM, Berger B, et al. Dynamics of infant gut microbiota are influenced by delivery mode and gestational duration and are associated with subsequent adiposity. mBio. 2015;6(1).
191. Vael C, Verhulst SL, Nelen V, Goossens H, Desager KN. Intestinal microflora and body mass index during the first three years of life: an observational study. Gut pathogens. 2011;3(1):8.
192. Scheepers LE, Penders J, Mbakwa CA, Thijs C, Mommers M, Arts IC. The intestinal microbiota composition and weight development in children: the KOALA Birth Cohort Study. International journal of obesity. 2015;39(1):16-25.
193. Freeland KR, Wolever TM. Acute effects of intravenous and rectal acetate on glucagon-like peptide-1, peptide YY, ghrelin, adiponectin and tumour necrosis factor-alpha. The British journal of nutrition. 2010;103(3):460-6.
194. Backhed F, Manchester JK, Semenkovich CF, Gordon JI. Mechanisms underlying the resistance to diet-induced obesity in germ-free mice. Proceedings of the National Academy of Sciences of the United States of America. 2007;104(3):979-84.
195. Koleva PT, Bridgman SL, Kozyrskyj AL. The Infant Gut Microbiome: Evidence for Obesity Risk and Dietary Intervention. Nutrients. 2015;7(4):2237-60.
196. Payne AN, Chassard C, Lacroix C. Gut microbial adaptation to dietary consumption of fructose, artificial sweeteners and sugar alcohols: implications for host-microbe interactions contributing to obesity. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2012;13(9):799-809.
197. Laforest-Lapointe I, Arrieta MC. Patterns of Early-Life Gut Microbial Colonization during Human Immune Development: An Ecological Perspective. Frontiers in immunology. 2017;8:788.
198. Penders J, Thijs C, Vink C, Stelma FF, Snijders B, Kummeling I, et al. Factors influencing the composition of the intestinal microbiota in early infancy. Pediatrics. 2006;118(2):511-21.
199. Shao X, Ding X, Wang B, Li L, An X, Yao Q, et al. Antibiotic Exposure in Early Life Increases Risk of Childhood Obesity: A Systematic Review and Meta-Analysis. Frontiers in endocrinology. 2017;8:170.
200. World Health Organization (WHO). Global Recommendations on Physical Activity for Health. Geneva; 2010. Report No.: 978 92 4 159 997 9.
201. Konstabel K, Veidebaum T, Verbestel V, Moreno LA, Bammann K, Tornaritis M, et al. Objectively measured physical activity in European children: the IDEFICS study. International journal of obesity. 2014;38 Suppl 2:S135-43.
202. te Velde SJ, van Nassau F, Uijtdewilligen L, van Stralen MM, Cardon G, De Craemer M, et al. Energy balance-related behaviours associated with overweight and obesity in preschool children: a systematic review of prospective studies. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2012;13 Suppl 1:56-74.
203. Pate RR, O'Neill JR, Liese AD, Janz KF, Granberg EM, Colabianchi N, et al. Factors associated with development of excessive fatness in children and adolescents: a review of prospective studies. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2013;14(8):645-58.
204. Thivel D, Rumbold PL, King NA, Pereira B, Blundell JE, Mathieu ME. Acute post-exercise energy and macronutrient intake in lean and obese youth: a systematic review and meta-analysis. International journal of obesity. 2016;40(10):1469-79.
205. Gori D, Guaraldi F, Cinocca S, Moser G, Rucci P, Fantini MP. Effectiveness of educational and lifestyle interventions to prevent paediatric obesity: systematic review and meta-analyses of randomized and non-randomized controlled trials. Obes Sci Pract. 2017;3(3):235-48.
206. Pratt KJ, Van Fossen C, Cotto-Maisonet J, Palmer EN, Eneli I. Mothers' Perspectives on the Development of Their Preschoolers' Dietary and Physical Activity Behaviors and Parent-Child Relationship: Implications for Pediatric Primary Care Physicians. Clinical pediatrics. 2017;56(8):766-75.
207. O'Connor SG, Maher JP, Belcher BR, Leventhal AM, Margolin G, Shonkoff ET, et al. Associations of maternal stress with children's weight-related behaviours: a systematic literature

review. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2017;18(5):514-25.
208. van Ekris E, Altenburg TM, Singh AS, Proper KI, Heymans MW, Chinapaw MJM. An evidence-update on the prospective relationship between childhood sedentary behaviour and biomedical health indicators: a systematic review and meta-analysis. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2017;18(6):712-4.
209. American Academy of Pediatrics, Council on Communications and Media. Media and Young Minds. Pediatrics. 2016.
210. Marsh S, Ni Mhurchu C, Maddison R. The non-advertising effects of screen-based sedentary activities on acute eating behaviours in children, adolescents, and young adults. A systematic review. Appetite. 2013;71:259-73.
211. Lissner L, Lanfer A, Gwozdz W, Olafsdottir S, Eiben G, Moreno LA, et al. Television habits in relation to overweight, diet and taste preferences in European children: the IDEFICS study. European journal of epidemiology. 2012;27(9):705-15.
212. Ford C, Ward D, White M. Television viewing associated with adverse dietary outcomes in children ages 2-6. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2012;13(12):1139-47.
213. Magee C, Caputi P, Iverson D. Lack of sleep could increase obesity in children and too much television could be partly to blame. Acta paediatrica. 2014;103(1):e27-31.
214. Bornhorst C, Hense S, Ahrens W, Hebestreit A, Reisch L, Barba G, et al. From sleep duration to childhood obesity--what are the pathways? European journal of pediatrics. 2012;171(7):1029-38.
215. Ruan H, Xun P, Cai W, He K, Tang Q. Habitual Sleep Duration and Risk of Childhood Obesity: Systematic Review and Dose-response Meta-analysis of Prospective Cohort Studies. Scientific reports. 2015;5:16160.
216. Cespedes EM, Hu FB, Redline S, Rosner B, Gillman MW, Rifas-Shiman SL, et al. Chronic insufficient sleep and diet quality: Contributors to childhood obesity. Obesity (Silver Spring, Md). 2016;24(1):184-90.
217. Hirshkowitz M, Whiton K, Albert SM, Alessi C, Bruni O, DonCarlos L, et al. National Sleep Foundation’s sleep time duration recommendations: methodology and results summary. Sleep Health: Journal of the National Sleep Foundation.1(1):40-3.
218. Taveras EM, Rifas-Shiman SL, Oken E, Gunderson EP, Gillman MW. Short sleep duration in infancy and risk of childhood overweight. Archives of pediatrics & adolescent medicine. 2008;162(4):305-11.
219. Chen X, Beydoun MA, Wang Y. Is sleep duration associated with childhood obesity? A systematic review and meta-analysis. Obesity. 2008;16(265-74).
220. Felso R, Lohner S, Hollody K, Erhardt E, Molnar D. Relationship between sleep duration and childhood obesity: Systematic review including the potential underlying mechanisms. Nutrition, metabolism, and cardiovascular diseases : NMCD. 2017;27(9):751-61.
221. Norman A, Berlin A, Sundblom E, Elinder LS, Nyberg G. Stuck in a vicious circle of stress. Parental concerns and barriers to changing children's dietary and physical activity habits. Appetite. 2015;87:137-42.
222. Sleddens EFC, Gubbels JS, Kremers SPJ, van der Plas E, Thijs C. Bidirectional associations between activity-related parenting practices, and child physical activity, sedentary screen-based behavior and body mass index: a longitudinal analysis. The international journal of behavioral nutrition and physical activity. 2017;14(1):89.
223. Nieman P, LeBlanc C, Canadian Paediatric Society, Healthy Active Living Committee. Psychosocial aspects of child and adolescent obesity. Paediatrics & child health. 2012;17(3):205-6.
224. Gray LA, Hernandez Alava M, Kelly MP, Campbell MJ. Family lifestyle dynamics and childhood obesity: evidence from the millennium cohort study. BMC Public Health. 2018;18(1):500.

225. Chen AY, Escarce JJ. Family structure and childhood obesity: an analysis through 8th grade. Maternal and child health journal. 2014;18(7):1772-7.
226. Sisson SB, Sheffield-Morris A, Spicer P, Lora K, Latorre C. Influence of family structure on obesogenic behaviors and placement of bedroom TVs of American children: National Survey of Children's Health 2007. Preventive medicine. 2014;61:48-53.
227. Moore TG, McDonald M, Carlon L, O'Rourke K. Early childhood development and the social determinants of health inequities. Health promotion international. 2015;30(suppl_2):ii102-ii15.
228. Bzostek SH, Beck AN. Familial instability and young children's physical health. Soc Sci Med. 2011;73(2):282-92.
229. Harrist AW, Topham GL, Hubbs-Tait L, Shriver LH, Swindle TM. Psychosocial Factors in Children's Obesity: Examples From An Innovative Line of Inquiry. Child Development Perspectives. 2017;11(4):275-81.
230. Walton K, Simpson JR, Darlington G, Haines J. Parenting stress: a cross-sectional analysis of associations with childhood obesity, physical activity, and TV viewing. BMC pediatrics. 2014;14:244.
231. Jarman M, Inskip HM, Ntani G, Cooper C, Baird J, Robinson SM, et al. Influences on the diet quality of pre-school children: importance of maternal psychological characteristics. Public health nutrition. 2015;18(11):2001-10.
232. Bel-Serrat S, Heinen MM, Mehegan J, O'Brien S, Eldin N, Murrin CM, et al. School sociodemographic characteristics and obesity in schoolchildren: does the obesity definition matter? BMC Public Health. 2018;18(1):337.
233. MacMahon B, Weld G. The cost of a minimum essential food basket in the Republic of Ireland. Study for six household types. Dublin: Safefood; 2015. Contract No.: ISBN: 978-1-905767-56-4.
234. Barriuso L, Miqueleiz E, Albaladejo R, Villanueva R, Santos JM, Regidor E. Socioeconomic position and childhood-adolescent weight status in rich countries: a systematic review, 1990-2013. BMC pediatrics. 2015;15:129.
235. World Health Organization Regional Office for Europe. Obesity and inequities - Guidance for addressing inequities in overweight and obesity. Copenhagen: World Health Organization; 2014.
236. Caprio S, Daniels SR, Drewnowski A, Kaufman FR, Palinkas LA, Rosenbloom AL, et al. Influence of race, ethnicity, and culture on childhood obesity: implications for prevention and treatment: a consensus statement of Shaping America's Health and the Obesity Society. Diabetes care. 2008;31(11):2211-21.
237. El-Sayed AM, Scarborough P, Galea S. Socioeconomic inequalities in childhood obesity in the United Kingdom: a systematic review of the literature. Obesity facts. 2012;5(5):671-92.
238. Layte R, Bennett A, McCrory C, Kearney J. Social class variation in the predictors of rapid growth in infancy and obesity at age 3 years. International journal of obesity. 2014;38(1):82-90.
239. Ahrens W, Pigeot I, Pohlabeln H, De Henauw S, Lissner L, Molnar D, et al. Prevalence of overweight and obesity in European children below the age of 10. International journal of obesity. 2014;38 Suppl 2:S99-107.
240. Lissner L, Wijnhoven TM, Mehlig K, Sjoberg A, Kunesova M, Yngve A, et al. Socioeconomic inequalities in childhood overweight: heterogeneity across five countries in the WHO European Childhood Obesity Surveillance Initiative (COSI-2008). International journal of obesity. 2016;40(5):796-802.
241. Ruijsbroek A, Wijga AH, Kerkhof M, Koppelman GH, Smit HA, Droomers M. The development of socio-economic health differences in childhood: results of the Dutch longitudinal PIAMA birth cohort. BMC Public Health. 2011;11:225.
242. Schrempft S, van Jaarsveld CH, Fisher A, Fildes A, Wardle J. Maternal characteristics associated with the obesogenic quality of the home environment in early childhood. Appetite. 2016;107:392-7.

243. Black L, Matvienko-Sikar K, Kearney PM. The association between childcare arrangements and risk of overweight and obesity in childhood: a systematic review. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2017.
244. Nuffield Foundation. The role of informal childcare: a synthesis and critical review of the evidence. 2012.
245. Aggio D, Smith L, Fisher A, Hamer M. Mothers' perceived proximity to green space is associated with TV viewing time in children: the Growing Up in Scotland study. Preventive medicine. 2015;70:46-9.
246. Kikuya M, Matsubara H, Ishikuro M, Sato Y, Obara T, Metoki H, et al. Alterations in physique among young children after the Great East Japan Earthquake: Results from a nationwide survey. Journal of epidemiology / Japan Epidemiological Association. 2017;27(10):462-8.
247. Johnson JA, 3rd, Johnson AM. Urban-rural differences in childhood and adolescent obesity in the United States: a systematic review and meta-analysis. Childhood obesity. 2015;11(3):233-41.
248. Gose M, Plachta-Danielzik S, Willie B, Johannsen M, Landsberg B, Muller MJ. Longitudinal influences of neighbourhood built and social environment on children's weight status. International journal of environmental research and public health. 2013;10(10):5083-96.
249. Li X, Memarian E, Sundquist J, Zoller B, Sundquist K. Neighbourhood deprivation, individual-level familial and socio-demographic factors and diagnosed childhood obesity: a nationwide multilevel study from Sweden. Obesity facts. 2014;7(4):253-63.
250. Ferrao MM, Gama A, Marques VR, Mendes LL, Mourao I, Nogueira H, et al. Association between parental perceptions of residential neighbourhood environments and childhood obesity in Porto, Portugal. European journal of public health. 2013;23(6):1027-31.
251. Datar A, Nicosia N, Shier V. Parent perceptions of neighborhood safety and children's physical activity, sedentary behavior, and obesity: evidence from a national longitudinal study. American journal of epidemiology. 2013;177(10):1065-73.
252. Reeske A, Spallek J, Bammann K, Eiben G, De Henauw S, Kourides Y, et al. Migrant background and weight gain in early infancy: results from the German study sample of the IDEFICS study. PLoS One. 2013;8(4):e60648.
253. de Hoog ML, van Eijsden M, Stronks K, Gemke RJ, Vrijkotte TG. Overweight at age two years in a multi-ethnic cohort (ABCD study): the role of prenatal factors, birth outcomes and postnatal factors. BMC Public Health. 2011;11:611.
254. Watt TT, Martinez-Ramos G, Majumdar D. Race/ethnicity, acculturation, and sex differences in the relationship between parental social support and children's overweight and obesity. Journal of health care for the poor and underserved. 2012;23(4):1793-805.
255. Kannan S, Acosta LM, Acevedo-Garcia D, Divjan A, Bracero LA, Perzanowski MS, et al. Sociocultural characteristics, obesity and inflammatory biomarkers in Puerto Rican toddlers born in New York City. Pediatric allergy and immunology : official publication of the European Society of Pediatric Allergy and Immunology. 2013;24(5):487-92.
256. Taveras EM, Gillman MW, Kleinman K, Rich-Edwards JW, Rifas-Shiman SL. Racial/ethnic differences in early-life risk factors for childhood obesity. Pediatrics. 2010;125(4):686-95.
257. Isong IA, Richmond T, Avendano M, Kawachi I. Racial/Ethnic Disparities: a Longitudinal Study of Growth Trajectories Among US Kindergarten Children. Journal of racial and ethnic health disparities. 2017.
258. Arcaya MC, Arcaya AL, Subramanian SV. Inequalities in health: definitions, concepts, and theories. Global health action. 2015;8:27106-.
259. Scambler G. Health inequalities. Sociology of Health & Illness. 2012;34(1):130-46.
260. Davidoff F, Dixon-Woods M, Leviton L, Michie S. Demystifying theory and its use in improvement. BMJ quality & safety. 2015;24(3):228-38.
261. Davies M, Macdowall W. Health Promotion Theory. Black N, Raine R, editors. Berkshire, England: Open University Press; 2006.
262. Cyranoski D. CRISPR-baby scientist fails to satisfy critics. Nature. 2018;564:13-4.

263. Growing Up in Ireland NLSCI. Report on pre-piloting, piloting and dress rehearsal phases of the infant cohort at wave one (9 months). Dublin: Department of Children and Youth Affairs; 2013 Dec. Report No.: 1.
264. Quail A, Williams J, McCrory C, Murray A, Thronton M. Sample design and response in wave 1 of the infant cohort (at 9 months) of Growing Up in Ireland. Dublin: The Economic and Social Research Institute, Office of the Minister for Children and Youth Affairs, University College Dublin; 2011 4/2011.
265. The Economic and Social Research Institute, Trinity College Dublin. Growing Up in Ireland - the National Longitudinal Study of Children, Data Workshop, Infant Cohort [Presentation slides]. Dublin, 2016.
266. Murray A, Williams J, Quail A, Neary M, Thornton M. A summary guide to wave 3 of the infant cohort (at 5 years) of Growing Up in Ireland. Dublin,: The Economic and Social Research Institute, Department of Children and Youth Affairs, Trinity College Dublin,; 2015 Oct.
267. Growing Up in Ireland NLSCI. Design, instrumentation and procedures for the infant cohort at wave one (9 months). Dublin: Department of Children and Youth Affairs; 2013 Dec.
268. Murray A, Quail A, McCrory C, Williams J. A summary guide to wave 2 of the infant cohort (at 3 years) of Growing Up in Ireland. Dublin: The Economic and Social Research Institute, Department of Children and Youth Affairs, University of Dublin Trinity College; 2013 May 2013.
269. Growing Up in Ireland NLSCI. Design, instrumentation and procedures for the infant cohort at wave two (3 years). Dublin: Department of Children and Youth Affairs; 2013 Dec.
270. Growing Up in Ireland NLSCI. Report on the pre-pilot, pilot and dress rehearsal exercises for wave two of the infant cohort at age three years. Dublin: Department of Children and Youth Affairs; 2014.
271. Growing Up in Ireland NLSCI. Report on the pilot phase of wave three, infant cohort (at 5 years of age). Dublin: Department of Children and Youth Affairs; 2016.
272. The Economic and Social Research Institute. Infant Cohort [updated 2016. Available from: http://www.esri.ie/growing-up-in-ireland/questionnaires/.
273. Department of Children and Youth Affairs. Growing Up in Ireland [Available from: https://www.dcya.gov.ie/viewdoc.asp?fn=%2Fdocuments%2FGrowingUpInIreland%2FGrowingUpInIrelandQuestions.htm.
274. World Health Organization. Child growth standards: WHO; [updated 2017. Available from: http://who.int/childgrowth/en/.
275. World Health Organization. Growth reference 5-19 years: WHO; [updated 2017. Available from: http://www.who.int/growthref/en/.
276. Mei Z, Grummer-Strawn LM. Standard deviation of anthropometric Z-scores as a data quality assessment tool using the 2006 WHO growth standards: a cross country analysis. Bulletin of the World Health Organization. 2007;85(6):441-8.
277. WHO Multicentre Growth Reference Study Group. WHO Child Growth Standards based on length/height, weight and age. Acta paediatrica (Oslo, Norway : 1992) Supplement. 2006;450:76-85.
278. Berkey CS, Colditz GA. Adiposity in adolescents: change in actual BMI works better than change in BMI z score for longitudinal studies. Annals of epidemiology. 2007;17(1):44-50.
279. Freedman DS, Berenson GS. Tracking of BMI z Scores for Severe Obesity. Pediatrics. 2017;140(3).
280. Freedman DS, Butte NF, Taveras EM, Goodman AB, Ogden CL, Blanck HM. The Limitations of Transforming Very High Body Mass Indexes into z-Scores among 8.7 Million 2- to 4-Year-Old Children. The Journal of pediatrics. 2017;188:50-6 e1.
281. Vidmar S, Carlin J, Hesketh K, Cole T. Standardizing anthropometric measures in children and adolescents with new functions for egen. Stata Journal. 2004;4(1):50-5.
282. Vidmar SI, Cole TJ, Pan H. Standardizing anthropometric measures in children and adolescents with functions for egen: Update. Stata Journal. 2013;13(2):366-78.

283. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. BMJ. 2000;320(7244):1240-3.
284. Growing Up in Ireland NLSCI. Infant Cohort Questionnaires 2016 [Available from: http://www.growingup.ie/index.php?id=236.
285. Burrows TL, Martin RJ, Collins CE. A systematic review of the validity of dietary assessment methods in children when compared with the method of doubly labeled water. Journal of the American Dietetic Association. 2010;110(10):1501-10.
286. Tognon G, Moreno LA, Mouratidou T, Veidebaum T, Molnar D, Russo P, et al. Adherence to a Mediterranean-like dietary pattern in children from eight European countries. The IDEFICS study. International journal of obesity. 2014;38 Suppl 2:S108-14.
287. Lioret S, Betoko A, Forhan A, Charles MA, Heude B, de Lauzon-Guillain B. Dietary patterns track from infancy to preschool age: cross-sectional and longitudinal perspectives. The Journal of nutrition. 2015;145(4):775-82.
288. Manios Y, Kourlaba G, Kondaki K, Grammatikaki E, Birbilis M, Oikonomou E, et al. Diet quality of preschoolers in Greece based on the Healthy Eating Index: the GENESIS study. Journal of the American Dietetic Association. 2009;109(4):616-23.
289. Bamia C, Trichopoulos D. An anatomy of the way composite scores work. European journal of epidemiology. 2015;30(6):473-83.
290. Poti JM, Duffey KJ, Popkin BM. The association of fast food consumption with poor dietary outcomes and obesity among children: is it the fast food or the remainder of the diet? The American journal of clinical nutrition. 2014;99(1):162-71.
291. Robinson SM, Marriott LD, Crozier SR, Harvey NC, Gale CR, Inskip HM, et al. Variations in infant feeding practice are associated with body composition in childhood: a prospective cohort study. The Journal of clinical endocrinology and metabolism. 2009;94(8):2799-805.
292. Healthy Ireland. Healthy food for life - The food pyramid guide to every day food choices for adults, teenagers and children aged five and over. 2016.
293. Vilela S, Oliveira A, Ramos E, Moreira P, Barros H, Lopes C. Association between energy-dense food consumption at 2 years of age and diet quality at 4 years of age. The British journal of nutrition. 2014;111(7):1275-82.
294. Walton J, McNulty BA, Nugent AP, Gibney MJ, Flynn A. Diet, lifestyle and body weight in Irish children: findings from Irish Universities Nutrition Alliance national surveys. The Proceedings of the Nutrition Society. 2014;73(2):190-200.
295. Pereira-da-Silva L, Rego C, Pietrobelli A. The Diet of Preschool Children in the Mediterranean Countries of the European Union: A Systematic Review. International journal of environmental research and public health. 2016;13(6).
296. Bornhorst C, Huybrechts I, Ahrens W, Eiben G, Michels N, Pala V, et al. Prevalence and determinants of misreporting among European children in proxy-reported 24 h dietary recalls. The British journal of nutrition. 2013;109(7):1257-65.
297. Manios Y, Kourlaba G, Grammatikaki E, Androutsos O, Moschonis G, Roma-Giannikou E. Development of a diet-lifestyle quality index for young children and its relation to obesity: the Preschoolers Diet-Lifestyle Index. Public health nutrition. 2010;13(12):2000-9.
298. Cools W, De Martelaer K, Vandaele B, Samaey C, Andries C. Assessment of Movement Skill Performance in Preschool Children: Convergent Validity Between MOT 4-6 and M-ABC. Journal of sports science & medicine. 2010;9(4):597-604.
299. Barnett LM, Morgan PJ, van Beurden E, Beard JR. Perceived sports competence mediates the relationship between childhood motor skill proficiency and adolescent physical activity and fitness: a longitudinal assessment. The international journal of behavioral nutrition and physical activity. 2008;5:40.
300. Santaliestra-Pasias AM, Mouratidou T, Reisch L, Pigeot I, Ahrens W, Marild S, et al. Clustering of lifestyle behaviours and relation to body composition in European children. The IDEFICS study. European journal of clinical nutrition. 2015;69(7):811-6.

301. Hinkley T, Salmon J, Okely AD, Crawford D, Hesketh K. The HAPPY study: development and reliability of a parent survey to assess correlates of preschool children's physical activity. Journal of science and medicine in sport. 2012;15(5):407-17.
302. Weng SF, Redsell SA, Swift JA, Yang M, Glazebrook CP. Systematic review and meta-analyses of risk factors for childhood overweight identifiable during infancy. Arch Dis Child. 2012;97(12):1019-26.
303. Gibson E, Nawab U. Gestational Age. Merck Manula Professional Version. Kenilworth, NJ, USA: Merck Sharp & Dohme Corp.; 2015.
304. Poulsen G, Kurinczuk JJ, Wolke D, Boyle EM, Field D, Alfirevic Z, et al. Accurate reporting of expected delivery date by mothers 9 months after birth. Journal of clinical epidemiology. 2011;64(12):1444-50.
305. Tate AR, Dezateux C, Cole TJ, Davidson L. Factors affecting a mother's recall of her baby's birth weight. International journal of epidemiology. 2005;34(3):688-95.
306. Voigt M, Rochow N, Hesse V, Olbertz D, Schneider KT, Jorch G. [Short communication about percentile values of body measures of newborn babies]. Zeitschrift fur Geburtshilfe und Neonatologie. 2010;214(1):24-9.
307. Voigt M, Fusch C, Olbertz D, Hartmann K, Rochow N, Renken C, et al. Analyse des Neugeborenenkollektivs der Bundesrepublik Deutschland. Geburtshilfe Frauenheilkd. 2006;66(10):956-70.
308. Ong KK, Preece MA, Emmett PM, Ahmed ML, Dunger DB, Team AS. Size at birth and early childhood growth in relation to maternal smoking, parity and infant breast-feeding: longitudinal birth cohort study and analysis. Pediatr Res. 2002;52(6):863-7.
309. Matthews EK, Wei J, Cunningham SA. Relationship between prenatal growth, postnatal growth and childhood obesity: a review. European journal of clinical nutrition. 2017.
310. Euro-Peristat Project with SCPE and EUROCAT. European perinatal health report. The health and care of pregnant women and babies in Europe in 2010. May 2013.
311. Berry JO, Jones WH. The Parental Stress Scale: Initial Psychometric Evidence. Journal of Social and Personal Relationships. 1995;12(3):463-72.
312. Braveman PA, Cubbin C, Egerter S, Chideya S, Marchi KS, Metzler M, et al. Socioeconomic status in health research: one size does not fit all. Jama. 2005;294(22):2879-88.
313. Sobal J. Obesity and socioeconomic status: a framework for examining relationships between physical and social variables. Medical anthropology. 1991;13(3):231-47.
314. Ali FM, Farah N, O'Dwyer V, O'Connor C, Kennelly MM, Turner MJ. The impact of new national guidelines on screening for gestational diabetes mellitus. Irish medical journal. 2013;106(2):57-9.
315. Growing Up in Ireland NLSCI. GUI infant cohort - small area population statistics. Personal email communication. ed2016.
316. Merriman B, Greene S, Doyle E, McDaid R. Report on the qualitative study of infants and their parents at wave 1 (nine months). Dublin: Department of Children and Youth Affairs; 2013 2013 Nov.
317. Growing Up in Ireland NLSCI. Report on the qualitative study of infants and their parents at wave 1 (nine months). Dublin; 2013.
318. The Economic and Social Research Institute. Infant cohort qualitative and quantitative data link. Personal email communication. ed2015.
319. Dong Y, Peng C-YJ. Principled missing data methods for researchers. SpringerPlus. 2013;2(1):222-.
320. StataCorp. Stata Statistical Software: Release 13. TX: StataCorp LP; 2013.
321. von Elm E, Altman DG, Egger M, Pocock SJ, Gotzsche PC, Vandenbroucke JP. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement: guidelines for reporting observational studies. International journal of surgery (London, England). 2014;12(12):1495-9.

322. Dinsdale H, Ridler C, Ells L. A simple guide to classifying body mass index in children Oxford: National Obesity Observatory; 2011.
323. Reilly JJ. Assessment of obesity in children and adolescents: synthesis of recent systematic reviews and clinical guidelines. Journal of human nutrition and dietetics : the official journal of the British Dietetic Association. 2010;23(3):205-11.
324. Rotella CM, Dicembrini I. Measurement of body composition as a surrogate evaluation of energy balance in obese patients. World journal of methodology. 2015;5(1):1-9.
325. Bluher S, Molz E, Wiegand S, Otto KP, Sergeyev E, Tuschy S, et al. Body mass index, waist circumference, and waist-to-height ratio as predictors of cardiometabolic risk in childhood obesity depending on pubertal development. The Journal of clinical endocrinology and metabolism. 2013;98(8):3384-93.
326. Reilly JJ, Dorosty AR, Ghomizadeh NM, Sherriff A, Wells JC, Ness AR. Comparison of waist circumference percentiles versus body mass index percentiles for diagnosis of obesity in a large cohort of children. Int J Pediatr Obes. 2010;5(2):151-6.
327. Reilly JJ, Kelly J, Wilson DC. Accuracy of simple clinical and epidemiological definitions of childhood obesity: systematic review and evidence appraisal. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2010;11(9):645-55.
328. Lindholm A, Bergman S, Alm B, Tangen GA, Dahlgren J, Roswall J. Infant body mass index growth patterns predicted overweight at five years, waist-to-height ratio did not add to this predictivity. Acta paediatrica. 2018.
329. Verweij LM, Terwee CB, Proper KI, Hulshof CT, van Mechelen W. Measurement error of waist circumference: gaps in knowledge. Public health nutrition. 2013;16(2):281-8.
330. Kyle UG, Earthman CP, Pichard C, Coss-Bu JA. Body composition during growth in children: limitations and perspectives of bioelectrical impedance analysis. European journal of clinical nutrition. 2015;69(12):1298-305.
331. Thivel D, Verney J, Miguet M, Masurier J, Cardenoux C, Lambert C, et al. The accuracy of bioelectrical impedance to track body composition changes depends on the degree of obesity in adolescents with obesity. Nutrition Research. 2018.
332. Nagy P, Kovacs E, Moreno LA, Veidebaum T, Tornaritis M, Kourides Y, et al. Percentile reference values for anthropometric body composition indices in European children from the IDEFICS study. International journal of obesity. 2014;38 Suppl 2:S15-25.
333. O'Shea B, Ladewig EL, Kelly A, Reulbach U, O'Dowd T. Weighing children; parents agree, but GPs conflicted. Arch Dis Child. 2014;99(6):543-5.
334. Lo K, Wong M, Khalechelvam P, Tam W. Waist-to-height circumference for screening paediatric cardio-me Reviews. 2016;17(12):1258-75.
335. Kromeyer-Hauschild K, Wabitsch M, Kunze D, Geller F, Geiss HC, Hesse V, et al. Percentiles of body mass index in children and adolescents evaluated from different regional German studies. Monatsschrift fur Kinderheilkunde. 2001;149:807-18.
336. Valerio G, Balsamo A, Baroni MG, Brufani C, Forziato C, Grugni G, et al. Childhood obesity classification systems and cardiometabolic risk factors: a comparison of the Italian, World Health Organization and International Obesity Task Force references. Italian journal of pediatrics. 2017;43(1):19.
337. Maalouf-Manasseh Z, Metallinos-Katsaras E, Dewey KG. Obesity in preschool children is more prevalent and identified at a younger age when WHO growth charts are used compared with CDC charts. The Journal of nutrition. 2011;141(6):1154-8.
338. Wijnhoven TM, van Raaij JM, Spinelli A, Starc G, Hassapidou M, Spiroski I, et al. WHO European Childhood Obesity Surveillance Initiative: body mass index and level of overweight among 6-9-year-old children from school year 2007/2008 to school year 2009/2010. BMC Public Health. 2014;14:806.

339. Lissau I, Overpeck MD, Ruan WJ, Due P, Holstein BE, Hediger ML. Body mass index and overweight in adolescents in 13 European countries, Israel, and the United States. Archives of pediatrics & adolescent medicine. 2004;158(1):27-33.
340. Whelton H, Harrington J, Crowley E, Kelleher V, Cronin M, Perry IJ. Prevalence of overweight and obesity on the island of Ireland: results from the North South Survey of Children's Height, Weight and Body Mass Index, 2002. BMC Public Health. 2007;7:187.
341. G. B. D. Obesity Collaborators. Health effects of overweight and obesity in 195 countries over 25 years - Supplementary materials. N Engl J Med. 2017;377(1).
342. McMaster C, Cullen L, Raymond N. Overweight and obesity in Irish primary schools: retrospective cohort study. Child: care, health and development. 2005;31(5):499-506.
343. Harrison M, Burns CF, McGuinness M, Heslin J, Murphy NM. Influence of a health education intervention on physical activity and screen time in primary school children: 'Switch Off--Get Active'. Journal of science and medicine in sport. 2006;9(5):388-94.
344. NCD Risk Factor Collaboration. Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults - Supplementary appendix. The Lancet. 2017;390(10113).
345. Heinen MM, Murrin C, Daly L, O'Brien J, Heavey P, Kilroe J, et al. The Childhood Obesity Surveillance Initiative (COSI) in the Republic of Ireland: Findings from 2008, 2010 and 2012. Dublin; 2014.
346. Evans DS, Glacken M, Goggin D. Childhood obesity: the extent of the problem among 6-year-old Irish national school children. Child: care, health and development. 2011;37(3):352-9.
347. Barron C, Comiskey C, Saris J. Prevalence rates and comparisons of obesity levels in Ireland. British journal of nursing (Mark Allen Publishing). 2009;18(13):799-803.
348. Geraghty A, O'Brien EC, Alberdi G, Horan MK, Donnelly J, Larkin E, et al. 756: Maternal protein intake during pregnancy impacts child growth up to five years of age: Findings from the ROLO study. American Journal of Obstetrics & Gynecology. 2018;218(1):S453.
349. Keane E, Layte R, Harrington J, Kearney PM, Perry IJ. Measured parental weight status and familial socio-economic status correlates with childhood overweight and obesity at age 9. PLoS One. 2012;7(8):1-9.
350. Murrin CM, Kelly GE, Tremblay RE, Kelleher CC. Body mass index and height over three generations: evidence from the Lifeways cross-generational cohort study. BMC Public Health. 2012;12:81.
351. Belton S, Brady P, Meegan S, Woods C. Pedometer step count and BMI of Irish primary school children aged 6-9 years. Preventive medicine. 2010;50(4):189-92.
352. Murtagh E, Mulvihill M, Markey O. Bizzy Break! The effect of a classroom-based activity break on in-school physical activity levels of primary school children. Pediatric exercise science. 2013;25(2):300-7.
353. O'Neill A, Dowd K, O'Gorman C, Hannigan A, Walsh C, Purtill H. Activity Profiles and the Associations With Weight Status in Population Studies of Young Children: Are There Gender Differences? Pediatric exercise science. 2017;29(1):131-44.
354. Keane E, Li X, Harrington JM, Fitzgerald AP, Perry IJ, Kearney PM. Physical Activity, Sedentary Behavior and the Risk of Overweight and Obesity in School-Aged Children. Pediatric exercise science. 2017;29(3):408-18.
355. Martin R, Murtagh EM. An intervention to improve the physical activity levels of children: design and rationale of the 'Active Classrooms' cluster randomised controlled trial. Contemporary clinical trials. 2015;41:180-91.
356. WHO European Childhood Obesity Surveillance Initiative 2015. COSI Round 4. 2018.
357. Coppinger T, Lacey S, O'Neill C, Burns C. 'Project Spraoi': A randomized control trial to improve nutrition and physical activity in school children. Contemp Clin Trials Commun. 2016;3:94-101.

358. Irish Universities Nutrition Alliance. National Children's Food Survey II [Available from: https://www.iuna.net/national-childrens-food-survey-ii.
359. Murray D, Hourihane J, Kenny L, Kiely M, Irvine A. BASELINE study in numbers. The Cork BASELINE Birth Cohort. Cork, Ireland: National Children's Research Centre; 2017.
360. Cole TJ, Lobstein T. Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. Pediatr Obes. 2012;7(4):284-94.
361. Wright CM, Williams AF, Elliman D, Bedford H, Birks E, Butler G, et al. Using the new UK-WHO growth charts. Bmj. 2010;340:c1140.
362. Grummer-Strawn LM, Reinold C, Krebs NF, Centers for Disease Control & Prevention. Use of World Health Organization and CDC growth charts for children aged 0-59 months in the United States. MMWR Recommendations and reports : Morbidity and mortality weekly report Recommendations and reports / Centers for Disease Control. 2010;59(RR-9):1-15.
363. Freeman JV, Cole TJ, Chinn S, Jones PR, White EM, Preece MA. Cross sectional stature and weight reference curves for the UK, 1990. Arch Dis Child. 1995;73(1):17-24.
364. World Health Organization. Training course on child growth assessment. Geneva; 2008.
365. Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, Mei Z, et al. 2000 CDC Growth Charts for the United States: methods and development. Vital and health statistics Series 11, Data from the national health survey. 2002(246):1-190.
366. Cole TJ, Freeman JV, Preece MA. Body mass index reference curves for the UK, 1990. Archives of Disease in Childhood. 1995;73(1):25-9.
367. Cole TJ, Wright CM, Williams AF, Group RGCE. Designing the new UK-WHO growth charts to enhance assessment of growth around birth. Arch Dis Child Fetal Neonatal Ed. 2012;97(3):F219-22.
368. Pediatric Obesity, World Obesity. Author Guidelines: John Wiley & Sons, Inc.; 2018 [Available from: https://onlinelibrary.wiley.com/page/journal/20476310/homepage/forauthors.html.
369. Rolland-Cachera MF. Childhood obesity: current definitions and recommendations for their use. Int J Pediatr Obes. 2011;6(5-6):325-31.
370. Ahluwalia N, Dalmasso P, Rasmussen M, Lipsky L, Currie C, Haug E, et al. Trends in overweight prevalence among 11-, 13- and 15-year-olds in 25 countries in Europe, Canada and USA from 2002 to 2010. European journal of public health. 2015;25 Suppl 2:28-32.
371. Oliveira GJ, Barbiero SM, Cesa CC, Pellanda LC. Comparison of NCHS, CDC, and WHO curves in children with cardiovascular risk. Revista da Associacao Medica Brasileira (1992). 2013;59(4):375-80.
372. Flegal KM, Carroll MD, Ogden CL. Effects of trimming weight-for-height data on growth-chart percentiles. The American journal of clinical nutrition. 2012;96(5):1051-5.
373. Edwards J, Evans J, Brown AD. Using routine growth data to determine overweight and obesity prevalence estimates in preschool children in the Capital Health Region of Alberta. Canadian journal of public health = Revue canadienne de sante publique. 2008;99(2):91-4.
374. Vidal E, Carlin E, Driul D, Tomat M, Tenore A. A comparison study of the prevalence of overweight and obese Italian preschool children using different reference standards. European journal of pediatrics. 2006;165(10):696-700.
375. Duran M, Gillespie J, Malina RM, Little BB. Growth and weight status of rural Texas school youth. American journal of human biology : the official journal of the Human Biology Council. 2013;25(1):71-7.
376. Serra-Majem L, Ribas-Barba L, Perez-Rodrigo C, Ngo J, Aranceta J. Methodological limitations in measuring childhood and adolescent obesity and overweight in epidemiological studies: does overweight fare better than obesity? Public health nutrition. 2007;10(10a):1112-20.
377. Meyers A, Joyce K, Coleman SM, Cook JT, Cutts D, Ettinger de Cuba S, et al. Health of children classified as underweight by CDC reference but normal by WHO standard. Pediatrics. 2013;131(6):e1780-7.

378. Silveira CR, Beghetto MG, Carvalho PR, Mello ED. Comparison of NCHS, CDC and WHO growth charts in the nutritional assessment of hospitalized children up to five years old. Nutricion hospitalaria. 2011;26(3):465-71.
379. Beau AB, Tauber M, Chollet C, Hurault-Delarue C, Bouilhac C, Montastruc JL, et al. A contemporary description of French newborns' growth using the Efemeris cohort. Archives de pediatrie : organe officiel de la Societe francaise de pediatrie. 2017;24(5):424-31.
380. Nilsen BB, Yngve A, Sjoberg A, Moraeus L, Lissner L, Werner B. Using different growth references to measure thinness and overweight among Swedish primary school children showed considerable variations. Acta paediatrica. 2016;105(10):1158-65.
381. WHO Working Group on Infant Growth, World Health Organization. An evaluation of infant growth : a summary of analyses performed in preparation for the WHO Expert Committee on Physical Status, the Use and Interpretation of Anthropometry / WHO Working Group on Infant Growth. Geneva; 1994.
382. Hassapidou M, Daskalou E, Tsofliou F, Tziomalos K, Paschaleri A, Pagkalos I, et al. Prevalence of overweight and obesity in preschool children in Thessaloniki, Greece. Hormones (Athens, Greece). 2015;14(4):615-22.
383. Pattinson CL, Staton SL, Smith SS, Trost SG, Sawyer EF, Thorpe KJ. Weighing in on international growth standards: testing the case in Australian preschool children. Obesity Reviews. 2017;18(10):1111-21.
384. Ogden CL, Flegal KM. Changes in terminology for childhood overweight and obesity. National health statistics reports. 2010(25):1-5.
385. Pigeot I, Barba G, Chadjigeorgiou C, de Henauw S, Kourides Y, Lissner L, et al. Prevalence and determinants of childhood overweight and obesity in European countries: pooled analysis of the existing surveys within the IDEFICS Consortium. International journal of obesity. 2009;33(10):1103-10.
386. Schmidt Morgen C, Rokholm B, Sjoberg Brixval C, Schou Andersen C, Geisler Andersen L, Rasmussen M, et al. Trends in prevalence of overweight and obesity in danish infants, children and adolescents--are we still on a plateau? PLoS One. 2013;8(7):e69860.
387. Keke LM, Samouda H, Jacobs J, di Pompeo C, Lemdani M, Hubert H, et al. Body mass index and childhood obesity classification systems: A comparison of the French, International Obesity Task Force (IOTF) and World Health Organization (WHO) references. Revue d'epidemiologie et de sante publique. 2015;63(3):173-82.
388. Lane A, Harrison M, Murphy N. Screen time increases risk of overweight and obesity in active and inactive 9-year-old Irish children: a cross sectional analysis. Journal of physical activity & health. 2014;11(5):985-91.
389. Dowd KP, Kirwan RP, Hannigan A, Purtill H, O'Gorman CS. The association between maternal perceptions of own weight status and weight status of her child: results from a national cohort study. Arch Dis Child. 2016;101(1):28-32.
390. Field A. Discovering statistics using SPSS. 3rd ed. London: Sage; 2009.
391. Barrett A, McGuinness S. The Irish Labour Market and the Great Recession. CESifo DICE Report. 2012;2:27-33.
392. Tabachnick BG, Fidell LS. Using multivariate statistics. 5th ed. Boston: Allyn & Bacon; 2007.
393. Curran PJ, Obeidat K, Losardo D. Twelve Frequently Asked Questions About Growth Curve Modeling. Journal of cognition and development : official journal of the Cognitive Development Society. 2010;11(2):121-36.
394. UCLA Institute for Digital Research and Education. Introduction to linear mixed models 2018 [Available from: https://stats.idre.ucla.edu/other/mult-pkg/introduction-to-linear-mixed-models/.
395. World Health Organization. Child growth standards BMI-for-age 2014 [updated 2014. Available from: http://www.who.int/childgrowth/standards/bmi_for_age/en/.
396. safefood. Minutes of the seventh meeting of the All-island Obesity Action Forum, held on Tuesday 15 November 2011 at The Morrison Hotel, Dublin. 2011.

397. Braun JM, Kalkwarf HJ, Papandonatos GD, Chen A, Lanphear BP. Patterns of early life body mass index and childhood overweight and obesity status at eight years of age. BMC pediatrics. 2018;18:161.
398. Perng W, Hajj H, Belfort MB, Rifas-Shiman SL, Kramer MS, Gillman MW, et al. Birth Size, Early Life Weight Gain, and Midchildhood Cardiometabolic Health. The Journal of pediatrics. 2016;173:122-30.e1.
399. McCormick EV, Dickinson LM, Haemer MA, Knierim SD, Hambidge SJ, Davidson AJ. What can providers learn from childhood body mass index trajectories: a study of a large, safety-net clinical population. Academic pediatrics. 2014;14(6):639-45.
400. Smego A, Woo JG, Klein J, Suh C, Bansal D, Bliss S, et al. High Body Mass Index in Infancy May Predict Severe Obesity in Early Childhood. The Journal of pediatrics. 2017;183:87-93.e1.
401. Cattaneo A, Monasta L, Stamatakis E, Lioret S, Castetbon K, Frenken F, et al. Overweight and obesity in infants and pre-school children in the European Union: a review of existing data. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2010;11(5):389-98.
402. Li C, Goran MI, Kaur H, Nollen N, Ahluwalia JS. Developmental trajectories of overweight during childhood: role of early life factors. Obesity (Silver Spring, Md). 2007;15(3):760-71.
403. Péneau S, Giudici KV, Gusto G, Goxe D, Lantieri O, Hercberg S, et al. Growth Trajectories of Body Mass Index during Childhood: Associated Factors and Health Outcome at Adulthood. The Journal of pediatrics. 2017;186:64-71.e1.
404. Guerrero AD, Mao C, Fuller B, Bridges M, Franke T, Kuo AA. Racial and Ethnic Disparities in Early Childhood Obesity: Growth Trajectories in Body Mass Index. Journal of racial and ethnic health disparities. 2016;3(1):129-37.
405. Eny KM, Chen S, Anderson LN, Chen Y, Lebovic G, Pullenayegum E, et al. Breastfeeding duration, maternal body mass index, and birth weight are associated with differences in body mass index growth trajectories in early childhood. The American journal of clinical nutrition. 2018;107(4):584-92.
406. Tu AW, Masse LC, Lear SA, Gotay CC, Richardson CG. Body mass index trajectories from ages 1 to 20: Results from two nationally representative canadian longitudinal cohorts. Obesity (Silver Spring, Md). 2015;23(8):1703-11.
407. Koontz MB, Gunzler DD, Presley L, Catalano PM. Longitudinal changes in infant body composition: association with childhood obesity. Pediatr Obes. 2014;9(6):e141-e4.
408. Dubois L, Girard M. Early determinants of overweight at 4.5 years in a population-based longitudinal study. International journal of obesity. 2006;30(4):610-7.
409. Crume TL, Ogden LG, Mayer-Davis EJ, Hamman RF, Norris JM, Bischoff KJ, et al. The impact of neonatal breast-feeding on growth trajectories of youth exposed and unexposed to diabetes in utero: the EPOCH Study. International journal of obesity. 2012;36(4):529-34.
410. Patro-Golab B, Zalewski BM, Kouwenhoven SM, Karas J, Koletzko B, Bernard van Goudoever J, et al. Protein Concentration in Milk Formula, Growth, and Later Risk of Obesity: A Systematic Review. The Journal of nutrition. 2016;146(3):551-64.
411. Azad MB, Konya T, Persaud RR, Guttman DS, Chari RS, Field CJ, et al. Impact of maternal intrapartum antibiotics, method of birth and breastfeeding on gut microbiota during the first year of life: a prospective cohort study. BJOG : an international journal of obstetrics and gynaecology. 2016;123(6):983-93.
412. Janssen PA, Thiessen P, Klein MC, Whitfield MF, Macnab YC, Cullis-Kuhl SC. Standards for the measurement of birth weight, length and head circumference at term in neonates of European, Chinese and South Asian ancestry. Open medicine : a peer-reviewed, independent, open-access journal. 2007;1(2):e74-88.
413. Boone-Heinonen J, Messer L, Andrade K, Takemoto E. Connecting the Dots in Childhood Obesity Disparities: A Review of Growth Patterns from Birth to Pre-Adolescence. Current epidemiology reports. 2016;3(1):113-24.

414. Roy SM, Chesi A, Mentch F, Xiao R, Chiavacci R, Mitchell JA, et al. Body mass index (BMI) trajectories in infancy differ by population ancestry and may presage disparities in early childhood obesity. The Journal of clinical endocrinology and metabolism. 2015;100(4):1551-60.
415. Wen X, Kleinman K, Gillman MW, Rifas-Shiman SL, Taveras EM. Childhood body mass index trajectories: modeling, characterizing, pairwise correlations and socio-demographic predictors of trajectory characteristics. BMC medical research methodology. 2012;12:38.
416. Queally M, Doherty E, Matvienko-Sikar K, Toomey E, Cullinan J, Harrington JM, et al. Do mothers accurately report their overweight/obesity status during early childhood? Evidence from a nationally representative cohort study. International Journal of Behavioral Nutrition and Physical Activity. 2018;15(1):56.
417. Martinson ML, McLanahan S, Brooks-Gunn J. Variation in child body mass index patterns by race/ethnicity and maternal nativity status in the United States and England. Maternal and child health journal. 2015;19(2):373-80.
418. Demment MM, Haas JD, Olson CM. Changes in family income status and the development of overweight and obesity from 2 to 15 years: a longitudinal study. BMC Public Health. 2014;14:417.
419. Shrewsbury V, Wardle J. Socioeconomic status and adiposity in childhood: a systematic review of cross-sectional studies 1990-2005. Obesity (Silver Spring, Md). 2008;16(2):275-84.
420. Chen AY, Escarce JJ. Family structure and childhood obesity, Early Childhood Longitudinal Study - Kindergarten Cohort. Preventing chronic disease. 2010;7(3):A50.
421. Carter MA, Dubois L, Tremblay MS, Taljaard M, Jones BL. Trajectories of childhood weight gain: the relative importance of local environment versus individual social and early life factors. PLoS One. 2012;7(10):e47065.
422. Castro PD, Kearney J, Layte R. A study of early complementary feeding determinants in the Republic of Ireland based on a cross-sectional analysis of the Growing Up in Ireland infant cohort. Public health nutrition. 2014;18(2):292-302.
423. Koning M, Hoekstra T, de Jong E, Visscher TL, Seidell JC, Renders CM. Identifying developmental trajectories of body mass index in childhood using latent class growth (mixture) modelling: associations with dietary, sedentary and physical activity behaviors: a longitudinal study. BMC Public Health. 2016;16(1):1128.
424. Danner FW. A national longitudinal study of the association between hours of TV viewing and the trajectory of BMI growth among US children. Journal of pediatric psychology. 2008;33(10):1100-7.
425. Simonato I, Janosz M, Archambault I, Pagani LS. Prospective associations between toddler televiewing and subsequent lifestyle habits in adolescence. Preventive medicine. 2018;110:24-30.
426. Bailey LC, Forrest CB, Zhang P, Richards TM, Livshits A, DeRusso PA. Association of antibiotics in infancy with early childhood obesity. JAMA pediatrics. 2014;168(11):1063-9.
427. Trasande L, Blustein J, Liu M, Corwin E, Cox LM, Blaser MJ. Infant antibiotic exposures and early-life body mass. International journal of obesity. 2013;37(1):16-23.
428. Block JP, Bailey LC, Gillman MW, Lunsford D, Boone-Heinonen J, Cleveland LP, et al. The PCORnet Antibiotics and Childhood Growth Study: Process for Cohort Creation and Cohort Description. Academic pediatrics. 2018.
429. Di Gioia D, Aloisio I, Mazzola G, Biavati B. Bifidobacteria: their impact on gut microbiota composition and their applications as probiotics in infants. Applied microbiology and biotechnology. 2014;98(2):563-77.
430. Cardon G, De Bourdeaudhuij I, Iotova V, Latomme J, Socha P, Koletzko B, et al. Health Related Behaviours in Normal Weight and Overweight Preschoolers of a Large Pan-European Sample: The ToyBox-Study. PLoS ONE. 2016;11(3):e0150580.
431. Donkor HM, Grundt JH, Juliusson PB, Eide GE, Hurum J, Bjerknes R, et al. Social and somatic determinants of underweight, overweight and obesity at 5 years of age: a Norwegian regional cohort study. BMJ open. 2017;7(8):e014548.

432. Townsend MS, Shilts MK, Styne DM, Drake C, Lanoue L, Ontai L. An Obesity Risk Assessment Tool for Young Children: Validity With BMI and Nutrient Values. Journal of nutrition education and behavior. 2018;50(7):705-17.
433. Bornhorst C, Huybrechts I, Hebestreit A, Vanaelst B, Molnar D, Bel-Serrat S, et al. Diet-obesity associations in children: approaches to counteract attenuation caused by misreporting. Public health nutrition. 2013;16(2):256-66.
434. Craigie AM, Lake AA, Kelly SA, Adamson AJ, Mathers JC. Tracking of obesity-related behaviours from childhood to adulthood: A systematic review. Maturitas. 2011;70(3):266-84.
435. Blake-Lamb TL, Locks LM, Perkins ME, Woo Baidal JA, Cheng ER, Taveras EM. Interventions for Childhood Obesity in the First 1,000 Days A Systematic Review. American journal of preventive medicine. 2016;50(6):780-9.
436. Kugelberg S, Jewell J, Breda J. Prevention Across Europe. [cited 6/3/2017]. In: The ECOG Free Obesity eBook [Internet]. [cited 6/3/2017].
437. Hennessy M, Byrne M, Laws R, Mc Sharry J, O'Malley G, Heary C. Childhood obesity prevention: priority areas for future research and barriers and facilitators to knowledge translation, coproduced using the nominal group technique. Translational behavioral medicine. 2018.
438. Jernigan J, Kettel Khan L, Dooyema C, Ottley P, Harris C, Dawkins-Lyn N, et al. Childhood Obesity Declines Project: Highlights of Community Strategies and Policies. Childhood obesity. 2018;14(S1):S32-s9.
439. Dattilo AM. Modifiable Risk Factors and Interventions for Childhood Obesity Prevention within the First 1,000 Days. Nestle Nutrition Institute workshop series. 2017;87:183-96.
440. Buscemi J, Berlin KS, Rybak TM, Schiffer LA, Kong A, Stolley MR, et al. Health Behavior and Weight Changes Among Ethnic and Racial Minority Preschoolers and Their Parents: Associations Across 1 Year. Journal of pediatric psychology. 2016;41(7):777-85.
441. Verbestel V, De Henauw S, Barba G, Eiben G, Gallois K, Hadjigeorgiou C, et al. Effectiveness of the IDEFICS intervention on objectively measured physical activity and sedentary time in European children. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2015;16 Suppl 2:57-67.
442. World Obesity Federation. Obesity prevention in childen in pre-school years: policies and evidence - Report of a high-level workshop held in Brussels, 11 April 2014. 2014.
443. Smith HA, O'Brien EC, Kiely M, O'Brien J, Warren P, Earlylife factors associated with the exclusivity and duration of breast feeding in an Irish birth cohort study. Midwifery. 2015;31(9):904-11.
444. Niedhammer I, O'Mahony D, Daly S, Morrison JJ, Kelleher CC. Occupational predictors of pregnancy outcomes in Irish working women in the Lifeways cohort. BJOG : an international journal of obstetrics and gynaecology. 2009;116(7):943-52.
445. Department of Health. Healthy Ireland - A framework for improved health and wellbeing 2013-2025. Dublin2013.
446. Department of Health. Non-broadcasting media advertising and marketing of food and non-alcoholic beverages, including sponsorship and retail product placement: Voluntary codes of practice. 2017.
447. Tatlow-Golden M, Hennessy E, Dean M, Hollywood L. Young children's food brand knowledge. Early development and associations with television viewing and parent's diet. Appetite. 2014;80:197-203.
448. Cradock AL, Barrett JL, Kenney EL, Giles CM, Ward ZJ, Long MW, et al. Using cost-effectiveness analysis to prioritize policy and programmatic approaches to physical activity promotion and obesity prevention in childhood. Preventive medicine. 2016.
449. Wang Y, Cai L, Wu Y, Wilson RF, Weston C, Fawole O, et al. What childhood obesity prevention programmes work? A systematic review and meta-analysis. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2015;16(7):547-65.

450. Muktabhant B, Lawrie TA, Lumbiganon P, Laopaiboon M. Diet or exercise, or both, for preventing excessive weight gain in pregnancy. Cochrane Database of Systematic Reviews. 2015(6).
451. Redsell SA, Edmonds B, Swift JA, Siriwardena AN, Weng S, Nathan D, et al. Systematic review of randomised controlled trials of interventions that aim to reduce the risk, either directly or indirectly, of overweight and obesity in infancy and early childhood. Maternal & child nutrition. 2016;12(1):24-38.
452. Matwiejczyk L, Mehta K, Scott J, Tonkin E, Coveney J. Characteristics of Effective Interventions Promoting Healthy Eating for Pre-Schoolers in Childcare Settings: An Umbrella Review. Nutrients. 2018;10(3).
453. Sisson SB, Krampe M, Anundson K, Castle S. Obesity prevention and obesogenic behavior interventions in child care: A systematic review. Preventive medicine. 2016;87:57-69.
454. Johnston Molloy C, Kearney J, Hayes N, Slattery CG, Corish C. Healthy incentive scheme in the Irish full-day-care pre-school setting. The Proceedings of the Nutrition Society. 2014;73(1):147-58.
455. Reznik M, Wylie-Rosett J, Kim M, Ozuah PO. A classroom-based physical activity intervention for urban kindergarten and first-grade students: a feasibility study. Childhood obesity. 2015;11(3):314-24.
456. Department of Children and Youth Affairs. Early Childhood Care and Education (ECCE) or Free pre-school 2018 [Available from: https://www.dcya.gov.ie/cat/EN/What-We-Do/211.htm.
457. Sosa ET, Parra-Medina D, He M, Trummer V, Yin Z. inverted exclamation markMiranos! (Look at Us! We Are Healthy!): Home-Based and Parent Peer-Led Childhood Obesity Prevention. Health promotion practice. 2016;17(5):675-81.
458. Department of Health and Children. Food and Nutrition Guidelines for Pre-School Services. 2004.
459. Healthy Ireland. Healthy Ireland Smart Start - Health promotion training for preschool practitioners. Healthy Ireland Smart Start e-newsletter 2018.
460. safefood. About Little Bites [Available from: https://www.safefood.eu/Education/Pre-school/Little-Bites-(ROI)/About-us.aspx.
461. Little Steps, Department of Health and Children, Health Service Executive. 60 minutes active play every day! Fact sheet for childcare providers.
462. Healthy Ireland. Get Ireland Active! National physical activity plan for Ireland. In: Health Do, editor. Dublin2016.
463. Early Childhood Ireland. Kids active programme 2017 [Available from: https://www.earlychildhoodireland.ie/kids-active-programme/.
464. Health Service Executive. Making Every Contact Count: A health behaviour change framework and implementation plan for health professionals in the Irish health service. 2016.
465. Health Service Executive. Form of agreement with registered medical practitioners for provision of services to children under 6 years old pursuant to the health (General practitioner service) act 2014 (under 6 year olds). 2015. p. 30.
466. Marchi J, Berg M, Dencker A, Olander EK, Begley C. Risks associated with obesity in pregnancy, for the mother and baby: a systematic review of reviews. Obesity reviews : an official journal of the International Association for the Study of Obesity. 2015;16(8):621-38.
467. Healthy Ireland, Department of Health, Patient Safety First. Creating a Better Future Together - National Maternity Strategy 2016-2026. 2016.
468. Allen-Walker V, Mullaney L, Turner MJ, Woodside JV, Holmes VA, McCartney DM, et al. How do women feel about being weighed during pregnancy? A qualitative exploration of the opinions and experiences of postnatal women. Midwifery. 2017;49:95-101.
469. Flannery C, McHugh S, Anaba AE, Clifford E, O'Riordan M, Kenny LC, et al. Enablers and barriers to physical activity in overweight and obese pregnant women: an analysis informed by the theoretical domains framework and COM-B model. BMC pregnancy and childbirth. 2018;18(1):178.

470. Anderson CK, Walch TJ, Lindberg SM, Smith AM, Lindheim SR, Whigham LD. Excess Gestational Weight Gain in Low-Income Overweight and Obese Women: A Qualitative Study. Journal of nutrition education and behavior. 2015;47(5):404-11.e1.
471. Food Safety Authority of Ireland. Best practice for infant feeding in Ireland - From pre-conception through the first year of an infant's life. 2012.
472. Health Service Executive, Irish College of General Practitioners. Healthy weight, management guidelines before, during and after pregnancy. A quick reference guide for primary care. Dublin.
473. safefood. Minutes of the fifteenth meeting of the All-island Obesity Action Forum, held on Tuesday 10th November 2015 at the Gibson Hotel, Dublin. 2015.
474. Huxtable A, Millar L, Love P, Bell C, Whelan J. Parental Translation into Practice of Healthy Eating and Active Play Messages and the Impact on Childhood Obesity: A Mixed Methods Study. Nutrients. 2018;10(5).
475. Wen LM, Baur LA, Simpson JM, Rissel C, Wardle K, Flood VM. Effectiveness of home based early intervention on children's BMI at age 2: randomised controlled trial. Bmj. 2012;344:e3732.
476. Paul IM, Savage JS, Anzman-Frasca S, et al. Effect of a responsive parenting educational intervention on childhood weight outcomes at 3 years of age: The insight randomized clinical trial. Jama. 2018;320(5):461-8.
477. Lanigan J. Prevention of overweight and obesity in early life. Proceedings of the Nutrition Society. 2018;77(3):247-56.
478. Mennella JA, Ventura AK, Beauchamp GK. Differential growth patterns among healthy infants fed protein hydrolysate or cow-milk formulas. Pediatrics. 2011;127(1):110-8.
479. Weber M, Grote V, Closa-Monasterolo R, Escribano J, Langhendries JP, Dain E, et al. Lower protein content in infant formula reduces BMI and obesity risk at school age: follow-up of a randomized trial. The American journal of clinical nutrition. 2014;99(5):1041-51.
480. Health Service Executive, Healthy Ireland, Better Outcomes Brighter Futures. The Nurture Programme - Infant health and wellbeing. 2016.
481. Health Service Executive. Brief updates from The Nurture Programme 2018 [Available from: https://www.hse.ie/eng/health/child/nurture/updates.html.
482. safefood. Minutes of the tenth meeting of the All-island Obesity Action Forum, held on Tuesday 25 June 2013 at the Stormont Hotel, Belfast. 2013.
483. Health Service Executive, Irish College of General Practitioners. Weight management treatment algorithm for children - A quick reference guide for primary care. 2013.
484. Klocke A, Stadtmüller S. Social Capital in the Health Development of Children. Child Indicators Research. 2018.
485. Knol LL, Myers HH, Black S, Robinson D, Awololo Y, Clark D, et al. Development and Feasibility of a Childhood Obesity Prevention Program for Rural Families: Application of the Social Cognitive Theory. American journal of health education. 2016;47(4):204-14.
486. Fiese BH. Context matters in pediatric obesity: commentary on innovative treatment and prevention programs for pediatric overweight and obesity. Journal of pediatric psychology. 2013;38(9):1037-43.
487. Doorley E, Young C, O'Shea B, Darker C, Hollywood B, O'Rorke C. Is primary prevention of childhood obesity by education at 13-month immunisations feasible and acceptable? Results from a general practice based pilot study. Irish medical journal. 2015;108(1):13-5.
488. Rietmeijer-Mentink M, Paulis WD, van Middelkoop M, Bindels PJ, van der Wouden JC. Difference between parental perception and actual weight status of children: a systematic review. Maternal & child nutrition. 2013;9(1):3-22.
489. Moore LC, Harris CV, Bradlyn AS. Exploring the relationship between parental concern and the management of childhood obesity. Maternal and child health journal. 2012;16(4):902-8.
490. Oude Luttikhuis H, Stolk RP, Sauer PJ. How do parents of 4-to 5-year-old children perceive the weight of their children? Acta paediatrica. 2010;99.

491. Hernandez RG, Cheng TL, Serwint JR. Parents' healthy weight perceptions and preferences regarding obesity counseling in preschoolers: pediatricians matter. Clinical pediatrics. 2010;49(8):790-8.
492. safefood. A guide for health professionals - Assisting parents and guardians in communicating with their children about body weight. 2013.
493. Jones AR, Tovee MJ, Cutler LR, Parkinson KN, Ells LJ, Araujo-Soares V, et al. Development of the MapMe intervention body image scales of known weight status for 4-5 and 10-11 year old children. Journal of public health. 2017:1-9.
494. Hammersley ML, Jones RA. Parent-Focused Childhood and Adolescent Overweight and Obesity eHealth Interventions: A Systematic Review and Meta-Analysis. 2016;18(7):e203.
495. Mack I, Bayer C, Schaffeler N, Reiband N, Brolz E, Zurstiege G, et al. Chances and Limitations of Video Games in the Fight against Childhood Obesity-A Systematic Review. European eating disorders review : the journal of the Eating Disorders Association. 2017.
496. Oliver J. Foundation [Internet]2018. [cited 2018]. Available from: https://www.jamieoliver.com/news-and-features/features/weve-adenough-of-junk-food-marketing/.
497. safefood. 'Lets take on childhood obesity' - Evaluation of a 3 year public health campaign. safefood; 2017.
498. Pont SJ, Puhl R, Cook SR, Slusser W. Stigma Experienced by Children and Adolescents With Obesity. Pediatrics. 2017;140(6).
499. Gillison FB, Lorenc AB, Sleddens EF, Williams SL, Atkinson L. Can it be harmful for parents to talk to their child about their weight? A meta-analysis. Preventive medicine. 2016;93:135-46.
500. Dominguez Rodriguez A, Cebolla Marti AJ, Oliver-Gasch E, Banos-Rivera RM. Online platforms to teach Nutrition Education to children: a non-systematic review. Nutricion hospitalaria. 2016;33(6):1444-51.
501. Irish Nutrition and Dietetic Institute. Links to useful resources on childhood nutrition: A guide for parents and teachers 2015 [Available from: https://www.indi.ie/fact-sheets/fact-sheets-on-nutrition-for-babies-children/365-links-to-useful-resources-on-childhood-nutrition-a-guide-for-parents-and-teachers.html.
502. Bryant M, Ashton L, Brown J, Jebb S, Wright J, Roberts K, et al. Systematic review to identify and appraise outcome measures used to evaluate childhood obesity treatment interventions (CoOR): evidence of purpose, application, validity, reliability and sensitivity. Health technology assessment (Winchester, England). 2014;18(51):1-380.
503. Nau C, Schwartz BS, Bandeen-Roche K, Liu A, Pollak J, Hirsch A, et al. Community socioeconomic deprivation and obesity trajectories in children using electronic health records. Obesity (Silver Spring, Md). 2015;23(1):207-12.
504. Gutilla MJ, Davidson AJ, Daley MF, Anderson GB, Marshall JA, Magzamen S. Data for Community Health Assessment in Rural Colorado: A Comparison of Electronic Health Records to Public Health Surveys to Describe Childhood Obesity. Journal of public health management and practice : JPHMP. 2017;23 Suppl 4 Supplement, Community Health Status Assessment:S53-s62.
505. Pimpin L, Jebb S, Johnson L, Wardle J, Ambrosini GL. Dietary protein intake is associated with body mass index and weight up to 5 y of age in a prospective cohort of twins1,2. The American journal of clinical nutrition. 2016;103(2):389-97.
506. Warthon-Medina M, Hooson J, Hancock N, Gibson LE, Bush LA, Hutchinson J, et al. Systematic review and website presentation of validated dietary assessment tools. Summer Meeting, 10-12 July 2018, Getting energy balance right: Proceedings of the Nutrition Society; 2018.
507. van de Beek C, Hoek A, Painter RC, Gemke R, van Poppel MNM, Geelen A, et al. Women, their Offspring and iMproving lifestyle for Better cardiovascular health of both (WOMB project): a protocol of the follow-up of a multicentre randomised controlled trial. BMJ open. 2018;8(1):e016579.

508. Fonseca H, Silva AM, Matos MG, Esteves I, Costa P, Guerra A, et al. Validity of BMI based on self-reported weight and height in adolescents. Acta paediatrica. 2010;99(1):83-8.
509. Dubois L, Girad M. Accuracy of maternal reports of pre-schoolers' weights and heights as estimates of BMI values. International journal of epidemiology. 2007;36(1):132-8.
510. Meleleo D, Bartolomeo N, Cassano L, Nitti A, Susca G, Mastrototaro G, et al. Evaluation of body composition with bioimpedence. A comparison between athletic and non-athletic children. European journal of sport science. 2017;17(6):710-9.
511. Everitt BSL, Sabine; Leese, Morven; Stahl, Daniel Cluster Analysis. 5 ed. Walter A. Shewhart SSW, editor. West Sussex, United Kingdom: John Wiley & Sons Ltd; 2011 26/1/2011.
512. Hamilton LC. Statistics with STATA: updated for version 12. Boston, USA: Brooks/Cole; 2012.
513. Bauer DJ. Observations on the Use of Growth Mixture Models in Psychological Research. Multivariate Behavioral Research. 2007;42(4):757-86.
514. World Health Organization. Body mass index - BMI [updated 2017. Available from: http://www.euro.who.int/en/health-topics/disease-prevention/nutrition/a-healthy-lifestyle/body-mass-index-bmi.
515. World Health Organization. Global database on body mass index [updated 2017. Available from: http://www.who.int/bmi.
516. Shelley E, Daly L, Kilcoyne D, Graham I. Obesity: a public health problem in Ireland? Irish journal of medical science. 1991;160 Suppl 9:29-34.
517. World Health Organization (WHO). Ireland World Health Survey 2003. Geneva, Switzerland; 2005.

Appendices

Appendix 1: Classification of body mass index in adult populations

Table 30: Classification of body mass index in adult populations

Category	Body mass index
Very severe underweight	Below 15
Severe thinness	From 15.0 to 15.99
Moderate thinness	16 to 16.99
Mild thinness	17 to 18.49
Underweight	Below 18.5
Normal (healthy weight)	From 18.5 to 24.99
Overweight	From 25 to 29.99
Obese Class I (Moderately obese)	From 30 to 34.99
Obese Class II (Severely obese)	From 35 to 39.99
Obese Class III (Very severely obese)	40 or higher

Note: Based on data retrieved from the World Health Organization (514, 515).

Appendix 2: Details of the variable cleaning: Body mass index

2.1 First step of data cleaning

In the original RMF, several children had weight or height missing, so no BMI could be calculated. The number of missing observations in the original dataset and after the first step of cleaning (outlier removal) can be seen in Table 31.

Table 31: Missing weight and length/height before and after the first step of data cleaning

	N missing in original data		**N missing after data cleaning**	
Age	**Weight**	**Height**	**Weight**	**Height**
9 months	147 (1.3%)	157 (1.4%)	207 (1.9%)	297 (2.7%)
3 years	211 (2.2%)	208 (2.1%)	255 (2.6%)	258 (2.6%)
5 years	72 (0.8%)	103 (1.1%)	104 (1.2%)	140 (1.6%)

Note: N at nine months = 11,134, N at three years = 9,793, N at five years = 9,001

2.2 Second step of data cleaning

Table 32 shows ranges of weight and height changes between waves that were considered valid and hence retained in the dataset. Weight, length/height and BMI of children were excluded for the respective waves if they were outside these ranges. Negative changes were excluded as an absolute weight loss within the two-year periods between waves appeared highly unlikely. Strong outliers also were excluded. Furthermore, skewness was looked at to define valid ranges.

Values that expressed the changes in length/height between two waves were normally distributed for each of the periods under investigation. The distribution of changes in weight was slightly skewed to the right for each of these periods. This indicates that a small proportion of children in the cohort gained weight on a much faster rate than most children, while gains in height were within the expected range and distribution of variation. The observed extreme weight gains were too numerous and the distributional patterns too consistent across waves to be considered random errors in data entry. The observations were maintained in the dataset as it was hypothesised in Chapter 2 that some young children in Ireland

gain an excessive amount of weight, relatively to their length/height. An exclusion of children with extremely high weight gains which do not appear to be outliers may have biased this study. Furthermore, as only weight changes were skewed but the outcome variable of BMI at each wave was distributed normally, the slight skewness of weight changes is not expected to lead to computational difficulties.

Table 32: Changes in weight and length/height that took place between waves

Variable	Number *	Missing in waves* (%)	Mean	Standard deviation	Range
Weight change (kg) 9 months – 3 years	9375	418 (4.3%)	5.9	1.4	0.6 – 16.5
Height change (cm) 9 months – 3 years	9353	440 (4.5%)	23.3	3.1	9 – 38.5
Weight change (kg) 3 years – 5 years	8385	327 (3.8%)	4.6	1.7	0.1-17.7
Height change (cm) 3 years – 5 years	8411	301 (3.5%)	15.2	2.5	2.9-33.1
Weight change (kg) 9 months – 5 years	8734	267 (3.0%)	10.6	2.5	3.8-27.1
Height change (cm) 9 months – 5 years	8696	305 (3.4%)	38.5	3.8	22.5-57.5

* Total number of children included in waves 1 (at nine months) and 2 (at three years): 9,793; in waves 2 and 3 (at five years): 8,712; in waves 1 and 3: 9,001

2.3 Comparison of body mass index data before and after cleaning

Table 33 provides an overview of missing values and data ranges that were seen in the data prior and after the two cleaning steps.

Table 33: Descriptive statistics of body mass index before and after data cleaning

Age	Number*	Missing (%)	Mean	SD	Range
9 months *prior cleaning*	10,964	170 (1.5%)	18.2	2.3	6.7 – 60.1
9 months *after cleaning*	10,810	324 (2.9%)	18.2	1.7	11.9 – 26.8
3 years *prior cleaning*	9,533	270 (2.8%)	16.8	1.5	10.7 – 27.2
3 years *after cleaning*	9,422	371 (3.8%)	16.8	1.5	10.6 – 25.0
5 years *prior cleaning*	8,880	121 (1.3%)	16.2	1.7	8.3 – 30.4
5 years *after cleaning*	8,781	220 (2.4%)	16.3	1.6	11.3 – 26.5

SD = Standard deviation

* N at nine months = 11,134, N at three years = 9,793, N at five years = 9,001

Appendix 3: Additional details of cleaning the explanatory variables

3.1 Diet composite score

Table 34 shows which food items (n=53) were included in the seven food groups.

Table 34: Full list and grouping of food items measured in wave 3 (at five years)

Food items	Food group
Bacon, rashers, ham	Processed foods*
Sausage, frankfurters	
Chicken and poultry, e.g. as nuggets or breaded chicken	
Chips, fried potatoes (e.g. waffles etc)	
Savoury breads, e.g. pizza	
Cakes, pastries, buns	Sweet and salty snacks (top shelf foods)
Biscuits - any	
Chocolate or confectionery	
Other sweets	
Ice cream or ice lollies	
Puddings and chilled desserts	
Fruit squash (tropical fruit, lemon barley, etc)	
Blackcurrant only drinks	
Fizzy drinks (not mineral water, sugar free or diet)	
Chips, fried potatoes (e.g. waffles etc)	
Crisps or other packet snacks	
Butter	Spreads and oils
Low fat spread	
Other spreads	
Oil (e.g. vegetable, olive, sunflower)	
Eggs (include in home cooking)	Protein
Baked beans – canned	
Peas , in any form	
Fish or shellfish including fish fingers	
Sausage, frankfurters	
Liver (but not liver products, e.g. pate)	
Beef, e.g. roast, steak, in stews	
Beef, e.g. minced, burgers	
Lamb , e.g. roast, steak, in stews	
Pork, e.g. as a roast or chops in stir fries etc.	
Bacon, rashers, ham	
Chicken and poultry, e.g. as a roast, in casseroles	
Chicken and poultry, e.g. as nuggets or breaded chicken	
Milk (cow's)	Dairy products
Yoghurt (flavoured or plain but not fromage frais)	
Fromage frais (e.g. Petit Filous)	
Cheese or cheese spread	
Ready to eat cereals	Carbohydrate-rich foods
Other breakfast cereals, e.g. porridge	
White bread and rolls	
Wholemeal, brown bread and rolls	
Other bread, e.g. scones, croissants	

Food items	Food group
Savoury breads, e.g. pizza	
Rice, pasta, noodles	
Potatoes	
Leafy green vegetables, e.g. spinach, cabbage	Fruit and vegetables
Other green vegetables, e.g. green beans, broccoli	
Carrots	
Other root vegetables apart from carrots and potatoes, e.g. parsnips	
Mushrooms	
Apples or pears (fresh)	
Soft fruits (e.g. peaches, nectarines, grapes)	
Citrus (e.g. oranges, tangerines, satsumas)	
Banana	
Cucumber	
Fresh tomatoes	
Salad (e.g. lettuce)	
Fruit juice (not squash)	

* the food items in this group appear twice in this table as 'processed food' is the only indicator of dietary quality that does not measure the macro-nutrient intake, whereas items in this group may as well contribute to the total protein or carbohydrate intake of children.

Based on the grouping in Table 34, Table 35 shows how many children under- or over-consumed each food group or met the food pyramid guidelines.

Table 35: Proportion of five-year-old children meeting the food pyramid guidelines

Food group	N below recom-mendation (%)	N recommmen-dation met (%)*	N above recom-mendation (%)	N total (%)
Processed foods	N.A.	7,012 (77.95%)	1,980 (22.05%)	8,995 (100%)
Sweet and salty snacks	N.A.	585 (6.51%)	8,405 (93.49%)	8,990 (100%)
Spreads and oils	384 (4.27%)	7,909 (87.95%)	700 (7.78%)	8,993 (100%)
Protein	43 (0.47%)	3,834 (42.66%)	5,111 (56.86%)	8,988 (100%)
Dairy products	178 (1.98%)	8,126 (90.36%)	689 (7.66%)	8,993 (100%)
Carbohydrate-rich foods	6 (0.07%)	7,928 (88.15%)	1,060 (11.79%)	8,994 (100%)
Fruit and vegetables	165 (1.84%)	8,826 (98.16%)	N.A.	8,991 (100%)

N= Number

N.A. = Not applicable as no minimum or maximum recommendation is mentioned in the food pyramid guidelines.

* = this includes children who were slightly below or above the recommendation but within range for inclusion as meeting the food recommendation

Based on the food pyramid guidelines for children aged five years, the food item frequencies assessed in wave 3 were converted into quality scores using the PDL score, as well as a simplified binary score, details of which are shown in Table 36.

Table 36: Conversion scale used in this thesis to generate composite food scores based on the food consumption labels assessed in wave 3

Food pyramid group	**Scoring system**	**Consumption frequency**								
		Never	< 1 per month	Min. 1 per month	Min. 1 per week	Most days	Once daily	2-3 per day	4-5 per day	≥6 per day
Processed foods	PDL	4	4	4	4	3	2	1	0	0
	binary	1	1	1	1	1*	1*	0	0	0
Sweet & salty	PDL	4	4	4	4	3	2	1	0	0
	binary	1	1	1	1	1*	1*	0	0	0
Spreads & oils	PDL	0	0	0	0	3	4	3	2	1
	binary	0	0	0	0	1*	1	1*	0	0
Protein	PDL	0	0	0	0	3	4	3	2	1
	binary	0	0	0	0	1*	1	1*	0	0
Dairy	PDL	0	0	0	0	1	3	4	3	2
	binary	0	0	0	0	1*	1*	1	1*	0
Carbs	PDL	0	0	0	0	1	1	4	3	2
	binary	0	0	0	0	1*	1*	1	1*	0
Fruit & vegetables	PDL	0	0	0	0	1	2	3	4	4
	binary	0	0	0	0	0	1*	1*	1	1

Note: The Physical activity, diet & lifestyle (PDL) score was developed by Manios et al. (297) and food pyramid recommendations categorised based on their scoring system. The PDL scores indicate: 4 = perfect consumption, 3 = slight over- or under-consumption, 2 = moderate over-consumption, 1 = high over-consumption or moderate under-consumption, and 0 = high under-consumption. The binary food consumption score was developed by the author of this thesis, and scores indicate: 1 = food recommendation was (almost) met, and 0 = the food recommendation was not met.

* = slightly below or above the recommendation but within range for inclusion as meeting the food recommendation

3.2 Unstructured physical activity

Table 37 summarises the distribution of unstructured physical activity in the five-year-old children. The adjusted score was used in this thesis due to low counts in the highest and lowest values of the original range.

Table 37: Distribution of the unstructured physical activity (PA) composite score

PA score	0	1	2	3	4	5	6	7	Total
N original score	52	517	1,358	1,821	2,071	1,729	1,232	140	8,920
%	0.58	5.80	15.22	20.41	23.22	19.38	13.81	1.57	100
N adjusted score	-	569	1,358	1,821	2,071	1,729	1,372	-	8,920
%	-	6.38	15.22	20.41	23.22	19.38	15.38	-	100

N = Number

3.3 Sleep

Table 38 shows the number of children in each group of the NSF sleep recommendations, as well as the number of children in adjusted sleep categories after the data cleaning. The adjusted categories were used in this study in order to increase the counts of children in the shortest and longest sleep categories.

Table 38: Number (N) of children with short, normal and long sleep duration. Sleep duration was grouped by the National Sleep Foundation (NSF) recommendations and in adjusted categories used in this thesis

N (%) in sleep category:	9 months		3 years		5 years	
	NSF	adjusted*	NSF	adjusted*	NSF	adjusted*
Shortest sleep	2,103 (19%)	=	120 (1%)	1,144 (12%)	187 (2%)	1,763 (20%)
Medium sleep (recommendation)	7,668 (69%)	=	8,313 (85%)	7,289 (75%)	8,606 (96%)	5,110 (57%)
Longest sleep	1,324 (12%)	=	1,333 (14%)	=	181 (2%)	2,101 (23%)
Total	11,095 (100%)	11,095 (100%)	9,766 (100%)	9,766 (100%)	8,974 (100%)	8,974 (100%)

* Unless “=” is stated, alternative cut-off values were used because counts in the NSF categories were small

3.4 Birth-weight-for-gestational-age

Table 39: Distribution of birth weight in gram after data cleaning. Descriptive statistics are shown separately for children remaining in the cohort at the ages of nine months, three years and five years

Wave	N	Missing in wave (%)	Type	Range	Mean (SD)
1	10,991	143 (1.30%)	Continuous	453.6 – 5443.2	3465.31 (590.05)
2	9,666	127 (1.30%)	Continuous	510.3 – 5443.2	3473.07 (587.92)
3	8,887	114 (1.27%)	Continuous	510.3 – 5443.2	3479.21 (585.54)

N = Number

SD = Standard deviation

3.5 Equivalised household income

The continuous measurement of annual equivalised household income was skewed, with a number of very high incomes reported. In wave 1, 39 families (0.35%) reported an equivalised income above EUR 80,000, in wave 2, 11 families (0.11%) had an equivalised income above this threshold and in wave 3, 33 families (0.37%). In each wave, there was high variation above this threshold, which led to the skewness of the variable. Due to the possibility that families may be identifiable based on their high income, no exact figures are provided.

3.6 Childcare arrangements

Table 40 presents the detailed distribution of children in childcare arrangements. A clear pattern was seen as to the age when the childcare arrangement had commenced. A likely explanation for this is that childcare arrangements tend to depend on the working routines of parents, changes in which occur infrequent for most families. In fact, most of the nine-month-old children started childcare between the ages of six to eight months, which reflects the length of maternity leave in Ireland (443). Most three-year-old children were in their current childcare

arrangement since their first year of life, and most five-year-old children were in their current childcare arrangement since the age of four years, when many children start attending preschool and childcare needs may change (456).

Table 40: Distribution of main type of childcare used in each wave

Type of childcare	Wave 1 N (%)	Wave 2 N (%)	Wave 3 N (%)
Relative in family home	667 (6.00%)	338 (3.45%)	410 (4.56%)*
Non-relative in family home	429 (3.85%)	432 (4.41%)	525 (5.84%)*
Relative in their home	1,089 (9.78%)	745 (7.61%)	918 (10.21%)*
Non-relative in their home	975 (8.76%)	820 (8.38%)	669 (7.44%)*
Centre-based care	1,222 (10.98%)	2,666 (27.23%)	844 (9.39%)
After School Service care	0	0	171 (1.90%)
Other type of childcare	38 (0.34%)	5 (0.07%)	60 (0.67%)*
No regular arrangement	6,714 (60.30%)	4,785 (48.87%)	5,496 (61.15%)
Total	11,134 (100.00%)	9,791 (100.00%)	8,988 (100.00%)

N = Number

* A number of families in wave 3 mentioned more than one type of informal childcare as their main arrangement, which is why all categories add up to n=9,093 (101.17%).

3.7 Exclusion of observations of explanatory variables

Numbers of observations removed during the data cleaning, in addition to values that were missing in the original dataset, and reasons for removal are provided for each wave.

Table 41: Removal of observations from explanatory variables

Variable	Wave 1	Wave 2	Wave 3	Reason for removal
Sports club membership			15	Caregiver indicated “Do not know”
Screen time		10	3	Caregiver indicated “Do not know”
Antibiotic intake		8	9	Caregiver indicated “Do not know”
Daily sleep duration	9	17	13	Caregiver indicated “Do not know”
Sleep duration during the night	26			Caregiver indicated “Do not know”
	9			Invalid values
				Outlier: child was reported to wake up at 1am on a normal day
Weight of the PCG		363	215	Invalid values
Height of the PCG		197	2	Invalid values
	1			Outlier: 120cm
Weight of the SCG		265	421	Invalid values
Height of the SCG		225	24	Invalid values
	1			Outlier: 122.5cm
Birth weight	12			Outliers: 5.9-8.0kg. This appeared unusual compared to birth weight charts (307) and may bias the analysis nine months later. Furthermore, one of these births was early (36 weeks) and ten within term (37 to 41 weeks), which made the occurrence of these high birth weights even more unlikely. In contrast, 36 children had a birth weight of 5.0-5.4kg who were mostly born after 40 gestational weeks or later, Thus, 5.5kg was used as the threshold for value exclusion.
Breastfeeding onset	3			Caregiver indicated “Do not know”
Onset of solid food	26			Caregiver indicated “Do not know”
PCG education	10	10		Caregiver indicated “Do not know”
		3		Caregiver refused to reply
SCG education	9	6		Caregiver indicated “Do not know”
Neighbourhood perceived as safe	44	35	13	Caregiver indicated “Do not know”
Neighbourhood perceived to offer green spaces	27	19	14	Caregiver indicated “Do not know”

PCG = Primary caregivers; SCG = Secondary caregiver

Appendix 4: Differences between the NCD Risk Factor Collaboration study and the Global Burden of Disease study

Table 42: Characteristics of the NCD Risk Factor Collaboration study and the Global Burden of Disease study

Study details	NCD Risk Factor Collaboration (12)	Global Burden of Disease (15)
Study period	1975-2016	1980-2015
Number of included countries	200	195
Sample size	128.9 million	68.5 million
Age range	5 years and older	2 years and older
Child growth criterion	World Health Organization Child Growth Reference	International Obesity Task Force Child Growth Reference
Type of data	Standardised measurement of height and weight	Standardised and self-reported height and weight data
Type of studies and surveys	Multi-country, national, sub-national and community-level data	Multi-country, national and sub-national data

Appendix 5: List of Irish data included in the NCD Risk Factor Collaboration study and the Global Burden of Disease study

Table 43: Data sources from Ireland included in recent estimates of overweight and obesity trends. Trends were investigated from 1975 to 2016 in the NCD Risk Factor Collaboration (NCD) study and from 1980 to 2015 in the Global Burden of Disease (GBD) study

Years of data collection	Data source	Represen-tation	Age range	Boys N	Girls N	Included in NCD study (344)	Included in GBD study (341)
1985	Kilkenny Health Project (341, 516)	Sub-national	35-64	784		No	Yes
1997-1999	North/South Ireland Food Consumption Survey (344)	National	18-64	613	698	Yes	No
1998	SLAN (341, 344)	National	18+	123	296	Yes	Yes
2001-2002	North/South Survey of Children's Oral Health (340, 341)	National	4-16	8,806	8,712	No	Yes
2002	SLAN (341, 344)	National	18+	164	215	Yes	Yes
2003	Ireland World Health Survey 2003 (341, 517)	National	18+	497	516	No	Yes
2003-2004	National Children Food Survey (344)	National	5-12	293	301	Yes	No
2005-2006	National Teens Food Survey (344)	National	13-17	223	217	Yes	No
2006-2007	SLAN (341, 344)	National	18+	945	1,225	Yes	Yes
2008	COSI 1 (344)	National	6-9	1,098	1,285	Yes	No
2008-2010	National Adult Nutrition Survey (341, 344)	National	18+	658	696	Yes	Yes
2009-1011	The Irish Longitudinal Study on Ageing (341, 344)	National	50+	2,693	3,170	Yes	Yes

Years of data collection	Data source	Represen-tation	Age range	Boys N	Girls N	Included in NCD study (344)	Included in GBD study (341)
2010	Bizzy Break intervention study (344, 352)	Community	7-12	49	41	Yes	No
2010	COSI 2 (344)	National	6-9	1,022	964	Yes	No
2012-2013	COSI 3 (344)	National	6-9	1,087	1,054	Yes	No
2013-2016	Project Spraoi (344)	Community	5-11	474	429	Yes	No
2015	Active Classroom Study (344)	Community	8-11	124	120	Yes	No
Total Children and adolescents			4-17	13,176	13,123	**8,781**	**18,302**
Total adults			18+	5,693	6,816	**11,496**	**11,198**
Total all ages			4+	18,869	19,939	**20,277**	**29,500**

N = Number

SLAN = Survey of Lifestyle, Attitudes and Nutrition in Ireland

COSI = Childhood Obesity Surveillance Initiative

Appendix 6: Distribution of body mass index according to World Health Organization percentiles

Table 44: Changes in the distribution of body mass index (BMI) in all children. BMI in the Growing Up in Ireland infant cohort was compared to the age- and sex-adjusted BMI percentiles defined by the World Health Organization (WHO) growth standard and growth reference

WHO centiles	9 months % (n=10,810)	3 years % (n=9,422)	5 years % (n=8,781)	WHO distribution %	Change from 9 months to 3 years *p-value**	Change from 3 years to 5 years *p-value**
< 0.1	0.11	0.18	0.10	0.10	0.99	0.99
0.1 – 0.99	0.40	0.27	0.23	0.90	0.99	0.99
1 – 2.99	0.52	0.21	0.34	2.00	**0.02**	0.99
3 – 4.99	0.59	0.33	0.49	2.00	0.11	0.99
5 – 9.99	1.81	0.92	1.36	5.00	**0.02**	0.08
10 – 14.99	1.71	0.81	1.82	5.00	**0.02**	**0.02**
15 – 24.99	3.93	2.70	4.42	10.00	**0.02**	**0.02**
25 – 49.99	14.87	11.70	17.93	25.00	**0.02**	**0.02**
50 – 74.99	23.40	23.48	28.72	25.00	0.99	**0.02**
75 – 84.99	13.37	15.19	14.55	10.00	**0.02**	0.99
85 – 89.99	9.14	9.84	8.64	5.00	0.99	0.08
90 – 94.99	10.74	12.18	8.80	5.00	**0.02**	**0.02**
95 – 96.99	5.55	6.09	3.47	2.00	0.99	**0.02**
97 – 98.99	6.98	7.72	4.33	2.00	0.70	**0.02**
99 – 99.89	5.16	6.00	3.12	0.90	0.14	**0.02**
≥ 99.9	1.71	2.40	1.67	0.10	**0.02**	**0.02**

N = Number

*Multiple comparisons adjusting for Bonferroni were used to test changes between two interview waves in the WHO centile groups for significance. Significant p-values ($p < 0.05$) are highlighted in bold.

Table 45: Changes in the distribution of body mass index (BMI) in children breastfed six months or longer only. BMI in the Growing Up in Ireland infant cohort was compared to the age- and sex-adjusted BMI percentiles defined by the World Health Organization (WHO) growth standard and growth reference

WHO centiles	9 months % (n=2,094)	3 years % (n=1,831)	5 years % (n=1,711)	WHO distribution %	Change from 9 months to 3 years *p-value**	Change from 3 years to 5 years *p-value**
< 0.1	0.10	0.33	0.12	0.10	0.99	0.99
0.1 – 0.99	0.62	0.22	0.18	0.90	0.91	0.99
1 – 2.99	0.62	0.16	0.29	2.00	0.38	0.99
3 – 4.99	0.57	0.44	0.58	2.00	0.99	0.99
5 – 9.99	2.24	1.20	1.69	5.00	0.21	0.99
10 – 14.99	2.24	0.87	2.16	5.00	**0.02**	**0.03**
15 – 24.99	5.11	3.22	4.97	10.00	**0.05**	0.13
25 – 49.99	17.05	12.73	19.70	25.00	**0.02**	**0.02**
50 – 74.99	24.45	25.78	29.40	25.00	0.99	0.26
75 – 84.99	12.80	15.29	14.55	10.00	0.40	0.99
85 – 89.99	9.17	8.85	8.24	5.00	0.99	0.99
90 – 94.99	9.46	11.20	7.60	5.00	0.99	**0.02**
95 – 96.99	4.92	5.30	3.21	2.00	0.99	**0.03**
97 – 98.99	5.64	7.05	3.80	2.00	0.99	**0.02**
99 – 99.89	3.87	5.79	2.51	0.90	0.08	**0.02**
≥ 99.9	1.15	1.58	0.99	0.10	0.99	0.99

N = Number

*Multiple comparisons adjusting for Bonferroni were used to test changes between two interview waves in the WHO centile groups for significance. Significant p-values ($p < 0.05$) are highlighted in bold.