Infant and young child growth and nutrition in urban informal settlements in Mumbai, India

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Thesis submitted for the degree of

Doctor of Philosophy

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Declaration

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

Signed: ……. Date: 22 October 2019

Abstract

The overarching question addressed in the thesis is: What are the relationships between socioeconomic position, parental characteristics, and infant and young child growth and nutrition in urban informal settlements (slums) in Mumbai, India?

I answer this question using data from the SNEHA Centres Infant Nutrition Cohort study, an epidemiologic birth cohort of 978 infants born between March 2013 and March 2014 in 20 informal settlements in Mumbai, and followed up till April 2016.

After introducing the topic in Chapter 1, I present a systematic review of longitudinal studies in Chapter 2 to identify the determinants of linear growth in infancy and early childhood. Chapter 3 details the cohort's study design, implementation, and data collection procedures. In Chapter 4 I describe how I used these data to derive my main study variables.

Chapter 5 presents a profile of the cohort at birth, outlining key infant, parental and household socioeconomic characteristics. I also investigate patterns and predictors of missing data and non-response in longitudinal data.

In Chapter 6 I identify the determinants of linear growth between 0-37 months using the SITAR model to fit growth curves to 16 753 length measurements for 944 children. I quantify the relationship between parental anthropometry and child growth.

In Chapter 7 I describe infant and young child feeding practices, and investigate the relationships between baseline characteristics and longitudinal feeding patterns using discrete-time survival and dynamic autoregressive models.

In Chapter 8 I investigate whether the relationship between predominant breastfeeding (0-5 months) and predicted length at 24 months is mediated by consumption of animal source foods at 6-23 months using causal mediation analysis.

Chapter 9 begins with a summary of the main findings of my research. I discuss the empirical and methodologic implications of my study.

Impact statement

My research makes several empirical and methodologic contributions which will be of use to researchers, policymakers, and practitioners in the field of global health, nutrition, and epidemiology.

- I present a profile of a contemporary birth cohort in urban informal settlements in Mumbai, India, providing a detailed understanding of the socioeconomic and household circumstances of children and their families. This will be useful background and contextual information for policymakers and practitioners in urban health and development.
- This is the first study to use the SuperImposition by Translation and Rotation (SITAR) method of growth curve modelling to understand the factors that shape linear growth in a population in urban informal settlements. My research highlights the application and suitability of a sophisticated analytic strategy, and raises additional ideas for expanding the method for future applied biostatistical research and life course epidemiology.
- I provide a detailed description of children's diets as they progress from breastfeeding to family foods through complementary feeding. These results will be of interest to policymakers and practitioners who aim to improve the health and nutrition of young children living in urban poverty. I identify several factors associated with children's diets, and that are amenable to individual and population health action.
- The findings of my systematic review will be of interest to researchers interested in longitudinal growth studies of linear growth, and provide a synthesis of the methods and tools used in current research.
- My thesis will also be of use to researchers in global nutrition with an interest in causal inference, and to epidemiologists and population health scientists who are interested in the application of causal inference methods to contemporary public health issues in urban low-income settings.
- Finally, my findings will complement the work of local government and non-governmental organizations that work to improve the health and nutrition of people, particularly young children, living in informal settlements in Mumbai or other urban contexts.

Dedication

For my mother

Acknowledgements

I've been immensely fortunate to have Professor David Osrin as my primary supervisor. His wisdom, wit, and intelligence have been invaluable in helping me develop my research skills and capacity. He gave me the freedom and support I needed to find my niche, and has been so generous in making me part of the brilliant team he leads at IGH. I also had the most fun chats about urban theory, global health, and epidemiology with him. Professor Jonathan Wells offered excellent advice, discussions, and ideas on all things nutrition, and his supervision and guidance have really helped me think, write, and research better. Professor Bianca De Stavola brought much clarity to my ideas, and helped me explore and develop skills in causal mediation analysis that I've wanted to learn for so long! I could not have asked for a better trio of PhD supervisors.

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Abbreviations

ALSPAC	Avon longitudinal study of parents and children
ANOVA	Analysis of variance
aOR	adjusted odds ratio
AR	Autoregressive
ASF	Animal source food
BF	Breastfeeding
BIC	Bayesian information criteria
BMI	Body mass index
CDE	Controlled direct effect
CF	Complementary feeding
CI	Confidence interval
DAG	Directed acyclic graph
DHS	Demographic and Health Survey
DOHaD	Developmental origins of health and disease
EBF	Exclusive breastfeeding
EED	Environmental enteric dysfunction
EPDS	Edinburgh postnatal depression score
FFQ	Food Frequency Questionnaire
FGR	Foetal growth restriction
GEE	Generalised estimating equations
GOS-ICH	Great Ormond Street Institute of Child Health
GPS	Global positioning system
HAART	Highly active anti-retroviral therapy
HAD	Height-for-age difference
HAZ	Height-for-age z-score
HH	Household
HIC	High income country
HR	Hazard ratio
ICDS	Integrated Child Development Services
IGH	Institute for Global Health
IQR	Interquartile range
IUGR	Intrauterine growth retardation
IYCF	Infant and young child feeding
LAZ	Length-for-age z-score
LBW	Low birth weight
LMIC	Lower middle income countries
LMUP	London measure of unplanned pregnancy
LRT	Likelihood ratio test
MAD	Minimum acceptable diet
MAR	Missing at random
MCAR	Missing completely at random
MDD	Minimum dietary diversity
MGRS	Multicentre Growth Reference Study

MI	Multiple imputation
MNAR	Missing not at random
MUAC	Mid-upper arm circumference
NDE	Natural direct effect
NGO	Non-governmental organization
NIE	Natural indirect effect
OR	Odds ratio
ORS	Oral rehydration solution
PAF	Population attributable fraction
PBF	Predominant breastfeeding
PCA	Principal components analysis
PM	Proportion mediated
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
RCT	Randomized controlled trial
RIA	Randomized interventional analogue
RR	Risk ratio
RRR	Relative risk ratio
RSD	Residual standard deviation
SD	Standard deviation
SDG	Sustainable Development Goals
SE	Standard error
SEM	Structural equation model
SEP	Socioeconomic position
SGA	Small for gestational age
SIM	Shape invariant model
SITAR	SuperImposition by Translation and Rotation
SNEHA	Society for Nutrition, Education, and Health Action
TCE	Total causal effect
TEM	Technical error of measurement
UCL	University College London
UNICEF	United Nations Children's Fund
WAMI	Water and sanitation, assets, incomes, maternal education
WASH	Water, sanitation and hygiene
WHO	World Health Organization
WLZ	Weight-for-length z-score

Chapter 1 Introduction

"People ask me: Why do you write about food, and eating and drinking? Why don't you write about the struggle for power and security, and about love, the way others do?

They ask it accusingly, as if I were somehow gross, unfaithful to the honour of my craft.

The easiest answer is to say that, like most other humans, I am hungry. But there is more than that. It seems to me that our three basic needs, for food and security and love, are so mixed and mingled and entwined that we cannot straightly think of one without the others. So it happens that when I write of hunger, I am really writing about love and the hunger for it, and warmth and the love of it and the hunger for it… and then the warmth and richness and fine reality of hunger satisfied… and it is all one.

I tell about myself, and how I ate bread on a lasting hillside, or drank red wine in a room now blown to bits, and it happens without my willing it that I am telling too about the people with me then, and their other deeper needs for love and happiness.

There is food in the bowl, and more often than not, because of what honesty I have, there is nourishment in the heart, to feed the wilder, more insistent hungers. We must eat. If, in the face of that dread fact, we can find other nourishment, and tolerance and compassion for it, we'll be no less full of human dignity.

There is a communion of more than our bodies when bread is broken and wine drunk. And that is my answer, when people ask me: Why do you write about hunger, and not wars or love?"

Mary Frances Kennedy Fisher (1943)

Summary

In this chapter I introduce the overarching question of my doctoral research:

What are the relationships between socioeconomic position, parental characteristics, and infant and young child growth and nutrition in urban informal settlements (slums) in Mumbai, India?

I discuss the wider literature underpinning my research question, the rationale for the topic and study population, and briefly describe the wider research programme and the SNEHA Centres Infant Nutrition Cohort study that my research was based on. I list the six specific research questions and corresponding objectives addressed in the thesis. I explain my contribution to the study and present an outline of the thesis.

1.1 Height and environmental conditions

The average height of populations is a marker of national health and development (Subramanian et al., 2011), an auxological barometer of a country's success at achieving positive change of one kind or another. But height and its derivatives are simultaneously popular metaphors for privilege (Robertson, 2015) and academic constructs of reproductive or obstetric success (Sear, 2006), the perception of beauty (Bogin and Varela-Silva, 2010), and social mobility and professional achievement (Judge and Cable, 2004). Is stature an all-round measure of triumphs future and past?

Height is also strongly under genetic control, though it is considered a polygenic trait rather than one determined by a small number of genes (Lango Allen et al., 2010). The magnitude of its heritability is much higher in affluent societies, but the genetic basis of height has been reliably demonstrated across populations rich and poor (Stulp and Barrett, 2016). If we, as individuals, largely inherit stature, and height indexes success, what hope exists for the offspring of short parents?

History provides some reassurance. The secular increases in height over successive generations that took place across the globe during the second half of the 20th century have been attributed to improved environments (for example, higher incomes, better healthcare, and less disease) rather than change in the genetic

make-up of populations (Deaton, 2007, McEvoy and Visscher, 2009, Stulp and Barrett, 2016). If genetic change had driven observed trends, rapid increases in such a short amount of time would have been impossible unless a large proportion of the shortest people in society simply failed to reproduce altogether (Stulp and Barrett, 2016). The World Health Organization (WHO) Multicentre Growth Reference Study (MGRS) that underpinned the WHO Growth Standards highlighted a similar view. The authors argued that, under optimal social and environmental conditions, the physiological growth of children from diverse ethnic groups across the world was extraordinarily similar (WHO Multicentre Growth Reference Study Group, 2006a). With a high standard of living, even children of relatively short parents can achieve adult heights that match or exceed their genetic potential. If environment matters, does a rising tide lift all boats?

The high heritability of height suggests, however, that in communities with homogeneous environmental conditions the phenotypic variation in stature would essentially be a result of genetic factors. Perhaps the greater stature of some African communities (compared to Asians) despite poorer environments implies that there are strong genetic factors that perpetuate differences between populations (Deaton, 2007). This is not strictly true. Even subtle variations in crucial conditions between groups living in similar (poor or prosperous) environments can (at least partially) explain observed differences (Stulp and Barrett, 2016). Milk consumption is a prominent example of a factor that can lead to height increases in a population (de Beer, 2012), and the secular trend among the Japanese population coincided with greater milk consumption (Takahashi, 1984). Adult stature is heritable, the growth process less so. All else being equal, do milk-drinking populations grow faster and taller than their lactose-averse neighbours or predecessors?

There is also evidence that the secular trend has its roots in the first two years of life (Cole, 2000, Cole, 2003). The upward trajectory reflects a reduction in the extent of stunting in a population (Cole, 2000), and environmental drivers of the increase in average (adult) height are likely to be those that facilitate less stunting. If countries want taller, and therefore healthier and more productive, adult citizens, should they invest in the linear growth of young children?

1.2 The heightened position of linear growth in the global health and development discourse

The current global health literature on children's linear growth (Branca et al. (2015) is one example) and its apparent consensus stem from two broader ideas. The first is the United Nations Children's Fund (UNICEF) framework adopted in the early 90s (UNICEF, 1990) on the determinants of maternal and child malnutrition (a broad definition encompassing chronic and acute malnutrition manifesting as low height and / or weight, or clinical symptoms that indicate poor nutrition). The second is the life course approach to human health encapsulated by the Developmental Origins of Health and Disease (DOHaD) concept and extended to nutrition by the Standing Committee on Nutrition in 2000 (ACC/SCN, 2000).

The UNICEF framework is embedded within a rights-based approach combined with a political economy perspective, and was first proposed by Urban Jonsson (1981). Maternal and child malnutrition occur as a result of poor dietary intake and frequent disease (immediate determinants), which are themselves related to household food insecurity, inadequate access to health, and poor care practices (underlying determinants). These stem from poverty, a consequence of the social, economic, and political superstructures (basic determinants) in a society. From a child rights view, nutrition does not constitute a separate right, but is implied in the 1990 UN Convention on the Rights of the Child in the sense that the most immediate and underlying determinants of nutrition embody children's right to food, health, and care (Jonsson, 1996). Child nutrition matters because children matter.

The life course perspective suggests that the effects of poor nutrition accumulate over the human life span (and across generations) from conception, in utero, infancy and childhood, adolescence, into the adult reproductive years and on to offspring (for women), with increased metabolic disease in adulthood (ACC/SCN, 2000). Short women are more likely to have preterm or small-for-gestational age babies (Kozuki et al., 2015). However, the life course and intergenerational mechanisms that influence growth in early life and final adult height are much more nuanced than the global discourse suggests. There are several questions and competing hypotheses around the aetiology (or, indeed, existence of a distinct phenotype), mechanistic pathways, and consequences of poor linear growth from evolutionary, developmental, and public health perspectives (see Lampl and Schoen (2017), Martorell (2017), Wells (2012), Wells (2017) for reviews and discussions).

The amalgamation of these two frameworks underlies current global priorities, though ironically cloaked in somewhat utilitarian language about the human capital and gross domestic product losses that widespread malnutrition inflicts on economies. The framework homes in on the nutrition of women, children, and adolescents as windows of opportunity, with particular emphasis on stunting among children under five (Branca et al., 2015). Linear growth faltering, which takes the form of stunting (defined as length-for-age (LAZ) or height-for-age (HAZ) z-score below -2 standard deviations (SD) of the WHO Growth Standards), reflects suboptimal nutrition (poor breastfeeding or complementary feeding) or poor health and living conditions. It largely occurs in the first two years of life, a critical time during which constraints to physical growth and cognitive development (which may or may not have the same causes as linear growth faltering) can prove irreversible (Martorell, 2017, Victora et al., 2010).

The juxtaposition of nutrition and linear growth in the discourse on (sustainable) development takes high-level form in the World Health Organization's (WHO) six Maternal and Child Nutrition targets for 2025 (WHO, 2016). These are linked closely with the Sustainable Development Goals (SDGs) for 2030 (WHO/UNICEF, 2018), and supported by the wider advocacy agenda of intervention in the first 1000 days (WHO, 2013). One of six targets is to achieve a 30% global reduction in the prevalence of stunting. The rest aim to improve breastfeeding practices, halt the rise in childhood overweight, and reduce childhood wasting, anaemia among women of reproductive age, and low birth weight.

1.3 Nutrition and food intake in early life

The importance of infant and young child feeding (IYCF) extends beyond its contribution to children's nourishment and nutritional status, and includes the lifelong immunological, cognitive and psychosocial benefits of breastfeeding (Victora et al., 2016), and the neurodevelopment and flavour preferences that young children acquire through high quality complementary feeding (Agostoni et al., 2008). These (and additional) benefits are applicable to populations across the world, although their protective properties are arguably more critical for children born in deprived settings.

However, the challenge of ensuring good IYCF is somewhat contextual. An infant's transition to solids is marked by the difficulty of meeting high nutrient requirements.

Infants have small stomachs and consume small quantities of food at each meal. Complementary foods need to be nutrient dense (Solomons and Vossenaar, 2013). Once an infant's iron stores are depleted by about six months, their main source of dietary iron is the complementary food they receive alongside iron-poor breastmilk as they transition towards a diet based on family foods. The micronutrient quality of complementary feeding largely reflects the family diet, which can be limited in quantity or bioavailability of iron and zinc in the cereal-laden diets of lower middle-income country (LMIC) populations (Dewey, 2013). Such discussions have a longer history in WHO work on complementary feeding in LMICs (Brown et al., 1998a).

These concerns, validated and reinvigorated by recent findings of large scale Demographic and Health Surveys (DHS) and national surveys showing poor complementary feeding across LMICs (White et al., 2017), have motivated dialogue, action, and calls for more research into how best to improve IYCF practices (Bégin and Aguayo, 2017). Additional research from such surveys suggests that certain components of complementary feeding, such as consumption of animal source foods (ASF), predict length-for-age z-scores (LAZ) (Krasevec et al., 2017), though a systematic review of observational studies on the role of ASF was inconclusive (Shapiro et al, 2017). The issue of deficits in complementary feeding is not the only concern, as other contemporary research suggests that young children's consumption of commercially produced snack foods is widespread across LMICs (Bentley et al., 2015, Huffman et al., 2014, Pries et al., 2017). However, the role of snack foods in shaping nutritional status, health, or early growth patterns remains a significant research gap (Michaelsen et al., 2017).

1.4 Informality and urban existence

If environmental factors shape secular trends rooted in linear growth in the first two years of life, what does that mean for children who grow up in urban poverty?

Informal settlements (or slums) are broadly defined as areas of inadequate housing, water, and sanitation, high density, poor access to basic services, potentially hazardous location, and insecure residential tenure (UN-Habitat, 2003). UN-HABITAT, the United Nations human settlements programme, estimated that in 2014 one in eight people in the world lived in informal settlements or under similar conditions, accounting for about 30% of the urban population in LMICs (UN-Habitat,

2016). In Mumbai, India, informal settlements are home to 40% of the city's households (Chandramouli, 2011).

In reality, informal settlements are evolving spaces that vary vastly in size and density, land entitlement and tenure, built environment, demographic and cultural mix, function, and relationship with the rest of the city (Agarwal and Taneja, 2005). Urban studies researchers have documented morphological features of informal settlements that occupy a spectrum of fluid categories (Dovey and King, 2011), and built environment indicators that are measured at multiple levels (Kohli et al., 2012). Informal settlements often embody (in varying and contextual ways) the sociality and economic activity that render urban life sustainable in the face of poverty and suboptimal living conditions (Dovey, 2015). The stereotypical informal settlement does not exist (Simon, 2011).

The recent global health literature (in the form of a two-paper series (Ezeh et al., 2017, Lilford et al., 2017) published in *The Lancet*) hints at consensus that 'slums' pose unique health (and nutrition, by inclusive extension) challenges distinct from those of urban settings or poverty. Briefly, these include the problem of translating evidence-based health interventions that work in other settings directly to the informal settlement environment, and the neighbourhood effects (the authors vaguely borrow a spatial construct) that operate independently of individual-level factors (Ezeh et al., 2017). Children, who (apparently) are at much greater risk of stunting in the informal settlement environment, can benefit from health promotion activities (to improve breastfeeding) and supplementary feeding interventions (Lilford et al., 2017). The authors suggest that informal settlements are all essentially 'unhealthy places' that merit (for a range of reasons) an academic sub-specialty of their own to prevent them being subsumed by the urban health and development disciplines.

Academic interest in the nutrition of children in informal settlements goes back several decades (see Fernandez et al. (1968) for a study of household diet and nutritional status in Puerto Rico in the 1960s). The tone of some studies is echoed by the more recent (and articulate) theories describing the social determinants of health (Marmot, 2005) and the uniquely urban causes of health that distinguish town from country (Galea et al., 2005), but also much older ideas about the social and economic production of ill health (see Doyal and Pennell (1979)). Cutting and

Kothari (1988) comment on the living conditions observed in Mumbai's Dharavi informal settlement in 1985:

> "Underlying the nutrition problems were the socio-economic problems. Low income, limited education, overcrowding, pollution, social insecurity, and psychological stress all contributed to frequent infections and inadequate nutrition. Of the mothers, a quarter had been born in Dharavi but a third had moved there within the past five years, many from country areas over 1000 km away. Over half the mothers had had no schooling and were illiterate, and 92% were unemployed. Over one quarter of the men were also illiterate; poor employment and bad housing correlated with a lack of education. Some 80% of the infants were born in nuclear family units, and their parents had exchanged the stable homogenous village life with its extended family structure for the linguistic and culturally heterogeneous conditions of the slum."

They then equate these conditions with those observed by Friedrich Engels in Victorian England. Indeed, many researchers have drawn parallels between the urban poverty of the LMIC informal settlement and historical conditions (of high morbidity and mortality) in metropolitan high-income settings; for example, in the slums of New York City in the late 19th and early 20th centuries (Wray, 1986). Vast reductions in child mortality in these now-affluent settings were attributed to the broad notion of 'improved standard of living', of which nutrition was a major component (McKoewn, 1976).

The older and newer global health narratives on informal settlements may seem platitudinous, but they still apply in some respects. While infant and child mortality have reduced sharply across LMICs in the last three decades, urban informal settlements in many countries have higher rates of mortality than even the poorest rural areas (Ezeh et al., 2017). Life in a 21st Century informal settlement can carry a psychological penalty (Subbaraman et al., 2012, Subbaraman et al., 2014), which often stems from the persistence of living conditions similar to those described by Cutting and Kothari.

But the heterogeneity of informality that signals 21st Century urban poverty means that most (if not all) informal settlements defy easy (and lazy) categorization as hell-holes of disease and despair where children are born to be stunted before their second birthdays (see UN-Habitat (2003) for a discussion of 'slums of hope' and 'slums of despair'). There is much variation, and sometimes it is subtle or unmeasured, or simply lacks an adequately theorized framework. Can we make

epidemiological inferences about the determinants of young children's nutrition and linear growth in settings with subtle differences in critical conditions?

1.5 Rationale for research topic and setting

My interest in the topic of child nutrition in informal settlements stems from a combination of theoretical and empirical concerns and the questions they raise.

My theoretical (and corresponding methodological) interests are tied to epidemiologic and public health approaches to linear growth outcomes. First, I am interested in the conceptualisation of the determinants of linear growth in informal settlements. What do we measure in our search for (hopefully modifiable) factors that shape outcomes? And, second, I want to identify (and apply) analytical methods and techniques that are effective and powerful, as obfuscation and lack of plausibility sometimes accompany advanced statistical analyses, and make the task more challenging. What methods can we use to analyse available information to produce answers that are valid?

To some degree, I also want to understand how these questions are related in the way global health research on this topic is conducted. Are intended theoretical aims of inquiry supported by the toolbox of methods currently available to those who study infant and young child growth and nutrition? Does our understanding of the topic stand on solid theoretical and methodological ground when we attempt to extrapolate inferences about child nutrition in LMICs to the urban informal settlement environment?

A more empirical and pragmatic purpose of this thesis is to focus on a population to which I have a personal and longstanding connection, families who live (in informal settlements) in Mumbai, the city I grew up in. Can I identify factors that shape the nutrition and growth of young children born in the city's most deprived areas so that those who seek to improve their health are supported in their efforts of experimentation or implementation by high quality observational evidence that applies to this context?

1.6 Research context

Specifically, I wanted to focus on informal settlements in the north-eastern parts of Mumbai because these are among the most vulnerable in the city, and to use a

longitudinal study design in my research because of the added granularity that sustained observation brings. I also wanted to complement the ongoing research efforts of the Society for Nutrition, Education, and Health Action (SNEHA), a local non-governmental organization (NGO) based in Mumbai. SNEHA have worked to improve the health of women and children in the city's informal settlements since 1999, with an active research collaboration with UCL's Institute for Global Health since 2004.

Between 2012 and 2016, the SNEHA and UCL team conducted a cluster-randomized trial of community resource centres to improve maternal and child health outcomes (see Shah More et al. (2013) for the trial protocol; Shah More et al. (2017) for the primary results paper; and Bentley et al. (2015) for an analysis of IYCF practices based on pre-intervention census data). As part of the SNEHA Centres trial surveillance activities, the team established a birth cohort study, the SNEHA Centres Infant Nutrition Cohort. The study was conducted between 2013 and 2016, focusing on nutrition and growth of children born in Mumbai's urban informal settlements. The aim was to follow up children born in the span of one calendar year for the first two years of their lives. The cohort is described in further detail in Chapter 3.

I had volunteered previously with SNEHA in 2010, working on an adolescent nutrition project, and again between 2011 and 2012 on a family planning project. I was aware of plans for the trial (but not the cohort study). In 2014, I approached Professor David Osrin to ask about PhD opportunities at UCL, with a draft proposal to look at the relationships between parental anthropometry and child growth outcomes in informal settlements. I originally approached the topic from the perspective of the nutrition transition (Popkin et al., 2011) in India's urban informal settlements. We agreed that this broad question could be refined further, and could also be examined using data from the birth cohort study which was to conclude in 2016. The management and programme teams at SNEHA agreed to have me collaborate with them for my doctoral research.

I began my PhD studies at UCL in January 2015, shaping my research to incorporate some of the cohort's original objectives, and refined the cohort analysis plan to align more closely with the most useful and important issues raised in recent empirical and methodological studies. I selected length as the main anthropometric outcome, choosing to focus on only one indicator for the purpose of a doctoral

thesis. The work presented in the thesis is based largely on statistical analysis of data from the SNEHA Centres Infant Nutrition Cohort and additional desk-based research comprising a systematically conducted literature review.

1.7 Research questions

The overarching research question addressed for the thesis is:

What are the relationships between socioeconomic position, parental characteristics, and infant and young child growth and nutrition in urban informal settlements (slums) in Mumbai, India?

I broke this down into six specific questions. The first two apply to the literature on the topic:

1. What analysis strategies are used in the longitudinal assessment of infant linear growth?
2. What are the determinants of infant linear growth?

The final four questions relate to the SNEHA Centres Infant Nutrition Cohort:

1. What were the baseline characteristics of the cohort and how do they relate to patterns of follow-up, attrition, and missing data?
2. What are the determinants of linear growth in infancy and early childhood?
3. What are the determinants of infant and young child feeding?
4. Does consumption of animal source foods in the complementary feeding period (6-23 months) mediate the relationship between predominant breastfeeding (0-5 months) and attained length at 24 months?

1.8 Objectives

I aligned the objectives of my research with the six research questions:

Objectives for questions 1 & 2:

1. Describe the exposures and confounders used in published studies on the determinants of infant linear growth and identify variables that could be included in analyses.

2. Understand the range of growth analysis techniques employed in longitudinal studies of linear growth in the first year of life.

Objectives for question 3:

1. Generate a profile of the cohort at baseline describing characteristics observed at or close to birth.
2. Describe parental anthropometric characteristics observed at least three months after the infant's birth.
3. Investigate relationships between baseline characteristics related to socioeconomic position, infant characteristics, parental health behaviours, and parental anthropometry.
4. Describe rates of participation, follow-up, non-response, and attrition over the study period.
5. Investigate and evaluate reasons for missing data in the cohort and specific analytic subsets of it.

Objectives for question 4:

1. Describe the linear growth patterns among children aged 0-37 months.
2. Identify socioeconomic, parental, and child characteristics associated with linear growth in infancy and early childhood.
3. Understand the relationship between parental overweight and obesity and linear growth in infancy and early childhood.

Objectives for question 5:

1. Describe frequencies of age-appropriate breastfeeding, complementary feeding, and snack food consumption in the cohort at each follow-up age.
2. Determine the probability of discontinuing exclusive or predominant breastfeeding at 0-5 months, and its associations with baseline variables.
3. Determine the probability of infants receiving soft, semi-solid and solid food for the first time at 6-8 months, if they had not yet been given any non-liquid items, and its association with baseline variables.
4. Determine the association of complementary feeding practices with feeding in adjacent periods as well as baseline variables.

Objectives for question 6:

1. Estimate the total causal effect of predominant breastfeeding on attained length at 24 months, and the proportion mediated by consumption of animal source foods in the complementary feeding period.

1.9 My contribution

1.9.1 Supervision and statistical guidance

Professor David Osrin, based at the UCL Institute for Global Health, was my primary supervisor and had overall responsibility for guiding my doctoral training and research activities. I had two subsidiary supervisors from the UCL Great Ormond Street Institute of Child Health (GOS-ICH). Professor Jonathan Wells was my subsidiary supervisor for the full duration of my doctoral degree (2015 to 2019), provided regular mentoring and supervision on all aspects of research, and also chaired the panel for my upgrade from MPhil to PhD in October 2015. Professor Bianca De Stavola joined my supervisory panel in 2018 and led the supervision of research presented in Chapter 8.

In addition, I received statistical guidance from Professor Tim Cole at GOS-ICH in relation to analyses presented in Chapter 6, which also formed the basis of some components of Chapter 8. Additional details of my contribution are presented at the start of both chapters.

1.9.2 Research collaboration and data collection

My PhD research was conducted within a wider research programme led by Professor David Osrin as part of his Wellcome Trust Senior Research Fellowship in Clinical Science (091561/Z/10/Z) from 2012 to 2016, in collaboration with SNEHA. The cohort study was designed by Professor Osrin and Ms Sushmita Das from SNEHA. The research was led by the trial manager, implemented by a team of field investigators, and assisted by two data managers. (See Chapter 3 and its accompanying appendices for a description of the cohort protocol and procedures.)

The conditions of my research collaboration with SNEHA were laid out in a memorandum of understanding (Appendix 1.1) at the start of my degree in 2015.

This covered my access to and analysis of data generated in the SNEHA Centres programme.

I did not collect any data for the cohort study used in the thesis. However, I lived in Mumbai between October 2015 and April 2016 and was able to observe the last six months of data collection and interact with the study team as the cohort neared completion of follow-up to two years of age. I presented at SNEHA Research Group Meetings and contributed to ongoing discussions related to the trial and SNEHA's work on nutrition. I received the cohort dataset for analysis in July 2016.

1.10 Ethical approval and funding

I received ethics approval for my PhD research from the UCL Research Ethics Committee in September 2015 (Appendix 1.2). I self-funded my PhD course fees and living expenses. I also paid for all travel and accommodation expenses in Mumbai between 2015 and 2016. While I did not receive any funding for my PhD, I did receive small grants to present my research at conferences in the UK and overseas. These included awards from the UCL School of Life and Medical Science Student Conference Fund (2016), the Society for the Study of Human Biology Postgraduate Travel Prize (2017), and the Yusuf Ali Travel Grant from UCL (2017).

1.11 Outline of the thesis

The thesis consists of nine chapters, including this chapter.

In **Chapter 2**, I present my systematic literature review, addressing Questions 1 and 2.

In **Chapter 3**, I describe the SNEHA Centres Infant Nutrition Cohort study protocol and data collection procedures.

In **Chapter 4**, I explain how I used the cohort dataset to derive my study variables and the ways in which they were coded or parameterized, and discuss some statistical considerations underpinning my research.

Chapter 5 addresses Question 3, providing a detailed description of the cohort at baseline, presented alongside an investigation of follow-up, attrition, and missing data in longitudinal measurements.

Chapter 6 focuses on Question 4, presenting the results of linear growth analysis based on the SuperImposition by Translation and Rotation (SITAR) model.

In **Chapter 7**, I examine IYCF practices in the cohort (Question 5), and quantify their associations with background factors using discrete-time survival analysis and dynamic autoregressive models for longitudinal data.

Chapter 8 aims to quantify the relationship between IYCF and attained length at 24 months (Question 6), which I examined using a counterfactual-based causal mediation analysis.

In **Chapter 9**, I summarise the key findings of my research, discuss my empirical and methodological contribution to the field, and provide concluding remarks.

Chapters 2, 5, 6, 7, and 8 are results-based chapters and are similarly structured. Each has its own methods and discussion sections book-ending the presentation of findings, preceded by a summary of theoretical or methodological issues pertaining to the research question.

Chapter 2. Literature review

Summary

In this chapter I present my literature review on the determinants of infant linear growth. The purpose of the review was two-fold: I wanted to identify covariates used in previous studies to inform my conceptual framework of the determinants of infant growth, and also to understand the range and type of metrics and statistical approaches commonly used to quantify infant linear growth. I conducted a systematic search of two databases, and carried out the screening, extraction, syntheses, and quality assessment alone. I carried out a narrative synthesis of the 77 studies included in my review, presented the findings alongside summaries of four recent reviews in the field.

2.1 Introduction

2.1.1 Conceptual grounding and recent reviews of child growth

The UNICEF framework on the causes of malnutrition (UNICEF, 1990) has been used extensively for research, programme planning and implementation, policy development, and global advocacy. It proposes a hierarchical set of immediate, underlying and basic factors that lead to maternal and child malnutrition. It has been flexible as a theoretical and conceptual tool, with several adaptations (Black et al., 2008, Black et al., 2013) and extensions (Engle et al., 1997a, Engle et al., 1997b) in the light of new empirical evidence and multidisciplinary thinking. The most recent iteration adopted by WHO (Stewart et al., 2013) on the context, causes and consequences of stunted growth and development retains an acyclic structure, but shifts the focus by putting stunted growth and child development – both of which result, fully or at least partly, from biological and psychosocial deprivation within the first 1000 days of life – at its centre. To incorporate stronger methods, metrics and evidence on food intake in early life (WHO, 2008a, WHO, 2010), the WHO framework explicitly recognizes the role of complementary feeding in promoting healthy growth and development. It also more specifically highlights age-appropriate milestones and markers of infant environment and feeding practices, indicating that any causal effects are time-sensitive. (The review article presenting the WHO framework is summarised in the Results section of this chapter).

Recent reviews have synthesised evidence on the determinants of poor linear growth in infancy and childhood, identifying contextual factors that contribute to stunting in LMICs (Black et al., 2013), in regions such as Sub-Saharan Africa (Akombi et al., 2017), or in a specific national context, such as Indonesia (Beal et al., 2018) and Ethiopia (Wirth et al., 2017). These narrative reviews often use the UNICEF framework on the causes of malnutrition (UNICEF, 1990) or the related WHO conceptual framework (Stewart et al., 2013) to guide data analysis. They also highlight factors that are not explicitly listed in the chosen framework, but have been identified as determinants of stunting, such as paternal height or parental smoking in Indonesia (Beal et al., 2018), or malaria in some African countries (Akombi et al., 2017) . While the determinants of stunting may vary by context, early growth faltering is widespread across LMICs.

2.1.2 Methodologic limitations

A recent pooled analysis of cross-sectional DHS data compared the growth of children 0-36 months of age in LMICs to the trajectory implied by the WHO growth standards, using mean HAZ at each age to construct a population-level growth curve (Roth et al., 2017). The authors interpreted decreasing mean HAZ with increasing age as an indication that children across the full HAZ distribution grow slower than the international standard. They also argued for a shift towards intervention that address community-level factors that shape population growth trajectories.

The reviews focus on indicators of malnutrition – usually wasting, underweight and stunting – which are measured cross-sectionally in surveys and case-control studies, or longitudinally in intervention studies that assess change in stunting incidence or risk in response to particular treatments or changes in an exposure in a defined period. While these reviews identify factors associated with poor growth outcomes that manifest as malnutrition, their focus on size attained at some age relative to an idealized growth standard does not position them to shed light on what factors shape the *rate* at which infants grow or the age at which growth faltering occurs for particular individuals or groups. Further, by adopting a standard definition of stunting as the outcome, the reviews exclude most birth cohort studies that do not use the WHO Growth Standards to assess linear growth for methodological reasons, including those examining the influence of poverty, food intake and parental factors on growth in early life in high-income country (HIC) populations.

Using cross-sectional survey data, as done in Roth et al's pooled analysis of DHS data, to derive population-level growth trajectories which can help identify intervention strategies is also problematic. The population average at each time point is not an adequate characterisation of the average trajectory or deviation from a growth norm of individual children (Tu et al., 2013), especially when the samples at each time point represent data from different sets of children rather than measurements on the same set of children followed from birth.

Longitudinal data enable analysis of temporal associations between earlier risk factors and concurrent growth processes or later growth outcomes. Determinants of growth identified from a review of cross-sectional studies would not indicate the direction of relationships, and would also leave out risks that can only be examined concurrently or longitudinally (e.g. biomarkers at specific time points, recurrent episodes of exposure, and markers of rapid feedback between exposure and outcome). The set of factors that influence attained length-for-age may not overlap completely with those that affect the process and pattern of linear growth in the first year of life.

Analysis of longitudinal growth data from the first year of life is particularly challenging (Hauspie et al., 1980), and these reviews of cross-sectional studies offer little insight into methodological issues in linear growth assessment, which needs to marry considerations of statistical modelling with growth physiology in order to accurately and meaningfully characterise growth (Lampl, 2012). As knowledge of the biology of human growth has evolved, methods to measure and assess it have also seen technological advances, such that older statistical methods are no longer sufficient to take account of new biological knowledge (Lampl and Mummert, 2014).

2.1.3 Review question

My objective was to describe the exposures and confounders used in published studies on the determinants of infant linear growth and identify variables that I could include in my analyses, and to understand the range of growth analysis techniques employed in longitudinal studies of linear growth in the first year of life. The two questions for my literature review were:

1. What growth analysis strategies are used in the longitudinal assessment of infant linear growth?

2. What are the determinants of infant linear growth?

2.2 Methods

2.2.1 Search strategy

I searched two databases, PubMed and Scopus, and combined results for screening, data extraction and quality assessment. I carried out the search on 19 June 2018.

I was interested in infant growth as an outcome, but did not select a specific exposure, keeping the search open to identify a range of causes or determinants. I used free text search terms, developing a list of keywords and synonyms related to 'infant linear growth' and 'causes'. I used the Boolean operator 'OR' to combine search terms within each concept, and 'AND' to combine results of the two concepts. This strategy was refined through exploratory searching. This led to an unmanageable number of results in the first instance, with many irrelevant studies. I decided to make my search more sensitive by excluding studies of animals (pigs, monkeys, etc), non-infant populations (foetus, adolescents), twins, preterm infants, studies of gestation and pregnancy outcomes, growth disorders (Down's Syndrome, achondroplasia, etc), paediatric illness (cancer, HIV, acute renal failure, etc), and markers of sensory development in infants (language, speech, hearing, etc). I used the Boolean operator 'NOT' to exclude these terms from the search results on infant linear growth and its determinants. (Appendix 2.1)

I was interested in infant length examined at two or more time points per participant, which implies a longitudinal or follow-up study design, but did not use search terms specifying this, choosing to categorise studies as cross-sectional or longitudinal at the filtration and screening stages. I did not specify the number of data points used to calculate growth from available measurements, as this could be one, two, or more than two. For example, studies that measured infants every month for a year could use all 12 data points to model an individual's growth curve and derive velocity (cm/month), or use the HAZ based on a single data point to describe size in each follow-up period. In both cases, the study would be classified as a longitudinal study of growth, but with the growth metric in the former derived from multiple data points, and from one data point in the latter.

I did not use search terms to exclude studies on infant weight from my strategy because authors often report analyses of weight and length in the same article. I did not specify a geographic focus as I wanted to identify studies in high-income countries that examine growth in low-income and vulnerable communities where the experience of certain risk factors would potentially be similar to that in urban informal settlements in LMICs.

I exported search results to EndNote X7 to conduct title and abstract screening and full-text review, and to identify studies eligible for data extraction.

2.2.2 Inclusion and exclusion criteria

I included quantitative, observational studies from peer-reviewed sources in which infant length was a study outcome, measured by trained investigators in the supine position at two or more time points between birth and 12 months, or linked to medical records; and in which statistical analysis to quantify the relationship between at least one exposure variable and an infant linear growth parameter was reported.

Infant linear growth was defined as any change in length with age, and parameters included any conditional growth measures, growth curves, and patterns of growth derived from serial measurements. I defined infancy as the full first year of life, but did not specify the duration as an inclusion criterion; studies that covered parts of the first year (0-6 months, 6-18 months) were also eligible. Studies on singleton, live-born infants in any part of the world were included.

Articles published between 1 January 2010 and 19 June 2018 were included, although this criterion was applied at the full-text screening stage to prevent filtering out older articles that were of conceptual and methodological relevance to other sections of the thesis. I chose this period in order to make the review process manageable.

Studies that were related to infant growth theory or research methods were excluded from the review, but were collated in a separate list of relevant literature. I also examined the reference lists of included studies to identify additional papers that met the inclusion criteria.

I applied the following exclusion criteria:

- Multiple births
- Infants with diseases or growth disorders
- Studies on pregnancy or birth complications
- Animal studies
- Growth in infancy as an exposure for outcomes later in life
- Growth not measured in the first year of life (0-12 months)
- Self-reported (by parents) length measurements
- Rare exposures at the population level (e.g., gas leaks, natural disasters)
- Descriptive studies without analysis of determinants
- Primary analyses of randomized or non-randomized intervention or evaluation studies
- Cross-sectional assessment of infant length
- Studies published before 1 January 2010 (applied at full-text stage)

2.2.3 Quality assessment and risk of bias

Rating observational studies is a challenging task due to the high risk of selection bias. A further challenge relates to the methodological flaws that can occur in the design, conduct and data analysis of observational studies. The following five domains are crucial components of checklists that are used to assess the quality of observational studies: comparability of subjects, exposure, outcome measurement, statistical analysis, funding or sponsorship (West et al., 2002). I adapted the Newcastle-Ottawa Quality Assessment Scale for Cohort Studies (Wells and Shea, 2018) which includes three categories: selection of participants (representativeness, selection, exposure assessment, and demonstration that the outcome was not present at the start of the study), comparability (control for confounders), and outcome (assessment, follow-up time, and adequacy of follow-up).

I adapted the tool for my review by specifying criteria in the comparability and outcome categories. In the comparability category, I listed infant sex as an essential confounder (or effect modifier) and modified the second item to include *a priori* justification for confounders, rather than a particular variable of interest. In the outcome category, I applied arbitrary cut-offs for two items. I specified the full first year of life as adequate follow-up time, and an attrition of 10% or less (without an examination of missingness) or examination of missingness (if >10%) as evidence

of adequate participant follow-up (Appendix 2.2). I calculated overall scores (out of 9) and within each category following the scale's star-based rating system to understand the sources of bias in included studies.

2.2.4 Data extraction and synthesis

I created a data extraction framework in MS Excel for full-text articles and online supplementary material (where applicable), and conducted quality assessment and analysis in linked spreadsheets. I carried out additional data management and produced summary statistics in Stata SE version 13.

I extracted information on study design, location, participants, follow-up ages and study time, growth analysis methods, variables included in the analysis (exposure, outcome, confounders, and mediators), analytical strategies, main results (determinants of linear growth), and conclusions (Appendix 2.3).

Since I did not focus on specific exposures or a particular definition of infant growth, a meta-analysis was not relevant; the heterogeneity of study design, exposures, outcome assessment, and analytical methods would have made quantitative analysis infeasible. I adopted a narrative synthesis approach, summarising the range and types of study design, growth metrics and analytical strategies used (Table 2.1). I used the definitions and categories of growth metrics (Appendix 2.4) summarized in (Leung et al., 2018), and adapted a list of analytical methods for observational studies summarised in a medical statistics textbook (Kirkwood and Sterne, 2009) as coding frameworks to organize my methodological findings. I also examined the data reduction strategies in each article to assess whether results were based on *a priori* selection and specification of exposures and confounders, and how final models were selected or evaluated. I generated a list of exposures and covariates and categorized them into thematic groups by age-group or unit of observation.

Table 2.1 Categories of study design, growth metrics, and statistical analysis methods

Study design	
Approach	**Type**
Observational	Follow-up of cross-sectional study participants Retrospective record linkage Prospective birth cohort Prospective cohort Prospective open cohort Multi-site cohort

	Pooled analysis of cohort studies	
Experimental or intervention	Secondary analysis of trial data Follow-up study of trial participants	
Hybrid	Pooled analysis of cohort and trial follow-up studies Prospective observational cohort nested in trial	
Growth metrics		
Component	**Range**	
Standardization	(1) Raw (2) Standardized	
Level of estimation	(1) Group (2) Individual	
Metric type	(1) Continuous (2) Categorical	
Quantity of data	(1) 1 data point (2) 2 data points (3) 2+ data points	
Metric sub-type	(1) Mean (2) Proportion (3) Incremental change (4) Incremental rate of change (5) Instantaneous rate of change (6) Proportional change (7) Proportional rate of change (8) Conditional difference (9) Age-scaling factor (10) Tempo (11) Maximum or minimum (12) Velocity z-score (13) Class (14) Other	
Analytical approach to derive metric	(1) Manual or simple calculation (2) Threshold values (3) Child-specific pre-defined structural model (4) Child-specific data-driven regression model (5) Linear fixed effect regression (6) Non-linear fixed effect regression (7) Linear mixed effect regression (8) Non-linear mixed effect regression (9) Conditional regression (10) SITAR (11) Growth mixture model (12) Latent growth curves (13) Machine learning (14) Generalized estimating equations* (15) Other	
Statistical analysis methods		
Type of outcome	**Strategy**	**Additional details**
Cross-sectional numerical	Linear regression	Simple linear regression; paired t-test, multiple linear regression; OLS regression; one-way ANOVA; multivariable ANOVA
Cross-sectional binary	Logistic regression	Chi-squared test, multivariable logistic regression, Mantel-Haenszel methods, Chi-squared test for trend
Rate	Poisson regression	Z-test, Mantel-Haenszel methods, Poisson Regression (extensions)
Survival time	Cox regression	Cox regression (with non-proportional odds); Weibull, Gompertz, or others; log rank test
Longitudinal or clustered outcome	Fixed effect regression	With or without robust standard errors to allow for clustering
Longitudinal or clustered outcome	Random effect regression	Dynamic models, linear mixed effects, non-linear mixed effects
Longitudinal or clustered outcome	Generalized estimating equations	GEE logistic, GEE linear, extensions of marginal models
Longitudinal or clustered outcome	Repeated Measures ANOVA	For balanced designs
Any (mediated outcome)	Mediation analysis or effect decomposition approaches	Path analysis; Structural Equation Modelling (SEM); other causal mediation analysis with or without time-varying exposures, outcomes, or covariates
Any	Other	Instrumental variable analysis (trial data), seemingly unrelated regression (panel data)

*was not included in Leung et al. (2018)

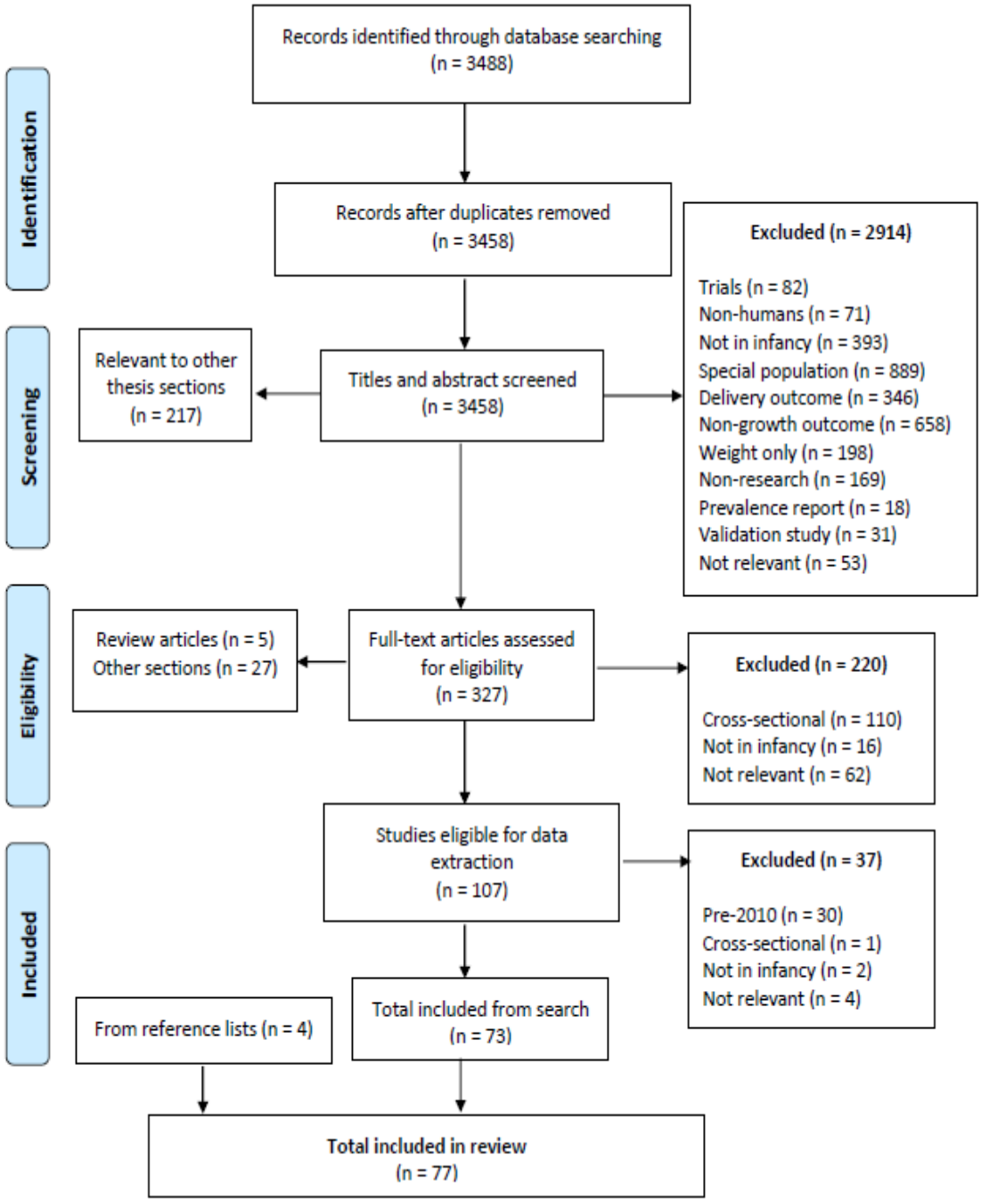
2.3 Search results

2.3.1 Details of included and excluded studies

I identified 3,488 articles, and after removing duplicates, screened titles and abstracts of 3,458. I included 327 articles for full-text screening, of which 107 were eligible for data extraction. I excluded 30 studies published before 2010, 7 that did not meet other inclusion criteria, and retained 73 for data extraction. I identified 4 articles from scanning reference lists of those already included for data extraction. The total number of articles included was 77 published since 2010. Figure 2.1 presents a PRISMA flowchart describing the number of studies and reasons for inclusion and exclusion.

At the full-text screening stage I also identified 5 review articles – 4 on the determinants of linear growth and 1 on growth metrics in early childhood – that were relevant to my topic but not eligible for data extraction. In the next section I summarise each of these articles to contextualise my study within the wider literature on linear growth in early life, followed by presentation of my review in the subsequent section.

Figure 2.1 PRISMA flow-chart of selection of studies for review



2.4 Summary of four recent review articles

2.4.1 Determinants of infant linear growth

I did not find any systematic reviews synthesizing evidence from longitudinal studies on factors affecting infant linear growth, characterized using two or more length measurements per participant. Instead, conceptual papers and systematic reviews identified in my search adopted stunting in early childhood as the outcome of interest, citing its importance as a marker of growth accumulated pre- and post-natally, and its relevance to the global SDG or national health agendas (Bhutta et al., 2013, Black et al., 2008, Black et al., 2013).

I describe four reviews that focus on the determinants of infant linear growth: Prendergast and Humphrey's (2014) narrative review describing the mechanisms leading to linear growth failure at different ages across the life course, which characterise the 'stunting syndrome' widespread in LMICs; Stewart et al's (2013) review positioning complementary feeding within a wider stunting-prevention framework; Danaei et al's (2016) systematic review and estimation study of the population attributable fraction (PAF) of stunting for 18 risk factors to estimate their causal effect on stunting; and Hermanussen and Wit's (2017) critical review questioning the causal link between nutrition and linear growth.

2.4.1.1 Review 1: Stunting syndrome

Prendergast and Humphrey (2014) define the stunting syndrome as a condition in which "multiple pathological changes marked by linear growth retardation increase morbidity and mortality and reduce physical, neurodevelopmental and economic capacity", with short-, medium- and long-term consequences that render the stunting process intergenerational and cyclical. They divide the life span into the first 1000 days – from conception to 2 years of age, including pregnancy and the neonatal period – and the period between 2 years and adulthood, punctuated by childhood (school age) and puberty. The cycle continues when women become pregnant. Within each period, they reviewed evidence on causative or aggravating factors that contribute to age-specific outcomes among neonates (LBW, SGA, prematurity) and in infancy (HAZ <-2SD, increased morbidity and mortality, delayed motor skills), with a divergence of outcomes from childhood until adulthood: further undernutrition or stunting in resource-constrained environments, or overweight and obesity in addition to extant stunting in an environment of excess food and calorie intake. These

divergent outcomes ultimately converge at conception, leading to stunting in the next generation.

Describing age-specific factors that shape the pathogenesis of stunting, the authors cite evidence for several maternal factors in pregnancy (inadequate diet, intrauterine infection, systemic infection or inflammation, environmental enteric dysfunction (EED), and ambient air pollution), and between birth and two years (introduction of non-breastmilk food before 6 months of age, inadequate complementary feeding practices, poor WASH which leads to diarrhoea and EED, recurrent infection, exposure to pollutants and toxins, poor infant stimulation, and maternal depression) that lead to stunted growth.

They acknowledge that stunting begins *in utero* and linear growth continues to falter for the first two years of life, with marked deceleration soon after birth in LMICs. However, population-level trajectories do not necessarily describe the process of linear growth that individual children experience. There is extensive between-child variability in growth curves and the timing at which growth faltering becomes apparent, which needs to be studied and characterised more clearly to inform action. They also cite debates on the validity of HAZ as an indicator with which to quantify change in length between two or more time points (Leroy et al., 2015, Lundeen et al., 2014). The denominator for HAZ – the age- and sex-specific standard deviation for height – increases with age, and a constant absolute deficit in height would lead to an increase in HAZ over the study period and result in apparent – and misleading – improvements in linear growth.

2.4.1.2 Review 2: Complementary feeding to address stunting

Stewart et al's review (2013) presents the WHO conceptual framework on childhood stunting. They argue that even though stunting begins *in utero*, complementary feeding is a central opportunity for intervention because most of the postnatal decline in HAZ occurs between 6-24 months, and stunting is associated with poor complementary feeding in this age group. Their review extends the UNICEF framework, first describing several immediate causes of stunted growth – including inadequate complementary feeding – and then demonstrating how contextual (basic and underlying) factors affect complementary feeding, eventually leading to stunting. Citing evidence for a range of factors that are immediate causes (household and family environment, inadequate breastfeeding and complementary feeding, and infections), they then examine the pathways through which six contextual causes

made up of community and societal factors act on complementary feeding. These six interacting and overlapping factors include political economy, health and healthcare, education, society and culture, agriculture and food systems, and water, sanitation and environment.

They argue for multidisciplinary thinking in addressing complementary feeding, and use evidence from a range of disciplines to describe crucial variables within each factor that are amenable to action. Within political economy, the most important variables are food prices and trade policy, marketing regulations, political stability, poverty, income and wealth, financial services, employment and livelihoods. Health and healthcare-related variables include access to healthcare, qualified healthcare providers, availability of supplies, infrastructure, and healthcare systems and policies. Education at the community or societal level refers to access to quality education, qualified teachers, qualified health educators, and infrastructure supporting schools and training institutions. Society and culture influence complementary feeding by shaping beliefs and norms, social support networks, child caregivers, and women's status. Agriculture and food systems play a role in food production and processing, availability of micronutrient rich foods, and food safety and quality. Finally, WASH and environmental variables include WASH infrastructure and services, population density, climate change, urbanization, and natural and manmade disasters.

The authors deliberately omit, but explicitly acknowledge, crucial factors that are not amenable to action, particularly genetic influences on infant growth and development.

2.4.1.3 Review 3: Causal effects of 18 risk factors for childhood stunting

Danaei et al's (2016) modelling study presents a global, pooled quantitative analysis of data from population surveys to estimate the relative contribution of risk factors implicated in the burden of linear growth faltering at the end of the 1000 days period. They interpreted these associations as causal because they include only factors for which they deemed the available evidence as convincing. An estimate of the number of cases of stunting among children aged 24 to 35 months in developing countries in 2011 was the study outcome.

Modifiable risk factors were selected for analysis based on three criteria: first, availability of nationally-representative exposure data of high quality; second, robust

evidence for an association with stunting; and third, recent effect size estimates from meta-analyses. Evidence was considered robust when the risk factor's relationship with stunting was convincing (corroborated by more than one study type, e.g. cohort studies, RCTs) or probable (from at least two independent RCTs or cohort studies, or at least five cross-sectional or case-control studies), and included in the estimation model if national data on risk factor prevalence were available for all countries.

Their model included 18 risk factors arranged in five groups: Group 1: maternal nutrition and infection (maternal short stature, underweight, malaria in pregnancy, and anaemia); Group 2: teenage motherhood and short birth intervals (age at delivery <20 years, and <24 month intervals between consecutive births); Group 3: foetal growth restriction (FGR) and preterm birth (preterm SGA, preterm AGA, term SGA, LBW); Group 4: child nutrition and infection (zinc deficiency, diarrhoea, non-EBF, discontinued BF, HIV without HAART); Group 5: environmental factors (unimproved sanitation, unimproved water, use of biomass fuels). With the exception of maternal malaria, childhood zinc deficiency, and use of biomass fuels, which were included based on evidence from systematic reviews of RCTs, all risk factors had an evidence base consisting of pooled analyses of DHS data or systematic reviews of observational studies (cohort or non-cohort studies).

In their statistical analysis, the authors calculated PAF for each risk factor using effect sizes from epidemiological studies, quantifying its independent effect on stunting while holding the rest constant. For maternal characteristics (malaria, underweight, and anaemia) and biomass fuels, meta-analyses reported on LBW as the outcome. The PAF for stunting was calculated by multiplying the PAF of LBW for each risk factor by the PAF of stunting attributable to LBW. This method was replicated for non-EBF and discontinued breastfeeding studies that reported diarrhoea as the outcome. Combined effects of factors in each cluster, with assumptions of multicausality or mediation, were also calculated, before producing country-specific attributable prevalence estimates for individual and clusters of risk-factors.

They identified being term SGA as the most important individual determinant of stunting (accounting for 10.8 million (95% CI 9.1 million, 12.6 million) of 44.1 million cases of stunting globally), followed by unimproved sanitation (7.2 million (95% CI 6.3 million, 8.2 million) cases) and diarrhoea (5.8 million (95% CI 2.4 million, 9.2

million) cases). Indicators of FGR and preterm birth formed the most important cluster of determinants (PAF of 32.5%), followed by environmental factors (21.7%), maternal nutrition and infection (14.4%), child nutrition and infection (13.5%), and teenage motherhood and short birth intervals (1.9%). While FGR and preterm birth was the leading risk factor across all regions, environmental factors (especially unimproved sanitation) were the second leading cluster in South Asia, East Asia and the Pacific, and sub-Saharan Africa. Child nutrition and infection (especially diarrhoea) were second leading in Latin America and the Caribbean, Middle East and North Africa, and Central Asia. The top five risk factors (with the greatest PAF) for India were term SGA, unimproved sanitation, childhood diarrhoea, maternal short stature, and biomass fuel use. The bottom five (with the lowest PAF) were short birth intervals, teenage motherhood, discontinued BF, unimproved water, and maternal malaria.

2.4.1.4 Review 4: Is there (really) a causal link between nutrition and linear growth?

Drawing on historical as well as more recent literature, Hermanussen and Wit (2017) argue that the relationship between nutrition and linear growth is not as clear as is generally assumed. Their historical analysis centres on Ancel Keys' observations in a text published in 1950 on the short- and long-term effects of food shortages and human starvation in the late 19th and first half of the 20th centuries in European countries (Keys et al., 1950). Historic data from the two World Wars showed that very severe calorie restriction was necessary to induce large decreases in birthweight (as a marker of prenatal growth), and many populations that endured difficult conditions in early life did not have marked deficits in adult height. Among older children who lived through food shortages of the 1940s, the drop in rate of growth in height was not substantial when the food crisis was short lived.

Citing recent Cochrane reviews and meta-analyses of supplementary feeding interventions in LMICs, they argue that evidence of catch-up growth after community-based (Sguassero et al., 2012) or targeted interventions in socioeconomically deprived groups (Kristjansson et al., 2015) is inconclusive (Sguassero et al., 2012) or limited in effect (Kristjansson et al., 2015). This contrasts with evidence from animal experiments and re-feeding of starved post-war European populations for whom improved nutrition resulted in marked catch-up growth. They also cite another meta-analysis of variability in height observed in 833 studies from 78 countries, showing that height and weight gains are independent of

each other (Mumm et al., 2016). They use this as further evidence that short stature is not a result of poor nutrition, questioning the causal link between nutrition and growth.

To provide an alternative view, they point to a more recent proposition based on experimental animal models of competitive growth in cooperative mammals, where size is a sign of dominance within a group and social status is a stimulus for strategic growth adjustments. They hypothesize that height could function as a signal of status in human populations too, such that an as yet poorly understood mechanism of social height targeting shapes height trends and increases during prolonged periods of equal opportunity or political turmoil enabling upward mobility. Nutrition, living conditions, health, and care are pre-requisites for growth – not to maximize height, but to allow for height to serve as a social signal among groups within populations. In many populations, secular height increases originate in the lowest social strata. Among lower social strata, probabilistic assessments at group level that lead to strategic growth adjustments and height increases are perceived as social challenges by higher social groups, which respond by smaller increments and thus set a new target. This hypothesis of community-based targeting in growth during childhood and adolescence is presented as an alternative explanation for rapid height increases among migrant groups, and the parallels between political changes and secular trends in attained height since the 19th century (Hermanussen and Wit, 2017).

Turning their attention to the WHO Growth Standards, Hermanussen and Wit raise methodological concerns about using normative growth charts to depict growth faltering in populations that exhibit wide within- and between-group variability in height. Since the WHO Growth Standards are about 1 SD score above healthy South Asian children, they are likely to over-diagnose stunting in otherwise healthy and normally-growing children. Citing the combination of poor causal effects of nutrition on growth and the methodological misfit of the WHO Growth Standards, they propose that local growth references might be more appropriate. They suggest that Synthetic Growth References (Hermanussen et al., 2016) – which combine local growth data from a population of interest and universal features of human population growth to produce reference curves from birth to maturity – could be a suitable context-specific alternative to the WHO Growth Standards.

2.4.2 Growth analysis strategies

Leung et al. (2018) published the only recent systematic review mapping metrics used in recent epidemiological studies of early childhood growth (length, weight and BMI). Their scoping review included eligible studies published between October 2015 and June 2016, and a 10% random sample of older studies (January 2010 to June 2016). The authors designed a data extraction tool for their review to identify metric labels used in studies (descriptive terms like 'length velocity' or 'conditional gain'), and metric content capturing the conceptual and statistical characteristics of a growth metric. They used six components to produce content signatures for each metric:

1. Standardization (raw measurement or standardized values)
2. Level of analysis (group or individual)
3. Metric type (continuous or categorical)
4. Quantity of data (2 or >2 measurements per individual)
5. Metric subtype (quantification or parametrization of the metric)
6. Analytical approach (method of categorizing, calculating or estimating growth)

(See Appendix 2.4 for full definitions of content components).

They found 40 unique growth assessment metrics in 122 studies; 64 studies (52%) measured linear growth using 20 unique metrics, and over three-quarters of these used length as a study outcome. The most common metric was incremental change in a standardized length parameter between two time points calculated manually, often specified as 'linear growth', 'gain', or 'change'. Conditional change in standardized length parameter between two time points derived from conditional regression (using the residual from regression of current HAZ on previous HAZ) was the second most common metric. Incremental rate of change in unstandardized length between two time points calculated manually (third most common), or based on more than two measurements estimated from a linear mixed effects model (fourth most common) were also reported.

In their critical appraisal, they highlighted several methodological gaps in current use of growth metrics. Few studies explicitly justified their choice of growth assessment approach, even when it was appropriate. The considerable heterogeneity in metric

use could be one explanation for the inconsistent relationships between growth and other factors across studies, especially since measures of conditional growth that account for previous size are interpreted differently from results of methods that do not adjust for baseline measurements. Further, between-child variance in length could be assessed by several comparable parametrizations of longitudinal data spread across metrics. The authors also found that study descriptions lacked precision: 'gain' or 'velocity' could be implied by a wide range of metrics that use very different statistical approaches or conceptual terms. This is particularly challenging for meta-analyses and systematic reviews since search terms in narrow strategies may not adequately reflect all published material on childhood growth.

2.5 Review results

2.5.1 Characteristics of included studies

The data extraction framework presented in Appendix 2.5 includes all relevant information drawn from the 77 articles, along with a critical appraisal of the risk of bias due to selection of participants, comparability, and outcome in each study. The greatest number of articles published in a year was in 2012 (14 articles, 18%), followed by 2018 (12, 16%), 2016 (11, 14%) and 2014 (10, 13%). Eight (10%) were published in 2013, seven (9%) each in 2015 and 2017, and four (5%) each in 2010 and 2011.

2.5.1.1 Location

The 77 articles were based on 62 unique studies. These are presented separately due to varying sub-samples, follow-up duration, and population groups included in analytical samples in separate articles based on the same study. The studies were conducted in 40 countries, including 12 high-income settings (Chile, Denmark, France, Germany, Hong Kong, Korea, Norway, Oman, Spain, the Netherlands, United Kingdom, and United States). Eight articles (10%) presented findings from multi-site studies. Bangladesh was the most common setting for single and multi-site studies (14 articles), followed by Brazil (13 studies). Nine articles (12%) reported findings from India, and five of these were from multi-site studies. The multi-site MAL-ED study and the Generation R study in the Netherlands had the greatest number of articles (four each) reported for an individual study.

41 articles (53%) reported on urban populations, of which six were in urban informal settlements and two in peri-urban populations. Sixteen articles (21%) were based on studies in rural settings, and 20 (26%) were conducted in both urban and rural settings.

2.5.1.2 Study design and timing of recruitment

Most studies (55 articles, 71%) adopted an observational approach, 17 (22%) were linked to a randomized or non-randomized intervention component, and five (six percent) had a hybrid design. The most common design was the prospective birth cohort study (47 articles, 61%), followed by follow-up studies of trial or intervention participants (nine articles, 12%) and secondary analysis of trial data (eight articles, 10%). Three (4%) prospective observational cohorts were nested (unrelated to evaluating the outcome of any treatment) within ongoing intervention studies, two (three percent) were follow-up studies of participants in previous cross-sectional observational studies, and four (six percent) were pooled analyses of multiple cohort studies (two of these also included data from follow-up studies of trial participants).

Forty articles (52%) were based on studies that began recruitment or data collection during pregnancy, 26 (34%) began at or within one month of the birth of the index infant, and 11 (14%) studies recruited infants in the post-neonatal period (between two and nine months). A higher proportion of observational studies (32 of 55 articles: 58%) recruited participants in pregnancy compared to those based on intervention participants (eight of 22 articles: 36%).

2.5.1.3 Follow-up and study size

The duration of follow-up across all studies varied between four months and 19 years. Twelve (16%) articles were based on studies that were reported as active at the time of publication (between 2010 and 2015). These were mostly population-based or large prospective birth cohort studies in the United Kingdom, the Netherlands, Denmark, and Brazil.

The youngest analytical sample with the shortest duration of follow-up was aged 0-4 months, the oldest 9-24 months, and the longest followed-up from birth to ten years. The median duration of follow-up was 24 months, (interquartile range (IQR) 12-36 months). Sixty-nine (90%) studies followed up infants through the first full year of life (0-12 months), with varying frequency of measurement in this period.

Measurement schedules varied across studies: 18 (23%) articles reported plans to measure infants every month, and 13 (17%) planned assessments every two or three months. Most studies had schedules that varied with infant age, with more frequent measurements in the first six or 12 months, and less frequent thereafter. The median number of expected length measurements per participant across 75 articles was six (IQR 4-12). Two articles did not provide enough information to make an assessment. Seven (9%) measured infant length less than three times during follow-up.

Due to unclear or incomplete reporting of response rates and measurements used in final models in many studies, I was unable to calculate total follow-up person-time across the 77 articles. Fifty-one (66%) articles did not mention the average or total number of length measurements per participant used in the main analysis. Of the rest, 14 (18%) reported the average (ranging between one and 12), seven (nine percent) reported the total number (between two and five), and five reported the range, or minimum number of measurements, or the proportion of participants who completed follow-up.

Ten studies reported multiple analytical samples of different sizes (to allow for varying response rates, age-groups or outcome of interest for each study objective). The rest included between 148 and 12,463 participants (median 872 participants, IQR 383-1972).

2.5.2 Quality assessment of included studies

Quality assessment scores for the articles by study design are presented in Table 2.2. Over 90% of articles scored stars for adequate selection of non-exposed groups, independent assessment of the outcome, and follow-up spanning the full first year of life. The weakest area of study quality was adequate follow-up of participants (outcome category): 49% did not make a statement about attrition or investigate bias due to missing data when non-response rates exceeded 10%. For the comparability category, 31% did not clearly justify their choice of confounders.

One main difference in study quality between observational and intervention studies was in the selection of participants: a higher proportion of observational studies (82% vs 59%) were able to demonstrate that study populations were somewhat or

truly representative of the average infant in the community. However, more intervention studies adjusted for infant sex in their analysis.

Table 2.2 Frequency distribution of articles that scored a star for each item of the Newcastle-Ottawa Scale, by study approach.

Item	Observational (n=55)	Interventional (n=17)	Hybrid (n=5)	Total (n=77)
Selection				
Representativeness of the exposed cohort	45 (82%)	10 (59%)	5 (100%)	60 (78%)
Selection of the non-exposed cohort	55 (100%)	17 (100%)	5 (100%)	77 (100%)
Ascertainment of exposure	47 (85%)	17 (100%)	4 (80%)	68 (88%)
Demonstrates that outcome (growth) was not present at baseline	44 (80%)	12 (71%)	5 (100%)	61 (79%)
Comparability				
Adjusts for infant sex	43 (78%)	16 (94%)	3 (60%)	62 (81%)
Additional confounders justified	37 (67%)	13 (76%)	3 (60%)	53 (69%)
Outcome				
Assessment of outcome	54 (98%)	17 (100%)	4 (80%)	75 (97%)
Follow-up spans first year of life	49 (89%)	15 (88%)	5 (100%)	69 (90%)
Adequate follow-up of participants	28 (51%)	9 (53%)	2 (40%)	39 (51%)

2.5.3 Growth metrics

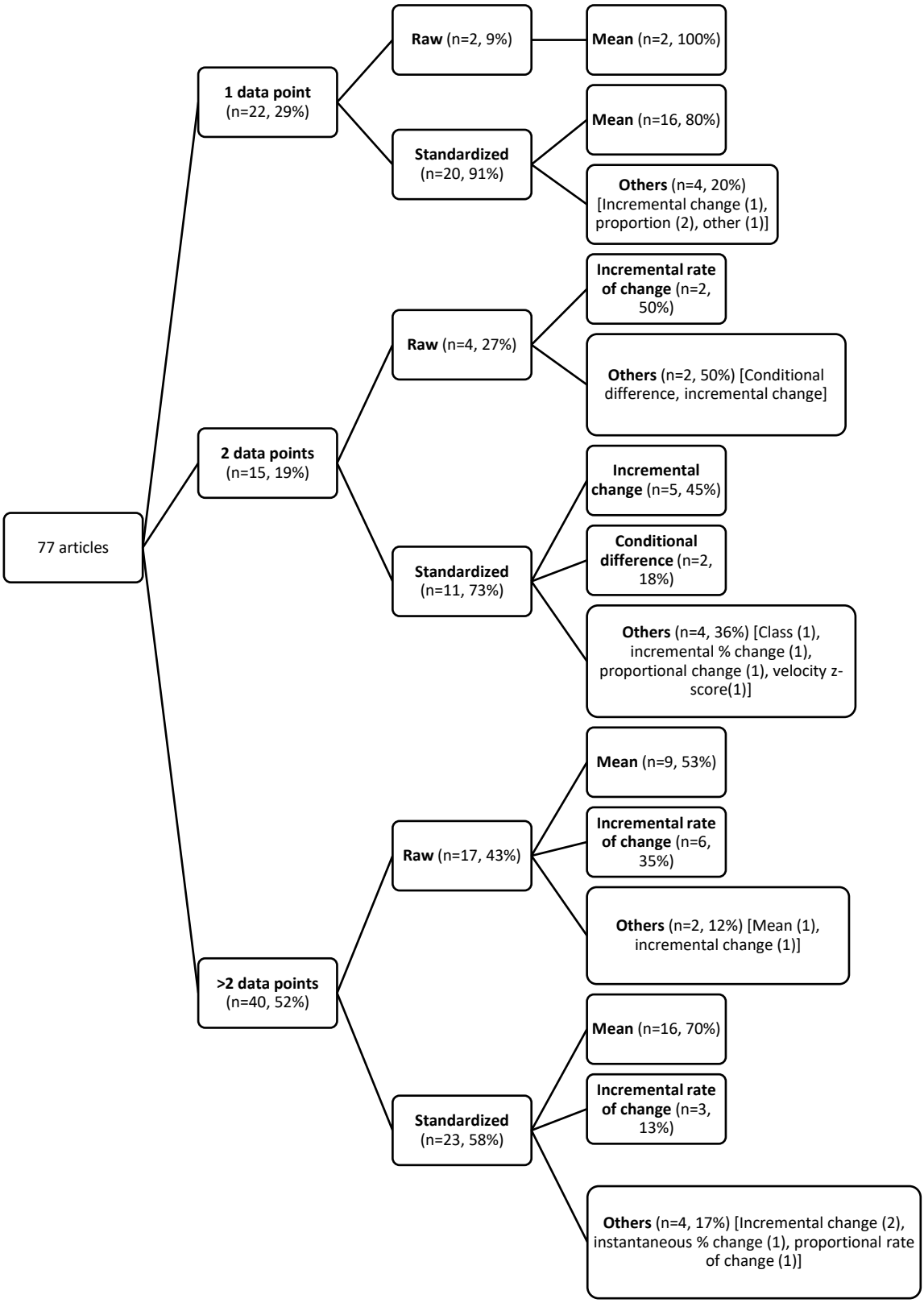
The frequencies with which approaches to quantifying growth metric components appeared in the 77 articles are summarised in Table 2.3. For articles that calculated more than one metric, the one most closely aligned with the study objective or hypothesis was summarised. When the level of estimation included group as well as individual levels, and when both categorical and continuous metrics were derived, a third category for ‘both’ was added to the component.

Table 2.3 Approaches to quantifying growth metric components (n=77 articles)

Component	Approach	N	%
Standardization	Raw	23	30
	Standardized	54	70
Level of estimation	Group	54	70
	Individual	19	25
	Both	4	5
Metric type	Continuous	68	88
	Categorical	3	4
	Both	6	8
Quantity of data	1 data point	22	29
	2 data points	15	19
	>2 data points	40	52
Metric sub-type	Mean	43	56
	Incremental rate of change	12	16
	Incremental change	10	13
	Conditional difference	3	4
	Instantaneous rate of change	2	3
	Proportion	2	3
	Class	1	1
	Proportional change	1	1
	Proportional rate of change	1	1
	Velocity z-score	1	1
	Other metrics	1	1
Analytical approach to derive metric	Manual	32	42
	Linear mixed effect model	26	34
	Generalized estimating equations	5	6
	Conditional regression	3	4
	Non-linear mixed effect model	3	4
	Other method	3	4
	Pre-designed structural model	3	4
	Linear fixed effect model	1	1
	Threshold or cut-off	1	1

A decision tree (Figure 2.2), based on the type produced by Leung et al (2018), shows the most common approaches to handling growth data with one, two, or more data points per participant. For example, mean length (usually LAZ) was used in 16 studies that standardized a single growth measurement at a given time point. Incremental change was used in five studies to describe the difference between standardized length measurements at two time points.

Figure 2.2 Decision tree for selection of metric sub-types (n=77)



I identified thirty-five unique 'content signatures' based on combinations of number of data points per child, standardization, metric sub-type, and analytical approach to derive metric (Appendix 2.6) Of these, five accounted for the primary growth metric used in over 50% of the articles reviewed (Table 2.4)

Table 2.4 Five most common content signatures

Rank	Description	Number (%) of articles using it
1	1 data point standardized to derive a mean value estimated manually	16 (21%)
2	>2 data points standardized to derive a mean value from a linear mixed effect model	9 (12%)
3	>2 data points used in raw form to derive a mean value from a linear mixed effect model	6 (8%)
4	>2 data points used in raw form to derive incremental rate of change from a linear mixed effect model	5 (6%)
5	2 data points standardized to calculate incremental change manually (or a simple calculation)	4 (5%)

The average number of expected length measurements collected per participant in the 75 articles that reported it was 7.4 for 21 articles that based growth metrics on one data point, 6.2 for 15 that used two data points, and 10.8 for 39 that used more than two data points to derive a growth metric.

2.5.4 Analytical strategies to investigate relationships between covariates and growth outcomes

2.5.4.1 Causal or multivariable approaches

The approaches to investigating the relationships between exposures or potential risk factors and infant growth varied. Based on the stated study aims and statistical analysis methods, 10 articles adopted a predictive modelling approach to identify significant factors from a range of possible (and some of particular interest) exposures that could predict infant growth outcomes; 67 aimed to quantify the effect of one or more exposures of interest on an infant growth outcome while accounting for known confounders.

The most common primary statistical approaches were random effect models (34 articles, 44%), linear regression (27 articles, 35%), generalized estimating equations (seven articles, 9%), mediation analysis (four articles, 5%), and logistic regression (three articles, 4%). Fixed effect models and repeated measures ANOVA were

uncommon (one article each). The 12 articles that employed a second analytical method to answer a main study hypothesis also used some of these strategies, but additionally included Cox regression, instrumental variable analysis, and seemingly unrelated regression analysis. In 22 articles based on an intervention approach, linear regression (nine articles), random effect models (eight articles) and mediation analysis (three articles) were most commonly used.

2.5.4.2 Model selection

Fifty-seven (74%) studies specified exposures or covariates that were of *a priori* interest, and 21 (26%) reported using variable reduction strategies such as step-wise regression or p-values from unadjusted analyses to select variables for inclusion in a final model. A quarter of those that reported covariates *a priori* subsequently also used variable reduction strategies based on p-values. Ten studies (13%) reported additional statistical considerations for model selection or evaluation, such as adjustment for multiple testing (two articles), change-in-estimate methods to control for confounding (three articles), assessing model fit using information criteria (four articles), and checking for collinearity of covariates before model fitting (one article).

Fourteen of 67 studies (21%) that included multivariable analyses did not specify covariates of *a priori* interest, and eight of ten studies that modelled predictors of infant growth described variable elimination strategies based on p-values.

2.5.5 Exposures, confounders, and mediators

A general list of study exposures, confounders, and mediators identified was collapsed into 18 categories (Table 2.5) pertaining to infants, parents, and households or environments.

Table 2.5 Description of exposures, confounders and mediators

Category	Description
Infant	
Diarrhoea	Episodes, duration, frequency, intensity, pathogen. Subsumes all gastrointestinal infections that manifest as diarrhoea
Other child illness	Fever, malaria, cough, or other infection, including treatment for illness with antibiotics or other drugs
Chemical substances and drugs	Insecticide, pesticide, antibiotics
IYCF-Breastfeeding	Breastfeeding, formula use
IYCF-Complementary	Non-breastmilk solid, semi-solid foods introduced after BF or

Category	Description
feeding	formula for a few months
IYCF-full continuum	Breastfeeding and complementary feeding in the same analysis
Infant characteristic	Sex, ethnicity, season of birth, age, preterm, gestational age, birth order
Infant anthropometry	LBW, birth weight, birth length, postnatal anthropometry
Parents	
Parental anthropometry	Height, weight, indices (such as BMI), change in anthropometry
Parental diet	e.g. food intake, protein intake, deficiency status
Parental behaviour	e.g. smoking, alcohol intake, health care-seeking, physical activity, use of dietary supplements
Parental health	Disease biomarker, mental health status, prevalent risk factor (e.g. blood pressure)
Parental characteristics	Socio-demographic factors: age, religion, place of birth, ethnicity, duration of residence
Household, environment or group	
Socioeconomic position	Includes asset-based, consumption, education, occupation, wealth, subjective measure of individuals or households
Household	Overcrowding, structure, size, composition. Household food insecurity. Also for similar family characteristics.
Water, Sanitation and Hygiene (WASH)	Water and sanitation (access, use)
Environmental exposure	No individual exposure, e.g. area level pollution, neighbourhood hygiene, presence of facility
Clustering	Intervention arm, area of residence, cohort, source of measurement, other group membership
Abbreviations: IYCF: Infant and young child feeding; BMI, Body mass index; LBW, Low birth weight	

2.5.5.1 Exposure variables

Fifty-eight (75%) articles reported exposure variables pertaining to one category, and 19 (25%) were based on more than one (up to four). Forty-nine (64%) included repeated measurements of exposure, 20 (26%) collected a biological sample (blood, urine, stool, cord blood, or breastmilk) to ascertain exposure status, and 38 (49%) first measured exposure at or close to the infant's birth.

The most common primary exposure was infant and young child feeding (IYCF), used in 17 articles (nine on breastfeeding, seven on complementary feeding, and one on the full continuum). Others included diarrhoeal disease (13 articles), socioeconomic factors (11 articles), parental anthropometry (nine articles), chemical substances and drugs (six articles), parental health (five articles), infant characteristics (four articles) and parental behaviour (four articles). Categories used as the primary exposure in fewer than four articles included infant anthropometry, parental diet, environmental or household exposures, and WASH. Parental

characteristics, other child illness and factors related to clustering were not examined as primary exposures in any study. However, these were more commonly used as confounders.

2.5.5.2 Confounders

The list of confounders in an article included a maximum of 10 categories, but 90% adjusted for confounders from 6 groups or fewer. Fifty-three (69%) articles adjusted for an SEP variable, 52 (68%) adjusted for infant characteristics, 38 (49%) for parental anthropometry, 32 (42%) for other parental characteristics, and 30 (39%) for infant anthropometry. Other frequently used confounders were breastfeeding (21, 27%), parental behaviours (19, 25%), clustering (16, 21%), other child illness (13, 17%), diarrhoea (11, 14%), and household factors (eight, 10%).

2.5.5.3 Mediators

The four studies that conducted mediation analyses included the following mediated relationships: (1) birth length and placental mitochondrial DNA mediate the relationship between NO2 exposure and infant length, (2) Maternal BMI, infant length at 2-3 months, infant birth length, and infant birth weight as mediators of the relationship between SEP factors and infant length; (3) birth weight as a mediator of maternal antenatal health and infant length; (4) maternal life stress and depression as a mediator of the effect of SEP on infant length.

2.5.6 Determinants of infant linear growth

2.5.6.1 Infant and young child feeding

Evidence on the relationship between IYCF quantity, quality, or timeliness and infant growth was mixed in the 22 studies that reported on it, with 13 reporting neutral findings. IYCF indicator and categorization used varied considerably (Table 2.6).

Table 2.6 Relationship between IYCF and linear growth

ID	Reference	Country	Exposure specification	Age group at exposure assessment	Effect on growth
4	(Sanin et al., 2018)	Bangladesh	Micronutrient adequacy ratio	9-12, 15-18, and 21-24 months	Neutral
7	(Kramer et al., 2018)	Belarus	Breastfeeding	2-3 months, 12 months	Neutral
8	(Moradi et al., 2018)	Bangladesh	Dietary diversity	9-12, 15-18, 21-24 months	Neutral
11	(Cheng et	Hong Kong	Exclusive	0-3 months	Neutral

ID	Reference	Country	Exposure specification	Age group at exposure assessment	Effect on growth
	al., 2018)		breastfeeding >3 months		
13	(Zhang et al., 2017)	Bangladesh	Exclusive breastfeeding >6 months (boys)	0-24 months	Negative
15	(MAL-ED Network Investigators, 2017)	Bangladesh, Brazil, India, Nepal, Peru, South Africa, Tanzania	Low energy intake and protein density	9-24 months	Negative
17	(Bork and Diallo, 2017)	Senegal	Minimum Meal Frequency	6-7, 9-10 months	Neutral
18	(Bell et al., 2017)	United States	Predominant Breastfeeding or Exclusive Formula Feeding	0-6 months	Neutral
21	(Owais et al., 2016)	Bangladesh	Minimum Acceptable Diet	9 months	Positive
21	(Owais et al., 2016)	Bangladesh	Exclusive breastfeeding	0-3 months	Neutral
23	(Kavle et al., 2016)	Egypt	Minimum Dietary Diversity	4-12 months	Neutral
27	(Busert et al., 2016)	Nepal	Dietary diversity	9-69 months, 29-89 months	Positive
29	(Bhargava, 2016)	Philippines	Calcium intake as a ratio of energy intake	4-24 months	Positive
31	(Wright et al., 2015)	Philippines	Continued breastfeeding up to 2 years (with or without minimum dietary diversity)	6-24 months	Positive
32	(Vail et al., 2015)	United Kingdom	Early age at weaning	3-7 months	Neutral
41	(Mallard et al., 2014)	Zambia	Iron rich food and dietary diversity	6 months	Positive
41	(Mallard et al., 2014)	Zambia	Iron rich food and dietary diversity	12 months	Neutral
44	(Betoko et al., 2014)	France	Type of formula	0-4 months	Neutral
45	(Woo et al., 2013)	United States, Mexico, China	Timing of introduction to CF	0-12 months	Neutral
57	(Queiroz et al., 2012)	Brazil	Duration of exclusive breastfeeding	0-6 months	Positive
65	(Bork et al., 2012)	Senegal	Meal Frequency Index and Complementary Feeding Index	6-36 months	Positive
69	(De Hoog et al., 2011)	The Netherlands	Exclusive breastfeeding	0-4 months	Negative
75	(Kattula et al., 2014)	India	Exclusive breastfeeding	0-6 months	Negative
77	(Johnson et al., 2012)	India	Exclusive breastfeeding	0-3 months	Neutral

The pattern of confounders selected for inclusion varied and no two studies adjusted for the same group of variables. All but three (Bork and Diallo, 2017, Wright et al., 2015, Mallard et al., 2014) adjusted for an SEP variable and 12 adjusted for infant sex or another characteristic. Diarrhoea was included as a confounder in six studies (Sanin et al., 2018, Islam et al., 2018, Zhang et al., 2017, Bhargava, 2016, Mallard et al., 2014, Betoko et al., 2014) and parental anthropometry in 12. Most other confounders were context specific, such as WASH in LMIC settings, a clustering variable for studies embedded within trials or across sites, as well as related IYCF variables for studies that addressed a later stage of the continuum but wanted to adjust for the effect of a previous one.

Four articles reported on studies conducted in urban informal settlements (Sanin et al., 2018, Islam et al., 2018, Kattula et al., 2014, Zhang et al., 2017), and none showed positive relationships between IYCF and linear growth. Two found negative effects of exclusive breastfeeding up to six months on linear growth: exclusively breastfed boys in an urban informal settlement in Dhaka, Bangladesh were more likely to have poor overall growth over 0-24 months defined by membership of a functional principal components stratum (Islam et al., 2018); breastfed infants in Vellore, India had lower length velocity (-0.06 cm/month; 95% CI -0.10, -0.01) than those breastfed for a shorter duration (Kattula et al., 2014). In the MAL-ED cohort Bangladesh site (Sanin et al., 2018), micronutrient adequacy of complementary feeding did not protect against stunting between 12 and 24 months (aOR 0.99; 95%CI 0.98, 1.01). Instead, low birth weight infants and boys had the highest odds of stunting (aOR 3.03; 95%CI 1.69, 5.44 for LBW infants and aOR 1.98; 95%CI 1.17, 3.33 for boys). In the same cohort (Islam et al., 2018), dietary diversity score also did not protect against stunting (aOR 0.93; 95%CI 0.74, 1.16), and birth length was a stronger predictor of stunting in the second year of life (aOR 0.40; 95%CI 0.26, 0.61).

Four of six articles that reported positive relationships between IYCF and linear growth were in rural settings, but their findings were not comparable. One article (Owais et al., 2016) reported a positive effect of acceptable complementary feeding at nine months, but not of exclusive breastfeeding to three months. Two other studies (Busert et al., 2016, Garced et al., 2012) reported beneficial effects of complementary feeding, but they included children older than two years in their sample (up to 89 and 36 months), making it difficult to assess the benefit in the early childhood. A study in rural Brazil found a small effect on mean LAZ in the first year

(β 0.0031, p<0.05), compared to a larger negative effect of maternal short stature (β -0.44, p<0.001) in the same analysis (Queiroz et al., 2012). A follow-up study (Mallard et al., 2014) of the CIGNIS trial in Lusaka, Zambia found a positive effect of dietary diversity and consumption of iron rich foods at six months of age on LAZ at 18 months, which mediated 13.4% of the effect of maternal education on infant length, but the benefit was not maintained for the same diet at 12 months. A different exposure specification on a sample of 2822 infants in the Cebu cohort study in the Philippines (Wright et al., 2015) found that continued breastfeeding up to 24 months conferred a benefit on LAZ at 6-24 months (β 0.16 for breastfeeding with high dietary diversity, and β 0.14 for breastfeeding with low dietary diversity), as well as predicting LAZ score at 24 months (β 0.25; 95%CI 0.19, 0.30 for boys and β 0.2; 95%CI 0.12, 0.28 for girls).

The methodologic issues around assessing the relationship between IYCF and growth were demonstrated in a recent re-analysis (Kramer et al., 2018) of data from the PROBIT trial of breastfeeding promotion in Belarus using three different approaches: intention to treat analysis comparing randomized and control groups, using observed duration of breastfeeding, and by the predicted probability of breastfeeding using randomization as an instrumental variable. The authors found that the two experimental approaches showed a different direction of effect to the observational one: infants in the intervention group and those breastfed for over 12 months grew faster than those in the control group at 2-3 months of age, with declining difference and near equivalence by one year. In the observational analysis, infants in the intervention group had lower LAZ at six, nine, and 12 months.

2.5.6.2 Diarrhoeal diseases

The relationship between diarrhoeal illness and linear growth was consistently negative across studies, with neutral findings in two. One found a negative effect of another infectious agent in the same study (Garzón et al., 2018), and another was a study in Egypt (Kavle et al., 2016) that used a crude measure to assess diarrhoea (monthly recall of diarrhoea that lasted more than 7 days) and did not follow-up infants beyond 12 months . Eleven of 14 articles assessed exposure using a biomarker, and six tested the association between specific pathogens and linear growth. A study in urban informal settlements in Peru found that parasitic infection in infancy had a stronger negative association with linear growth than one that only appeared in the second year (Jaganath et al., 2014); in Sao Tome and Principe sub-clinical infection led to mild growth faltering in LAZ between 0-24 months (Garzón et

al., 2018); in two overlapping pooled analyses across LMICs recurrent diarrheal episodes and high cumulative burden over 0-24 months led to growth faltering (Richard et al., 2014, Richard et al., 2013). All studies on diarrhoea and growth were conducted in LMICs (Table 2.7).

Table 2.7 Relationship between diarrhoeal exposures and linear growth

ID	Reference	Country	Exposure specification	Age group at exposure assessment	Effect on growth
1	(Syed et al., 2018)	Pakistan	Anti-LPS IgA (marker of environmental enteric dysfunction)	6-9 months	Negative
2	(Steiner et al., 2018)	Bangladesh	Cryptosporidium infection	0-24 months	Negative
3	(Schnee et al., 2018)	Bangladesh	Cryptosporidium and campylobacter attributable diarrhoea in first year	0-12 months	Negative
6	(Lima et al., 2018)	Bangladesh, Brazil, India, Pakistan, South Africa, Tanzania	Subclinical entero-aggregative E.coli infection	0-6 months	Neutral
9	(Garzón et al., 2018)	Sao Tome and Principe	Subclinical parasitic infection (Giardia lamblia and helminth)	3-24 months	Negative
9	(Garzón et al., 2018)	Sao Tome and Principe	Cryptosporidium infection	3-24 months	Neutral
22	(Nagata et al., 2016)	Guatemala	Diarrhoea in past week	12 months	Negative
23	(Kavle et al., 2016)	Egypt	Diarrhoeal episode (7+ days)	2-12 months	Neutral
37	(Richard et al., 2014)	Peru, Brazil, Guinea-Bissau, and Bangladesh	Diarrhoeal episodes (lagged and cumulative)	0-24 months	Negative
42	(Jaganath et al., 2014)	Peru	H.pylori infection in late infancy and more than 3 episodes per year	6-23 months	Negative
46	(Richard et al., 2013)	Peru, Brazil, Guinea-Bissau, and Bangladesh	Average diarrhoea burden (days)	0-24 months	Negative
47	(Peterson et al., 2013)	Bangladesh, Peru	REG1B concentrations in stool	3 months	Negative
48	(Lee et al., 2013)	Peru	Campylobacter infection and severity	0-72 months	Negative
70	(Moore et al., 2010)	Brazil	Episode of prolonged or acute diarrhoea	6-12 months	Negative
74	(LaBeaud et al., 2015)	Kenya	Parasitic infection (species specific) and polyparasitism	0-36 months	Negative

2.5.6.3 Socioeconomic position

Fourteen studies explored the link between socioeconomic position (SEP) and linear growth, focusing on maternal or parental education, measures of income, standard of living, or a composite marker based on access to water and sanitation, assets, income, and maternal education (WAMI) (Table 2.8). Studies were based in LMICs as well as HICs.

Table 2.8 Relationship between socioeconomic position and linear growth

ID	Reference	Country	Indicator	Effect on growth
10	(Devakumar et al., 2018)	Nepal	Maternal education and asset score	Positive
15	(MAL-ED Network Investigators, 2017)	Bangladesh, Brazil, India, Nepal, Peru, South Africa, Tanzania	WAMI Index (water and sanitation, assets, maternal education, household income)	Positive
20	(Svefors et al., 2016)	Bangladesh	Maternal illiteracy	Negative
24	(Griffiths et al., 2016)	India	Standard of Living Index	Positive
25	(Gough et al., 2016)	Zimbabwe	Maternal education	Positive
38	(Patel et al., 2014)	Belarus	Maternal education	Positive
40	(Murasko, 2014)	United States	Household permanent income	Positive
49	(Kwok et al., 2013)	Hong Kong	Parental education	Positive
54	(Silva et al., 2012)	The Netherlands	Low maternal education	Positive
58	(Matijasevich et al., 2012)	Brazil	Maternal education	Positive
61	(Kang Sim et al., 2012)	Chile	SEP (Graffar Index)	Positive
76	(Howe et al., 2012b)	United Kingdom	Maternal education	Positive
77	(Johnson et al., 2012)	India	Middle or low SEP (Standard of Living Index) vs High	Negative

Household standard of living was associated with length in a study in rural India (Griffiths et al., 2016, Johnson et al., 2012). In a pooled analysis of data from seven of eight MAL-ED study sites (Bangladesh, Brazil, India, Nepal, Peru, South Africa, Tanzania) where SEP was measured in the same way, a 10% increase in the WAMI index was associated with a 0.018 LAZ (SE 0.003) increase per month from birth to 24 months (MAL-ED Network Investigators, 2017).

Most studies found that greater maternal educational attainment had a positive effect on linear growth, except one, the Generation R cohort in Rotterdam, the

Netherlands (Silva et al., 2012). The protective effect of low maternal education (less than ten years schooling or below O-level grade) on linear growth was observed due to the higher length velocity (cm/month) between 1-18 months of infants born to women who were less educated, leading to greater length at 14 months (0.4cm; 95%CI 0.08, 0.72) than children of more educated mothers despite shorter length at two months (-0.8cm; 95%CI -1.16, -0.58).

2.5.6.4 Parental anthropometry

Fifteen studies spread across LMICs and HICs reported on parental anthropometry as a determinant of infant linear growth (Table 2.9). The link between maternal height and linear growth was positive across several studies, and maternal short stature was associated with reduced linear growth; one of these was conducted in an urban informal settlement in Vellore, India (Kattula et al., 2014).

The effect of maternal weight or BMI was ascertained at different time points or assessed as gestational weight gain, but all found that higher weight was associated with greater linear growth, in rural Vietnam (Hanieh et al., 2015) and urban United States (Deierlein et al., 2011), or that low maternal BMI was negatively associated with linear growth in rural Benin (Padonou et al., 2014).

The influence of paternal anthropometry was apparent in two studies that examined the relationship between mid-parental height and growth. A large population-based cohort in Hong Kong (Kwok et al., 2013) found a positive relationship with infant length gain z-score within 3-9 months (β 0.04; 95%CI 0.04-0.05), though it did not persist into later childhood. A secondary analysis of the multi-country WHO MGRS data (Garza et al., 2013) found that mid-parental height explained a greater proportion of variability (mean 16%; 11% in Ghana to 21% in India) in attained child length at 24 months than maternal or paternal height alone. In an analysis of 4116 infants in the Generation R study (Durmus et al., 2013), the effect of maternal pre-pregnancy and paternal height, weight, and BMI were associated with higher LAZ at birth, 3, 6, 12, 24, 36, and 48 months, and increased with age (β 0.24 at birth to β 0.36 at 48 months for maternal anthropometry and β 0.21 to β 0.33 for paternal anthropometry, all $p < 0.05$). Combined maternal and paternal heights explained 16% of the variance in child length measurements at 24 months.

No study examined the effect of parental obesity (based on a cut-off) on linear growth to examine whether the apparently protective effect of higher BMI has an upper threshold.

Table 2.9 Relationship between parental anthropometry and linear growth

ID	Reference	Country	Indicator	Effect on growth
20	(Svefors et al., 2016)	Bangladesh	Maternal short stature	Negative
21	(Owais et al., 2016)	Bangladesh	Maternal height	Positive
24	(Griffiths et al., 2016)	India	Maternal height	Positive
25	(Gough et al., 2016)	Zimbabwe	Maternal height	Positive
35	(Hanieh et al., 2015)	Vietnam	Maternal BMI in pregnancy and gestational weight gain	Positive
39	(Padonou et al., 2014)	Benin	Maternal short stature	Negative
39	(Padonou et al., 2014)	Benin	Maternal low BMI	Negative
49	(Kwok et al., 2013)	Hong Kong	Mid-parental height	Positive
50	(Garza et al., 2013)	United States, Oman, Norway, Brazil, Ghana, India	Mid-parental height	Positive
52	(Durmus et al., 2013)	The Netherlands	Combined parental BMI, and height and weight	Positive
53	(Addo et al., 2013)	Brazil, Guatemala, India, the Philippines, South Africa	Maternal height	Positive
57	(Queiroz et al., 2012)	Brazil	Maternal short stature	Negative
60	(Lourenço et al., 2012)	Brazil	Maternal height	Positive
63	(Hambidge et al., 2012)	Guatemala	Maternal height	Positive
68	(Deierlein et al., 2011)	United States	Gestational weight gain	Positive
75	(Kattula et al., 2014)	India	Maternal height	Positive

2.5.6.5 Parental behavioural, dietary, and health-related factors

Evidence for parental health and behavioural factors included the influence of smoking, alcohol intake, maternal diet in pregnancy and lactation, mental health, hypertension, malaria in pregnancy, and oxidative stress (Table 2.10).

In stratified analyses of the 1993 and 2004 Pelotas cohorts in Brazil (Matijasevich et al., 2011), maternal smoking during pregnancy was associated with lower LAZ score at birth (β -0.34; 95%CI -0.40, -0.27 and β -0.24; 95%CI -0.33, -0.16), three months (β -0.35; 95%CI -0.56, -0.15 and β -0.24; 95%CI -0.32, -0.15), 12 months (β -0.20; 95%CI -0.35, -0.05 and β -0.20; 95%CI -0.28, -0.11), and 24 months (β -0.20; 95%CI -0.28, -0.12), adjusted for SEP, parental anthropometry and paternal smoking. A subsequent analysis of the 1993 Pelotas cohort (Martínez-Mesa et al., 2012) showed a dose-response relationship between number of cigarettes smoked per day and infant LAZ at 12 months (β -0.39; 95%CI -0.56, -0.22 for less than 10 cigarettes per day, β -0.70; 95%CI -0.98, -0.42 for 10-19 per day, and β -0.67; 95%CI -0.97, -0.37 for more than 20 per day, Wald test for trend p<0.001). This was attenuated after additional control for LAZ at birth (p= 0.042). The relationship persisted at 11 and 15 years even after adolescent smoking was taken into account.

In the Generation R study (Durmuş et al., 2011), maternal smoking in the first trimester did not affect length SDS at any age, but continued smoking had negative effects across all ages studied: β -0.4; 95%CI -0.49, -0.31 at birth, β -0.30; 95%CI -0.38, -0.23 at three months, β -0.14; 95%CI -0.21, -0.06 at six months, β -0.14; 95%CI -0.21, -0.06 at 12 months, β -0.13 95%CI -0.21, -0.05 at 24 months, β -0.11; 95%CI -0.20, -0.03 at 36 months, and β -0.10; 95%CI -0.19, -0.01 at 48 months.

A study in rural Uganda (De Beaudrap et al., 2016) showed that infants born to women who had malaria in pregnancy had lower length gain between 0-12 months (-2.71cm; 95%CI -4.17, -1.25), highlighting its importance as a risk factor for growth faltering in settings where malaria is endemic.

Most other factors had neutral effects, or were assessed in special groups, such as infants from high-income families in the United States (Switkowski et al., 2016, Ertel et al., 2010) who were not likely to be representative of the average American infant.

Table 2.10 Parental behavioural, dietary and health-related factors related to linear growth

ID	Reference	Country	Indicator	Effect on growth
Behaviour				
66	(Matijasevich et al., 2011)	Brazil	Maternal smoking in pregnancy and partner smoking	Negative
59	(Martínez-Mesa et al., 2012)	Brazil	Maternal smoking in pregnancy and partner smoking	Negative
67	(Durmus et al., 2013)	The Netherlands	Maternal smoking in pregnancy	Negative
34	(O'Keeffe et al., 2015)	United Kingdom	Maternal light drinking during pregnancy	Neutral
Diet				
5	(Moradi et al., 2018)	Iran	Maternal dietary density during lactation	Neutral
19	(Switkowski et al., 2016)	United States	Maternal protein intake in pregnancy	Negative
Health				
72	(de Beer et al., 2010)	The Netherlands	Pre-pregnancy or pregnancy-induced maternal hypertension	Neutral
71	(Ertel et al., 2010)	United States	Postpartum depression	Positive
71	(Ertel et al., 2010)	United States	Antenatal depression	Neutral
26	(De Beaudrap et al., 2016)	Uganda	Maternal malaria in pregnancy	Negative
43	(Hong et al., 2014)	Korea	Maternal antioxidant and oxidative stress levels	Neutral
62	(Husain et al., 2012)	United Kingdom	Maternal depression	Neutral

2.5.6.6 Exposure to chemical substances and antibiotics

Evidence for exposure to chemical substances was limited as studies examined very different and sometimes context-specific exposures (Table 2.11).

One study in an urban informal settlement in Vellore, India that followed-up infants monthly found no short-term (0-6 months) effects of exposure to antibiotics in a month on growth in the following month, or long-term (6-36 months) effects of exposure in the first six months. Exposure to antibiotics was high (57% had received antibiotics by six months, and 28% more than one dose). Linear growth was assessed as absolute change in z-score from longitudinal linear regression models with robust variance, controlling for infant sex, previous z-score, SEP, household factors, infant illness and breastfeeding. There were no effects on growth in the short-term (LAZ -0.03; 95%CI -0.10, 0.04), or long-term (LAZ -0.05; 95%CI -0.17, 0.06). In adjusted longitudinal Poisson regression models, girls had higher short-

term risk of stunting at six months (aRR 1.27; 95%CI 1.04, 1.56), but the effect did not persist beyond early infancy (Rogawski et al., 2015).

Table 2.11 Effect of exposure to chemical substances and antibiotics on infant growth

ID	Reference	Country	Indicator	Effect on growth
55	(Saha et al., 2012)	Bangladesh	Postnatal arsenic exposure (among girls)	Negative
33	(Rogawski et al., 2015)	India	Antibiotic use	Neutral
64	(Garced et al., 2012)	Mexico	Prenatal DDE exposure	Neutral
30	(Alkhalawi et al., 2016)	Germany	Perfluorohexanesulfonic acid (PFHxS)	Positive
36	(Costet et al., 2015)	Guadeloupe*	Pre- and postnatal chlorodecone (insecticide) exposure	Negative
73	(Andersen et al., 2010)	Denmark	perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA)	Neutral

*French overseas territory

2.5.6.7 Infant characteristics and anthropometry

Three articles (Admassu et al., 2018, Padonou et al., 2014, Richard et al., 2012) examined the effects of an infant's anthropometric characteristics on subsequent growth, two addressed ethnicity, and five examined sex-differences in growth (Table 2.12).

In Benin (Padonou et al., 2014), intrauterine growth restriction (IUGR) and low birth weight (LBW) led to lower length gain (LAZ) between zero and 18 months in multivariable mixed models (β -0.49; SE 0.09, p<0.001, and β -0.43; SE 0.14, p= 0.002). In the same model, length at birth was associated with greater length gain (β 0.16; SE 0.02, p<0.001). A pooled analysis of eight cohort studies (Richard et al., 2012), which included the cohort in an urban informal settlement in Vellore, India, showed that fluctuating WLZ (≥0.5 SD) up to 17 months led to lower LAZ at 18-24 months of age (β -0.51; 95%CI -0.67, -0.36). Positive change in WLZ between 6-12 and 18-24 months was associated with greater length at 18 months (0.33 cm; 95%CI 0.11, 0.54) and 24 months (0.72 cm; 95%CI 0.52, 0.92).

A body composition study of the iABC cohort in Ethiopia (Admassu et al., 2018) examined the effect of changes in body composition from 0-6 months on later linear growth using a linear mixed-effects model, and showed a positive relationship between fat-free mass accretion and LAZ at one year (β 0.64; 95%CI 0.19, 1.09) and linear growth up to five years (β 0.63; 95%CI 0.19, 1.07). In addition, infants of

mothers with higher BMI measured 2.5 months after birth had faster linear growth from one to five years.

Many studies adjusted for infant sex in multivariable analyses to take into account sexual dimorphism in growth, as well as sex-specific social or cultural factors. In the Vellore cohort in India (Kattula et al., 2014), girls had lower length velocity between 0-24 months (0.05 cm/month; 95%CI -0.10, -0.01). In the Infant Feeding Study in rural India (Johnson et al., 2012), girls were shorter between three and 15 months (-1.5 cm; SE 0.2, p<0.001) in mixed-effects models adjusted for a range of SEP, IYCF and infant morbidity factors. Two studies in Africa found contrasting effects: boys in rural Senegal (Bork and Diallo, 2017) had lower height for age difference (HAD) per month (-0.025 cm, p<0.001), but boys in Zimbabwe (Gough et al., 2016) were less likely to have poor LAZ growth trajectories between 0-24 months.

Table 2.12 Effect of infant characteristics on linear growth

ID	Reference	Country	Indicator	Effect on growth
Anthropometry				
12	(Admassu et al., 2018)	Ethiopia	Fat-free mass accretion (0-6 months)	Positive
39	(Padonou et al., 2014)	Benin	Length at birth	Positive
39	(Padonou et al., 2014)	Benin	IUGR, LBW	Negative
56	(Richard et al., 2012)	Peru, Brazil, Guinea-Bissau, India, and Bangladesh	Wasting or highly variable WLZ at 6-11 or 12-17 months	Negative
Ethnicity				
14	(Matos et al., 2017)	Ecuador	Ethnicity	Neutral
51	(Fairley et al., 2013)	United Kingdom	Ethnicity (Pakistani vs White British)	Positive
Sex				
17	(Bork and Diallo, 2017)	Senegal	Male sex	Negative
25	(Gough et al., 2016)	Zimbabwe	Male sex	Positive
28	(Broere-Brown et al., 2016)	The Netherlands	Male sex	Neutral
75	(Kattula et al., 2014)	India	Female sex	Negative
77	(Johnson et al., 2012)	India	Female sex	Negative

2.5.6.8 Other factors

Table 2.13 summarises findings of studies on the effects of other environmental factors such as WASH, environmental exposures, and household characteristics.

Seven studies adjusted for the effects of WASH on infant growth in multivariable analysis. In a predictive modelling study in urban informal settlements in Bangladesh (Zhang et al., 2017), girls from households with access to municipal water supply had lower risk of poor growth (β -0.411, p-value adjusted for false discovery rate = 0.0185). In the same analysis, boys from households with more than five family members were more likely to experience growth faltering (β 0.273, p= 0.0226). A study in the Philippines (Bhargava, 2016) also reported negative effects of large household size, and studies in Ecuador (Matos et al., 2017) and Chile (Kang Sim et al., 2012) found that the number of young children in the household also had negative consequences. Only one study in Guatemala (Nagata et al., 2016) suggested that large households with many children posed greater risk for infant growth.

Table 2.13 Other factors related to linear growth

ID	Reference	Country	Indicator	Effect on growth
WASH				
13	(Zhang et al., 2017)	Bangladesh	Access to municipal water supply	Positive
Household				
13	(Zhang et al., 2017)	Bangladesh	Number of people in the household	Negative
29	(Bhargava, 2016)	Philippines	Number of people in the household	Negative
14	(Matos et al., 2017)	Ecuador	Number of children in the household	Negative
61	(Kang Sim et al., 2012)	Chile	Number of children in the household	Negative
22	(Nagata et al., 2016)	Guatemala	Number of people in the household	Negative
22	(Nagata et al., 2016)	Guatemala	Number of children in the household	Negative
Environmental				
16	(Clemente et al., 2017)	Spain	Prenatal NO2 exposure	Negative
Season				
20	(Svefors et al., 2016)	Bangladesh	Season of conception (monsoon)	Negative

2.6 Discussion

2.6.1 Determinants of growth

2.6.1.1 Recent reviews – focus on prenatal growth, and postnatal feeding

Three of the four recent summarised reviews made the case for stunting as a condition with a distinct causal web that surrounds linear growth faltering in early life. These factors perpetuate its long-term, intergenerational consequences in the absence of positive changes within the first 1000 days to break the cycle.

While these reviews look at a wide range of factors that lead to poor growth (or stunting) in infancy and early childhood, they do not sufficiently explore how these factors are interrelated, or the direction of observed relationships. The cross-sectional nature of most studies that form the current evidence base is a major barrier to exploring issues of temporality. Even when cohort studies were included (Danaei et al., 2016), they were not used to develop temporal chains of causation, but merely as more 'robust' sources of relationships between risk factors and stunting.

Prenatal growth featured prominently in three of four reviews (Danaei et al., 2016, Prendergast and Humphrey, 2014, Stewart et al., 2013). The frameworks on the stunting syndrome (Prendergast and Humphrey, 2014) and the importance of complementary feeding (Stewart et al., 2013) both stated that growth faltering begins in utero, and that the first two years of life provide an opportunity to mitigate its effects and prevent further faltering. The pooled analysis of 18 risk factors (Danaei et al., 2016) attributed the largest influence on stunting to being SGA and term.

The central importance given to markers of foetal growth restriction and gestational age in these reviews is in broad agreement with another pooled analysis of 19 birth cohorts that quantified (as odds ratios) the contribution of foetal and prenatal factors to stunting (Christian et al., 2013). While the odds of stunting (compared to term, AGA children) were highest for children born SGA and preterm (OR 4.51; 95%CI 3.42, 5.93) than those born SGA and term (OR 2.43; 95%CI 2.22, 2.66), the authors concluded that SGA had strong associations with nutritional status independent of gestational age (i.e., term or preterm). They also highlighted that birth length is more strongly correlated with length gain than SGA, and so a shift away from birth weight

in research would add more mechanistic insight across contexts (Christian et al., 2013).

In relation to FGR and size at birth, none of the reviews discuss an auxological phenomenon called canalization (Czerwinski and Towne, 2004). Canalization is the propensity of a growth-related characteristic for a certain trajectory. Applied to birth weight or birth length, infants would be more likely to track the growth centile in which they are born, a result of strong genetic influence that remains somewhat insensitive to environmental changes. Therefore some of the correlation between size at birth and childhood length is expected because height is a highly heritable trait. None of the reviews comment on the importance of separating genetic and environmental components in understanding their influence on growth faltering in contexts where it is common.

The reviews also highlight the lack of clarity around IYCF which is reflected in the empirical evidence. Danaei et al (2016) don't account for IYCF sufficiently and so it is not quantified in their review. They focus on BF, which appears to have a low PAF, but leave out complementary feeding. Conversely, Stewart et al (2013) give complementary feeding prime position in their framework, but do not quantify its influence and state that the exact mechanisms will be context-specific rather than globally applicable. However, evidence on the effect of breastfeeding promotion trials on infant length is inconclusive, as a meta-analysis of 17 studies (Giugliani et al., 2015), based on intention-to-treat analyses, showed no effect on LAZ at six months (β 0.03; 95%CI -0.02, 0.08). But the trials did not report on growth into early childhood, or account for subsequent complementary feeding, and so the question of context-specific effects in experimental evidence remains open.

Further, breastfed infants, in studies comparing their growth to that of formula-fed infants, generally exhibit faster growth up to two months and then slower growth up to 12 months (Lind et al, 2018). This finding is attributed to the higher protein content of formula, which promotes production of two growth-promoting hormones, insulin-like growth factor 1 (IGF-1) and insulin. The lower IGF-1 and insulin levels in breastfed infants are sustained into the second year of life when breastfeeding continues alongside complementary feeding. This is known as the early protein hypothesis (Koletzko et al, 2005).

Hermanussen and Wit's (2017) position deviates most from the other reviews. They club all modifiable and non-modifiable factors together, and propose that they have little to do with the aetiology of malnutrition in LMICs or the ways in which it can be successfully addressed. While their social targeting of height hypothesis posits an interesting mechanism, the supporting evidence is unconvincing because they cite trials that have failed to confirm widespread beliefs rather than support the ones they hold. If social targeting of height cannot be tested experimentally, it could be interrogated using existing studies and triangulation using a range of analytical techniques.

This divergent conceptual framing across reviews does not provide a clear picture of the determinants of infant linear growth, but it highlights that the underlying empirical base is also full of contradictions.

2.6.1.2 My review

In my systematic review I identified several infant, parental, and environmental or household-level determinants of infant linear growth, which I grouped into 18 categories. The influence of parental heights, maternal weight, and favourable SEP and WASH conditions on linear growth was consistently positive. Factors that had a consistently negative influence were diarrhoeal disease, maternal and partner smoking, and large household size. Evidence on the role of IYCF was mixed, as were sex-differences in various linear growth outcomes.

An important finding from studies in urban informal settlements is that WHO-recommended IYCF practices were negatively associated with growth patterns, growth velocity, and risk of stunting. Qualitative evidence suggests that the factors that prevent urban poor women from exclusively breastfeeding are largely social and structural (Kimani-Murage et al., 2015b), with the more deprived ones less likely to breastfeed, and there is often a trade-off between work and childcare (Kabir and Maitrot, 2017). However, it is unclear whether the paradoxical negative or neutral association between IYCF and growth observed in informal settlements is a result of real mechanisms that operate in the unique context of urban poverty in such settings.

The influence of a wide range of SEP indicators on growth is not surprising, since favourable conditions protect children from a range of health and nutrition disorders. Findings from across LMIC and HIC settings were mostly congruent, lending

strength to the importance of SEP in early life. Given the strength of evidence, it is surprising that Danaei et al's risk factor analysis (2016) did not include any SEP factors. However, it is interesting that two studies from HIC settings supported Hermanussen and Wit's social targeting hypothesis. Dutch children born to women with low educational attainment showed accelerated length velocity (Silva et al., 2012), and British Pakistani children grew faster and taller than white British children despite lower length at birth (Fairley et al., 2013).

The list of exposures and covariates identified in my review broadly overlaps with those described in the four reviews. However, my review identified three others that are missing from these frameworks.

First, maternal smoking and partner smoking had a consistently negative influence on growth. While cigarette smoking among women is less common in LMICs than HICs, use of smokeless tobacco products can be widespread in some settings, with deleterious consequences for health and pregnancy outcomes (Gupta and Ray, 2003, O'Connor, 2012). A cross-sectional survey of women in Mumbai's low-income suburban community found that 22% of adult women consumed some form of tobacco (Mishra et al., 2015).

Second, paternal anthropometry had a positive influence on infant length, but the role of fathers was not mentioned in any of the frameworks. Paternal height is a strong marker of offspring height as well as several SEP indicators, and also associated with maternal height (Perkins et al., 2016) and so its omission is a potential source of unmeasured confounding in observational studies.

Third, household overcrowding had a negative relationship with infant linear growth across several settings, and this applied to increasing numbers of adults as well as children in a household. However, this leaves out nuance that merits further investigation. A recent study among a Maya community in rural Mexico elucidates the potential trade-off between early childhood growth and family size (Kramer et al., 2016). Younger siblings are more likely to pose competition to the breastfeeding child who may be displaced at the breast, and this is more likely when birth intervals are short and fertility is high. Older siblings can be a source of hazard or support. If the infectious load among children in a community is high, older siblings will expose younger siblings with less developed immune systems to greater disease risk. On the other hand, in communities where older siblings are a source of childcare or

contribution to the household economy, there is likely to be a positive association between large family size and child health and growth outcomes. Children are more susceptible to the effects of sibling competition during the complementary feeding period, which is also likely to be a time of greater vulnerability to growth faltering. And so the role of older siblings needs to be examined in more detail.

However, my review did not identify one factor that I was interested in *a priori*: pregnancy intention. There is evidence of poor growth outcomes among children born as a result of unplanned pregnancy in cross-sectional studies (Shapiro-Mendoza et al., 2005, Upadhyay and Srivastava, 2016). However, this has not been examined in longitudinal studies with robust measurement of pregnancy intention in urban informal settlements.

2.6.2 Methodological issues in analytical techniques used

In my review of longitudinal studies, 29% of 77 articles used only one data point to produce a growth metric, comparing several manually calculated values at each age instead of modelling curves derived from multiple serial length measurements. While calculating a metric at each age is not wrong per se, it wastes much available information, and answers a limited set of research questions. This implies that nearly a third of recent available evidence on the determinants of infant growth is based on suboptimal analytical methods. Further, the full range of growth modelling techniques available was not used across the included studies. Non-linear mixed effects models, which can be a powerful growth modelling technique (Johnson, 2015), were particularly under-represented.

Another missed opportunity was the limited focus on growth parameters other than size. Few studies assessed growth velocity or tempo, with the result that across the review there is very little description of patterns of growth (velocity, acceleration) and how these differ between and within groups.

The widespread under-reporting of missing data and attrition in the 77 articles hints at possible bias. If the underlying mechanism the leads to non-response or loss to follow-up is related to the exposures or covariates under investigation, study results will be biased. In my review, 49% either had >10% missing data or did not investigate missing data patterns at all.

2.6.3 Limitations of my review

I conducted all steps of the review process alone, and the results presented here were not independently replicated by another researcher. This may have introduced bias in screening and data extraction, quality assessment, and misclassification of growth metrics and study variables.

I relied on published systematic reviews and narrative reviews to identify risk factors for linear growth in cross-sectional studies. These reviews covered a different time frame (most recent one was published in 2016) to my review of longitudinal studies (2010 to 19 June 2018). I may have missed more recent cross-sectional studies that would have appeared in a primary review and enabled more thorough comparison.

My quality assessment was based on the Newcastle-Ottawa Scale, a generic tool for observational studies. Longitudinal growth studies have particular features which contribute to their validity, such as justification of the growth parameter used and its biological meaning. There is currently no validated quality assessment checklist or tool that offers tailored items to conduct a thorough critical appraisal and evaluate whether the growth analysis or modelling was appropriate. It is possible that my quality assessment excludes aspects of studies that were biased.

I also did not consider the heterogeneity of growth metrics in a systematic way (for example, by assigning weights to particular growth analysis methods that are demonstrably more robust than other approaches). However, despite their importance for making inferences about the causes of growth (Leung et al., 2018), there is little guidance on how best to account for methodological heterogeneity in a formal review.

Finally, my review was based on a qualitative synthesis of the literature, and I did not quantify the effect of different risk factors on infant growth and pool the magnitude of association from comparable studies.

2.6.4 Conclusion

Despite greater availability of longitudinal data on linear growth and several advances in growth modelling techniques, the findings of my literature review indicate that there is scope for improving growth studies to better serve the objectives of global health and nutrition. I discuss two key concerns below. First, few

studies embrace causal approaches to conceptualizing or investigating linear growth. And second, the current state of methodology and evidence raises several ethical issues around the conduct of growth research studies.

2.6.4.1 Lack of causal thinking

The limited causal thinking on the causes of linear growth failure is problematic. There have been several trials and meta-analyses of interventions to improve growth in early life, and these have either shown small or no effects on normalising linear growth faltering (Giugliani et al., 2015, Sguassero et al., 2012). It is possible that studies are targeting causes that are poorly understood, and so well-intentioned action is largely ineffective. Second, if the underlying mechanisms of linear growth faltering are poorly understood, a shift towards mechanistic thinking and methods for causal inference based on longitudinal data is timely. If we understand how certain exposures make individuals and groups grow differently in early life, we can address these more effectively.

Further, size is one of several growth parameters, but its assessment dominates research and policy action. Factors that affect the rate at which infants grow are not as well defined, and whether these represent a distinct set of causes to those that result in shorter length has not been established. Given the large amount of longitudinal data now available from developing countries, it is possible to ask questions that relate to growth velocity, and also to look at the influence of exposures that vary over time.

The lack of causal thinking also has consequences for the relative importance of different exposures, as well as the estimated size of their impact. Few studies use causal mediation analysis, and so there is little research showing the direct and indirect effect of exposures. It is possible that the direct effect of some exposures that form the focus of much intervention is very small, and that their impact on linear growth is largely due to an indirect effect mediated by another factor that does not receive much attention. Such relationships could vary by context, and are worth examining in existing datasets from longitudinal studies.

2.6.4.2 Ethical considerations

The recent rise in longitudinal studies of infant growth in developing countries is encouraging because they address important scientific questions. However, these studies are also often conducted in populations where participation in research

brings time-related and other unobserved costs for study participants, who are often from low-income or deprived communities. In infant growth studies, the burden of participation tends to fall on the child's mother. Frequent data collection can place additional demands on individuals who give up their time to participate, and the degree of compensation (monetary or non-monetary, if any) is not reported in studies. Among socioeconomically deprived communities, the decision to participate can sometimes be based on access to indirect benefits offered to study members rather than on the informed consent process (Ravinetto et al., 2015), raising the risk of exploitation.

When researchers require intensive participation from low-income families for long periods of time, they have an ethical duty towards research subjects to use data appropriately in analyses. For example, several studies measured infants every month from birth to two years, but used only the first and last measurement in their analysis, wasting the thousands of data points and participant-hours in between. Further, 29% of articles derived their growth metric from just one data point, despite having an average of 7.2 measurements per child. If longitudinal data are not intended to be used as such, they should not be collected in the first place. Researchers should be required to justify intensive data collection in their statistical analysis plans and demonstrate adequate expertise to undertake appropriate data modelling. The argument for improving the methodological quality of health research to prevent unnecessary data collection is seldom made, but its ethical ramifications must be considered more carefully when participants are drawn from vulnerable communities (Chalmers and Glasziou, 2009).

Chapter 3 Methods

Summary

This chapter presents the data collection methods for the cohort study used in my research. It begins with a description of study design, aims, setting, and process. I describe the measurement protocol for the main variables I used in my research. These relate to infant and young child anthropometry, feeding practices, and morbidity, parental anthropometry, socioeconomic position, and parental health and behavioural measures. I also briefly discuss additional aspects related to study size.

I used data from the SNEHA Centres Infant Nutrition Cohort to answer my research questions. I did not design the cohort study or collect any data used in the thesis. However, I was present at the study site in Mumbai for the last six months (October 2015 to April 2016), and this gave me an opportunity to understand the trial and cohort processes through interaction with investigators and project staff. The following description of the cohort study design and data collection activities is based on unpublished protocols and documentation, and the baseline and follow-up questionnaires prepared by David Osrin at IGH, Sushmita Das at SNEHA, and other study investigators (see Appendices 3), related procedures described in the trial publications (Shah More et al., 2017, Shah More et al., 2013), my reading of the study design and methodological literatures, and additional discussions with the study team in Mumbai.

3.1 SNEHA Centres Infant Nutrition Cohort

3.1.1 Study design

The SNEHA Centres Infant Nutrition Cohort is a prospective, observational, birth cohort in a closed population nested within the intervention arm of a cluster-randomized controlled trial (RCT). The trial site comprises 40 informal settlement clusters, 20 in the intervention arm and 20 in the control arm, in M-East and L wards in Mumbai, India. Infants born in the 20 intervention clusters over a year between March 2013 and March 2014 were recruited into the study at birth and followed-up until March 2016. Infants' parents and siblings were also included in the study as participants or proxy respondents linked to the index infant.

The study is **prospective** in the following ways. First, data on exposures and covariates, even if they took place before the study began, were collected concurrently or before the outcome of interest had occurred, such that the exposure information could not have been influenced by the outcome (Rothman et al., 2008a). Second, data collection took place after the main research questions and study design had been developed, and baseline characteristics were collected for the explicit purpose of later relating them to the outcome of interest. This differs from a record linkage or historical cohort study in which data are obtained from existing scientific or administrative databases – which may have been set up for a purpose other than to link with the outcome of interest – after research questions have been formulated (Vandenbroucke, 1991).

The study is **observational** in that estimates of relationships between variables of interest are based on data derived from observation rather than experimentation, and exposure status was not assigned randomly (Rothman et al., 2008a). The cohort was nested within an ongoing cluster RCT at onset, but does not aim to evaluate the short- or long-term effects of the trial interventions on health outcomes. All participants in the cohort were in the intervention arm of the trial and were offered the health services provided at SNEHA centres. However, the duration spent in the trial implementation period differed between cohort participants due to the staggered nature of the trial phases and the distribution of births over a calendar year.

It is a **birth cohort in a closed population** because individuals in a population of interest born within a defined period were recruited into the study if they met certain criteria, and these same individuals were followed up over time. Individuals born before or after the defined period were not added to the study. The composition of the cohort did not change over the study period, in contrast with a dynamic population which may gain new members through birth or immigration (Greenland and Rothman, 2008). However, for some time-varying measures of interest for which an individual's status could change with time, an individual participant could move between exposure groups during the follow-up period. For example, an infant who received a food supplement package of fortified flour (as complementary feeding) from the Integrated Child Development Services (ICDS) scheme at seven, eight and ten months would switch between supplementation exposure groups several times in the first year because they did not receive any at six, nine, eleven, or twelve months. In this sense, the cohort is not a fixed cohort with exposure status ascertained at and constant since baseline. In the event of no losses from the

cohort, it could not be classified as a closed cohort with respect to these time-varying measures, making it hard to calculate incidence rates accurately (Rothman and Greenland, 2008).

3.1.2 Original aims and research questions

Appendix 3.1 is the original cohort study protocol written by David Osrin and Sushmita Das. Briefly, the original aim of the cohort study was to develop detailed and contextual understanding of infant growth in Mumbai's informal settlements. The impetus for this stemmed from exploratory work using data from children in a previous trial that ran from 2007 to 2010 (Shah More et al., 2012). Analysis of growth in early life failed to identify a particular age in infancy at which growth faltering begins among children living in the city's informal settlements (Das et al., 2012). This ambiguity was attributed partially to lack of sufficient data in the first year of life with which to investigate a 'downturn', highlighting the need for a more focused prospective, longitudinal study. The SNEHA Centres trial provided an opportunity to embed an observational study within ongoing surveillance activities, and to ask questions that would help identify suitable and optimally-timed interventions to address linear growth faltering in informal settlements.

Primary questions

1. At what point does growth faltering begin in slum-dwelling children?
2. How does their growth relate to parental body size?

Secondary questions

1. What sort of diet do infants and young children have?
2. How does growth faltering relate to morbidity?
3. Is there a gender dimension to growth faltering, diet, morbidity and care seeking?

3.2 Study setting and participants

3.2.1 Trial intervention clusters – source population

The SNEHA Centres trial site was spread across two of 24 municipal wards in eastern Mumbai. These wards had among the lowest Human Development Indices,

0.05 in M-East ward and 0.29 in L-ward (Municipal Corporation of Greater Mumbai, 2010), in the city and a high density of informal settlements. They are low-lying, flood prone areas, and some settlements are situated close to the city's largest waste-disposal site. Three-quarters of the settlement populations are migrants from the northern states of Uttar Pradesh and Bihar, and 15% are from Maharashtra. Informal settlements identified in these two wards were included in a sample frame of 159 clusters of approximately 600 households. Settlements that had been part of a previous trial were excluded, and larger areas were divided into clusters along distinct physical boundaries. All clusters in the sample frame were visited by a team of investigators who used a scorecard (Osrin et al., 2011) to conduct a rapid vulnerability assessment, identifying clusters at high maternal and child health risk. Forty clusters with the lowest scores were included in the study and were randomly assigned to the intervention or the control group.

SNEHA centres were set up in the 20 intervention clusters to deliver a range of integrated health services to local residents. Each cluster was run by three community organizers. The study intervention addressed maternal and reproductive health, neonatal and child health, child nutrition, and prevention of violence against women and children. These issues were woven into activities delivered through home visits, group meetings, day care for malnourished children, community events, service provision and referral, and liaison with local municipal and public service providers to improve uptake of services. Community organizers also addressed infant and young child feeding practices during home visits and group meetings, and conducted regular growth monitoring of children below five years to identify those at risk of malnutrition (Shah More et al, 2017).

Control clusters did not receive any intervention during the course of the trial, and families in these neighbourhoods were only visited for pre- and post-intervention data collection activities. In contrast, eligible families in intervention clusters were contacted at least once a month by SNEHA staff during the intervention period to carry out trial surveillance activities.

The trial's three primary outcomes were met need for family planning among married women (15-49 years), proportion of fully immunized children (12-23 months), and proportion of children below five years with wasting (weight-for-height or weight-for-length z-score <-2 SD of the WHO Growth Standards).

Post-intervention, met need for family planning was higher in intervention areas (OR 1.31; 95%CI 1.11, 1.53). However, the trial arms did not differ with respect to childhood wasting (OR 0.92; 95%CI 0.78, 1.12) and immunization (OR 1.30, 95%CI 0.84, 2.01) in intention-to-treat analysis. In per-protocol analysis, the proportion of children with wasting decreased by 2.5% in the control arm and 6.4% in the intervention arm (difference between group = 0.020), indicating some beneficial effect of the intervention on child growth (Shah More et al, 2017).

The SNEHA Centres Infant Nutrition Cohort study was conducted in the 20 intervention clusters, as the ongoing surveillance system and support offered a feasible setting and infrastructure for an embedded cohort study involving high-frequency data collection and regular contact with participants. Further, the health promotion and services available at SNEHA Centres ensured that children in an embedded cohort study who exhibited growth faltering could be easily referred to the local centre for assistance. A cohort study which included children born in control clusters would have proved logistically challenging and added significantly to the cost of the observational study.

The trial's preintervention census in 2011-13 of 12,239 households in 6976 homes sheds some light on the demographic and health characteristics of the cohort's source population. A summary of key population characteristics is as follows.

The median number of households per cluster was 625. Sixty percent of homes were owned by occupants, and 64% families had ration cards granting them access to government welfare programmes and services. Twelve percent of homes were temporary structures, 26% were partly robust, and the remaining 62% were made of robust materials. Over 99% had electricity and two-thirds had access to a metered supply. Sixty percent purchased drinking water from tankers or in containers, 21% had a private tap, and 19% used a community tap stand. Of 7947 women of reproductive age (15-49 years), 27% had had a pregnancy in the last two years, ten percent were nulliparous, and 36% had four or more children. Twenty-nine percent reported using some method of family planning. Nearly half (49%) were educated up to secondary school, 37% had no formal education, and 6% had some higher education. Eighty-three percent were Muslim, 16% Hindu, and less than 1% were from other religions. Six percent had lived in Mumbai for less than a year; 25% had lived in the city for more than ten years and 33% were lifelong residents. Forty-two

percent had lived in their current home for all the years that they had lived in the city (Shah More et al., 2017).

Of 1905 births in the two years before the trial, 83% were institutional deliveries and 15% were home births. Sixty-five percent of 945 children aged 12-23 months had been fully immunized. Eighteen percent of 3550 children below five years were wasted (weight-for-length z-score (WLZ) below -2 SD of the WHO Growth Standards), indicative of acute malnutrition; and 46% of 3541 were stunted (length-for-age z-score (LAZ) below -2 SD of the WHO Growth Standards). Use of ICDS was low: 11% of 4767 eligible children under five used it at all, and less than 10% accessed daily food supplements, monthly health check-ups, regular early childhood development intervention, or quarterly weight monitoring (Shah More et al., 2017).

Forty-four percent of infants had been breastfed within an hour of birth, and 62% had been exclusively breastfed for the WHO recommended duration of six months. Complementary feeding practices were poor: only 13% of children aged 6-23 months were fed diverse diets consisting of four or more different food groups (Minimum Dietary Diversity – MDD), and five percent had diets of adequate nutritional quantity and quality (Minimum Acceptable Diet – MAD) in the previous 24 hours (Shah More et al., 2017).

Intervention areas were broadly similar to control areas at baseline with respect to demographic, environmental and health characteristics (Shah More et al., 2017).

3.2.2 Study inclusion criteria

The main inclusion criteria, as described in the protocol (Appendix 3.1), for recruitment into the cohort were:

1. Family live in a SNEHA Centres intervention cluster.
2. Family say that they intend to stay in SNEHA Centres intervention cluster for at least 6 months.
3. If weight not measured by Investigators within 72 hours of birth, possession of an institutional birth weight record.
4. If birth outside the cluster, institutional birth weight record available and infant visited and weighed within 2 months.
5. Live singleton infant born at 8 months gestation or greater

6. Infant born from 1st March 2013.

3.2.3 Exclusion criteria

I applied one further criterion for data analysis reported in the thesis by excluding infants who had any clinical or congenital disorders which affect patterns of linear growth. During a visit to the study site in 2015-16, I came across a study participant who had Down's syndrome and was in the process of accessing specialized care. Children with Down's syndrome exhibit different growth patterns to children without the condition (Van Gameren-Oosterom et al., 2012, Zemel et al., 2015, Zemel, 2017), and including this child's data to study growth patterns would have introduced bias. I decided to exclude any participants with diagnoses of medical conditions associated with growth disorders. Condition-specific growth references have been developed for some populations (Myrelid et al., 2002), but such disorders are rare and it was unlikely that this cohort would have had sufficient numbers to merit subgroup analyses. I asked cohort investigators to identify other children in the study who had known health conditions that impaired normal growth so that I could exclude them from data analysis.

Further restrictions for main analyses were related to completeness or other features of data independent of any baseline characteristics, and are discussed in relevant sections in Chapters 6-8.

3.3 Study process

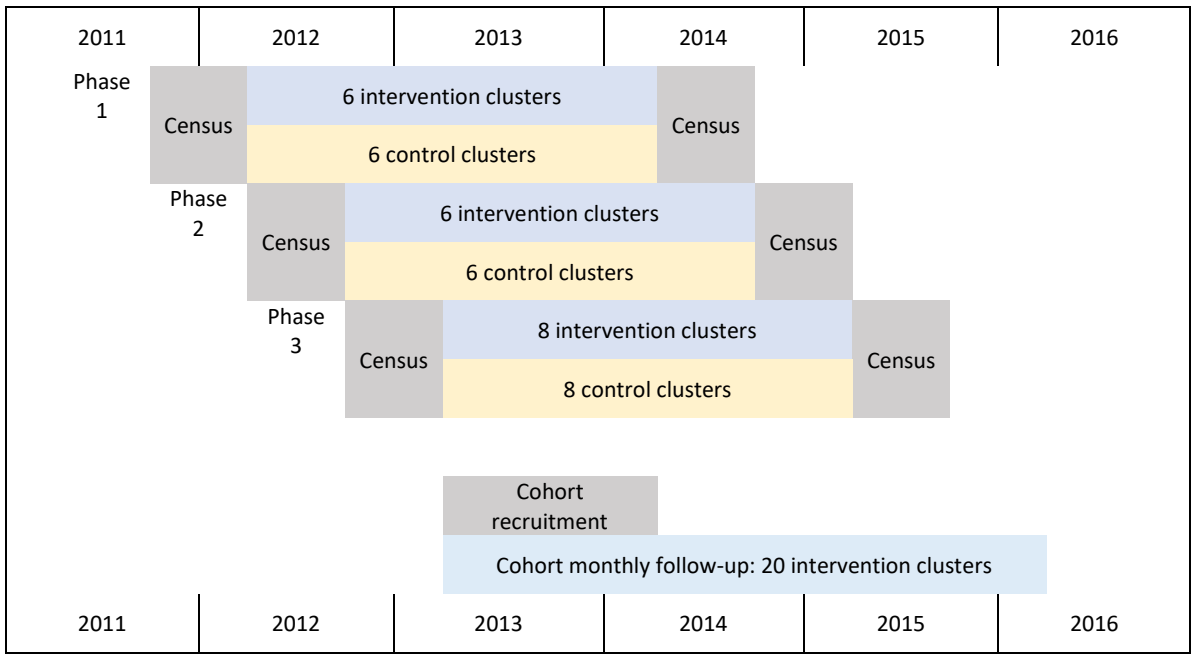
3.3.1 Cohort and trial study teams and timelines

The cohort was embedded within the trial implementation and evaluation structures, but conducted by a dedicated team of investigators. Ten field investigators carried out all data collection activities, working in five pairs. Two project officers supervised investigators and reported to a programme coordinator. The cohort team were supervised by the trial manager.

The trial's three implementation phases were initiated at six-monthly intervals in August 2011. Each phase consisted of a preintervention census over the first six months, two years of intervention implementation, and a further six months of census following the intervention. Recruitment to the cohort began after the trial's preintervention census was completed in all clusters in January 2013, running from

March 2013 to March 2014. Follow-up data collection activities began when the cohort study commenced in March 2013, and ended in March 2016, when the youngest participant (born in March 2014) turned two years old (Figure 3.1). Participants who were two years old by March 2015 remained in the study until March 2016. All data used in the thesis were administratively censored at the end of this follow-up period. Additional data collection beyond March 2016 is not covered.

Figure 3.1 Trial and cohort timeline



Source: Adapted from Shah-More et al., 2017.

3.3.2 Participant recruitment and follow-up

The trial targeted women of reproductive age (15-49 years) and children below five years. Community organizers carried out regular home visits during which they identified and referred women and children for health services. During the cohort recruitment period (March 2013 to March 2014) community organizers and cohort investigators identified pregnant women in each cluster and encouraged them and their families to inform SNEHA staff at the centre of the birth of a baby. Other members of the community were also asked to inform SNEHA staff of births in their vicinity. Community organizers informed cohort investigators of new births in each cluster, who then visited homes as close to the identification of births as possible to speak to families about the cohort study. Primary caregivers, usually the mother, of infants who met the inclusion criteria were given a participant information sheet in Hindi and further details about the study process. Caregivers who agreed to

participate gave signed consent and were enrolled in the study (see Appendix 3.2 for English translations of the participant information sheet and consent form).

Infants were followed up monthly; cohort investigators visited infants at home or requested that families bring children to the local SNEHA centre for anthropometry and questionnaire data collection. If the mother (or primary caregiver) was not able to respond to questionnaire modules then, investigators measured the child and returned later that day or another convenient day to collect questionnaire responses. Additional, one-time measurements of parents or siblings were also incorporated into monthly visits. Investigators requested families to inform them of imminent travel plans so that subsequent visits could be re-scheduled if necessary. If families were travelling with the infant for longer than a month and likely to miss a follow-up visit, investigators kept their records open for the duration of their absence. Upon the family's return, investigators re-established contact and participant follow-up continued.

If families were moving to a home within the cluster, they were retained in the study. If families moved to a home in another intervention cluster, the child's paper and digital records were transferred to that cluster, and if necessary, re-assigned to the investigator team responsible for that area.

If families migrated to another part of the city or country, records were kept open for up to six months, during which time no data were collected for the participant, and subsequently closed if it was confirmed that the family would not be returning to the cluster. Records of infants who died after enrolment were closed once a project officer trained in verbal autopsy was able to visit the home to record the cause of death based on an institutional death certificate or, if unavailable, conduct a verbal autopsy.

3.3.3 Data collection instruments

An electronic data capture system was set up on password-protected smartphones using open source software, CommCare (Dimagi, Cambridge, MA, USA) in Google Android (versions 3.0 to 4.4). Electronic data collection was in use in several projects at SNEHA at the time, including the Centres trial. Questionnaires were programmed onto phones with validation constraints and skips. Investigators entered questionnaire responses directly into phones, but anthropometric

measurements were first recorded in notebooks before duplicate manual entry into smartphones. The data collection form fields were programmed to appear in Hindi on the smartphone, with numerical input in English. Forms and data could be viewed in Hindi or English on the online server. Smartphones were also used to collect Global Positioning System (GPS) coordinates for each infant's home at the baseline interview. Data were submitted at the end of each month through a Wi-Fi network and uploaded to the CommCare Open Data Kit project server. Data managers generated Excel spreadsheets of each month's data and shared these with project officers, who checked for errors and completeness and discussed any clarification with cohort investigators at site visits or monthly meetings.

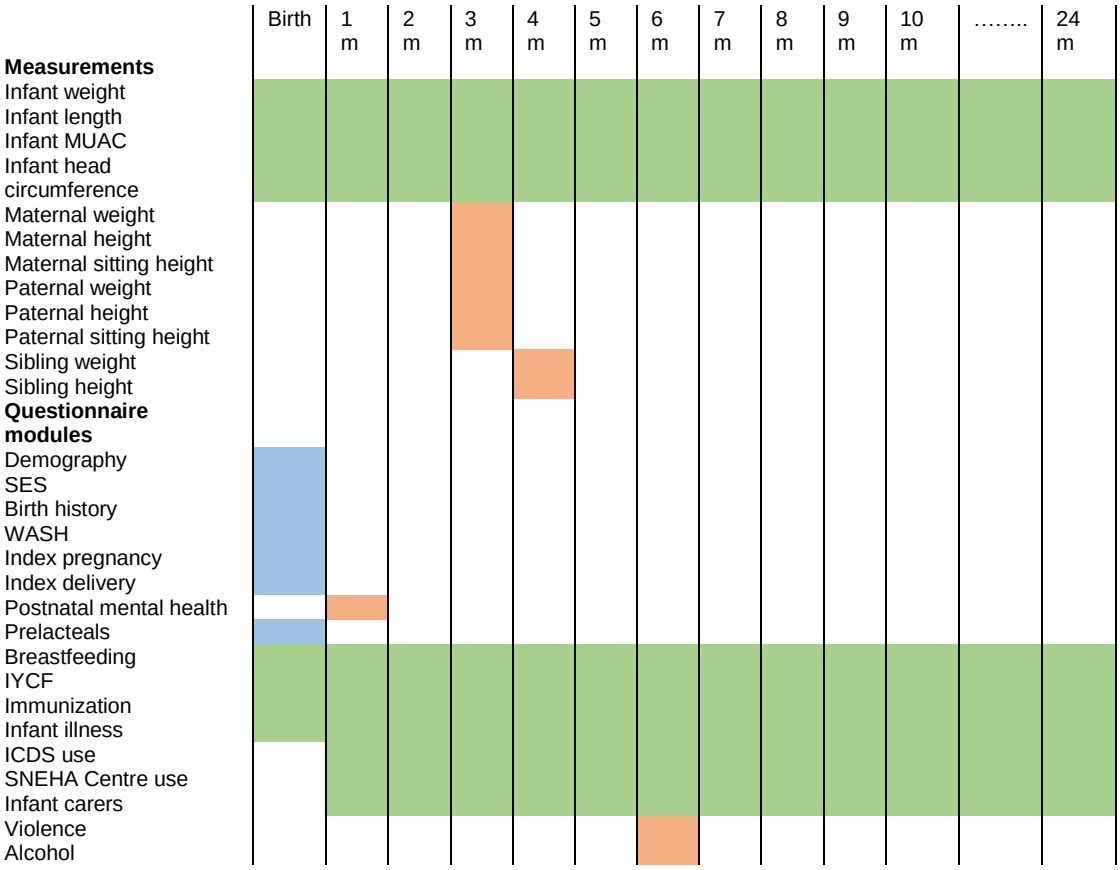
The cohort's electronic records database included a registration questionnaire completed for each infant at enrolment, detailing household and identity information. This master registration list was then linked to all participant data at baseline and follow-up. Baseline questionnaire modules were included as one form which could be filled in only once per participant. Infant anthropometry, IYCF, morbidity and care-seeking modules were incorporated into another form which could be filled in multiple times for each participant, linked to their registration details. Modules for parent and sibling data collection were created as separate forms which could only be filled in once per participant for each parent and for up to 3 siblings. All study forms were made visible on investigators' handsets. Records were grouped by the ID numbers of investigators responsible for data collection in each cluster. Investigators entered their ID numbers upon opening the application on their smartphones, and were directed to a cascading list of clusters and participants currently allocated to them.

3.3.4 Baseline and follow-up

Data collection over the study period included three components (see Appendix 3.3 for the full study questionnaire). At baseline, primary caregivers responded to an interviewer-administered questionnaire and investigators measured infant anthropometry. Questionnaire modules covered a range of socio-demographic and health-related topics pertaining to the infant, parents, and households. At monthly follow-up visits, investigators assessed infant anthropometry, and asked caregivers about infant feeding and care practices in the last 24 hours, and infant morbidity, health, and use of welfare services in the past month. Additional questionnaire or anthropometric assessments of parents and siblings were incorporated into monthly

visits following a schedule of milestones or optimal periods for observation (Figure 3.2). No biological samples were collected at baseline or follow-up.

Figure 3.2 Study diagram



Source: Adapted from cohort study protocol (Appendix 3.1). Blue = measured at baseline only; orange = measured only once post-baseline; Green = serial measurement. MUAC: mid upper-arm circumference; SES: socioeconomic status; WASH: water, sanitation and hygiene; IYCF: infant and young child feeding; ICDS: Government of India Integrated Child Development Services; SNEHA: Society for Nutrition, Education and Health Action.

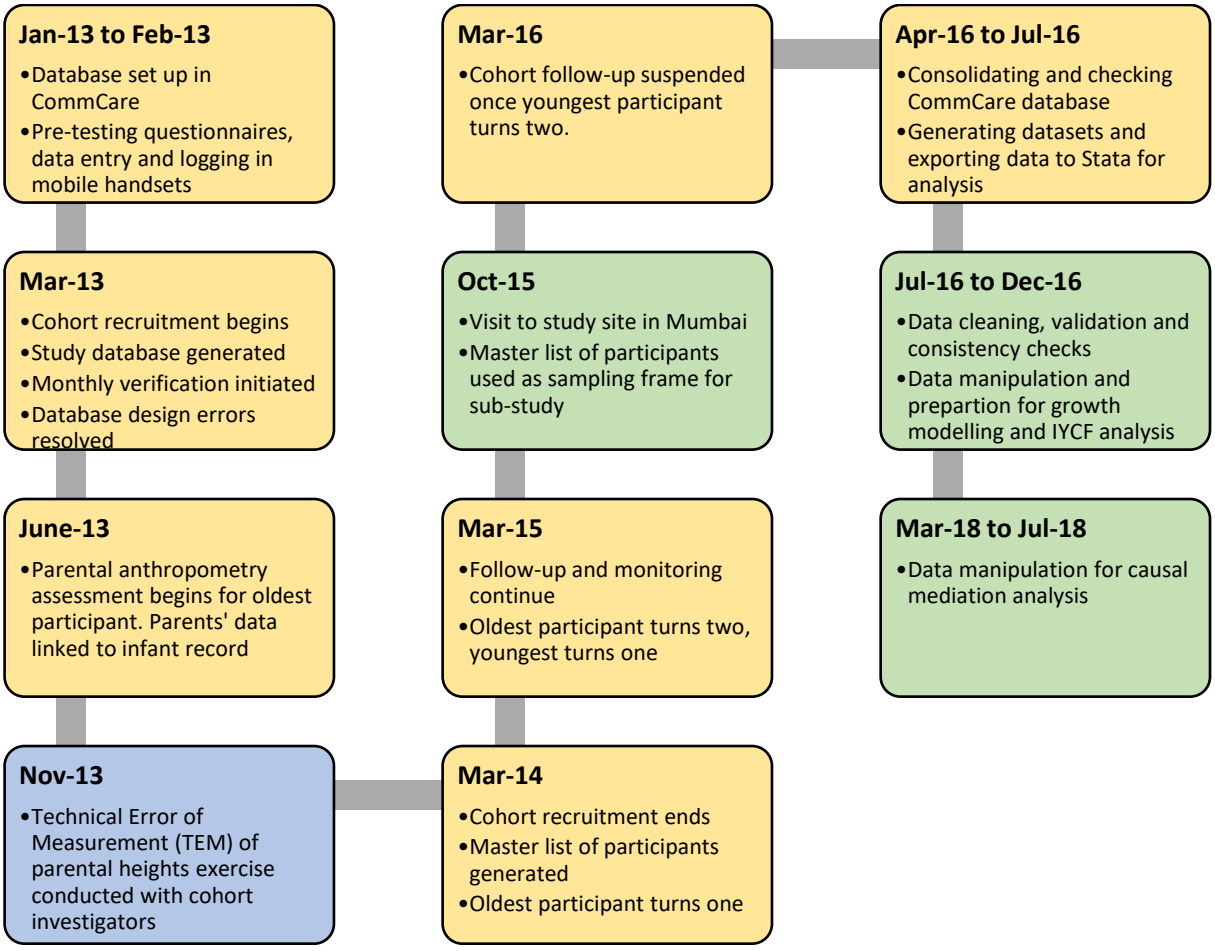
3.3.5 Training

David Osrin and Sushmita Das trained cohort investigators in 2013 before the study began. Investigators received information and training on recruitment and enrolment procedures, baseline and follow-up survey administration, and correct infant anthropometric assessment. Ethical considerations related to each procedure were also explained. Refresher training was conducted as required over the follow-up period. The Technical Error of Measurement (TEM) assessment outcome is reported in Appendix 3.4.

3.3.6 Data timeline

I did not design or manage the cohort database while the study was in progress. Sushmita Das supervised two data managers at SNEHA who worked closely with the cohort field investigators and managers to monitor data entry and address any issues over the study period (March 2013 to March 2016). Data validation and consistency checks were built into the measurement protocol, data entry and data management processes, and also regularly addressed in additional training and quality control activities. Database management activities began three months prior to recruitment and continued for three months after follow-up was suspended in order to consolidate the database and generate Stata files for analysis. I was given copies of the cohort datasets in July 2016, after which I began data cleaning and management to prepare and merge several datasets for analysis. Figure 3.3 describes data management activities carried out between 2013 and 2018.

Figure 3.3 Data management timeline and activities



Colour code for researcher leading activity: Yellow – Sushmita Das; Blue – David Osrin; Green – Komal Bhatia.

3.4 Infant and young child anthropometry

3.4.1 Length / height

3.4.1.1 Rationale

Childhood anthropometry is an important marker of the health and wellbeing of children in developing countries, and its measurement in longitudinal birth cohort studies as a health outcome is recommended (Golding, 2009). Frequent measurement in the first year of life allows for detailed characterization of the rapid growth that takes place during infancy (de Onis et al., 2004), which was an important consideration in this study population (Das et al., 2012).

Recumbent or supine length is measured in individuals who are not able to stand up straight, and is widely used to assess linear growth in infants and young children up to two years old. Standing height, or stature, is measured thereafter (WHO, 2008b).

3.4.1.2 Measurement protocol

The first length measurement was recorded at or close to enrolment, ideally within 72 hours of birth if the infant was brought home by then. Subsequent measurements were taken at follow-up visits at least 28 days apart, until the end of March 2016. Assessment took place in SNEHA Centres or participants' homes, with the participant's mother or caregiver present in the room. Investigators followed standard procedures and used identical anthropometry instruments, Seca length boards accurate to 1 mm for supine length (children aged 0-24 months) and Leicester stadiometers accurate to 1 mm for standing height (after 24 months), throughout the study. Measurements followed the procedures recommended by Cameron (2004), with deliberate observation of the Frankfort plane during assessment.

The Frankfort or Frankfurt plane is used in anthropometric assessment to obtain a standardized position of the skull at the time of measurement, eliminating measurement error due to the variation in head shapes. It is obtained when the outer canthus or lower margin of the eye is in the same horizontal plane as the

external auditory meatus. The supinated Frankfort plane is vertical (Cameron, 2004).

Supine length was measured by two investigators. Investigators placed the length board on a flat, firm surface, usually the floor of the Centre or home, lay the infant on the board, and kneeled on the floor with their line of sight perpendicular to the instrument. One investigator removed the infant's headgear (caps, hats, wraps or bands) or footwear. They then positioned the infant's head in a supinated Frankfort plane with the top of the head touching the fixed end of the board and eyes looking straight up. The second investigator positioned the infant's body such that there was no arching of the spine or bending of the knees. The investigator held the infant's ankles, keeping the feet together, ankles at right angles and heels touching the moving plate of the board. They also ensured that the legs were extended and aligned with the board and the shoulders were not lifted off the board. After checking that the child was positioned correctly, they moved the plate to make contact with the infant's feet, applied slight pressure to the ankles to straighten the legs, and took a length measurement to the last complete millimetre. The investigator then took a second measurement by moving and re-positioning the plate, taking care to ensure that the infant's position was maintained or corrected if he or she had moved. If the difference between the two measurements was more than 5 mm, two further measurements were taken, and the final pair of measurements were recorded in a notebook. The average of the two length measurements was used in growth analysis.

Standing height for children above 24 months was measured by one investigator. The investigator placed the free-standing stadiometer on the floor of the SNEHA Centre or participant's home, ensuring that the area was firm and flat. The child was asked to remove any footwear or headgear, and was assisted by their caregiver or the investigator if necessary. The investigator instructed the child to stand upright against the backboard of the stadiometer with their feet together. The investigator then checked that the child's heels and back were in contact with the backboard, and the arms were relaxed by the side of the body, before positioning the child's head in the Frankfort plane. The headboard of the stadiometer was lowered to make contact with the top of the child's head. The investigator kneeled on the floor so that their eyes were level with the Frankfort plane, to check that the child's head was in the correct position. The investigator asked the child to take a breath, and then applied light pressure to the mastoid processes to hold the head in the slightly

raised position upon inhalation. The investigator asked the child to exhale fully, and applied slight upward pressure on the mastoid processes, while checking that the child's heels remained on the ground. Once the child had exhaled fully, the investigator took a height measurement read at eye level, to the last complete millimetre. The investigator took a second measurement by raising and lowering the headboard and repeating the inhale-exhale instruction to the child to ensure consistent positioning between measurements. As in supine length assessment, if the difference between the two measurements exceed 5 mm, two further measurements were taken, and the final pair of readings were recorded.

3.4.1.3 Strengths and limitations

A major strength of assessing body length or height is that anthropometry is an inexpensive method and can be measured accurately by non-specialist individuals who receive the appropriate amount and quality of training. Length is measured in studies across the globe and, if measured using a standardized protocol across settings, can be used to compare cohorts of contemporaneous children in different settings or several cohorts across time and geography. As a marker of growth, length measurements are amenable to several data transformations and manipulations that can highlight different parameters of growth. It is also possible to quantify intra and inter-observer measurement error by calculating the Technical Error of Measurement.

Two key limitations relate to measurement error and statistical manipulation. If the TEM of a group of observers is very large, the data are likely to produce biased estimates, which may or may not vary with participants' age and time in a follow-up study. This limitation can be minimized with adequate training and regular monitoring over the course of a research study.

A further limitation relates to the technical expertise and theoretical knowledge required to analyse longitudinal growth data. Taking multiple measurements per participant may not significantly increase the amount of training observers require to collect data with precision and validity, but handling longitudinal growth data requires greater knowledge of statistical modelling and human biology than working with a single measurement per child. This limitation can be harder to overcome, and failing to analyse growth data appropriately can result in misleading conclusions based on biased or incorrect estimates (Tu et al., 2013).

3.5 Infant and young child feeding

3.5.1 Rationale

Five broad components of diets are commonly measured using objective or subjective methods in research studies: energy intake, micronutrient or macronutrient intake, food item or food group consumption, dietary patterns, or dietary behaviours. In large studies, objective measures (direct observation, duplicate diets, or nutritional biomarkers) are seldom feasible due to their time- and resource-intensive application, and subjective measures are more amenable to epidemiologic research. Common subjective methods that involve participant report of food intake include food diaries (also known as food records or dietary diaries), 24-hour dietary recall, food frequency questionnaires (FFQ), dietary checklists, and dietary history. These subjective methods can be adapted to assess multiple dietary components, and have varying levels of participant burden, cost, and risk of bias (DAPA Measurement Toolkit, 2018). FFQs have been validated for use in longitudinal and birth cohort studies in developed and developing countries to provide information on nutrient intakes among individual infants and children in prospective cohort studies. Other methods that have been applied include 24-hour food recall, dietary diaries or food records, and short versions of existing validated FFQs (Emmett, 2009).

The assessment and interpretation of IYCF in the thesis is informed largely by the WHO schedule of IYCF indicators (WHO, 2008a, WHO, 2010) using point-in-time methods to assess population-level practices. Since 2010, these revised indicators have been used in a large number of Demographic and Health Surveys (DHS) and other national-level studies in LMICs. Based on the WHO guide, data to calculate IYCF indicators must be collected using a structured, interviewer-administered questionnaire. The tool consists of retrospective questions about initiation of breastfeeding, and an IYCF module based on a 24-hour recall period covering current breastfeeding and complementary feeding practices.

In most cases, the questionnaire must be adapted to suit study objectives, often integrated into larger survey schedules, and to the local setting to take into account the age groups and socio-cultural characteristics of the study population. For this cohort study, data on initiation of breastfeeding were collected as close to birth as possible and integrated within the baseline questionnaire on demographic and

health characteristics. The IYCF module was adapted by reducing the number of questions, modifying the list of food items, and including additional questions related to the consumption of salty and sugary snack foods. At each follow-up visit, the IYCF module covered three main practices: breastfeeding, complementary feeding, and consumption of snack foods. The main caregiver, usually the mother, responded to questions. If another caregiver was responsible for the child at the measurement occasion, this was noted at the start of the IYCF module.

3.5.2 Pre-lacteal and initiation of breastfeeding

3.5.2.1 Rationale

Administration of pre-lacteals is common practice across many cultures in South Asia (Khanal et al., 2016, Patel et al., 2013) where infants may be given honey, sugar water, non-breast milk, or other liquids before breastfeeding is initiated. The WHO definition of breastfeeding does not allow infants to receive any pre-lacteals, and therefore it is important to account for pre-lacteal feeding when summarising data to describe exclusive and predominant breastfeeding. However, the 24-hour recall method to assess breastfeeding practices at any age does not capture pre-lacteal feeds. These must be assessed separately, especially so that breastfeeding status in the first month of life is as accurate as possible. In the absence of direct observation of pre-lacteal feeding in the birth facility or at home, studies use self-reported data. Assessing pre-lacteal feeding and initiation of breastfeeding as close to birth as possible also minimizes recall bias.

3.5.2.2 Measurement protocol

In the baseline survey investigators asked respondents about any pre-lacteal feeding and timing of initiation of breastfeeding by asking how soon after birth infant (Table 3.1)

Table 3.1 Baseline survey questions on pre-lacteals and breastfeeding initiation

Question	Response constraints
Have you ever breastfed (NAME)?	Yes / No
[If yes to previous question] How long after birth did you first put (NAME) to the breast?	Number of hours OR number of days if >24 hours
In the first three days after delivery, was (NAME) given anything to drink other than breast milk?	Yes / No
[If yes to previous question] Which of the following was (NAME) given to drink? [programmed as separate questions to allow multiple responses]	Milk other than breast milk / Plain water / Sugar or glucose water / Gripe water / Sugar-salt solution / Fruit juice / Infant formula / Tea / Honey / JanamGhutti* / Other

**Janam ghutti* or *bal ghutti* is a home-made herbal paste used as a pre-lacteal, or to prevent and treat colic in infants.

3.5.3 Breastfeeding

3.5.3.1 Rationale

Breastfeeding has short and long-term benefits for maternal and child health. In LMICs in particular, longer duration of breastfeeding protects infants against infectious morbidity and mortality, and infants breastfed for longer have higher intelligence than non-breastfed infants and those breastfed for shorter durations. Longer duration of breastfeeding protects women against breast cancer and increases birth spacing (Victora et al., 2016). Despite several methodological limitations impeding causal inference (Kramer et al., 2018), measuring breastfeeding is useful in observational epidemiologic studies.

Determinants of breastfeeding at multiple levels (structural, setting-specific, and individual factors) operate by affecting different aspects of breastfeeding, including early initiation, exclusive breastfeeding for the first six months of life, any breastfeeding up to six months, and continued breastfeeding alongside complementary feeding in the second year of life (Rollins et al., 2016). The relationships between breastfeeding and other factors are therefore best articulated using time-dependent information on infants' intake of breastmilk and non-breastmilk liquids and solids over the first two years of life.

A single question asking women how long they exclusively breastfed an infant is prone to measurement error because the definition of EBF as understood by

participants may influence responses. There are two alternative ways to assess EBF in a survey (Greiner, 2014). The first method, point-in-time, is to use a 24-hour recall questionnaire asking respondents to list everything the infant received yesterday. The proportion of children who were exclusively or predominantly breastfed at that point in time can be calculated using this information. The second method is to obtain lifelong data, in which respondents are asked to report the age at which each of a list of liquids and solids was first introduced to the child. The duration of exclusive and predominant breastfeeding is then calculated using this life-long data. Both point-in-time and life-long data methods should be reported in studies where this information is available.

3.5.3.2 Measurement protocol

The first part of the IYCF module asked about infants' consumption of breastmilk and non-breastmilk liquids in the last 24 hours (Table 3.2). This was a point-in-time assessment of breastfeeding repeated at every follow-up visit. The length of questions was reduced to shorten the interview. Some questions were re-worded to provide greater clarity. For example, the question on daytime and night time feeding was split into two, and daytime was described as 'during sunlight hours' and night time as 'between sunset and sunrise'.

Table 3.2 Follow-up IYCF questions related to breastfeeding and non-breastmilk liquids

Question	Response constraints
How did you / they [caretaker] feed the baby?	Only breastfeeding / Bottle / Spoon/ Finger or hand / Cotton wick / Other
Are you still breastfeeding (NAME)?	Yes / No
How many times did you breastfeed (NAME) last night between sunset and sunrise?	0-10
How many times did you breastfeed (NAME) yesterday during daylight hours?	0-10
Was (NAME) given any vitamin drops or other medicine as drops yesterday during the day or at night?	Yes / No
Next I would like to ask you about some liquids that (NAME) may have had yesterday during the day or at night. Did (NAME) have any of the following?	(Read only)
Plain water	Yes / No / Don't know
Infant formula such as Lactogen	Yes / No / Don't know
Other milk such as tinned, powdered or fresh animal milk	Yes / No / Don't know
[if infant formula or other milk were give] How many times did (NAME) have (formula or non-formula) milk of any kind yesterday during the day or at night?	0-7
Lassi, *chaas* or other yoghurt drinks	Yes / No / Don't know
Fruit juice	Yes / No / Don't know
Clear broth	Yes / No / Don't know

Question	Response constraints
Tea or coffee	Yes / No / Don't know
Cold drinks such as Pepsi, Coke and Frooti*	Yes / No / Don't know
Any other liquids	Yes / No / Don't know

Notes: Adapted from WHO 2010. *Frooti, brand name of a popular mango flavoured juice drink.

Data from the breastfeeding component were combined with other IYCF data to generate variables based on criteria recommended by the WHO (2010) (Table 3.3).

Table 3.3 Criteria for breastfeeding practices used to generate variables

Breastfeeding practice	Infants must receive	Infants are allowed to receive	Infants are not allowed to receive
Exclusive breastfeeding	Breastmilk (including milk expressed or from a wet nurse)	ORS, drops, syrups (vitamins, minerals, medicines)	Anything else
Predominant breastfeeding	Breastmilk (including milk expressed or from a wet nurse) as the predominant source of nourishment	Certain liquids (water and water-based drinks, fruit juice), ritual fluids and ORS, drops or syrups (vitamins, minerals, medicines)	Anything else (non-human milk and food-based liquids in particular
Breastfeeding	Breastmilk (including milk expressed or from a wet nurse)	Anything else: any food or liquid including non-human milk and formula	NA
Bottle-feeding	Any liquid (including breastmilk) or semi-solid food from a bottle with nipple / teat	Anything else: any food or liquid including non-human milk and formula	NA

Source: (WHO, 2010)

3.5.4 Complementary feeding and consumption of snacks

3.5.4.1 Rationale

The importance of measuring and addressing complementary feeding practices to improve the health and wellbeing of children has gained increasing prominence globally (Bégin and Aguayo, 2017) and in South Asia (Menon et al., 2015). With greater availability of standardized IYCF data from large scale cross-sectional surveys (DHS and MICS), a clearer image of the poor quality of complementary feeding has emerged. Dietary diversity is of particular concern – only 28% of 6-23 month children in LMICs were fed diets comprising more than four food groups (White et al., 2017). In South Asia, only 17% of children received complementary foods from animal sources including meat, fish, poultry, or eggs (Aguayo, 2017). Evidence from 39 DHS surveys highlighted the increased odds of stunting (OR 1.4; 95%CI 1.3, 1.5) among children 6-23 months who did not consume any animal

source foods compared to those who consumed meat, eggs, and dairy (Krasevec et al., 2017).

At the same time, young children's consumption of commercially produced snacks and soft drinks in LMICs (Huffman et al., 2014, Pries et al., 2017), and in Mumbai's urban informal settlements (Bentley et al., 2015), is an emerging phenomenon. Patterns of snack food consumption in the complementary feeding period and their associations with child health and longer-term risk of adult obesity are not yet well understood (Michaelsen et al., 2017).

Measuring the different components of complementary feeding, in addition to breastfeeding, over time within the same study would bring additional granularity to assessing longitudinal IYCF and growth effects.

3.5.4.2 Measurement protocol

The second part of the IYCF module asked about infants' consumption of solid or semi-solid foods in the last 24 hours (Table 3.4). The list of food items was shortened by removing foods not applicable to the local context (grubs, snails, or insects, and foods made with red palm oil) or that are universally used in cooking in India (spices and herbs) and therefore uninformative. Within each question, names of food items were adapted to include varieties and items available and consumed locally. The WHO list includes one question describing 'sugary foods such as chocolates, sweets, candies, pastries, cakes or biscuits', which was retained to incorporate information on consumption of high-sugar foods. However, three additional high-fat and high-salt items from the trial's baseline questionnaire module on IYCF were also included. These included deep-fried crisps and extruded cereal-based puffs (referred to using local names and brand names), a local street food snack of a fried potato patty eaten with a white bread roll (*vada pav*), and instant noodles (referred to using a popular brand name).

Following the WHO measurement guidelines, investigators probed respondents who reported feeding children mixed dishes by asking them about ingredients used to prepare the dish, and selecting corresponding food items from the list.

The questionnaire did not include the optional questions on fortified foods (iron-fortified food, micronutrient powders and sprinkles, or lipid-based nutrient supplements). The questions on consumption of solid and semi-solid foods were

also asked from the first follow-up visit at 1 month, in order to capture the age at which each child was first fed any solids or particular foods. Since follow-up continued until children were aged 24 to 36 months, some children's diets were measured even after they turned two and had crossed the upper age group covered by the indicators.

Table 3.4 Follow-up IYCF questions related to solid and semi-solid foods

Question	Constraints
I would like to ask you about the food (NAME) ate yesterday during the day or at night, either separately or combined with other foods. Did (NAME) eat any of the following?	(Read only)
Commercial baby food like *Cerelac* or *Farex*	Yes / No / Don't know
Porridge, bread, roti, chapatti, rice, noodles, idli, or any other foods made from grains	Yes / No / Don't know
Pumpkin, carrots, sweet potatoes that are yellow or orange inside	Yes / No / Don't know
White potatoes, white yams, cassava, or any other foods made from roots	Yes / No / Don't know
Dark green leafy vegetables	Yes / No / Don't know
Ripe mangoes, papayas, cantaloupe or jackfruit	Yes / No / Don't know
Other fruits or vegetables	Yes / No / Don't know
Liver, kidney, heart or other organ meats	Yes / No / Don't know
Chicken, duck or other birds	Yes / No / Don't know
Other meat	Yes / No / Don't know
Eggs	Yes / No / Don't know
Fresh or dried fish or shellfish	Yes / No / Don't know
Foods made from beans, peas or lentils?	Yes / No / Don't know
Nuts	Yes / No / Don't know
Cheese, yoghurt or other milk products	Yes / No / Don't know
Food made with oil, fat, ghee or butter	Yes / No / Don't know
Sugary foods such as chocolates, sweets, candies, pastries, cakes or biscuits	Yes / No / Don't know
Nalli or wafers such as Lays, Kurkure, and Pogo	Yes / No / Don't know
Vada Pav	Yes / No / Don't know
Maggi noodles	Yes / No / Don't know
Any other solid or semi-solid food	Yes / No / Don't know
How many times did (NAME) eat solid, semi-solid, or soft foods other than liquids yesterday during the day or at night? *If 7 or more times, record 7*	0-7
Notes: Adapted from WHO (2010). *Nalli (tube or pipe in Hindi) refers to deep-fried cereal based extruded salty snacks in various shapes, available to buy in branded or unbranded form.	

3.5.5 Strengths and limitations of IYCF measurement

A key strength of the WHO IYCF indicators is that food consumption can be measured with a simple tool that has been used in a large number of surveys in LMICs, enabling comparison with other populations. The tool was developed to ensure that the indicators, especially minimum dietary diversity, are able to signal higher quality diets among breastfed as well as non-breastfed children (WHO,

2008a). The IYCF questionnaire also covers a variety of food groups, including several snack foods, making it easy to understand the influence of single or multiple food groups on child health outcomes. Indicators of age-appropriate feeding practices also make it possible to examine health or growth differences between children who receive these time-sensitive feeding practices and those who do not.

Further strengths relate to repeated measurement of ICYF using a simple tool. Longitudinal assessment allows us to assess how the average diet of the cohort changes over time, the age at which certain food groups are most likely to be introduced, and which aspects of dietary quality are particularly problematic at key ages. Assessing diet in the same child allows for a detailed examination of the ways in which individual children are fed in the first two years. This means that it is possible to identify predictors of cumulative exposure to good feeding practices, and also the characteristics of children who are most likely to have consistently poor diets.

Using breastfeeding data collected prospectively from birth is also a more accurate method of calculating duration of exclusive breastfeeding in HIC and LMIC settings (Aarts et al., 2000, Agampodi et al., 2009). A recent cohort study in Nepal found that the prevalence of exclusive breastfeeding was 9% based on recall-since-birth method and 19% using the 24-hour recall method (Khanal et al., 2016).

Longitudinal assessment of snack food consumption is another strength. Previous studies have used data from cross-sectional surveys and little information is available on when snacking behaviour emerges in early life or whether habitual snack food consumption can be explained by parental, SEP or household characteristics in an urban informal settlement. This information can only be generated by repeatedly observing children in prospective studies.

However, IYCF measurement has some limitations. Subjective measures of diet are always prone to recall bias (DAPA Measurement Toolkit, 2018). The recall period in the questionnaire was 24 hours and data are unlikely to have more bias than data from a week-long recall period, but it is still a limitation. I use the previous day's food consumption as a proxy for food intake in the full age-month in which it was recorded, which would produce bias in instances where the previous day was unusual or not representative of children's habitual diets over a 28-day period.

The WHO IYCF indicators do not contain any information on the amount of food eaten since any quantity consumed is sufficient to count towards dietary quality, and it is not possible to assess a dose-response relationship between consumption of a food item and an outcome of interest at a particular age. It is nevertheless possible to assess whether greater diversity (i.e. more food groups vs fewer or none) at a particular age is associated with larger effects.

The age range for follow-up in the cohort study extended beyond two years for some children born early in the study, but the IYCF questionnaire does not capture food items that older children eat (Chinese fast food, ice cream, or meat kebabs and rolls). Some of these are likely to be introduced in the second year of life due to the influence of older siblings and adolescents in the household. In this sense, the list of food items, particularly snacks, does not comprehensively capture diet in children aged 24-36 months of age.

The WHO indicators also lead to some loss of information by dichotomising dietary data, which are inherently continuous (as calories or grams). Dichotomising leads to lower statistical power, increases the risk of biased associations, and also conceals any non-linearity that would have been identified had data been continuous (Altman and Royston, 2006).

Finally, the WHO indicators were designed for use in large cross-sectional surveys to understand population-level IYCF practices, and not to categorise individual children (WHO, 2008a). However, in a longitudinal study spanning three years of monthly data collection, using an objective measure of diet would not have been feasible. These indicators are therefore crude measures of habitual diet, but are nonetheless useful and informative due to the wide range of foods covered and repeated observation of the same cohort of children.

3.6 Infant morbidity

3.6.1 Diarrhoeal morbidity

3.6.1.1 Rationale

It is important to assess childhood morbidity in a birth cohort study, focusing on diseases and symptoms that are most relevant in a given context (Golding et al., 2009). The cohort protocol included diarrhoeal and respiratory illnesses as

secondary outcomes, but I treated diarrhoeal illness as a time-varying mediator-outcome confounder of the relationship between IYCF and growth when investigating whether IYCF mediated the association of parental anthropometry with infant growth.

Recent research from the MAL-ED cohort study has focused on pathogen-specific aetiological effects of diarrhoea on growth in low-resource settings (Rogawski et al., 2018), using appropriate molecular diagnostic methods to produce more refined insight (Platts-Mills et al., 2018). However, measuring caregiver-reported illness at each monthly follow-up would be a sufficient measure of the associations of diarrhoeal episodes with IYCF practices in the corresponding or adjacent period as well as growth across the first two years of life. Several longitudinal studies have used caregiver reports of diarrhoea in order to understand lagged, cumulative, or dose-response associations with linear growth (Moore et al., 2010, Nagata et al., 2016, Richard et al., 2014, Richard et al., 2013).

3.6.1.2 Measurement protocol

The cohort follow-up questionnaire module on infant illness and treatment collected information at each visit on symptoms and treatment of diarrhoea and respiratory illness in the past month, drawing on questions commonly used in DHS surveys. I did not use data on respiratory illness. The module on illness was administered after the IYCF module. Investigators asked about the duration of any diarrhoeal illness, and details of any treatment (Table 3.5).

Table 3.5 Follow-up questions on infant illness and treatment related to diarrhoea

Question	Constraints
Has [Name] had diarrhea in the last month?	Yes / No
How many days did the diarrhea last?	(Number of days)
Is the diarrhoea better or still going?	Better / Still going
How much was [name] given to drink during the diarrhea? Was s/he given less than usual to drink, about the same amount, or more than usual to drink?	Less / usual / more
Did you seek advice or treatment for the diarrhea from any source?	Yes / No
[If no, skip to question about ORS] If yes, where did you seek advice or treatment?	BMC health post / BMC hospital / Private practitioner / private hospital / government hospital / urban health centre
Did [name] have to stay in hospital?	Yes / No
For how many days?	(Number of days)

Has [name] been given any fluid from a special packet called ORS (local name)?	Yes / No
Has [name] been given any gruel made from rice or other grain?	Yes / No
Has [name] been given anything else to treat the diarrhea?	Yes /No
If yes, what else? (select all that apply)	Antibiotic syrup / antimotility syrup / zinc syrup / other syrup / unknown syrup / antibiotic injection / non-antibiotic injection / unknown injection/ intravenous (IV) / home remedy or herbal medicine

3.6.1.3 Strengths and limitations

Asking about diarrhoeal morbidity every month provides a time-varying measure of disease occurrence, making it possible to assess longitudinal relationships between morbidity and other factors. Using a simple questionnaire-based method ensured lower participant and investigator burdens than collecting biological samples like stool or blood repeatedly over three years of follow-up, while also minimizing the cost of collecting data frequently.

However, a month-long recall period introduces the possibility of bias in caregiver recall of duration or subjective intensity of diarrhoea. More frequent contact with participants would have reduced bias, but would have added significantly to the cost and feasibility of conducting the study. However, despite the 30-day recall, it is unlikely that occurrence of any diarrhoea would be forgotten.

Diarrhoea was measured subjectively, and participant reports were not validated by an objective measure such as direct observation in the home or laboratory testing of stool samples.

Social desirability bias is another limitation associated with using caregiver reports of diarrhoea, since parents may not wish to disclose that infants had been ill in order to demonstrate that their children received adequate care or for fear of being judged by investigators. This could lead to an underestimate of incidence of diarrhoea.

The Hawthorne effect is another bias associated with longitudinal observational studies. It is possible that repeated visits to ask about child morbidity coupled with exposure to health messages through SNEHA Centre activities increased caregiver awareness of transmission modes, leading to successful changes in their practices to prevent diarrhoea as children grew older. However, an objective measure of

diarrhoea would not have helped to prevent this bias. Blinding participants to assessment would not have been possible as direct observation at home as well as stool sample collection require participant cooperation at each measurement occasion.

3.7 Parental anthropometry

3.7.1 Height and weight

3.7.1.1 Rationale

The relationship between parental (maternal, paternal, or both) anthropometry and infant linear growth has been studied extensively. In Chapter 2, I identified 15 studies that examined the association of parental anthropometric characteristics with infant growth. Parental heights signal the genetic growth potential of offspring, and to some extent reflect the adequacy of parents' own growth and health in early life and adolescence (Wells, 2017). Parental weights, adjusted for heights, are markers of the current nutritional status of caregivers in a household, and a mother's weight partially reflects her ability to respond to the increased nutritional and health demands of pregnancy and lactation (Wells, 2018).

Measuring both parents' heights and weights is also one way to understand the differential associations of each parent's height and weight with infant health outcomes (Griffiths et al., 2007).

3.7.1.2 Measurement protocol

Investigators measured parental heights and weights on one occasion three months after the birth of the infant. An interval of three or more months postpartum was applicable only to mothers, in order to allow early post-partum weight loss to occur (Gunderson, 2009), but was applied to fathers as well to make data collection more convenient. There is no specific cut-off for when women should return to their pre-pregnancy weight and most research studies measure weight after at least 6 weeks postpartum (Gunderson and Abrams, 2000). Women who became pregnant again during the follow-up period were excluded. Participants were measured at home or the local SNEHA Centre.

One investigator measured parental weight using a portable electronic weighing scale accurate to the nearest 100 grams. Participants were asked to remove

footwear, any heavy clothing or headgear, and empty pockets before standing on the scales. Two weight readings were recorded and the average was used in analysis.

Standing height was measured using a portable Leicester stadiometer accurate to 1 mm. Investigators placed the stadiometer on a flat and firm surface and instructed the participant to stand upright against the backboard of the stadiometer with their feet together. The investigator checked that the participant's heels and back were in contact with the backboard and positioned their head in the Frankfort plane. If necessary, the investigator stood on a low stool to ensure they were able to assess the Frankfort plane at eye level. The headboard was lowered to make contact with the top of the adult's head. The investigator asked the participant to inhale and exhale and took a height measurement to the last complete millimetre at the end of expiration. Two height readings were recorded and the average was used in analysis.

The TEM of the cohort's field investigators (Appendix 3.4) was low (0.143%), indicating a low risk of biased estimates due to measurement error in parental heights.

3.8 Socio-economic position (SEP)

3.8.1 Asset-based measure of household wealth

3.8.1.1 Measurement protocol

The cohort baseline questionnaire included questions on socioeconomic status (home ownership, access to welfare services), housing structure (type of house and material of floor), ownership of a range of items, and access to basic services such as electricity, water, fuel, and sanitation.

I created a table of assets based on a subset of these questions, generating variables coded zero for responses representing the poorest or least favourable (Table 3.6). I tabulated the frequency of each item, excluding any held by less than five percent or more than 95% of households. I then converted data from 17 variables into an asset score using a Principal Components Analysis (PCA). A PCA is a data reduction technique used to generate a set of uncorrelated principal components from correlations between as many indicators. Each component has an

Eigenvalue, or a weight by which each standardized original indicator would have to be multiplied in order to obtain the component score. Eigenvalues are used to interpret each component's relevance. The first component explains the greatest proportion of variance of the indicators, and subsequent ones explain additional but a lower proportion of variance. In a PCA for an asset index, only the first component is retained, using factor scores as weights to generate a variable, with a mean of zero and SD of one, representing the household's asset score (Vyas and Kumaranayake, 2006). Higher scores imply higher household wealth. I split the asset score into quintiles, with a single variable identifying each child as belonging to the highest, second highest, middle, second lowest, or lowest wealth quintile.

Table 3.6 Baseline survey questions used to generate asset index

Question	**Response categories representing poorest households**	**Response category representing least poor households**
Socioeconomic status		
Do your family own or rent your home?	Rent	Own
What colour is your ration card?*	Orange / Yellow	White
Housing structure		
Interviewer: select the type of house the respondent lives in	Partly robust (semi-pucca) / Temporary (kaccha)	Robust (pucca)
Interviewer: select the type of flooring in the home	Dirt, sand, mud	Brick, concrete, tile
Ownership of durable assets		
Do you own any of the following household items (select all that apply): Mattress / pressure cooker / gas cylinder* / stove / chair / bed / table / clock / fan / mixer / radio* / phone / fridge / washing machine* / television / bicycle* / two-wheel vehicle* / car*	No	Yes
Access to basic services		
What type of electricity supply does your home have?	None / Family pay landlord for supply / Other	Metered electricity supply
Note: *items dropped prior to PCA due to very low (<5%) or very high (>95%) ownership.		

3.8.2 Parental education and occupation

3.8.2.1 Measurement protocol

In the baseline survey investigators asked respondents, usually the infant's mother, to report the highest school grade they (or the infant's mother) had completed, recorded as numerical responses ranging from 0 to 17. The respondent was also asked the same question with respect to the infant's father's education.

Maternal and paternal occupation were also recorded in the baseline survey. The question for each parent pertained to current livelihood, measured at or close to the infant's birth. The question had ten response categories, ranging from unskilled work to a professional or high level government job.

3.8.3 Water and sanitation

3.8.3.1 Measurement protocol

The baseline survey questionnaire included questions on the main source of drinking water for the household, with 14 response options. Two of these captured an improved source of water that was accessible in the home or yard plot, which I used as measures of adequate access to piped water on or close to premises. The survey did not capture information on whether the water was available when needed, or test water for quality and contaminants.

Questions related to sanitation focused on the type of facility, whether it was shared with other households, and the number of households that used it. I used these two variables to identify houses that used an improved toilet facility which they did not have to share with other households.

3.8.4 Household composition

3.8.4.1 Measurement protocol

After asking about whether both parents lived in the home, investigators posed five questions to respondents about household composition (Table 3.7). I used these to calculate the number of children and adults in the household.

Table 3.7 Baseline survey questions on household composition

Question	Variable name	Response constraints
How many of her / your own children under 18 years live here? (Including the index one)	ownkidsunder18	1 to 15
How many other children (not her own) live here? (Less than 18 years)	otherchildren	0 to 8
How many of her / your own children above 18 years live here?	ownkidsabove18	0 to 8
How many other men over 18 live here apart from the infant's father?	othermales	0 to 8
How many other women over 18 live here apart from you / infant's mother?	otherfemales	0 to 8

3.9 Parental health and behavioural measures

3.9.1 Pregnancy intention

3.9.1.1 Rationale

Pregnancy intention was of a priori interest as a determinant of infant growth and nutrition in Mumbai's informal settlements. DHS surveys identify intended, mistimed, or unwanted pregnancies based on a single question "At the time you became pregnant did you want to become pregnant then, did you want to wait until later, or did you want to have no (more) children at all?". This method is prone to several methodological biases, especially when used retrospectively, and does not sufficiently capture the different dimensions of pregnancy intention. The London Measure of Unplanned Pregnancy (LMUP) is a psychometrically validated multi-item tool to assess pregnancy intention on a continuous scale from 0 to 12 (Barrett et al., 2004). It has been adapted and validated for use in urban South India (Rocca et al., 2010), and covers the multi-dimensional nature of pregnancy intention by including its attitudinal, behavioural, and contextual dimensions.

3.9.1.2 Measurement protocol

The LMUP was translated into Hindi and included as a module in the baseline questionnaire, after the component on water, sanitation and hygiene, and before the module on prelacteal and breastfeeding initiation. Investigators asked the infant's mother to respond to six questions about the index pregnancy (Table 3.8). If the mother was not the main respondent at that visit, investigators returned to administer the LMUP at a convenient time when she would be available.

Table 3.8 Baseline survey questions on pregnancy intention and response scoring

Question	Response constraints	Score
Please tick the statement which most applies to you:		
In the month that I became pregnant.....	I/we were not using contraception	2
	I/we were using contraception, but not on every occasion	1
	I/we always used contraception, but knew that the method had failed (i.e. broke, moved, came off, came out, not worked etc) at least once	1
	I/we always used contraception	0
In terms of becoming a mother (first time or again), I feel that my pregnancy happened at the.....	Right time	2
	Ok, but not quite right time	1
	Wrong time	0
Just before I became pregnant.....	I intended to get pregnant	2
	My intentions kept changing	1
	I did not intend to get pregnant	0
Just before I became pregnant....	I wanted to have a baby	2
	I had mixed feelings about having a baby	1
	I did not want to have a baby	0
In the next question, we ask about your partner:		
Before I became pregnant....	My partner and I had agreed that we would like me to be pregnant	2
	My partner and I had discussed having children together, but hadn't agreed for me to get pregnant	1
	We never discussed having children together	0
Before you became pregnant, did you do anything to improve your health in preparation for pregnancy? *Please select all that apply*	Took folic acid	2 or more actions = 2 1 action = 1
	Stopped or cut down smoking	
	Stopped or cut down drinking alcohol	
	Ate more healthily	
	Sought medical/health advice	
	Took some other action (specify)	
	I did not do any of the above before my pregnancy	0

LMUP scores had already been calculated based on the LMUP scoring guide within the dataset when I received it, but I recalculated scores and verified them against the raw responses and scores. Each question is scored 0, 1, or 2. Scores for the six questions were added to produce an overall score for each woman, which was used in analysis alongside a binary variable.

3.9.2 Postnatal maternal and paternal smoking

3.9.2.1 Measurement protocol

Questions on smoking, including use of smokeless tobacco, were included in the household module of the baseline survey, as the last of a list of questions pertaining

to each parent's education, occupation, and other age-related characteristics. Respondents were asked if the father or mother currently smoked any of four types of tobacco product commonly used in India: cigarettes, *bidis* (cigarettes made with unprocessed tobacco wrapped in leaf), *gutka* (chewing tobacco), or *mishri* (tobacco-based tooth cleaning powder). Responses were coded as a single 'yes' or 'no' and the type of product used was not recorded. If the mother was present, she reported her own as well as her husband's current smoking status; if the respondent was another member of the household, they reported both maternal and paternal smoking status.

3.10 Additional variables

The cohort study protocol also specified several other variables which I did not use in my analysis. I present summary statistics for some of them in Chapter 5 in order to provide more detailed contextual information about the participants (Table 3.9).

Table 3.9 Cohort study variables summarised but not used for analysis

Concept	Method or tool	Timing of assessment
Maternal postnatal depression	Edinburgh Postnatal Depression Scale	Baseline questionnaire
Low birth weight	Institutional birth weight record	Baseline questionnaire
Maternal antenatal care and delivery	Structured interview	Baseline questionnaire
Maternal birth history	Structured interview	Baseline questionnaire
Household disposal of young children's faecal matter	Structured interview	Baseline questionnaire
Infant care, care-seeking practices and use of welfare services	Structured interview	Monthly follow-up questionnaire

The study also included serial measurement of four other anthropometric markers of infant growth: weight, mid-upper arm circumference (MUAC), head circumference, and abdominal circumference. Heights and weights of up to three siblings under five years were recorded once during the course of the study, including any younger siblings born after the index infant. These measurements are outside the scope of the thesis.

Further, I did not use low birth weight data as a study variable for a number of reasons. First, there is no particular justification for 2500 g as a cut-off for elevated infant mortality risk, and LBW can be affected by factors that are not related to

mortality risk (Wilcox, 2001). Second, preterm birth is a strong determinant of LBW in a population, and the small preterm births are usually at highest risk of mortality. The cohort excluded preterm infants, among whom birthweight is a marker of foetal growth because gestational age no longer has a clinically important effect on birthweight. However, the cohort did not include any prenatal exposures in order to study their association with birthweight. Third, any relationship between birthweight and infant linear growth would be contaminated if length at or close to birth was also included in growth data, because birth weight also encompasses length. Fourth, birthweight data are prone to digit heaping, especially close to the LBW cut-off, often making the data unreliable in analyses.

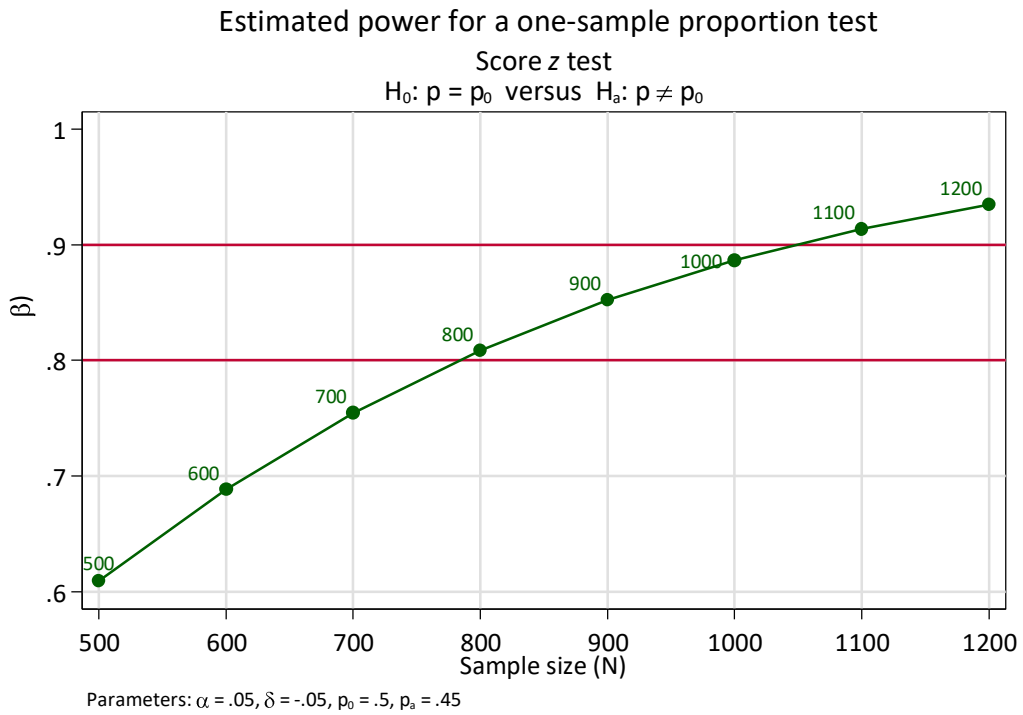
3.11 Study size

Birth cohort studies vary vastly in size and may have more than one outcome of interest. Studies that examine rare outcomes require larger numbers of participants than those that investigate more common conditions. Sample size and power calculations are also determined by whether outcomes, exposures, covariates and environmental variables are continuous, dichotomous or categorical. In addition to being powered to address the main research hypothesis, cohort studies must also have sufficient numbers for ancillary investigations. Practical considerations such as funding constraints, feasibility of data collection and follow-up in a given population, and expected attrition also influence final cohort size (Golding and Steer, 2009).

The main outcome of interest during the early stages of study planning was stunting (height-for-age z-score below -2 SD of the WHO Growth Standards), with the objective of estimating the proportion of stunted children in the cohort with a certain level of precision. Previous research in Mumbai's informal settlements found that 47% of children below 5 were stunted (Das et al., 2012). The number of births expected annually was 1000, of whom 20% would be lost to follow-up.

These two parameters were used to calculate required sample size given an expected stunting prevalence of 45% within a two-sided significance level (α) of 5%, and ascertain the statistical power of the study to detect this prevalence based on 1000 expected births. Different scenarios can be visualised in a power analysis using the Stata command **power**, comparing the estimated power of study sizes ranging from 500 to 1200 participants to detect a prevalence of 45% (p_a) against a null hypothesis (p_0) of 50% (Figure 3.4).

Figure 3.4 Sample size and statistical power scenarios



Note: Stata code for the figure

power oneproportion 0.5 0.45, n(500 (100) 1200) graph(yline(0.8 0.9) plotopts(mlabel(N)))

power oneproportion 0.5 0.45, power (0.8 0.9)

A cohort of at least 783 infants would be required to have 80% power, or 1047 infants for a study with 90% power. A much larger study of 1200 infants would lead to little additional power while adding significantly to the cost and feasibility of conducting the study.

The target cohort size was thus set at 1000, assuming that a 20% loss to follow up would still allow the study to have enough statistical power and a desired sample size of 800.

3.12 Limitations of study design

While the longitudinal element of the cohort brings several advantages, there are two limitations that should be noted.

First, attrition is a very real possibility in longitudinal studies. Urban informal settlements can have very high turnover, and families with young children might be

more likely to move out if they have insecure residential status. Depending on the specific hypothesis and age group of interest, it is possible that substantial loss to follow-up, beyond that accounted for in the target cohort size, could make it difficult to conduct valid statistical analyses if insufficient numbers remain in the study for the full duration. Since my data are drawn from a birth cohort in a closed population born in a defined period in a particular setting, there is no way to 'replenish' or 'boost' the study size by recruiting additional participants with similar characteristics if they move in to the study area. For example, this cohort would exclude infants whose mothers usually lived in the study area in a permanent home but spent six months post-partum living in their natal home in another part of the country.

Second, the cohort is nested in a trial that directly addressed an anthropometric indicator of child growth as a primary outcome. While my research addresses linear growth whereas the trial targeted weight-for-height, this is a nevertheless a study limitation because length is closely associated with weight. Study participants actively received information or services that could influence children's linear growth in the first two years of life. One anticipated impact of cohort children's participation in the trial activities and their success in achieving study objectives is that the overall incidence of growth faltering and acute malnutrition would be lower than if the children had not received any intervention (for example, among children born in the trial's control arm). The magnitude of association between exposures and linear growth outcomes in my research would be biased downwards. However, since the findings of the trial in relation to the primary anthropometric outcome were ambivalent, I feel that using data from a cohort restricted to the trial's intervention arm would not have a major impact on my findings.

Chapter 4 Study variables

Summary

In this chapter I describe the methods used to derive variables used in my study. I detail how I used and interpreted the cohort data and measures, and discuss the strengths and limitations of the variables or specifications I used. I also discuss some key statistical considerations that have shaped my overall analytic approach.

4.1 Introduction

Birth cohort studies should ideally aim to measure a broad range of health outcomes pertaining to the parents (e.g., body mass index or postnatal depression), the pregnancy outcome (e.g., birthweight or placental characteristics), and childhood anthropometry, morbidity, cognitive health and other developmental outcomes in early life. Outcomes should be selected after considering topical local health issues, emerging problems that could be of interest in a few years' time, and the relevance of outcomes commonly collected in other longitudinal birth cohorts (Golding, 2009). Variables for this cohort were selected for their relevance to the aims of the study, as described in the protocol, and based on practical considerations of feasibility and contextual suitability.

A .do file describing variable coding is in Appendix 4.1.

4.2 Length and height measurements (0-37 months)

Length measurements are often combined with age and sex to produce indices and reported in relation to growth references and standards. In the thesis, however, I used length measured in centimetres accurate to the nearest millimetre in raw, unstandardized format (i.e., I did not convert values to z-scores). The date of measurement was used to calculate the individual's age – in days, months (integer and continuous), and years – at each measurement occasion by subtracting the individual's date of birth. For index children who turned two during the follow-up period, standing height was measured subsequently, which I then converted to length. I did this by adding 0.73 cm to height measurements after 24 months, based on the methods described by the WHO MGRS methods manual (WHO, 2006).

I used length and age measurements to model infant growth trajectories over the follow-up period. Serial or longitudinal measurements on the same children over a particular period also allow for the calculation of growth velocity of length gain (WHO Working Group, 1986). I used the SITAR (SuperImposition by Translation and Rotation) method (Cole et al., 2010) to model infant growth (see Chapter 6).

4.3 Prelacteal and initiation of breastfeeding

4.3.1.1 Use and interpretation

I used data on initiation of breastfeeding to calculate the mean number of hours or days after which infants were first put to the breast. I also calculated the proportion of infants who received breastmilk within one hour of birth. I described proportions of infants given pre-lacteal feeds, and the types most commonly used in this population.

I created additional variables for exclusive and predominant breastfeeding ignoring any pre-lacteal feeding. This contradicts WHO definitions of these practices, but would enable identification of infants whose mothers go on to exclusively or predominantly breastfeed for several months despite pre-lacteal feeding, as is the case in many LMICs (Greiner, 2014). Even though pre-lacteal feeding may prevent breastfeeding from being established, expose infants to early infection through contaminated herbs or utensils, and reduce the immunological benefits of colostrum as the first feed (Debes et al., 2013), it would not inhibit the benefits of subsequent exclusive breastfeeding on infant health in informal settlements.

4.3.1.2 Strengths and limitations

Incorporating data on pre-lacteal feeding into assessment of breastfeeding duration enables greater flexibility in using IYCF indicators to meet study objectives. Since pre-lacteal feeding was assessed before information on breastfeeding practices was collected, the relationship between pre-lacteal feeding and duration of breastfeeding would not be subject to reverse causality.

Assessment of pre-lacteal feeding and initiation of breastfeeding is prone to social desirability bias, and recall bias may be higher among participants whose baseline interviews took place several days or weeks after birth.

4.4 Breastfeeding

I calculated several indicators of breastfeeding practices by classifying every child's data at each measurement occasion as a binary indicator of whether or not they had achieved that indicator, and used the data to quantify population-level prevalence at each age and across age groups (Table 4.1). Since IYCF was measured repeatedly, I applied the criteria for the exclusive breastfeeding (EBF) indicator more stringently. On each measurement occasion, infants who met WHO criteria for EBF were only categorised as such if they had met the criteria on all previous measurement occasions. In this way, I attempted to use repeated point-in-time data to approximate life-long data (Greiner, 2014).

I also used these repeated binary data to describe breastfeeding patterns of individuals to calculate the duration of exclusive breastfeeding or predominant breastfeeding, and the age up to which breastfeeding continued for each individual child. I used each measurement as a proxy for the breastfeeding practice for the full month in which it was collected in the absence of data based on a longer duration of recall. I also used data on each type of non-breastmilk liquid listed in the questionnaire to understand group and individual-level patterns of consumption.

Interpretation of breastfeeding data for specific analyses is reported in Chapter 7.

Table 4.1 WHO population-level breastfeeding indicators: definitions and age groups

Indicator	Definition	Age group	Further disaggregation
Exclusive breastfeeding under 6 months	Proportion of infants 0-5 months of age who are fed exclusively with breastmilk	0-5 months	0-1 months, 2-3 months, 4-5 months, 0-3 months
Predominant breastfeeding under 6 months	Proportion of infants 0-5 months of age who are predominantly breastfed	0-5 months	0-1 months, 2-3 months, 4-5 months, 0-3 months
Continued breastfeeding at 1 year	Proportion of children 12-15 months of age who are fed breastmilk	12-15 months	12, 13, 14, and 15 months
Continued breastfeeding at 2 years	Proportion of children 20-23 months of age who are fed breastmilk	20-23 months	20, 21, 22, 23 months

Source: (WHO, 2008a)

4.5 Complementary feeding

Following the WHO definition, any amount of food from a group was deemed sufficient to 'count' towards a complementary feeding indicator. As with breastfeeding, I used data on complementary feeding to generate group as well as individual-level summaries. However, I selected some indicators from the WHO core list and created additional ones using different criteria (Table 4.2). I included Minimum Dietary Diversity (MDD) and timely introduction of solid, semi-solid, and soft foods using WHO definitions. MDD is achieved when children receive food from at least four of seven food groups. These include grains, roots and tubers, legumes and nuts, dairy products (milk, yogurt), flesh foods (meat, fish, poultry and organ meat), eggs, vitamin-A rich fruits and vegetables, and any other fruits and vegetables. Using these 7 groups, I also created an indicator of any fruit and vegetable consumption in the last 24 hours (combining any and vitamin-A fruit and vegetables groups), and another describing consumption of animal source foods by combining data on dairy, eggs, and flesh foods (Krasevec et al., 2017). I did not count breastmilk as a food group as these indicators are intended to evaluate quality of complementary feeding (WHO, 2008a).

Table 4.2 Population-level complementary feeding indicators

Indicator	Definition	Age group	Further disaggregation
Timely introduction of solid, semi-solid and soft foods	Infants fed solid, semi-solid or soft foods	6-8 months	6, 7, 8 months
Minimum dietary diversity	Infants fed foods from 4 or more food groups	6-23 months	Every month across 6-23 months, 6-11 months, 12-17 months, and 18-23 months
Consumption of fruit and vegetables	Infants fed any fruit or vegetables		
Consumption of animal source foods	Infants fed any animal source foods (dairy, eggs, or flesh foods)		

Source: (Krasevec et al., 2017, WHO, 2008a, WHO, 2010).

Indicators of snack food consumption were derived from both components of the IYCF module to quantify consumption of any snacks, sugar-sweetened beverages, sugary snacks, salted snacks, and hot-cooked snacks (Table 4.3). I included tea and coffee in the sugar sweetened beverages category because they are normally made with added milk and sugar, and excluded yogurt-based drinks as sweet and salty versions were combined within the same question. I calculated these indicators for individual children at all ages covered in the study.

Interpretation of complementary feeding data for specific analyses is reported in Chapter 7.

Table 4.3 Classification of snack food and drink consumption.

Category	Items consumed in the last 24 hours
Sugar sweetened beverages	Tea, coffee, or cold drinks (Pepsi, Coke, Frooti)
Sugary snacks	Sugary foods such as chocolates, sweets, candies, pastries, cakes or biscuits
Salted snacks	*Nalli* or wafers such as Lays, Kurkure, and Pogo
Hot-cooked snacks	*Vada pav* or Maggi noodles
Any snacks	Any sugar sweetened beverages, sugary, salty, or hot-cooked snacks

4.6 Diarrhoea

I used information on the occurrence, duration, and treatment of diarrhoeal episodes to understand patterns of morbidity in the cohort.

For mediation analysis, I did not discriminate between episodes of longer duration or type of treatment, using a binary variable at each measurement occasion to indicate whether or not the child had suffered any diarrhoea in that month. I subsequently collapsed data in 3-monthly intervals from birth to 24 months, creating two variables for each interval. A binary variable indicated whether or not the child had experienced any diarrhoea in the interval, and a categorical variable indicated the number of months in which the child had experienced diarrhoea (none, one, two, or all three months).

4.7 Parental anthropometry

4.7.1.1 Use and interpretation

I used parental height and weight measurements in several ways (Table 4.4), calculating indices and z-scores, applying cut-offs where applicable, and creating variables to group participants based on these categorizations.

I summarised raw height and weight measurements to understand the population distribution of parental size, and calculated the proportion of women who were shorter than 145 cm to identify those were at elevated risk of poor pregnancy and health outcomes (Ozaltin et al., 2010, Subramanian et al., 2009).

I calculated Body Mass Index (BMI) by dividing weight in kilograms by the square of height in metres, and used cut-offs for Asian populations (WHO Expert Consultation, 2004) to categorize underweight (BMI ≤18.5), overweight (BMI ≥23.5), and obesity (BMI≥27.5). These cut-offs are lower than international cut-offs (WHO, 2000) for overweight (BMI ≥25) and obesity (BMI ≥30), but I selected these because they identify those at elevated health risk. Asian populations are at increased risk of metabolic diseases such as diabetes and hypertension at lower BMI values than those observed in non-Asian populations (WHO Expert Consultation, 2004).

Table 4.4 Indicators derived from parental height and weight measurements

Measurement and unit	Parent	Indicator and summary
Raw values		
Height (cm)	Mother, father	Height: Mean, SD Low maternal height <145 cm (%)
Weight (kg)	Mother, father	Weight: Mean, SD
Height (cm) and weight (kg)	Mother, father	BMI (kg/m^2): Mean, SD Underweight (BMI ≤ 18.5) (%) Normal (BMI between 18.5 and 23.5) (%) Overweight (BMI ≥ 23.5) (%) Obese (BMI ≥ 27.5) (%)
Height (cm) and weight (kg)	Both parents	Parental overweight (BMI ≥23.5) category (1) Neither parent overweight (2) Mother overweight (3) Father overweight (4) Both parents overweight
Internal z-scores		
Height (internal z-score)	Mother, father	Height z-score
Weight (internal z-score)	Mother, father	Weight z-score
Height (internal z-score)	Both parents	Sum of parental height z-scores Half difference of parental height z-scores
Weight (internal z-score)	Both parents	Sum of parental weight z-scores Half difference of parental weight z-scores

Note: BMI, Body Mass Index; SD, Standard Deviation

Using BMI, I categorized parents based on overweight status (BMI ≥23.5), creating four mutually-exclusive groups describing each couple's discordance with respect to BMI category. This variable was used to understand whether the influence of parental overweight on infant growth depended on whether either or both parents were categorized as overweight compared to when both parents had BMI values below 23.5. Dichotomizing continuous variables leads to loss of information, but I wanted to understand the relationship between parental size using a measure that is of public health significance and easy to interpret.

I also used a parameterization suggested by Griffiths et al. (2007) to understand the differential contributions of parental anthropometry to child anthropometry in

regression models. I converted maternal and paternal heights and weights to sex-specific internal z-scores by subtracting the mean and dividing by the SD. I used these as four separate variables, but also constructed two summary variables for each anthropometric measurement. I calculated the half difference in maternal and paternal height coefficients (maternal height z-score – paternal height z-score)/2, and the sum of maternal and paternal height z-scores. I repeated this for weight z-scores. I subsequently used these in pairs (of difference z-scores and sum z-scores) in growth models. I interpreted a positive difference z-score coefficient as greater maternal influence, and the sum of z-scores coefficients as the mean parental contribution, with height and weight adjusted for each other in both analyses. This parameterization has the advantage of producing standard errors for the difference or mean values that would otherwise be calculated manually from models where maternal and paternal values were specified separately.

4.7.1.2 Strengths and limitations

A key strength of parental anthropometric assessment is that both parents were included, providing additional information on the genetic and environmental factors that influence infant growth outcomes. Height and weight are highly informative measurements, amenable to statistical analysis in a number of ways that provide insight on different dimensions of health and nutritional status. Anthropometry was measured by trained investigators in this cohort, providing more reliable data than self-reported measurements.

Using several parameterizations of height and weight also facilitates sensitivity analyses to test whether associations observed using continuous variables hold when cut-offs are applied to the data.

However, BMI is a crude measure of adiposity, and I was unable to ascertain the distribution of body fat based only on height and weight. In reality, the distribution of fat in the abdomen and around internal organs (visceral fat), is more metabolically relevant as a marker of elevated risk of disease than BMI. However, more recent research suggests that BMI is strongly correlated with abdominal adiposity and suitable for detecting raised cardiometabolic risk (Bell et al., 2018).

Measuring postpartum weight in women has its drawbacks because I was unable to account for the relative influence of three crucial determinants of postpartum weight retention: parity, pre-gravid weight and gestational weight gain (Gunderson and

Abrams, 2000, Hollis et al., 2017). Excessive gestational weight gain is associated with short- and long-term postpartum weight retention (Mannan et al., 2013). Further, the rate of postpartum weight loss exhibits high inter-individual variability. Data on pre-pregnancy maternal BMI would have enabled an investigation of the effect of higher postpartum weight retention or incident obesity following pregnancy on infant growth patterns (Gunderson, 2009).

4.8 Socioeconomic position (SEP)

I employ socio-economic position (SEP) as a concept describing the social and economic factors that are associated with the position of individuals or groups within a society. SEP encompasses two aspects: actual resources and rank-related characteristics (Krieger et al., 1997). No single indicator comprehensively captures all dimensions of SEP or its effects on health outcomes and the choice of indicators is best determined by the research question. When SEP is conceptualized as a potential confounder, using multiple measures becomes important in order to minimize residual confounding in observational epidemiological studies. SEP can be measured at different times across the life course, including childhood, young adulthood, active professional or working age life, and retirement (Galobardes et al., 2006a). The timing of measurement and its interpretation is often determined by hypothesized causal pathways (Krieger et al.,1997).

Based on my research objectives, I use measures of SEP as exposures of interest for infant growth and IYCF outcomes, but also as baseline confounders of the relationship between parental anthropometry and infant growth.

The most commonly used indicators of SEP in high-income countries are based on education, housing, income, occupation, wealth, labour market participation, and proxy measures such as number of siblings or family characteristics. Composite indicators of multiple SEP measures as well as area-level measures of deprivation are also used (Galobardes et al., 2006b, Galobardes et al., 2006a).

Epidemiologic investigations in LMIC settings use contextually relevant measures of SEP, many of which are conceptually similar to those used in HICs. The most prominent ones include objective measures of household assets to describe wealth and material living standards, consumption expenditure, education, income, occupation, and participatory wealth ranking. Additional subjective measures of

these factors are sometimes employed to allow participants to report perceived SEP in relation to income or other factors (Howe et al., 2012a). More recently in the MAL-ED multi-country cohort study, researchers developed a novel composite indicator of socioeconomic status called the WAMI index (Water and sanitation, Assets, Maternal education, and household Income) for use in resource limited settings (Psaki et al., 2014).

Based on my literature review (Chapter 2) and methodological considerations I focus on five markers of SEP from variables included in the original cohort protocol: household asset-based wealth, parental education, occupation, WASH, and household composition. All were measured at baseline, and represent the conditions into which infants were born, or early childhood SEP. These also assess the accumulated resources and current circumstances of parents, capturing their working age SEP at one time point. I used these to understand how absolute material conditions as well as the relative position of individuals and households within the study site are associated with health outcomes.

I did not use any area-level measures of deprivation. The study sites were selected because they were at higher risk of poor maternal and child health outcomes among the city's informal settlements (Osrin et al., 2011, Shah More et al., 2017).

4.8.1 Asset based measures of household wealth

4.8.1.1 Rationale

The asset or wealth index approach to generating an indicator of household material living standards in the absence of data on consumer expenditure and income is used widely in LMIC settings. The approach was first developed using DHS survey data on ownership of a range of durable assets, housing characteristics, and access to basic services, all of which potentially affect health directly (Filmer and Pritchett, 2001). It has been used in previous work on health in Mumbai's informal settlements, including the SNEHA Centres pre- and post-intervention surveys (Shah More et al., 2017, Shah More et al., 2013).

4.8.1.2 Use and interpretation

I used the asset index quintiles primarily to assess socio-economic gradients in health outcomes. Since it is measured at household level, I interpreted it as an

indicator of the SEP to which infants were exposed in the home environment, and of aspects of SEP which were shared by both parents.

The asset index's composite specification encapsulates relative as well as absolute measures of SEP in informal settlements. It separates families into an assumed hierarchy of home owners and renters, and those with access to welfare services within the study population. It also describes the absolute material conditions of housing structure and ownership of consumer durables. With respect to access to basic services, while questions are directed towards individual households, the supply of basic services is often pre-determined by municipal governance or local conditions for entire informal settlements. In this way, I interpret the asset score as indicative of SEP across multiple domains relevant to health.

I did not use the asset quintile and additional water and sanitation variables in the same multivariable model, since some contribution of water and sanitation to SEP is already measured in the asset quintile. I did, however, use asset quintile alongside individual, parent-specific markers of SEP such as education and occupation, as well as household-level indicators of family composition in the same model.

4.8.1.3 Strengths and limitations

The asset index approach is a simple and quick method for assessing SEP in population surveys. In LMICs, it can be a more stable measure than consumer expenditure, which can vary markedly with fluctuations in income or price shocks (Howe et al., 2012a). It also provides a way to examine the effects of relative wealth or social stratification on health, complementing approaches that focus on the effects of absolute, categorical measures of SEP (Krieger et al., 1997).

However, it also has some drawbacks. The asset index does not indicate the quality of assets or basic services, which may be important determinants of health (Falkingham and Namazie, 2002). Despite being an 'asset' index, it measures items that are provided at the neighbourhood level if these are included in the PCA, such as piped water supply or a functioning sewage system to facilitate installation of toilets in the home. In some studies, the asset index can therefore be correlated more with local infrastructure than household-level consumer expenditure (Howe et al., 2011). Finally, it also masks any mechanistic effects of SEP factors, such as the effect of poor sanitation on health that may operate through increased risk of infectious diseases (Howe et al., 2012a).

4.8.2 Parental education and occupation

4.8.2.1 Rationale

Education is an important marker of SEP in LMICs due to its effect on an individual's occupational and economic status, social mobility, as well as health related behaviours (Buchmann and Hannum, 2001). To some extent, the completion or attainment of formal education by adolescence is an indicator of adults' SEP in early life (Galobardes et al., 2006a).

Occupation reflects an individual's social position related to type of work and their potential contribution to household income, as well as their intellectual abilities and skills. It also reflects societal norms around women's participation in economic activity (Galobardes et al., 2006a). Informal employment is common in LMIC settings and may vary widely in the scale of economic activity. Occupational status of the main income earner also signals the SEP of the entire household (Howe et al., 2012a).

4.8.2.2 Use and interpretation

I used maternal and paternal educational achievement as binary variables to measure whether parents had completed a basic level of formal education to be able to read and write. I interpreted it as the summary effect of studying up to at least primary level, rather than the incremental effects of each grade of formal schooling as implied by a continuous measure. I also interpreted these as individual level SEP which, though likely correlated within a couple, signalled each parent's independent knowledge, literacy, and ability to influence their own and their child's health.

I split paternal occupation responses into three categories, unskilled or low skilled work, skilled work, and formal clerical or professional work (Table 4.5). I used maternal occupation to categorize maternal employment status only, as I expected that a large number of women would not be economically active at the time of the infant's birth.

Table 4.5 Paternal occupation groups based on baseline survey responses

Occupation
Unskilled or low skilled work
Does not work
Unskilled work, like *pheriwalla*, domestic servant, watchman, labourer
Plant or machine operator or assembler, or driver
Skilled work
Skilled craftsperson like potter, tailor, plumber, electrician, jewellery maker
Agriculture or fishery worker
Formal clerical or professional work
Service worker, shop or market sales worker, caterer, bus conductor
Clerk in an office, computer operator, typist
Technician (KG or primary school teacher, nurse)
Professional (Doctor, lawyer, engineer, school or college teacher, *pandit*, *moulvi*)
High level government job (Legislator, senior official or manager, local corporator)

4.8.2.3 Strengths and limitations

Education and occupation are simple measures for use in surveys. Educational attainment is a relevant factor for people at any age, unlike other indicators such as income which may be more important at certain ages. However, measuring the number of grades of education does not indicate the quality of education or the resources to use one's knowledge to influence health. Measuring occupation is prone to misclassification in this setting where men may move across categories frequently, or may work in more than one type of job.

4.8.3 Water and sanitation

4.8.3.1 Rationale

While the availability of water piped into the home and use of a flush toilet are part of the asset index variable, they measure technological aspects of water and sanitation relevant for health. A further level of classification is based on service level criteria, recently employed in the WHO / UNICEF Joint Monitoring Programme for Water Supply and Sanitation in the context of the Sustainable Development Goals. These indicators are normative interpretations of data, with additional criteria to account for water as a human right and sanitation services that should be safely managed (WHO / UNICEF, 2018).

Water from an improved source (piped, tap stand or tubewell) must be accessible in the dwelling, yard or plot, and take no more than 30 minutes to collect, be available when needed, and must be of adequate quality free from contaminants (WHO / UNICEF, 2017). In order to be safely managed, households should use an improved facility (flush or pour flush latrine connected to piped sewers or septic tanks, or ventilated improved pit latrines) that is not shared with other households, and from which excreta are removed in ways that prevent human contact at the household and community levels (WHO / UNICEF, 2018).

4.8.3.2 Use and interpretation

I used the water and sanitation variables as binary exposure variables to understand their relationship with infant growth and feeding outcomes in uni- and multi-variable analyses. I also used them as confounders in analyses with parental anthropometry as the main exposure variable.

I interpreted water piped to the home or yard plot as indicative of ease of access to a municipal supply and greater stability over time, without the need for household members to make trips to fetch water from a public tap or well outside the area or wait for a private tanker to deliver water (which would be from an unimproved source).

I interpret use of a shared toilet, whether a privately-owned one shared with neighbouring households or a public toilet in the cluster, to mean that these households had more restricted access and less privacy than households that did not share a toilet, and that shared toilets were possibly less hygienic than private ones due to greater use.

4.8.3.3 Strengths and limitations

This specification of water and sanitation allows some examination of the quality of services associated with access. It is possible to use these indicators to assess whether the lack of adequate access to an improved source, rather than merely whether or not the source is adequate, has implications for health and nutrition in informal settlements. As a marker of SEP, adequate access differentiates families with poor access from those with more secure access.

Since the indicators did not include information on other normative criteria, they do not comprehensively capture the quality of water and sanitation facilities, especially availability of water when needed and safety of water and waste management.

4.8.4 Household composition

4.8.4.1 Rationale

Number of siblings is used as a proxy variable for poor SEP in settings where there is greater risk of infection, or the number of siblings represents one of the mechanisms through which family size affects health outcomes (Galobardes et al., 2006b). In my literature review I identified five studies that examined the effects of household composition on infant growth (Zhang et al., 2017, Matos et al., 2017, Nagata et al., 2016, Bhargava, 2016, Kang Sim et al., 2012). All, including one in informal settlements (Zhang et al., 2017), found that poor linear growth outcomes were associated with larger household size or the number of children in the home. However, siblings or other children in the household can be a source of benefit or harm. Older children are sometimes a source of childcare and economic contribution, but may also expose younger children to greater risk of infection in early life, when they are more vulnerable to growth faltering (Kramer et al., 2016). Similarly, adult household members other than the child's parents, such as older siblings aged over 18, grandparents, aunts, and uncles, may or may not contribute to the household economy and childcare. It is important to examine the effects of number of children and adults in the household separately in order to determine their differential relationships with infant growth and nutrition.

4.8.4.2 Use and interpretation

I created a continuous variable for the total number of children in the household (ownkidsunder18 + otherchildren), and another one for the total number of adults (ownkidsabove18 + othermales + otherfemales). I also dichotomised both variables at the median value.

I used the continuous and binary variables describing numbers of children and adults in the household as separate exposure variables in uni- and multivariable analyses to evaluate their influence on infant growth and nutrition, and as confounders in analyses using parental anthropometry as the main exposure for infant growth outcomes.

When using continuous variables, I interpreted any association as the effect of each additional child or adult in the household on infant outcomes. For binary variables dichotomized at the median value, I interpreted any association as the difference in growth or nutrition outcomes between those from more and less crowded households. Since the data were collected at baseline, very close to birth, the effect of children in the household only captured the effect of older children.

4.8.4.3 Strengths and limitations

Both adults and older children may contribute to childcare or economic activities, and so evaluating their effects separately helps disentangle the contribution of younger and older members of a household. Further, in multivariable analyses in which both adult and child variables are included, it is possible to see their mutually adjusted effects, signalling any potential compensation by one generation in households with fewer members of the other. By excluding parents, whose contribution is captured in other SEP measures, these variables are proxies for the household circumstances that parents and children are both exposed to. These were assessed at baseline. Household composition in informal settlements can vary by month or season, and the effect of the additional resources or constraints attributable to household size could change frequently. In addition, the effect of any younger sibling or child born into the house after the index infant's birth cannot be assessed. Younger children may compete for maternal or household resources, especially in the complementary feeding period (Kramer et al., 2016).

4.9 Parental health and behavioural measures

4.9.1 Pregnancy intention

4.9.1.1 Use and interpretation

I used the London Measure of Unplanned Pregnancy data as continuous and binary variables. I created a binary variable using a cut-off between nine and ten for unplanned / planned pregnancy (Hall et al., 2017a). Use of the full range of scores is recommended (Barrett et al., 2004), and increasing scores indicate increasing degrees of pregnancy planning or intention. In multivariable analyses using LMUP as a continuous covariate I interpreted any association as the effect of greater pregnancy intendedness on infant and child outcomes. When used as a binary variable, I interpreted the association as the difference in outcome between children born as a result of planned and unplanned pregnancies.

4.9.1.2 Strengths and limitations

Using the LMUP presents a methodological advantage over more crude methods of assessment, enabling more nuanced examination of the role that pregnancy intention plays in determining child nutrition and health outcomes within a wider web of factors. The use of continuous as well as binary LMUP variables provided greater statistical flexibility.

However, assessment of pregnancy intention is most reliable when measured during pregnancy (Hall et al., 2017b). In this study, it was measured soon after the birth of the infant, and it is possible that pregnancy outcome, for example sex or birthweight of the infant, could influence women's perception of its intendedness. However, since growth and nutrition outcomes were unlikely to have already occurred when pregnancy intention was assessed, its direct effect unrelated to pregnancy outcome (e.g. sex) is unlikely to be prone to reverse causality. A further limitation of using the LMUP, administered after women gave birth to live singleton babies, is that the prevalence of unplanned pregnancies in the cohort is unlikely to be generalizable to urban informal settlements as a whole. It would be an underestimate of the true prevalence since it does not capture pregnancies (planned or unplanned) that ended in abortion or miscarriage.

4.9.2 Postnatal maternal and paternal smoking

4.9.2.1 Rationale

The studies in my literature review that included parental smoking were set in The Netherlands (Durmuş et al., 2011) and Brazil (Martínez-Mesa et al., 2012, Matijasevich et al., 2011). Self-reported measures of maternal and partner smoking, and the number of cigarettes smoked per day, were used to assess exposure to tobacco in pregnancy. However, use of tobacco products in India is not limited to conventional cigarettes. Many people use locally made cigarettes, chewing tobacco, and other smokeless tobacco products instead of or in addition to cigarettes (Mishra et al., 2016). Assessing current smoking status of both parents in pregnancy is a way to use fathers as negative controls for maternal intrauterine exposures in epidemiological investigations (Davey Smith, 2012). Parents who smoke during pregnancy are likely to continue to smoke postnatally; those who quit or cut down may relapse. Postnatal smoking in the Indian context can also serve as a marker of a socioeconomically patterned health behaviour among men (Bhan et al., 2016, Mishra et al., 2016).

4.9.2.2 Use and interpretation

I used data on maternal and paternal smoking as binary variables to describe the prevalence of smoking in the cohort, and as confounder variables in multivariable analyses. In the absence of disaggregated information on type of tobacco product used, I could not interpret parental smoking as a valid measure of infants' exposure to environmental tobacco smoke. I interpreted parental smoking as possibly correlated indicators of health behaviours which would influence their own and their infant's health.

4.9.2.3 Strengths and limitations

A key strength of smoking assessment in this study is that data on both parents were collected in the baseline survey and the question captured a range of tobacco products. However, the data are subject to several caveats.

Asking participants or proxy-respondents about current smoking status is a subjective and crude method of assessment. The questionnaire did not collect information on type of tobacco product, frequency of use, or number of years since the parent began smoking, making it hard to further investigate dose-response patterns of any observed effect of smoking status. These data are also prone to recall and social desirability biases (Florescu et al., 2009). Data on tobacco products that expose adults and children to first- or second-hand environmental tobacco smoke were combined with information on smokeless products from the same question, preventing any assessment of their differential effects on infant outcomes.

Since maternal smoking was assessed soon after women gave birth, it is possible that some women who usually smoked would have stopped for the duration of the pregnancy and not yet resumed smoking if intending to. Smoking in pregnancy or during lactation is also viewed as socially unacceptable, making maternal smoking data more prone to social desirability bias than paternal smoking. These data are therefore likely to underestimate the true extent of (postnatal) maternal smoking in this cohort. Further, there is no information on smoking during pregnancy, precluding analysis of the relationship between intrauterine exposure to maternal smoking and postnatal growth.

4.10 Two key statistical considerations

While the statistical methods for multivariable analyses I use vary across questions, I have attempted to follow common principles in two key areas: selection of confounders in multivariable regression (without mediation or time-varying exposures), and investigation of interaction or effect modification. These are discussed below as general topics of relevance to the whole thesis.

Additional statistical considerations including investigating and handling missing data, dealing with loss to follow-up, and bias and sensitivity analysis to assess validity of findings are addressed for specific research questions in subsequent chapters.

4.10.1 Selection of confounders

I relied on my systematic review, contextual information and knowledge of urban informal settlements, and *a priori* research interests to select variables for inclusion in my thesis. However, the number of variables I identified from the cohort study was still large, raising concerns about multicollinearity and data sparsity in multivariable analyses (Greenland et al., 2016b).

Predictive analyses attempt to find a model that predicts observed data well using a small number of variables. Change-in-estimate approaches to selecting variables attempt to control most or all confounding with a small number of variables (Greenland et al., 2016a).

The goal of my analysis of the relationship of baseline exposures with infant growth and nutrition (without mediation) was to produce valid and precise associations based on available data and statistical methods (Greenland et al., 2016a, Greenland and Pearce, 2015).

In subsequent chapters, I first conducted univariable analysis to quantify the relationship between a baseline factor and outcome, identifying those with a p-value below 0.1 for use in a reduced multivariable model. I also used the full set of baseline factors in a multivariable analysis. I presented both reduced and full models for all analyses.

4.10.2 Interaction, effect modification and sub-group analyses

Interaction is the effect of two exposures together on an outcome, and effect modification is the effect of one exposure within strata of another exposure (VanderWeele, 2009).

Infant sex was the only variable which was hypothesized *a priori* to be a potential effect modifier or interaction variable, where the associations of environmental variables with infant growth, diet, morbidity, and careseeking are different for male and female infants. Interest in early life sex differences in nutrition and growth has received much attention in public health, epidemiology, anthropology (Miller, 1997), human biology, gender studies and development economics (Jayachandran and Kuziemko, 2011). Gender disparities in child health track into adulthood and examining their early manifestation in finer detail is of scientific interest.

There are several reasons for assessing interaction, or effect modification if of greater interest (Knol and VanderWeele, 2012, VanderWeele and Knol, 2014). First, assessing interaction could identify environmental covariates which can be altered to have the largest effect on health outcomes in either sex. Second, it could tease out factors or interventions that may be beneficial for one sex but harmful for the other, known as ‘crossover’ or ‘qualitative’ interaction. Third, interaction analyses could shed light on the mechanisms which lead to observed sex disparities in health outcomes. A fourth reason is that since infant sex is a non-modifiable factor, it is important to find the most relevant covariate which can be intervened on to reduce any negative effect associated with infant sex. A fifth reason, not determined by scientific or policy considerations, is that statistical models with interaction terms have greater flexibility and can therefore improve overall model fit in some instances.

The joint effect of two exposures is measured in two ways: using risk differences on an additive scale or using relative risk on a multiplicative scale. While it is good practice to report interaction on both scales (Knol and VanderWeele, 2012), additive interaction is more informative than multiplicative interaction from a public health perspective (Greenland and Rothman, 2008). Interaction between a risk factor and an intervention on an outcome on the additive scale would indicate whether those with the risk factor would see a larger benefit from the intervention than those without the risk factor – in which case a larger number of people would benefit if the

intervention were given to a hundred individuals with the risk factor than if it were administered to those without the risk factor. This is important for prioritizing or targeting subgroups for intervention and resource allocation. Multiplicative interaction would not provide this information, and only indicate the subgroup for which the risk ratio effect size of both risk factor and intervention is greater (VanderWeele and Knol, 2014). The usefulness of additive versus multiplicative interaction measures in ascertaining causality is largely context dependent (Poole, 2010).

Interpreting interaction analyses is also determined by whether adjustment for confounders has been made for either, neither or both exposures of interest. For example, in assessing interaction between two exposures, water supply and infant sex, on growth outcomes, if we are interested chiefly in estimating the effect of water supply on growth within sexes then it is sufficient to control for confounders of water supply only, and to interpret the analysis as effect modification. This would allow us to understand whether the growth of girls would benefit from improved water supply. However, it is possible that the secondary exposure, infant sex, is a proxy for another exposure that is causally related to growth. If we were interested in understanding the mechanistic interaction between infant sex and water supply, or if the secondary exposure were a factor amenable to intervention, we would need to adjust for confounders of the relationship between water supply and growth as well as a set of those for infant sex and growth. Adjusting for confounders of the relationships between both exposures and the outcome would allow us to assess causal interaction rather than just effect modification (VanderWeele, 2009).

The distinction between examining causal interaction and effect modification in the thesis rests first on whether any action to improve infant growth would involve one or two interventions: one on whichever environmental variable is considered for analysis, like water supply, and a second intervention related to infant sex. A further consideration is mechanistic interaction, and whether the analysis would attempt to examine whether the observed outcome is turned ‘on’ if both exposures are present and turned ‘off’ when either or neither are present (VanderWeele and Robins, 2007). In either case, it would be essential to control for confounding of both exposures with the outcome.

In the thesis I assume that it is not possible to intervene on the sex of an infant, though an intervention on gender-related care practices is possible. However,

examining the latent properties of infant sex informed by sociocultural constructs goes beyond the research focus in my thesis. And so I have focused on effect modification or stratified analysis by infant sex as appropriate.

The cohort study protocol did not mention any planned subgroup analyses, and I did not identify any baseline groups in the cohort that I wanted to include in a subgroup analysis. I used restricted samples of the cohort when carrying out complete-case analyses, but this decision was informed by statistical considerations in handling missing data (see Chapter 5) rather than for theoretical reasons related to the baseline characteristics of those with complete information. I do not present any sub-group analyses in this thesis.

Chapter 5 Cohort profile

Summary

In this chapter I present a profile of the cohort using descriptive statistics to outline the main characteristics of infants and their families recorded soon after birth. I investigate the relationships between baseline socioeconomic, infant, and parental variables. I also describe parental anthropometric data collected three months after birth. I calculate total duration of follow-up, dropout, and attrition. I investigate patterns of missing data and their causes at each post-baseline occasion and for subsequent analytic samples. Finally, I discuss the cohort baseline characteristics and participation rates in the context of recent research, and highlight the implications of missing longitudinal data for further analyses.

5.1 Introduction

A cohort profile can provide useful information on the methods and key findings of an established and large-scale longitudinal study, including on subsets of participants (see for example Boyd et al. (2013) and Fraser et al. (2013) for profiles of the ALSPAC index children and their mothers). While such a profile would be premature for this study, the format provides a useful template for describing the main characteristics of the study population and contextualising it within the context of recent research. The purpose of this chapter is to combine descriptive epidemiology with additional analyses of missing data patterns. A profile highlighting the circumstances in which the participants were born, and the ways in which they did or did not take part across components of the longitudinal study, is a useful building block for understanding subsequent sections of the thesis.

5.2 Research question and objectives

In this chapter, I address Research Question 3: What are the baseline characteristics of the cohort and how do they relate to patterns of follow-up, attrition, and missing data?

The five specific objectives were to:

1. Generate a profile of the cohort at baseline describing characteristics observed at or close to birth.
2. Describe parental anthropometric characteristics observed at least three months after the infant's birth.
3. Investigate relationships between baseline characteristics related to socioeconomic position, infant characteristics, parental health behaviours, and parental anthropometry.
4. Describe rates of participation, follow-up, non-response, and attrition over the study period.
5. Investigate and evaluate reasons for missing data in the cohort and specific analytic subsets of it.

5.3 Overview of missing data issues in cohort studies

While descriptive analyses of baseline survey data tend to be fairly straightforward statistical exercises, missing data pose certain analytic challenges.

Rubin's (1976) concept of missing data mechanisms is applied widely in epidemiologic studies, and can be understood in the context of the cohort. These mechanisms include missing completely at random (MCAR), missing at random (MAR), and missing not at random (MNAR). Data are MCAR when missingness of the outcome is independent of the real values of the outcome (for example, infant length) and exposure (for example, maternal education), such that infants with missing length data are not systematically different on length and maternal education values from those with length measured. Data are MAR when the missingness of an outcome is independent of the value of the outcome given exposure data, such that infants with missing length data are not systematically shorter or longer than those with length observed for the same level of maternal education. Data are MNAR if they are neither MAR nor MCAR, which would imply that infants with missing length values differ systematically in length from those with length observed, even after conditioning on maternal education. It is difficult to concretely test data for MCAR, MAR and MNAR. Most statistical analyses instead employ one of these mechanisms as an assumption when handling data with missing values, though using directed acyclic graphs to complement and guide the analysis strategy offers greater clarity (Daniel et al., 2012, Howe et al., 2016a).

The missing data challenges for cohort studies are slightly different. While Rubin's classification is useful and applicable to life course studies, it does not comprehensively capture mechanisms that lead to missing longitudinal data or characterize patterns of missingness. Three relevant concepts in life course studies are missing data patterns, missing value patterns, and non-response mechanisms (Clarke and Hardy, 2007).

Missing data patterns can be observed in the overall study as well as in subsets of variables used in each piece of analysis. Missing value patterns include four types that describe how missing data values can arise. A univariate pattern consists of a group of participants with all variables fully observed and another group with some missing data for one variable. A multivariate pattern includes several variables that are unobserved for the second group. A monotone pattern consists of several groups: a first, fully observed group, a second with some missing data, a third with more missing data than the first two, and so on. This pattern is observed in longitudinal studies when participants drop out at different times, such that those who remain in the study will have more data than those who dropped out at previous stages. A non-monotone pattern is one that is not univariate, multivariate, or monotone, and is observed in longitudinal studies where data present with intermittent missingness, indicating that participants dip in and out of the study and few have fully-observed information at all measurement occasions (Clarke and Hardy, 2007).

Non-response can stem from four mechanisms. Some participants may choose to not participate, leading to unit non-response. Some may not respond to certain questions or measurements (such as anthropometry), leading to item non-response. Attrition will occur when participants are lost to follow-up for whatever reason. Wave non-response is identified by successfully making contact with participants during a wave after they did not participate in previous wave(s) (Clarke and Hardy, 2007).

Selection bias due to non-response can lead to inaccurate or invalid estimates when missing data at successive measurement occasions or dropout are associated with exposure, outcome, or confounding variables (Kleinbaum et al., 1981). Restricting analysis to those who do not drop out, in effect conditioning on covariate-induced attrition if there is differential loss to follow-up, would lead to bias (Hernan et al., 2004). While there are several methods for dealing with selection bias in cross-sectional studies (for overviews, see Seaman and White (2013) or Laird (1988)),

there is no specific cut-off to classify the magnitude of potential bias as negligible or non-negligible in cohort studies.

Methodological work to quantify the extent of selection bias in cohort studies (see Pizzi et al. (2011) for a theoretical discussion and Pizzi et al. (2012) for an applied example from an Italian cohort) has focused on the magnitude of the odds of selection associated with an exposure or covariate, and the effect of this association on the exposure-outcome relationship. In cohort studies or analyses of cohorts that comprise specific sub-groups selected from a restricted source population, it is possible that the magnitude of the relationship between an exposure and an observed covariate in the selected cohort differs from that in the source population if the selection process is influenced by the exposure and covariate. Based on Monte Carlo simulations, the authors concluded that if exposure-selection odds ratios lie between 0.5 and 2.0, the bias in exposure-outcome log odds will be less than ±0.02 (Pizzi et al., 2011).

Such selection bias would apply in this context if my analyses in subsequent chapters relied on complete cases of participants who remained in the study for a longer duration (effectively a selected cohort) than the overall cohort (restricted source population, since the cohort comprises infants who met specific criteria rather than the general population of infants). An example of this would be an analysis of the relationship between maternal education (exposure) and duration of exclusive breastfeeding (outcome), adjusting for access to piped water (covariate). If maternal education and access to piped water are not related in the overall cohort, but if both are associated with longer duration of follow-up (and thus predict selection into an analysis sample of complete cases), they will be related in the analytical sample. If the odds ratio of selection associated with either variable is <0.5 or >2.0, the estimated relationship between maternal education and duration of breastfeeding in the analytic sample would be biased. In such a case, it is important to examine the relationship between all covariates and participation across ages, in order to rule out the possibility that a restricted cohort will lead to non-negligible bias in estimates.

In summary, in addition to describing the baseline characteristics of a cohort, it is essential to conduct a more thorough examination of missing longitudinal data and value patterns, and assess whether using restricted samples of the cohort would lead to biased estimates.

5.4 Methods

I first identified all eligible participants who had been successfully recruited to the cohort and met all inclusion criteria for my study. I retained their data in a master dataset, which formed the basis of analyses in this chapter as well as the rest of the thesis. I then carried out data analysis in three segments.

In the first segment I analysed responses to the baseline survey questionnaire, summarising data (missing and non-missing) on each indicator or question. I then conducted univariable and multivariable analyses to examine relationships between pairs or sets of variables. The purpose of this was to understand the baseline confounding structure of the cohort.

In the second segment I calculated the total follow-up time in the cohort in study time and participant time (age, as child-months), as well as the proportion of participants who dropped out of the study. I examined the association of baseline characteristics with attrition.

In the third segment I quantified the amount of missing data across follow-up visits for indicators of infant anthropometry, infant and young child feeding practices, and diarrhoeal illness, and mapped missing value patterns for each indicator of interest. I conducted multivariable analyses to identify baseline determinants of non-response at each age. I also examined analysis patterns of missingness: assessed if those included in analyses presented in Chapters 6-8 were systematically different from those who were excluded due to missing or insufficient data, and if the restricted samples were likely to produce biased estimates.

I conducted data analysis primarily in Stata 13, with additional analysis in R to produce graphs of missing value patterns. Stata .do files and R Script files are included in Appendix 5.1 and 5.2.

5.4.1 Analysis of cohort profile at baseline

I tagged one observation for each participant to identify their responses to baseline questions.

For each baseline variable of interest described in Chapters 3 and 4 for use in subsequent analyses, I calculated descriptive statistics as appropriate, focusing on

mean and standard deviation, median and IQR, frequency and proportion. Where appropriate, I also dichotomized continuous variables at the median value or at pre-specified cut-offs.

I also examined a handful of background variables that were not intended for further analyses but provided contextual detail relevant for an overview of the cohort's characteristics. These included month of birth, place of delivery, and maternal obstetric history. I summarized data on maternal postnatal depression measured using the Edinburgh Postnatal Depression Scale, treating it as a baseline variable even though it was measured four weeks after the infant's birth, as it would have made little practical difference to the rest of my analysis.

I generated histograms, scatter plots, box and whiskers plots, and bar charts, combining similar variables to examine their distributions within the same graph. I conducted tests of normality using a Shapiro-Wilk test (Stata command *swilk*) or a skewness and kurtosis test of normality (Stata command *sktest*). I used a combination of statistical tests and visual inspection of frequency histograms to assess departures from normality in each variable.

I tested relationships between pairs of characteristics in univariable analyses, examining differences in groups or measures of central tendency using appropriate parametric or non-parametric tests, based on whether the outcome variable of interest was (continuous and) normally or non-normally distributed, binary, or categorical (Table 5.1).

Table 5.1 Tests for univariable analyses of baseline characteristics

Test	Type	Stata command
Two-sample t-test to examine the difference between two groups	Parametric test for continuous data	ttest
K-sample test of equality of medians	Non-parametric test for continuous data	median
Kruskal-Wallis test of difference between two or more groups	Non-parametric test for continuous data	kwallis
Wilcoxon rank sum test of difference between two groups	Non-parametric test for continuous data	ranksum
Kendall's tau test of association between two variables	Non-parametric test for continuous data	ktau
Chi-squared test of association between two categorical variables	Test for contingency table	chi; tabodds
Chi-squared test for trend	Test for contingency table with ordered exposure	contrast; tabodds
Linear regression	Test of association for continuous outcome	regress

Test	Type	Stata command
Exposure odds ratio	Test of association for categorical outcome	tabodds
Logistic regression	Test of association for binary outcome	logistic
Multinomial regression	Test of association for multinomial outcome	mlogit

When extending univariable associations to stratified analysis, I restricted my analyses to stratification by infant sex, household wealth quintile, and parental age variables. I examined multivariable relationships between sets of variables using linear or logistic regression as appropriate. I also examined the relationships between background variables for evidence of multicollinearity in a multiple linear regression analysis (using the cluster ID number as a continuous outcome, since the relationships between covariates were independent of the outcome). I calculated the variance inflation factor using *estat vif*, a regression post-estimation command in Stata. I applied a cut-off for the variance inflation factor, with a value >10 indicative of multicollinearity, although much lower values below 3 or 5 would be better at ruling out multicollinearity.

I also conducted descriptive analyses for parental anthropometric measurements. I first summarised each variable individually and then looked at data for both parents together. I assessed variables for non-normality in the tails of distribution (5^{th} and 95^{th} percentile) using quantile normal plots (Stata command *qnorm*), and visually inspected graphs for evidence of departure from normality. I explored relationships between pairs of anthropometric measurements and examined the association of baseline socioeconomic, infant, and parental variables with parental anthropometry.

I assessed the proportion of missing data in each baseline variable of interest, and the proportion of incomplete cases to identify children who were missing any baseline data. I plotted a graph displaying the pattern of missing values in each variable (see Section 5.4.3 for more details on how missing value graphs were produced).

For parental anthropometry, I analysed the determinants of missing data to understand the extent to which non-response was covariate-induced. Based on the methods outlined by Pizzi et al. (2011), exposure-missingness odds ratios between 0.5 and 2.0 were unlikely to introduce non-negligible bias in analyses based on a

subset from the main population. (See section 5.4.4 for further detail on how I used these methods in my analyses).

5.4.2 Assessing follow-up time and dropout

I calculated total and mean follow-up time using 17,929 observations for 976 participants. These were occasions at which at least one of two main study variables (length and IYCF) had been recorded. While the dataset contained 23,134 observations, 5205 of these represented visits at which neither length or IYCF data had been collected (if the family were away for the school holidays or festive season, had migrated recently, the child was unwell, or parents refused to participate on that occasion), or other ancillary data (weight or immunization, for example) which are not relevant to my thesis had been recorded.

I used Stata's survival analysis suite of commands (*sts*) to investigate follow-up time. I calculated study-time in the full dataset to assess the average time spent in the cohort between March 2013 and April 2016. I calculated person-time of follow-up as child-months by truncating each child's data at their second birthday, even if they had been followed up for longer, to assess the average success in following children up to two years. The 'failure' event was the last occasion at which the participant's length was observed (before 24 months, or the end of the study), capturing the number of months for which each child was followed up. I also examined median person-time and study-time by baseline variables, using the *stci* command. I tested the equality of follow-up experience with a Log-rank test using the *sts test* command, which examines whether the expected contribution of groups at each duration of total follow-up time differed from observed values. For the household wealth quintile variable, I included the *trend* option for ordered categorical variables. I plotted survival curves, using the *sts graph* command, displaying follow-up time for each category of any variable that exhibited some group-differences.

Field investigators kept records open for children who had migrated recently in case the family returned to the study site. However, they did close cases when they were certain that the participant had dropped out. I tabulated reasons for case closure and examined the relationships between case closure and baseline characteristics using univariable and multivariable logistic regression.

In addition, I applied another definition of attrition. I designated those who did not have any data between 18 and 24 months as having dropped out, and examined the association of dropout with baseline characteristics.

5.4.3 Investigating missing longitudinal data

I first rectangularized the cohort dataset using Stata's *fillin* command such that all children had the same number of observations, one per integer month, corresponding to 24 visits per child under a perfect follow-up scenario. For children with gaps in follow up, the command created additional observations for months in which they did not have any visits recorded. For each visit (real or synthetic) in the dataset, I created binary variables for length, IYCF, and diarrhoea, coded zero if observed and one if missing (synthetic visits were coded 1 for all variables). For IYCF, I used the breastfeeding variable up to the fifth month and the complementary feeding variable thereafter to derive missingness.

Using Stata's looping commands *foreach* and *forvalues*, I tabulated response rates at each age for length, IYCF, diarrhoea, and any of the three, assessing the proportion of children that responded to data collection. I examined patterns of incomplete cases at each age, assessing the proportion of children who lacked data for none, one, two, or all three variables. I created bar charts to describe these proportions. I used survival analysis to inspect the age at which children first had missing data at a follow-up visit, plotting the data as a Kaplan-Meier survival curve.

I then created graphs to visualise missing value patterns. I used the *amelia* package in R, exporting a wide version (one row per participant) of the Stata dataset. I plotted ID numbers on the y-axis and age on the x-axis, creating a graph similar to a stacked area chart, but with several hundred values on the y-axis. A black shaded area in each x-y coordinate indicated that a child had data for a certain visit, and a grey one indicated that they did not. Each child's missing value pattern could (in theory, in a magnified view) be traced across the x-axis from the first visit up to the 24th month. The full map showed those who dipped in and out of the study (a chequered pattern) as well as ages at which non-response was particularly high or low (large black or grey patches) across the follow-up period. The graph also displayed the proportion of missing values in the dataset. I first produced graphs for length, IYCF, and diarrhoea missing value patterns for all 978 children, and then created separate length graphs for those who dropped out of the study by 18

months (using their data from 0-17 months) and those who were in active follow-up at 24 months.

When creating missing value graphs for baseline responses, I plotted variables on the x-axis and ID numbers on the y-axis. The baseline missing value graph served to visualise variables (non-response) as well as individuals (incomplete cases) with missing values.

In order to identify the determinants of wave non-response, I partially replicated the analysis presented in Pizzi et al. (2012). I investigated the relationships between baseline variables and missing data for length, IYCF and diarrhoea at each follow-up visit from 1 to 24 months (but included length at the baseline visit). I assessed whether any baseline characteristics consistently predicted non-response in a way that would lead to bias (covariate-visit OR <0.5 or >2.0) over several visits.

I did not use a straightforward definition of how frequently (or in which periods) a covariate would need to influence wave non-response for it to become problematic. Instead, I decided that covariate-induced selection bias would be problematic if the magnitude was consistently less than 0.5 or greater than 2.0 at multiple waves and for more than one indicator (IYCF, length, diarrhoea) in multivariable analyses.

Using logistic regression with response at each wave as the outcome variable, I calculated univariable ORs of missing data for each background variable, and then multivariable ORs adjusted for all background variables. I used the Stata *foreach* command to loop over length, IYCF, and diarrhoea missingness at successive visits. For this component, I converted the asset quintile variable into a binary variable coded 1 for the highest quintile, and 0 for the other four, since Pizzi et al (2011) recommend that all continuous and categorical variables be made binary for simplicity. In a separate analysis I examined the relationship between parental response to anthropometric data collection (maternal and paternal heights and weights) and baseline variables.

5.4.4 Analysis patterns of missing data

For analysis patterns of missing data, I used subsets of participants who met data requirements for each piece of analysis. Once again, I replicated the analysis

described by Pizzi et al (2011) to understand if using a restricted sample of the cohort resulted in non-negligible selection bias.

For each analytic sample representing <90% of the cohort (or fewer than 880 participants, implying over 10% loss to follow-up), I examined baseline differences between those included in the analysis and those who were excluded due to missing data (in exposure, outcome, or covariates) in univariable and multivariable logistic regression models. Once again, I used a binary variable to indicate highest asset quintile.

When the baseline covariate-inclusion odds ratio for missing data was <0.5 or >2.0 for any variable – planned pregnancy, for example – I examined whether the association of planned pregnancy with other covariates differed between those in the analytic sample and the overall cohort. Essentially, this further step in the analysis enabled me to understand if the confounding structure of the restricted sample was different to that in the overall cohort as a result of covariate-induced self-selection.

The statistical methods I employed for subsequent research questions (explained in detail in Chapters 6-8) were a mix of methods that required complete cases and methods that accommodated unbalanced longitudinal data, and so the number of participants I included varied considerably. I was able to include 97% of the cohort in my analysis of the baseline determinants of linear growth, but only 45% when examining the relationship between predominant breastfeeding and length at 24 months mediated by diet in the complementary feeding period (Table 5.2). I did not investigate missingness further for Chapter 6 since the first analysis included 97% of the cohort and the second sample has already been addressed in analysis of the determinants of missing parental anthropometry data (see section 5.5.8).

Table 5.2 Participants in multivariable analyses in Chapters 6-8

Chapter	Analysis	Analytic method	N (%)	Type of subset
Chapter 6	Relationship between baseline variables (covariate) and linear growth (0-37 months) (longitudinal outcome)	SITAR	949 (97%)	Complete covariate and unbalanced outcome data
	Relationship between parental anthropometry (exposure) and linear growth (0-37 months) (longitudinal outcome)	SITAR	508 (52%)	Complete exposure /covariate and unbalanced outcome data

Chapter	Analysis	Analytic method	N (%)	Type of subset
Chapter 7	Relationship between baseline variables (covariates) and cessation of exclusive / predominant breastfeeding (0-5 months) (survival outcome)	Discrete-time survival analysis	533 (54%)	Complete covariate and outcome data (complete case analysis)
	Relationship between baseline variables (covariates) and introduction to solid foods (6-8 months) (survival outcome)	Discrete-time survival analysis	550 (56%)	Complete covariate and (partially complete) outcome data
	Baseline determinants (covariates) of achieving minimum dietary diversity (outcome) of complementary feeding (6-23 months)	Dynamic autoregressive model	746 (76%)	Complete covariate and unbalanced outcome data
	Baseline determinants (covariates) of consuming animal source foods (outcome) in the complementary feeding period (6-23 months)	Dynamic autoregressive model	746 (76%)	Complete covariate and unbalanced outcome data
	Baseline determinants (covariates) of consuming snack foods (outcome) in the complementary feeding period (6-23 months)	Dynamic autoregressive model	746 (76%)	Complete covariate and unbalanced outcome data
Chapter 8	Effect of predominant breastfeeding (exposure) on predicted length at 24 months (outcome) mediated by consumption of animal source foods (mediator)	Causal mediation analysis (linear regression)	438 (45%)	Complete case analysis

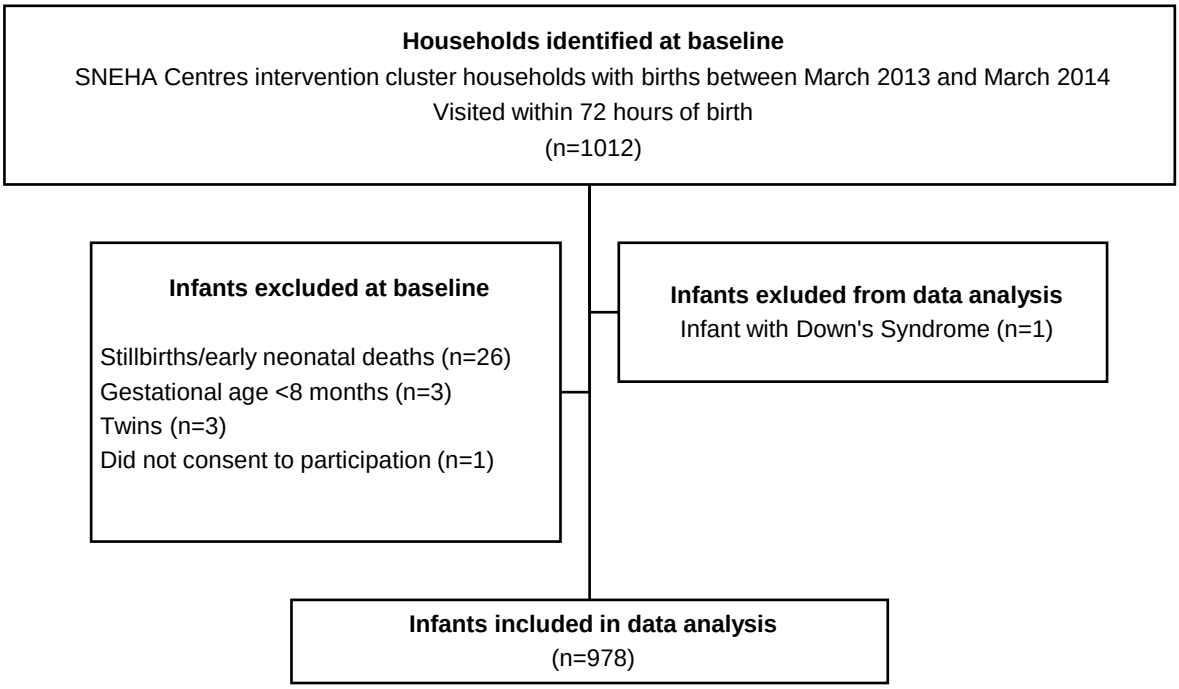
5.5 Description of the cohort

5.5.1 Recruitment profile

Between March 2013 and March 2014 investigators identified 1012 households in which women had given birth. Three were twin births, one woman did not consent to participation, and three infants were born before eight months' gestation. Twenty-five births were either stillbirths or the infant had died before study investigators visited families at home, and their mothers were not enrolled in the study. One infant was alive when their mother consented to participation at the initial visit, but when investigators visited the family at home nine days after birth for a baseline visit, the parents reported that the infant had died. The cause of death was not reported. Some baseline information about the household was available, but I did not include the infant in any analyses. I also excluded one infant with Down's syndrome from all analysis despite the availability of baseline and follow-up information. I included 978

infants (97% of households identified at baseline) who met all inclusion criteria (Figure 5.1).

Figure 5.1 Participant flow diagram

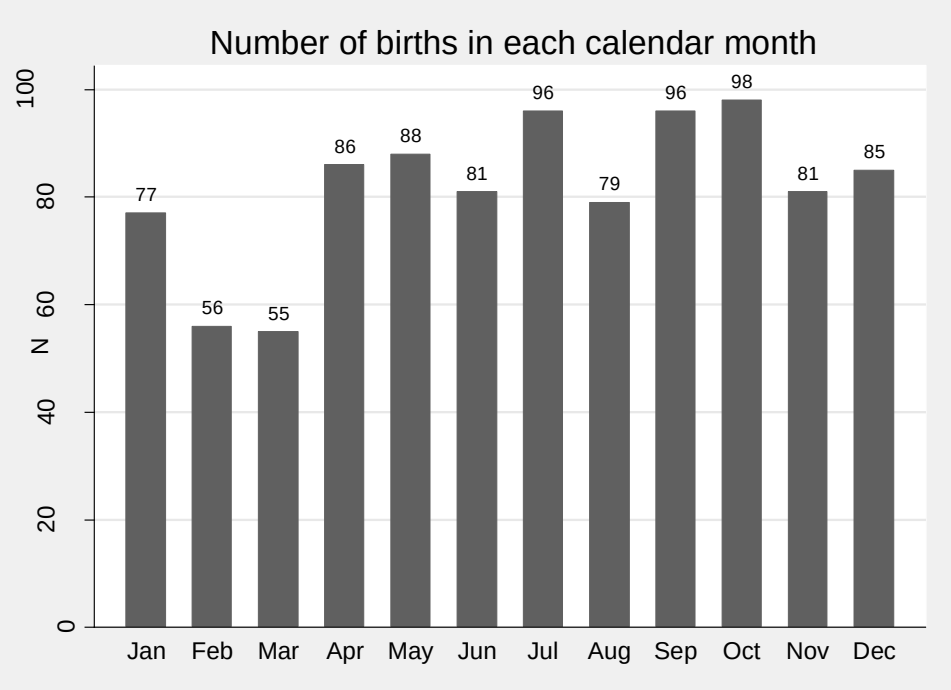


5.5.2 Infant characteristics

5.5.2.1 Infant sex and month of birth

Of 978 infants, 473 (48%) were male and 505 (52%) were female. The highest number of births per month (Figure 5.2) occurred in October (98, 10%), and the lowest in March (55, 6%).

Figure 5.2 Number of births in each month



5.5.2.2 Place of delivery

Of 971 infants with available information, 941 (96%) were born in Mumbai, and 62 (6%) were home births. Of the 94% born in health facilities, 118 (13%) were in municipal maternity homes, 421 (46%) in municipal hospitals, 336 (37%) in private hospitals, and 34 (4%) in larger government hospitals.

5.5.2.3 Maternal obstetric history

Twenty four percent of mothers reported that they were married before the age of 18; the median age at marriage was 19 years (IQR 18-20 years). The median age at first pregnancy was 20 years (IQR 19-22 years). Eighty-eight percent had never experienced the loss of a child either through stillbirth or in the first five years of its life. A similar proportion (84%) reported that they had never had an abortion or miscarriage. Of those who had, 45% had experienced the loss in the three years preceding the survey.

5.5.2.4 Maternal and paternal age at infant birth

Mean maternal age at infant birth was 25.6 years (SD, 4.5 years; range 17-42 years), and mean paternal age was 30.2 years (SD, 5.3 years, range 20-55 years). T-tests showed that there was little evidence of differences in maternal (p= 0.2331) or paternal (p= 0.9665) age by infant sex. Median maternal and paternal ages were

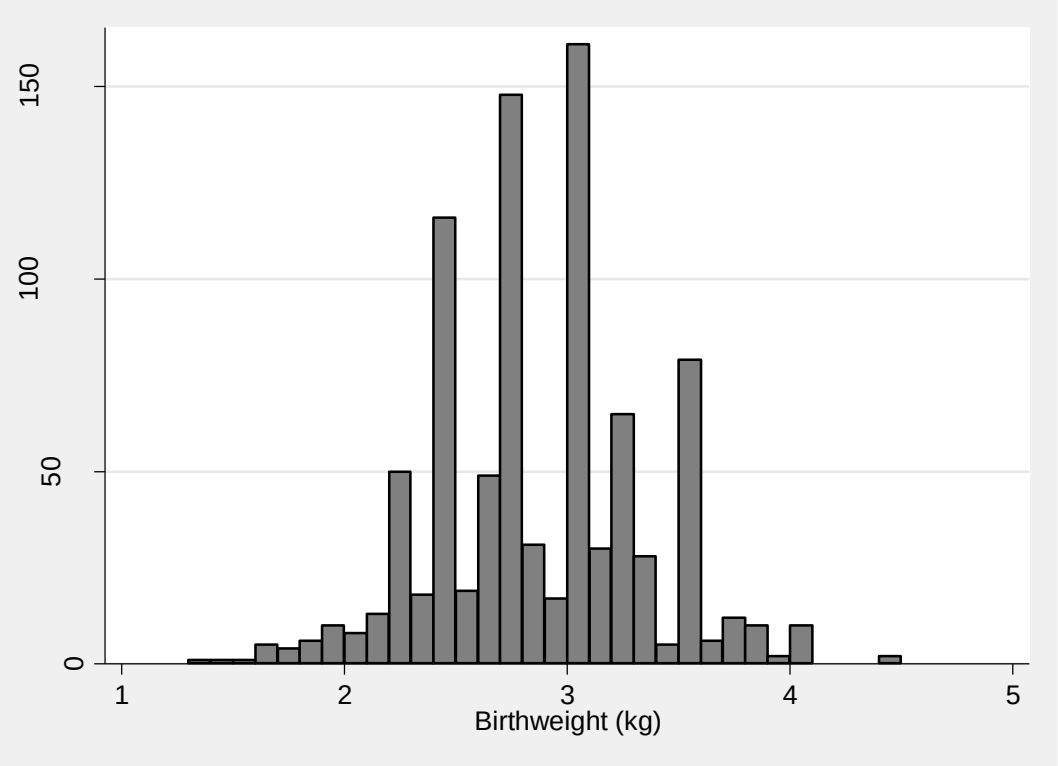
25 and 30 years, respectively, which I used subsequently to generate binary variables to categorize younger and older parents. Chi-squared tests showed that neither maternal age ≥25 years (p= 0.105) nor paternal age ≥30 years (p= 0.482) was associated with infant sex. However, parental ages were highly correlated (linear regression coefficient 0.70; 95%CI 0.67, 0.73). Women aged 25 years or over were 18.7 times (95%CI 13.4, 26.1) more likely to be married to men who were 30 or over.

5.5.2.5 Birthweight

A Shapiro-Wilk test for normality suggested that birthweight data were not normally distributed (p= 0.0014). A histogram (Figure 5.3) showed that values were heaped at 2.5kg (16%), 3.0kg (12%), and 3.5kg (8%).

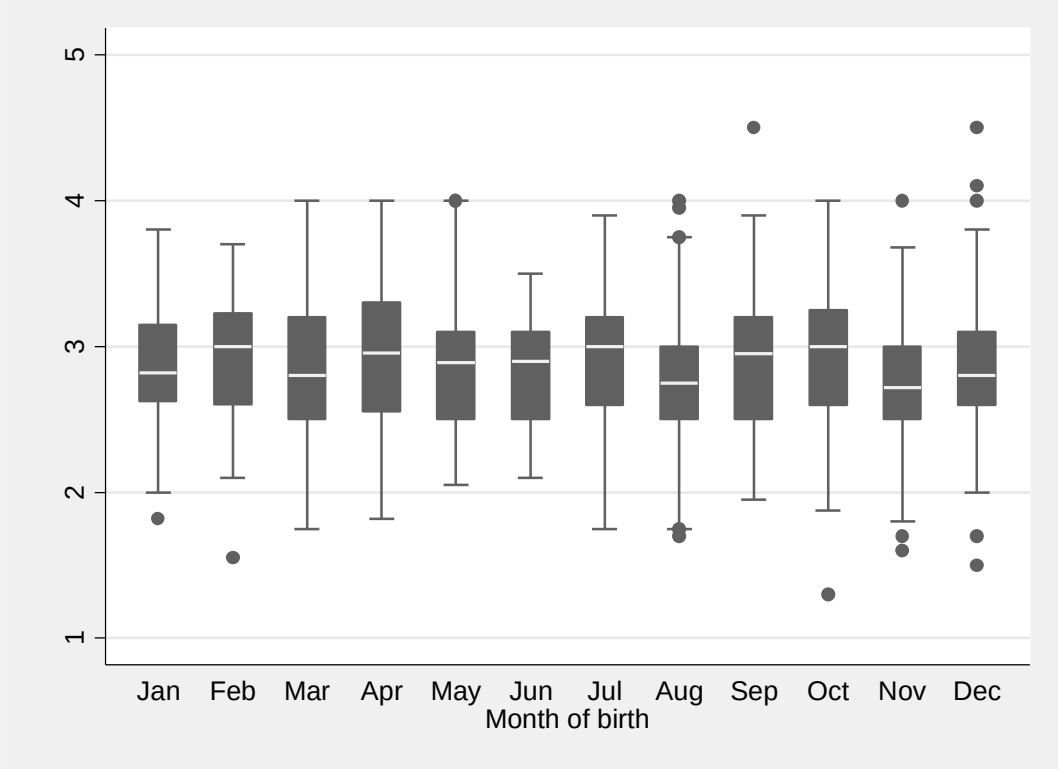
Mean birthweight for 907 infants with data was 2.86 kg (SD 0.46 kg, range 1.3 kg to 4.5 kg), and the median 2.84 kg (IQR 2.5 kg to 3.1 kg). Mean birthweight for girls was 92 g lower than that for boys; and a test of equality of medians indicated that there was evidence of a difference in birthweight between the sexes (p= 0.002). 128 infants (14%) weighed less than 2500 g at birth. One infant weighed less than 1500 g at birth.

Figure 5.3 Histogram of birthweight data



Birthweights were similar over the calendar year (Figure 5.4), with little evidence to support a hypothesis that birthweight differed by month of birth (Kruskal-Wallis test p= 0.4014).

Figure 5.4 Box-and-whiskers plot of birthweight data by month of birth



5.5.3 Socio-economic position (SEP)

5.5.3.1 Household asset score and quintiles

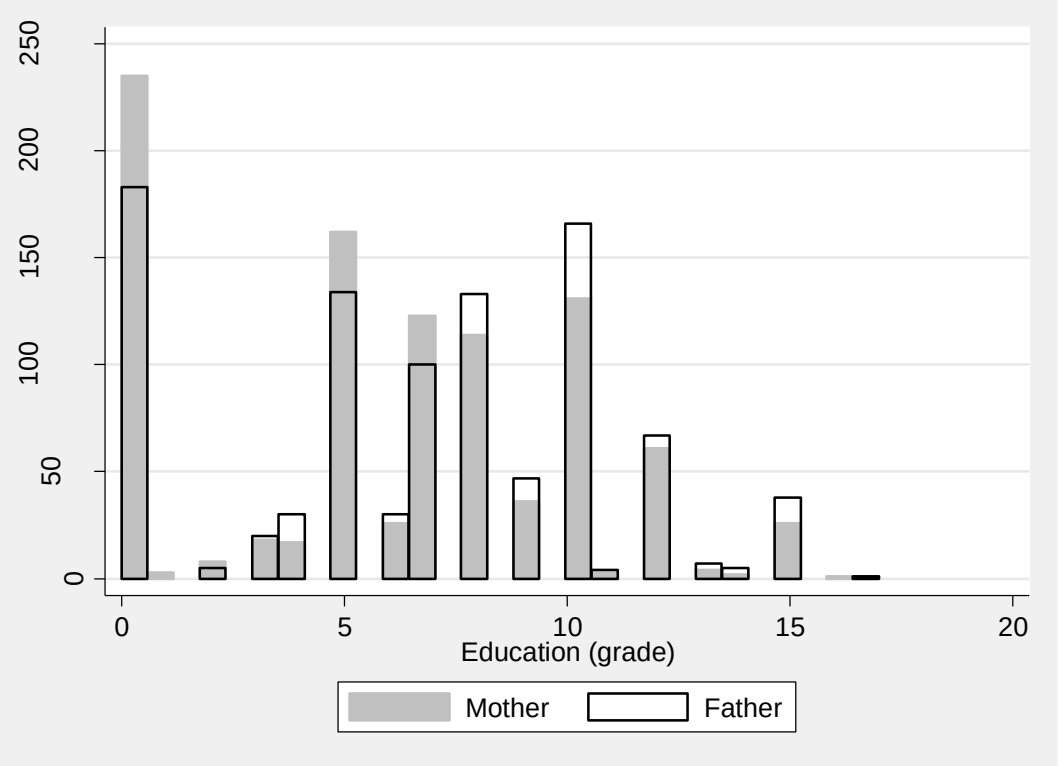
Household asset score and quintile were both related to infant sex. Girls were less likely to be born into families with higher asset scores (OR 0.86; 95%CI 0.76, 0.98). Across quintiles, there was some evidence that girls were more likely to be born into households in the lowest asset quintile than the highest (OR 1.52; 95%CI 1.01, 2.27), although the association did not hold for the three middle quintiles. A chi-squared test for trend showed weak evidence of a linear trend (p= 0.0261).

5.5.3.2 Parental education and occupation

Among 971 children, 24% of mothers and 18% of fathers had not completed any formal schooling (Figure 5.5); the median grade of educational attainment was seven (corresponding to lower secondary education) for both parents (IQR 2-9 among mothers and 5-10 among fathers). Fifty-four percent of mothers and 62% of

fathers had studied beyond the fifth grade. Maternal and paternal education were correlated; for every grade of maternal education, paternal education increased by 0.44 of a grade (95%CI 0.43, 0.45). Women whose husbands had studied beyond the fifth standard were more likely to have attained a similar level of education themselves (OR 4.5, 95%CI 3.45, 6.02).

Figure 5.5 Frequency distribution of maternal and paternal education



There was some evidence of a relationship between maternal education and infant sex. Girls were less likely to be born to women with greater educational attainment (OR 0.96, 95%CI 0.93, 0.99), or those who had studied beyond the fifth standard (OR 0.71, 95%CI 0.55, 0.92). There was no association between paternal education and infant sex, but it weakened the influence of maternal educational attainment (aOR 0.96; 95%CI 0.93, 1.00), as well as maternal education beyond the fifth standard (aOR 0.70; 95%CI 0.54, 0.93).

Nearly all (99%) mothers reported that they were not currently engaging in any economic activity at the time of the baseline survey. The most commonly reported occupations among fathers were skilled crafts like pottery or tailoring (55%), factory or machine work (17%), and unskilled work like vending or door-to-door trade (15%).

5.5.3.3 Water and sanitation

The most common source of drinking water was piped water accessed in the yard plot (39%), followed by use of a public tap standpipe (30%), and piped water into the home or dwelling (23%). Use of privately-managed water tanker / trucks was less common (7%). 92% of families used flush toilet facilities and 8% used ventilated improved pit latrines. Only one family reported that they did not have access to any toilet facility or used an open field or road.

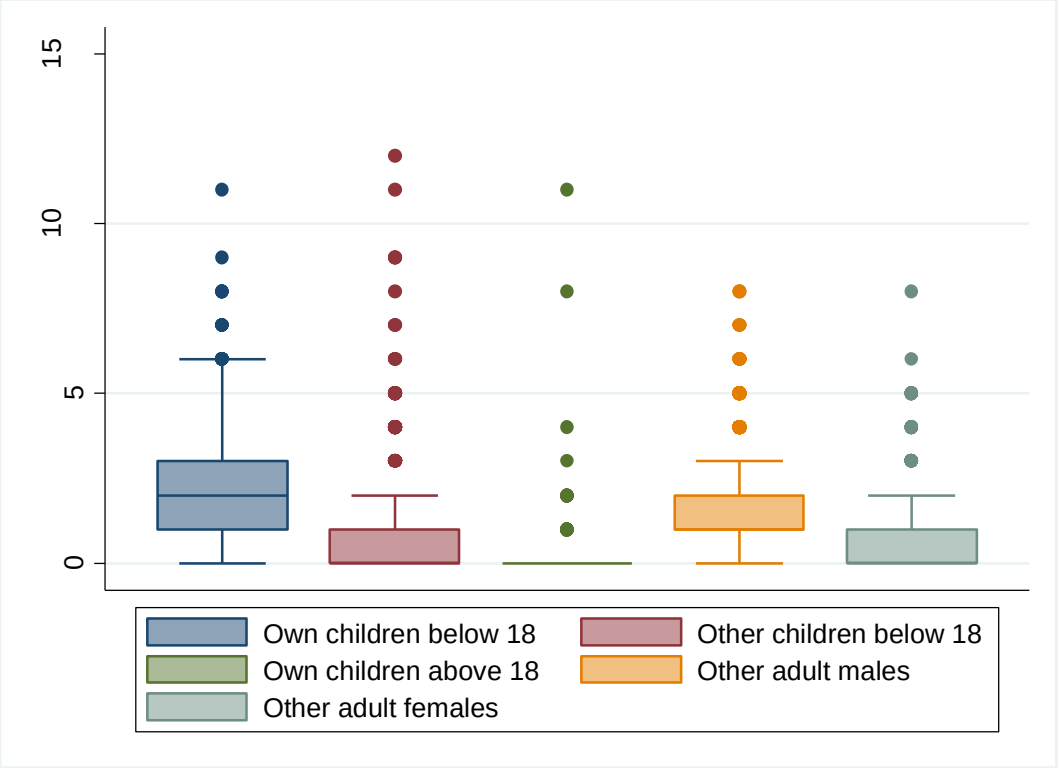
Based on normative criteria for access to water and sanitation, 84% of families shared a toilet facility with other households and 38% did not have access to piped water in the home or the yard plot. Families who had access to piped water were 90% less likely to use a shared toilet facility (OR 0.11; 95%CI 0.06, 0.21).

Neither lack of access to piped water nor use of shared toilet was related to infant sex in univariable or mutually adjusted logistic regression analyses (mutually adjusted OR 0.87; 95%CI 0.64, 1.11 for water and 1.21; 95%CI 0.65, 1.12 for shared toilets).

5.5.3.4 Household composition

Sixty percent of mothers had at least two children, including the index infant, below the age of 18 years. Four responses were not valid as women reported that they did not have any children. (However, I retained these observations when creating a binary variable.) Ninety-seven percent did not have any children older than 18 years. In 75% of households, there were no other children (the index infant's cousin or non-related child) below the age of 18. 98% of families had an adult male other than the index infant's father living in the home. The analogous proportion for adult women other than the index infant's mother was 59%. Household composition varied widely across the study (Figure 5.6), and Shapiro-Wilk tests for normality indicated that data in all variables were non-normally distributed ($p<0.001$ for all five).

Figure 5.6 Box and whiskers plots of household composition variables.



The median number of adults (including the index infant's siblings ages 18 and over) in the household was one, and the median number of children was three. Binary household composition variables (≥2 adults or ≥4 children) were not related to the sex of the infant (OR 0.82; 95%CI 0.63, 1.07 for ≥4 children and OR 0.93; 95%CI 0.73, 1.20 for ≥2 adults).Households with ≥2 adults other than the parents were more likely to have ≥4 children (OR 1.73; 95%CI 1.32, 2.26).

5.5.4 Parental smoking

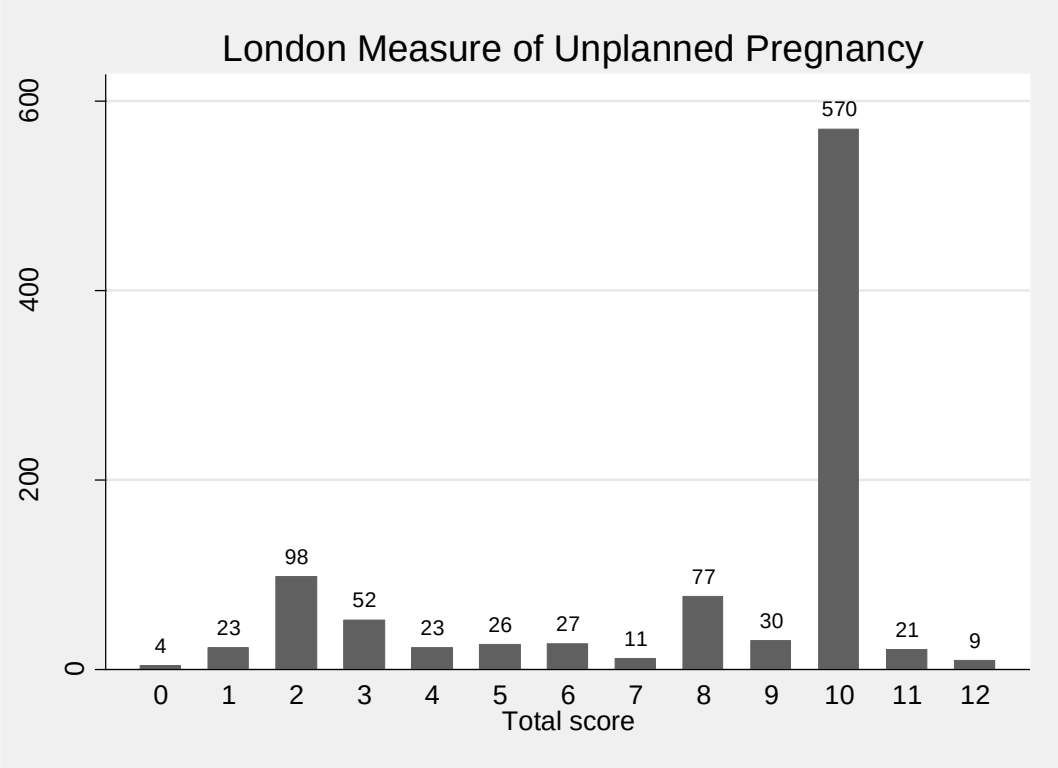
Smoking was more prevalent among fathers (55%) than mothers (13%), with evidence of a relationship between maternal and paternal smoking (chi-squared p<0.001). Infant sex was not associated with either parent's smoking behaviour (p= 0.406 for fathers and p= 0.353 for mothers).

5.5.5 Pregnancy intention

Responses (n=971) to the London Measure of Unplanned Pregnancy (LMUP) covered the full range of scores (0 to 12), indicating increasing intendedness. The median score was ten (IQR 6-10). A Shapiro-Wilk test for normal data indicated that

values were not normally distributed (p<0.001), and a histogram confirmed this; a large proportion (59%) of respondents scored 10 out of 12 (Figure 5.7). Kruskal-Wallis tests indicated that median LMUP scores did not differ by infant sex (p= 0.5653). However, there was strong evidence of differences by maternal and paternal age groups and number of children in the household (p<0.0001 for all three), with weaker differences by paternal smoking (p= 0.0006) and paternal education (p= 0.0141).

Figure 5.7 Distribution of unplanned pregnancy scores



Six hundred (62%) infants were born as a result of planned or highly planned pregnancies (LMUP score of more than 9). There was little evidence of sex differences in pregnancy intention (p= 0.368).

5.5.6 Relationships between infant, parental, and SEP variables

All univariable logistic regression odds ratios for pairs of infant, parental, and SEP variables are reported in a matrix in Table 5.3.

Girls were less likely to be born to more educated women and in higher wealth quintiles. Older parents were less educated, had households with more children and

fewer adults, were more likely to smoke, and were more likely to have an unplanned pregnancy.

Both maternal and paternal education (which were themselves positively correlated) were associated with higher wealth quintile, access to piped water, lower odds of a shared toilet, fewer children in the household, more adults, and lower odds of either parent smoking.

Further, children born in higher wealth quintile households were more likely to have access to piped water, less likely to use a shared toilet, and have parents who smoked. Access to piped water was also associated with households with two or more adults and fewer than four children. Households that used a shared toilet were more likely to have four or more children and parents who smoked, and less likely to have more than two adults. Children born in households with four or more children were more likely to have parents who smoked.

In Mantel-Haenszel tests of homogeneity of odds ratios in stratified analyses, there was little evidence (all $p>0.05$) of quintile-specific or sex-specific relationships between pairs of SEP variables. For example, there was no evidence that the relationship between having more than two adults and four or more children in a household differed across asset score quintiles ($p= 0.1818$), or that the relationship between maternal education and use of a shared toilet differed between boys and girls ($p= 0.6672$).

In analyses stratified by maternal and paternal age groups, there was some evidence of parental age-specific relationships between background variables. There was evidence of stratum-specific differences in the relationships between maternal education and number of children in the household ($p= 0.002$ for maternal age and $p= 0.0098$ for paternal age), access to piped water and number of adults ($p= 0.0328$ for maternal age), number of adults and children ($p<0.0001$ for both parents), and of the association of asset quintile with number of children ($p= 0.0002$ for maternal age and $p= 0.0003$ for paternal age) and adults in the household ($p= 0.0099$ for maternal age and $p= 0.0163$ for paternal age). For maternal age, the relationships between paternal smoking and maternal education ($p= 0.0343$), and maternal smoking and adults in the house ($p= 0.0185$), showed evidence of stratum-specific associations. For paternal age, there was also evidence of age-specific

differences in the relationship between access to piped water and number of children in the household (p= 0.0035).

Table 5.3 Univariable associations between infant, parental, and SEP characteristics

	OR (95%CI)											
Variable [OR (95%CI)]	**Female**	**Maternal age ≥25**	**Paternal age ≥30**	**Maternal education**	**Paternal education**	**Water**	**Toilet**	**Adults**	**Children**	**Father smokes**	**Mother smokes**	**LMUP**
Maternal age ≥25	0.81 (0.6, 1.0)											
Paternal age ≥30	0.91 (0.7, 1.2)	18.7 (13.4, 26.1)										
Maternal education ≥6th standard	0.72 (0.6, 0.9)	0.37 (0.3, 0.5)	0.34 (0.3, 0.4)									
Paternal education ≥6th standard	0.92 (0.7, 1.2)	0.68 (0.5, 0.9)	0.67 (0.5, 0.9)	4.6 (3.4, 6.0)								
Access to piped water	0.82 (0.6, 1.1)	0.92 (0.7, 1.2)	0.98 (0.7, 1.2)	1.77 (1.3, 2.3)	1.83 (1.4, 2.4)							
Use of shared toilet	1.29 (0.9, 1.8)	0.88 (0.6, 1.2)	0.98 (0.7, 1.4)	0.42 (0.3, 0.6)	0.27 (0.2, 0.4)	0.11 (0.1, 0.2)						
≥2 adults in the HH	0.9 (0.7, 1.2)	0.43 (0.3, 0.6)	0.47 (0.3, 0.6)	2.82 (2.2, 3.7)	1.9 (1.5, 2.5)	2.22 (1.7, 2.9)	0.46 (0.3, 0.6)					
≥4 children in the HH	0.82 (0.6, 1.1)	4.1 (3.1, 5.5)	3.21 (2.4, 4.2)	0.44 (0.3, 0.6)	0.52 (0.4, 0.6)	0.88 (0.7, 1.2)	1.06 (0.7, 1.5)	1.72 (1.3, 2.2)				
Father smokes	1.11 (0.8, 1.4)	1.55 (1.2, 2.0)	1.34 (1.0, 1.7)	0.53 (0.4, 0.7)	0.41 (0.3, 0.5)	0.74 (0.6, 0.9)	2.32 (1.6, 3.3)	0.56 (0.4, 0.7)	1.51 (1.2, 1.9)			
Mother smokes	1.19 (0.8, 1.7)	2.4 (1.6, 3.6)	2.24 (1.5, 3.3)	0.40 (0.3, 0.6)	0.52 (0.3, 0.7)	0.84 (0.6, 1.2)	1.41 (0.8, 2.4)	0.57 (0.4, 0.8)	2.13 (1.4, 3.1)	2.37 (1.6, 3.6)		
Planned pregnancy	1.12 (0.9, 1.4)	0.45 (0.4, 0.6)	0.42 (0.3, 0.6)	1.16 (0.9, 1.5)	1.26 (0.9, 1.6)	0.83 (0.6, 1.1)	1.02 (0.7, 1.4)	1.11 (0.8, 1.4)	0.52 (0.4, 0.7)	0.65 (0.5, 0.8)	0.74 (0.5, 1.1)	
Asset quintile												
Second lowest	0.77 (0.5, 1.1)	0.83 (0.6, 1.2)	0.93 (0.6, 1.4)	1.7 (1.1, 2.5)	1.71 (1.1, 2.5)	1.40 (0.9, 2.1)	0.22 (0.1, 0.7)	2.14 (1.3, 3.3)	1.07 (0.7, 1.6)	0.62 (0.4, 0.9)	0.69 (0.4, 1.2)	0.78 (0.5, 1.2)
Middle	0.77 (0.5, 1.1)	1.13 (0.8, 1.7)	1.3 (0.9, 2.0)	1.6 (1.1, 2.4)	2.02 (1.3, 3.0)	2.22 (1.5, 3.3)	0.15 (0.1, 0.4)	3.11 (1.9, 4.9)	1.5 (1.0, 2.3)	0.81 (0.5, 1.2)	0.97 (0.5, 1.6)	0.79 (0.5, 1.2)
Second highest	0.63 (0.4, 0.9)	0.84 (0.6, 1.3)	1.16 (0.8, 1.7)	5.12 (3.3, 7.8)	3.91 (2.5, 5.9)	2.67 (1.7, 4.0)	0.07 (0.0, 0.2)	5.87 (3.7, 9.22)	1.32 (0.8, 2.0)	0.45 (0.3, 0.7)	0.7 (0.4, 1.2)	1.06 (0.7, 1.6)
Highest	0.65 (0.4, 0.9)	0.74 (0.5, 1.1)	1.16 (0.4, 1.0)	8.9 (5.5, 14.3)	7.51 (4.6, 12.2)	8.15 (4.9, 13.5)	0.02 (0.0, 0.1)	18.18 (10.8, 30.3)	0.93 (0.6, 1.4)	0.27 (0.2, 0.4)	0.1 (0.0, 0.3)	1.06 (0.7, 1.6)

5.5.7 Parental anthropometry

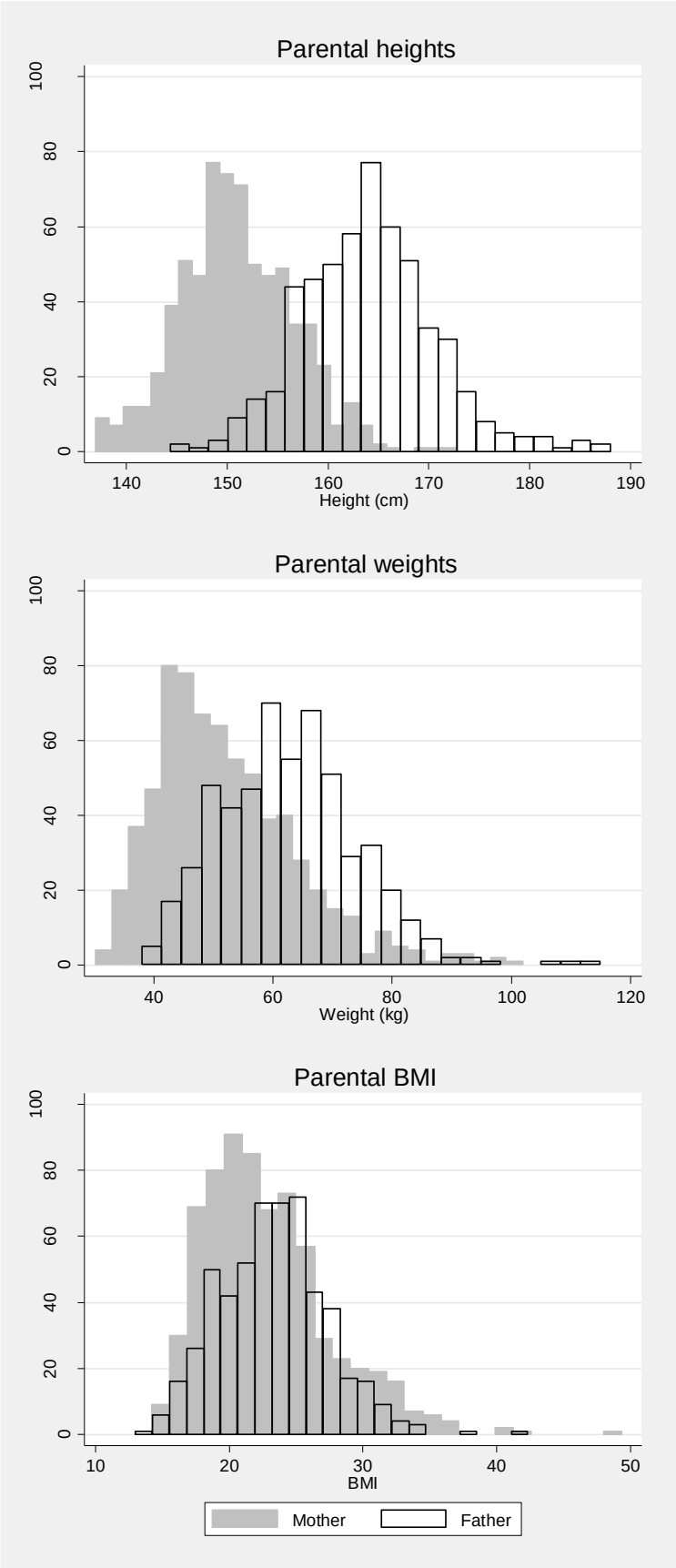
Height and weight data were available for 690 (71%) mothers and 537 (55%) fathers (Table 5.4). Data on both parents were available for 522 (53%) infants.

Table 5.4 Summary of parental anthropometry

	N	Mean	SD	Median	Min	Max
Father						
Height (cm)	537	163.9	6.6	164	144.4	188.0
Weight (kg)	537	62.7	11.3	62	38.0	115.0
BMI (kg/m^2)	537	23.3	4.0	23.3	13.0	42.4
Mother						
Height (cm)	690	150.9	5.6	150.6	137.0	172.7
Weight (kg)	690	52.1	11.8	49.9	30.3	102.0
BMI (kg/m^2)	690	22.8	4.7	21.9	14.2	49.4

The distribution of height, weight and BMI values for men and women is presented in Figure 5.8.

Figure 5.8 Histograms of superimposed maternal and paternal anthropometry variables



Shapiro-Wilk tests for normal data indicated that maternal and paternal height, weight, and BMI values were not normally distributed (p= 0.03165 for maternal height, and p<0.001 for the other five variables). Quantile normal plots to check for normality in the tails of distribution for each variable showed that data points in the top and bottom 5% of the distribution deviated from values that would be expected if data were normally distributed (Figure 5.9). Weight and BMI variables appeared to be slightly skewed to the right in histograms (Figure 5.10)

Figure 5.9 Quantile normal plots of maternal and paternal anthropometry variables

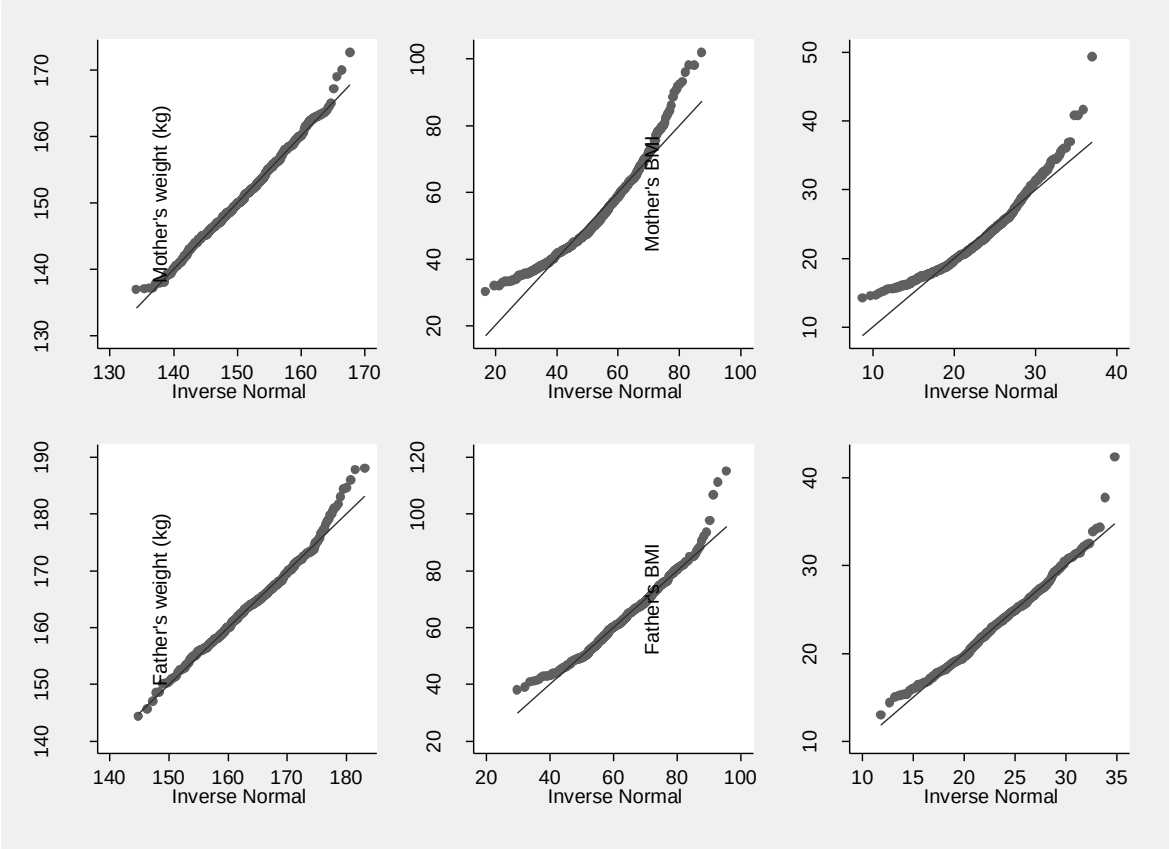
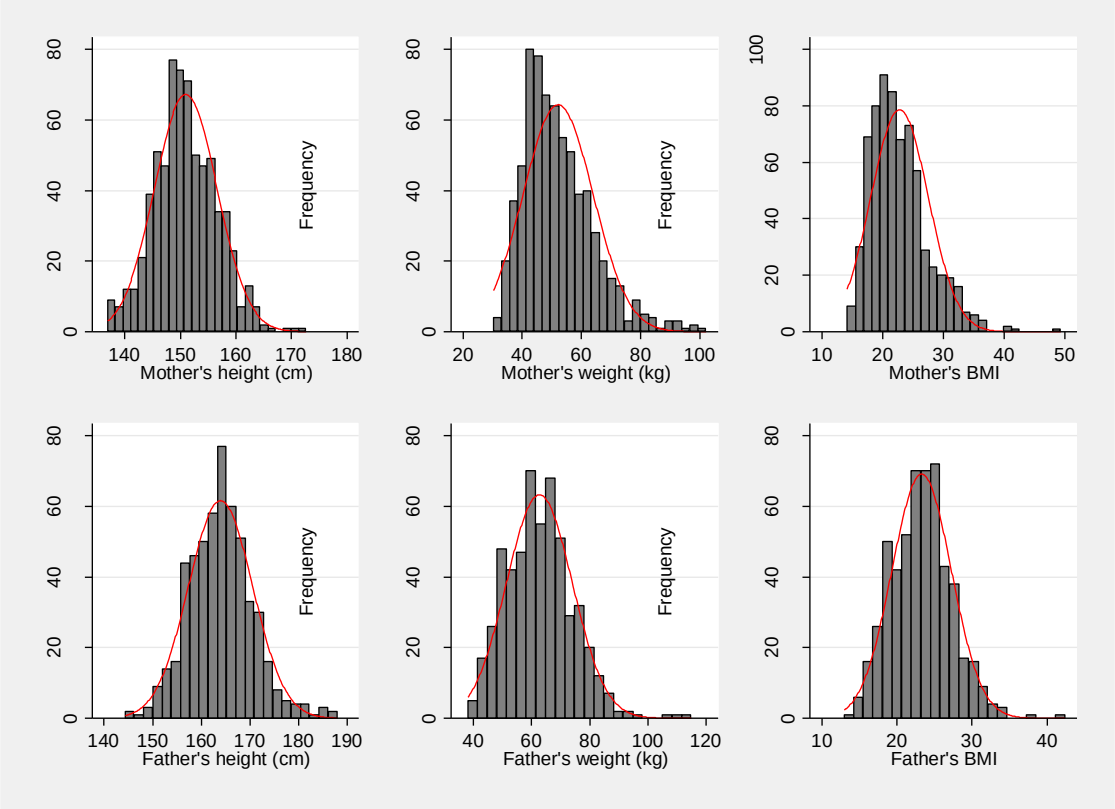


Figure 5.10 Histograms of maternal and paternal anthropometry variables



Wilcoxon signed rank tests of no difference between paired observations indicated that there was evidence of differences between median maternal and paternal heights ($p<0.0001$) and weights ($p<0.0001$), but not in BMI ($p= 0.0559$).

Kendall tau tests of independence between two variables showed that maternal and paternal measurements were correlated for height, weight, and BMI (all $p<0.0001$). Visual inspection of scatterplots also indicated that taller and heavier women had taller and heavier husbands (Figure 5.11 and Figure 5.12).

Figure 5.11 Scatter plots of maternal and paternal heights and weights

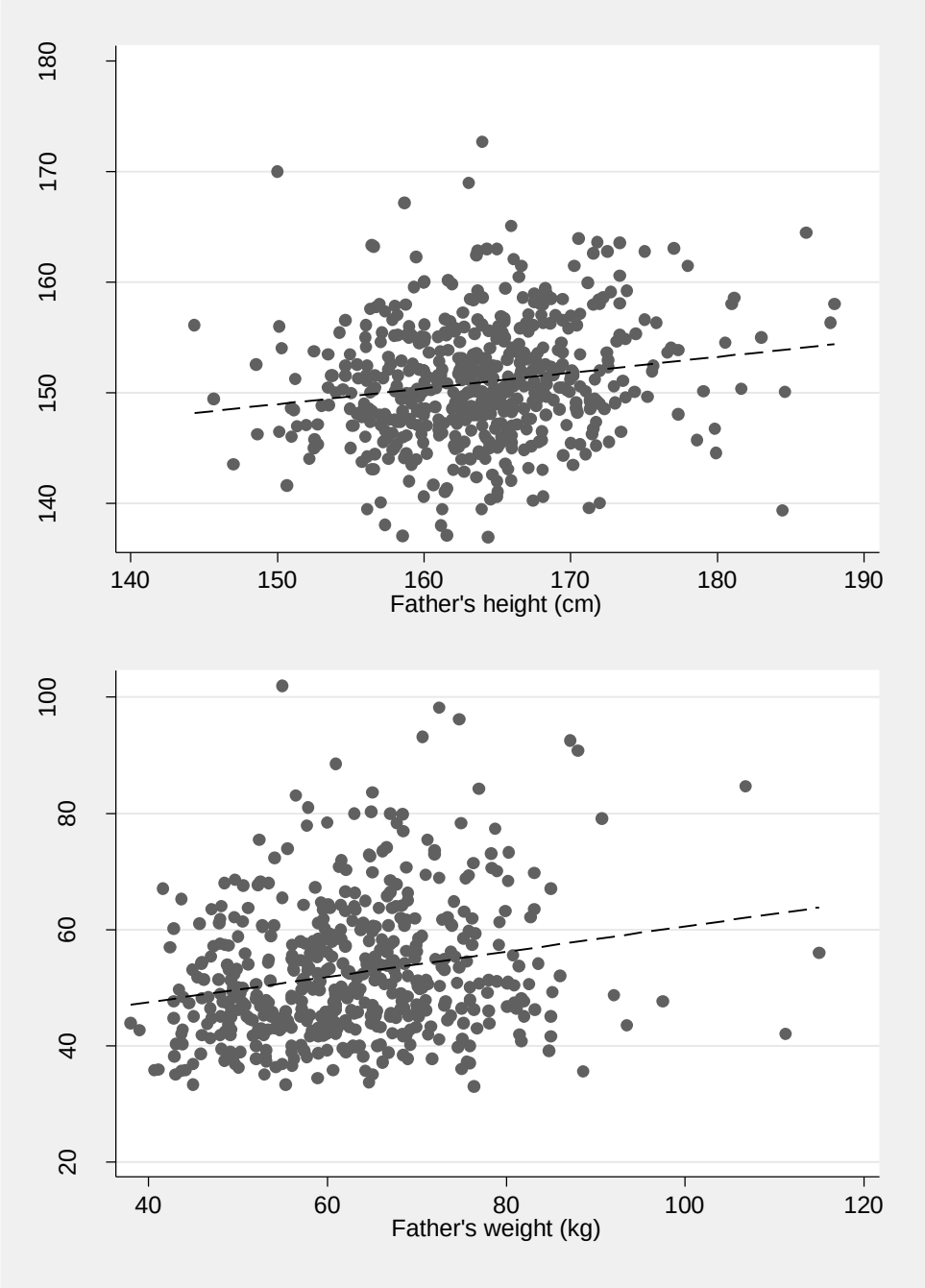
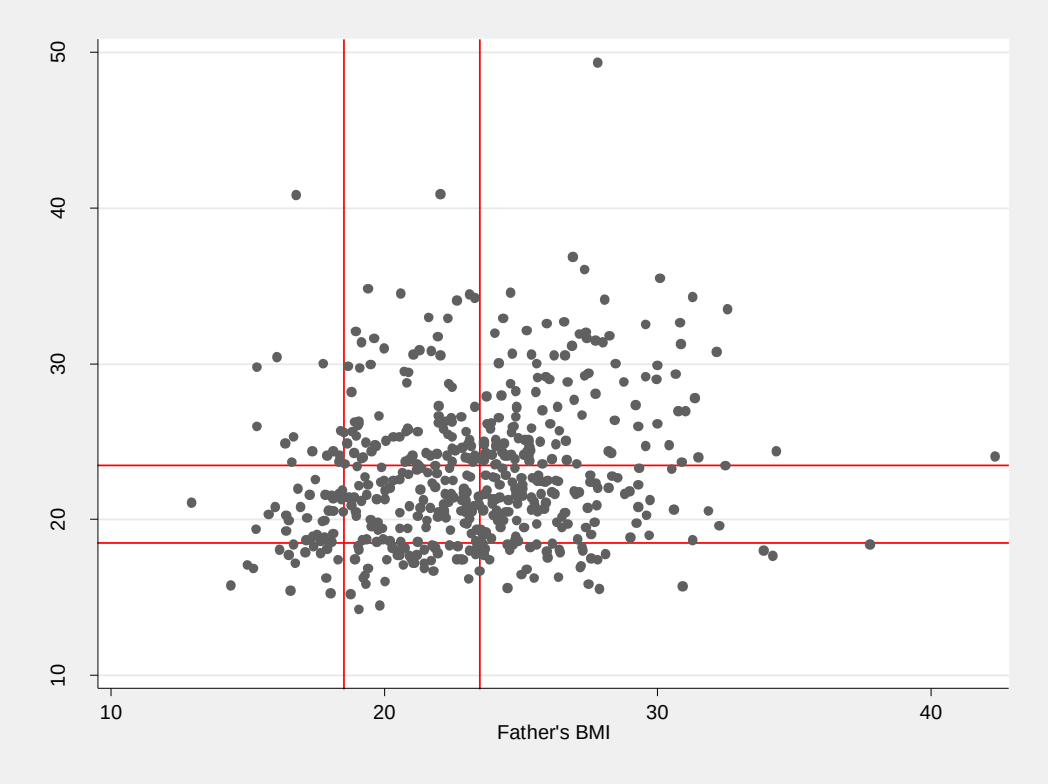
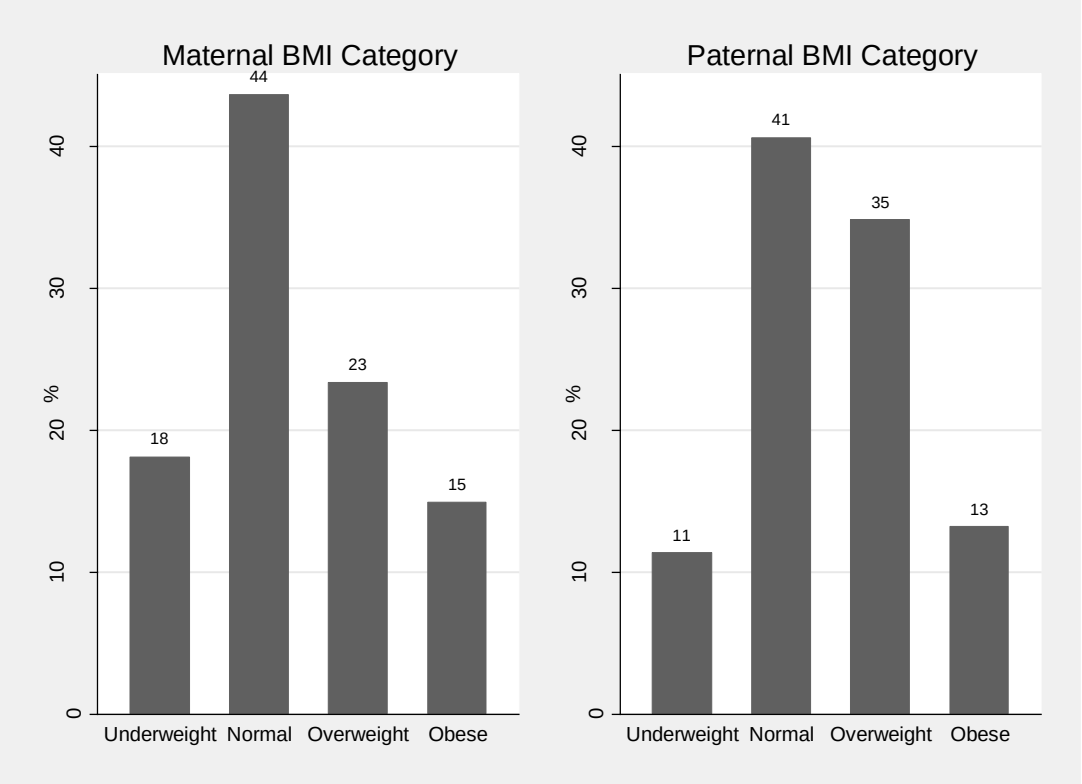


Figure 5.12 Scatter plot of maternal and paternal BMI



The proportions of individuals categorized as underweight (BMI ≤18.5), normal (BMI > 18.5 but <23.5), or obese (BMI ≥27.5) were higher among mothers, but overweight (BMI ≥23.5 but <27.5) was more common among fathers (Figure 5.13).

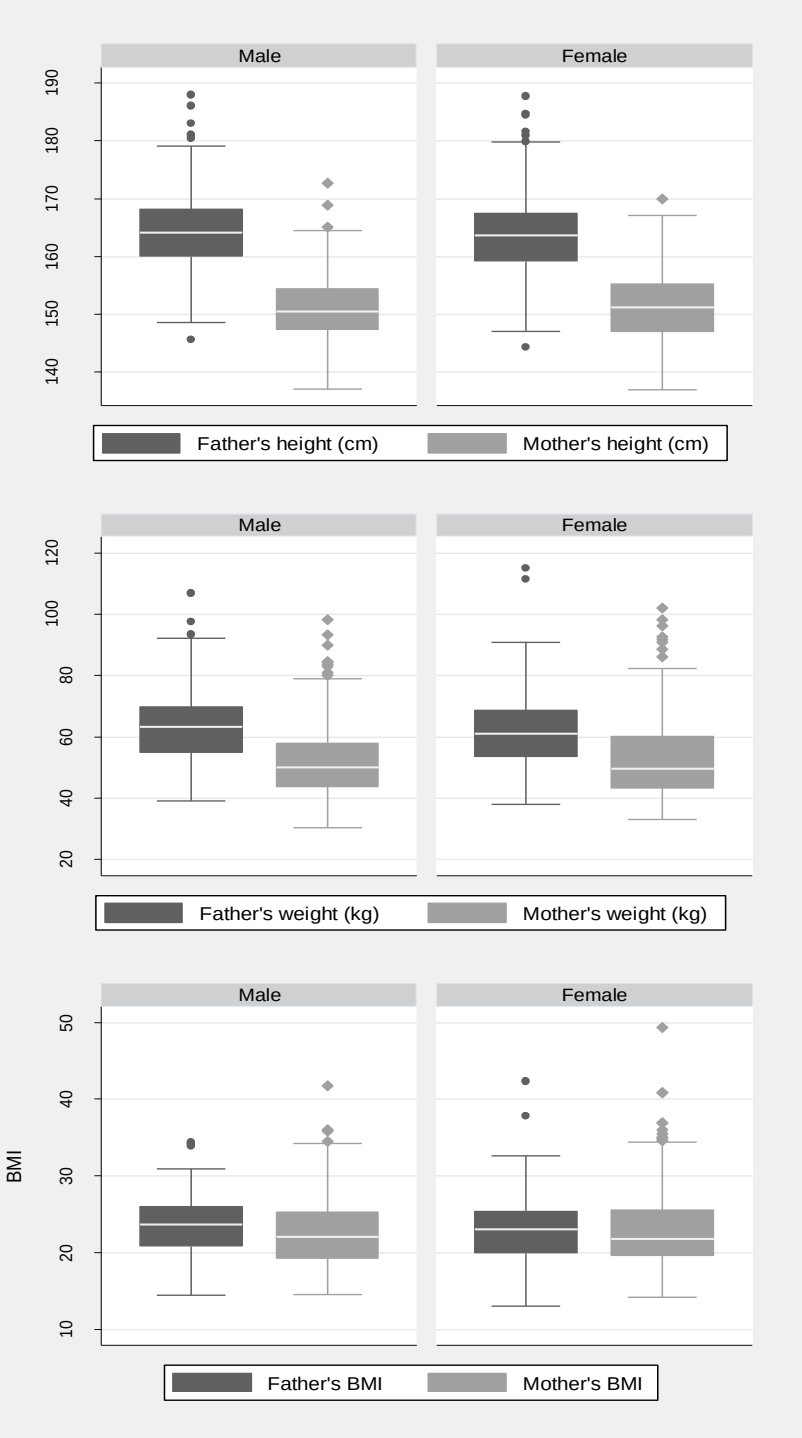
Figure 5.13 Proportion of underweight, normal, overweight, and obese parents



The numbers of observations in some cells of a cross-tabulation of maternal and paternal BMI with four groups were too small to carry out a chi-squared test, so I collapsed the overweight and obese categories. Chi-squared tests showed that maternal and paternal BMI categories were correlated when using three categories (p for trend 0.01), as well as binary categories using 23.5 as a cut-off (p= 0.009). Overweight women were more likely to have overweight husbands (OR 1.59; 95%CI 1.12, 2.27).

Among 522 pairs of parents, in 180 (34%) neither parent was overweight, in 134 (26%) only the father was overweight, in 95 (18%) only the mother was overweight, and in 113 (22%) both parents were categorized as overweight. There was little evidence of difference in maternal or paternal height (p-values 0.4339 and 0.3287), weight (p-values 0.1252 and 0.5781) or BMI (p-values 0.1713 and 0.8019) by infant sex in Wilcoxon rank sum tests (Figure 5.14). Kruskal-Wallis tests for trend in BMI categories (underweight, normal, overweight, and obese) did not show any differences by infant sex for maternal (p= 0.5005) or paternal status (p= 0.1637). Chi-squared tests showed that infant sex was not related to maternal (p= 0.519) or paternal (p= 0.077) overweight (BMI ≥23.5).

Figure 5.14 Box-and-whiskers plots of maternal and paternal anthropometry by infant sex



Maternal age over 25 years was associated with maternal height (p= 0.0058), weight (p<0.0001), BMI (p<0.0001), BMI category (p-value for trend 0.0001), and overweight status (p<0.0001), but paternal age (over 30 years) did not show associations with any paternal anthropometric markers (all p-values were >0.05).

There was some evidence that maternal height was associated with maternal education (Wilcoxon rank-sum test p= 0.0415), but not with any other SEP variable. Maternal weight was associated with household asset quintile, access to piped water, use of a shared toilet, and more than four children in the household (all p <0.05). Maternal BMI, BMI category, and overweight status were all associated with household asset quintile, access to piped water, and use of a shared toilet (all p <0.05).

Similarly, paternal height was associated with paternal education, smoking, use of a shared toilet, and households with four or more children. Paternal weight, BMI, BMI category, and overweight were associated with access to piped water, use of a shared toilet, paternal smoking, and household asset quintile. The ordinal variable encoding four parental overweight groups (neither, mother only, father only, both) was associated with access to piped water (p= 0.003), use of a shared toilet (p= 0.037), household asset quintile (p= 0.004), paternal age over 30 years (p= 0.002), and maternal age over 25 years (p= 0.001).

In multivariable multinomial regression analysis adjusted for all background factors as well as parental height internal z-scores, children whose parents were both overweight were more likely to be from the top three asset quintiles and from households with access to piped water (relative risk ratio 2.29; 95%CI 1.3, 4.1) compared to those with neither parent categorized as overweight (Table 5.5).

Table 5.5 Baseline determinants of parental overweight (BMI≥23.5) in a fully-adjusted multinomial regression model

	Overweight father		Overweight mother		Both parents overweight	
Covariate	**RRR (95%CI)**	**p**	**RRR (95%CI)**	**p**	**RRR (95%CI)**	**p**
Female	0.74 (0.5, 1.2)	0.205	1.66 (1.0, 2.9)	0.070	1.17 (0.7, 2.0)	0.564
Maternal age ≥25	1.24 (0.7, 2.3)	0.508	2.47 (1.2, 5.0)	0.012	3.60 (1.8, 7.0)	<0.0001
Paternal age ≥30	0.65 (0.4, 1.2)	0.169	0.98 (0.5, 1.9)	0.950	1.13 (0.6, 2.2)	0.705
Maternal height z-score	1.06 (0.8, 1.4)	0.640	0.98 (0.7, 1.3)	0.903	1.37 (1.1, 1.8)	0.020
Paternal height z-score	0.96 (0.7, 1.2)	0.756	1.39 (1.1, 1.8)	0.021	0.64 (0.5, 0.8)	0.002
Maternal education ≥6th standard	0.73 (0.4, 1.3)	0.271	1.08 (0.6, 2.0)	0.825	0.84 (0.4, 1.6)	0.571
Paternal education ≥6th standard	1.30 (0.8, 2.2)	0.329	0.77 (0.4, 1.4)	0.400	0.67 (0.4, 1.2)	0.182
Asset quintile (ref lowest)						
Second lowest	0.73 (0.4, 1.5)	0.379	0.55 (0.2, 1.3)	0.171	2.03 (0.8, 4.9)	0.114
Middle	1.53 (0.7, 3.2)	0.252	1.96 (0.9, 4.4)	0.102	3.18 (1.2, 8.2)	0.017
Second highest	1.13 (0.5, 2.5)	0.757	1.48 (0.6, 3.6)	0.386	5.15 (2.0, 13.4)	0.001
Highest	0.89 (0.4, 2.2)	0.794	0.86 (0.3, 2.5)	0.784	5.09 (1.8, 14.6)	0.003
Access to piped water	1.96 (1.2, 3.3)	0.009	1.59 (0.9, 2.8)	0.116	2.29 (1.3, 4.1)	0.005
Use of shared toilet	0.63 (0.3, 1.4)	0.238	0.50 (0.2, 1.2)	0.104	0.65 (0.3, 1.4)	0.280
≥4 children in the household	1.20 (0.7, 2.1)	0.523	1.17 (0.6, 2.2)	0.616	0.93 (0.5, 1.7)	0.810
≥2 adults in the household	0.59 (0.3, 1.0)	0.061	0.76 (0.4, 1.4)	0.392	0.82 (0.5, 1.5)	0.507
Planned pregnancy	1.11 (0.8, 1.5)	0.494	1.25 (0.9, 1.8)	0.209	1.19 (0.8, 1.7)	0.308
Paternal smoking	0.88 (0.5, 1.4)	0.617	1.43 (0.8, 2.6)	0.238	0.59 (0.3, 1.0)	0.056
Maternal smoking	1.07 (0.5, 2.2)	0.855	1.08 (0.5, 2.4)	0.843	1.72 (0.8, 3.6)	0.157

Note: RRR, relative risk ratio. Reference group: neither parent overweight

5.5.8 Missing data in background and parental characteristics

5.5.8.1 Proportion of missing data for each variable

Infant sex was the most complete variable, with no missing data. Data on most sociodemographic variables were complete for 99% of participants. However, data on household composition, home ownership, source of water supply, and initiation of breastfeeding were missing for 3% of participants. Birthweight data were missing for 7% of infants. The most incomplete variables were for the Edinburgh Postnatal Depression Scale (15% missing data in each item).

Parental anthropometric data were most incomplete for paternal height and weight, with 45% values missing. Maternal height (30%) and weight (29%) data had fewer missing values (Table 5.6).

Table 5.6 Proportion of missing data for parental anthropometry variables

	Missing		**Observed**		**Total**
Variable	**n**	**%**	**n**	**%**	
Father's height	441	45	537	55	978
Father's weight	441	45	537	55	978
Mother's height	289	30	689	70	978
Mother's weight	288	29	690	71	978

5.5.8.2 Proportion of incomplete cases

Three-quarters of infants had complete information on all core baseline survey variables, and only 2% were missing data on more than ten variables (Table 5.7).

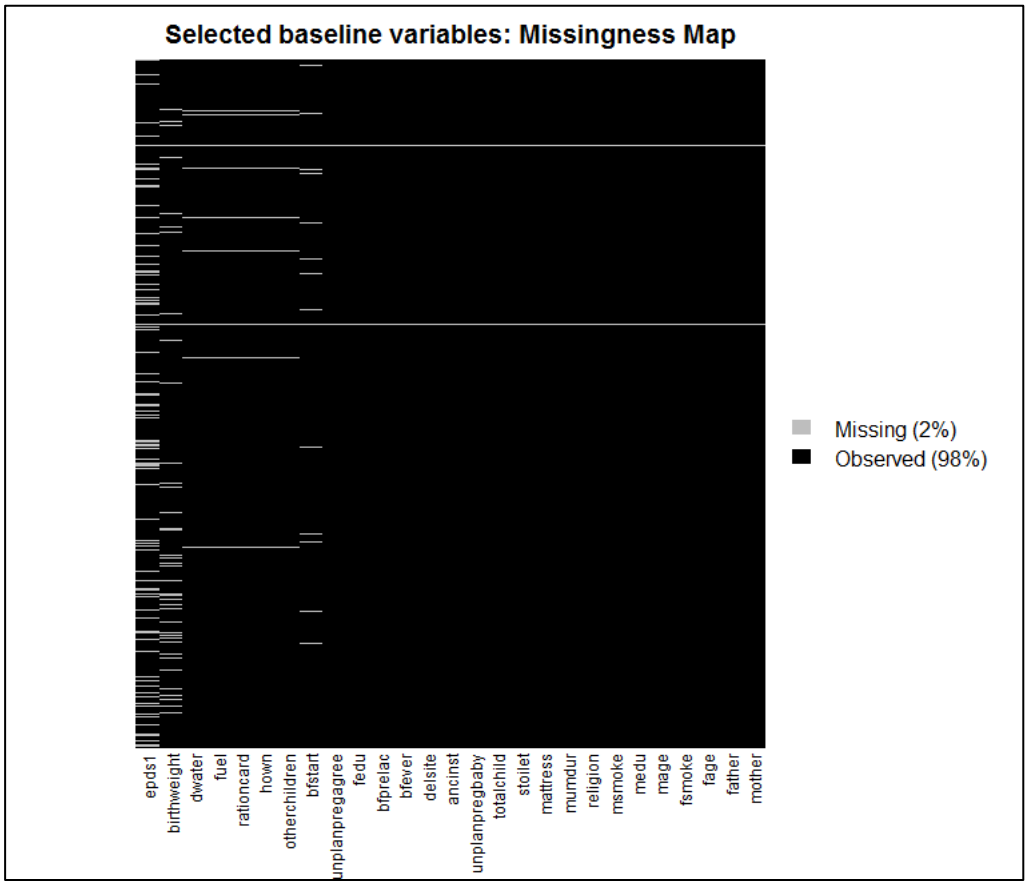
Table 5.7 Pattern of missing values for background variables

Pattern (number of variables with missing values)	**Number of infants**	**%**
Complete cases	743	76
1	67	7
2	2	0
10	143	15
11	12	1
20	4	0
70	2	0
80	5	1
Total	**978**	**100**

The amount of missing data across the baseline survey was low. In a set of 27 variables for 978 children which covered key background and parental

characteristics, 2% of values were missing (Figure 5.15), and much of this was for EPDS and birthweight.

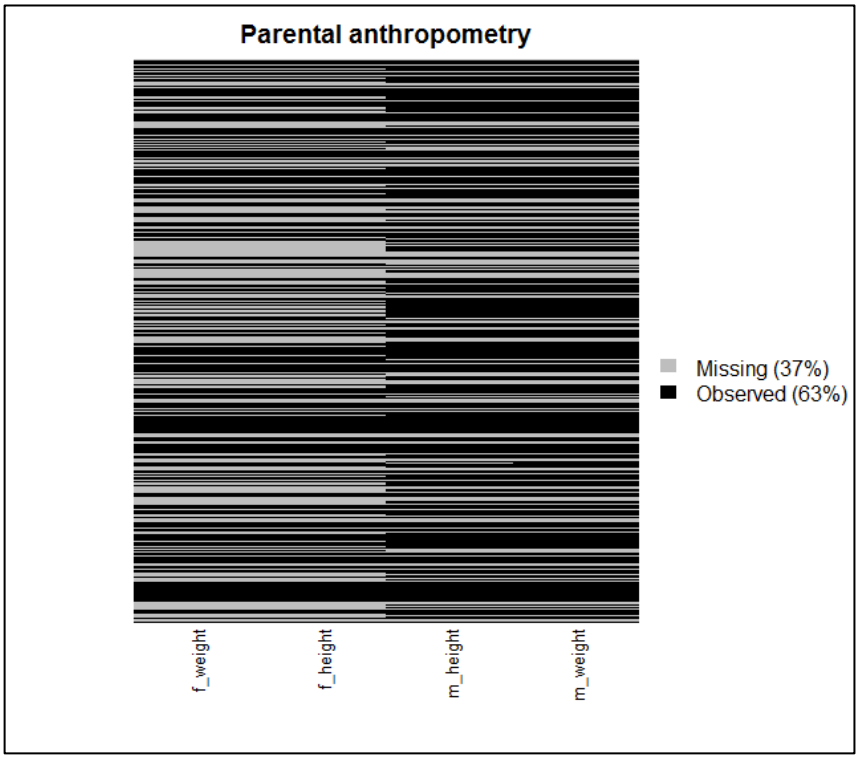
Figure 5.15 Missingness map of selected background and parental variables



Note: y axis: child ID, x axis: variable

The proportion of missing data was higher for parental anthropometry (Figure 5.16), with 28% of infants missing heights and weights of both parents. Only 521 infants (53%) had data for paternal and maternal heights and weights. A higher proportion (17%) were missing only paternal data than those missing only maternal data (2%).

Figure 5.16 Missingness map of maternal and paternal heights and weights



In multivariable analyses of relationships between response to parental anthropometry and baseline variables, none (maternal, paternal, or both parents' anthropometry) had odds ratios for non-response below 0.5 or greater than 2.0 (Table 5.8), indicating that the bias introduced due to self-selection for parental anthropometry was unlikely to be substantial.

Table 5.8 Determinants of missing parental anthropometry data

Covariate	Maternal anthropometry		Paternal anthropometry		Parental anthropometry	
	aOR (95%CI)	p	aOR (95%CI)	p	aOR (95%CI)	p
Female	0.80 (0.6, 1.1)	0.1382	1.13 (0.9, 1.5)	0.3729	1.08 (0.8, 1.4)	0.5642
Maternal age ≥25	0.74 (0.5, 1.1)	0.1302	0.69 (0.5, 1.0)	0.0350	0.73 (0.5, 1.0)	0.0809
Paternal age ≥30	0.73 (0.5, 1.1)	0.1164	0.94 (0.7, 1.3)	0.7456	0.89 (0.6, 1.3)	0.5181
Maternal education ≥6th standard	1.14 (0.8, 1.6)	0.4371	1.19 (0.9, 1.6)	0.2660	1.17 (0.9, 1.6)	0.3193
Paternal education ≥6th standard	1.06 (0.8, 1.5)	0.7280	0.97 (0.7, 1.3)	0.8592	0.96 (0.7, 1.3)	0.7719
Highest asset quintile	0.78 (0.5, 1.2)	0.2482	0.86 (0.6, 1.3)	0.4430	0.87 (0.6, 1.3)	0.4847
Access to piped water	0.95 (0.7, 1.3)	0.7335	0.99 (0.7, 1.3)	0.9316	0.99 (0.7, 1.3)	0.9639
Use of shared toilet	1.01 (0.7, 1.6)	0.9570	0.93 (0.6, 1.4)	0.7299	0.91 (0.6, 1.3)	0.6427
≥2 adults in the household	1.08 (0.8, 1.5)	0.6303	1.37 (1.0, 1.8)	0.0405	1.35 (1.0, 1.8)	0.0507
≥4 children in the household	0.82 (0.6, 1.2)	0.2632	0.94 (0.7, 1.3)	0.6701	0.88 (0.6, 1.2)	0.4009
Paternal smoking	0.86 (0.6, 1.2)	0.3418	0.84 (0.6, 1.1)	0.2263	0.85 (0.6, 1.1)	0.2518
Maternal smoking	1.00 (0.6, 1.6)	0.9855	0.96 (0.6, 1.4)	0.8415	0.97 (0.6, 1.4)	0.8798
Planned pregnancy	1.52 (1.1, 2.1)	0.0087	1.37 (1.0, 1.8)	0.0273	1.40 (1.1, 1.8)	0.0184

5.6 Follow-up and attrition

5.6.1 Follow-up time

The total study-time of follow-up recorded in the study was 20,042 months between March 2013 and April 2016, with children contributing between 0.1 and 36.4 months. The estimated median duration for which children were followed-up was 26.2 months (95%CI 25.3, 26.8). The total person-time of follow-up represented 16,711 child-months over the first two years of life, with duration ranging between 0.03 and 24.9 months. The estimated median age up to which children were followed up was 23.9 months (95%CI 23.6, 24.0).

There was some evidence of differences in study-time of follow up by maternal age, household asset quintile, and pregnancy intention. Children of older women, those in higher asset quintiles, and those born as a result of unplanned or ambivalent pregnancies were more likely to still be in active follow-up at each age (Table 5.9 and Figure 5.17). In relation to person-time of follow up, there was strong evidence of differences by maternal and paternal age categories (Table 5.10 and Figure 5.18). However, there was weak evidence of an association with asset quintile or type of toilet facility. In both sets, the absolute difference in median follow-up times between groups was greatest for household wealth quintiles (lowest vs highest).

Table 5.9 Duration and equality of follow-up times by baseline characteristics

Variable (Category)	**Study-time**		**Person-time**	
	Median follow-up (months)	Log-rank p	Median follow-up (months)	Log-rank p
Sex	26.2	0.3872	23.9	0.4749
Male	25.9		23.8	
Female	26.4		23.9	
Maternal age	26.4	0.0017	23.9	<0.0001
<25 years	24.9		23.0	
≥25 years	27.5		24.2	
Paternal age	26.4	0.1346	23.9	0.0011
<30 years	25.2		23.4	
≥30 years	27.4		24.1	
Maternal education	26.4	0.3309	23.9	0.2552
Below 6th standard	26.4		24.0	
6th standard and above	26.1		23.8	
Paternal education	26.4	0.3318	23.9	0.2534
Below 6th standard	26.4		23.9	
6th standard and above	26.2		23.9	
Household asset quintile*	26.4	0.0086	23.9	0.0791
Lowest	23.8		22.6	
Second lowest	25.4		23.7	
Middle	26.7		24.1	
Second highest	27.1		24.0	
Highest	27.1		24.1	
Access to piped water	26.4	0.1447	23.9	0.2351
No	25.6		23.7	
Yes	26.5		23.9	
Use of shared toilet	26.4	0.6768	23.9	0.0703
No	27.1		24.2	
Yes	26.1		23.9	
Adults in the household	26.2	0.5118	23.9	0.9299
<2 adults	26.2		23.9	
≥2 adults	26.2		23.9	
Children in the household	26.2	0.1556	23.9	0.0893
<4 children	25.4		23.6	
≥4 children	27.4		24.1	
Paternal smoking	26.4	0.4654	23.9	0.8354
No	26.6		23.9	
Yes	26.1		23.9	
Maternal smoking	26.4	0.2776	23.9	0.3366
No	26.2		23.9	
Yes	26.6		23.9	
Pregnancy intention	26.4	0.0139	23.9	0.2262
Unplanned or ambivalent	27.4		24.0	
Planned	25.4		23.8	

Note: * p-value for Log-rank test for trend

Figure 5.17 Study time by baseline characteristics

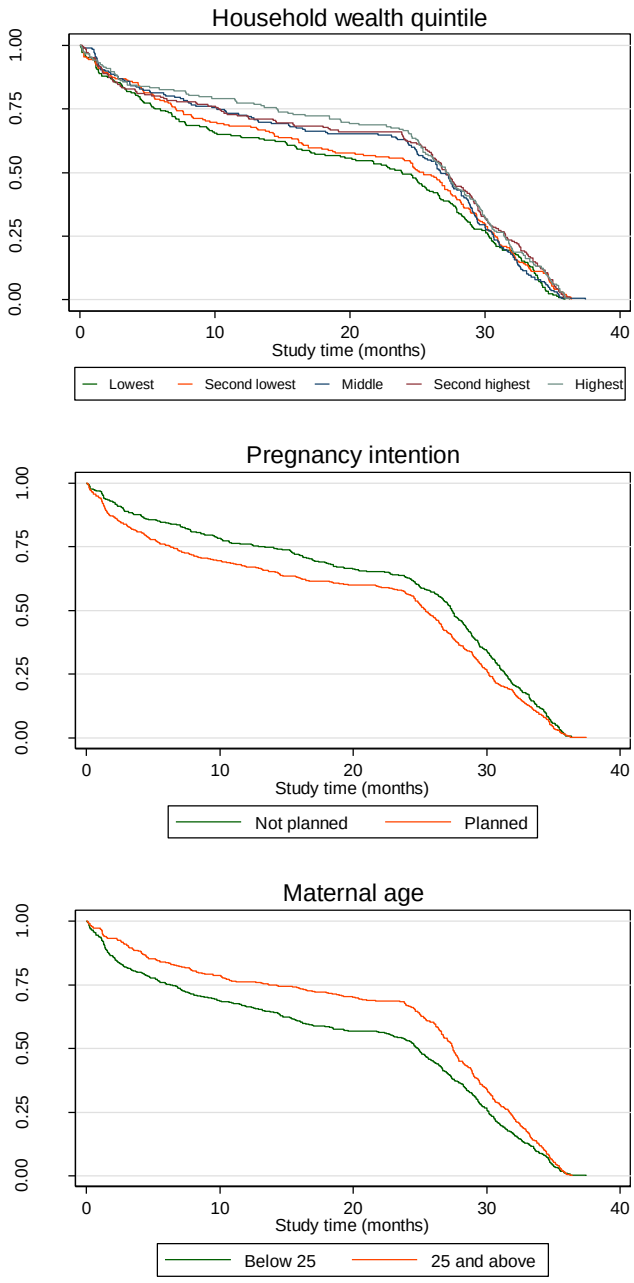
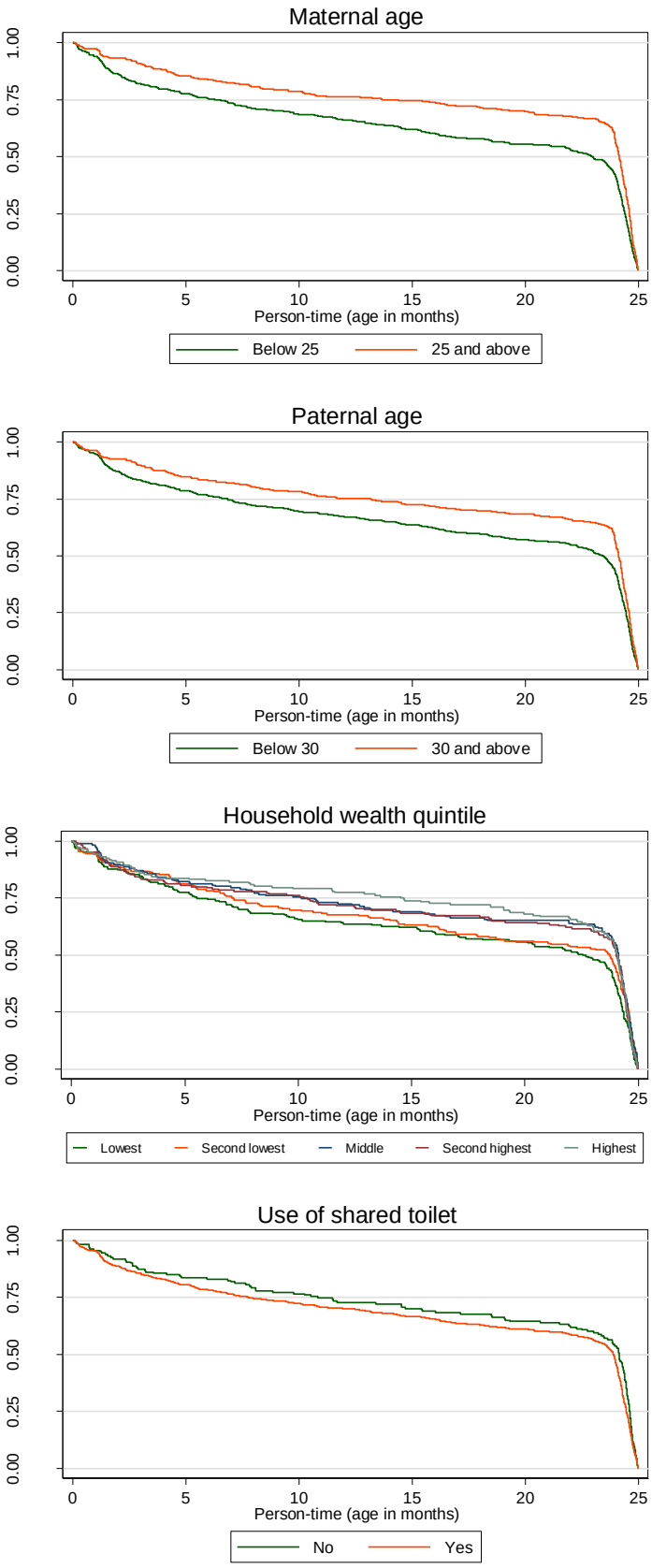


Figure 5.18 Person-time by baseline characteristics



5.6.2 Attrition

Investigators closed 350 (36%) cases during the follow-up period, 90% of whom were children whose families moved out of the study site. Thirteen infants (4%) died during the study, 16 cases (5%) were closed because the parent(s) no longer consented to participation, and five (1%) were closed because investigators failed to re-establish contact after three months.

My analysis of dropout showed that, by 24 months, 371 infants (38%) had been lost to follow-up, i.e., they had not completed a single follow-up visit between 18 and 24 months. Dropout and case closure were strongly correlated (chi-squared $p < 0.0001$). Ninety percent of those who dropped out also had their records closed by field investigators. Similarly, 97% of children who did not drop out had open records at two years. Both case closure and dropout were associated with younger maternal age and negatively with the highest asset quintile in multivariable analyses (Table 5.10). However, the magnitudes of association were between 0.5 and 2.0, indicating that the bias introduced into analyses restricted to those who did not drop out or have their cases closed would not be substantial.

Table 5.10 Associations of baseline characteristics with case closure during the follow-up period and dropout before 18 months

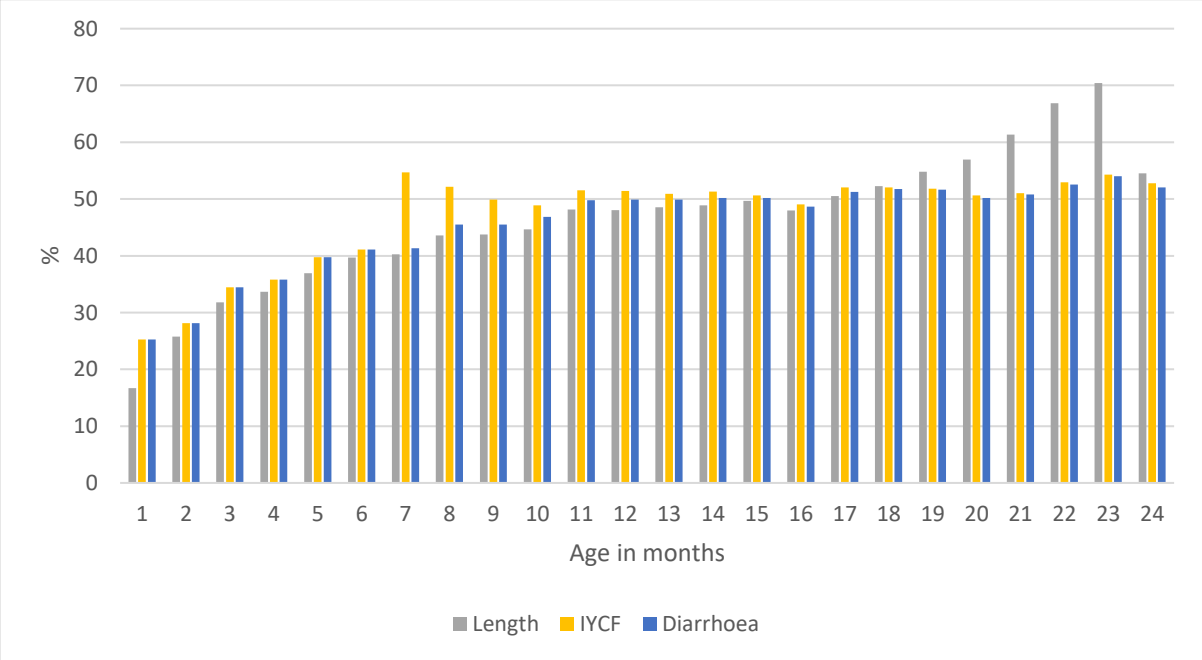
Variable	Case closure				Dropout			
	Univariable		Fully-adjusted		Univariable		Fully-adjusted	
	OR (95%CI)	**p**	**OR (95%CI)**	**p**	**OR (95%CI)**	**p**	**OR (95%CI)**	**p**
Female	0.95 (0.7, 1.2)	0.716	0.91 (0.7, 1.2)	0.487	0.91 (0.7, 1.1)	0.463	0.84 (0.6, 1.1)	0.215
Maternal age ≥25	0.59 (0.4, 0.8)	<0.0001	0.67 (0.5, 1.0)	0.033	0.55 (0.4, 0.7)	<0.0001	0.63 (0.4, 0.9)	0.013
Paternal age ≥30	0.66 (0.5, 0.9)	<0.001	0.87 (0.6, 1.3)	0.458	0.65 (0.5, 0.8)	0.002	0.97 (0.7, 1.4)	0.865
Maternal education ≥6th standard	1.16 (0.9, 1.5)	0.253	1.18 (0.9, 1.6)	0.299	1.26 (0.9, 1.6)	0.082	1.19 (0.9, 1.6)	0.265
Paternal education ≥6th standard	1.01 (0.7, 1.3)	0.940	1.01 (0.7, 1.4)	0.966	1.03 (0.8, 1.3)	0.828	1.02 (0.8, 1.4)	0.896
Highest asset quintile	0.57 (0.4, 0.8)	0.003	0.57 (0.4, 0.9)	0.010	0.63 (0.4, 0.9)	0.013	0.59 (0.4, 0.9)	0.014
Access to piped water	0.78 (0.6, 1.0)	0.075	0.86 (0.6, 1.1)	0.301	0.76 (0.5, 1.0)	0.046	0.79 (0.6, 1.1)	0.111
Use of shared toilet	1.42 (0.9, 2.1)	0.060	1.13 (0.7, 1.7)	0.555	1.33 (0.9, 1.9)	0.113	1.05 (0.7, 1.6)	0.819
≥2 adults in the household	0.88 (0.7, 1.1)	0.326	0.93 (0.7, 1.3)	0.659	0.97 (0.7, 1.3)	0.828	1.08 (0.8, 1.5)	0.623
≥4 children in the household	0.74 (0.6, 0.9)	0.038	0.94 (0.7, 1.3)	0.700	0.66 (0.5, 0.9)	0.004	0.81 (0.6, 1.1)	0.205
Paternal smoking	0.96 (0.7, 1.2)	0.758	0.93 (0.7, 1.2)	0.613	1.03 (0.8, 1.3)	0.770	1.05 (0.8, 1.4)	0.731
Maternal smoking	1.02 (0.7, 1.5)	0.922	1.10 (0.7, 1.7)	0.642	0.88 (0.6, 1.3)	0.524	0.98 (0.6, 1.5)	0.914
Planned pregnancy	1.39 (1.1, 1.8)	0.019	1.22 (0.9, 1.6)	0.176	1.38 (1.1, 1.8)	0.020	1.21 (0.9, 1.6)	0.189

5.7 Study patterns of missing longitudinal data over follow-up period

5.7.1 Non-response

The proportion of non-response increased with age. At one month, 17% of children were missing length data. The proportion increased to 48% at 12 months (Figure 5.19). While non-response to length was particularly high at 23 months (70%), at 24 months it was much lower (53%). The proportion of non-response to diarrhoea was similar to that for IYCF in most months, except between seven and nine months when there was a higher proportion of non-response to IYCF.

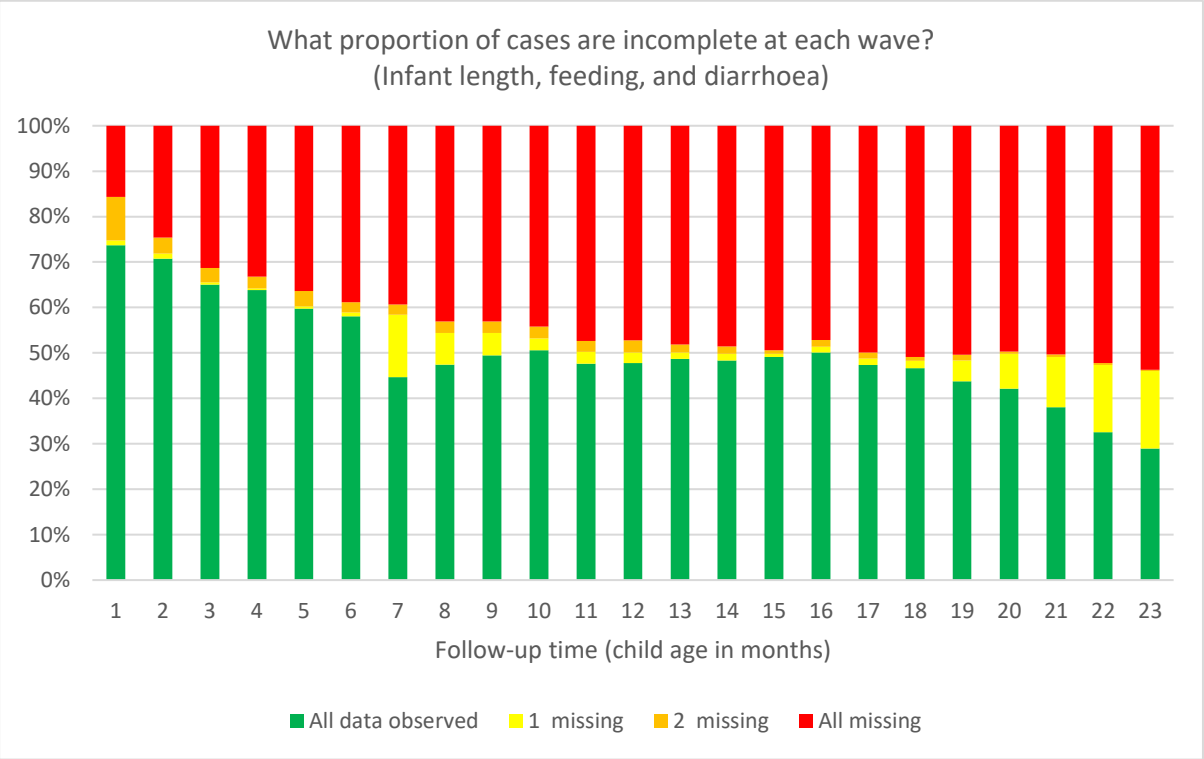
Figure 5.19 Non-response to length, IYCF, and diarrhoea at follow-up visits up to 2 years



The proportion of incomplete cases with respect to length, IYCF, and diarrhoea, i.e. those missing data on one, two, or all three variables, was highest at 23 months. While 74% of children had complete information at 1 month, at 23 months the figure was 29% (I did not compute case completeness at 24 months since I only used IYCF data up to 23 months). However, due to the increase in response to anthropometry at 24 months, the proportion of complete cases at 24 months was 45%. The proportion missing data for one or two indicators (i.e., partially incomplete cases) did not exceed 20% in any month, indicating that wave non-response was generally in relation to all three variables (unit non-response) rather than selective non-response to any one component in a wave (item non-response).

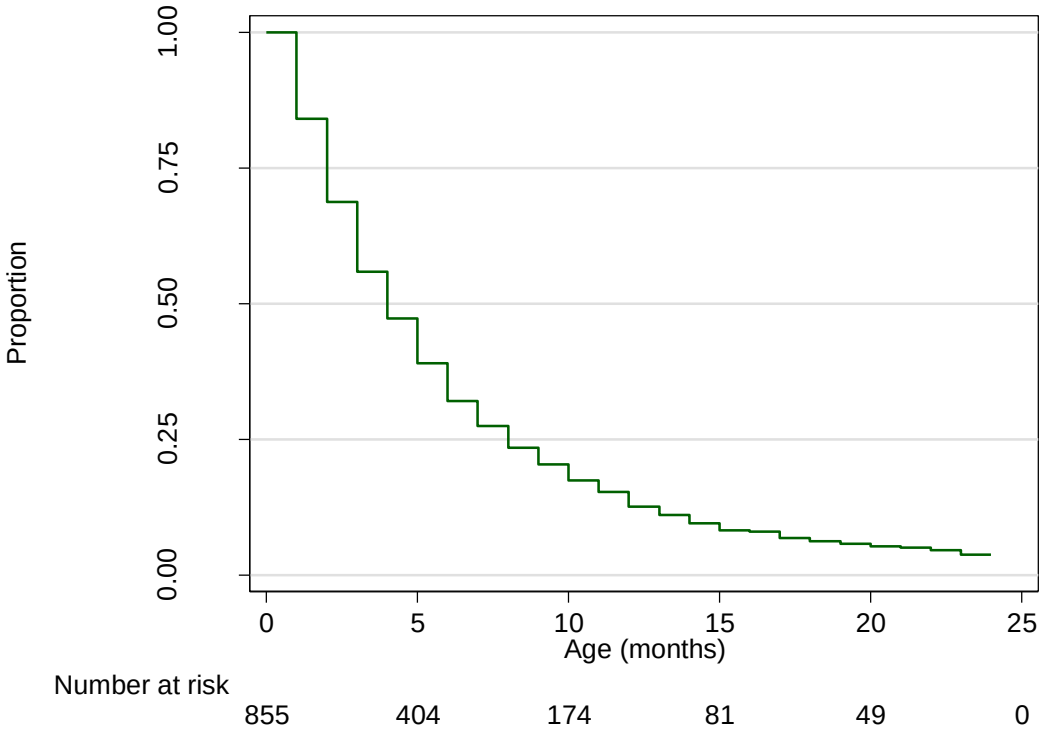
At 7 months of age, over 50% had some missing data for a follow-up visit, and at the 23 month visit 71% of cases were incomplete (Figure 5.20).

Figure 5.20 Proportion of incomplete cases at each age (1-23 months)



At four months, over 50% of the cohort already had some missing data for a follow-up visit (Figure 5.21).

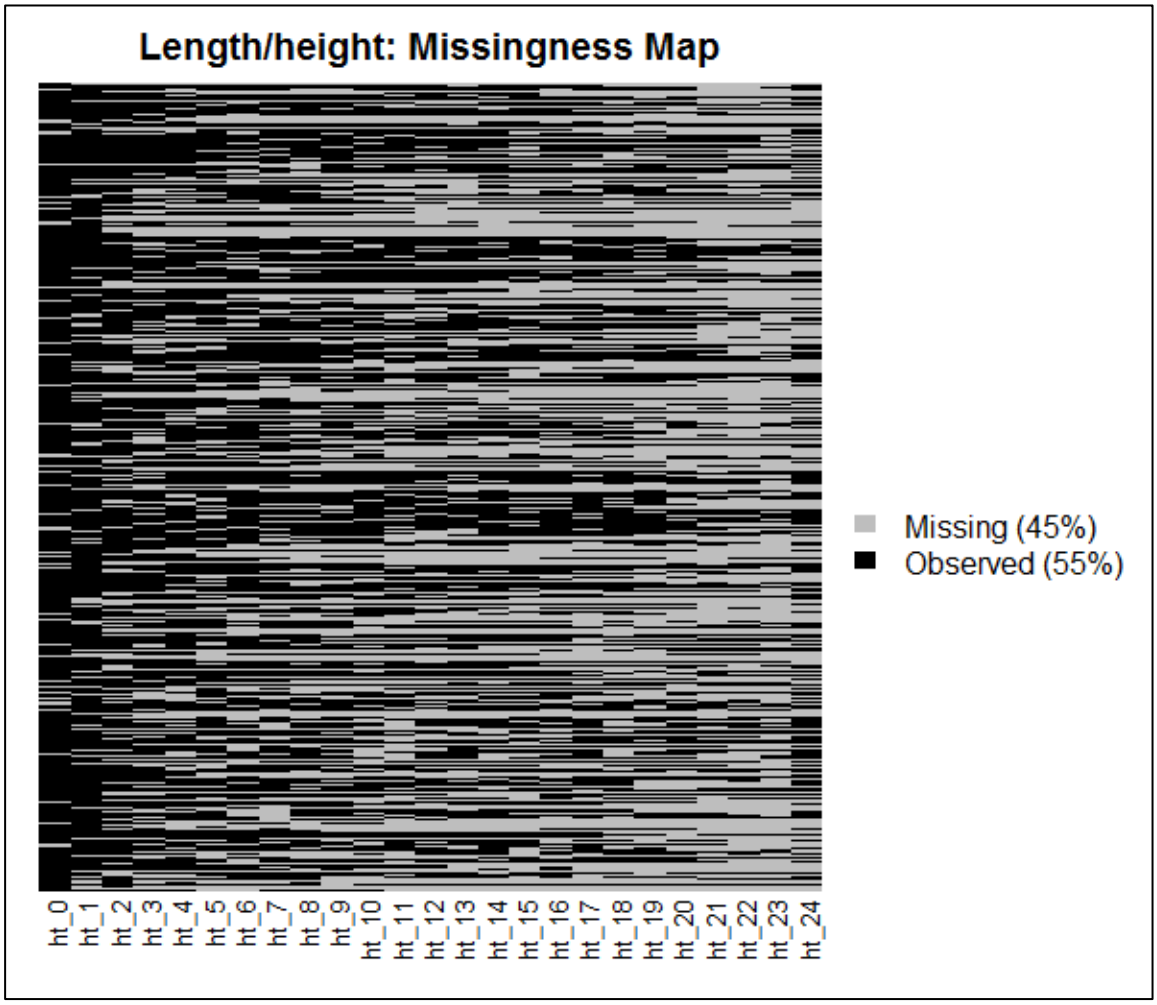
Figure 5.21 Survival curve of age at which participants first had missing data for a follow-up visit



5.7.2 Missing value patterns

Graphs of missing value patterns displaying the amount of missing information for each child up to 24 months showed that there were many children who were successfully contacted after non-response at one or more previous waves. However, there were many who had no data for a large part of the follow-up period. For graphs up to 24 months including all 978 children in the study, the proportion of missing values was 45% for length measurements, including the first one at the baseline visit, (Figure 5.22), 47% for IYCF (Figure 5.23), and 46% for diarrhoea (Figure 5.24).

Figure 5.22 Length missingness map



Note: ht_0 to ht_24 denote length measurements from 0-24 months

Figure 5.23 IYCF missingness map

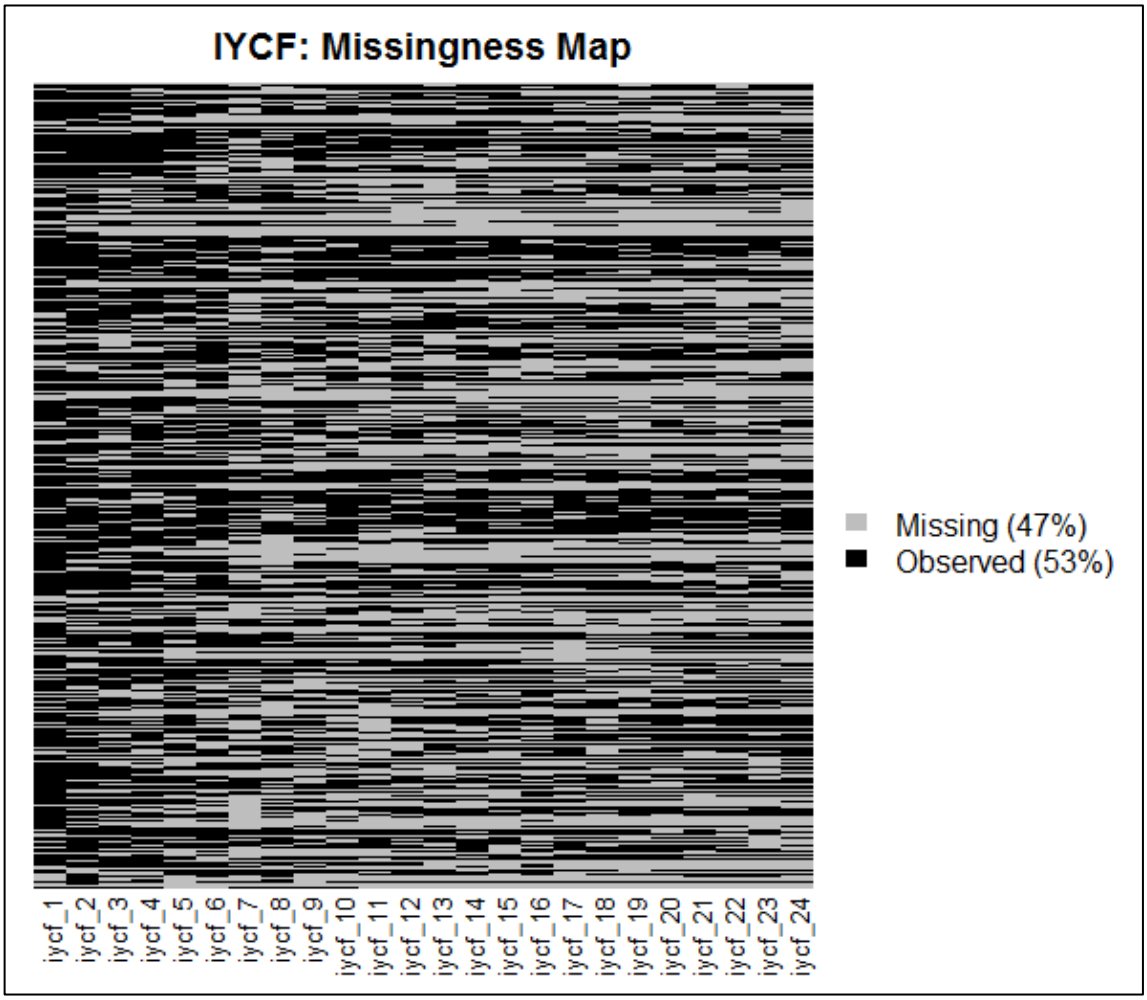
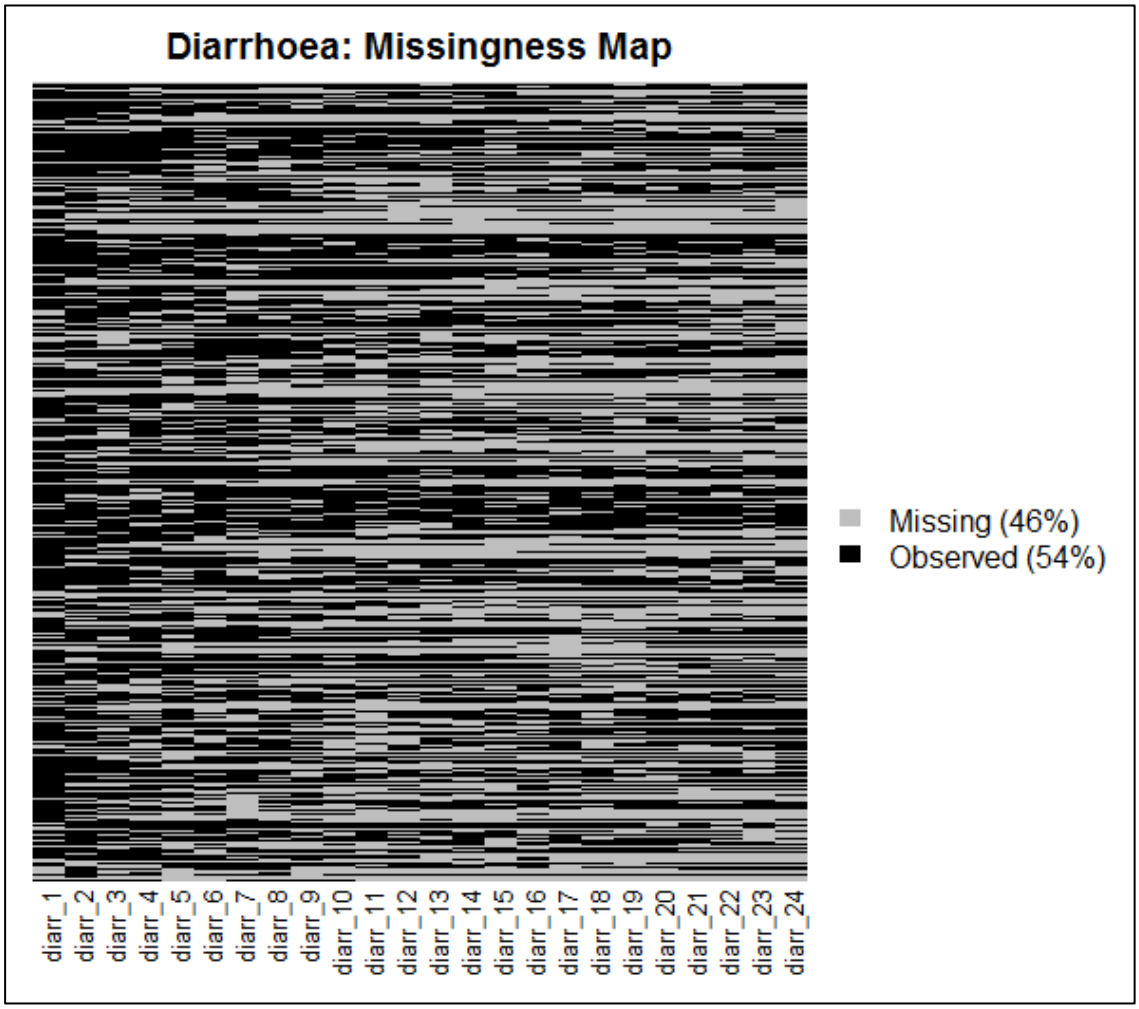


Figure 5.24 Diarrhoea missingness map



The missing value patterns in length measurements for those who were lost to follow-up by 18 months (Figure 5.25) were in stark contrast to the density of data among those who were still in the study at 24 months (Figure 5.26). Excluding those who dropped out would have led to greater completeness in length data, as 75% of expected observations were recorded for those who participated. However, among those who dropped out, 31% of expected observations were nonetheless available, indicating that they did not comprise only one group with little or no data available. Several participants who dropped out appeared to have a large number of longitudinal data points up to 18 months which would be sufficient for use in growth modelling based on unbalanced data. The difference in data availability between those who dropped out and those who did not was therefore not very meaningful despite large differences in the overall quantity of data points.

Based on these graphs, a non-monotone pattern of missing values is a realistic description of the cohort's missing data.

Figure 5.25 Missing data (length) map for children lost to follow up by 18 months

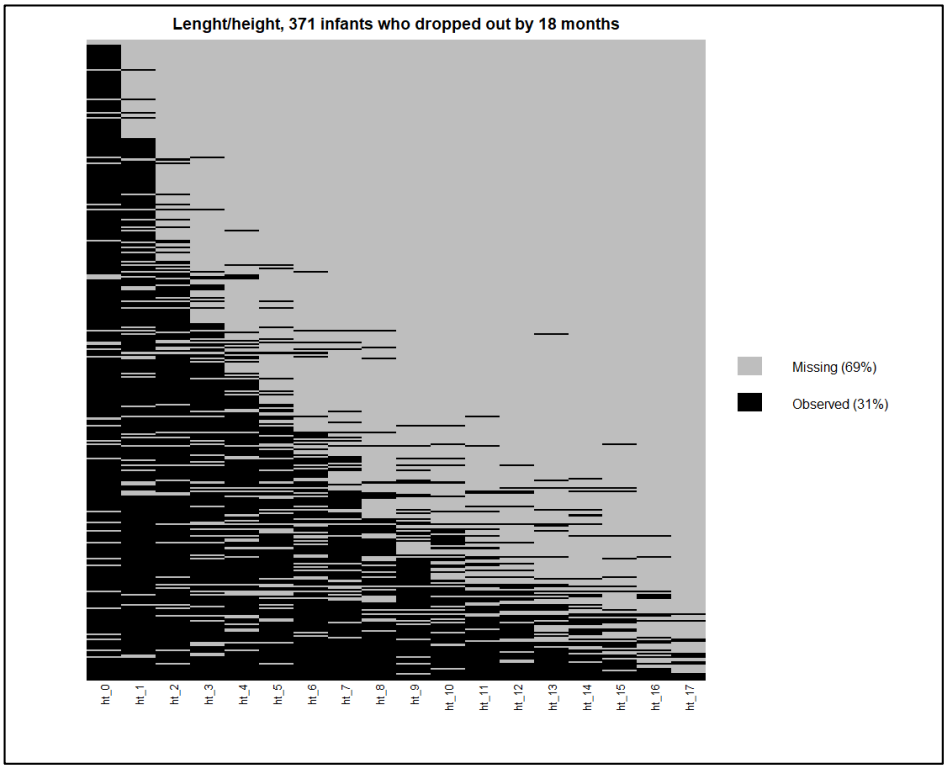
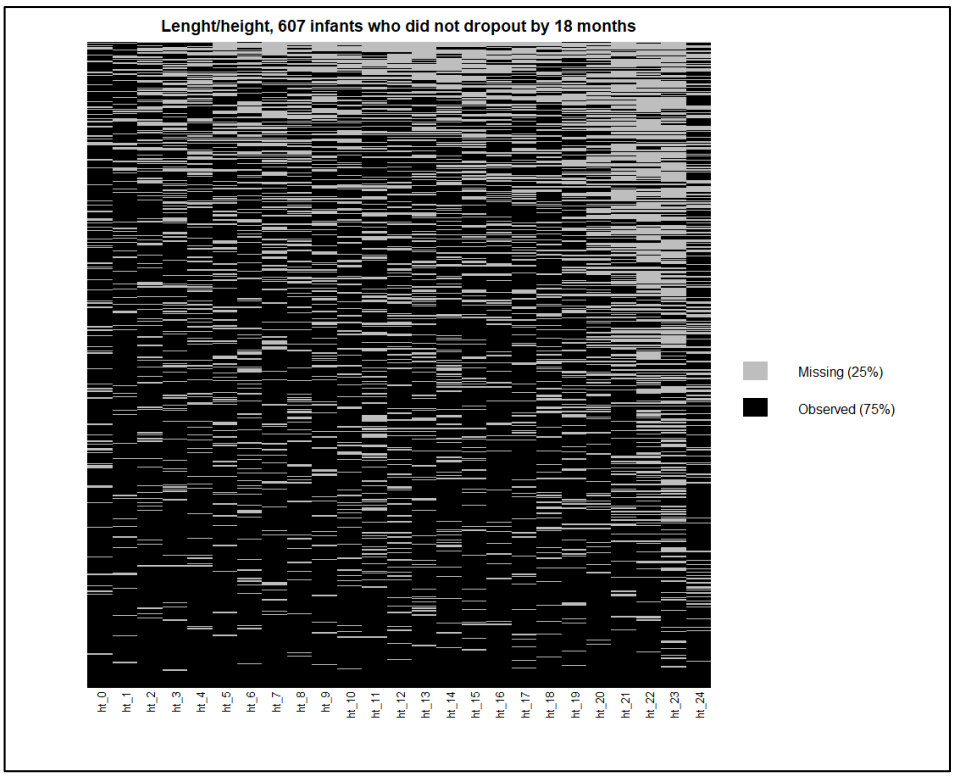


Figure 5.26 Missing data (length) map for children still in study at 24 months



5.7.3 Selection bias in wave non-response

In multivariable analyses regressing missingness on baseline variables at each age (0-24 months) for length, diarrhoea, and IYCF, there were a few instances of covariate-visit odds ratios below 0.5, but none greater than 2.0 (Table 5.11). Maternal age over 25 years was associated with lower odds of missing length measurements at 1 month (aOR 0.48; 95%CI 0.3, 0.8) and 2 months (aOR 0.49; 95%CI 0.3, 0.8). At 12 months, children in the highest asset quintile were less likely to have missing data for length (aOR 0.46; 95%CI 0.3, 0.7), IYCF (aOR 0.42; 95%CI 0.3, 0.6), as well as diarrhoea (aOR 0.43; 95%CI 0.3, 0.6). However, my analyses taken as a whole suggest that the intermittent non-response patterns observed in the cohort were not consistently non-negligible or induced by baseline factors across the study period.

Table 5.11 Multivariable analyses with instances of non-negligible selection bias in wave non-response (n=947)

Covariate	Length at 1 month		Length at 2 months		Length at 12 months		IYCF at 12 months		Diarrhoea at 12 months	
	aOR (95%CI)	p-value	aOR (95%CI)	p-value	aOR (95%CI)	p-value	aOR (95%CI)	p-value	aOR (95%CI)	p-value
Female	1.03 (0.7, 1.5)	0.879	0.84 (0.6, 1.1)	0.279	1.04 (0.8, 1.4)	0.749	1.00 (0.8, 1.3)	0.984	0.98 (0.8, 1.3)	0.881
Maternal age ≥25	0.48 (0.3, 0.8)	0.004	0.50 (0.3, 0.8)	0.001	0.80 (0.6, 1.1)	0.212	0.82 (0.6, 1.2)	0.270	0.79 (0.6, 1.1)	0.186
Paternal age ≥30	1.07 (0.7, 1.7)	0.794	1.23 (0.8, 1.9)	0.330	0.72 (0.5, 1.0)	0.063	0.77 (0.5, 1.1)	0.136	0.80 (0.6, 1.1)	0.201
Maternal education ≥6th standard	0.85 (0.6, 1.3)	0.427	1.10 (0.8, 1.6)	0.594	1.39 (1.0, 1.9)	0.033	1.26 (0.9, 1.7)	0.131	1.21 (0.9, 1.6)	0.209
Paternal education ≥6th standard	1.13 (0.7, 1.7)	0.551	1.55 (1.1, 2.2)	0.017	0.91 (0.7, 1.2)	0.537	0.82 (0.6, 1.1)	0.182	0.85 (0.6, 1.2)	0.301
Highest asset quintile	1.30 (0.8, 2.1)	0.307	1.28 (0.8, 2.0)	0.259	0.46 (0.3, 0.7)	<0.0001	0.43 (0.3, 0.6)	<0.0001	0.43 (0.3, 0.6)	<0.0001
Access to piped water	0.56 (0.4, 0.8)	0.004	0.62 (0.4, 0.9)	0.006	0.90 (0.7, 1.2)	0.455	0.94 (0.7, 1.3)	0.694	0.94 (0.7, 1.3)	0.682
Use of shared toilet	0.76 (0.5, 1.3)	0.290	1.31 (0.8, 2.1)	0.249	0.74 (0.5, 1.1)	0.125	0.62 (0.4, 0.9)	0.019	0.60 (0.4, 0.9)	0.011
≥2 adults in the household	1.47 (1.0, 2.2)	0.064	1.60 (1.1, 2.3)	0.008	1.07 (0.8, 1.4)	0.674	1.09 (0.8, 1.5)	0.554	1.05 (0.8, 1.4)	0.732
≥4 children in the household	0.79 (0.5, 1.2)	0.286	0.94 (0.7, 1.4)	0.742	1.00 (0.7, 1.4)	0.999	0.91 (0.7, 1.2)	0.559	0.93 (0.7, 1.3)	0.663
Paternal smoking	0.77 (0.5, 1.1)	0.162	1.16 (0.8, 1.6)	0.358	0.91 (0.7, 1.2)	0.487	0.90 (0.7, 1.2)	0.469	0.89 (0.7, 1.2)	0.399
Maternal smoking	0.74 (0.4, 1.4)	0.357	0.53 (0.3, 0.9)	0.026	1.00 (0.7, 1.5)	0.982	0.98 (0.7, 1.5)	0.917	0.91 (0.6, 1.4)	0.636
Planned pregnancy	1.24 (0.8, 1.8)	0.280	1.55 (1.1, 2.2)	0.010	1.39 (1.0, 1.8)	0.021	1.38 (1.0, 1.8)	0.024	1.43 (1.1, 1.9)	0.011

5.8 Analysis patterns of missing longitudinal data

The odds ratios for exclusion from analytic samples for longitudinal IYCF patterns by baseline characteristics (Chapter 7) were between 0.5 and 2.0 in multivariable analyses of the determinants of missingness. This suggests that any self-selection for inclusion in subsets of the cohort for breastfeeding (n=533), introduction to solids (n=550), or complementary feeding (n=746) based on baseline characteristics was unlikely to create substantial bias in estimates. For the mediation analysis reported in Chapter 8, which included the smallest analytic sample with 438 children, the findings were similar. Multivariable logistic regression of missingness showed little evidence of relationships between baseline factors and inclusion in analysis (Table 5.12).

Overall, my findings suggest that patterns of missing longitudinal data in analytic samples would not lead to confounding patterns that deviated from those observed in the overall cohort.

Table 5.12 Multivariable logistic regression of determinants of inclusion in analytic samples

Variable	Exclusive / predominant breastfeeding		Introduction to solid foods		Complementary feeding		IYCF and length (mediation analysis)	
	aOR (95%CI)	p-value	aOR (95%CI)	p-value	aOR (95%CI)	p-value	aOR (95%CI)	p-value
Female	0.79 (0.6, 1.0)	0.085	0.72 (0.6, 0.9)	0.017	0.81 (0.6, 1.1)	0.197	0.94 (0.7, 1.2)	0.667
Maternal age ≥25	0.78 (0.5, 1.1)	0.159	0.68 (0.5, 1.0)	0.032	0.70 (0.5, 1.1)	0.114	0.64 (0.5, 0.9)	0.013
Paternal age ≥30	0.84 (0.6, 1.2)	0.327	0.94 (0.7, 1.3)	0.720	0.92 (0.6, 1.4)	0.713	0.86 (0.6, 1.2)	0.377
Maternal education ≥6th standard	1.43 (1.1, 1.9)	0.021	1.30 (1.0, 1.8)	0.095	1.19 (0.8, 1.7)	0.346	1.29 (1.0, 1.7)	0.096
Paternal education ≥6th standard	1.03 (0.8, 1.4)	0.854	1.11 (0.8, 1.5)	0.489	1.06 (0.7, 1.5)	0.765	1.16 (0.9, 1.6)	0.337
Highest asset quintile	1.06 (0.7, 1.6)	0.753	0.76 (0.5, 1.1)	0.173	0.69 (0.4, 1.1)	0.145	0.71 (0.5, 1.1)	0.091
Access to piped water	0.70 (0.5, 0.9)	0.016	0.80 (0.6, 1.1)	0.124	0.74 (0.5, 1.0)	0.090	0.98 (0.7, 1.3)	0.869
Use of shared toilet	0.90 (0.6, 1.3)	0.601	1.02 (0.7, 1.5)	0.932	1.16 (0.7, 1.9)	0.566	0.98 (0.7, 1.5)	0.925
≥2 adults in the household	1.39 (1.0, 1.9)	0.033	1.16 (0.9, 1.6)	0.341	1.01 (0.7, 1.5)	0.937	1.25 (0.9, 1.7)	0.155
≥4 children in the household	1.01 (0.7, 1.4)	0.939	0.90 (0.7, 1.2)	0.514	0.88 (0.6, 1.3)	0.502	0.74 (0.5, 1.0)	0.051
Paternal smoking	1.00 (0.8, 1.3)	0.984	0.95 (0.7, 1.3)	0.714	0.79 (0.6, 1.1)	0.169	0.97 (0.7, 1.3)	0.852
Maternal smoking	0.60 (0.4, 0.9)	0.017	0.82 (0.5, 1.2)	0.342	1.08 (0.7, 1.8)	0.771	0.95 (0.6, 1.4)	0.816
Planned pregnancy	1.49 (1.1, 2.0)	0.006	1.32 (1.0, 1.8)	0.051	1.58 (1.1, 2.2)	0.011	1.20 (0.9, 1.6)	0.193

5.9 Discussion

5.9.1 Characteristics of the cohort

In this chapter I have described the main characteristics of the birth cohort's 978 index infants and their parents. The cohort participants were born in households that were comparable to those included in the original trial pre-intervention census (Shah-More et al., 2017) on several indicators. In the trial and cohort, similar proportions were born in a health facility (83% and 94%), used a shared or public toilet facility (88% and 83%), and had mothers who had completed secondary education (44% and 54%).

However, one major difference related to source of water supply. While only 7% of cohort families reported that they bought water from a private tanker service, 60% of trial households purchased water for daily use, implying that the cohort was drawn from a more restricted population with more secure water supply (or that this indicator was under-reported). During the course of the trial, purchase of water from private tankers decreased by 53% in this community (Shah-More et al., 2017). It is possible that the cohort was recruited after this change took place.

One cohort characteristic that is unusual for informal settlements is the proportion of women (99%) who reported that they were not engaged in paid work in response to questions on occupation. Female participation in economic activity is generally widespread among the urban poor, and it is possible that the low proportion is a result of under-reporting. The question was asked soon after women gave birth, and many who usually worked were unlikely to be working at the time. Posing the question a few months after birth could have led to a different result. However, this proportion is similar to the 96% for mothers of children under five in the pre-intervention census (Bentley et al., 2015), indicating that the low level of maternal employment is probably unique to this community rather than a fault of study design.

There were few sex differences in baseline characteristics (though maternal education and household asset quintile are critical markers of SEP), and no observed sex-specific relationships between characteristics in stratified analyses. While the data indicated that the odds of a girl being born into a higher SEP family were about a third lower, the confidence intervals in both cases suggest that the difference could be as little as 1% or 8%. Further, while maternal education and

asset quintile were strongly correlated, their relationship did not show any sex-specific differences, which would have raised additional concerns about greater disadvantage among female children in the cohort.

The relationships between SEP markers and parental health behaviours suggest that younger parents were more educated, less likely to smoke, and from more households with more secure access to water and sanitation, and fewer children and more adults. This indicates that children born to younger parents were more likely to benefit from multiple socioeconomic advantages. On the other hand, older parents were often from households with many children, fewer adults, and were more likely to smoke and have an unplanned pregnancy. Children born to older parents were possibly exposed to multiple markers of low SEP and health behaviours.

Parental anthropometric data also suggested some nuanced socioeconomic patterning. While having one overweight parent (compared to neither) was related to parental age (for mothers) or water supply (for fathers), the determinants of having overweight parents were more strongly socioeconomic (water supply and top three asset quintiles). In this cohort, having overweight parents indicates socioeconomic advantage, but also hints at a wider problem. Parental anthropometric data suggest that, even in some of the city's most deprived communities, overweight and obesity among working age adults is an issue (15% of mothers and 13% of fathers were obese), and that higher living standards come with rising BMI for both sexes, a pattern observed in other urban poor communities in India (Gupta et al., 2016). Simultaneously, 18% of mothers and 11% of fathers were underweight, signalling a double burden of underweight and overweight among adults. This pattern is characteristic of populations undergoing a nutrition transition (Popkin et al., 2011). A similar transition was also observed recently in Nairobi's informal settlements (Kimani-Murage et al., 2015a).

The prevalence of low birth weight was 14%, lower than the 22% (Das et al., 2012) and 34% (Potdar et al., 2014) reported in other informal settlements that were part of intervention studies in Mumbai. However, 16% of birth weight values were heaped at 2500 g, suggesting that up to a third of cohort participants, all of whom were born after 37 weeks of gestation, were hovering quite close to having a low birth weight. I do not use birthweight in a subsequent analysis of the determinants of

linear growth (Chapter 6), but these data nonetheless indicate that a large proportion of cohort children were at risk of poor growth and health outcomes.

5.9.2 Overall follow-up and attrition

The cohort's high enrolment rate (97% of identified women who gave birth met inclusion criteria and consented to participation) corresponds to that observed for birth cohorts in other LMICs (Golding and Birmingham, 2009), which ranged from 74% to 98% of eligible births. However, attrition in the cohort compares poorly to the follow-up success of the study with the lowest enrolment among these older cohorts. The Birth to 20 cohort in South Africa, which identified only 74% of pregnancies in a very mobile population during a time of political upheaval, nevertheless reported 70% follow-up at 16 years (Richter et al., 2007), in stark contrast to the 62% at just two years in this study. Maintaining high rates of participation in a cohort study is a resource-intensive process (Golding and Birmingham, 2009), which is perhaps more feasible in larger cohorts with dedicated teams rather than a small one nested within an intervention study.

Arguably, attrition could have been minimized through a different study design or management strategies, such as an open cohort whose composition was allowed to change as residents moved in and out of informal settlement clusters, or following children up even after they were in another part of the city where SNEHA's programmes operate. However, the first would have made it difficult to examine the effect of time-varying factors such as infant feeding where data on diet at younger ages are just as important as data at older ages for the same infant. In the second scenario, it would have been difficult to relate later outcomes to any intermediate changes in children's environmental conditions (household asset quintile, for example) which were assumed constant since baseline for all participants.

Migration, which was the main reason for attrition, is a feature of urban poverty and life in informal settlements, which often comprise a mix of stable, long-term residents and highly mobile families (Zulu et al., 2011). The SNEHA Centres trial documented high annual turnover in this population (Shah-More et al., 2017) and the attrition observed represents the reality of doing research in urban informal settlements. Dropout is par for the course. Selecting a very stable cohort from the general population to ensure high response and retention rates would on one hand have reduced the risk of selection bias and / or statistical inefficiency in complete case

analysis, but on the other would have been unrepresentative of more vulnerable informal settlements. It would then be difficult to generalize study findings to the wider population of children born in these communities.

How much attrition is too much? One simulation study suggested that loss to follow-up between 5% to 60% of participants is not problematic if missing data arise due to MAR or MCAR mechanisms, but data that are missing due to MNAR can lead to bias even if attrition is low (Kristman et al., 2004). My analysis suggested that attrition was related to observed characteristics (maternal age and highest asset quintile), and that 38% loss to follow-up under a MAR assumption would not be very problematic.

I partially overcame the problem of attrition by using a more sophisticated analytic method. For growth analysis (Chapter 6), I used the SITAR model, which accommodates unbalanced designs and uses all available data to model a population trajectory as well as individual patterns for the full analysis period. Coupling anthropometry with detailed baseline socioeconomic indicators, I was able to examine the growth of almost all children (n=944, regardless of the number of length measurements, retaining available data for children who did not complete follow-up to 24 months. However, attrition did lead to a much smaller sample (n=438; 45%) in my analysis of the relationship between breastfeeding, complementary feeding, and length at two years (Chapter 8), as it was difficult to examine the longitudinal diets (exposure and mediator variables) of those who dropped out of the study.

5.9.3 Missing data patterns and their implications for subsequent analyses

My findings on analysis patterns of missing data presented in this chapter suggested that confounding structures were unlikely to differ between the whole cohort and subsets of participants included in subsequent analyses. Overall, the cohort's missing data are unlikely to lead to biased results presented in Chapter 6, 7, and 8.

The cohort's longitudinal dataset had a large number of incomplete cases and missing values due to intermittent non-response or dropout. Even those who completed follow-up to their second birthdays had missed 25% of scheduled visits in between. The patterns of missing longitudinal data are less problematic for the main

outcome (length) than they are for post-baseline (parental anthropometry) and time-varying (IYCF) factors which serve as exposure, mediator, or outcome variables. Only 53% of children had complete parental anthropometry data, and 47% of age-appropriate IYCF values were missing. While the missingness of neither was strongly related to observed background characteristics, the gaps in follow-up present some analytic challenges.

A complete case analysis would be near impossible since very few children had longitudinal information for every month from birth to two years. For longitudinal IYCF as an outcome, a realistic strategy would be to adopt a more flexible definition of time, using data from adjacent periods where possible and expanding the definition of an interval from one month to perhaps two or three months. Another approach would be to use a longitudinal analysis method that allows individuals to contribute varying numbers of observations across time, such that those who skip one or two visits are still able to contribute data collected at all other visits. While these strategies would require additional assumptions, they nonetheless offer a way to avoid wasting data. I combined these strategies to shape my analysis of the determinants of IYCF practices in Chapter 7.

For IYCF as an exposure or mediator, it would be difficult to use multiple imputation (MI) to impute missing values. MI is a good choice when data on confounders are missing, or if auxiliary variables are available (auxiliary variables are not used in an MI model, but are associated with missing data and are correlated (>0.3) with the variable which has missing data). However, MI is not a good choice when exposure data are missing and auxiliary variables that are not already in the substantive model are not available (or conceptualized) for an imputation model. It is also harder to implement MI when the missing data are for repeated measures (Tan et al., 2018). Further, datasets with large proportions of missing values make MI prone to errors. Complete case analysis (with or without weights for probability of prolonged participation) would be statistically inefficient but less biased than MI. In Chapter 8, I conducted a complete case analysis with expanded time intervals to use all available longitudinal IYCF data for those who completed follow-up with gaps.

Chapter 6 Linear growth in infancy and early childhood

Summary

In this chapter I examine how infant, parental and socioeconomic factors are associated with linear growth in infancy and early childhood (0-37 months). I present my rationale for selecting the SuperImposition by Translation and Rotation (SITAR) model and provide an overview of its main features. I explain how I used the SITAR model, including simple model fitting and curve-plotting, and multivariable analyses to identify how background factors shape children's size, tempo and velocity. I also present findings on the relationships between parental anthropometry and linear growth outcomes. I discuss the implications of my findings and my contribution to methodology.

Statement of contribution

I conducted all statistical analyses reported in this chapter. I received guidance from Professor Tim Cole, who shared basic R code for the SITAR model, helped me understand and interpret the basic model output, and provided periodic input on methodological and empirical issues related to multivariable analyses using SITAR.

6.1 Introduction

As described in Chapter 2, there are multiple strategies that researchers use to model infant growth in longitudinal observational studies. My review identified 35 unique metrics, and Leung et al. (2018) identified 40 in their more comprehensive study (which also included weight and BMI). Three of the five most common metrics in my review were based on linear mixed effects models, accounting for metrics used in 26% of included articles. Some approaches are obviously more common than others, possibly because they can be implemented in a more straightforward fashion or are suited to hypotheses and study designs that were most popular between 2010 and 2018.

Growth models should ideally be selected based on the research question and study design, number of growth measurements per participant and age at measurement (Wit et al., 2017). Several review articles (for example, see Johnson (2015)) describe the range of growth modelling strategies and provide guidance on selecting

the most appropriate model as well as examples of studies that have previously used it. Others focus on the application of one type of model; for example, linear spline multilevel models (Howe et al., 2016b) or linear parametric multilevel models (Johnson et al., 2013).

A slightly different approach to selecting a modelling strategy involves identifying a novel growth analysis model (from a broader class of applicable strategies) and applying it to study a unique population or a specific substantive area. What does the new method tell us that we did not know previously? Does it work as well in a population or age group very different to the one it was first used in? What additional methodologic questions does it raise? This approach shifts the research from a purely empirical or applied study to one with some methodologic intent.

I adopted such an approach in my analysis of factors that shape children's linear growth in early life. I was interested in identifying systematic differences in growth outcomes as a result of socioeconomic, parental, and child factors measured at or close to birth, a line of inquiry which could be best served by mixed effects growth curve modelling (Johnson, 2015). I also wanted to employ a method that would use all available longitudinal data and produce parameters that could be interpreted in a biologically meaningful way.

I chose the SuperImposition by Translation and Rotation (SITAR) model developed by Cole et al. (2010) to analyse cohort children's length data. SITAR is an example of a relatively new method that is not as commonly used as other mixed effects models. It has been used to model longitudinal weight outcomes in infancy and early childhood in a range of settings (Fuemmeler et al., 2016, Johnson et al., 2011, Johnson et al., 2014, Pizzi et al., 2014, Popovic et al., 2016). The only published paper on its application to linear growth of children in an urban setting is based on a Mexican study in which SITAR was used to model length in infancy as an exposure for BMI outcomes at seven years (Jones-Smith et al., 2013). Its application for outcome modelling of linear growth in the LMIC urban informal settlement environment has not yet been demonstrated.

While the novelty of the method for my study population sets it apart from other mixed effects models, SITAR is also a sophisticated and powerful tool for growth analysis. It is a significant development in the growth modelling toolkit, which made it a valid methodological choice for my research question (see Section 6.3).

6.2 Research question and objectives

In this chapter I address Research Question 4: What are the determinants of linear growth in infancy and early childhood?

The three specific objectives were to use the SITAR model to:

1. Describe the linear growth of children in the cohort.
2. Identify socioeconomic, parental and child characteristics associated with linear growth in infancy and early childhood.
3. Quantify the relationship between parental anthropometry and linear growth in infancy and early childhood.

6.3 Overview of growth modelling and SITAR

6.3.1 Growth modelling

Statistical analysis of longitudinal growth data generally consists of three steps. First, distance and velocity curves, and possibly also an acceleration curve, are estimated using a parametric or non-parametric smoothing function. Second, individual curves are used to derive parameters (such as age at maximum velocity) and their corresponding values for distance, velocity, and acceleration. Third, the parameters are used in analyses comparing groups or populations (Molinari and Gasser, 2004).

A basic statistical model of growth assumes that length *l(t)* consists of the sum of a 'true' age-dependent length *l(t)*, which is unknown but fixed for any given infant, and a 'random' part. The random part includes components that are not the objective of the research, such as the error of measurement, and short term growth effects such as daily variations in length, seasonality in growth velocity, and catch-up and catch-down growth. This implies that what can be regarded as the 'true' length can change depending on the aims of the research (since the random part is determined by the study and context). The selected analytic method (non-parametric or parametric function) must either allow or not allow deviation from a pre-defined pattern or model of growth (Molinari and Gasser, 2004).

Parametric non-linear models do not adequately express the shape of the underlying regression function used to analyse noisy data. This problem can be

overcome by using non-parametric estimators such as splines, local polynomials, or kernel estimators (Gasser et al., 2004). Non-parametric (or non-structural) models do not specify a particular functional form for the growth curve, and are often easier to fit than structural ones such as Jenss-Bayley and Berkey-Reed models that are used for growth in infancy (Hauspie and Molinari, 2004).

A cubic spline or cubic polynomial, specified over the range of the data, is a set of age cubed terms used to give a smooth shape to the growth function so it fits the data better. The terms are joined at knots. The location and number of knot points in a smoothing spline is generally a subjective decision, involving a trade-off between introducing small bias from fewer knots (and thus minimal smoothing) and larger bias from having many knots that potentially lead to overfitting (Johnson, 2015). A regression cubic spline places knots at equidistant points or quantiles of the age distribution. A natural cubic spline is constrained to be linear beyond boundary knot points such that data points beyond the boundaries can also be fitted.

Another important concept is the shape invariant model (SIM) applied to growth modelling, which stems from the knowledge that normal growth is regulated by general biological mechanisms which give a common shape to a set of curves. This common curve can be derived by using an appropriate model to describe a growth process that differs across individuals in quantitative terms but is the same in qualitative terms. The quantitative differences between individuals can be expressed as shifting and scaling model parameters. The parameters of a SIM are interpretable in a biologically meaningful way (Gasser et al., 2004).

Mixed effects or multilevel models offer a statistical method to express any structural or non-structural growth function. They address a common problem associated with longitudinal growth studies: participants often have unequal numbers of growth measurements which are unequally spaced over time. Further, multilevel models enable researchers to fit growth curves for all participants in one model rather than fitting individual growth curves separately for each child (Johnson, 2015). It is also easy to estimate an average sample curve and examine associations of covariates with growth outcomes (Johnson et al., 2013). Mixed effects growth curve models produce the sample average as fixed effects and individual-specific parameters as random effects, with residuals for between- and within-individual differences. However, with the exception of SITAR and linear spline models, the between-individual residuals do not have any biological interpretation. Non-linear mixed

effects models allow for the growth outcome variable to be a non-linear function of the model parameters (Johnson, 2015). They are also more flexible and parsimonious (producing fewer parameters) than linear mixed models.

6.3.2 SITAR

The SITAR model's underlying method is based on two pieces of work. The first paper by Beath (2007) described a longitudinal growth model for weight gain in infancy and its relationship with duration of breastfeeding in the Childhood Asthma Prevention Study trial. Beath used a shape invariant model with a natural cubic spline function, and fitted it as a non-linear mixed effects model. The second paper, an analysis by Cole et al. (2008), presented a longitudinal analysis of height data for children aged 9-20 years from the Christ's Hospital School study conducted between 1927 and 1956. They used a non-linear mixed effects model, citing Beath's paper. They estimated the mean height curve with a fixed effect regression spline with 11 degrees of freedom, and used a cubic regression spline to compare population and individual growth curves of children. The two spline curves were used to derive mean peak height velocity and mean age at peak height velocity.

The SITAR model was published as a paper in 2010 with an accompanying R package (*sitar*) for curve fitting and plotting released in 2013. Its main features are explained below, based on the description in the paper by Cole et al. (2010), and modified for modelling length in infancy and early childhood.

For a dataset containing child age in continuous months and length measurements in centimetres, the SITAR model for a set of length curves can be expressed as the following equation for a random effects model:

$$y_{it} = \alpha_i + l\left(\frac{t - \beta_i}{\exp(-\gamma_i)}\right)$$

Where y_{it} is the length for child *i* at age *t*, *l(t)* is a natural cubic spline curve of length versus age, and α_i, β_i, and γ_i are child-specific random effects, corresponding to terms for size, tempo, and velocity. When the model fits the data well, suitable values of the child-specific random effects can define how each child's growth differs from the mean curve, and as such the three terms are meant to be interpreted together.

The size term adjusts for differences in mean length (interpreted in cm), and corresponds to an up-down shift in the mean spline curve, with smaller values for shorter children. The tempo term adjusts for differences in timing of peak length velocity (interpreted as age in months), and corresponds to a left-right shift in the spline curve, with negative values for those who achieve it early. The size and tempo terms are, geometrically, translations in the mean curve.

The velocity term represents an individual's duration of growth as an age-scaling factor (expressed as a fraction). The average curve has a velocity of zero, so the term can take on positive and negative values. A positive velocity indicates a stretching of the age scale and a steep growth curve (altered slope), such that the child's growth is faster than average across the entire period. A negative velocity indicates a shrinking of the age scale, a shallow growth curve, and slower than average length gain. Geometrically, the shrinking-stretching of the age scale rotates each child's curve to make it similar to the mean spline curve.

The three random effects thus enable each individual curve (adjusted for α, β, and γ) to be superimposed (by translation and rotation) on the average curve.

There are two ways to relate SITAR parameters to covariates. One way is to include covariates as fixed effects in the model (additively or multiplicatively, though the latter significantly increases the model's complexity) to look at their association with any one, two or all three parameters. This gives the difference in average size, tempo, and / or velocity between two or more groups of children. The second way is to fit a simple model without any covariates, or adjust minimally for 'forced' variables such as sex, and then export the child-specific values of size, tempo, and velocity for use in further analysis.

6.4 Methods

This section begins with a description of the cohort dataset and data checking and cleaning tasks conducted prior to analysis. I then describe the model fitting, multivariable analyses, and model checking tasks.

6.4.1 Dataset

The cohort dataset was retained in its long form, consisting of multiple measurements per child. Each observation was indexed by identifiers (ID number,

cluster, household details, date of birth), and contained unique time variables (date of measurement and derived age variables in days and months) and corresponding child anthropometric (and other time-varying) data collected at the occasion, as well as time-invariant baseline survey and parental anthropometry variables which were fixed across a child's set of observations.

After cleaning the data and manipulating them to derive analysis variables, I created two analysis datasets. The first included data on all children who had complete information on background covariates of interest, and the second was a reduced subset of children whose parents had responded to anthropometry data collection. I did this because the SITAR model uses only complete cases in a dataset, even if individuals are missing data on variables that are not in the substantive model.

Variables common to both analysis datasets included length, age, binary variables for parental ages and education, access to piped water, use of shared toilet, two or more adults and four or more children in the household, maternal and paternal smoking, and pregnancy intention. Two variables encoded household asset quintile (ordered categorical) and scores (continuous).

Additional variables for the parental anthropometry dataset included height and weight z-scores, BMI, BMI category (underweight, normal, overweight and obese), and overweight status (binary variable) for each parent. Combined parental anthropometry variables included sums of and differences in maternal and paternal height and weight z-scores (see Chapter 4 and Griffiths et al., 2007), and a categorical variable encoding four groups of parental overweight (either, neither, and both).

6.4.2 Data checking and cleaning

The purpose of data cleaning was to ensure that the length data I used for growth modelling were as free of error as possible. However, I also wanted to carry out data cleaning without altering the data to an extent that would introduce bias. Further, since the cohort's data management and supervision teams had already carried out quality control and random checks during the study and on the final dataset, I did not expect to conduct extensive cleaning.

One general data cleaning strategy consists of three phases that involve screening, diagnosis, and treatment or editing, and an additional one of excluding observations (Van den Broeck et al., 2005). I used this as a general framework to structure my data cleaning activities, though the process was more recursive than linear.

In the screening phase, I examined the data for lack of or excess data and inconsistencies. This included data that had not been entered (missing), entered more than once (duplicates), or entered in the wrong field (for example, weight recorded in the length field). I looked for inconsistencies that were a likely result of incorrect data entry or programming error.

In the diagnostic phase I emphasized plausibility and longitudinal coherence. I examined any observations that were measured on dates outside the study period (March 2013 to April 2016), and corresponding ages (0-1127 days and 0-37 months). I examined data (at any age) reporting length below 45 cm or over 100 cm to identify possibly implausible values. Those below 45 cm were categorized as either true outliers or errors. For longitudinal coherence, I assessed whether the data made sense given the child's age and other measurements. Once inconsistencies and potentially implausible values had been identified, I examined whether they made sense given the child's age and other length measurements.

Since children cannot technically become shorter with age, I flagged instances of decrease in length between successive visits as potential errors. I then examined decreases greater than 1 cm to assess whether these were due to obvious mistakes that could be resolved without dropping the observation. Since length was measured in duplicate, I checked variables for both measurements, and used the paired value as well as adjacent values to make an assessment before marking observations for editing or deletion, or leaving them unchanged. For example, if a length measurement was 56.5 cm, its pair was 65.5 cm, the ones taken 30 days previously were both 64.3 cm, and those 30 days later were 66.1 cm, I decided that it was safe to change the 56.5cm to 65.5cm to correct an obvious digit entry error. When such straightforward corrections were not possible and the difference was very large, I chose to drop the suspect observation as the rest of the child's measurements would be sufficient to contribute data towards the population and individual curves.

After making changes or deleting observations, I re-screened data for any obvious or large inconsistencies. Once I addressed these, I decided not to clean the data

further. I recalculated the mean length at each visit after the first and second measurement variables had been checked and cleaned.

As described in Chapter 4, for data collected after 24 months (730 days), I added 0.7 cm to height measurements to convert them to length measurements before I began growth analysis.

I carried out data cleaning in Stata, and then exported the dataset to R in order to divide it into two analysis datasets.

6.4.3 Model fitting

I conducted all SITAR modelling in R using the *sitar* package (versions 1.0.8 to 1.1.1). The analysis code is presented in a script file in Appendix 6.1.

I fitted the basic SITAR model using the larger dataset which comprised 16 753 length observations on 944 children with complete covariate information. The model's natural cubic spline function included four internal knots placed at equal intervals on the age distribution. The code for my simple model is described below.

```
e0 <- sitar (agemonths, lt, id, na.omit (df), 4)
```

Where *e0* is the name of the object which holds the fitted model; *agemonths, lt,* and *id* are variables for age, length, and id. The dataset is identified by *df*, with an option (*na.omit*) to exclude any observations with missing data, and 4 indicates the degrees of freedom for fitting the spline curve.

In order to describe the cohort's linear growth, I subsequently fitted two separate models by updating the basic model, *e0*.

```
e2 <-  update (e0, a.formula=~sex, b.formula=~sex, c.formula=~sex)
```

```
es2 <- update (e0, a.formula=~sex+sint+cost, b.formula=~sex+sint+cost,
                c.formula=~sex+sint+cost)
```

The first (e2) was a model adjusted for infant sex, which was included as a fixed effect. The second (es2) was adjusted for sex and seasonality. I used a Fourier transformation to describe seasonal effects on linear growth. A Fourier term

decomposes a periodic function into oscillating sine and cosine functions (*sint* and *cost* in the model above), and offers a way to examine seasonal effects on growth outcomes (see Fulford et al. (2006) for a detailed description of its application to growth modelling). Including it as a fixed effect in SITAR would indicate whether seasonality, adjusted for sex, affected size, tempo, or velocity.

After fitting the sex and sex-seasonality models, I summarised and compared their outputs using sitar post-estimation commands. I looked at the standard deviations of the size, tempo, and velocity parameters and the extent of their correlations. I plotted the mean spline curve and estimated average age at peak length velocity. I also plotted all individual growth curves, before and after SITAR adjustment, and used the *predict* option to calculate predicted lengths at different ages.

Since the model fit well using age in continuous months, I did not log-transform the age variable.

6.4.4 Multivariable analyses

I used the sex and sex-seasonality adjusted models as the basic models in multivariable analyses for the second and third objectives of the chapter.

To identify the determinants of linear growth from among the background variables, I first conducted univariable analysis. I added each covariate as fixed effects on size, tempo, and velocity in a model adjusted for sex. I added household asset quintile and score variables separately, since the categorical variable was more likely to run into data sparsity problems or make the model too complex to fit. I then fitted a full model adjusting for all covariates. I repeated the univariable and multivariable analyses using the sex-seasonality specification as the basic model. I did this in order to assess whether seasonality altered the effect of any or all factors associated with size, tempo, or velocity. I also tested a basic model with an interaction term for sex, but the model failed to converge. I did not examine this further.

To quantify the relationship between parental anthropometry and linear growth, I conducted two sets of analyses after fitting a basic model to 12 208 length measurements for 509 children with complete exposure and covariate information.

In the first stage, I fitted four sex-seasonality adjusted models with different exposure specifications. One included the categorical variable encoding parental overweight status (either, neither, both parents overweight). A second included four separate variables for maternal and paternal height and weight z-scores to look at their mutually adjusted associations with linear growth. A third included two variables for the sum of maternal and paternal z-scores, for height and weight. The fourth exposure specification included two variables for the difference in maternal and paternal z-scores, for height and weight. These four models enabled me to understand whether the crude influence of parental anthropometry was due to a combined effect of both parents' size (sum of z-scores) or the relative contribution of one parent (difference in z-scores).

In the second stage, I fitted separate multivariable models adjusted for sex, seasonality and all background covariates. One model included the categorical parental overweight variable as an exposure of a priori interest. The second model included whichever of the two combined parental specifications (sum or difference in parental heights or weights) showed an association with growth in the first stage.

6.4.5 Model checking

I assessed model fit using Bayesian Information Criterion (BIC) values. Lower BIC values indicate better fit. Poor model specification or an inadequate set of covariates generally result in penalized fit, leading to larger BIC values.

I examined the residual standard deviation (SD) of the spline curve. A residual SD that is similar to the error of measurement associated with the measure, length data in this case, generally indicates that the model is a good fit for the observed data (Johnson, 2015).

I plotted child-level residuals as scatter plots and quantile normal plots, checked that they were normally distributed, and examined their range and IQR. Departures from normality would indicate heteroscedasticity or poor model fit.

For each model, I checked the proportion of variance in the length data that it explained, and compared this between models and to values in other studies that have used SITAR. The proportion of variance explained by differences between

children tends to be particularly high for mixed effects models because children's individual growth shows much population variability (Johnson, 2015).

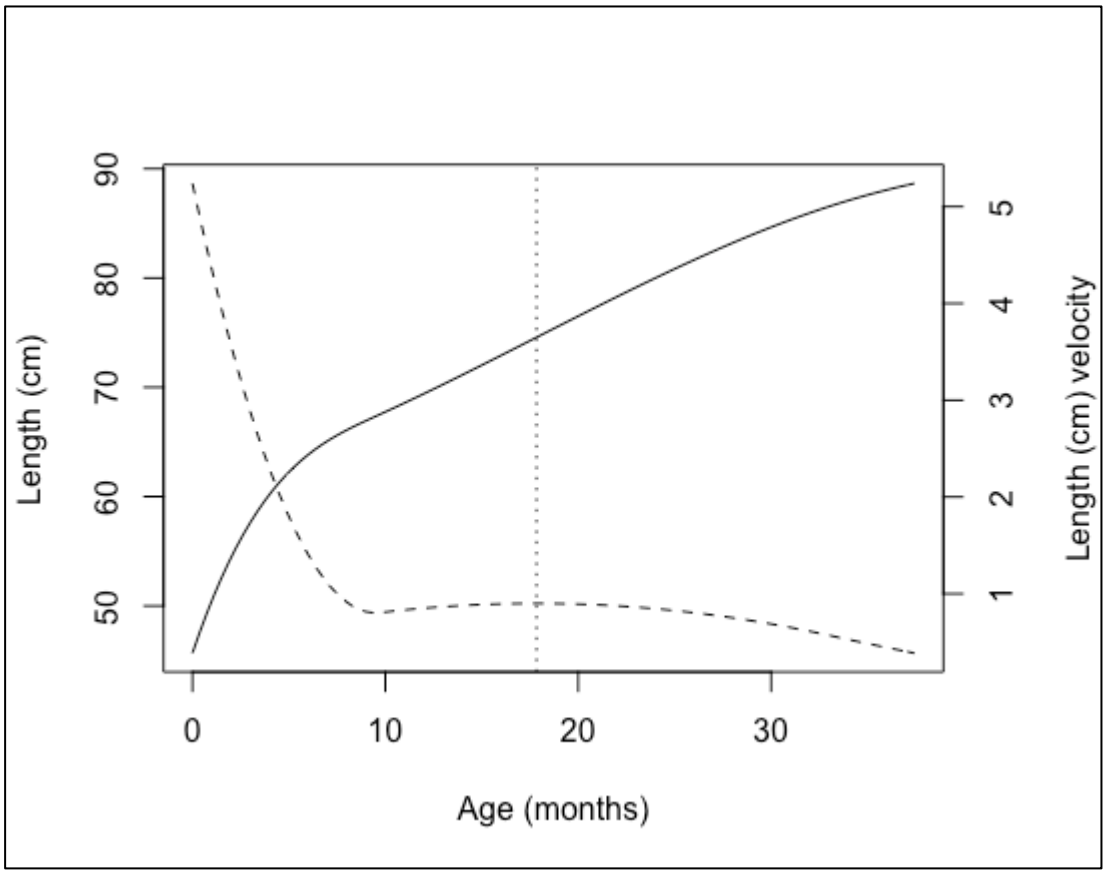
6.5 Description of the linear growth curve for the cohort

6.5.1 Basic model

The median number of length measurements per child was five in the first year and 11 across the full period.

The mean population curves for length gain (solid black curve) and velocity (dashed black curve) are shown in Figure 6.1. The length curve extends from birth to 37 months with a predicted average length of 45.6 cm at birth and 88.4 cm at 37 months. Length velocity decreased steadily from 4.9 cm/month in the first month to 0.81 cm/month at 11 months. Soon after, there was a slight, gradual increase and velocity peaked (vertical, dotted black line) at 0.8994 cm/month at 17.8 months, decreasing to 0.41 cm/month by the time children were 37 months old.

Figure 6.1 Size and velocity curves, and age at peak length velocity produced by the basic SITAR model



The standard deviations of the three random effects and their correlations are presented in Table 6.1.

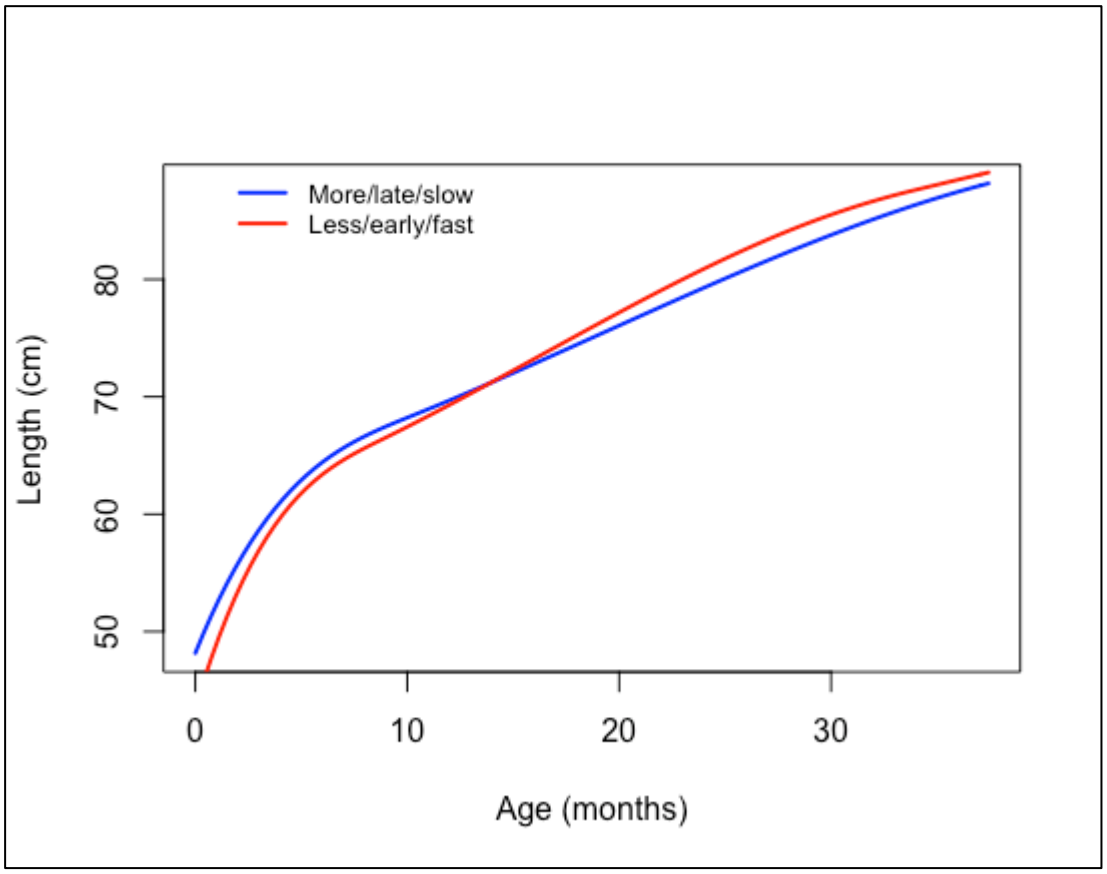
Table 6.1 Correlations between size, tempo, and velocity in the basic SITAR model

Model parameter	SD	Correlations	
Size (cm)	4.54	**Size**	**Tempo**
Tempo (months)	3.52	0.7	
Velocity (%)	23	-0.6	-0.9
Residual (cm)	1.10		

The positive correlation between size and tempo indicates that children who gained more length over the follow-up period tended to achieve peak length velocity at older ages. The negative correlation between size and velocity indicates that greater length gain was accompanied by lower length velocity. The strong inverse correlation between tempo and velocity implies that children who reached peak length velocity later had slower growth across the follow-up period. The direction of these correlations suggests that those who gained more length tended to do so slower and peaked later, probably starting out much shorter than average, and this growth pattern likely resulted in lower attained length. The lengths and velocities predicted by the model attest to this. Children who reached peak velocity one month later than average were shorter than those who reached it one month before the average child at six (62.2 cm vs 65.1 cm) and twelve (68.5 cm vs 70.2 cm) months.

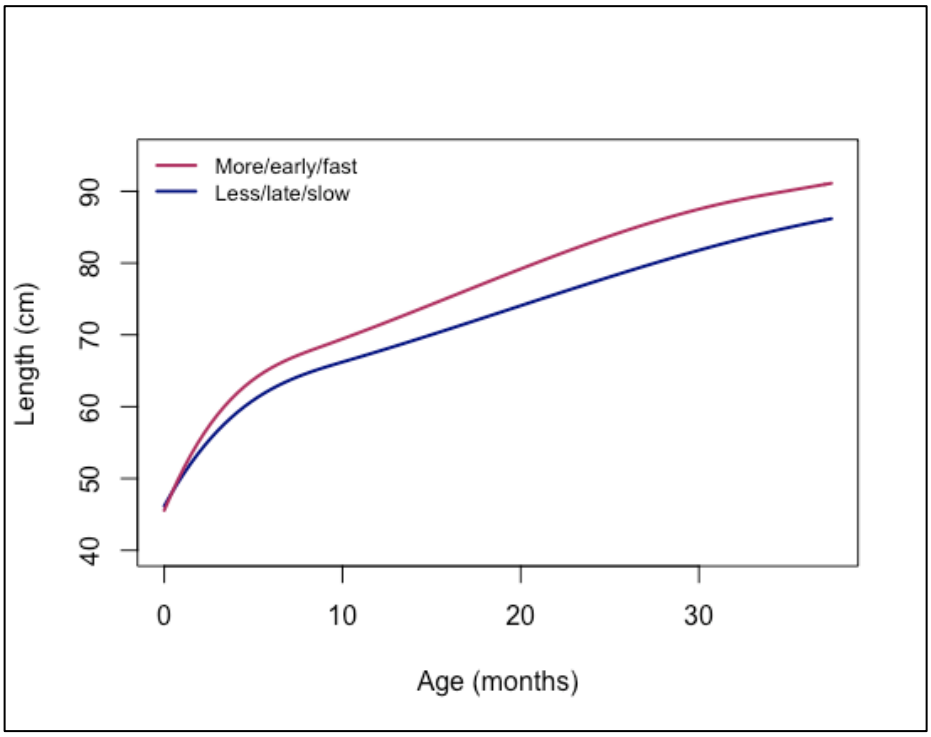
Figure 6.2 shows that those who grew less (in the first 10 months), but peaked earlier and had higher velocity (at around 1 year), eventually attained greater length in the second and third years of life, even if they started out slightly shorter than children who grew more, peaked later and had lower than average velocity.

Figure 6.2 Distance curves comparing more / late / slow growth and less / early / fast growth



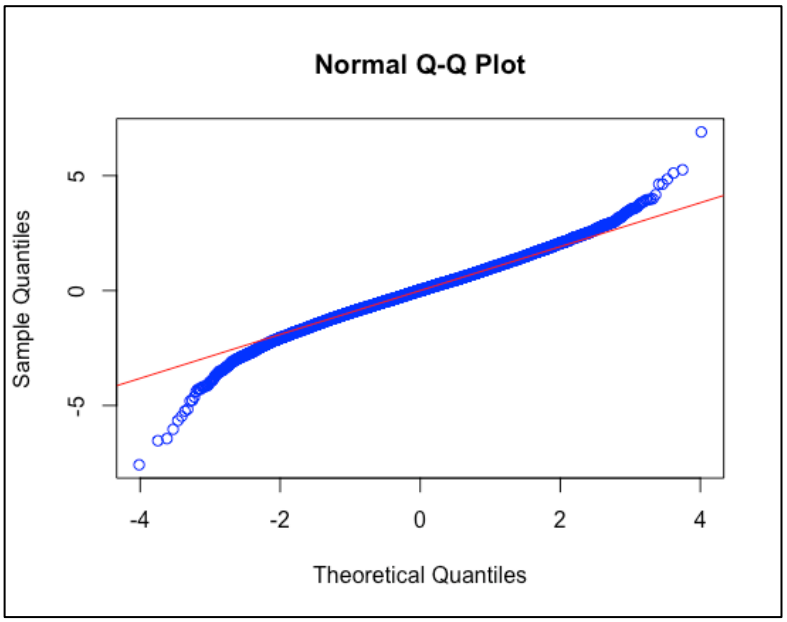
Further, despite starting out at similar lengths (45.6 cm and 46.1 cm), attained length was much greater when early tempo and faster velocity were accompanied by more length gain (+1SD), compared to a growth process of lower length gain (-1SD) with delayed tempo and slower velocity (Figure 6.3). At 24 months, this translated to predicted lengths of 82.9 cm and 77.3 cm.

Figure 6.3 Distance curves comparing more / early / fast growth and less / late / slow growth



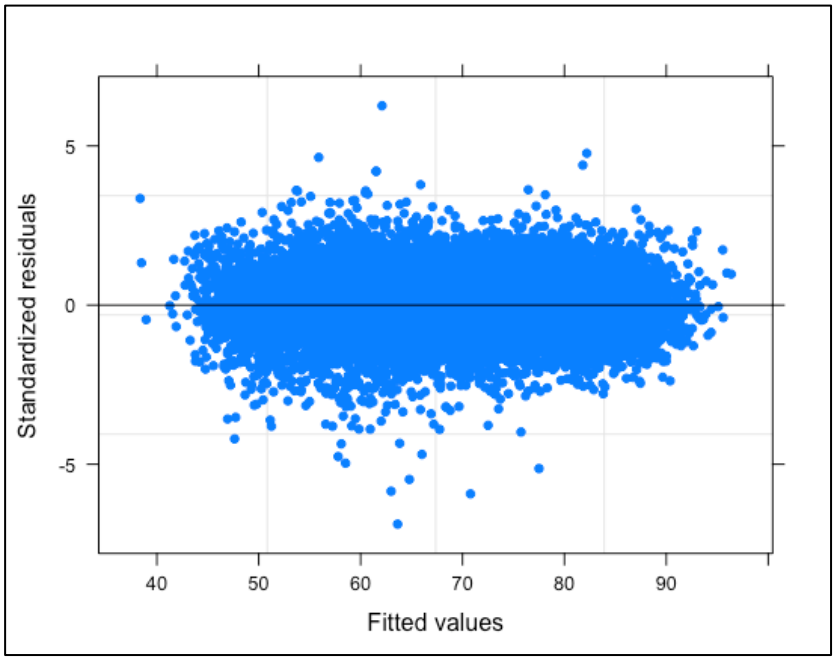
The model explained 88% of the variance in length, with a residual standard deviation (RSD) of 1.1 cm. A quantile normal plot (Figure 6.4) of the residuals showed that they were normally distributed, although there was some evidence of non-normality at the tails.

Figure 6.4 Quantile normal plot for between-child residuals



A plot of the standardized within-child residuals (Figure 6.5) showed that they were tightly centred around zero (IQR -0.58 cm to 0.58 cm), though there were some outlier values with large residuals (-6.8cm to 6.2cm), indicating that for a small number of observations the difference in raw and SITAR-predicted length was quite large.

Figure 6.5 Plot of standardized residuals from the basic SITAR model



6.5.2 Sex and sex-seasonality adjusted growth curves

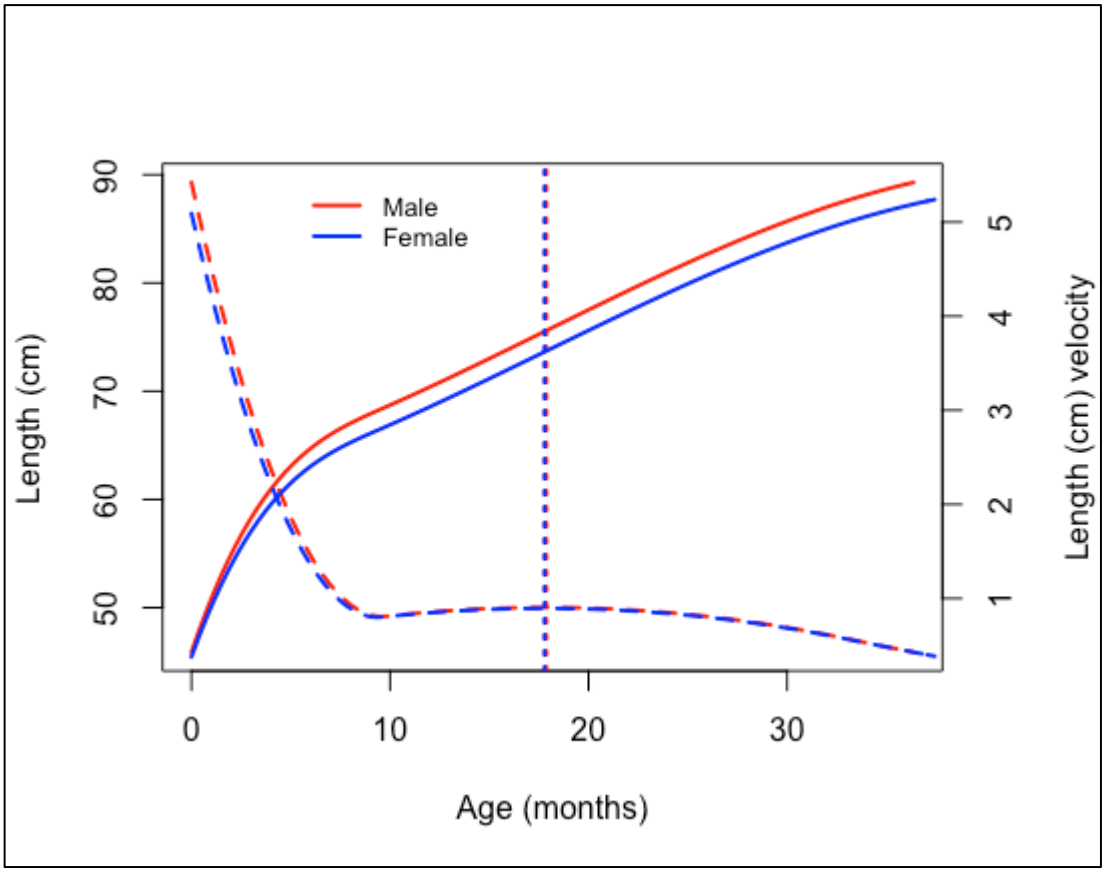
Models adjusted for sex and sex-seasonality generally fit better than the simple model (BIC values 59157 and 59190 vs 59214). The random effect and residual SD values did not change much in the sex-adjusted model, though the sex-seasonality model reduced the residual SD to 1.08 and increased the SD for size, tempo, and velocity random effects. The fixed effects showed that female children had lower size, but not tempo or velocity (Table 6.2).

Table 6.2 Size, tempo, and velocity fixed and random effects for sex and sex-seasonality adjusted models.

	Sex-adjusted model				Sex-seasonality adjusted model			
	Random effects	Fixed effects (Female)			Random effects	Fixed effects (Female)		
	(SD)	Value	SE	p	(SD)	Value	SE	p
Size (cm)	4.44	-1.97	0.33	<0.0001	4.91	-2.27	0.36	<0.0001
Tempo (months)	3.52	-0.16	0.27	0.547	3.83	-0.35	0.29	0.2226
Velocity (proportion)	0.24	-0.01	0.02	0.5562	0.26	-0.003	0.02	0.9896
Residual (cm)	1.10				1.08			

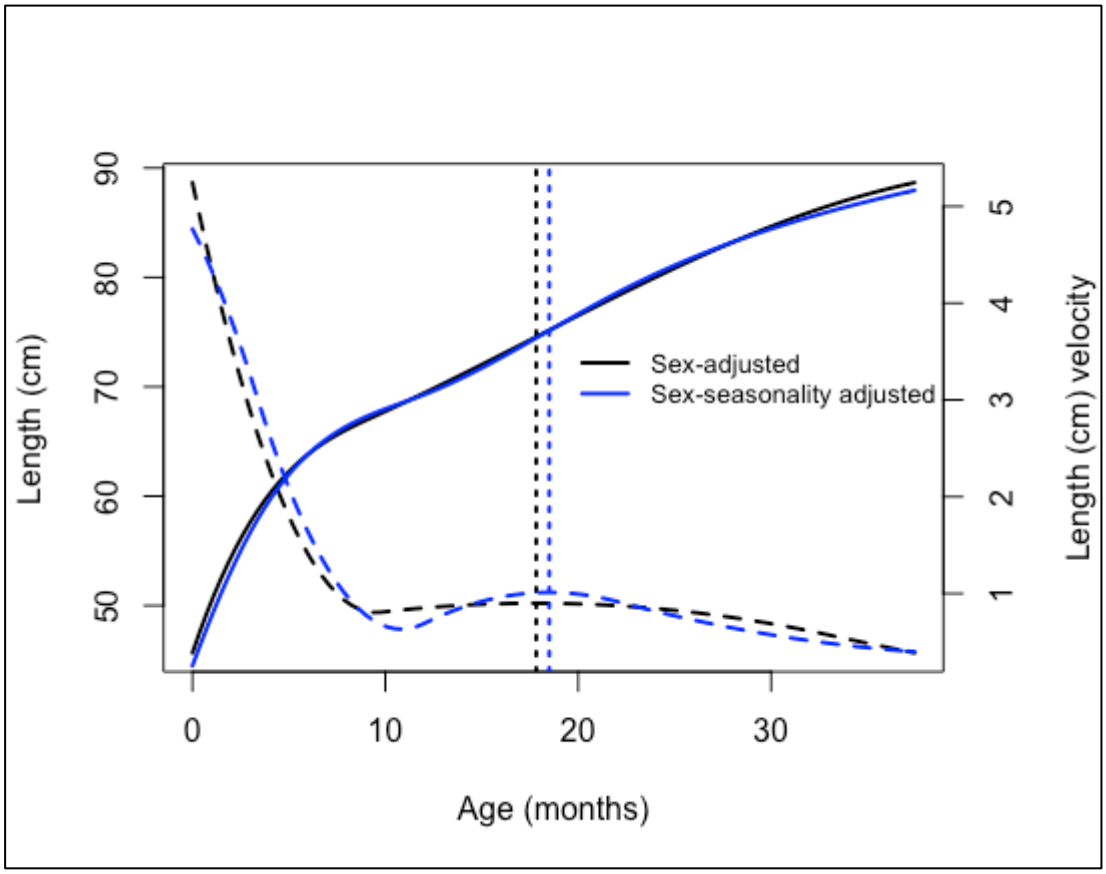
The mean distance curves also showed that female children were, on average, shorter than males, and gained 1.97 cm less, but there were no appreciable differences in their growth velocity or the age at which they reached peak length velocity (Figure 6.6).

Figure 6.6 Sex-adjusted growth curves for male and female children



The sex-seasonality adjusted velocity curve showed a distinct bump, beginning with slightly lower velocity than the sex-adjusted model between 9 and 12 months, and a gradual rise again between 15 and 22 months. The adjustment for seasonality delayed the age at peak length velocity from 17.8 to 18.5 months, when it was 1.01 cm/month, but the distance curve remained largely unchanged (Figure 6.7).

Figure 6.7 Sex and sex-seasonality adjusted growth curves



6.6 Environmental determinants of linear growth

6.6.1 Univariable relationships

In univariable models with infant sex included as a forced variable (Table 6.3), most baseline factors, except parental age variables and maternal smoking, had some effect on one or more growth parameters. Most associations were preserved (and more pronounced) in sex-seasonality adjusted models (Table 6.4), with a further association between maternal smoking and tempo (0.93 months; SE 0.41, p= 0.0247).

The relationships of SEP variables with size, tempo, and velocity differed in magnitude but were similar in nature. Higher household asset score and quintile were both associated with lower length gain, but earlier tempo and faster velocity. Compared to children in the lowest quintile, those in the highest grew 16% faster (increasing to 19% after accounting for seasonality) and reached peak velocity 2.25 months earlier. The inverse pattern was observed for use of a shared toilet, with greater length gain, but delayed tempo (by 1.65 months) and 13% lower velocity, which increased to 15% in the seasonality model.

Greater parental education, and households with more than two adults, were also associated with earlier tempo and higher velocity, and a similar pattern was observed for pregnancy intention in the seasonality model. The inverse pattern was associated with households with four or more children.

Access to piped water was associated with 4% greater velocity, and paternal smoking with 4% lower velocity.

Table 6.3 Estimates of associations between background factors and linear growth outcomes in univariable sex-adjusted models

			Size (a)			Tempo (b)			Velocity (c)		
Model and covariates	**BIC**	**Variance explained (%)**	**Estimate (cm)**	**SE**	**p**	**Estimate (months)**	**SE**	**p**	**Estimate (proportion)**	**SE**	**p**
Maternal age	59184	88.08									
Female			-1.99	0.33	<0.0001	-0.16	0.27	0.5512	-0.01	0.02	0.5459
Mother ≥25 years			-0.15	0.33	0.6551	-0.13	0.27	0.6232	-0.01	0.02	0.5549
Paternal age	59191	88.08									
Female			-2.01	0.33	<0.0001	-0.19	0.27	0.4825	-0.01	0.02	0.6192
Father ≥30 years			-0.18	0.33	0.5954	0.29	0.27	0.2838	-0.02	0.02	0.2399
Asset score	59151	88.08									
Female			-1.96	0.33	<0.0001	-0.19	0.26	0.4637	-0.01	0.02	0.6643
Asset score			-0.39	0.17	0.0182	-0.78	0.13	<0.0001	0.06	0.01	<0.0001
Asset quintile	59237	88.06									
Female			-1.92	0.33	<0.0001	-0.15	0.26	0.5539	-0.01	0.02	0.5476
Second lowest			-0.91	0.50	0.0677	-1.26	0.40	0.0019	0.08	0.03	0.0042
Middle			-0.56	0.51	0.2751	-1.16	0.41	0.0048	0.07	0.03	0.0071
Second highest			-1.10	0.52	0.0328	-2.15	0.41	<0.0001	0.15	0.03	<0.0001
Highest			-1.11	0.52	0.0314	-2.25	0.41	<0.0001	0.16	0.03	<0.0001
Maternal education	59161	88.06									
Female			-1.92	0.33	<0.0001	-0.16	0.26	0.5275	-0.01	0.02	0.6116
≥6th standard			0.01	0.33	0.9649	-0.81	0.26	0.0022	0.07	0.02	<0.0001
Paternal education	59164	88.07									
Female			-1.96	0.33	<0.0001	-0.13	0.26	0.6199	-0.01	0.02	0.4785
≥6th standard			-0.19	0.33	0.5716	-0.73	0.27	0.0071	0.06	0.02	0.0004

			Size (a)			Tempo (b)			Velocity (c)		
Model and covariates	**BIC**	**Variance explained (%)**	**Estimate (cm)**	**SE**	**p**	**Estimate (months)**	**SE**	**p**	**Estimate (proportion)**	**SE**	**p**
Water supply	59169	88.08									
Female			-1.98	0.33	<0.0001	-0.20	0.27	0.4412	-0.01	0.02	0.7215
Access to piped water			0.28	0.34	0.4068	-0.26	0.28	0.3487	0.04	0.02	0.0473
Toilet	59158	88.08									
Female			-2.04	0.33	<0.0001	-0.26	0.26	0.3272	-0.003	0.02	0.8572
Shared toilet			1.14	0.44	0.0095	1.65	0.34	<0.0001	-0.13	0.02	<0.0001
Household children	59215	88.03									
Female			-1.93	0.33	<0.0001	-0.08	0.26	0.752	-0.02	0.18	0.3412
≥4 children			0.42	0.34	0.2188	1.11	0.28	0.0001	-0.09	0.02	<0.0001
Household adults	59174	88.08									
Female			-1.97	0.33	<0.0001	-0.16	0.26	0.5318	-0.01	0.02	0.5738
≥2 adults			-0.20	0.33	0.5401	-0.69	0.26	0.0088	0.06	0.02	0.0009
Pregnancy intention	59169	88.08									
Female			-2.02	0.33	<0.0001	-0.20	0.26	0.456	-0.01	0.02	0.6435
Planned			0.20	0.33	0.5488	-0.46	0.27	0.0868	0.05	0.02	0.0117
Paternal smoking	59177	88.08									
Female			-2.01	0.33	<0.0001	-0.21	0.27	0.4305	-0.01	0.02	0.6948
Father smokes			-0.09	0.33	0.7925	0.46	0.27	0.0859	-0.04	0.02	0.0359
Maternal smoking	59386	87.96									
Female			-1.95	0.33	<0.0001	-0.13	0.27	0.6496	-0.01	0.02	0.4662
Mother smokes			0.10	0.47	0.8372	0.73	0.40	0.0683	-0.04	0.03	0.1194

Table 6.4 Estimates of associations between background factors and linear growth outcomes in univariable sex-seasonality adjusted models

			Size (a)			Tempo (b)			Velocity (c)		
Model and covariates	**BIC**	**Variance explained (%)**	**Estimate (cm)**	**SE**	**p**	**Estimate (months)**	**SE**	**p**	**Estimate (proportion)**	**SE**	**p**
Maternal age	59207	88.4									
Female			-2.32	0.36	<0.0001	-0.38	0.29	0.1912	0.002	0.02	0.9283
Mother ≥25 years			-0.36	0.36	0.3185	0.04	0.29	0.8812	-0.004	0.02	0.8377
Paternal age	59211	88.39									
Female			-2.32	0.36	<0.0001	-0.40	0.29	0.1639	0.004	0.02	0.8562
Father ≥30 years			-0.04	0.36	0.9163	0.37	0.29	0.2043	-0.03	0.02	0.1758
Asset score	59278	88.33									
Female			-2.35	0.36	<0.0001	-0.44	0.28	0.1165	0.01	0.02	0.6622
Asset score			-0.55	0.18	0.0024	-0.84	0.14	<0.0001	0.06	0.01	<0.0001
Asset quintile	59293	88.36									
Female			-2.26	0.36	<0.0001	-0.35	0.28	0.1984	0.002	0.02	0.9069
Second lowest			-1.00	0.55	0.0678	-1.24	0.43	0.0091	0.08	0.03	0.0091
Middle			-0.17	0.56	0.7620	-0.74	0.44	0.1052	0.05	0.03	0.1052
Second highest			-0.90	0.56	0.1107	-1.91	0.44	<0.0001	0.14	0.03	<0.0001
Highest			-1.68	0.56	0.0029	-2.50	0.43	<0.0001	0.19	0.03	<0.0001
Maternal education	59212	88.34									
Female			-2.25	0.36	<0.0001	-0.40	0.28	0.1532	0.01	0.02	0.7457
≥6th standard			-0.13	0.36	0.7088	-0.93	0.28	0.0009	0.08	0.02	<0.0001
Paternal education	59196	88.38									
Female			-2.28	0.37	<0.0001	-0.36	0.29	0.211	0.001	0.02	0.9723
≥6th standard			-0.12	0.36	0.7424	-0.70	0.29	0.0159	0.06	0.02	0.0009

			Size (a)			Tempo (b)			Velocity (c)		
Model and covariates	**BIC**	**Variance explained (%)**	**Estimate (cm)**	**SE**	**p**	**Estimate (months)**	**SE**	**p**	**Estimate (proportion)**	**SE**	**p**
Water supply	59278	88.35									
Female			-2.27	0.36	<0.0001	-0.38	0.29	0.1876	0.004	0.02	0.8338
Access to piped water			0.26	0.37	0.4923	-0.29	0.30	0.3372	0.04	0.02	0.0355
Toilet	59226	88.37									
Female			-2.31	0.36	<0.0001	-0.38	0.28	0.1729	0.00	0.02	0.8412
Shared toilet			1.49	0.48	0.0019	1.97	0.37	<0.0001	-0.15	0.03	<0.0001
Children in the HH	59200	88.37									
Female			-2.24	0.36	<0.0001	-0.30	0.28	0.2889	-0.004	0.02	0.8475
≥4 children			-0.01	0.37	0.9775	0.73	0.30	0.0138	-0.07	0.02	0.001
Adults in the HH	59213	88.37									
Female			-2.35	0.36	<0.0001	-0.45	0.28	0.108	0.01	0.02	0.6751
≥2 adults			-0.49	0.36	0.1754	-0.95	0.28	0.0008	0.08	0.02	<0.0001
Pregnancy intention	59205	88.37									
Female			-2.29	0.36	<0.0001	-0.37	0.28	0.1954	0.001	0.02	0.942
Planned			0.03	0.36	0.9279	-0.66	0.29	0.0226	0.06	0.02	0.0022
Paternal smoking	59208	88.39									
Female			-2.29	0.36	<0.0001	-0.39	0.29	0.1738	0.003	0.02	0.887
Father smokes			-0.22	0.36	0.542	0.37	0.29	0.1963	-0.03	0.19	0.0768
Maternal smoking	59202	88.39									
Female			-2.32	0.36	<0.0001	-0.42	0.29	0.1462	0.005	0.02	0.813
Mother smokes			0.32	0.51	0.5376	0.93	0.41	0.0248	-0.05	0.03	0.0579

6.6.2 Multivariable relationships

In the sex-adjusted multivariable model incorporating the full set of hypothesised covariates, the associations of household asset score and use of a shared toilet were preserved for all three growth parameters (Table 6.5). In addition, access to piped water was positively associated with the size parameter despite no previous relationship in a univariable analysis. Associations of other variables were either attenuated (positive association of four or more children in the household with tempo and a negative one with velocity), or no longer maintained (parental education variables, two or more adults in the household, pregnancy intention, paternal smoking, access to piped water) after adjustment for other factors in sex-adjusted models. However, pregnancy intention was associated with higher velocity in the sex-seasonality adjusted model (Table 6.6).

A one SD increase in household asset score was associated with lower length gain (-0.49 cm; SE 0.2, p= 0.0172), earlier tempo (-0.65 months, SE 0.16, p <0.0001), and greater velocity (4%, SE 1%, p= 0.0002). Children from households that used a shared toilet facility gained more length (1.19 cm, SE 0.48, p= 0.0132), but had later age at peak velocity (1.24 months, SE 0.3, p= 0.0007), and lower velocity (-9%, SE 2%, p= 0.0002). Children from households with four or more other children had delayed tempo (0.96 months, SE 0.3, p= 0.0014) and lower velocity (-8%, SE 2%, p= <0.0001). In the seasonality model, children born as a result of planned pregnancies grew 5% faster (SE 2%, p= 0.0161) over the course of the study.

Growth curves plotted by household asset score quartile (highest vs lowest) and type of toilet facility (shared vs own) showed that the early tempo and higher velocity among those who lived in more favourable conditions resulted in greater attained length at the end of follow-up (Figure 6.8 and 6.9). The maximum length velocity was greater among the higher SEP groups (1.123 cm/month vs 0.9442 cm/month for asset score and 1.162 cm/month and 0.9939 cm/month for type of toilet facility). For both SEP markers, the divergence in length between groups seemed to occur at around 12 months. The sex-adjusted model without seasonality terms fit slightly better than the one with sex-seasonality adjustment (BIC 59309 vs 59498).

Figure 6.8 Growth by asset score quartile from a sex-seasonality adjusted multivariable model

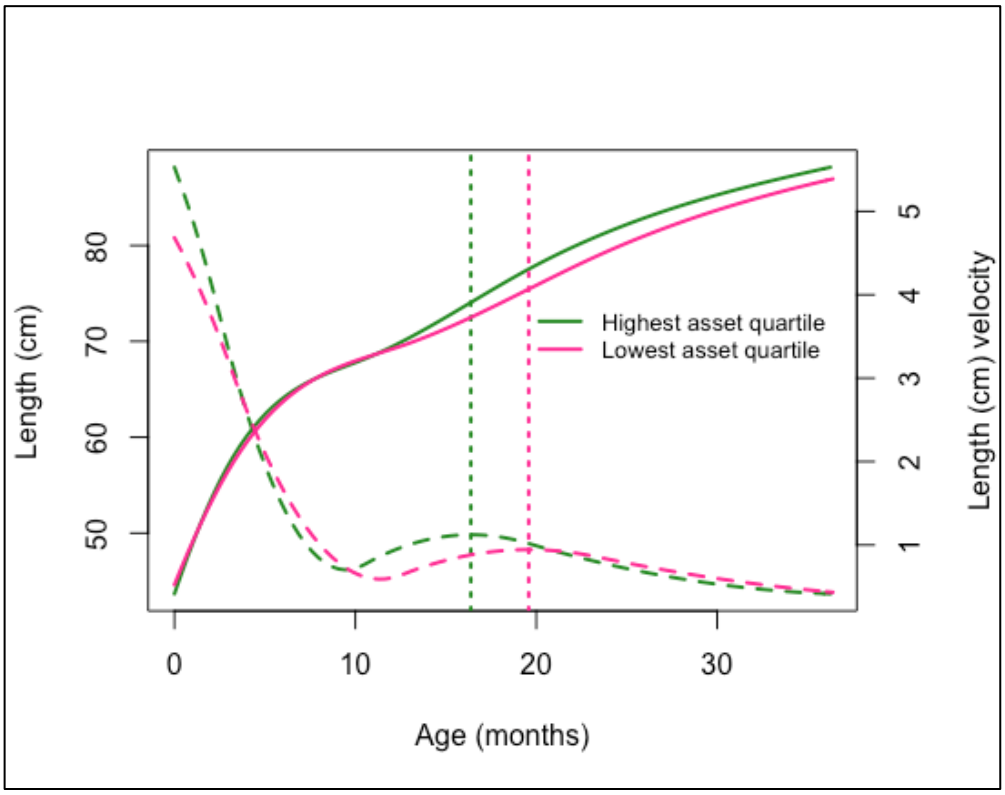


Figure 6.9 Growth curves by type of toilet facility from a sex-seasonality adjusted multivariable model

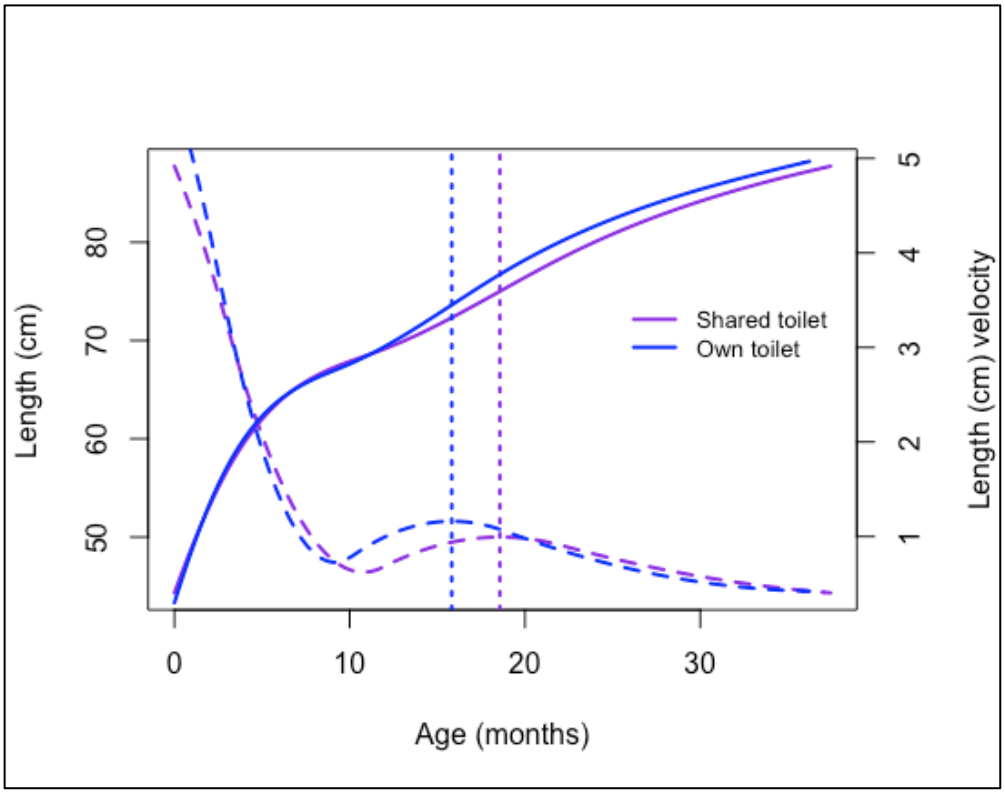


Table 6.5 Sex-adjusted multivariable SITAR model

	Size (a)			Tempo (b)			Velocity (c)		
Covariate	**Estimate (cm)**	**SE**	**p**	**Estimate (months)**	**SE**	**p**	**Estimate (proportion)**	**SE**	**p**
Female	-1.87	0.33	<0.0001	-0.14	0.26	0.5793	-0.010	0.02	0.5427
Maternal age ≥25	0.11	0.43	0.7893	-0.15	0.33	0.6544	0.02	0.02	0.4386
Paternal age ≥30	-0.31	0.42	0.4674	-0.11	0.33	0.7282	0.01	0.02	0.5441
Asset score	-0.49	0.20	0.0172	-0.65	0.16	0.0001	0.04	0.01	0.0002
Maternal education ≥6th standard	0.31	0.38	0.4241	-0.17	0.30	0.5786	0.03	0.02	0.1907
Paternal education ≥6th standard	0.00	0.35	0.9946	0.001	0.27	0.9921	0.01	0.02	0.7075
Access to piped water	0.76	0.36	0.037	0.41	0.28	0.1491	-0.01	0.02	0.5857
Shared toilet	1.19	0.48	0.0132	1.24	0.37	0.0007	-0.09	0.02	0.0002
≥4 children in the HH	0.49	0.38	0.1959	0.96	0.30	0.0014	-0.08	0.02	0.0001
≥2 adults in the HH	-0.01	0.38	0.9856	-0.18	0.30	0.5385	0.03	0.02	0.1935
Planned pregnancy	0.45	0.35	0.1896	-0.11	0.27	0.6904	0.02	0.02	0.2007
Paternal smoking	-0.12	0.35	0.7185	0.09	0.27	0.7451	-0.01	0.02	0.6985
Maternal smoking	-0.25	0.49	0.6009	0.05	0.39	0.8978	0.01	0.03	0.6859

Table 6.6 Sex-seasonality adjusted multivariable SITAR model

	Size (a)			Tempo (b)			Velocity (c)		
Covariate	**Estimate (cm)**	**SE**	**p-value**	**Estimate (months)**	**SE**	**p-value**	**Estimate (proportion)**	**SE**	**p-value**
Female	-2.31	0.36	<0.0001	-0.45	0.27	0.0968	0.01	0.02	0.5651
Maternal age ≥25	-0.50	0.47	0.2894	-0.52	0.35	0.1395	0.04	0.02	0.0642
Paternal age ≥30	0.11	0.46	0.8086	0.09	0.35	0.8009	-0.001	0.02	0.9771
Asset score	-0.58	0.22	0.0089	-0.63	0.17	0.0002	0.04	0.01	0.0005
Maternal education ≥6th standard	0.21	0.42	0.6211	-0.24	0.32	0.4596	0.03	0.02	0.1311
Paternal education ≥6th standard	0.12	0.41	0.7666	0.059	0.31	0.8495	0.002	0.00	0.9073
Access to piped water	0.71	0.39	0.0715	0.34	0.30	0.2621	-0.004	0.02	0.8355
Shared toilet	1.34	0.52	0.0104	1.42	0.39	0.0003	-0.11	0.03	0.0001
4+ children in the HH	0.37	0.42	0.3758	0.84	0.32	0.0084	-0.07	0.02	0.0006
2+ adults in the HH	-0.19	0.41	0.6378	-0.35	0.31	0.2629	0.04	0.02	0.0818
Planned pregnancy	0.10	0.38	0.7957	-0.45	0.29	0.1151	0.05	0.02	0.0161
Paternal smoking	-0.32	0.38	0.4015	-0.08	0.29	0.7805	0.002	0.02	0.9023
Maternal smoking	0.02	0.53	0.9731	0.26	0.40	0.5126	0.0004	0.03	0.9893

6.7 Relationships between parental anthropometry and linear growth

The associations of parental anthropometry with growth outcomes in sex-seasonality adjusted models are presented in Table 6.7. Exposure specifications with parental overweight as a categorical variable and two variables for the difference in maternal and paternal heights and weights did not show any associations with size, tempo, or velocity.

When maternal and paternal height and weight z-score were included as four separate variables, children with fathers who were 1 SD taller than average had greater length velocity (3%), after adjustment for both parents' weights and mother's height. In the model with sums of parents anthropometry z-scores as two variables, having taller parents was associated with earlier tempo (0.3 months) and 2% higher velocity, and children with heavier parents gained 0.37 cm more length. These patterns suggest that the influence of parental anthropometry works primarily through the combined contribution of both parents.

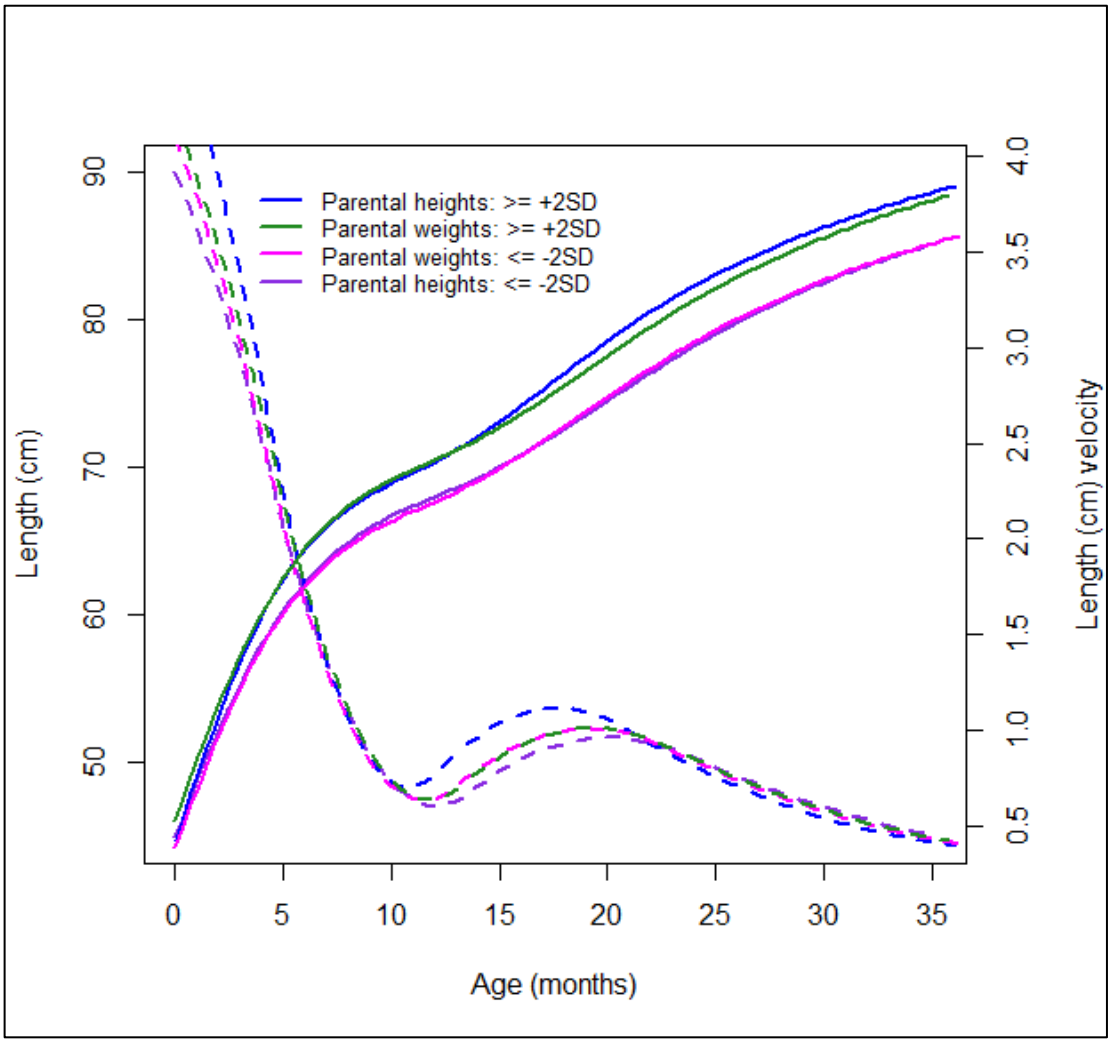
Table 6.7 Association of parental anthropometry with growth using different exposure specifications in sex-seasonality adjusted models

	Size (cm)			Tempo (months)			Velocity (proportion)		
Exposure specification	**Value**	**SE**	**p**	**Value**	**SE**	**p**	**Value**	**SE**	**p**
Parental overweight (ref Neither)									
Overweight father	0.24	0.62	0.6993	0.09	0.52	0.8543	0.01	0.03	0.8096
Overweight mother	0.35	0.69	0.6133	0.44	0.58	0.4462	-0.01	0.03	0.5946
Both parents	0.67	0.65	0.301	0.33	0.54	0.546	-0.01	0.03	0.8796
Sum of parental z-scores									
Height	0.18	0.17	0.303	-0.32	0.14	0.024	0.02	0.01	0.0011
Weight	0.37	0.17	0.0281	0.17	0.13	0.2338	-0.01	0.01	0.3945
Parental z-scores									
Maternal height	0.40	0.26	0.1275	-0.24	0.22	0.2774	0.03	0.01	0.0563
Paternal height	-0.04	0.25	0.8878	-0.39	0.21	0.0613	0.03	0.01	0.0183
Maternal weight	0.35	0.27	0.1882	0.27	0.22	0.227	-0.02	0.01	0.2393
Paternal weight	0.37	0.26	0.1477	0.06	0.22	0.7678	0.001	0.01	0.9206
Difference between parental height and weight z-scores									
Height	0.45	0.39	0.2479	0.18	0.33	0.5742	-0.01	0.02	0.7349
Weight	-0.05	0.40	0.883	0.20	0.34	0.5454	-0.02	0.02	0.3877

In a multivariable model for the influence of sum of parental anthropometry, the relationships with SITAR parameters were preserved after adjustment for all

background factors (Table 6.8). The positive influence of taller and heavier parents (+2 SD of the sums of height and weight distributions) was apparent in children's growth curves (Figure 6.10). Greater parental size conferred an advantage in terms of overall length across the full period, although the trajectories diverged substantially in the first few months of life. The velocity curves for children of lighter and heavier parents were similar, but those with taller parents had much greater velocity in the second year of life than those with short parents, indicating that after adjustment for parental weights and SEP, children of taller parents had faster linear growth.

Figure 6.10 Growth curves by parental size from a sex-seasonality adjusted multivariable model



In a multivariable model, a categorical parental overweight exposure variable did not show any relationship with growth outcomes (Table 6.9).

Table 6.8 Association of sum of parental heights and weights with SITAR growth parameters in a multivariable model

	Size (a)			Tempo (b)			Velocity (c)		
Variable	**Estimate (cm)**	**SE**	**p**	**Estimate (months)**	**SE**	**p**	**Estimate (proportion)**	**SE**	**p**
Parental heights	0.24	0.17	0.1496	-0.23	0.13	0.0986	0.02	0.008	0.0116
Parental weights	0.48	0.17	0.0061	0.25	0.14	0.0778	-0.01	0.008	0.091
Female	-2.07	0.48	<0.0001	-0.35	0.39	0.3615	0.0005	0.02	0.9824
Maternal age ≥25	-0.74	0.62	0.2282	-0.21	0.51	0.6688	0.03	0.03	0.3368
Paternal age ≥30	0.04	0.59	0.9344	-0.10	0.49	0.833	0.009	0.03	0.7643
Asset score	-0.61	0.29	0.0364	-0.58	0.23	0.0139	0.03	0.01	0.0235
Maternal education ≥6th standard	0.76	0.56	0.173	0.24	0.45	0.5935	0.01	0.02	0.6564
Paternal education ≥6th standard	-0.25	0.53	0.634	0.020	0.43	0.9492	0.003	0.02	0.9084
Access to piped water	0.83	0.51	0.1084	0.77	0.42	0.0686	-0.010	0.02	0.5463
Shared toilet	0.79	0.71	0.2639	1.24	0.58	0.0329	-0.08	0.03	0.016
≥4 children in the HH	0.43	0.54	0.4317	1.06	0.45	0.0188	-0.08	0.02	0.0023
≥2 adults in the HH	-0.16	0.53	0.7584	-0.27	0.44	0.5338	0.03	0.02	0.2594
Planned pregnancy	-0.25	0.50	0.6085	-0.68	0.41	0.0948	0.06	0.02	0.0064
Paternal smoking	0.73	0.49	0.1448	0.79	0.41	0.0524	-0.030	0.02	0.1775
Maternal smoking	0.18	0.69	0.7952	0.23	0.57	0.6794	0.0070	0.03	0.8342

Table 6.9 Association of parental overweight status with SITAR growth parameters in a multivariable model

	Size (a)			Tempo (b)			Velocity (c)		
Variable	**Estimate (cm)**	**SE**	**p**	**Estimate (months)**	**SE**	**p**	**Estimate (proportion)**	**SE**	**p**
Parental overweight (ref Neither)									
Overweight father	0.22	0.63	0.7249	0.09	0.51	0.8611	0.003	0.03	0.9016
Overweight mother	0.37	0.71	0.5956	0.42	0.57	0.4609	-0.02	0.03	0.4644
Both parents overweight	0.93	0.68	0.1738	0.67	0.56	0.2269	-0.03	0.03	0.2841
Female	-2.01	0.48	<0.0001	-0.38	0.39	0.3352	0.001	0.00	0.8894
Maternal age ≥25	-0.36	0.62	0.5554	-0.29	0.51	0.5593	0.03	0.03	0.2023
Paternal age ≥30	0.09	0.61	0.8738	-0.08	0.49	0.871	0.01	0.01	0.7799
Asset score	-0.50	0.29	0.0882	-0.59	0.24	0.0135	0.034	0.01	0.0199
Maternal education ≥6th standard	0.81	0.56	0.1497	0.22	0.46	0.623	0.01	0.02	0.6037
Paternal education ≥6th standard	-0.26	0.54	0.6179	0.040	0.44	0.9254	0.001	0.02	0.9477
Access to piped water	0.94	0.52	0.073	0.79	0.42	0.063	-0.010	0.02	0.5158
Shared toilet	0.78	0.72	0.2764	1.27	0.58	0.0295	-0.08	0.03	0.0142
≥4 children in the HH	0.23	0.55	0.6727	1.11	0.44	0.0129	-0.09	0.02	0.0009
≥2 adults in the HH	-0.11	0.54	0.8351	-0.28	0.44	0.5263	0.03	0.02	0.2326
Planned pregnancy	-0.26	0.51	0.5971	-0.69	0.41	0.0913	0.06	0.02	0.0064
Paternal smoking	0.62	0.51	0.2185	0.77	0.41	0.0608	-0.030	0.02	0.1901
Maternal smoking	0.21	0.70	0.763	0.22	0.57	0.6964	0.0100	0.03	0.8148

6.8 Discussion

6.8.1 Summary of findings

This is the first study to use the SITAR model to analyse linear growth in infancy and early childhood in urban informal settlements, and I was able to identify several factors that have an association with size, tempo, and velocity of growth in this age group.

I fitted the basic SITAR model to 16 753 length measurements for 944 children, modelled the average growth curve of children in the cohort, and estimated the relationships between size, tempo, and velocity. Higher length gain velocity was correlated with earlier tempo and lower length gain, indicating that children who had faster growth across the study were likely to attain peak velocity at younger ages and end up longer at the end of follow-up. On average, girls grew 1.97 cm less than boys, though there were no sex differences in velocity or tempo. Seasonality adjustment altered all three random effect SDs, delayed the tempo of the overall cohort, but did not affect the distance curve substantially.

In univariable analyses most markers of low SEP were associated with low velocity and later peak. Conversely, higher SEP was associated with high velocity and early tempo. After adjustment for multiple SEP markers and background variables in a sex-seasonality model, use of a shared toilet and being born into a household with four or more children were associated with lower length velocity (11% and 7% slower than average) and delayed age at peak velocity (1.4 and 0.8 months later than average). A one SD increase in asset score was associated with 4% higher velocity and an earlier peak (0.63 months). Distance curves showed that children from households with higher asset score and families with their own toilet attained greater length.

Children of taller parents were more likely to have higher growth velocity after adjustment for multiple SEP markers and parental weights.

6.8.2 Socioeconomic and parental determinants of linear growth

Since SITAR has not been used for linear growth outcome modelling in this age group before, it is difficult to make direct comparisons with other studies. The consistency of the pattern of relationships between SEP markers and growth in

univariable models is remarkable, though somewhat unsurprising since these factors were highly correlated (see Chapter 5). Most factors showed some relationship with growth parameters, in directions that indicated that children from more deprived families were likely to be shorter and grow slowly.

The three strongest predictors of lower length velocity and later tempo (which also manifested as shorter length in distance curves) in the fully adjusted model hint at an interesting web of factors. Collectively, they signify greater material deprivation, household overcrowding, and inadequate sanitation. These characteristics essentially define informal settlements (UN-Habitat, 2006). Greater informality was associated with lower length gain velocity and delayed tempo in this cohort. The three factors also operate at different levels, although they arguably represent nested relationships. While household asset score and number of children in the household operate in the home environment, type of toilet facility is likely to represent the constraints of the neighbourhood built environment that expose many children in an area to a similar level of sanitation.

The timing of influence is also meaningful. A recent longitudinal study from rural Ecuador looked at the 'herd immunity' that high coverage of sanitation in a community provides for linear growth among children under five, concluding that the protective influence was strongest in the second year of life for girls (Fuller et al., 2016). My analysis shows that the growth velocity of children from households with their own toilet was higher in the second year than that of those using a shared facility. The same pattern was evident for the other two SEP factors. While poor household and environmental conditions that children are born into could be associated with much lower length at birth, which leads to lower attained size in childhood, their association with children's growth trajectory appears to act strongly in the second year.

I was also able to tease apart the influence of maternal and paternal heights and weights. The more 'favourable' growth trajectory of higher velocity and early tempo was clearly driven by combined parental heights rather than weights, hinting at a likely heritable influence a genetic influence. But it is difficult to disregard the underlying influence of SEP factors past and present. The attenuation of the association with length velocity (3% to 2%) after SEP adjustment indicates that some of the influence of parental heights works through the better environment they provide for their children. Taller parents possibly grew better as children and

adolescents in supportive environments, leading to higher final height in adulthood, and were able to maintain these beneficial living conditions into their reproductive lives.

The manifestation of intergenerational relationships of maternal height and differential environments in infant linear growth outcomes has been observed in the MINIMat birth cohort in rural Bangladesh, with additional long-term associations with children's height at 10 years (Svefors et al., 2016). However, in the Hong Kong children of 1997 cohort, parents' education and mid-parental heights were associated with linear growth in infancy, but the influence did not extend into greater height in later childhood (Kwok et al., 2013), suggesting that the pathways acting to increase stature in each age group are probably context specific. It is therefore unclear whether the higher velocity of children of taller parents in my study will translate into greater height in later childhood or adulthood.

Further, since the cohort children were all born at term, it is possible that average maternal height was higher in the cohort than in the community they were drawn from, since shorter women are at greater risk of having preterm infants (Kozuki et al., 2015) and their infants would not have met the study inclusion criteria. If such a selection bias operates in this study, my current estimates of the relationship between parental anthropometry and growth could be biased downwards.

6.8.3 Does sex matter?

My finding that the pattern of growth (tempo and velocity adjusted for size) did not differ between the sexes corroborates findings from the urban Generation R cohort in the Netherlands. In a mixed effects growth model, boys were longer than girls, and the difference was statistically significant after 9 months, but there was no overall difference in the pattern of growth between birth and two years (Broere-Brown et al., 2016). However, the Alimfert cohort study in rural Senegal, which focused on sex differences in growth between birth and 39 months, found that boys had lower length velocity than girls (-0.025 cm/month) based on absolute height-for-age deficits in a mixed effects model (Bork and Diallo, 2017).

The SITAR model parameters are mutually adjusted and my interpretation is that, after taking into account differences in size across the study period, female children have the same growth velocity and tempo patterns as males. The difference in size

was magnified by only 0.3 cm across the full period after accounting for seasonality, and the lack of difference in velocity and tempo was sustained. If seasonality is interpreted as an age-related effect that manifests in the second year of life, female children fare slightly worse than males, but the additional influence is insignificant.

6.8.4 Strengths and limitations

The large number of length measurements used to model growth in this cohort is a major strength. I also used a method that utilized all available data. As the first study to use SITAR to model infant length as an outcome, I related several SEP and parental characteristics to size, tempo, and velocity. An important empirical finding is that factors that are most indicative of urban informality have the strongest influence on linear growth, and that their associations are most prominent in the second year of life. Using SITAR has the added advantage of examining these associations for each growth parameter while taking into account the other two. The only other study that used SITAR to model length in infancy in a LMIC urban population found that it explained 86% of the variance in length from 0-24 months (Jones-Smith et al., 2013), so the 88% for this cohort is slightly better.

However, the model did not fit perfectly. The residual standard deviation for the mean spline curve was 1.10 cm, and 1.08 cm in a sex-seasonality adjusted model. A TEM value for length is not available for the cohort as the exercise was conducted on adult heights. However, the residual is higher than the TEM reported for the WHO MGRS team (0.23 cm to 0.58 cm) that measured length data for young children (WHO Multicentre Growth Reference Study Group, 2006b). The child-specific residuals also showed that there were some outliers with unusually large values, though the IQR was -0.5 cm to +0.6 cm, indicating that the model fit most children's data well.

Another limitation is that it is difficult to compare my findings with other empirical studies that address the same topic, because none have applied the same modelling technique. Having demonstrated that the SITAR model can be used to identify factors associated with linear growth trajectories in early life, it is possible that other studies (new or old) would benefit from applying the model to length measurements in infancy.

The tempo parameter in the model is difficult to square with the growth process across childhood and adolescence. The infancy phase represents one part of human growth from conception to adulthood, and subsequent prepubertal and pubertal phases will see further spikes in the velocity of linear growth. It would be interesting to examine later stages of growth in this cohort to understand if the lower velocity of low SEP groups extends into prepubertal and pubertal growth and tempo.

6.8.5 Methodologic implications

My analysis raises further empirical questions about the determinants of linear growth in this cohort. Does the influence of SEP variables on growth in the second year of life reflect complementary feeding practices? Do exclusively breastfed infants have higher or lower growth velocity than mixed fed infants? How different are the growth trajectories of children who were fed according to WHO guidelines across the full 0-24 month period and those fed largely suboptimal diets? Since sanitation is associated with subsequent growth trajectories, does sanitation-induced diarrhoea across infancy and early childhood influence length? What happens when size at seven months influences caregivers' feeding decisions in that month and alters subsequent growth patterns?

The analysis also raises questions about methodology which are not related to how we can better analyse growth by choosing one model specification over another. The issue relates to whether a growth model like SITAR that uses all available data and produces interpretable growth parameters can incorporate theorized pathways and temporal relationships for hypothesis testing or estimation of effects.

In my analysis, I looked at associations between time-invariant factors and subsequent growth. However, several factors that have hypothesized causal effects on linear growth, such as diet and disease, change over time or act in specific periods. One common framework for the causes of linear growth faltering focuses on the time-sensitive effects of complementary feeding (Stewart et al., 2013). Beath's original shape invariant model provides one solution which could be extended to SITAR. The model can accommodate time-varying covariates, such that exposure at a particular age (which remains constant in that period) influences growth rate and size in that period, which then has a sustained effect for the rest of the follow-up period. The change in growth rate in response to such a covariate is achieved by varying the time scale, and the influence of the covariate accumulates

over time. Operationally, the covariate's value in each period is the cumulative duration for which it has each of its values. Beath demonstrated this by comparing the mean weight trajectory of infants who breastfed for six months to those who breastfed for the full first year.

While such a solution could work for my study and is straightforward enough to implement, it comes with several limitations. Based on the life course paradigm in epidemiology, the accumulation hypothesis tests the cumulative effect of an exposure as its duration or severity increases such that it leads to long term consequences that are irreversible (Kuh et al., 2003). Calculating the total time spent in a particular 'state' measured as a binary exposure would create a score for use as an exposure variable in regression analyses. The hazard of such an approach is that it disregards the timing of the exposure. When the greatest risk associated with an exposure is particular to a period (critical period model) or strengthens when an individual switches categories (mobility model), the likelihood of misclassification in an accumulation model is high. An individual with a high score who was unexposed in the critical period will have the same risk as someone who had a lower score but was exposed in the critical period. The accumulation assumption can produce misleading findings (Mishra et al., 2009).

Applying this to a time-varying exposure like infant and young child feeding (IYCF), the accumulation hypothesis would imply that two children with the same score (18 out of 24) have the same risk of a certain linear growth outcome irrespective of when they were fed the more nutritionally adequate diet. It is possible that one would have accumulated this score solely in the complementary feeding period (6-23 months) and the other at 0-11 and 18-23 months through a combination of exclusive breastfeeding and intermittent high quality complementary feeding. The analytic model would draw an equivalence between a feeding trajectory based on poor breastfeeding and good complementary feeding and another based on somewhat poor complementary feeding following good breastfeeding. The relationship between IYCF and growth would shed no light on whether exclusive breastfeeding plays a critical role in promoting good nutrition, or if complementary feeding is most beneficial when children successfully accumulate its effects across the full period.

As a general principle, applying more than one modelling approach in a life course study produces greater mechanistic insight (De Stavola et al., 2006). Latent growth

curve modelling is more suited to such questions because it can incorporate the structural equation modelling framework (Johnson, 2015). It is currently not possible to test competing life course mechanisms of the relationship between IYCF and growth outcomes in the SITAR model, and this area needs further development if SITAR is to be successfully applied to more mechanistic inquiry in early childhood nutrition and growth.

Chapter 7 Infant and young child feeding

Summary

In this chapter I describe infant and young child feeding practices in the cohort. I begin with descriptive cross-sectional analysis of data at each age, focusing on breastfeeding from 0-5 months and complementary feeding (6-23 months), summarising indicators of exclusive / predominant breastfeeding, dietary diversity, animal source foods, and sweet and salty snack foods. I then present longitudinal analyses of the determinants of discontinuation of exclusive / predominant breastfeeding before five months, and introduction to solid foods by eight months in discrete time survival analyses. I analyse the determinants of dietary diversity, consumption of animal source foods, and snacks between 6-23 months using autoregressive models. I contextualise my findings in relation to recent studies, and discuss the implications of my findings for global child nutrition and methodologic development.

7.1 Introduction

Infant and young child feeding (IYCF) is a time-related process. Nutritional requirements change between birth and two years, and patterns of feeding exhibit a progression from breastfeeding to complementary feeding as children transition to family foods (Brown et al., 1998a, Agostoni et al., 2008) and increasing calorie requirements (Figure 7.1).

Figure 7.1 Increasing energy requirements of infants and young children

Note: figure removed due to copyright restrictions. See Brown et al., 1998a (pp.5) for the original figure.

Source: (Brown et al., 1998a)

Findings from qualitative studies in urban informal settlements suggest that several factors lead to suboptimal IYCF practices, and that some may be specific to local culture and social structures. These include a trade-off between economic activities and exclusive breastfeeding (EBF) in the absence of adequate support at home or in the workplace for lactating women in informal settlements in Dhaka, Bangladesh (Kabir and Maitrot, 2017), and the cultural belief among women in Nairobi that breastfeeding in public attracts the 'evil eye' (Wanjohi et al., 2016). A study in Lima, Peru, highlighted that women attempted to stop breastfeeding in the first year of life, sometimes as early as three months, due to time commitments or health-related reasons. However, if infants reacted negatively to discontinuation of breastfeeding, mothers re-instated it. The authors suggested that such a weaning-relactation cycle was considered acceptable in the local community, and many women went through several cycles before stopping breastfeeding permanently (Marquis et al., 1998).

Another study in flood-prone areas of Dhaka described how heavy monsoons each year disrupted daily life in informal settlements and affected breastfeeding as well as complementary feeding practices. Increased household food insecurity and reduced cooking facilities meant that children were fed suboptimal complementary foods, and women's experience of food insecurity led to lactation difficulties (Goudet et al., 2011).

These qualitative findings suggest that IYCF patterns in informal settlements may be influenced by factors that are particular to the environment or socioeconomic features of urban poverty, as well as temporal influences such as seasonality or fluctuating economic activity. Quantifying such temporal relationships requires longitudinal data, as well as suitable analytic methods to operationalise hypotheses of IYCF practices.

In the context of urban informal settlements in Mumbai, longitudinal data provide an opportunity to quantify IYCF patterns and their temporal properties, and understand how practices are influenced by socioeconomic and household factors. Repeated measurements can be used to identify factors that shape the maintenance of exclusive or predominant breastfeeding in the first six months of life and the age at which infants are most likely to first receive solid food. It is also possible to understand how complementary feeding practices are correlated over time – for example, if previous consumption determines current consumption – and also with background covariates: for example, if current consumption is associated with socioeconomic position or parental characteristics after accounting for any influence of previous consumption.

7.2 Research question and objectives

In this chapter, I address Research Question 5: What are the determinants of infant and young child feeding?

The four specific objectives were:

1. Describe frequencies of age-appropriate breastfeeding, complementary feeding and snack food consumption in the cohort at each follow-up age.
2. Determine the probability of discontinuing exclusive / predominant breastfeeding between 0-5 months, and its associations with baseline variables.
3. Determine the probability of infants receiving soft, semi-solid and solid food for the first time between 6-8 months, if they had not yet been given any non-liquid items, and its association with baseline variables.
4. Determine the association of complementary feeding practices with feeding in adjacent periods as well as baseline variables.

7.3 Methodologic challenges

Analysis of longitudinal IYCF data poses methodologic challenges related to sources of autocorrelation and the depiction of feeding practices as states, events, or processes. These challenges shape the choice of statistical approach and the validity of implicit assumptions, and expose the limitations of standard methods of analysis.

Repeated IYCF data on the same child are correlated and the magnitude and strength of these correlations can have different sources. While socioeconomic factors (Joshi et al., 2012, Nguyen et al., 2018, Patel et al., 2012) and innate taste preferences (Beauchamp and Mennella, 2009, Mennella et al., 2001) influence IYCF practices at a given point in time, they can also shape individual feeding patterns and trajectories. Some factors may engender habitual intake more than others. Part of the autocorrelation of IYCF data will stem from the long-acting impact of observed baseline factors that affect food consumption at any age. The influence of food intake at younger ages, and that of unobserved or unknown factors, are other sources of autocorrelation.

For example, a child's consumption of carrot purée at eight months is likely to influence whether they eat carrots again at nine months. This correlation could depend on the underlying influence of family income that affects the caregiver's ability to buy and cook carrots regularly, and also the child's preference for carrot acquired through in-utero exposure to carrots in the maternal diet (Mennella et al., 2001, Mennella et al, 2012). The unmeasured extent of the mother's exposure to a nationwide health campaign to improve IYCF could also contribute to any observed correlation between carrot consumption at eight and nine months.

Further, many IYCF practices can be described as states rather than events because infants are expected to spend some time in the state of being breastfed or eating cereal-based food for breakfast every morning once they have been introduced to solid foods. A feeding practice can therefore span more than one time point. However, some components of IYCF can also be characterised as events. For example, when an infant exclusively breastfed since birth is given cow's milk for the first time, the cessation of exclusive breastfeeding is an 'event' that occurs at a particular time (e.g. 12.2 weeks) or within a broader time interval (e.g. third month). Introduction to semi-solid and solid foods for the first time could also be an event,

which ought to ideally occur at 6-8 months according to WHO guidelines. The availability of repeated or time-related measurements on the same individual is essential for statistical analysis to estimate probabilities of an 'event' occurring. Repeated measurements are also required to accurately classify information on the timing of changes from one feeding 'state' to another, or the probability of being in a certain state at a given time.

IYCF data with repeated observations on the same individuals over time could be analysed using two (of several) types of analysis in order to maximise all available information and answer complementary research questions. These comprise, first, survival analysis to analyse time to an event of interest, and second, longitudinal analysis (random-effects, multi-state or other models for recurrent data) to characterise recurrent feeding practices accounting for the correlated nature of a child's diet over time.

Depending on the research question, there are several ways to analyse data on recurrent events or phenomena. A common motivation across methods is to avoid wasting information on subsequent events which would be discarded in regular survival analysis techniques which only use data on the first event of interest (Twisk et al., 2005). A further consideration is use of information on the timing of events, and whether specific methods are able to address the relationship between events and time, either as rates, durations, or times at which they occur (Amorim and Cai, 2015). Finally, it is important to also account for dependence between recurring events where this is a possibility, an assumption violated by straightforward logistic regression or single-failure survival analysis techniques (Twisk et al., 2005).

Several recent studies have adopted survival analysis and longitudinal techniques to understand IYCF practices across a range of contexts. One sub-group analysis of trial surveillance data from Uganda included single- and multiple-event Cox regression and two-state Markov models to study the dynamic nature of switching between predominant breastfeeding and mixed-feeding, and the risk of breastfeeding cessation (Chola et al., 2013). A more recent study in rural Bolivia used dynamic or autoregression models to understand changes in maternal sleep behaviour over the seasons in response to nocturnal breastfeeding (Vitzthum et al., 2018). The association of pre-pregnancy maternal BMI and gestational weight gain with duration of breastfeeding among the 2004 wave of the Pelotas cohort in Brazil was examined using Cox proportional hazards regression (Castillo et al., 2016). A

pooled analysis of three birth cohorts in South India also used Cox regression to understand determinants of EBF in urban informal settlements (Velusamy et al., 2017).

Use of longitudinal techniques within the complementary feeding period with IYCF as an outcome is less common, though not entirely absent from the literature on LMICs (Bhargava, 2016, Saha et al., 2008a, Saha et al., 2008b). Even when longitudinal data are available, studies sometimes use simplistic analysis techniques. For example, a recent article describing complementary feeding in the Brazilian arm of the MAL-ED cohort used simple logistic regression to understand reasons for very early introduction of complementary feeding, despite having twice-weekly IYCF data up to the eighth month in an urban low-income community (Maciel et al., 2018).

In each of these examples, use of survival or longitudinal techniques has led to novel insights on the epidemiology and context of IYCF practices.

7.4 Methods

I carried out data analysis in four segments.

First, I used data at each age to produce population-level descriptive summaries of age-appropriate IYCF practices, ignoring drop-out and intermittent non-response. I did this for indicators of breastfeeding (0-5 months) and then for complementary feeding (6-23 months).

Second, I used survival analysis techniques to estimate the mean duration of exclusive breastfeeding and predominant breastfeeding (0-5 months) as survival functions, and determined their association with baseline characteristics.

Third, I estimated the probability of infants being introduced to semi-solid and solid foods between six and eight months, if they had not yet been given any non-liquid items, as a hazard function, and examined its association with baseline characteristics.

Fourth, I used dynamic autoregressive models for repeatedly measured complementary feeding data (6-23 months) to understand the influence of previous complementary feeding and baseline characteristics on current practices.

A common feature of analyses in this chapter is the treatment of time variables as discrete rather than continuous. The date of IYCF data collection was always recorded, and in principle it was possible to calculate each child's exact age on each measurement occasion. However, even though IYCF data collection in each month was based on a 24-hour recall period, I have made the simplifying assumption that this measurement represents the child's habitual diet in that integer month. For survival analysis (Objectives 2 & 3), I assume that the outcome (discontinuation of EBF, or introduction to solids) could have occurred at any point in that integer month rather than on the day before the home visit. For repeated measures analysis (Objective 4), I assume that children spent the full month of measurement in that 'state', for example, receiving animal source foods or eating two types of snack item.

This treatment of time as categorical is known as interval censoring (Zhang and Sun, 2010). I grouped age values into monthly intervals. For example, age data from four children at 13.4, 13.1, 13.7, and 13.9 months would all take the value 13. While this could be problematic since those assessed at 13.1 months are in reality closer to those of age 12.9 months rather than those who are 13.9 months old, I did not consider this problematic. Aggregation could in theory lead to loss of information on the timing of change and also bias estimates (Steele, 2011), but in this cohort the distribution of exact age is a marker of the distribution of IYCF data collection activities over a calendar month rather than the distribution of ages at which breastfeeding cessation (or another IYCF practice) took place. It would not have been feasible to obtain a direct and exact measure of IYCF every day of the month, or at exactly the same age for all children.

Treatment of time as a discrete interval for IYCF differs from the analyses presented in Chapter 6 where age was treated as a continuous measurement for growth modelling. The assumption was valid for anthropometry because the date of observation accurately reflected the real magnitude (as measured, momentarily disregarding measurement error) of the variable of interest at that time point.

Interval censored data preclude certain types of single or multiple failure survival analyses, and their use is limited to methods that are less flexible than models such as Cox regression (and its more flexible equivalents) for continuous outcomes.

Stata .do files used for all analyses presented in this chapter are in Appendix 7.1.

7.4.1 Dataset

The original cohort dataset was in long format and unbalanced. It contained multiple observations per participant (long format), but with gaps such that individuals had data only for occasions on which they had responded (unbalanced). Responses to baseline questions were recorded as variables that did not change across observations. A sample of the original cohort data format in Table 7.1 shows unequal numbers of observations for three children. This person-time format is the structural basis for several longitudinal analysis methods, and can incorporate time-fixed as well as time-varying information.

Table 7.1 Mock data for three participants showing long format of original dataset

ID	Date of birth	Measurement occasion	Age	Baseline survey variables			Variables containing time-varying IYCF data				
id	**dob**	**visitdate**	**agem**	**mage**	**fed**	**….**	**ebf**	**pbf**	**mdd**	**asf**	**…**
1	01jan2018	05jan2018	0	27	1		1	1	0	0	
1	01jan2018	07feb2018	1	27	1		0	1	0	0	
1	01jan2018	10mar2018	2	27	1		0	0	0	0	
2	10jan2018	13jan2018	0	31	0		0	1	0	0	
2	10jan2018	19feb2018	1	31	0		0	1	0	0	
2	10jan2018	……	…	..							
2	10jan2018	11sep2018	8	31	0		0	0	0	1	
3	19jan2018	23jan2018	0	23	1		1	1	0	0	
3	19jan2018	28mar2018	2	23	1		1	1	0	0	

For analysis of baseline information on initiation of breastfeeding, I tagged one observation per individual and used it to analyse a subset of observations. For descriptive analysis of data at each age, I tagged one response per integer month per child, using the first measurement in each month for those who had been visited more than once within the interval. Longitudinal data analysis was based on the same dataset, but with additional manipulation to prepare data for survival or longitudinal analyses (described in subsequent sections).

I right-truncated the dataset at 24 months, such that all information on IYCF collected after children's second birthday was excluded from analysis. For analysis

of exclusive and predominant breastfeeding, I right-truncated the dataset at six months, such that any additional information on breastfeeding after children turned six months old was ignored, even if they continued to be exclusively breastfed in the sixth month. For analysis of timing of introduction to semi-solid and solid food, I right-truncated the dataset at 12 months, once the window of interest (6-8 months) had passed and infants had been observed beyond that period while they were still likely to be introduced to food. For complementary feeding, I left-truncated the dataset at six months, such that any information on the indicator of interest before six months was ignored, even if individuals had met the indicator at younger ages. Intervals selected for truncation were based on durations indicated in the WHO guidelines on age-appropriate IYCF practices (WHO 2008, 2010).

7.4.2 Descriptive cross-sectional analysis at baseline and each follow-up age

I calculated summary statistics (means, medians, proportions) for variables related to prelacteal feeding and initiation of breastfeeding using data collected at the baseline visit. I cross-tabulated different types of prelacteal feed to identify items commonly given together.

I analysed data collected at each post-baseline age in two groups. First, I analysed data on exclusive breastfeeding (follow-up visits from 1 to 5 months), and then on complementary feeding (follow-up visits from 6 to 23 months). I did not summarise data on timing of introduction to solid, semi-solid and soft foods using repeated cross-sectional information, restricting their use to subsequent survival analysis.

For the early breastfeeding period (0-5 months), I first plotted the number of times infants were breastfed in the 24-hours before data collection for each month as box-and-whiskers plots of number of total, day-time, and night-time feeds. Using point-in-time methods (Greiner, 2014), I calculated the proportion of children at each age who were exclusively or predominantly breastfed in that month. I ignored breastfeeding status at previous time points and infants could switch from one type to another. I thus created snapshots of the population prevalence of breastfeeding practices at each age. I also examined the frequency of reported formula feeding. I then identified items that were most commonly responsible for loss of exclusive or predominant breastfeeding status in each month.

For the complementary feeding period (6-23 months), I followed a similar strategy, creating snapshots of the cohort at each age. I focused on indicators of complementary feeding quality and calculated the proportion of children at each age whose diets met criteria for minimum dietary diversity (four of seven food groups), consumption of animal source foods (dairy, flesh foods, or eggs), and consumption of any fruit or vegetables. For children who consumed any animal source foods, I calculated the proportions who consumed one, two or all three types at each age. I also calculated the proportion of infants who continued to receive breastmilk alongside complementary feeding.

I analysed data on snacks using three broad categories: sugary or sweet foods (e.g. chocolate, candy), salty foods (crisps or puffs, instant noodles, *vada pav*), and sweet beverages (tea, cold drinks). I first described the proportions consuming none, one, two, or all three types of snack in each age group. I then disaggregated data on solid snacks (any, sweet, or salty) and beverages (any, tea, or cold drinks) to identify items that were more frequently consumed at each age.

I did not examine associations between baseline characteristics and IYCF practices at each age, as this would have wasted the longitudinal element of the cohort study data and would also have led to spurious associations.

7.4.3 Life-long definitions of exclusive and predominant breastfeeding

Based on methods for life-long data on breastfeeding (Greiner, 2014), I used follow-up information on breastfeeding status in each month to calculate the proportion of infants who were exclusively or predominantly breastfed from 0-5 months. I first identified infants who had completed all follow-up visits in this period, and then a further subset who had at least one measurement in two-month intervals in this period (0-1, 2-3, 4-5 months). I categorized breastfeeding status in each interval using two binary variables, EBF and PBF, for both subsets of infants (data in every month and data in two-month intervals).

In order to understand how many infants continued to be exclusively or predominantly breastfed at each age, I used descriptive survival analysis methods. Data for each child were identified by a unique ID number, a variable for integer age (0-5) and their EBF or PBF status in each month to declare the dataset as survival data. The first age at which infants' EBF or PBF status was compromised was used

to identify ‘events’. I plotted these as survival curves displaying the proportion of infants still being exclusively / predominantly breastfed in each month.

I also calculated this information as survival probabilities in each month using Stata’s *sts list* command for survival data, treating time as a continuous variable (range 1-6). I did this for illustrative purposes. I wanted to simplify analysis and graphical display, to produce confidence intervals for estimated survival probabilities, and to aid comparison of duration of breastfeeding calculated using life-long (longitudinal) and point-in-time (cross-sectional) methods from the same dataset.

7.4.4 Breastfeeding (0-5 months)

I used discrete-time logit models to analyse data on breastfeeding (0-5 months). I first used age at exclusive breastfeeding cessation as an outcome variable, and then repeated the analysis with age at predominant breastfeeding cessation as the outcome to understand when discontinuation was most likely to occur and which baseline factors were associated with cessation at any age.

In order to maximise the amount of information available despite data gaps for many children, I used wider time-intervals for this analysis. I used information from the first follow-up visit (at one month) as each child’s status in the first interval. I combined data from the second and third months as the second interval, and from the fourth and fifth months as the third interval. Within each of these, I used either the second (later) measurement if there was no change in breastfeeding status, or the measurement that recorded discontinuation of EBF or PBF if it occurred in that period.

The person-period dataset contained one observation for each interval in which an (exclusively or predominantly breastfed) infant was at risk of stopping exclusive or predominant breastfeeding. The dataset included a variable indicating time interval (range 1-3), and another variable indicating the failure event. The failure variable was coded 0 for every month in which the infant continued to be breastfed exclusively or predominantly and 1 for the interval in which the infant was no longer exclusively or predominantly breastfed. Infants who had not yet experienced cessation of EBF or PBF by five months month (third interval) were censored, in that their value for the failure variable was 0 in all intervals. Additional identifier and time-

invariant variables of interest were also retained, and these had the same value for all observations for a child. Table 7.2 shows the data format for analysis, in which subject 1 was censored in the third interval, subject 2 stopped being exclusively breastfed in the third interval, and subject 3 stopped in the second interval.

Stata 13 SE does not offer a dedicated suite of commands for discrete-time survival models similar to those available for continuous-time models (such as *stcox*), so I used existing tools for logit models for a binary outcome (Rabe-Hesketh and Skrondal, 2012). These are discrete-time approximations of Cox regression models, with effect estimates interpreted as hazard ratios. Logit models produce regression co-efficients which can be exponentiated using post-estimation commands to produce hazard ratios.

Table 7.2 Data format for discrete-time logit models

ID	Baseline survey variables			Time interval	Failure variable
id	**water**	**fed**	**….**	**int**	**stop**
1	1	1		1	0
1	1	1		2	0
1	1	1		3	0
2	0	0		1	0
2	0	0		2	0
2	0	0		3	1
3	1	0		1	0
3	1	0		2	1

Note: *int* variable coded: 1 = 1st month; 2 = 2nd to 3rd month; 3 = 4th to 5th month.

I calculated the discrete-time hazard, or the conditional probability of infants stopping EBF or PBF, in an interval if they had been exclusively or predominantly breastfed until then. I fitted a logit regression model using dummy variables for intervals 2 and 3 as covariates, and used predicted probabilities from the model as estimates of discrete-time hazards in each interval, which were the same for all children. I plotted the hazard to understand how it increased or decreased with age.

After fitting a basic model, I examined the effect of background factors on hazard of discontinuing EBF or PBF in any time interval. The change in probability of discontinuation with time is captured by the baseline hazard function, defined as the hazard when all covariate values are equal to zero. In discrete-time models the time-dependency of the baseline function must be specified as either a polynomial (usually quadratic) function or a step function (dummy variable for each interval) (Steele, 2011). I specified the time-dependence of the hazard function by including a

quadratic term for age, with *time interval* and (*time interval*)^2 as covariates in one model, and a step function in another. I compared coefficients between the two and used the quadratic term since there was little difference in results.

I then tested crude associations between background covariates and the hazard of discontinuing EBF or PBF. After identifying those that had a p-value below 0.1 in crude analysis, I fitted a reduced model with just these covariates. I also fitted a full model with all hypothesized covariates, regardless of crude associations. I plotted predicted hazards for groups defined by background covariates that had the largest association with discontinuation of EBF / PBF.

The non-proportional hazards assumption is implicit in standard Cox regression as well as discrete-time models which approximate a Cox model (Steele, 2011). In order to test for non-proportional effects of background covariates, i.e., to allow the ratio of hazard of discontinuing EBF / PBF between two groups (for example, male and female infants) to change over time, I extended the model by including interactions between the background covariate and the time interval variable, and also with (*time interval*)^2, i.e., (covariate x time and covariate x time^2) in the same model. I carried out a Wald test to assess non-proportionality by testing whether the coefficients of the two interaction terms (*baseline variable x time* and *baseline variable x time^2*) were both zero.

I interpreted the hazard odds ratios from the discrete-time models as the odds of discontinuing exclusive or predominant breastfeeding in any given interval if cessation had not yet occurred.

7.4.5 Introduction to solid, semi-solid and soft foods

For analysis on timing of introduction to solid, semi-solid and soft foods I replicated the discrete-time survival analysis described above, but with some key differences.

First, I used data from the full first year of life, and divided it into four time intervals of varying width (t_1 = 1-3 months, t_2 = 4-5 months, t_3 = 6-8 months, and t_4 = 9-11 months). I also tested the analysis with six intervals (t_1 = 1-3 months, t_2 = 4-5 months, t_3 = 6 months, t_4 = 7 months, t_5 = 8 months, t_6 = 9-11 months), but with reduced sample size due to intermittent non-response, and compared results from both. Second, in crude and multivariable analyses of the influence of background

covariates, I restricted the data to below nine months of age since few children had not been introduced to any non-liquid food by the end of this period.

I interpreted the hazard odds ratios as the odds of introducing complementary feeding in any interval (before nine months) if it had not yet taken place.

7.4.6 Complementary feeding period (6-23 months)

I used autoregressive (AR) response or dynamic panel or lagged outcome modelling for repeated measures data to understand how complementary feeding in one period was influenced by feeding in the previous period as well as baseline characteristics.

Autoregressive models allow an individual's outcome at occasion *t* to be predicted by their outcome at occasions before *t*, by including previous responses as covariates or predictors in a multi-level model. Models that only include the outcome at occasion *t*-1 as a predictor of the outcome at occasion *t* are known as first-order lagged models, sometimes called AR (1) dynamic models. A key assumption made in first-order models is that the outcome at time *t*-1 is an adequate measure of the effects of the outcome at occasions before *t*-1. This is known as the first-order Markov assumption (Steele, 2014b).

Two key concepts in autoregressive models are state dependence and unobserved heterogeneity. State dependence, encapsulated by the first-order Markov assumption in AR (1) models, refers to the pattern of dependence between consecutive measurements due to the (assumed) causal effect of the outcome at *t*-1 on the outcome at *t*. Unobserved heterogeneity is a further source of autocorrelation, implying that there is an individual effect that has an even influence on an individual's outcome at any occasion. Unobserved heterogeneity could arise due to a combination of unmeasured factors, for example, a child's innate taste preferences and the family's cultural practices around food. Both state dependence and unobserved heterogeneity and their relative contribution to observed associations can be quantified in AR (1) models (Steele, 2014).

Data for autoregressive models must be derived from fixed measurement occasions that are equally spaced, as the correlations between consecutive measurements are assumed to be constant for any given lag (Steele, 2014). I divided the

complementary feeding period into six discrete three-month intervals, t_1 = 6-8 months, t_2 = 9-11 months, t_3 = 12-14 months, t_4 = 15-17 months, t_5 = 18-20 months, and t_6 = 21-23 months. I summed each child's feeding information within each interval into separate binary indicators of their diet at that age, for minimum dietary diversity (MDD), consumption of animal source foods (ASF), and consumption of two or more types of snack food or drink item.

In this analysis I did not restrict the dataset to children with complete data or a certain amount of data in each interval. Autoregressive models accommodate unbalanced datasets in which individuals contribute varying numbers of observations across the analysis period. I first calculated the proportion of visits at which each infant had met the indicator within each time interval. I tabulated the proportions who met the indicator at all visits (always), some visits (sometimes), or none (never) in each interval. I then created a binary indicator for each IYCF practice by combining the category that was consistently smallest over time with the adjacent category. For example, IYCF indicators were coded as Never vs Ever (MDD, and consumption of two or more snacks) or Always vs Never/Sometimes (ASF) within each time interval.

I also created a variable encoding the response in the previous period for intervals t_2 to t_6, but only when there were no gaps between consecutive intervals. For example, if an infant had data for t_1, t_3, t_4, t_5, and t_6, I calculated the lagged response for t_4, t_5, and t_6, but not for t_3 since the child was missing data for t_2. Using the value of the outcome at t_1 as a proxy for data at t_2 would have led to unequal spacing, violating a key assumption of AR(1) models.

An obvious problem that arises due to the one-lagged response model is that the lagged outcome for t_1 will always be missing, and therefore all observations at t_1 will be dropped from a regression model. A further problem is that residuals in an AR model will be correlated with covariates, violating an assumption made in regression analysis, which makes estimation of AR models using standard regression approaches problematic. This is known as the initial conditions problem. Ignoring this can lead to estimates of state dependence that are biased upwards, a further downward bias in estimates of unobserved heterogeneity, and biased estimates of the influence of covariates. The initial conditions problem can be addressed by joint modelling of outcomes at all occasions.

Following the methods outlined in Steele (2014), I carried out autoregression analysis using random effects models in two stages, with identical methods for three separates sets of analyses for MDD, ASF, and snack foods. In the first stage I ignored the initial conditions problem, and in the second stage I attempted to tackle it.

I first fitted a random-effects logistic regression model, Model 1, using *xtlogit* in Stata for longitudinal panel data for time intervals t_2 to t_6, with the child's ID as the group identifier. IYCF (MDD, ASF, or snacking) in the current interval was the outcome variable and the lagged outcome and time interval were covariates. The model was fitted using the Gauss-Hermite quadrature with 12 integration points, which is the default option in Stata 13 SE. After fitting the models, I carried out quadrature checks, which showed that changing the number of integration points to 8 or 16 would not affect the model coefficients substantially.

I examined the coefficient and p-value of the lagged outcome variable to assess evidence of state dependence. I looked at the between-child variance (by squaring the *sigma_u* parameter from the Stata output), proportion of total variance attributable to the child-level variance component (using the *rho* parameter from the Stata output or $sigma\_u^2/(sigma\_u^2 + 3.29)$, where 3.29 is the fixed occasion-level variance in a logit model (Steele, 2014a)), and likelihood ratio test of no difference between the occasion-level and child-level estimator (i.e, of *rho*=0) to assess evidence of unobserved heterogeneity. I converted the coefficients of the lagged outcome and between-child standard deviation to odds ratios and 95% confidence intervals to aid interpretation.

In order to model the initial condition, I specified the same substantive model as in the first stage (t_2 to t_6) and estimated it jointly with a model for the outcome in the first interval (t_1), the initial condition. I did this using the *gsem* suite of commands in Stata for generalized structural equation models that work with multi-level data. I modelled the initial condition in two ways. Model 2 was estimated simultaneously with Model 1, but the random effect coefficient in the logit model was constrained to one. Model 3 was also estimated simultaneously with Model 1, but the random effect coefficient was unconstrained. The difference between Models 2 and 3 is that Model 3 allows for the relationship of the child-level variance with occasion to differ (and therefore be freely estimated) between the first (t_1) and subsequent (t_{2-6}) occasions,

whereas Model 2 is simpler by constraining the occasion-level random effect co-efficient to 1 for both sets of intervals (t_1 and $t_{2\text{-}6}$).

I then compared estimates of the lagged outcome and between-child random effect across Models 1, 2, and 3 in order to understand whether ignoring the initial condition (Model 1) overstated state dependence and underestimated unobserved heterogeneity (Models 2 and 3). I also compared Models 2 and 3 using a likelihood ratio test (LRT).

I fitted the appropriate model (2 or 3) to estimate crude associations of each complementary feeding practice with background covariates, using those with a p-value <0.1 in a reduced multivariable model. I also fitted a full model with all hypothesized covariates. I calculated odds ratios for each model using a post-estimation command, *estat form*, from the gsem suite.

I interpreted the OR for the lagged outcome as the odds of an infant eating animal source foods in an interval if they had consumed some in the preceding interval. I interpreted the OR for the between-child random effect as the odds of consuming ASF for an infant whose unobserved characteristics put them one SD above the mean compared to an average infant with the same observed characteristics. I interpreted the *rho* estimate as the proportion of variance in the latent propensity to consume ASF, in addition to that explained by observed background covariates, attributable to other characteristics of the infant. Similar interpretation applied for analyses of dietary diversity and snack food consumption.

7.5 Cross-sectional IYCF summaries

7.5.1 Initiation of breastfeeding

Of 971 (99% of 978) women who responded to the baseline survey questions on breastfeeding, 953 (98%) had ever breastfed their infants and 820 (86%) of them had initiated breastfeeding on the day of birth (mean 1.6 hours (SD, 3.7) after birth). Among the 133 who initiated breastfeeding after the day of birth, the median number of days was two (IQR 1-3), and 95% began breastfeeding within the first week.

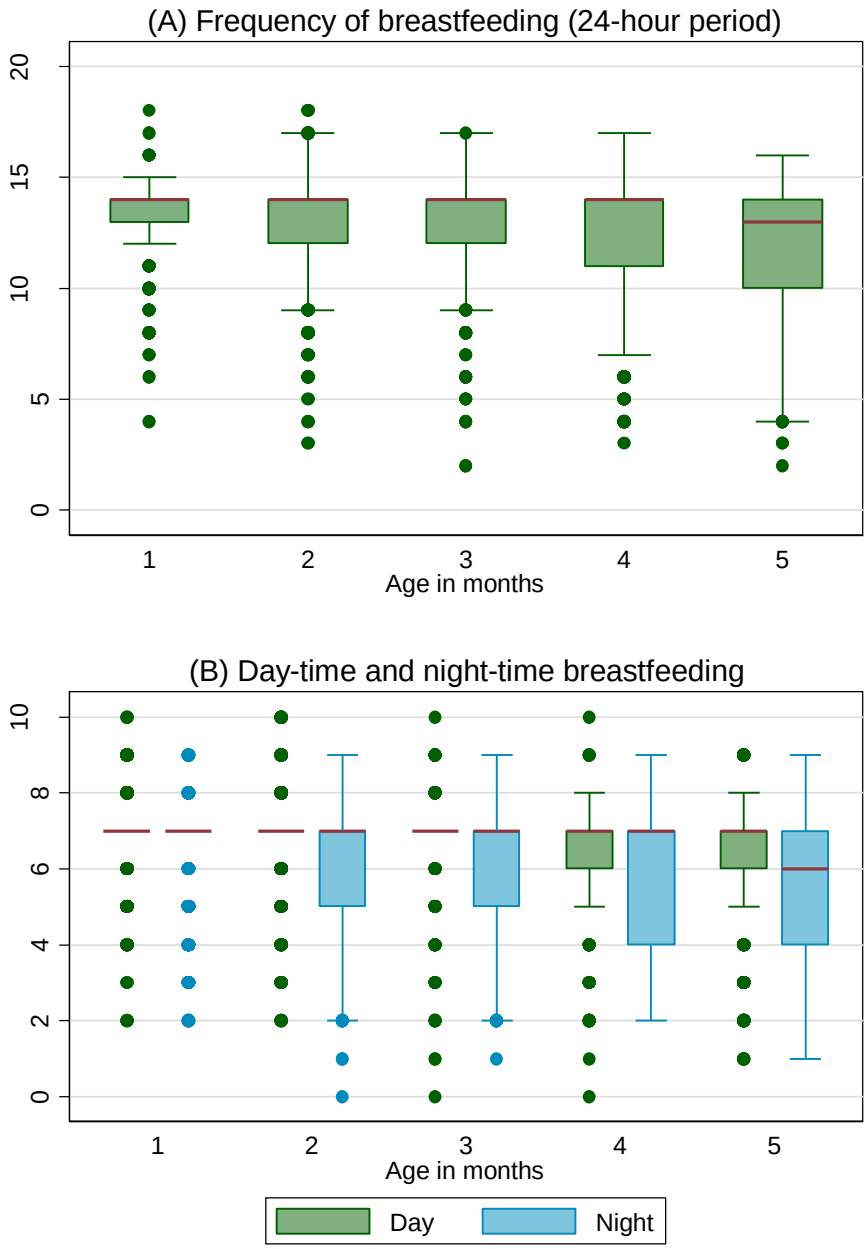
Thirty percent of infants (296 of 971) were given pre-lacteal feeds. The most common pre-lacteals were milk other than breastmilk (63%), honey (31%), and infant formula (13%). Sugar-sweetened water (4%), plain water (3%), and sugar-

salt-water solution (1%) were less common. Honey was the most common item administered in combination with another pre-lacteal. For example, 39% of infants who were given a non-breastmilk milk as a pre-lacteal feed also received honey.

7.5.2 Early breastfeeding period (0-5 months)

The median number of times infants were breastfed in the 24 hours before the survey was constant at follow-up visits up to four months (14 times) and decreased to 13 in the fifth month (Figure 7.2 - A). The variability in the number of feeds at older ages was greater for night-time feeding (IQR 4-7) than day-time feeding (IQR 6-7) (Figure 7.2 - B).

Figure 7.2 Box-and-whiskers plots of breastfeeding frequency (1-5 months)

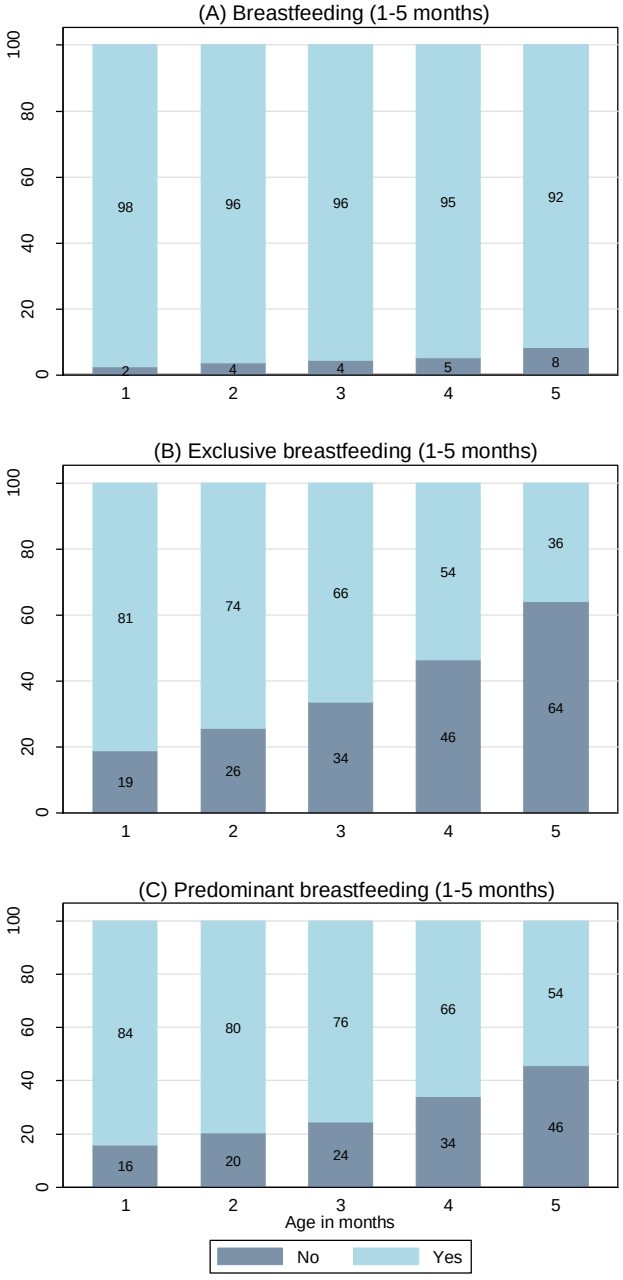


Note: Number of infants at each age = 741, 724, 646, 634, and 581.

Breastfeeding was widely practised up to six months (Figure 7.3 - A). At one month, 98% of infants were being breastfed, and this declined to 92% at five months. While breastfeeding was common, the proportions who were exclusively or predominantly breastfed were much lower.

Predominant breastfeeding was slightly higher than exclusive breastfeeding (as expected) at the first follow-up visit (84% vs 81%), but the difference was larger in the fifth month (54% vs 36%) (Figure 7.3 - B and C). In these graphs, it is possible that those exclusively breastfed in a month had been non-exclusively breastfed in a previous month, since I treated each data point cross-sectionally, ignoring an individual's previous responses.

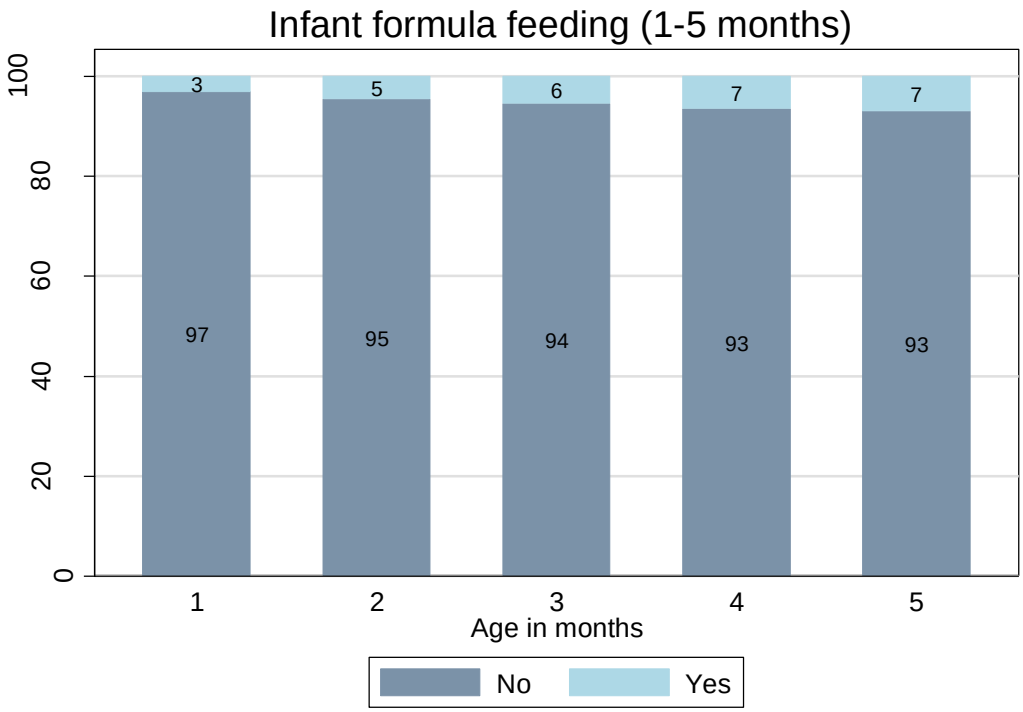
Figure 7.3 Breastfeeding practices at each follow-up visit



Note: Number of infants at each age = 719, 688, 626, 604 and 569.

Based on the criteria for exclusive breastfeeding, infants are allowed breastmilk and medication (drops, ORS), but no other liquids or solids. Predominant breastfeeding differs from exclusive breastfeeding in that it allows for breastfed infants to receive medication as well as some other liquids (water, fruit juice). The proportion of infants who met the exclusive and predominant breastfeeding indicators decreased at successive follow-up visits. Formula feeding was uncommon and did not appear to increase in proportion with decreases in prevalence of EBF or PBF (Figure 7.4); the proportion of infants who received infant formula was 3% at one month and 7% at five months.

Figure 7.4 Proportion of infants fed infant formula

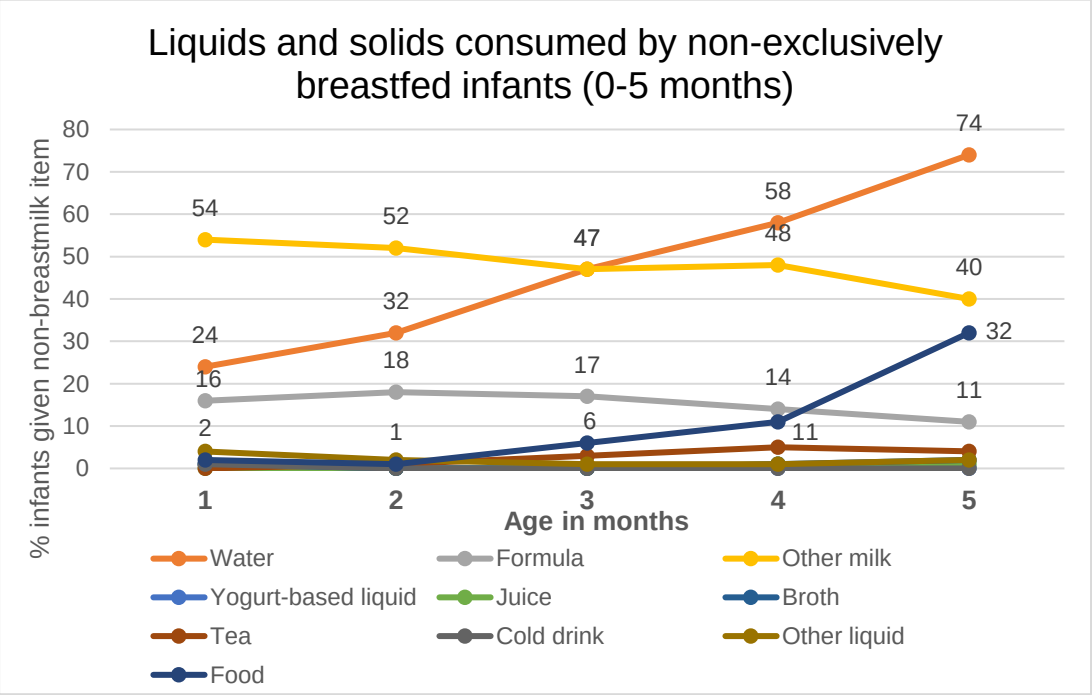


Note: Number of infants at each age = 719, 688, 626, 604 and 569.

Exclusive and predominant breastfeeding statuses were therefore more commonly compromised by items other than infant formula, though disaggregation by EBF and PBF showed more frequent use of formula than for the cohort as a whole.

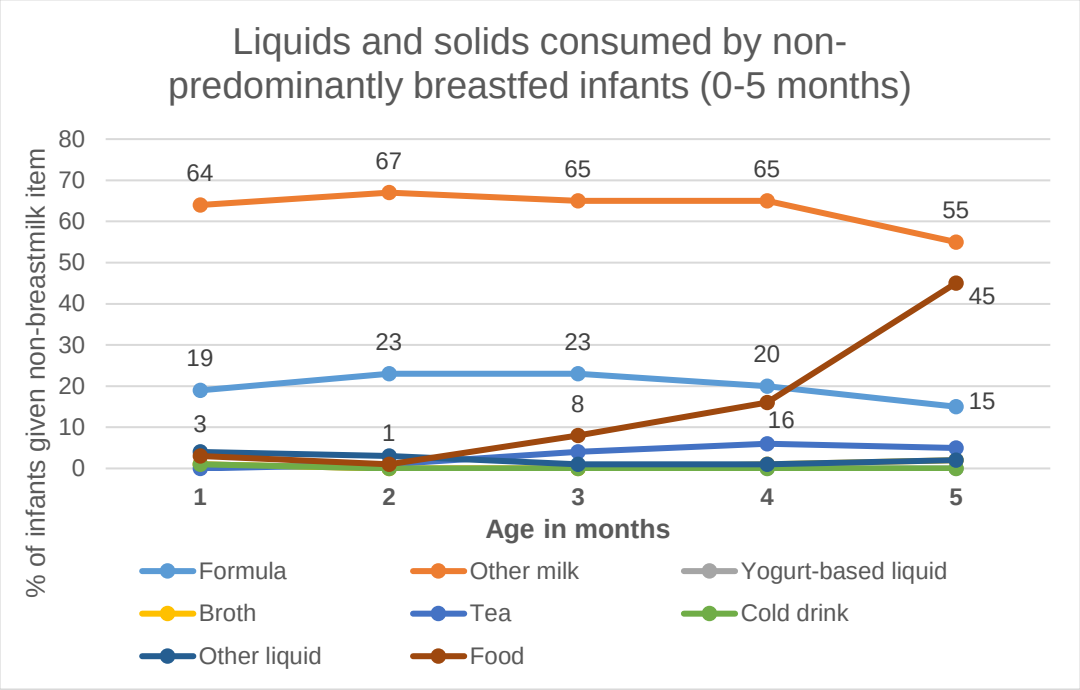
Non-human milk and water, in addition to infant formula, and, at older ages, semi-solid foods were the most common items that compromised EBF status (Figure 7.5). For example, 54% of infants who did not meet the criteria for EBF were given non-human milk at one month, and 24% received water. Non-exclusively breastfed infants more commonly received multiple non-breastmilk items at older ages (33% at four months and 48% at five months) than early in life (10%, 11% and 20% at one, two, and three months, respectively). PBF status (Table 7.6) was also compromised by non-human milk and formula in the first three months, though soft and semi-solid foods became more common in the fourth and fifth months (16% and 45%, respectively).

Figure 7.5 Items consumed by infants who did not meet EBF criteria



Note: Number of non-exclusively breastfed infants in each month = 135, 177, 211, 280, 364.

Figure 7.6 Items consumed by infants who did not meet PBF criteria

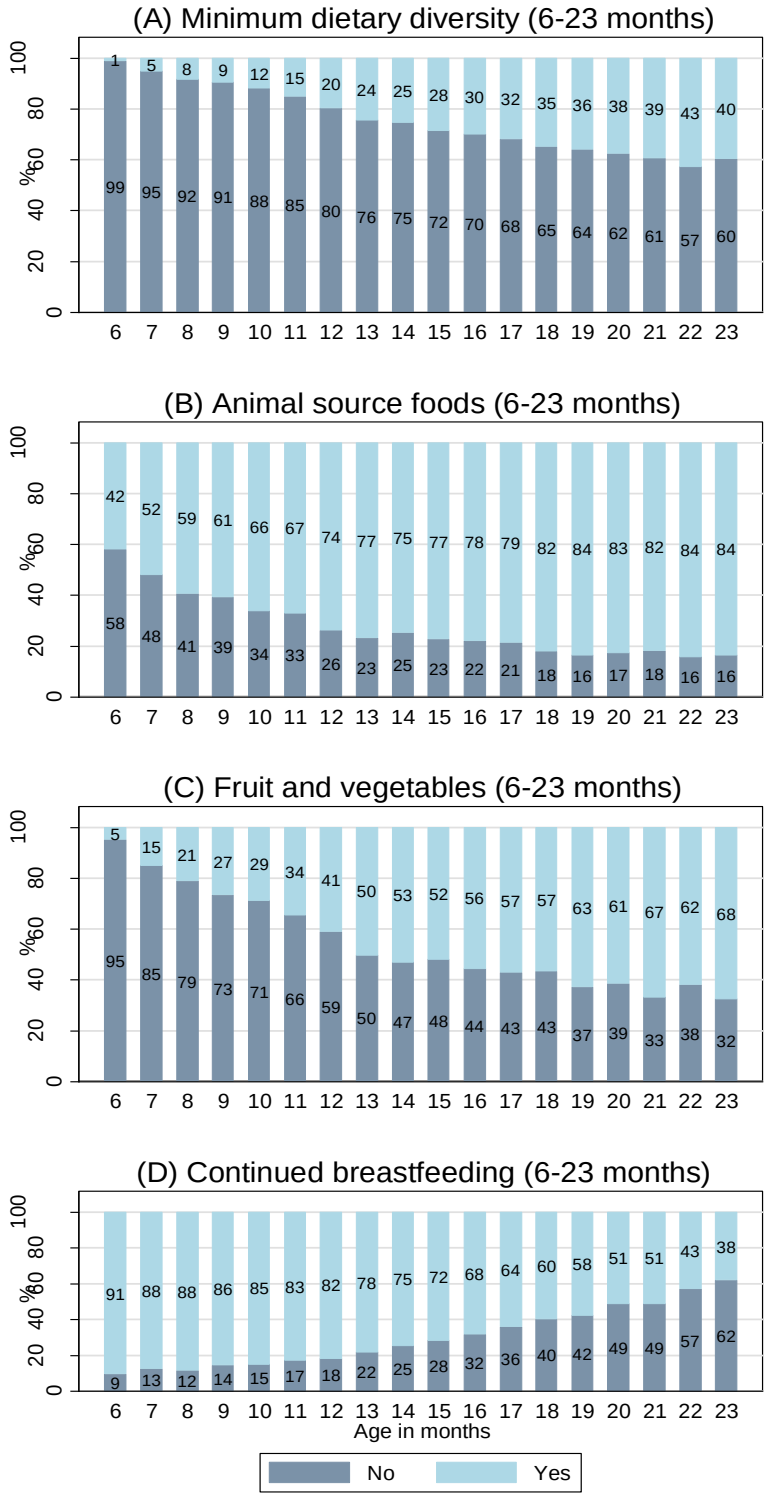


Note: Number of non-predominantly breastfed infants in each month = 114, 138, 153, 205, 260

7.5.3 Complementary feeding period (6-23 months)

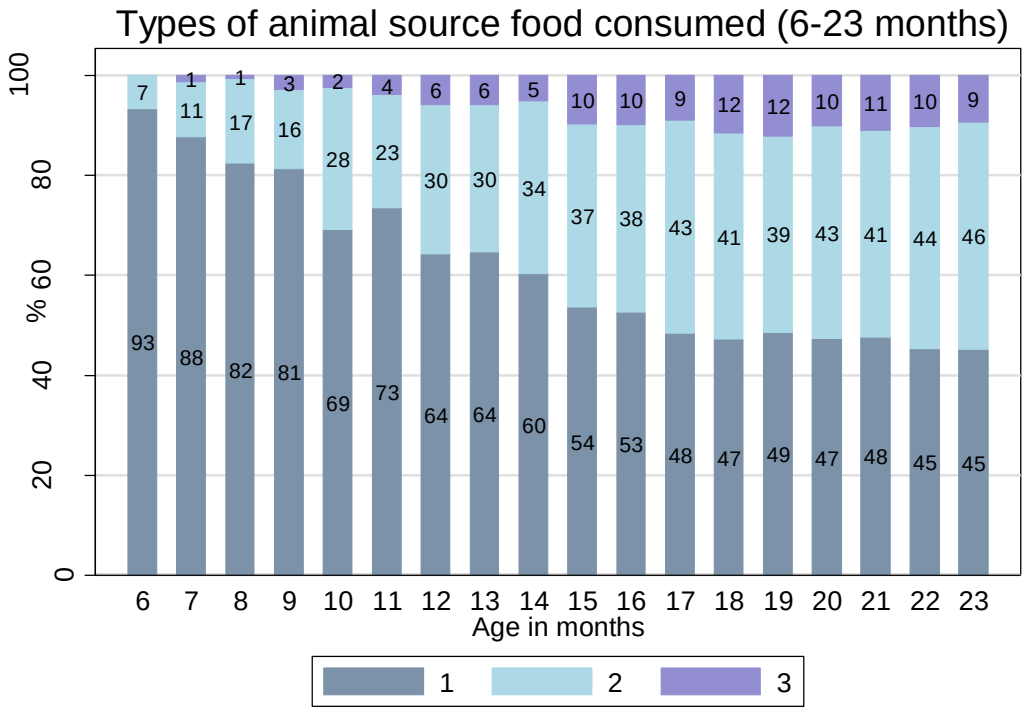
The progression to complementary feeding in the sixth month was marked by low proportions of infants consuming diverse diets (1%) (Figure 7.7 A), and consumption of any fruit and vegetables was also rare (5%) (Figure 7.7 C). These were 40% and 68%, respectively, at 23 months. The proportion of infants who received breastmilk was high in the early stages of the complementary feeding period (91% at six months), but was 38% at 23 months (Figure 7.7 D). Consumption of animal source foods was more frequent across the full period – 42% at 6 months and 84% at 23 months (Figure 7.7, B) – though at younger ages this was largely due to consumption of only one of three types of animal source food (dairy, flesh foods, or eggs) (Figure 7.8). At six months, 7% had consumed two types, but at 23 months 55% of those who achieved the ASF indicator had consumed two or three types.

Figure 7.7 Key complementary feeding practices and continued breastfeeding at each age (6-23 months)



Note: Number of children at each age (denominator) = 556, 560, 512, 520, 500, 476, 473,476, 475, 473, 495, 468, 463, 463, 475, 475, 462, and 444.

Figure 7.8 Number of types of animal source food consumed by infants who met ASF criteria

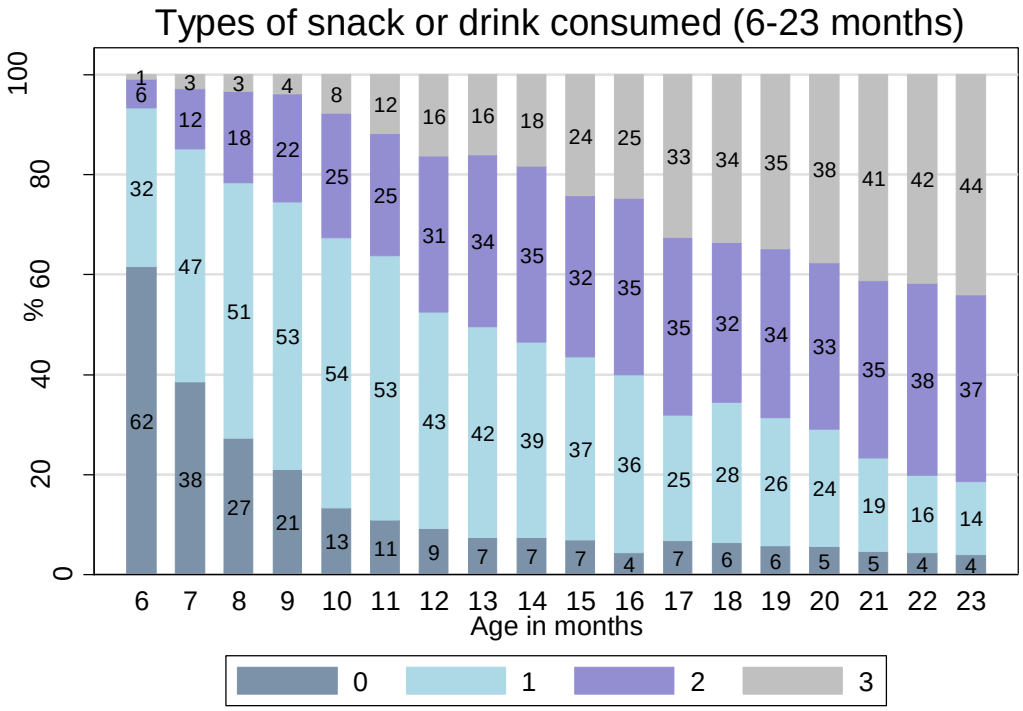


Note: Number of children who achieved ASF at each age (denominator) = 233, 291, 304, 316, 330, 319, 349, 366, 386, 368, 380, 387, 392, 389, 389, 371

Few infants (5%) consumed any snack food items or beverages in the first four months of life (less than five percent in each month, data not shown), but at five months 14% (82 of 569 infants) had consumed at least one type of snack or drink.

Consumption of snack foods and sweet beverages was common across the full complementary feeding period. At six months 38% of infants had consumed at least one type of sweet or salty snack food or sweet beverage; at 23 months this was 96% (Figure 7.9).

Figure 7.9 Number of types of sugary or salty snack or sweet beverage consumed

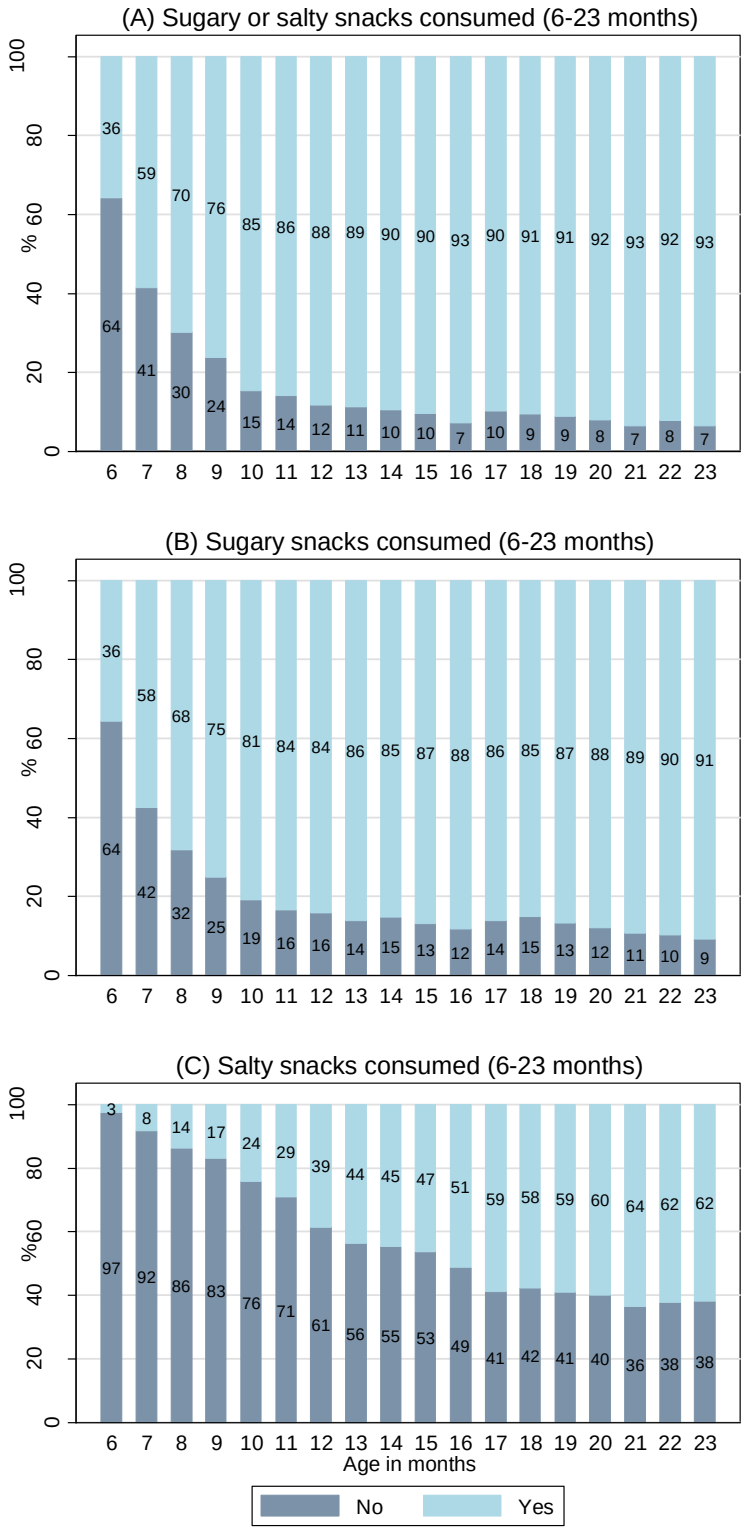


Note: Number of children at each age (denominator) = 556, 560, 512, 520, 500, 476, 473,476, 475, 473, 495, 468, 463, 463, 475, 475, 462, and 444.

Consumption of any snack food items (sweet or salty, soft, semi-solid or solid) increased from 36% at six months to 88% by 12 months, and then to 93% at 23 months (Figure 7.10 A). Sweet snacks were more frequently consumed than salty items at all ages (Figure 7.10 B and C).

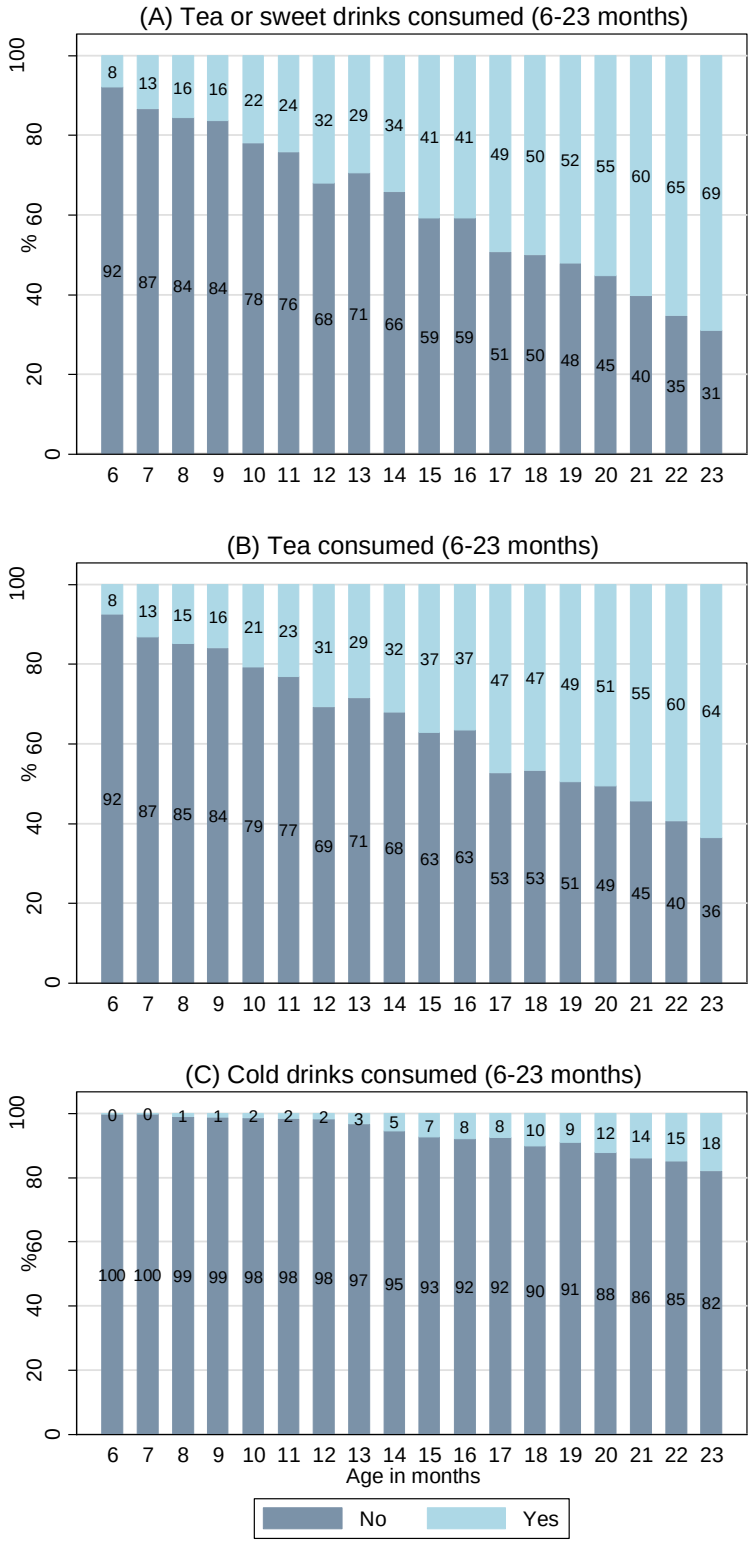
A slightly different pattern was observed for consumption of sweet beverages, which increased from 8% at six months to 32% at 12 months, and then increased by more than two-fold in the second year of life to 69% at 23 months (Figure 7.11 A). Tea was more frequently consumed (Figure 7.11 B) than cold drinks (Figure 7.11 C) at all ages.

Figure 7.10 Consumption of sweet or salty snacks (6-23 months)



Note: Number of children at each age (denominator) = 556, 560, 512, 520, 500, 476, 473,476, 475, 473, 495, 468, 463, 463, 475, 475, 462, and 444.

Figure 7.11 Consumption of sweet beverages (6-23 months).



Note: Number of children at each age (denominator) = 556, 560, 512, 520, 500, 476, 473,476, 475, 473, 495, 468, 463, 463, 475, 475, 462, and 444.

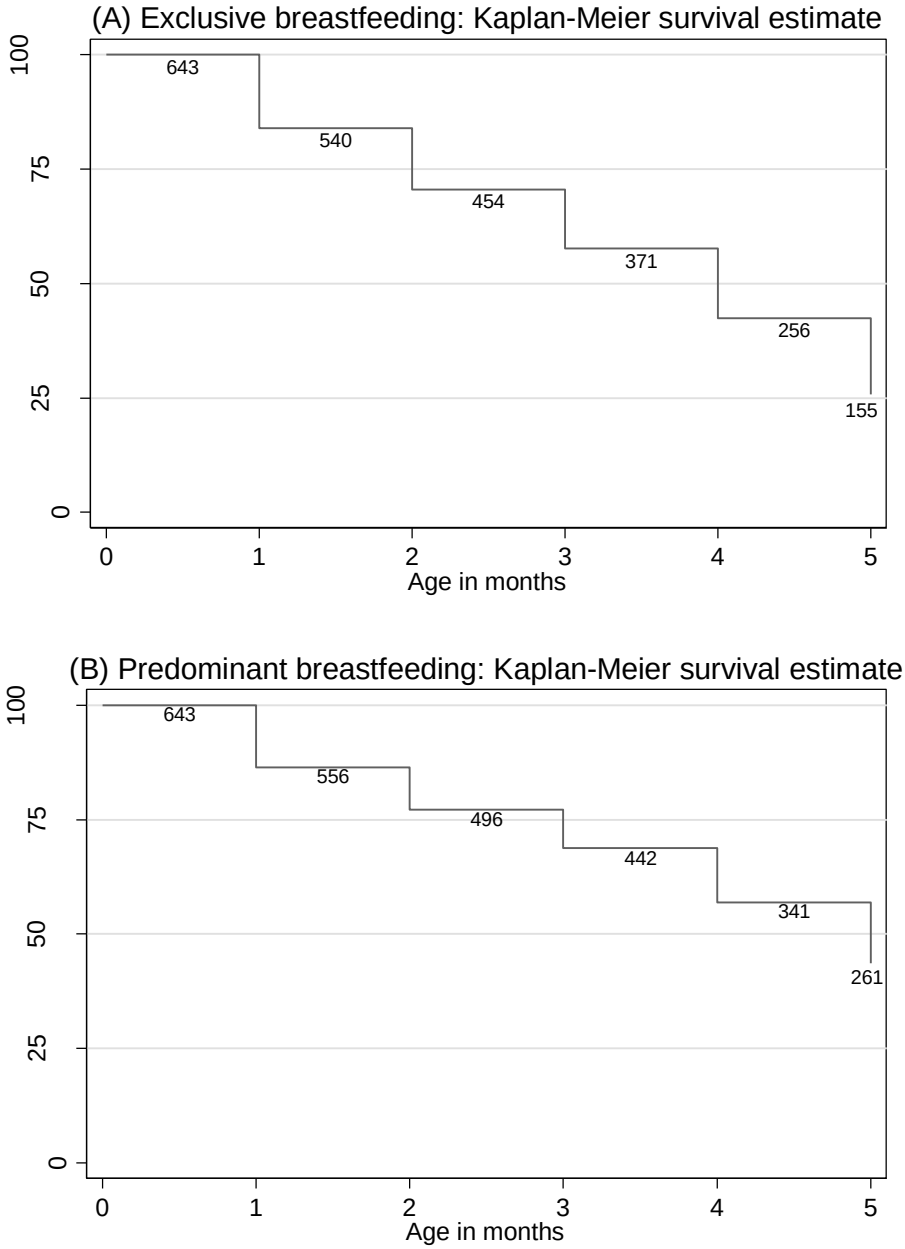
7.6 IYCF practices: findings from longitudinal data

7.6.1 Duration of exclusive and predominant breastfeeding based on life-long data

Complete case analysis of exclusive and predominant breastfeeding practices was possible for 270 (28%) infants who had IYCF data for every measurement occasion from 0 to 5 months. Using lifelong data definitions of exclusive breastfeeding and predominant breastfeeding, the proportion of infants who remained exclusively breastfed at every successive measurement occasion up to 5 months was 21% (58 infants), and 40% for predominant breastfeeding (109 infants).

When breastfeeding data were collapsed into two-month age bands (0-1 month, 2-3 months, and 4-5 months), allowing for one missing value in each period such that the last observation was carried forward to the next measurement occasion (or the previous missing one ignored), 643 infants (66%) had between three and six observations on breastfeeding practices. Based on survival estimates, 26% (172 infants) were still exclusively breastfed (Figure 7.12 A) and 44% (286 infants) were still predominantly breastfed (Figure 7.12 B) in the 4-5 month interval. The number of infants in each month who stopped receiving EBF or PBF and the estimated survivor function at each age are presented in Table 7.3. Infants were exclusively breastfed for a median duration of four months and predominantly breastfed for five months.

Figure 7.12 Exclusive and predominant breastfeeding. Kaplan-Meier survival curves for 643 infants



Note: Numbers below curves indicate number at risk of stopping at the start of the period

Table 7.3 Number at risk of stopping EBF and PBF and estimated survival probabilities in each month

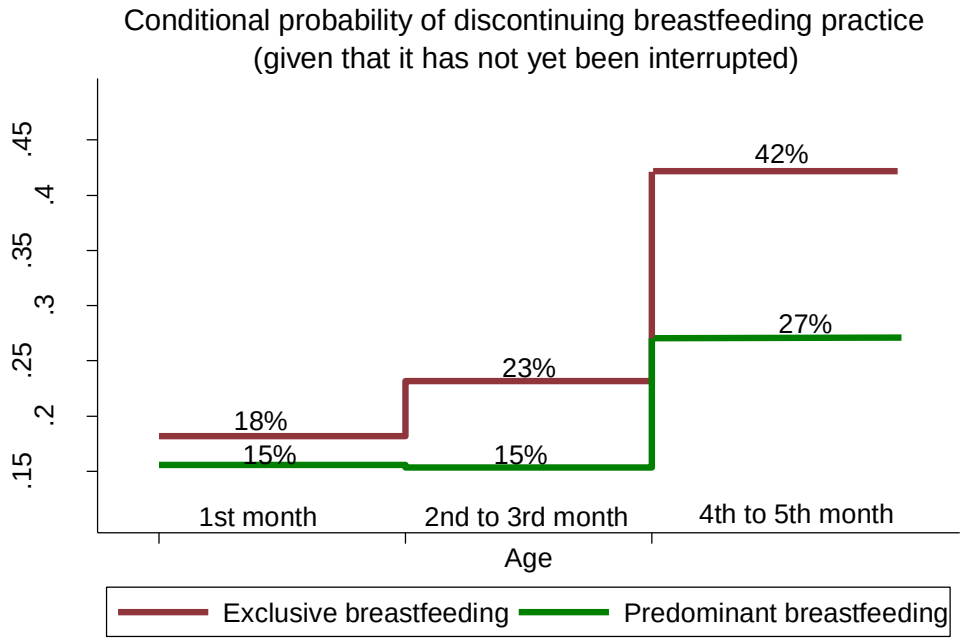
Age (months)	Number still breastfeeding at start of the month	Stopped during the month	Censored*	Survival probability	Lower 95%CI	Upper 95%CI
Exclusive breastfeeding						
1	643	103	0	0.84	0.81	0.87
2	540	86	0	0.71	0.66	0.74
3	454	83	0	0.58	0.54	0.61
4	371	98	17	0.42	0.39	0.46
5	256	101	155	0.26	0.22	0.29
Predominant breastfeeding						
1	643	87	0	0.86	0.84	0.89
2	556	60	0	0.77	0.74	0.80
3	496	54	0	0.69	0.65	0.72
4	442	76	25	0.57	0.53	0.61
5	341	80	261	0.44	0.40	0.47

*Censored: at 4 months, indicates the number of infants who had not yet experienced discontinuation but did not have a measurement in the 5th month; at 5 months, indicates the number of infants who were still breastfeeding at the end of the month, i.e., those EBF or PBF up to five months.

7.6.2 Determinants of exclusive and predominant breastfeeding (0-5 months)

Complete data in all three age bands (1st month, 2-3 months, and 4-5 months) were available for 533 (54%) infants. The discrete-time hazard for discontinuation of EBF and PBF was similar in the 1st month (18% and 15%), and remained the same for PBF in the 2-3 month interval (15%), but was much lower for PBF than for EBF in the 4-5 month period (27% vs 42%) (Figure 7.13).

Figure 7.13 Discrete-time hazard of discontinuing breastfeeding practices



In crude analyses of univariable associations, higher maternal education, access to piped water, households with two or more adults, paternal smoking, and greater household asset score were associated with greater hazard of discontinuing exclusive breastfeeding. In fully adjusted (all covariates) and reduced (covariates with p<0.1 in crude analysis) models, the associations of higher maternal education (adjusted HR 1.54; 95%CI 1.1, 2.1) and paternal smoking (adjusted HR 1.38; 95%CI 1.0, 1.8) remained (Table 7.4), though the HR for maternal education was slightly attenuated in the reduced model (adjusted HR 1.42; 95%CI 1.1, 1.9). The predicted probabilities for categories of maternal education and paternal smoking from the reduced model, with all other covariates held at their baseline values, are displayed in Figure 7.14. Wald tests of the non-proportional hazards assumption showed that there was little evidence that the associations of maternal education (p= 0.8591) and paternal smoking (p= 0.5860) with EBF cessation varied over time.

Figure 7.14 Predicted hazard of discontinuation of EBF

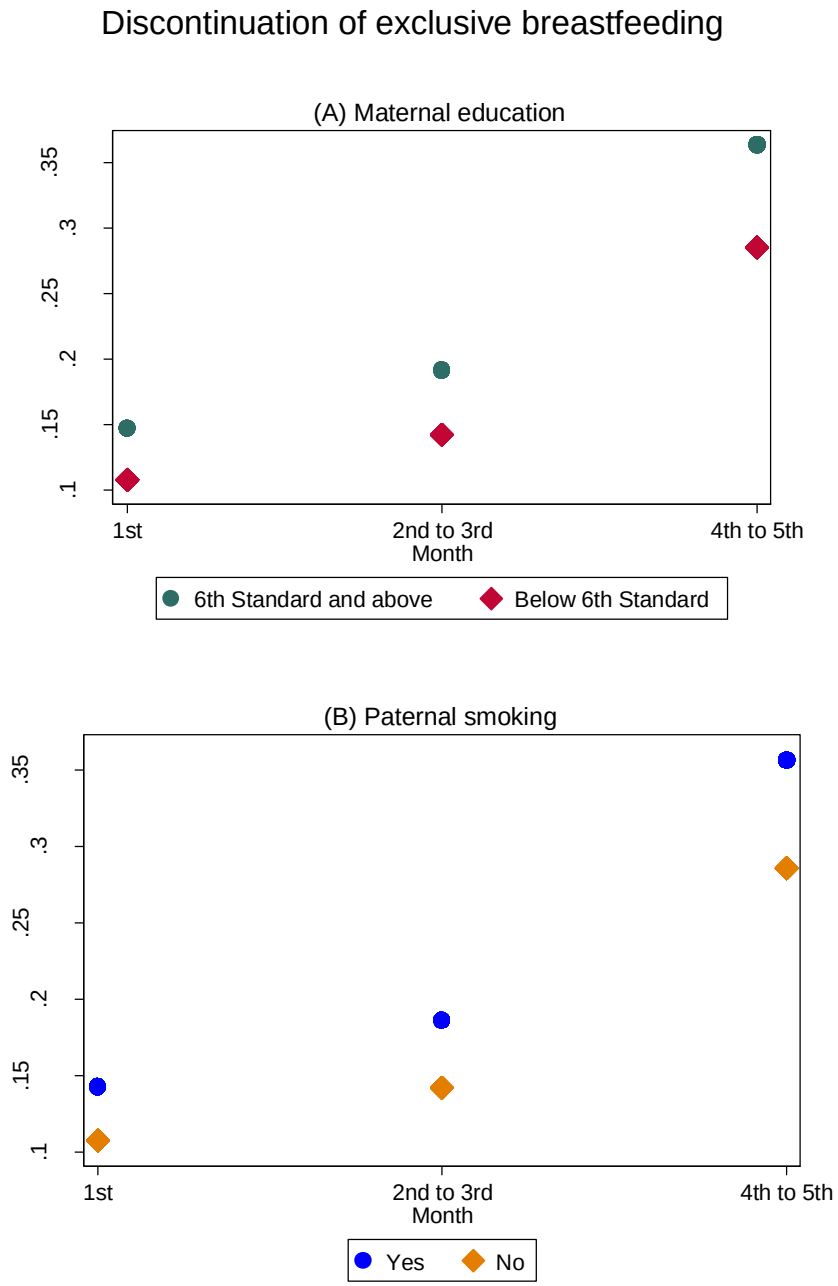


Table 7.4 Crude and adjusted hazard ratios for exclusive breastfeeding cessation in any interval (1, 2-3, or 4-5 months) (n=533)

	Crude associations		Full model		Reduced model	
Covariate	**HR (95%CI)**	**p-value**	**HR (95%CI)**	**p-value**	**HR (95%CI)**	**p-value**
Female	0.98 (0.8, 1.3)	0.850	1.02 (0.8, 1.3)	0.884		
Maternal age > 25	1.04 (0.8 1.4)	0.741	1.04 (0.7, 1.5)	0.807		
Paternal age > 30	1.11 (0.9, 1.4)	0.415	1.26 (0.9, 1.8)	0.183		
Maternal education (6th standard and above)	1.49 (1.2, 1.9)	0.002	1.54 (1.1, 2.1)	0.007	1.42 (1.1, 1.9)	0.014
Paternal education (6th standard and above)	1.03 (0.8, 1.3)	0.778	0.88 (0.7, 1.2)	0.406		
Asset score	1.12 (0.9, 1.3)	0.085	1.00 (0.9, 1.2)	0.988	1.02 (0.9, 1.2)	0.814
Asset quintile (ref Lowest)						
Second lowest	1.13 (0.8, 1.7)	0.514				
Middle	0.99 (0.7, 1.5)	0.985				
Second highest	1.41 (0.9, 2.1)	0.090				
Highest	1.34 (0.9, 2.0)	0.157				
Access to piped water	1.33 (1.0, 1.7)	0.034	1.27 (0.9, 1.7)	0.107	1.28 (1.0, 1.7)	0.082
Use of shared toilet	0.90 (0.6, 1.3)	0.562	1.03 (0.7, 1.5)	0.886		
2+ adults in the household	1.33 (1.0, 1.7)	0.030	1.30 (1.0, 1.8)	0.091	1.21 (0.9, 1.6)	0.191
4+ children in the household	0.93 (0.7, 1.3)	0.618	0.91 (0.7, 1.2)	0.543		
Paternal smoking	1.26 (0.9, 1.6)	0.076	1.38 (1.0, 1.8)	0.025	1.39 (1.1, 1.8)	0.017
Maternal smoking	0.89 (0.6, 1.3)	0.510	0.88 (0.6, 1.3)	0.481		
Planned pregnancy	0.83 (0.6, 1.1)	0.153	0.93 (0.7, 1.2)	0.584		

In crude analyses of univariable associations between predominant breastfeeding and covariates, higher maternal education, access to piped water, households with two or more adults, and higher household asset score were associated with greater hazard of discontinuing PBF at any age. Infants born as a result of planned pregnancies had a lower hazard of PBF cessation (unadjusted HR 0.72; 95%CI 0.6, 0.9). Higher maternal education showed a strong positive association with PBF cessation in both multivariable models (aOR 1.97; 95%CI 1.4, 2.8, in the full model and aOR 1.78; 95%CI 1.3, 2.4, in the reduced model). The results of fully adjusted and reduced models were slightly different for pregnancy intention, its protective influence retained only in the reduced model. Further, the full model showed a slightly attenuated relationship between households with two or more adults and hazard of discontinuation of PBF (Table 7.5), an association not preserved in the reduced model. The predicted probabilities for categories of maternal education and pregnancy intention from the reduced model, with all other covariates held at their baseline values, are displayed in Figure 7.15. Wald tests of the non-proportional hazards assumption showed that there was little evidence that the association of higher maternal education (p= 0.5038) and planned pregnancy (p= 0.8874) with PBF cessation changed over time.

Figure 7.15 Predicted hazard of discontinuation of PBF

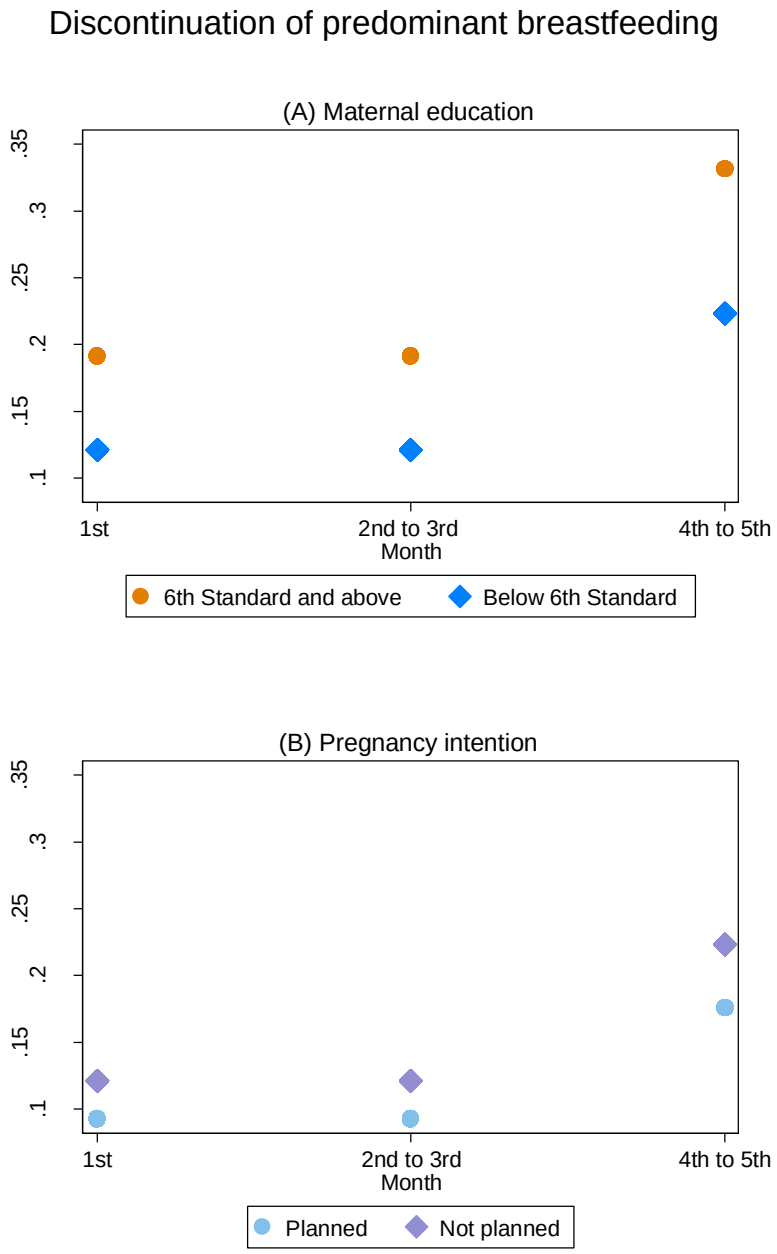


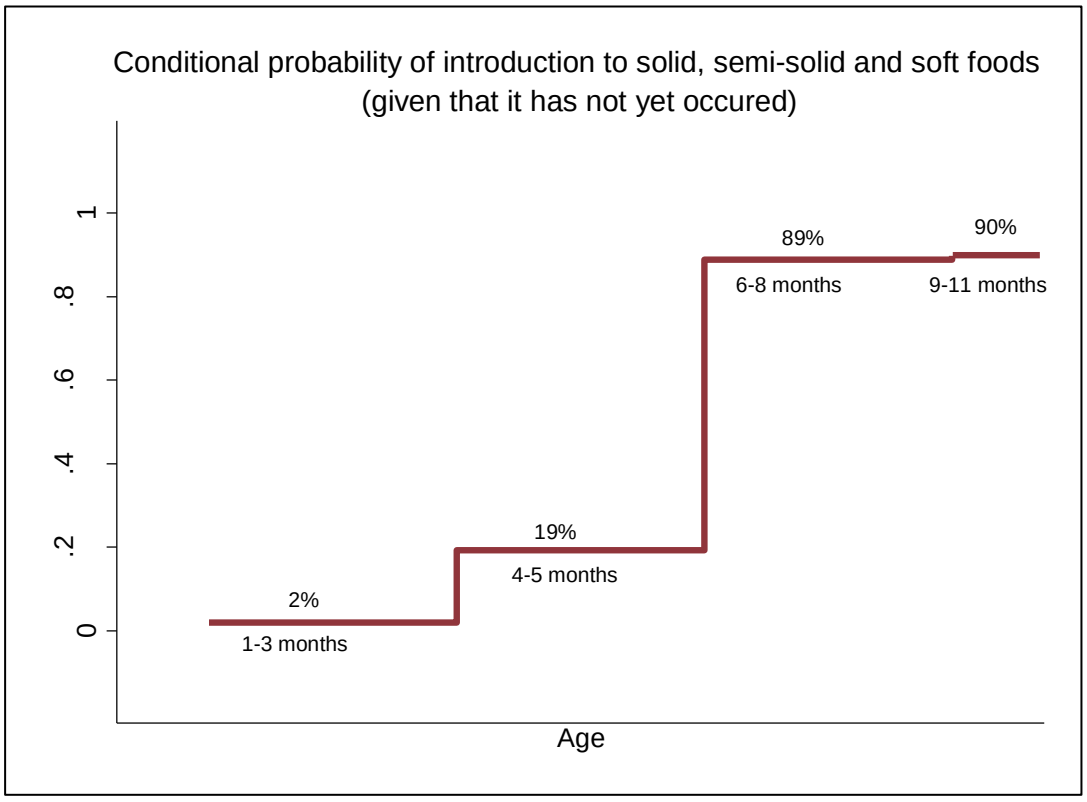
Table 7.5 Crude and adjusted hazard ratios for PBF cessation in any interval (1 month, 2-3 months, or 4-5 months)

	Crude association		Full model		Reduced model	
Covariate	**HR (95%CI)**	**p-value**	**HR (95%CI)**	**p-value**	**HR (95%CI)**	**p-value**
Female	0.96 (0.7, 1.3)	0.767	1.00 (0.7, 1.3)	0.988		
Maternal age > 25	1.16 (0.9, 1.5)	0.304	1.28 (0.9, 1.9)	0.201		
Paternal age > 30	1.13 (0.9, 1.5)	0.369	1.14 (0.8, 1.6)	0.485		
Maternal education (6th standard and above)	1.82 (1.4, 2.4)	<0.0001	1.97 (1.4, 2.8)	<0.0001	1.78 (1.3, 2.4)	<0.0001
Paternal education (6th standard and above)	1.20 (0.9, 1.6)	0.203	0.91 (0.7, 1.3)	0.580		
Asset score	1.12 (0.9, 1.3)	0.102	0.92 (0.8, 1.1)	0.315	0.94 (0.8, 1.1)	0.490
Asset quintile (ref Lowest)						
Second lowest	1.13 (0.8, 1.72)	0.537				
Middle	0.91 (0.6, 1.4)	0.658				
Second highest	1.45 (0.9, 2.2)	0.086				
Highest	1.35 (0.9, 2.1)	0.183				
Access to piped water	1.28 (0.9, 1.7)	0.100	1.15 (0.8, 1.6)	0.395	1.14 (0.8, 1.5)	0.407
Use of shared toilet	0.89 (0.6, 1.3)	0.546	1.02 (0.7, 1.6)	0.912		
2+ adults in the household	1.45 (1.1, 1.9)	0.009	1.41 (1.0, 2.0)	0.037	1.32 (1.0, 1.8)	0.084
4+ children in the household	1.00 (0.8, 1.3)	0.991	0.96 (0.7, 1.3)	0.829		
Paternal smoking	0.95 (0.8, 1.2)	0.691	0.97 (0.7, 1.3)	0.856		
Maternal smoking	0.85 (0.6, 1.3)	0.414	0.89 (0.6, 1.3)	0.570		
Planned pregnancy	0.73 (0.6, 0.9)	0.023	0.78 (0.6, 1.0)	0.100	0.74 (0.6, 1.0)	0.040

7.6.3 Determinants of introduction of solid, semi-solid and soft foods

Based on four age bands (1-3 months, 4-5 months, 6-8 months, and 9-11 months), complete data were available for 565 (58%) children. The probability of being introduced to solid, semi-solid and soft foods was 2% before four months, 19% between 4-5 months, rose to 89% at 6-8 months, and was 90% at 9-11 for the small number who had not yet been give non-liquid food (Figure 7.16).

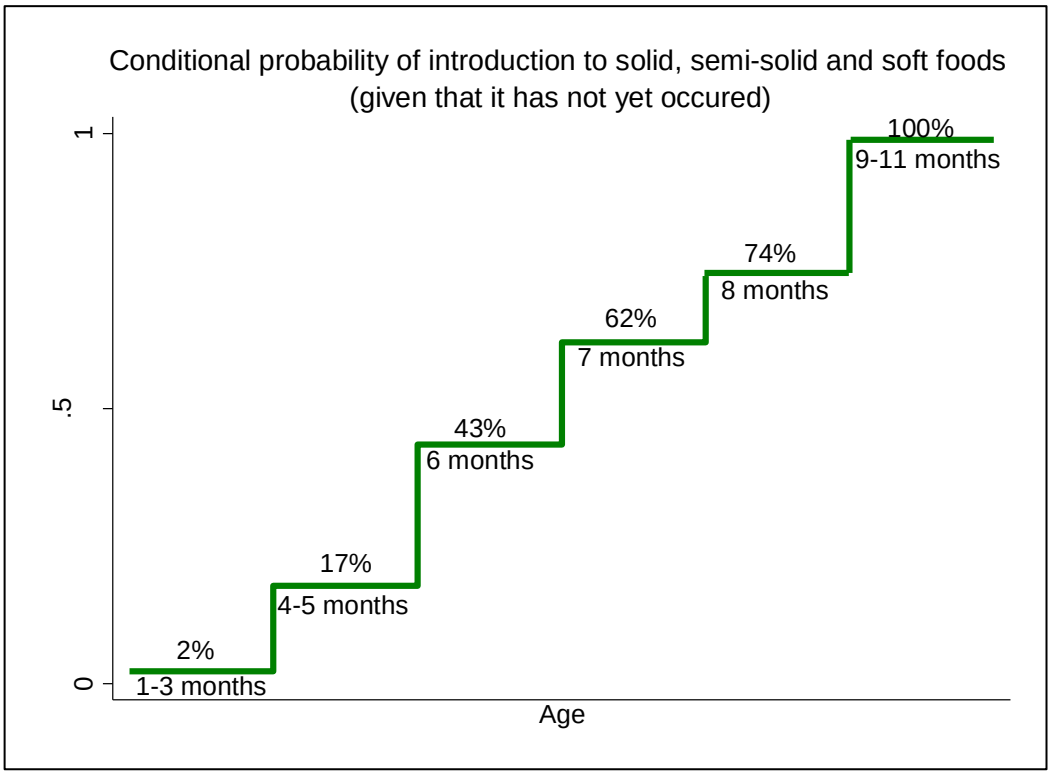
Figure 7.16 Discrete time hazard of introduction to solid, semi-solid and soft foods (n=565)



Note: X axis not to scale.

Data were available for 312 (32%) infants when I used six age bands (1-3, 4-5, 6, 7, 8, and 9-11 months), and this resulted in a slightly different picture. All children in this sample were eventually given solid food within the first year (Figure 7.17). In the period 6-8 months, the ideal window for introduction of complementary foods, the conditional probability was 43% at six months and 74% at eight months.

Figure 7.17 Discrete time hazard of introduction to solid, semi-solid and soft foods, using six time intervals (n=312)



Note: X axis not to scale

In crude models of the relationship between covariates and introduction to complementary food, maternal age over 25, paternal smoking, and households with four or more children were associated with lower hazard of introduction of complementary feeding in any interval (1-3 months, 4-5 months, or 6-8 months). Children of mothers who had studied beyond the sixth standard were more likely to be introduced to solids at any time, as were those from households with higher asset score.

Fully-adjusted and reduced models showed slightly different relationships (Table 7.6). In the fully adjusted model, older fathers (adjusted HR 1.84; 95%CI 1.2, 2.8) and more educated mothers (adjusted HR 1.55; 95%CI 1.0, 2.3) were more likely to introduce solids, while there was a negative relationship with households with four or more children (adjusted HR 0.60; 95%CI 0.4, 0.9). The positive association of maternal education (adjusted HR 1.40; 95%CI 1.0, 2.1) and negative association of number of children (adjusted HR 0.68; 95%CI 0.5, 1.0) were attenuated in the reduced model.

Wald tests of the non-proportional hazards assumption showed that there was little evidence that the associations of number of children in the household (p= 0.6638) and maternal education (p= 0.4003) with introduction to solids changed over time.

Table 7.6 Crude and adjusted hazard ratios for introduction to solid, semi-solid and soft foods in any interval (1-3, 4-5, or 6-8 months).

	Crude association		Full model		Reduced model	
Covariate	**HR (95%CI)**	**p-value**	**HR (95%CI)**	**p-value**	**HR (95%CI)**	**p-value**
Female	0.84 (0.6, 1.2)	0.309	0.89 (0.6, 1.3)	0.493		
Maternal age > 25	0.68 (0.5, 1.0)	0.026	0.68 (0.4, 1.1)	0.085	0.90 (0.6, 1.3)	0.592
Paternal age > 30	1.10 (0.8, 1.5)	0.592	1.84 (1.2, 2.8)	0.005		
Maternal education (6th standard and above)	1.67 (1.2, 2.3)	0.003	1.55 (1.0, 2.3)	0.035	1.40 (1.0, 2.1)	0.085
Paternal education (6th standard and above)	1.18 (0.8, 1.7)	0.331	0.85 (0.6, 1.2)	0.399		
Household asset score	1.17 (1.0, 1.4)	0.074	1.07 (0.9, 1.3)	0.559	1.07 (0.9, 1.3)	0.497
Household asset quintile (ref Lowest)						
Second lowest	1.17 (0.7, 2.0)	0.549				
Middle	0.96 (0.6, 1.6)	0.889				
Second highest	1.74 (1.0, 2.9)	0.038				
Highest	1.39 (0.8, 2.4)	0.225				
Access to piped water	1.03 (0.7, 1.5)	0.874	0.89 (0.6, 1.3)	0.555		
Use of shared toilet	0.87 (0.6, 1.4)	0.542	1.00 (0.6, 1.6)	0.986		
2+ adults in the household	1.19 (0.9, 1.7)	0.316	1.13 (0.8, 1.7)	0.527		
4+ children in the household	0.58 (0.4, 0.8)	0.003	0.60 (0.4, 0.9)	0.013	0.68 (0.5, 1.0)	0.046
Paternal smoking	0.64 (0.5, 0.9)	0.009	0.74 (0.5, 1.1)	0.104	0.76 (0.5, 1.1)	0.133
Maternal smoking	0.76 (0.5, 1.2)	0.255	0.94 (0.6, 1.6)	0.819		
Planned pregnancy	1.04 (0.7, 1.5)	0.815	0.93 (0.6, 1.3)	0.692		

7.6.4 Complementary feeding

The number of infants with sufficient data on complementary feeding in each three-monthly interval ranged from 724 (74%) at 6-8 months to 578 (59%) at 21-23. The frequency of dietary diversity, animal source food intake, and consumption of two or more types of snack food in each interval are described in Table 7.7.

Table 7.7 Frequency of complementary feeding practices in analytic sample

	Minimum dietary diversity, N (%)		Animal source foods, N (%)		Snacks (two or more types), N (%)		
Age interval	**Never**	**Ever**	**Never or Sometimes**	**Always**	**Never**	**Ever**	**Total**
6-8 months	627 (87)	97 (13)	475 (66)	249 (34)	524 (72)	200 (28)	724
9-11 months	455 (68)	213 (32)	331 (50)	337 (50)	311 (47)	357 (53)	668
12-14 months	298 (47)	341 (53)	234 (37)	405 (63)	176 (28)	463 (72)	639
15-17 months	215 (35)	396 (65)	226 (37)	385 (63)	107 (18)	504 (82)	611
18-20 months	154 (26)	435 (74)	179 (30)	410 (70)	79 (13)	510 (87)	589
21-23 months	128 (22)	450 (78)	164 (28)	414 (72)	43 (7)	535 (93)	578

The number of children with sufficient data to carry out multivariable autoregression analysis ranged between 746 (76%) and 767 (78%).

Preliminary unadjusted autoregressive models for each indicator, fitted without accounting for the initial condition (Model 1) or adjusting for covariates, showed strong evidence of state dependence as well as unobserved heterogeneity. The odds of consumption in an interval were strongly and positively associated with consumption in the previous period for minimum dietary diversity (OR 2.32; 95%CI 1.8, 3.0), animal source foods (OR 4.34; 95%CI 3.3, 5.8), and snacks (OR 2.38; 95%CI 1.8, 3.1). Likelihood ratio tests (LRT) of no difference between child and population-level estimators indicated strong evidence of unobserved heterogeneity ($p<0.0001$ for all three indicators). After adjusting for the lagged outcome and age interval, the proportion of variance in the propensity to receive an IYCF practice attributable to between-child characteristics was 22% for MDD, 24% for ASF, and 22% for snacks.

A comparison of unadjusted models for each indicator (Table 7.8) showed that ignoring the initial conditions (Model 1) over-estimated the state dependence (OR for consumption in previous interval) and underestimated the unobserved heterogeneity (OR for between-child random effect). However, models with an unconstrained occasion-level random effect coefficient (Model 3) were no different from those with the coefficient constrained to one (Model 2) for all three indicators, indicating that a simpler model specification (Model 2) could be used.

Table 7.8 Comparison of state dependence and unobserved heterogeneity between models

	Model 1	Model 2	Model 3	LRT for Model 2 vs 3
Minimum Dietary Diversity				p= 0.7821
Consumption in previous interval, OR (95%CI)	2.32 (1.8, 3.0)	1.78 (1.3, 2.3)	1.76 (1.3, 2.3)	
Between-child random effect, OR (95%CI)	2.66 (2.2, 3.5)	3.69 (2.5, 6.6)	3.81 (2.4, 7.4)	
Coefficient of occasion-level random effect, estimate (SE)	-	1*	0.93 (0.22)	
Animal Source Foods				p= 0.7880
Consumption in previous interval, OR (95%CI)	4.34 (3.3, 5.8)	2.43 (1.9, 3.2)	2.40 (1.8, 3.2)	
Between-child random effect, OR (95%CI)	2.76 (2.2, 3.8)	8.11 (4.6, 17.7)	8.61 (4.4, 22.9)	
Coefficient of individual random effect, estimate (SE)	-	1*	0.95 (0.16)	
Snacks				p= 0.1422
Consumption in previous interval, OR (95%CI)	2.38 (1.8, 3.1)	1.76 (1.3, 2.4)	1.69 (1.2, 2.3)	
Between-child random effect, OR (95%CI)	2.62 (2.1, 3.5)	3.01 (2.1, 5.3)	3.78 (2.3, 8.1)	
Coefficient of individual random effect, estimate (SE)	-	1*	0.68 (0.18)	
Notes Model 1: Model ignoring initial condition Model 2: Joint model of occasion-level random effect coefficient = 1 and Model 1 Model 3: Joint model of unconstrained occasion-level random effect coefficient and Model 1 Odds ratios are unadjusted *coefficient constrained to 1 Abbreviations: OR, odds ratio; 95%CI, 95% confidence interval; SE, standard error; LRT, likelihood ratio test				

Crude, fully adjusted, and reduced models for associations of background covariates with MDD, ASF, and snacks based on the strategy for Model 2 are

reported in Tables 7.9 to 7.11. Results of fully adjusted models were broadly similar to those of reduced models. Of the three dietary indicators, ASF consumption at any time had the strongest positive association with consumption in the previous period (crude OR 2.43; 95%CI 1.9, 3.2).

Paternal smoking had the strongest negative association with MDD; children of fathers who smoked were 38% less likely to consume diverse diets (aOR 0.62; 95%CI 0.5, 0.8), after adjusting for the effect of previous consumption and other covariates (Table 7.9). Planned pregnancies were also associated with lower odds of dietary diversity (aOR 0.77; 95%CI 0.6, 1.0).

Consumption of animal source foods was also associated negatively with paternal smoking and planned pregnancy, but had a strong positive relationship with access to piped water (Table 7.10). Children from homes with water piped into the dwelling or yard were more likely to consume ASF regularly (aOR 2.33; 95%CI 1.7, 3.2).

Consumption of snack foods was negatively associated with higher maternal education (aOR 0.65; 95%CI 0.5, 0.9), but positively with households that had four or more children (aOR 1.61; 95%CI 1.1, 2.2) in the fully adjusted model (Table 7.11).

Table 7.9 Relationships between background covariates and minimum dietary diversity (MDD) in Model 2

Minimum Dietary Diversity (MDD)	Crude association		Full model (n=746)		Reduced model (n=747)	
Covariate	**OR (95%CI)**	**p-value**	**OR (95%CI)**	**p-value**	**OR (95%CI)**	**p-value**
Lagged response (previous consumption)	1.78 (1.3, 2.3)	<0.0001	1.89 (1.4, 2.5)	<0.0001	1.87 (1.4, 2.5)	<0.0001
Time interval	1.73 (1.6, 1.9)	<0.0001	1.71 (1.6, 1.9)	<0.0001	1.71 (1.6, 1.9)	<0.0001
Female	1.07 (0.8, 1.4)	0.605	1.15 (0.9, 1.5)	0.265		
Maternal age > 25	1.12 (0.9, 1.4)	0.363	1.30 (1.0, 1.8)	0.098		
Paternal age > 30	1.01 (0.8, 1.3)	0.959	0.99 (0.7, 1.3)	0.938		
Maternal education (6th standard and above)	1.34 (1.0, 1.7)	0.020	1.21 (0.9, 1.6)	0.187	1.15 (0.9, 1.5)	0.294
Paternal education (6th standard and above)	1.23 (1.0, 1.6)	0.107	1.07 (0.8, 1.4)	0.637		
Household asset score	1.13 (1.0, 1.3)	0.048	1.05 (0.9, 1.2)	0.526	1.04 (0.9, 1.2)	0.567
Household asset quintile (ref Lowest)						
Second lowest	1.26 (0.9, 1.9)	0.248				
Middle	1.06 (0.7, 1.6)	0.768				
Second highest	1.26 (0.9, 1.9)	0.249				
Highest	1.45 (1.0, 2.1)	0.059				
Access to piped water	0.85 (0.7, 1.1)	0.210	0.74 (0.6, 1.0)	0.032		
Use of shared toilet	0.81 (0.6, 1.1)	0.200	0.95 (0.7, 1.4)	0.792		
2+ adults in the household	1.30 (1.0, 1.7)	0.039	1.25 (0.9, 1.7)	0.113	1.17 (0.9, 1.5)	0.259
4+ children in the household	1.02 (0.8, 1.3)	0.874	0.98 (0.7, 1.3)	0.909		
Paternal smoking	0.61 (0.5, 0.8)	<0.0001	0.62 (0.5, 0.8)	<0.0001	0.63 (0.5, 0.8)	<0.0001
Maternal smoking	0.95 (0.7, 1.4)	0.789	1.09 (0.8, 1.6)	0.648		
Planned pregnancy	0.80 (0.6, 1.0)	0.077	0.77 (0.6, 1.0)	0.041	0.76 (0.6, 1.0)	0.034
Between-child random effect, OR (95%CI)			3.06 (2.1, 5.3)		3.29 (2.2, 5.8)	

Table 7.10 Relationships between background covariates and consumption of animal source foods (ASF) in Model 2

Animal Source Foods (ASF)	Crude association		Full model (n=746)		Reduced model (n=746)	
Covariate	**OR (95%CI)**	**p-value**	**OR (95%CI)**	**p-value**	**OR (95%CI)**	**p-value**
Lagged response (previous consumption)	2.43 (1.9, 3.2)	<0.0001	2.42 (1.8, 3.2)	<0.0001	2.44 (1.9, 3.2)	<0.0001
Time interval	1.28 (1.2, 1.4)	<0.0001	1.29 (1.2, 1.4)	<0.0001	1.29 (1.2, 1.4)	<0.0001
Female	0.89 (0.7, 1.2)	0.431	0.99 (0.7, 1.3)	0.952		
Maternal age > 25	0.86 (0.6, 1.1)	0.304	1.01 (0.7, 1.4)	0.977		
Paternal age > 30	0.82 (0.6, 1.1)	0.182	0.88 (0.6, 1.3)	0.483		
Maternal education (6th standard and above)	1.70 (1.3, 2.3)	<0.0001	1.23 (0.9, 1.7)	0.211	1.28 (0.9, 1.7)	0.113
Paternal education (6th standard and above)	1.55 (1.2, 2.1)	0.004	1.08 (0.8, 1.5)	0.640	1.08 (0.8, 1.5)	0.611
Household asset score	1.32 (1.1, 1.5)	<0.0001	1.06 (0.9, 1.3)	0.505	1.05 (0.9, 1.2)	0.577
Household asset quintile (ref Lowest)						
Second lowest	1.03 (0.7, 1.6)	0.881				
Middle	0.99 (0.6, 1.6)	0.962				
Second highest	1.42 (0.9, 2.2)	0.128				
Highest	2.67 (1.7, 4.3)	<0.0001				
Access to piped water	2.84 (2.1, 3.8)	<0.0001	2.33 (1.7, 3.2)	<0.0001	2.33 (1.7, 3.2)	<0.0001
Use of shared toilet	0.42 (0.3, 0.6)	<0.0001	0.76 (0.5, 1.2)	0.196	0.76 (0.5, 1.2)	0.191
2+ adults in the household	1.53 (1.1, 2.1)	0.005	1.11 (0.8, 1.5)	0.523	1.13 (0.8, 1.5)	0.447
4+ children in the household	0.83 (0.6, 1.1)	0.223	0.95 (0.7, 1.3)	0.750		
Paternal smoking	0.55 (0.4, 0.7)	<0.0001	0.64 (0.5, 0.9)	0.003	0.64 (0.5, 0.9)	0.003
Maternal smoking	0.75 (0.5, 1.1)	0.190	1.02 (0.7, 1.5)	0.936		
Planned pregnancy	0.68 (0.5, 0.9)	0.010	0.64 (0.5, 0.9)	0.003	0.66 (0.5, 0.9)	0.004
Between-child random effect, OR (95%CI)			5.5 (3.4, 11.1)		5.39 (3.3, 10.8)	

Table 7.11 Relationships between background covariates and consumption of snacks in Model 2

Snacks	Crude association		Full model (n=746)		Reduced model (n=762)	
Covariate	**OR (95%CI)**	**p-value**	**OR (95%CI)**	**p-value**	**OR (95%CI)**	**p-value**
Lagged response (previous consumption)	1.76 (1.3, 2.4)	<0.0001	1.76 (1.3, 2.4)	<0.0001	1.75 (1.3, 2.4)	<0.0001
Time interval	1.86 (1.7, 2.1)	<0.0001	1.86 (1.7, 2.1)	<0.0001	1.86 (1.7, 2.1)	<0.0001
Female	1.14 (0.9, 1.5)	0.320	1.13 (0.9, 1.5)	0.365		
Maternal age > 25	1.06 (0.8, 1.4)	0.653	0.92 (0.7, 1.3)	0.609		
Paternal age > 30	1.09 (0.8, 1.4)	0.538	0.97 (0.7, 1.3)	0.847		
Maternal education (6th standard and above)	0.65 (0.5, 0.8)	0.001	0.65 (0.5, 0.9)	0.005	0.76 (0.6, 1.0)	0.053
Paternal education (6th standard and above)	0.71 (0.5, 0.9)	0.013	0.88 (0.7, 1.2)	0.383	0.87 (0.7, 1.1)	0.320
Household asset score	0.97 (0.8, 1.1)	0.613	1.11 (0.9, 1.3)	0.188		
Household asset quintile (ref Lowest)						
Second lowest	0.94 (0.6, 1.4)	0.761				
Middle	1.19 (0.8, 1.8)	0.408				
Second highest	0.91 (0.6, 1.4)	0.641				
Highest	0.83 (0.6, 1.2)	0.369				
Access to piped water	0.81 (0.6, 1.1)	0.123	0.89 (0.7, 1.2)	0.433		
Use of shared toilet	0.38 (0.1, 0.7)	0.024	1.43 (1.0, 2.1)	0.055	1.34 (1.0, 1.9)	0.085
2+ adults in the household	1.02 (0.8, 1.3)	0.902	1.00 (0.7, 1.3)	0.993		
4+ children in the household	1.65 (1.3, 2.2)	<0.0001	1.61 (1.2, 2.2)	0.002	1.51 (1.1, 2.0)	0.003
Paternal smoking	1.05 (0.8, 1.4)	0.695	0.93 (0.7, 1.2)	0.576		
Maternal smoking	0.87 (0.6, 1.3)	0.466	0.76 (0.5, 1.1)	0.164		
Planned pregnancy	0.83 (0.6, 1.1)	0.158	0.88 (0.7, 1.1)	0.344		
Between-child random effect, OR (95%CI)			2.66 (1.9, 4.7)		2.77 (1.9, 4.8)	

7.7 Discussion

7.7.1 Prevalence of IYCF practices

In this chapter I have described time-appropriate IYCF practices in the cohort and their relationship with background characteristics.

Depending on the type of analysis, the proportion of infants below six months who were exclusively breastfed was 21% (lifelong data, 270 (28%) complete cases), 26% (lifelong data, 643 (66%) partially complete cases), or 36% (cross-sectional analysis, 569 (58%) five-month old infants). Analogous proportions for predominant breastfeeding were 40%, 44%, and 54%. While the first result is based on the most stringent criteria, the last is based on the loosest, and estimates the proportion who were breastfed after possibly dipping in and out of exclusive / predominant breastfeeding at younger ages.

The proportion of infants introduced to solid, semi-solid and soft foods at 6-8 months was 89%. However, more granular analysis using a subset of complete cases showed that within this window less than half of infants who had not yet been given complementary food would begin in the sixth month.

Cross-sectional analysis of complementary feeding practices indicated poor dietary diversity (less than 20% in any month before 12 months), common consumption of animal source foods (nearly a third consumed two or more types at ten months), and early establishment of snacking behaviour (88% at 12 months). Autoregressive models for complementary feeding showed that children's diets were highly correlated between 6 and 23 months. Consumption of a diverse diet, animal source foods, or snacks in the previous period had a strong positive influence on a child's consumption in the current period.

The very low prevalence of exclusive breastfeeding in the cohort based on longitudinal methods confirms findings from a pooled analysis of prospective data from three birth cohorts in informal settlements in Southern India, which showed that EBF prevalence was as low as 11% by six months (Velusamy et al., 2017). In a peri-urban South African birth cohort it was 13% (Budree et al., 2017). By contrast, cross-sectional studies report much higher rates, with one study in Ethiopian informal settlements finding EBF as high as 84% (Demilew et al., 2017), and 57% in

a study in urban and rural West Bengal, India (Sinhababu et al., 2010). The average rate of exclusive breastfeeding of infants under six months was 41% globally in 2018 and 55% in India in 2016 (WHO, 2018a).

One explanation for my findings is that women's perceptions and definitions of optimal IYCF practice in early infancy could be very different from recommended practice. Maternal conceptualizations of IYCF can be highly contextual and framed by cultural beliefs about food and child health (Moffat, 2001, Monterrosa et al., 2012). Women who report exclusively breastfeeding in Mumbai's informal settlements may on closer inspection through in-depth interviews have very occasionally given infants honey, water, or other items that do not replace breastmilk, but expose infants to non-breastmilk items nonetheless, and in most research studies such instances would count as EBF cessation.

Such deviations from EBF may not alter women's description of their own practice as exclusive breastfeeding (Ramani et al., 2019). The monthly data in my study were derived from questions about infants' dietary intake rather than whether they were exclusively breastfeeding, and may have identified a large proportion of self-described exclusively-breastfeeding women and categorized their early lapses as 'failure events'. Women in the cohort who 'stop' EBF or PBF in survival analyses probably resume the practice at a later stage, but this is not captured in single-failure survival techniques. This could also explain why my cross-sectional analyses, in which previous status was ignored, showed higher prevalence of EBF and PBF in the fifth month.

An alternative analytical approach to these data, such as the Markov-chain model used by Chola et al. (2013), might give some insight into the probabilities of switching in and out of EBF or PBF at each age and paint a clearer picture of the dynamic nature of breastfeeding practices in Mumbai's informal settlements. However, a different method is unlikely to present a more favourable image, as even with the most lax definition using point-in-time data, only 36% of infants in the cohort were exclusive breastfed at five months. The estimate of 26% based on prospective two-monthly interval data for 66% of the cohort participants is probably a more realistic description of EBF practices.

Similar substantial gaps in quality of complementary feeding among Indian children have also been well documented, though largely by cross-sectional studies, and do

not match my findings on all components of complementary feeding. Nationally-representative data indicate that the diets of Indian children aged 6-23 months are low in non-dairy animal source foods (17%) and dietary diversity (33%) (Aguayo, 2017). A systematic review of observational studies in India found that 6-33% of children achieved dietary diversity (Manikam et al., 2018). Three cohort studies included in the review indicated that the age at introduction to complementary feeding varied, with 38% starting at four months in one cohort (Caleyachetty et al., 2013), 42% at 9-12 months in a pooled comparison of five cohorts (Fall et al., 2011), and 64% by six months in another cohort (Samuel et al., 2012). Widespread consumption of snacks among 6-23 month-old children (74% consumed sugary snacks, 57% consumed salty snacks) based on cross-sectional data has been reported in a previous study in this population (Bentley et al., 2015), as well as in urban Nepal, where 74% consumed a commercially produced snack (Pries et al., 2016).

7.7.2 Socio-economic patterning of IYCF practices

My findings on the socioeconomic and parental determinants of IYCF practices have shown that different factors operate for each stage and type of IYCF, and some factors that were associated with feeding across periods or indicators change direction at older ages or between practices. While household asset score was largely unrelated to any recommended practice, three other markers of SEP that were related to asset quintile (see Chapter 5), maternal education, (no) paternal smoking, and access to piped water, were also strongly associated with IYCF.

Maternal education was a strong predictor of nearly every component of IYCF. Educated women were more likely to stop exclusive or predominant breastfeeding, but were more likely to introduce solid foods. In the complementary feeding period, children of more educated mothers were less likely to consume snack foods. A study in rural Nicaragua identified the same pattern among rural households (Contreras et al., 2015). This dual relationship between maternal education and IYCF practices in LMIC contexts has been observed in several studies (Kimani-Murage et al., 2011, Malhotra et al., 2008, Zhao et al., 2017), though such studies often focus on breastfeeding or complementary feeding only.

Higher maternal education could be a proxy indicator for parental resourcefulness as well as more favourable socioeconomic position. Educated women might be able

to afford and confidently use non-breastmilk items (such as cow's milk or infant formula) early on if they encounter any lactation problems or are unable to continue to breastfeed exclusively. Their knowledge of IYCF educational messages could translate into greater likelihood of timely introduction to solid foods, though a study in Nairobi's informal settlements found a negative relationship (Kimani-Murage et al., 2011). As children grow older, educated mothers might be more successful in curbing their snack food consumption (Pries et al., 2017), possibly by restricting or limiting access to snacks. However, the lack of an influence on quality of complementary feeding in multivariable analyses does not support my resourcefulness hypothesis, although there was strong evidence of a protective effect in univariable analyses.

Paternal smoking was more consistently associated with sub-optimal IYCF practices. Children of fathers who smoked were 37% less likely to have diverse diets and 36% less likely to eat animal source foods, and 39% more likely to stop being exclusively breastfed. While paternal smoking was strongly associated with low household asset score, it is plausible that its negative association with IYCF works primarily through an SEP mechanism. However, it is possible that different mechanisms operate in the early breastfeeding and complementary periods.

Prospective studies in China and more recently in Hong Kong, two cultures where smoking among men is very common, have shown that paternal smoking was associated with shorter duration of any (Lok et al., 2018) as well as exclusive breastfeeding (Xu et al., 2010). The hazard of EBF cessation was 31% higher in children whose fathers smoked compared to children of non-smokers (Xu et al., 2010). Lok et al. (2018) suggest two possible reasons for the observed relationship. First, fathers who smoked preferred mixed feeding (and use of infant formula) to EBF, thus influencing the duration of EBF. Second, exposure to nicotine in second-hand smoke could reduce maternal milk production and ejection, encouraging women to reach for breastmilk substitutes. Either of these two explanations is plausible in the urban Indian context, though this merits further research to produce more conclusive answers.

The association of paternal smoking with complementary feeding is more likely a direct reflection of low SEP. Several studies have shown that poor quality IYCF practices, particularly dietary diversity and consumption of ASF, are linked to household food insecurity (Macharia et al., 2018) and family access to affordable

ASF (Cornelsen et al., 2016) in urban informal settlements in Kenya and in the Indian context (Aguayo, 2017), as well as maternal consumption of ASF (de Bruyn et al., 2017).

A similar explanation could apply to the strong association between ASF consumption and access to piped water (OR 2.33; 95%CI 1.7, 3.2). Animal source foods are expensive, and may also lead to greater domestic water use for food preparation and cooking. Water used to wash meat or poultry cannot be recycled for other purposes in the way that it can after washing rice or vegetables. Household water poverty in Mumbai's informal settlements is well documented (Subbaraman and Murthy, 2015, Subbaraman et al., 2012), and on a daily basis the lack of sufficient water can interfere with cooking (Subbaraman et al., 2015). Households with piped water are more likely to use it for cooking than those who access water from other improved sources (Muntalif et al., 2017). Families with secure access to piped water in this population were therefore more likely to buy and eat animal source foods regularly.

7.7.3 Infant and young child snacking

While maternal education had a protective influence on children's snack food consumption, its influence was much weaker in the reduced model (OR 0.76; 95%CI 0.6, 1.0; p= 0.053). The strongest predictor of snack food consumption at any age (across univariable, full and reduced models) was the presence of four or more children in the household, which increased the odds by as much as 61% in the full model and 51% in the more parsimonious model.

This finding suggests that children's snack food consumption is influenced not by adults but by other children. It is possible that older children are probably consuming snacks anyway, and share these with their younger siblings and cousins, or play an active role in buying food or feeding the younger ones. This hints at a wider custom of snacking behaviours originating in the first year of life that stretch into later childhood. Further, the effect can be interpreted as an even influence across the full complementary feeding period, suggesting that older children sustain the habit even if they do not initiate it. Another possibility is that poorer households have more children in them and parents enlist older children to look after younger ones, and snacks replace cooked meals that an adult would have provided.

Cross-sectional analyses also showed that salty and savoury foods were more frequently consumed at older ages, with sweet snacks favoured in the first year of life. Consumption of sugar sweetened beverages was largely due to tea-drinking (consumed as milky, sugary *chai* in most Indian homes), as soft drinks and soda may be expensive and inaccessible for most people in this population.

These dietary patterns raise questions about the nutritional quality of complementary feeding. First, regular tea consumption exposes young children to caffeine and added sugar, both of which are not recommended by the WHO (2008a), even though most caregivers use tea as a medium for softening crusty bread or biscuits for babies unable to chew (Palwala et al., 2009). Salty and savoury snacks tend to be deep fried, contributing calories as well as dietary lipid. The lipid quality of complementary foods is important for children's development and immune function (Agostoni et al., 2008). The source of cooking oil used by food manufacturers was not investigated, but previous research has shown that the quality of fatty acids is generally poor in LMICs, and insufficient to meet the increased nutritional demands of women and children in the 1000-day period (Michaelsen et al., 2011).

My findings also raise other non-nutritional questions. What makes consumption of snacks so widespread in Mumbai's informal settlements? In what contexts and situations do snacking instances take place? Why are children given snacks so early? What purpose do snacks serve in the cultural and social lives of young children? And why, when older children seem to be the primary custodians of the snacking culture in informal settlements, and by extension the quality of complementary feeding of their younger counterparts, do health and nutrition promotion programmes chiefly target mothers and adult caregivers?

Available evidence from four LMICs (Senegal, Tanzania, Nepal, and Vietnam) indicates that the most common reasons given by parents for feeding their children snacks were that children liked them, snacks were convenient, children demanded snacks, and that snacks were considered healthy or advertised as such (Pries et al., 2017). The normalization of snacks was also reported in Egypt, with parents often saying that sweet biscuits and sponge cake were 'ideal' complementary foods in routine diets and did not constitute 'outside' foods unsuitable for young children (Kavle et al., 2015).

A review of studies in high-income countries suggests that critical situations in which parents are more likely to offer infants snacks were often related to the competing demands of difficult circumstances and in social contexts where others encourage snacking, other children eat snacks, or in groups that have experienced food insecurity in living memory (Moore et al., 2017).

The extent to which these explanations apply in this cohort is debatable in the absence of more data, but presents a starting point for further investigation into the snacking phenomenon.

7.7.4 Long-term implications of observed IYCF practices

Most children (89% of those who had not yet received any solids) were introduced to complementary food within the 6-8 month window. However, information on the texture of foods given in this interval is not available, as the current WHO indicators do not capture this dimension of complementary feeding. Evidence from the ALSPAC cohort suggests that introduction to lumpy foods after nine months can lead to poor diet quality and more feeding problems at seven years (Coulthard et al., 2009). The probability of receiving solids for the first time between 4-5 months, that is, earlier than recommended, was 19%. Evidence from the Dutch ABCD cohort indicates that shorter duration of breastfeeding and early introduction of solids was associated with higher blood pressure in adulthood (de Beer et al., 2016).

Recent evidence from the Generation R cohort in the Netherlands suggests that protein intake, especially from animal sources, at one year is associated with higher BMI, height, and weight at nine years (Braun et al., 2016), although the effects were small (0.03 SD (95%CI 0.00, 0.06) among those with a 10 g/day higher total protein intake.

The long-term implications of frequent consumption of snack foods high in sugar, salt, and fat have not been studied (Michaelsen et al., 2017). However, there are obvious implications for the development of children's flavour preferences and later dietary practices, both of which are shaped by the quality of breastfeeding and complementary feeding (Anzman-Frasca et al., 2017, Mennella and Trabulsi, 2012). Some recent research suggests that exposure to sugar sweetened beverages at three years was associated with shorter telomere length at four years, indicating

faster cellular ageing. In the same study, exclusive breastfeeding had a protective effect on telomere length (Wojcicki et al., 2016).

7.7.5 Incorporating seasonality in longitudinal IYCF analysis

I did not account for seasonality in my analyses, as addressing such questions in the cohort would require additional methodological work.

The relationship between seasonality and IYCF practices is perhaps more difficult to quantify, and could differ between breastfeeding and complementary feeding. For breastfeeding, the seasonal influence possibly acts primarily at baseline, such as the season or month of birth which influences the initiation, maintenance, and cessation of breastfeeding (Das et al., 2016, Gonzalez-Chica et al., 2012). Data from rural Bangladesh and the Gambia have shown that infants consumed lower quantities of breastmilk in the main farming season when women devote more time to agricultural work (Rowland, 1986), indicating that those born in the six months before this season were more vulnerable to suboptimal exclusive breastfeeding. Complementary feeding is measured over an 18-month period, with starting dates often distributed over age as well as calendar time. For example, in this cohort, complementary feeding could begin between four and nine months of age, and data could cover all twelve months for a cohort of children born over a full calendar year. This provides a challenge as well as an opportunity.

It is unclear whether any seasonal influences on complementary feeding can be quantified using simple indicator variables for month of measurement, a periodic function that captures the oscillation of seasonal food availability or rainfall, or requires a polynomial to enable more flexible characterization. This is made more complicated when age is measured as a categorical variable. However, methodological insight could be gained from adapting approaches to time-series data in environmental epidemiology (Bernal et al., 2017, Bhaskaran et al., 2013).

Lagged entry to the complementary feeding stage and the calendrical distribution of births over a year is an opportunity to examine interactions between age and season. For example, do children born in the winter months (December to February) show delayed establishment of complementary feeding practices and dietary diversity because they are ready to begin consuming solids in the monsoon months

(June – August), a time of greater adversity due to household flooding in informal settlements (Subbaraman et al., 2014)?

7.7.6 Alternative modelling strategies

The analyses presented in this chapter look at the relationships between time-invariant characteristics and IYCF practices. I have not looked at how time-varying IYCF practices interact. For example, it would be useful to examine whether consumption of a diverse diet in the second year of life is influenced by introduction to snack foods in the first year of life, as well as concurrent and frequent consumption of snacks in the second year.

I looked at each IYCF stage separately, using a different technique within each analysis. When analysing data from the longest follow-up period, complementary feeding from 6-23 months, I used methods for un-ordered multiple failure time data. It would be possible to use methods for multiple ordered multiple failure-time data to understand progression along the full IYCF continuum from birth. Since complementary feeding cannot temporally precede breastfeeding, the order in which events occur is an important consideration in specifying the type of analyses and assumptions about the correlations between multiple events within the same person.

In some ways, IYCF is a multi-state process with competing risks. Competing risks are events that preclude the occurrence of an outcome of interest (Austin et al., 2016). The various states include exclusive, predominant, or partial breastfeeding in early infancy, and then complementary feeding once nutritional requirements are no longer met by breastmilk. Within the period of complementary feeding, children may transition between adequate or inadequate diets. At each point, they may switch states for a range of different reasons, or competing risks. For example, infants may switch from predominant to partial breastfeeding either due to use of infant formula or cow's milk alongside breastfeeding, or the very early introduction of complementary feeding. The differences between cross-sectional and longitudinal prevalence estimates of PBF and EBF in the cohort hint at such a possibility. In the complementary feeding period they may switch from adequate to inadequate diets either because they have had too many snack foods or are not being fed any animal source foods and vegetables. In both examples, it is likely that the competing risks have different causal structures. Use of formula could be linked to distribution of free samples in a community which women tried, whereas very early introduction of

complementary feeding could be due to the influence of traditional beliefs about nutrition or the practice of baby-led weaning. Similarly, inadequate diets characterised by low animal source food consumption might be due to household food insecurity, whereas diets comprising frequent snacking could be related to the influence of older children's consumption practices.

It is also possible that competing risks are correlated such that the presence of one risk has an effect on the other. For example, children who eat snack foods frequently (imitating the behaviour of older siblings) may have little appetite for a chicken and spinach soup (even though the family are able to afford chicken and spinach). Such situations violate the assumption of independence of irrelevant alternatives made by multinomial logit models, but could be estimated in correctly specified multilevel discrete-time competing risks models (Steele, 2011).

The analysis of longitudinal IYCF data presents many opportunities for methodological development.

7.7.7 Limitations

My analyses have several limitations. My approach of examining one IYCF component at a time is somewhat narrow. Multivariate (using multiple outcomes) analysis of ASF, snacks and continued breastfeeding from 6 to 23 months would have presented a more comprehensive understanding of IYCF patterns.

In my analyses of complementary feeding, I did not allow the influence of baseline factors to change over time. Autoregressive models use a random intercept model, which means that the effect of baseline characteristics is not allowed to vary within a child's set of measurements. The association of maternal education with dietary diversity is fixed across a child's measurements, i.e., the same at each age. Using a random intercept random slope model to allow the influence of background variables would be insightful, though incompatible with autoregressive modelling, effectively investigating whether maternal education becomes more or less important for dietary diversity as children grow.

Another limitation is that the suite of indicators focuses largely on dietary quality and duration of practices. The varied psychosocial aspects of child feeding, responsive feeding, food preparation practices, texture and consistency of food, and the amount

of food consumed were not measured. This is largely due to the lack of suitable indicators with which to measure these principles of IYCF (Ruel, 2017). While this limitation is attributable to methodology, it nevertheless raises the possibility of unmeasured confounding due to other aspects of feeding behaviour or caregivers' feeding decisions in response to children's appetite or growth. It would be worthwhile to examine if the magnitude of unobserved heterogeneity in the autoregressive models for complementary feeding would be reduced if some of these were taken into account. There is additional methodological work underway to develop new indicators (Ruel, 2017).

Chapter 8 Relationship between infant and young child feeding (IYCF) and linear growth: causal mediation analysis

Summary

In this chapter I explore the causal relationships between IYCF and child length, drawing on the counterfactual or potential outcomes approach. I justify my use of causal mediation analysis, provide an overview of counterfactual thinking and its application to public health problems, and describe its extension to longitudinal data with time-varying information. I then outline the statistical approach used to analyse data on IYCF and child length, and describe data preparation methods and the steps involved in carrying out exploratory regression analyses and causal mediation analysis using the parametric g-formula. I present findings from exploratory and mediation analyses and interpret them in light of current evidence on the relationship between IYCF and growth. I discuss the implications of my findings and key strengths and limitations of my research, and highlight areas for future work.

Statement of my contribution

I conceptualised the research question and hypothesized causal mechanisms presented in this chapter, with input from my supervisors on appropriate statistical methods to operationalise the analysis. After familiarising myself with the g-formula approach and methods, I prepared the cohort dataset for analysis along with an accompanying causal diagram. Bianca De Stavola (subsidiary supervisor) carried out the analyses presented here. I interpreted the findings, and wrote this chapter based on the output of the analysis and additional reading of the causal inference literature. Bianca De Stavola assessed the first draft of the chapter for accuracy and consistency with the statistical analysis carried out.

8.1 Introduction

In previous chapters I explored how the socioeconomic and household environment that children in urban informal settlements are born into affects their linear growth (Chapter 6) and time-appropriate IYCF practices (Chapter 7) in early life. I used SITAR to model growth and its relationship with baseline covariates, and discrete-

time survival analysis and autoregressive modelling to examine relationships between feeding patterns and baseline covariates.

In my systematic review of longitudinal studies on infant linear growth (Chapter 2), I identified IYCF and diarrhoeal disease as two crucial determinants of infant growth. While diarrhoea had a detrimental effect on growth, despite different exposure specifications and timing of measurement, the association of IYCF with growth was inconsistent across the 22 empirical studies that reported it (see Table 2.7). The emphasis on IYCF has also varied across recent conceptual papers on nutrition and growth ((Danaei et al., 2016, Hermanussen and Wit, 2017, Prendergast and Humphrey, 2014, Stewart et al., 2013)).

One study (Mallard et al., 2014) in my systematic review conceptualized IYCF as a mediator of the relationship between maternal education and growth. The rest treated it as an exposure or confounder. Few attempted to disentangle the temporal effects of feeding stages or components, focusing instead on the combination of different practices. For example, Bhargava (2016) focused on the cumulative effects of calcium and protein intake between 2 and 24 months on linear growth in the Cebu cohort in the Philippines.

A key challenge to understanding the relationship between IYCF practices and linear growth, after accounting for contextual factors, relates to the specification of temporal associations between variables, and the effect this has on choice of analytic approach. Socioeconomic position (SEP) and parental characteristics temporally precede and also shape infant growth and feeding practices. This means that IYCF cannot be a confounder of the effect of SEP or parental characteristics on growth, since a confounder must not lie on the causal pathway between an exposure and an outcome (Rothman et al., 2008b).

In addition, progression to certain complementary feeding practices can be influenced by breastfeeding practices at younger ages. Breastfeeding and complementary feeding are both independent prerequisites for growth, though any effect of breastfeeding on complementary feeding would mean that some of the influence of exposure to breastfeeding on growth outcomes is expressed indirectly through its relationship with complementary feeding. The relationship between breastfeeding and growth would therefore be *mediated*, not confounded, by complementary feeding.

Diarrhoeal disease in the first two years of life, linked to baseline factors such as water and sanitation, would affect linear growth, but its occurrence in a given period could also influence IYCF practices in subsequent periods through effects on the child's appetite or changes in caregiving practices. In turn, inadequate IYCF might increase the risk of diarrhoea in a subsequent period. Diarrhoea, when induced by baseline exposures or factors, can be considered a confounder of the mediating effect of IYCF on growth.

The presence of mediators or intermediate variables warrants special analytic methods that account for the nature of the mediating relationships (Robins, 1986, Robins and Greenland, 1992), especially when data are longitudinal (VanderWeele and Tchetgen Tchetgen, 2017).

I therefore framed my research questions as hypothesized mediated relationships, in order to decompose the direct and indirect (through a mediator) effects of exposures on child length at 24 months. In order to address my question, I selected a method that could adequately account for mediators, as well as confounders of the mediator-outcome relationship.

8.1.1 Research question

I address the following research question in this chapter:

Does consumption of animal source foods (ASF) in the complementary feeding period (6-23 months) mediate the relationship between predominant breastfeeding (0-5 months) and attained length at 24 months?

8.2 Overview of causal mediation analysis

8.2.1 Methods for dealing with mediators

The mediation analysis literature is based on two main approaches (De Stavola et al., 2015) a path analysis approach linked to path-specific effect estimation implemented using structural equation modelling (SEM), and a causal inference approach tied to the counterfactual or potential outcomes framework. The path analysis and counterfactual approaches are not mutually exclusive or antagonistic. Path analysis is often viewed as a subset of causal inference, and many causal inference studies use SEM (Brown et al., 1998b).

The counterfactual or potential outcomes framework, terms I use interchangeably despite some subtle differences in their interpretation (Daniel and De Stavola, 2019), is based on the notion of comparing an observed outcome to one that would have been observed in a counterfactual scenario. It is a way to understand how an outcome would have been different if an exposure had been different. The estimated effects are counterfactual because at least one of the two scenarios being compared is contrary to fact (Greenland et al., 2008). The methodological literature on potential outcomes explicates formal definitions of direct and indirect effects (Pearl, 2001, Robins and Greenland, 1992), thus enabling treatment of mediators, with clear assumptions under which these can be successfully identified. Identification is the process through which features of the distribution of observed data are linked to causal estimands, relying on assumptions to reconcile the absence of counterfactual information.

8.2.2 Key definitions in causal mediation analysis

Causal mediation analysis is the estimation of direct and indirect effects based on counterfactual models. It incorporates causal formalisms with unambiguously defined direct and indirect effects which apply to a linear or non-linear relationship between any outcome and mediator (VanderWeele, 2016b).

In this section Y refers to an outcome, X is an exposure, M is a mediator, and C represents a set of background confounders of the relationships between X and Y and between X and M.

The total causal effect (TCE) is the difference between the mean outcomes that would have been observed if everyone in a population were to receive an exposure compared to a scenario in which no one received the exposure.

Direct and indirect effects have been defined in several ways, including the controlled direct effect, the natural direct effect and natural indirect effect (Pearl, 2001, Robins and Greenland, 1992), and the more recently developed randomized interventional analogues of the natural direct effect and natural indirect effect (Vansteelandt and Daniel, 2017).

The controlled direct effect (CDE) of an exposure X on an outcome Y when the value of the mediator M is controlled (at *m*) compares hypothetical scenarios of X=1

vs X=0, with M held constant at *m* (across the population) in both situations. The CDE captures the direct effect of X on Y that is not mediated through M, but it does not capture a corresponding indirect path. In practice the CDE would implicitly prescribe a set value of the mediator for the whole population, which may not be a feasible intervention in some contexts (Pearl, 2001, Robins and Greenland, 1992).

The natural direct effect (NDE) of X on Y is a comparison of two hypothetical scenarios, with the value of the exposure set to X=0 vs X=1, but in both settings the value of M for each individual takes the value that would have occurred under no exposure (X=0). The natural indirect effect (NIE) is a comparison of two hypothetical scenarios where the exposure is set to 1 in both cases, while the mediator M changes from the value it would have taken under X=1 to the value it would have taken under X=0. The TCE can be interpreted as the sum of the NDE and NIE (Pearl, 2001, Robins and Greenland, 1992).

The interventional direct and indirect effects use the concept of randomized interventional analogues (RIA) of NDE and NIE. The RIA-NDE is the direct effect obtained when comparing X=1 vs X=0 with the mediator M randomly drawn from its counterfactual distribution under X=0 (controlled for confounders C). The RIA-NIE sets X=1 but compares scenarios where the mediator is drawn from its counterfactual distribution under X=1 vs X=0. The difference between RIA-NDE and RIA-NIE and their counterpart NDE and NIE is that in the former two the hypothetical shifts are applied to the distribution of the mediator conditional on the exposure, rather than at the individual level. However, the sum of RIA-NDE and RIA-NIE is not equal to the TCE, but it can be viewed as the overall effect of X on Y. In empirical settings, the TCE and the overall effect can both be estimated (Vansteelandt and Daniel, 2017).

The interventional analogues possess certain strengths in that they correspond to feasible interventions, and so have policy relevance, and their identification requires weaker assumptions than those for NDE and NIE (Vansteelandt and Daniel, 2017). The definitions of RIA-NDE and RIA-NIE also rely on population-level interventions in contrast to the individual-level ones implied by NDE and NIE, with practical implications for their use in public health (Moreno-Betancur and Carlin, 2018). As an example of its application, a recent article used data from the Framingham Heart Study to estimate the RIA-NDE and RIA-NIE of smoking behaviour on blood pressure over a ten-year period, accounting for weight change as a time-varying

mediator. The authors found that weight change conceals part of the harmful effect of smoking on hypertension that is not transmitted via weight (Lin et al., 2017).

The estimation of these mediation effects requires consideration of possible confounders that may introduce bias if not accounted for. More specifically, estimation of natural direct and indirect effects (and of their RIA counterparts) requires the assumption of no unmeasured confounding of the X-M, X-Y and M-Y relationships

8.2.3 Counterfactual thinking in public health

Use of the potential outcomes framework in epidemiology has increased in recent years in an attempt to understand the public health consequences of actions that are not easily or ethically testable using randomized experiments. Its use alongside traditional causal approaches such as Austin Bradford Hill's framework (Hill, 1965) has been reported, for example, in the decision-making process to set outdoor air quality standards in the US by evaluating the potential public health outcomes of varying levels of specific pollution reduction interventions (Glass et al., 2013).

Incorporating counterfactual thinking in public health research is also gaining greater attention as large scale population-based cohort studies are becoming more common. These are a rich source of health-related information which can be used to understand the longer-term health implications of early life exposures and their mechanistic nuances (Jackson et al., 2015, Pearce et al., 2016). Longitudinal observational datasets also provide an opportunity to understand, using potential outcomes approaches, how future health would have been affected by public health intervention in previous periods (Zhang et al., 2015). However, this translation to life course epidemiology has not yet been achieved across contexts and health issues, despite its potential to enhance life course investigations (Daniel et al., 2016, De Stavola and Daniel, 2016).

There is currently much debate around the suitability of the potential outcomes framework to address public health. Some of the most pressing public health challenges are influenced by factors such as ethnicity, gender, or race, which are arguably not amenable to well-defined intervention that can be simulated and easily interpreted in counterfactual scenarios (for a discussion see, for example, (Daniel et al., 2016, Vandenbroucke et al., 2016, VanderWeele, 2016a)).

8.2.4 Methods for dealing with time-varying information

Applications of counterfactual models to wider public health issues have also been encumbered by methodological limitations, though many of these have been addressed by recent advances in causal mediation analysis. Briefly, a large number of mediation analysis approaches have been applied only to study designs with an exposure and mediator measured once. In longitudinal studies where repeated measurement of multiple exposures and mediators is the norm, these methods had limited use.

The g-computational formula suggested by Robins in 1986 (Robins, 1986) overcame this partially by enabling estimation of the total effect for time-varying exposures and confounders. The effect of mediators can be estimated as the controlled direct effect by specifying fixed values of mediators and comparing exposure levels given these mediator values.

The more recent mediational g-formula (VanderWeele and Tchetgen Tchetgen, 2017) proposes a method to deal with time-varying mediators in causal mediation analysis. It relies on definitions of direct and indirect effects based on the RIA approach.

Another extension of Robins' g-computational formula attempts to tackle the problem of intermediate confounding (De Stavola et al., 2015). Most counterfactual models focus on non-parametric identification of direct and indirect effects, which are based on the assumption of no exposure-induced confounding of the mediator-outcome relationship. This is known as intermediate confounding, with variables inducing such confounding often denoted by the letter L. The proposed approach borrows from the parametric methods of SEM (which can easily deal with intermediate confounding) and incorporates the formalisms of the potential outcomes framework, coupled with an extension of the g-computational formula to include mediation analysis. Using this, it is possible to calculate TCE, NDE and NIE, and CDE in the presence of intermediate confounding, albeit under some additional assumptions (to those generally invoked for mediation analysis). This method has been previously illustrated in an analysis of binge-eating among adolescent girls from the ALSPAC cohort. Briefly, the analysis investigated whether the effect of maternal pre-pregnancy BMI (X) on offspring bingeing or overeating in adolescence (Y) was mediated by offspring BMI in childhood (M). The analysis included the

additional influence of birthweight as an intermediate confounder (L), and maternal education and mental health as baseline confounders (C).

Another study applied this parametric g-computational formula using a population record-linkage database in Finland to understand how antidepressant use (Y) would change if unemployment (X) was eliminated, accounting for the time-varying mediating and intermediate confounding effects of income, health conditions, and household status (M and L variables) (Bijlsma et al., 2017).

Recent advances in causal mediation analysis thus provide an opportunity to investigate the influence of breastfeeding practices on child growth mediated by complementary feeding in urban informal settlements.

Breastfeeding and complementary feeding are amenable to public health and community-based action, together and separately, making them suitable candidates for causal inference based on well-defined hypothetical interventions. Further, it would be unethical to randomize babies to varying levels and duration of exclusive breastfeeding in a low-income setting (Binns et al., 2017). Using observational data to simulate the effects of exclusive / predominant breastfeeding and complementary feeding on growth in a particular context thus provides an additional way to gain reliable and robust insight without conducting an RCT, or in addition to RCTs.

8.3 Methods

8.3.1 Outline of causal mediation analysis

I used the parametric g-computational formula applied to mediators and intermediate confounders (De Stavola et al., 2015) in my analysis of the effect of predominant breastfeeding (0-5 months) on attained length at 24 months mediated by consumption of ASF in the complementary feeding period (6-23 months), accounting for baseline confounding as well as intermediate confounding by diarrhoea.

The causal mediation analysis involved two stages.

In the first stage, I translated my research question into an appropriate causal Directed Acyclic Graph (DAG). The purpose of the causal DAG was to explicitly state the hypothesized relationships between the variables underpinning my

research question, and to understand whether the assumptions made about these relationships would be sufficient to carry out mediation analysis. I drew the causal DAG based on my knowledge of nutrition and health in urban informal settlements, and the findings of my systematic review (Chapter 2). I then used the cohort data to produce an analysis dataset that corresponded exactly to the variables in the casual DAG that were relevant for the analyses. Next, exploratory analyses following the hypothesized relationships in the DAG were carried out using regression models to help the interpretation of the mediation analysis results.

In the second stage, I specified hypothetical interventions corresponding to the exposure and mediator based on counterfactual notation, and explicitly defined the direct and indirect effects they would correspond to. I then stated the assumptions under which these effects would be estimated. Finally, model fitting and data simulation were conducted using the g-computation procedure, which led to the targeted estimated effects.

The g-computation procedure is implemented using the user-defined *gformula* command (Daniel et al., 2011) in Stata 13. The command can be used for time-varying confounders or mediation analysis. The procedure for an outcome measured at the end of follow-up involves two steps.

First, the relationships between variables in an observational dataset are modelled using linear, logistic, ordinal, or multinomial regression models. The models can either be fitted separately for each period using the same substantive model at each time point, or data can be pooled across periods. Second, the models fitted in the first step are used to simulate the potential outcomes for individuals if their exposure and mediator values had been set by intervention rather than those naturally observed in the dataset (Daniel et al., 2011).

In general, the modelling and simulation steps proceed sequentially over time intervals and are implemented by stating the full procedure in one command. Data at t_1 are modelled based on data at t_0. Data at t_1 are then simulated under alternative hypothetical interventions delivered as exposure at t_0 which can be compared. Next, data at t_2 are modelled based on data observed at t_1 and t_0, and then simulated under hypothetical interventions at t_1 and t_0. At each stage, the simulation is based only on previous values of exposures, confounders and

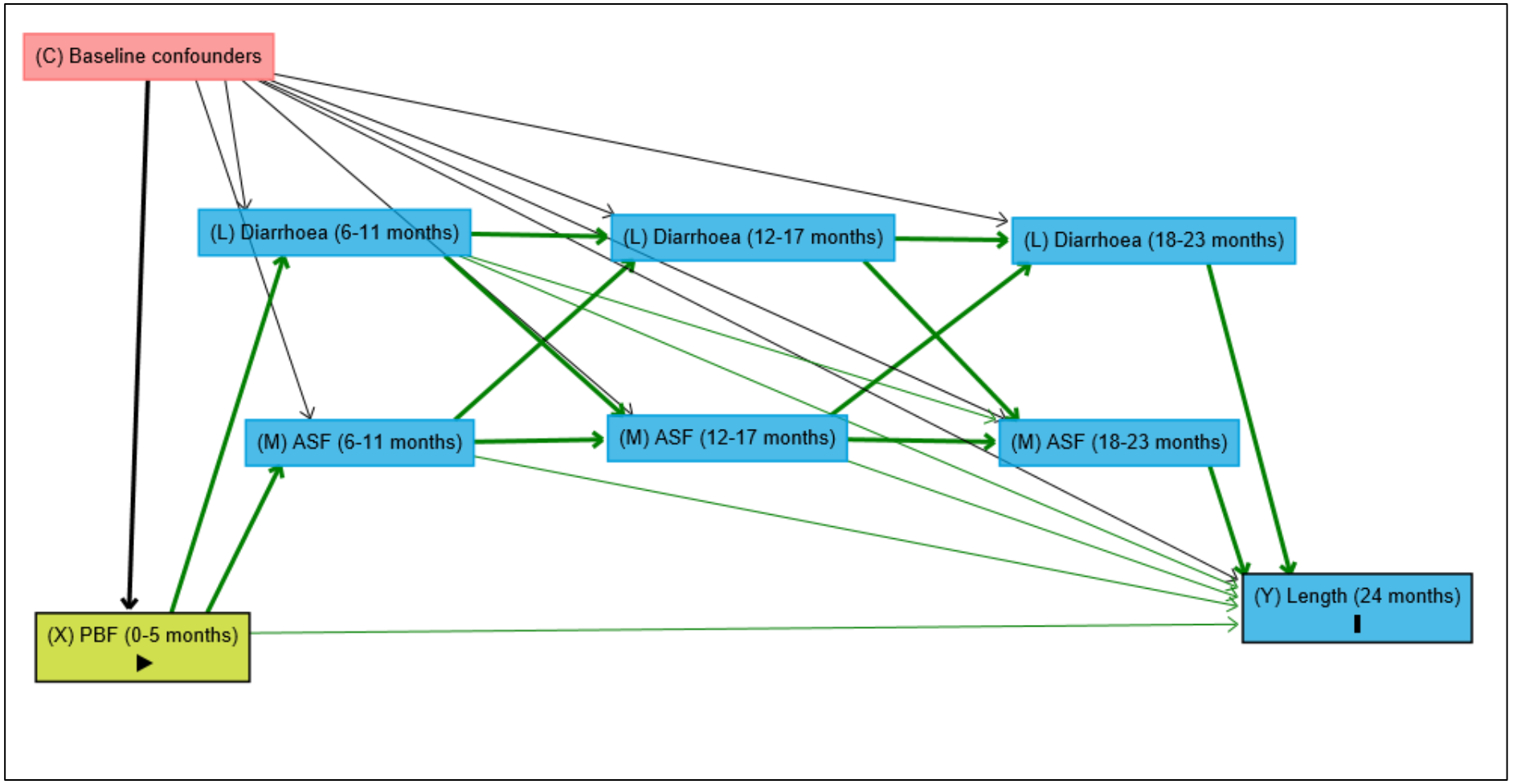
mediators. Standard errors and confidence intervals for the estimands are obtained using bootstrapping (Daniel et al., 2011).

8.3.2 Hypothesised causal Directed Acyclic Graph (DAG)

The causal DAG in Figure 8.1 describes the presumed associations between the exposure (X), baseline confounders (C), time-varying mediator (M), intermediate confounder (L), and the outcome (Y). The DAG describes all relationships that would need to be considered in order to decompose the causal effect of predominant breastfeeding from 0-5 months (X) on attained length at 24 months (Y) into an indirect effect acting through ASF consumption between 6-23 months (M) and a direct effect not mediated by ASF consumption, in the presence of intermediate confounding by diarrhoeal disease in the same period (L).

Based on my literature review (Chapter 2), I hypothesized that predominant breastfeeding from 0-5 months (X) would have a positive relationship with ASF consumption in the complementary feeding period at each time point between 6-23 months (M), which in turn would positively affect attained length at 24 months (Y). I also hypothesized that the direct effect of predominant breastfeeding (X), which would not involve the role of subsequent complementary feeding, would also have a positive influence on the outcome (Y). Further, I hypothesized that the exposure (X) would have a negative influence on the intermediate confounder (L), with greater duration of predominant breastfeeding protecting children from diarrhoea and inversely, shorter duration of predominant breastfeeding would predispose infants to diarrhoea. Finally, I hypothesized that the intermediate confounder (L) would have a negative effect on the outcome (Y), due to the deleterious influence of diarrhoeal illness on children's linear growth in early childhood.

Figure 8.1 Presumed directed acyclic graph (DAG) for causal mediation analysis



Note: Bold green arrows indicate pathways from X to Y that involve M and L variables. See Table 8.1 for descriptions of X, Y, L, M, and C variables.

The DAG also shows that there could be feedback between diarrhoea and ASF consumption, such that ASF in one period is affected by diarrhoea in the previous period, and in turn influences diarrhoea in the subsequent period.

I used the same set of baseline confounders used as exposures or confounders in analyses presented in previous chapters, with two additions. I included the first weight measurement obtained after the infant's birth. I hypothesized that the infant's size close to birth could shape breastfeeding practices (X), future complementary feeding (M_t), as well as the infant's length at 24 months (Y). I also included diarrhoea between 0 and 2 months as a baseline confounder, as it could affect breastfeeding practices (X), future diarrhoea (L_t), complementary feeding practices (M_t), as well as length at 24 months (Y).

I specified the exposure as an ordered categorical variable to compare mixed feeding with varying durations of PBF (0-2 months or 0-5 months). I wanted to understand if the effect of breastfeeding on length was different when recommended practice was adhered to for longer.

I chose PBF over EBF as the breastfeeding exposure for two reasons. First, the distinction between EBF and PBF is not strictly nutritional in this cohort. Consumption of dilute juice was very low (see Chapter 7), and so water was the main liquid that separated the two practices. Second, using EBF would have equated PBF with mixed feeding, which includes the use of formula, non-human milk and other liquids alongside breastfeeding, as well as the very early introduction of food in the fourth or fifth month. Using three types of feeding (mixed, EBF, and PBF) and varying durations would have led to a larger number of exposure groups (five or six depending on permissible feeding transitions) and reduced statistical power to detect meaningful differences.

8.3.3 Data preparation

The main objective of data manipulation was to collapse available information for each individual over the required number of intervals, and produce the correct data structure with just one observation for each participant. All study variables used in this chapter are summarised in Table 8.1. The Stata .do file I used to generate these from the cohort dataset is in Appendix 8.1.

Table 8.1 List of variables for proposed causal mediation analysis

Variable	Description	Labels or values
Outcome (Y)		
Length at 24 months	SITAR-predicted length at 24 months	Length in centimetres (continuous)
Exposure (X)		
Breastfeeding (0-5 months)	Predominant or mixed feeding	1= Mixed feeding (0-5 months), 2 = PBF (0-2 months), 3 = PBF (3-5 months)
Mediator (M)		
Animal source foods (6-11 months)	Consumption of ASF in four of six months	0 = No, 1= Yes
Animal source foods (12-17 months)	Consumption of ASF in three of six months	0 = No, 1= Yes
Animal source foods (18-23 months)	Consumption of ASF in four of six months	0 = No, 1= Yes
Intermediate confounder (L)		
Diarrhoea (6-11 months)	Any diarrhoea at 6-11 months	0 = No, 1= Yes
Diarrhoea (12-17 months)	Any diarrhoea at 12-17 months	0 = No, 1= Yes
Diarrhoea (18-23 months)	Any diarrhoea at 18-23 months	0 = No, 1= Yes
Baseline confounder (C)		
Weight in first month	Weight at 0-30 days	Weight in kilograms (continuous)
Diarrhoea 0-2 months	Any diarrhoea at 0-2 months	0 = No, 1 = Yes
Water	Access to piped water	0 = No, 1= Yes
Sanitation	Use of shared toilet	0 = No, 1= Yes
Household SEP	Household asset score	1 = Lowest, 2 = Second lowest, 3 = Middle, 4 = Second highest, 5 = Highest
Children in the household	4+ children in the household	0 = No, 1= Yes
Adults in the household	2+ non-parent adults in the household	0 = No, 1= Yes
Maternal age	Mother's age > 25 years	0 = No, 1= Yes
Maternal education	Mother studied beyond 6th standard	0 = No, 1= Yes
Maternal smoking	Mother smokes	0 = No, 1= Yes
Maternal height	Mother's height z-score	Height z-score (continuous)
Paternal age	Father's age > 30 years	0 = No, 1= Yes
Paternal education	Father studied beyond 6th standard	0 = No, 1= Yes
Paternal smoking	Father smokes	0 = No, 1= Yes
Pregnancy intention	LMUP score category	0 = Not planned, 1 = Planned
Infant sex	Female child	0 = No, 1= Yes
Abbreviations: LMUP, London Measure of Unplanned Pregnancy; SEP, Socioeconomic position		

I first prepared data for the outcome variable describing the length for each child at 24 months predicted by the SITAR model. I fitted a simple SITAR model to 16833 length observations for 975 infants, covering data from birth to 37 months of age, and then updated it to include terms for infant sex and seasonality (Fourier's term) as fixed effects. I used the *predict* function in the SITAR package to estimate each child's length at exactly 24 months from the sex-seasonality adjusted model. I

exported the predicted length measurements and ID for each child into a .dta file for use with Stata. I later merged this with the mediation analysis dataset, matching observations on the child ID variable. (See Chapter 6 for further details on SITAR).

I summarised IYCF in five age bands: 0-2 months, 3-5 months, 6-11 months, 12-17 months, and 18-23 months. I used the first two age bands to create the exposure variable for 0-5 months, and the last three to create the mediator variables.

Using breastfeeding data up to five months, I assigned infants a score of 1 for each month in which they were predominantly breastfed, and 0 if they were mixed fed. I calculated total scores within each time band. Infants who received PBF in all three months from 0-2 months were given a score of 1 for that time band, and those who received PBF in two of three months from 3-5 months were given a score of 1 for that time band. Based on a cross-tabulation of these two variables I created an ordered categorical variable encoding increasing duration of PBF as the exposure variable. Infants who were mixed fed from 0-2 months (and therefore could not qualify as PBF in the next period) formed the baseline group, those who received PBF from 0-2 months were coded 1, and those who received PBF into the 3-5 month period were coded 2.

In the complementary feeding period, I assigned infants a score of 1 for each integer month in which they were given ASF, and 0 if they were not. I calculated total scores within each of the three six-monthly time bands. I arbitrarily assigned a higher threshold in the first and last age bands of ASF consumption in four of six months, and a lower one in the middle band (ASF consumption at least three times between12-17 months). The mediator data were thus encoded in three binary variables, one for each time band.

I categorized data on diarrhoea in the same five time bands as the IYCF data, but coded any reported occurrence of diarrhoea as 1, rather than counting the frequency of diarrhoea within each period. The variable for the first time band (0-2 months) was used as a baseline confounder, and the rest were candidate intermediate confounders.

For the set of baseline confounders, I used 14 variables generated previously, related to SEP, parental characteristics, and infant characteristics (Table 8.x). I chose the research measurement of infant weight obtained by cohort investigators

within 30 days of the infant's birth over the institutional birth weight record because the latter exhibited significant digit preference. I compared the two data sources in scatter plots (Appendix 8.2) to check that research measurements were a satisfactory indicator of weight in early life.

8.3.4 Exploratory analysis

Exploratory data analysis began with baseline data descriptions and then the estimation of the associations between the exposure and outcome and the mediator and outcome. This included multivariable linear regressions of length at 24 months (Y) on breastfeeding practices (X) adjusted for baseline confounders (C), and also on ASF consumption (M) in each period adjusted for breastfeeding (X), and baseline confounders (C).

Next, associations of M and L variables with X and previous M and L variables were estimated in multivariable logistic regression analyses. Specifically, separate models were fitted to examine whether ASF consumption (M) in each interval was related to breastfeeding practices (X) after adjusting for baseline confounders. For diarrhoea in the complementary feeding period, the analysis examined the association between diarrhoea (L) in each of the three intervals (6-11 months, 12-17 months, 18-23 months) and feeding practice in the preceding period (X or M), adjusting for baseline confounders and previous feeding practice and diarrhoea.

Crude examination of the mediating role of M in the relationship between X and Y was carried out by including ASF at 6-11 months in the regression models of length at 24 months that included PBF and all baseline confounders.

8.3.5 Hypothetical interventions in the IYCF period and mediation effects of interest

The aim of this analysis is to quantify the contribution of ASF from 6 to 23 months to the causal association of PBF in early infancy with length at 24 months. I express this contribution in terms of natural direct and indirect effects, which add up to the total causal effect, i.e NDE+ NIE=TCE, and hence allow the calculation of percentage mediated effects. In this section I give a more detailed description of these entities and discuss the assumptions invoked to estimate them from the data.

As already discussed, I use counterfactual reasoning to express these quantities. Consider a hypothetical intervention on X (delivered, for example, through breastfeeding counselling or support groups) in which infants are exposed to varying levels of PBF or mixed feeding from 0-5 months, such that X=0 indicates mixed feeding from 0-5 months, X=1 indicates PBF from 0-2 months and mixed feeding thereafter (3-5 months), and X=2 indicates PBF that extends beyond the fourth month (0-5 months). X=0 is the natural baseline value of the exposure when infants are given mixed feeding. Mixed feeding would involve partial breastfeeding alongside use of other non-breastmilk items such as cow's milk, infant formula, and at older ages, some solid, semi-solid food, or soft foods. The two levels of intervention represent all *k* possible values of the exposure in this study.

Consider also a hypothetical intervention on M to increase consumption of animal source foods among young children once they have crossed the early breastfeeding period. This ASF intervention could be delivered through access to food subsidies for families in informal settlements or strengthening existing food value chains in urban areas to guarantee access in more deprived areas. Through this intervention children in the complementary feeding period (6-23 months) are fed one or more types of animal source food (dairy, eggs, or flesh foods) daily in at least four of six months in the first and last of three feeding stages (6-11 months, 18-23 months), and in three of six months in the middle stage (12-17 months), such that M=1 for those who receive the intervention. The mediator would take the value M=0 in a control group of children who consume ASF rarely or intermittently in each stage in the absence of more frequent access conferred by the intervention.

Let Y(X=0) be the potential outcome had X been set to 0, i.e. mixed feeding at 0-5 months, and Y(X=1) and Y(X=2) be respectively the potential outcome had X been set to 1 or 2. The total causal effect comparing level *1* of X versus level 0 [TCE_1], for example, is a comparison between the mean potential length at 24 months if, hypothetically, all children were predominantly breastfed from 0-2 months and mixed-fed thereafter, and the mean potential length at 24 months if all children received only mixed feeding from 0-5 months, is then defined as

$$TCE_1 = E\,[Y\,(X=1)] - E\,[Y\,(X=0)].$$

The total causal effect comparing level *2* of X versus level 0, is

$$TCE_2 = E\,[Y\,(X=2)] - E\,[Y\,(X=0)].$$

Using a similar notation for the mediator, Let M(*X*=0), M(*X*=1) and M(*X*=2) be the potential mediators had X been set to 0, 1 or 2. Since the mediator is downstream from X, we can then express the potential outcomes above, Y(X=k), as $Y\,[X = k, M\,(X = k)]$, without changing their meanings, for k=0, 1, 2. It follows that the total causal effects (for k=1, 2) can be expressed as:

$$TCE_k = E\left[Y\left(X = k, M\,(X = k)\right)\right] - E\left[Y\left(X = 0, M\,(X = 0)\right)\right]$$

This expanded notation allows us to express formally the natural direct and indirect effects introduced earlier. The natural direct effect for level *k* of X [NDE_k], for example when *k*=1, is a comparison between the mean potential length at 24 months if, hypothetically, all children were predominantly breastfed from 0-2 months and mixed fed thereafter (i.e., when X=1), and the mean potential length at 24 months if, hypothetically, all children received only mixed feeding from 0-5 months, while at the same time ASF consumption in both scenarios is set to its potential values under baseline exposure (i.e., when X=0). Since ASF consumption does not vary between scenarios, the NDE estimates the effect of breastfeeding on length that is not transmitted through subsequent ASF consumption. This can be written as:

$$NDE_k = E\left[Y\left(X = k, M(X = 0)\right)\right] - E\left[Y\left(X = 0, M(X = 0)\right)\right]$$

The natural indirect effect for level k of X [NIE_k], for example when k=1, is the difference between the TCE_k and the NDE_k. It compares two hypothetical scenarios, in both of which all children have exposure level k, but the values of ASF consumption vary. In the first, ASF consumption is set at the value it would have taken when children have exposure level k [NIE_1], and in the second scenario ASF consumption is set at the value it would have taken if all children had been mixed fed from 0-5 months (i.e., M when X=0). Since ASF consumption varies between scenarios while breastfeeding is held constant, the NIE estimates the effect of breastfeeding on length transmitted through ASF consumption. This can be written as:

$$NIE_k = E\left[Y\left(X = k, M(X = 1)\right)\right] - E\left[Y\left(X = k, M(X = 0)\right)\right]$$

The proportion mediated [PM_k] is the NIE_k divided by the TCE_k.

8.3.6 Assumptions

Estimation of the TCE, NDE, and NIE described above relies on assumptions central to the formulation of these contrasts as causal effects

The first is the assumption of no interference, which states that an individual's exposure status does not influence the outcome of another, and one individual's mediator value does not affect the outcome of another. This assumption implies that it is not possible for the breastfeeding status of one child to affect the ASF consumption and growth of another, and that one child's ASF consumption does not influence another's child's attained length.

The second is the consistency assumption, which states that the hypothetical interventions used to compare counterfactual scenarios and interpret causal effects are very close to the phenomenon described by the data. For set values of X and M, the potential outcome Y(*x, m*) corresponds to the observed outcome Y among individuals with X=*x* and M=*m,* and for each value X, the potential mediation M(*x*) corresponds to the observed value M among those for whom X is observed to take that value *x*. This assumption implies that predominant breastfeeding from 0-2 months or 0-5 months, and consumption of ASF in each of three complementary feeding periods correspond to actions that would result from well-defined and specific interventions.

The third is the conditional exchangeability assumption, which states that after stratification by confounders C and intermediate confounders L, individuals' exposure status X is randomly distributed within strata of C. Further, once individuals are stratified by X, C, and the intermediate confounder L, their allocation within strata of M is essentially random. Conditional exchangeability can be understood as an assumption of no X-Y confounding and no X-M confounding conditional on C, and no M-Y confounding conditional on C, X, and L. This implies that after conditioning on baseline factors there is no further confounding of the relationship between breastfeeding and ASF or infant growth, and that conditioning on baseline factors, breastfeeding, and subsequent diarrhoea, there is no further unmeasured confounding of the effect of ASF on growth.

An additional assumption made in the presence of intermediate confounding, as it occurs in our application, is of no X-L interaction, i.e. no effect modification of the exposure due to the intermediate confounder.

8.3.7 Estimation

There are several possible approaches to estimating the natural effects described above. Given the time-varying nature of the mediator we have used g-computation as implemented in the the gformula command in Stata (Daniel et al., 2011). Estimation is achieved by Monte Carlo simulation, whereby large numbers of potential mediators and potential outcomes are simulated under different scenarios, with simulations generated from models that are first fitted on the observed data. Assuming these models are correctly specified, these simulations lead to average potential outcomes under different hypothetical interventions that are then summarised and compared. Comparisons are expressed here in terms of expected mean differences in length at 24 months, with standard errors calculated via bootstrap (with 1000 bootstrap samples). Details of the syntax used are given in Table 8.2.

First, all analysis variables were listed. Second, the analysis was specified as a mediation analysis, and then the Y, X, M, L, and C variables were listed in groups. Baseline and control values for the X and M variables were also specified, and a further option indicated that the analysis included a single categorical variable for X. The next component listed the type of parametric models to be used in data simulation for the outcome, mediator variables, and intermediate confounder. This was followed by equations corresponding to these models, with the outcome and exposure variables separated by a colon for each equation. Interpretation of the results relies on the assumptions described in section 8.3.6. Given the very weak association found between ASF and diarrhoea at later ages, and to avoid introducing bias because of unmeasured confounding between diarrhoea at these later ages and the outcome, we only included diarrhoea at 6-11 months as an intermediate confounder. Hence five equations were specified: one for Y at 24 months, three for M, at 6-11, 12-17, and 18-23 months, and one for L at 6-11 months. Finally, options for the Monte Carlo simulation were specified. These included the number of bootstrap samples, the size of the Monte Carlo simulated dataset, and a random number seed.

Results of running this command include estimates of the total causal effect, the natural direct and indirect effects, and the proportion mediated.

Table 8.2 Explanation of components of g-formula syntax

Component	Syntax
Invoke g-computation command and list all variables in analysis	gformula y m_12 m3 m4 m5 l3 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5 c6 c7 c8 c9 c10 c11 c12 c13 c14,
Specify as mediation	mediation
Outcome variable	outcome(y)
Exposure variable	exposure(m_12)
Mediator variables	mediator(m3 m4 m5)
Intermediate confounder	post_confs(l3)
Baseline confounders	base_confs (wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5 c6 c7 c8 c9 c10 c11 c12 c13 c14)
Specify exposure as one categorical exposure	oce
Specify baseline value for exposure and control values for mediators	baseline(0) control(m3:0, m4:0, m5:0)
Specify type of parametric model for each equation for simulation	commands(y:regress, m5:logit, m4:logit, m3:logit, l3:logit)
List equations for each model	equations (y: i.m_12 m5 m4 m3 l3 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5 c6 c7 c8 c9 c10 c11 c12 c13 c14, m5: i.m_12 m4 m3 l3 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5 c6 c7 c8 c9 c10 c11 c12 c13 c14, m4: i.m_12 m3 l3 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5 c6 c7 c8 c9 c10 c11 c12 c13 c14, m3: i.m_12 l3 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5 c6 c7 c8 c9 c10 c11 c12 c13 c14, l3: i.m_12 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5 c6 c7 c8 c9 c10 c11 c12 c13 c14)
Monte Carlo simulation options	minsim samples(1000) moreMC simulations(30000) replace seed(1202)

8.4 Results of descriptive and multivariable analyses

8.4.1 Description of sample

Complete data on all variables were available for 438 of 978 (45%) children, of whom 52% were female (Table 8.3). (See Chapter 5 for details on determinants of missing data). Nineteen percent were predominantly breastfed from 0 to 2 months,

and 42% from 0 to 5 months. Consumption of ASF was more common at older ages (38% between 6 and 11 months and 71% between 18 and 23 months).

Table 8.3 Characteristics of 438 children with complete data included in analysis

Variable	Mean (SD) or %
Outcome (Y)	
Length at 24 months (cm)	79.7 (3.4)
Exposure (X)	
Mixed feeding at 0-5 months	39
PBF at 0-2 months	19
PBF at 0-5 months	42
Mediator (M)	
ASF at 6-11 months	38
ASF at 12-17 months	62
ASF at 18-23 months	71
Intermediate confounder (L)	
Diarrhoea at 6-11 months	56
Baseline confounder (C)	
Weight in first month (kg)	2.9 (0.5)
Diarrhoea 0-2 months	22
Access to piped water	61
Use of shared toilet	84
Baseline confounder (C)	
Household asset quintile	
Lowest	21
Second lowest	21
Middle	21
Second highest	18
Highest	19
4+ children in the household	41
2+ adults in the household	42
Mother's age ≥ 25 years	53
Mother studied beyond 6th standard	47
Mother smokes	15
Maternal height (z-score)	-0.3 (0.9)
Father's age ≥ 30 years	47
Father studied beyond 6th standard	57
Father smokes	57
Planned pregnancy	57
Female child	52

8.4.2 Multivariable models and crude mediation analysis

In a multivariable model of the relationship between breastfeeding and length at 24 months adjusted for all baseline confounders, PBF at 0-2 months had a weak and

not significant negative association with length (-0.49 cm; 95%CI -1.3, 0.3; p= 0.214), with stronger and larger negative associations for infants who were predominantly breastfed for longer (-1.1 cm; 95%CI -1.7, -0.5, p= 0.001). The relationships were slightly larger when the first weight measurement and diarrhoea 0-2 months were excluded from the model (-0.6 cm; 95%CI -1.4, 0.2; p= 0.138 for PBF 0-2 months, and -1.2 cm; 95%CI -1.8, -0.6; p<0.0001 for PBF 0-5 months) indicating confounding by these factors.

Predominantly breastfed infants were less likely to consume ASF in the complementary feeding period. The negative association was stronger for 6-11 months (aOR 0.33; 95%CI 0.2, 0.5) and 12-17 months (aOR 0.34; 95%CI 0.2, 0.6) than 18-23 months (aOR 0.58; 95%CI 0.3, 0.9) among infants who were PBF from 0-5 months.

ASF consumption in each period was positively associated with length at 24 months after accounting for breastfeeding and baseline confounders. The association was largest for the early complementary feeding period (0.96 cm; 95%CI 0.4, 1.6; p= 0.002 for ASF consumption at 6-11 months), with smaller effects in later periods (0.88 cm; 95%CI 0.3, 1.5; p= 0.003 at 12-17 months and 0.67 cm; 95%CI 0.01, 1.3; p= 0.038 at 18-23 months).

In a multivariable model adjusted for baseline confounders and previous diarrhoea (3-5 months), there was very weak evidence of a negative relationship between predominant breastfeeding and subsequent diarrhoea at 6-11 months (aOR 0.9; 95%CI 0.6, 1.6 among infants PBF 0-2 months, and aOR 1.45; 95%CI 0.9, 2.3 among infants PBF 0-5 months). Diarrhoea in this period was more strongly related to previous diarrhoea (aOR 1.79; 95%CI 1.2, 2.7).

Results of a crude mediation analysis showing total effects and direct effects (accounting for ASF at 6-11 months only) are presented in Table 8.4.

Table 8.4 Results of crude mediation analysis

	Total causal effect				Direct effect			
	Estimate	**95%CI**		**p**	**Estimate**	**95%CI**		**p**
PBF 0-2 months	-0.50	-1.29	0.29	0.214	-0.36	-1.15	0.43	0.369
PBF 0-5 months	-1.10	-1.72	-0.48	0.001	-0.87	-1.50	-0.25	0.007
ASF (6-11 months)					0.96	0.37	1.56	0.002
Diarrhoea (0-2 months)	-0.05	-0.73	0.63	0.889	-0.02	-0.69	0.65	0.962
Weight in first month	1.48	0.89	2.07	<0.0001	1.47	0.88	2.05	<0.0001
Piped water	0.82	0.21	1.42	0.009	0.70	0.10	1.31	0.022
Shared toilet	-0.66	-1.50	0.17	0.121	-0.65	-1.47	0.18	0.125
Asset quintile								
Lowest	-0.91	-1.78	-0.04	0.041	-0.82	-1.69	0.04	0.062
Secondlowest	-0.55	-1.41	0.31	0.209	-0.54	-1.39	0.32	0.216
Secondhighest	-0.09	-1.01	0.82	0.84	-0.13	-1.04	0.78	0.777
Highest	0.36	-0.64	1.35	0.478	0.38	-0.61	1.36	0.454
Household children ≥4	-1.08	-1.73	-0.44	0.001	-1.12	-1.76	-0.48	0.001
Household adults ≥2	0.53	-0.10	1.16	0.098	0.54	-0.09	1.16	0.091
Maternal age ≥25	0.29	-0.45	1.03	0.442	0.27	-0.46	1.00	0.468
Mother smokes	0.54	-0.29	1.36	0.2	0.59	-0.23	1.40	0.158
Maternal education ≥6th	0.90	0.26	1.54	0.006	0.78	0.14	1.42	0.017
Maternal height z-score	0.72	0.42	1.02	<0.0001	0.72	0.42	1.02	<0.0001
Paternal age ≥30	0.10	-0.62	0.82	0.777	0.09	-0.63	0.80	0.81
Paternal education	-0.27	-0.88	0.35	0.396	-0.25	-0.86	0.36	0.417
Paternal smoking	0.09	-0.51	0.68	0.778	0.08	-0.51	0.67	0.787
Planned pregnancy	1.00	0.41	1.59	0.001	1.13	0.54	1.71	<0.0001
Female	0.40	-0.17	0.96	0.169	0.38	-0.18	0.94	0.186

8.5 Mediation analysis

The estimated total causal effect (TCE), natural direct and indirect effects (NDE and NIE), and proportion mediated (PM) of predominant breastfeeding on attained length at 24 months are presented in Table 8.5. They are all mean differences expressed in cm.

Table 8.5 Estimation of the total causal effect of predominant breastfeeding on attained length at 24 months, and of the effects mediated and not mediated by ASF consumption

Estimand	PBF at 0-2 months (X=1 vs X=0)		PBF at 0-5 months (X=2 vs X=0)	
	Estimate (95%CI)	p	Estimate (95%CI)	p
Total causal effect (TCE)	-0.51 (-1.24, 0.22)	0.173	-1.10 (-1.75, -0.46)	0.001
Natural direct effect (NDE)	-0.58 (-1.34, 0.19)	0.140	-0.77 (-1.41, -0.13)	0.018
Natural indirect effect (NIE)	0.06 (-0.14, 0.27)	0.531	-0.33 (-0.55, -0.12)	0.003
Proportion mediated	-0.13	0.994	0.30	0.079

Abbreviations: PBF, predominant breastfeeding; SE, standard error

There was strong evidence of a negative total causal effect of predominant breastfeeding at 0-5 months on length at 24 months, relative to no PBF. If all infants were predominantly breastfed at 0-5 months, the average length at 24 months among the population would be 1.1 cm lower [95%CI: -1.75, -0.46; p= 0.001] than if they were all mixed fed from 0-5 months.

This negative total effect was only partially (30%) mediated by the effect of ASF consumption in the complementary feeding period (-0.33 cm; 95%CI -0.55, -0.12; p= 0.003), with most of the effect (-0.77 cm, 95%CI -1.41, -0.13; p= 0.018) being attributable to pathways from breastfeeding to linear growth that do not involve ASF (at least as measured).

The equivalent TCE for PBF for 0-2 months was -0.51 (-1.24, 0.22), indicating a weaker but still negative effect of PBF, with most of this effect not being mediated by ASF.

8.6 Discussion

This is the first study to decompose the effects of IYCF on attained length at 24 months into the direct effect of predominant breastfeeding in early infancy and its indirect effect expressed through ASF consumption in the complementary feeding period in urban informal settlements. My causal mediation analysis showed that if infants in this population had been predominantly breastfed from 0 to 5 months, they would have been 1.1 cm shorter than infants mixed-fed for the same period. Less than a third of this effect was mediated by subsequent complementary feeding with animal source foods from 6-23 months.

The results provided some nuance. The strong negative effect of PBF on length was apparent only for a longer duration of PBF. If predominantly breastfed infants switched to mixed feeding at three months, it would make little difference to length at 24 months in this population, though it could have detrimental consequences for children in many other ways. PBF was also negatively associated with subsequent ASF, which predicted greater attained length, explaining why the negative effect of PBF on length was largely direct.

The estimated total causal effect (1.1 cm) corresponds to about a third of the standard deviation (3.4 cm) of the cohort's mean length (79.7 cm). Predominantly breastfed infants would be 0.3 SD shorter than mixed fed infants. The proportion of this effect that is independent of complementary feeding is 70%, about 0.77 cm, which translates to less than a quarter of an SD. Comparison with corresponding values for 24 month old children based on the WHO Growth Standards yields similar results (the SD is 3.2 cm for girls and 3.1 cm for boys). While these effects are statistically significant, they are not substantial. This cohort of children is much shorter than the WHO growth standard, with mean length below -2SD for both sexes. They are much closer to the median length-for-age of 18 month old children (WHO, 2019a, WHO, 2019b). Given the large deviation from the global standard, a third of a standard deviation is unlikely to prevent growth faltering. My analysis also did not capture any change in length between birth and two years, and so it is difficult to assess whether the small difference in attained length at 24 months masks a much larger effect on growth velocity over 24 months.

Diarrhoea, included as a baseline and intermediate confounder, had a weak relationship with attained length. This contradicts a vast body of literature on the

deleterious effects of diarrhoea for linear growth, especially in early infancy (see Chapter 2). It is possible that length gain is preserved at the expense of weight among children who experience diarrhoea in this setting. Further, reported use of infant formula was very low in this community (see Chapter 7). Therefore the likelihood of it contributing to diarrhoea through use of contaminated water for reformulation is low. A more plausible explanation is that I specified diarrhoea too crudely by using data on any reported diarrhoea over three months (0-2 months) or six months (6-11 months).

However, a recent large WASH trial in Zimbabwe has shown very limited effects on linear growth (Humphrey et al., 2019). It is possible that the threshold of WASH infrastructure or dose is much higher than that tested in LMIC trials in order to produce larger improvements in linear growth outcomes. WASH requirements are probably much closer to the levels in HICs (Husseini et al., 2018).

8.6.1 Why are predominantly breastfed infants shorter at 24 months?

I based my definitions of hypothetical interventions on IYCF practices that are considered highly desirable according to international guidelines (WHO 2010). So why would predominant breastfeeding have a negative effect on length at 24 months? My findings are consistent with recent evidence suggesting that exclusively or predominantly breastfed infants exhibit poorer linear growth (De Hoog et al., 2011, Kattula et al., 2014, Zhang et al., 2017). There are two possible explanations for the relationship observed in my analysis.

One, mixed-fed infants, who are frequently exposed to cow's milk in this population, could have higher levels of insulin-like growth factor 1 (IGF-1) and insulin. These hormones have stimulating effects on linear growth in infancy (Hoppe et al., 2006, Michaelsen, 2013). Predominantly breastfed infants thus gain less length in the first six months, and this early deficit translates into lower length at 24 months, one which is largely direct. However, the long term effects of greater exposure to cow's milk in infancy on later adult health and cardiometabolic outcomes are unclear (Martin et al., 2011).

Two, discontinuation of PBF or EBF is partly related to socio-economic position in this cohort. Women with higher educational attainment were more likely to stop EBF or PBF at any point between 0 and 5 months (see Chapter 7), such that longer

duration of EBF or PBF was more common among those of lower SEP. While higher SEP prevented adherence to PBF in the first six months, it was a strong determinant of favourable complementary feeding practices. PBF thus becomes a proxy for low SEP variables that act pre- and post-natally. It is also possible that there is residual confounding of these relationship which is not captured by the SEP variables I used in my analysis.

Another interpretation is that the difference in length at 24 months between PBF and mixed-fed children is insignificant in the long term, if the former show compensatory growth in the mid childhood and pubertal phase, and both groups end up with similar adult heights.

8.6.2 Methodological significance

8.6.2.1 Applying counterfactual thinking to study nutrition in childhood

A key contribution of this analysis is to epidemiological methods for investigating nutrition and growth in early childhood using longitudinal data. By successfully conducting a causal mediation analysis to study the effect of IYCF on growth, I have demonstrated the potential of counterfactual thinking to understand a real world public health issue in a vulnerable population.

The results of a crude mediation analysis using multivariable linear regression were almost identical to the outcome of the formal analysis using the g-formula. While the potential outcomes approach seems to have limited added value in this context, comparing the two methods has been a useful exercise for an issue on which conflicting evidence abounds. Applying the causal formalisms of counterfactual thinking to my research question encouraged me to be more transparent and explicit about the relationships between variables in my analysis, and the assumptions that underpinned them. Arguably, one of the intended aims of the counterfactual approach is to increase transparency in causal analyses in epidemiology (Bollen and Pearl, 2013). Given the ambiguity and debate in the theoretical, empirical and methodologic literatures on nutrition and growth and the heterogeneity of analytical methods currently in use (Leung et al., 2018), I would argue that the greater transparency required by the potential outcomes approach would alone serve to address some of these ambiguities more effectively and efficiently.

Child nutrition as a health issue is also rooted in a rights based approach (Jonsson, 1981), with the duty of providing children care and nutrition firmly on parents and society. This, along with clarity on the benefits of many nutritional interventions (Bhutta et al., 2013), imposes ethical bounds on experimental research. Turning to observational data to make causal inference presents a useful alternative. This paradigm has been adopted by epidemiologists to study the health benefits of exclusive breastfeeding, using cross-cohort comparisons to exploit different confounding structures across contexts (Brion et al., 2011). But it has not been expanded to include the counterfactual approach. My research provides some evidence of its usefulness and scope.

8.6.2.2 Life course mechanisms of IYCF

This analysis also corroborates a methodologic argument I made in Chapter 6 on the mechanisms linking IYCF and linear growth in informal settlements. The original shape invariant model (Beath, 2007) which formed the foundation of the SITAR model accommodates time-varying covariates but assumes that their effects are cumulative. I argued that attempting to include IYCF data under the accumulation hypothesis would produce biased results if the mechanisms linking IYCF and growth were closer to the critical period or mobility hypotheses. While my causal mediation analysis was not set up to test competing hypotheses of life course mechanisms, an endeavour that would itself need to overcome problems similar to those of disentangling age-period-cohort effects (Hallqvist et al., 2004), my analysis is nonetheless insightful.

Predominant breastfeeding does not have a positive effect on attained length in this population, and neither does it lead to greater ASF consumption in the complementary feeding period. Early success in adhering to IYCF guidelines does not form a protective chain of practices (Kuh et al., 2003) that stretches into the second year of life. This is an area that requires more methodological work to understand how best to tease apart the effects of feeding over the IYCF continuum by integrating life course approaches into current methods. Integrating such mechanistic analysis with growth modelling using SITAR is a further research challenge.

8.6.2.3 Simulation and the consistency assumption

In the methodological literature on applied causal inference, the consistency assumption is concerned with whether hypothetical interventions are ‘well-defined’

enough to be meaningful and useful to public health issues (see for example,)(Hernan, 2016, Hernan and Taubman, 2008, Pearl, 2010). I compared counterfactual scenarios that correspond to interventions on the exposure and mediator which can be feasibly delivered in the real world.

Encouraging women to continue predominant breastfeeding for longer is certainly feasible through individual and community-level intervention. Intensive interpersonal counselling, mass-media campaigns, and community mobilization to improve breastfeeding practices have shown significant impact in the large scale Alive and Thrive project in Bangladesh and Viet Nam. Intervention fidelity was high (85% to 98% for different services) for interpersonal counselling in Bangladesh and 70% for the mass media in Viet Nam (Menon et al., 2016a). The intervention also improved complementary feeding practices (Menon et al., 2016b). Direct provision of animal source foods in the complementary feeding period in populations with high linear growth faltering has also been tested successfully. In a recent trial in rural Ecuador, children were given an egg per day for a three month period between 6 and 9 months, leading to a 0.63 SD increase in LAZ (Iannotti et al., 2017).

Further, the baseline or control values also correspond to realistic situations. In the absence of intervention or support, it is possible that babies would be mixed fed at 0-5 months because it proves the most convenient practice in an informal settlement environment. Mixed feeding would reduce the burden on the mother by allowing her to partially breastfeed when domestic or economic tasks compete with the demands of EBF, and to also rely on other caregivers to feed the infant (Kimani-Murage et al., 2015b). In the absence of material or food assistance to boost children's ASF consumption in deprived urban areas, most children would receive ASF items with a frequency that matches the family's intermittent access. There is evidence that low consumption of animal source foods among young children is widespread in LMIC settings (White et al., 2017).

By mapping my analysis to strategies employed in experimental research, I have demonstrated the application of causal inference from observational data by simulating intervention scenarios that are already common in trials. There is further potential to exploit the policy-relevance of counterfactual thinking to push forward causal inference in public health nutrition.

8.6.3 Limitations

My research has some limitations. There was significant attrition and the analytic sample of complete cases included less than half the original cohort. In some ways this is a potential source of selection bias, though my analysis of missing data in Chapter 5 suggest it is unlikely. It also represents children from families who have sustained, long-term exposure to the urban informal settlement environment. The smaller sample size nonetheless indicates statistical inefficiency.

Unmeasured confounding is a possibility in all observational cohort studies. Despite including a range of socioeconomic and parental confounders measured at or close to birth, there are possible omissions in my analysis. Household-level food security, as well as how it differentially affects women and children, would have provided information on how access to food affects IYCF, diarrhoea episodes, and child growth. Its relationship with LAZ outcomes has been well-documented (Psaki et al., 2012), but data on food security were not collected as part of this cohort's baseline survey. Children's exposure to living areas contaminated by animal faeces is a risk factor for environmental enteric dysfunction and linear growth faltering (Ngure et al., 2014), but keeping chickens or goats in an informal settlement (Dominguez-Salas et al., 2016) or urban areas (Fierstein et al., 2017) provides families with a readily-available source of ASF or additional income. Data on livestock-keeping at the household or community level were not collected in this study.

An additional source of unmeasured confounding could be maternal autonomy or another allied construct that reflects women's empowerment. Several studies have documented negative relationships between lack of female autonomy and child growth and feeding practices. In the South Asian context, women's control of resources and autonomy, workload and time, and social support are specific domains that reflect the ways in which maternal empowerment influences child nutrition (Cunningham et al., 2015). Data used in my research did not capture female autonomy directly, as this was not hypothesized as a confounder in the original protocol or my research questions. It is possible that maternal autonomy exerted an influence on child feeding, illness, as well as linear growth in the cohort, making it a plausible candidate for unmeasured baseline confounding.

The outcome variable I used is a crude measure of linear growth. While the predicted measurements were based on a large longitudinal dataset, there was

some loss of information. The SITAR model's triplet of growth parameters for each child are meant to be interpreted together, and use of any one parameter would not be meaningful without the other two (Cole et al., 2010). The causal mediation analysis approach I used does not accommodate multiple outcome variables, which shaped my decision to use predicted length at 24 months, but precluded any inference of how the hypothesized mediated relationships might influence different parameters of growth in early life. This requires further methodological work.

8.7 Conclusion

My findings corroborate Stewart et al's insight that the link between IYCF and linear growth is largely context dependent. The WHO recommended practices do not form a natural chain of positive events in urban informal settlements. Those who adhere to optimal breastfeeding guidelines are less likely to switch to optimal complementary feeding. While the benefit of predominant breastfeeding, after accounting for its indirect effect through complementary feeding, for linear growth in Mumbai's deprived communities is not apparent, the positive effect of consumption of animal source foods in the complementary feeding period is clearer.

I also provide evidence of the usefulness of the potential outcomes framework for thinking about the first 1000 days, and offer mechanistic insight in the context of urban poverty. This area of research could be further strengthened by borrowing methods and approaches to mechanistic understanding from life course epidemiology, and improved methods for longitudinal IYCF measurement in cohort studies.

Chapter 9 Conclusion

I begin this chapter with a summary of the key findings of my research and state my thesis in response to the main research question. In subsequent sections, I discuss some additional insights offered by my research and outline recommendations for future empirical and methodologic research.

9.1 Summary of findings and overarching thesis

There were five main research findings.

1. My literature review identified 77 articles that analysed linear growth in infancy in 35 unique ways, although 29% used only one data point to produce a summary of growth, despite the availability of an average 7.2 measurements. The determinants of growth examined in these studies were spread across 18 categories of infant, parental, and household or environmental factors. Recent narrative reviews on the determinants of linear growth had divergent framings of the factors that most strongly influence growth.
2. The 978 children (52% female) in the SNEHA Centres Infant Nutrition Cohort were followed up for a total duration of 20 042 child-months, with an average duration of 26.2 months between March 2013 and April 2016. The relationships between factors describing baseline parental and socioeconomic position showed that younger parents were more likely to have greater educational attainment, to live in households with secure water and sanitation, fewer children and more adults, and were less likely to smoke. By two years of age, 38% of the cohort were lost to follow-up. Analysis of missing data and value patterns in analytic samples suggested that non-response was unlikely to lead to biased estimates.
3. My analysis of the determinants of linear growth using the SITAR model showed that socioeconomic position factors most indicative of urban informality (material deprivation, inadequate sanitation, and household overcrowding) were related to lower length velocity and delayed age at peak velocity, and lower attained length. These factors appeared to affect growth velocity in the second year of life. Children from households that used a shared toilet facility grew 11% slower than those who had a toilet at home. Parental heights, rather than weights, were associated with higher velocity,

and children with taller parents attained greater length at the end of follow-up.

4. Adherence to exclusive (26%) and predominant (44%) breastfeeding practices for the first six months was low. Complementary feeding in the first year was characterised by diets that were infrequently diverse (<20%) and early establishment of snacking behaviour (88% at 12 months), although consumption of animal source foods was common (36% consumed two or more types at 12 months). Complementary feeding practices were correlated over time. Higher maternal education was associated with greater hazard of discontinuing exclusive (42%) or predominant (78%) breastfeeding before six months, and 35% lower odds of consumption of snack foods at 6-23 months. Children of fathers who smoked were 38% less likely to have diverse diets. In households that had access to piped water, children were 2.3 times more likely to regularly consume animal source foods in the complementary feeding period.
5. Predominant breastfeeding and consumption of animal source foods were also negatively related. Predominant breastfeeding had a negative association with attained length at 24 months, while consumption of animal source foods had a positive association. In causal mediation analysis, children who were predominantly breastfed at 0-5 months had 1.1cm lower attained length at 24 months; 70% (0.77 cm) of this effect was direct, transmitted through pathways that did not involve consumption of animal source foods between 6 and 23 months.

In response to the overarching research question posed in Chapter 1, my thesis can be stated as follows:

> Linear growth in infancy and early childhood is negatively affected by adverse socioeconomic conditions that index urban informality. The infant and young child feeding continuum is also socioeconomically determined, but the inverse relationship between adequate breastfeeding and complementary feeding highlights that stage-specific factors operate to shape children's earliest feeding experiences in Mumbai's informal settlements. Favourable socioeconomic position at birth, and the higher quality complementary feeding it facilitates, have positive relationships

with linear growth, but this pattern is interrupted by suboptimal breastfeeding between birth and six months.

9.2 Implications of empirical findings

Arguably, my thesis is an example of circular epidemiology (Kuller, 1999), 'the endless repetition of the well known' (Davey Smith, 2013), echoing prosaic reflections on child nutrition in informal settlements that have been made for decades about Mumbai (Cutting and Kothari, 1988) and centuries elsewhere. In a broad sense, such criticism would be fair. In defence, however, having used a longitudinal study design and an overall analytic approach that emphasized theoretical operationalization, the thesis offers *nuanced* reflection. This relates, perhaps unsurprisingly, to WASH and IYCF in the context of urban informality.

9.2.1 Water to eat, toilets to grow: a bit more meat, not too slow?

My findings allow me to draw a distinction between the relationships of water and toilets with IYCF and linear growth, and hint at slightly different pathways that link them to nutrition and growth in the cohort. Access to piped water was the strongest predictor of regular consumption of animal source foods, but had little influence on linear growth. This suggests that water facilitates better diets when other SEP conditions allow secure household food access, rather than solely offering protection against infection as a way to promote growth. Water is essential for cooking and other domestic activities (Subbaraman et al., 2015), and items that signal dietary diversity (such as meat, vegetables, fish, lentils) also need more water for washing and preparation. Secure access to water means that families are able to eat better and feed their children better.

Using a shared toilet was associated with lower length velocity, but not with any IYCF practice. Its main influence on linear growth in this setting, adjusted for other SEP markers, is likely to operate through the infection and environmental enteric dysfunction pathway, compounded by the fact that it is a shared environmental exposure.

WASH interventions often club the two components together, but I would argue that it is worth examining their relative contributions to nutrition and growth.

9.2.2 The synthetic curvature of the IYCF continuum

Breastfed infants are often shorter than those who are mixed-fed in LMIC contexts (Eriksen et al., 2017), and predominant breastfeeding did not lead to greater attained length at 24 months in the cohort. The IYCF continuum takes idealised form in the WHO recommendation to exclusively breastfeed from birth to six months and introduce a nutritious diet thereafter to ensure optimal growth (WHO, 2008a). In the absence of adequate stage-specific support in this setting, children receive one rather than the other, and this is largely determined by the socioeconomic circumstances they are born into. While women of lower SEP are more likely to predominantly breastfeed, they probably do it because non-breastmilk items that lead to departures from optimal practice are out of reach. The absence of these items a few months later makes it difficult for the child to continue along the optimal IYCF trajectory in the complementary feeding period; their diet reflects the family diet.

If the first stage of IYCF is achieved in conditions of lower SEP (by force of circumstance rather than choice), and the second in conditions of higher SEP (either by choice or circumstance), few infants will ever have a feeding curve that corresponds to WHO guidance unless families transition (rapidly) to better living conditions just as the infant begins to eat solids, but not before, as this would possibly raise the risk of sub-optimal breastfeeding.

Such a scenario is exaggerated, but it highlights the inherent trap in treating the IYCF continuum as a smooth curve that can be understood and shifted in a monolithic interventional sweep. Depending on the strategy, the intervention could also exaggerate growth differentials by SEP. A health promotion-based intervention would not affect the poorest women, who are also likely to have shorter infants, since they have no choice but to breastfeed exclusively and cannot afford good complementary feeding. It would leave their IYCF practices and growth curves untouched, while possibly encouraging better breastfeeding and even better complementary feeding among those of higher SEP, whose children would then attain greater length.

A focus on improving IYCF and growth in the poorest groups in these communities would probably offer the greatest population benefit, as the larger increases in growth rates of the poorest would offset any stagnation in less poor groups, leading

to greater average length (Blum, 2013). Perhaps such a change would be best achieved by improving the living conditions and SEP status of the poorest families, while supporting and encouraging them to continue exclusive breastfeeding because of all the other benefits it confers.

9.3 Recommendations for future work

9.3.1 Empirical research

I recommend further research on four specific topics.

First, the trend in snack food consumption so early in life is worrying from a nutritional perspective. However, I believe that any action to 'solve the problem' must wait until there is more detailed understanding of the pathways from urban poverty to snacking and we have identified corresponding intervention approaches that could be tested. Qualitative research would work well to generate a range of plausible explanations.

Second, we need to examine how relationships between SEP, nutrition, and growth are altered when families move from informal settlements to formal social housing. What protection does an apartment block offer, and what other challenges does it bring? A discordant-sibling analysis or case-control study would be well suited to such questions.

Third, we need to think about how to support women in urban informal settlements to exclusively breastfeed. Are their needs specific to the conditions they live in? Is an interpersonal approach such as breastfeeding counselling suitable? Recent WHO guidelines on counselling of women to improve breastfeeding practices (WHO, 2018b) did not mention the challenges of breastfeeding or breastfeeding counselling that are particular to cities or informal settlements. This reflects either the lack of empirical evidence on the topic, the absence of a suitable conceptual approach in existing studies, or both.

Fourth, children's nutrition was associated with characteristics of other family members, reflecting the social relationships that exert an influence on how children learn to eat and grow. Fathers are largely absent from the breastfeeding narrative and interventions in LMICs (Tadesse et al., 2018), and few interventions think about the role that older siblings play in introducing young children to snacks. We need

more empirical evidence on what role other family members play in child nutrition, and how they can share some of the responsibility for feeding and caregiving which public health and society place squarely and solely on the often overburdened shoulders of the mother.

9.3.2 Methodologic research

I recommend methodologic development in four specific domains.

First, we need tailored reporting guidelines and a critical appraisal tool for observational longitudinal growth studies, which lay out the most critical aspects of transparency and quality that are emblematic of well-conducted research. As a starting point, the STROBE checklist could be adapted to include aspects of growth modelling, and the framework of metrics review by Leung et al. (2018) could be incorporated into an appraisal tool.

Second, we need better indicators to measure IYCF longitudinally such that its psychosocial and appetitive dimensions can be studied using simple tools. This needs to be coupled with new analytic methods to investigate IYCF that borrow from life course epidemiology or another quantitative discipline that incorporates elements of time-dependent processes.

Third, the study of urban nutrition and poverty would benefit from drawing on concepts used in urban studies, and combining them with methods in environmental epidemiology and the study of neighbourhood effects on health. Nutrition in informal settlements will be understood better if we can situate communities within their urban context and tackle questions with methods that can incorporate such ideas. An added advantage would be that casual inference methods are already being developed to study neighbourhood effects on health (Diez Roux, 2004, Diez Roux, 2019).

Fourth, on a related note, I strongly recommend the expansion of nutritional epidemiology and associated auxological research to incorporate the formalisms of counterfactual thinking. A healthy dose of causal inference would bring clarity and rigour to the global health and development discourse on child nutrition, though it does come with the substantial challenge of incorporating and measuring all sources of confounding in an epidemiologic study. Recent concerns about the misuse of

indicators (Frongillo et al., 2019, Perumal et al., 2018) and misleading claims about ‘causal’ relationships between cognitive development, linear growth, and nutrition (Leroy and Frongillo, 2019) attest to this. Further, advances in causal inference and a unifying counterfactual framework for epidemiology provide an opportunity to improve the quality of evidence in health-related quantitative disciplines (Hernán et al., 2019). We must offer observational data the chance to speak causally.

9.3.3 Policy and programmatic action

Observational research offers an opportunity to identify avenues for action, strengthen current efforts and programmes, and plug gaps in policy. The strength of my research findings lies in the nuanced analysis of nutrition and growth in the first two years of life of children in Mumbai’s informal settlements. The work of local non-governmental organizations, municipal bodies, and public health agencies would benefit from the data presented here.

However, the relevance of my study for specific policies or programmes needs to be interpreted in a more participatory way, and any recommendations I make here would be cursory. For the purpose of SNEHA’s work on child nutrition in particular and urban health in general, my findings must be complemented by their teams’ comprehensive knowledge and wide-ranging experience of running public health programmes in urban informal settlements and the reality of the health and nutrition policy landscape in Mumbai. I hope to guide their health and nutrition teams through my research findings and the literature it is embedded in, and arrive at its policy and programmatic relevance, if any, through a collaborative discussion.

References

AARTS, C., KYLBERG, E., HORNELL, A., HOFVANDER, Y., GEBRE-MEDHIN, M. & GREINER, T. 2000. How exclusive is exclusive breastfeeding? A comparison of data since birth with current status data. *Int J Epidemiol,* 29**,** 1041-6.

ACC/SCN 2000. Fourth Report on the World Nutrition Situation. Geneva: ACC/SCN in collaboration with IFPRI.

ADDO, O. Y., STEIN, A. D., FALL, C. H., GIGANTE, D. P., GUNTUPALLI, A. M., HORTA, B. L., . . . MARTORELL, R. 2013. Maternal height and child growth patterns. *Journal of Pediatrics,* 163**,** 549-554.e1.

ADMASSU, B., RITZ, C., WELLS, J. C. K., GIRMA, T., ANDERSEN, G. S., BELACHEW, T., . . . KÆSTEL, P. 2018. Accretion of fat-free mass rather than fat mass in infancy is positively associated with linear growth in childhood. *Journal of Nutrition,* 148**,** 607-615.

AGAMPODI, S. B., AGAMPODI, T. C. & DE SILVA, A. 2009. Exclusive breastfeeding in Sri Lanka: problems of interpretation of reported rates. *Int Breastfeed J,* 4**,** 14.

AGARWAL, S. & TANEJA, S. 2005. All slums are not equal: child health conditions among the urban poor. *Indian Pediatr,* 42**,** 233-44.

AGOSTONI, C., DECSI, T., FEWTRELL, M., GOULET, O., KOLACEK, S., KOLETZKO, B., . . . VAN GOUDOEVER, J. 2008. Complementary feeding: a commentary by the ESPGHAN Committee on Nutrition. *J Pediatr Gastroenterol Nutr,* 46**,** 99-110.

AGUAYO, V. M. 2017. Complementary feeding practices for infants and young children in South Asia. A review of evidence for action post-2015. *Maternal & Child Nutrition,* 13.

AKOMBI, B. J., AGHO, K. E., HALL, J. J., WALI, N., RENZAHO, A. M. N. & MEROM, D. 2017. Stunting, wasting and underweight in Sub-Saharan Africa: A systematic review. *International Journal of Environmental Research and Public Health,* 14.

ALKHALAWI, E., KASPER-SONNENBERG, M., WILHELM, M., VÖLKEL, W. & WITTSIEPE, J. 2016. Perfluoroalkyl acids (PFAAs) and anthropometric measures in the first year of life: Results from the Duisburg Birth Cohort. *Journal of Toxicology and Environmental Health - Part A: Current Issues,* 79**,** 1041-1049.

ALTMAN, D. G. & ROYSTON, P. 2006. The cost of dichotomising continuous variables. *Bmj,* 332**,** 1080.

AMORIM, L. D. & CAI, J. 2015. Modelling recurrent events: a tutorial for analysis in epidemiology. *Int J Epidemiol,* 44**,** 324-33.

ANDERSEN, C. S., FEI, C., GAMBORG, M., NOHR, E. A., SØRENSEN, T. I. A. & OLSEN, J. 2010. Prenatal exposures to perfluorinated chemicals and anthropometric measures in infancy. *American Journal of Epidemiology,* 172**,** 1230-1237.

ANZMAN-FRASCA, S., VENTURA, A. K., EHRENBERG, S. & MYERS, K. P. 2017. Promoting healthy food preferences from the start: a narrative review of food preference learning from the prenatal period through early childhood. *Obesity Reviews,* 19**,** 576-604.

AUSTIN, P. C., LEE, D. S. & FINE, J. P. 2016. Introduction to the Analysis of Survival Data in the Presence of Competing Risks. *Circulation,* 133**,** 601-9.

BARRETT, G., SMITH, S. C. & WELLINGS, K. 2004. Conceptualisation, development, and evaluation of a measure of unplanned pregnancy. *J Epidemiol Community Health,* 58**,** 426-33.

BEAL, T., TUMILOWICZ, A., SUTRISNA, A., IZWARDY, D. & NEUFELD, L. M. 2018. A review of child stunting determinants in Indonesia. *Matern Child Nutr,* 14**,** e12617.

BEATH, K. J. 2007. Infant growth modelling using a shape invariant model with random effects. *Statistics in Medicine,* 26**,** 2547-2564.

BEAUCHAMP, G. K. & MENNELLA, J. A. 2009. Early flavor learning and its impact on later feeding behavior. *J Pediatr Gastroenterol Nutr,* 48 Suppl 1**,** S25-30.

BÉGIN, F. & AGUAYO, V. M. 2017. First foods: Why improving young children's diets matter. *Maternal & Child Nutrition,* 13**,** n/a-n/a.

BELL, J. A., CARSLAKE, D., O'KEEFFE, L. M., FRYSZ, M., HOWE, L. D., HAMER, M., . . . DAVEY SMITH, G. 2018. Associations of Body Mass and Fat Indexes With Cardiometabolic Traits. *J Am Coll Cardiol,* 72**,** 3142-3154.

BELL, K. A., WAGNER, C. L., FELDMAN, H. A., SHYPAILO, R. J. & BELFORT, M. B. 2017. Associations of infant feeding with trajectories of body composition and growth. *American Journal of Clinical Nutrition,* 106**,** 491-498.

BENTLEY, A., DAS, S., ALCOCK, G., SHAH MORE, N., PANTVAIDYA, S. & OSRIN, D. 2015. Malnutrition and infant and young child feeding in informal settlements in Mumbai, India: findings from a census. *Food Science & Nutrition,* 3**,** 257-271.

BERNAL, J. L., CUMMINS, S. & GASPARRINI, A. 2017. Interrupted time series regression for the evaluation of public health interventions: a tutorial. *Int J Epidemiol,* 46**,** 348-355.

BETOKO, A., CHARLES, M. A., HANKARD, R., FORHAN, A., BONET, M., REGNAULT, N., . . . DE LAUZON-GUILLAIN, B. 2014. Determinants of infant formula use and relation with growth in the first 4 months. *Maternal and Child Nutrition,* 10**,** 267-279.

BHAN, N., KARAN, A., SRIVASTAVA, S., SELVARAJ, S., SUBRAMANIAN, S. V. & MILLETT, C. 2016. Have Socioeconomic Inequalities in Tobacco Use in India Increased Over Time? Trends From the National Sample Surveys (2000-2012). *Nicotine Tob Res,* 18**,** 1711-8.

BHARGAVA, A. 2016. Protein and micronutrient intakes are associated with child growth and morbidity from infancy to adulthood in the philippines. *Journal of Nutrition,* 146**,** 133-141.

BHASKARAN, K., GASPARRINI, A., HAJAT, S., SMEETH, L. & ARMSTRONG, B. 2013. Time series regression studies in environmental epidemiology. *Int J Epidemiol,* 42**,** 1187-95.

BHUTTA, Z. A., DAS, J. K., RIZVI, A., GAFFEY, M. F., WALKER, N., HORTON, S., . . . GROUP, T. M. A. C. N. S. 2013. Evidence-based interventions for improvement of maternal and child nutrition: what can be done and at what cost? *The Lancet,* 382**,** 452-477.

BIJLSMA, M. J., TARKIAINEN, L., MYRSKYLA, M. & MARTIKAINEN, P. 2017. Unemployment and subsequent depression: A mediation analysis using the parametric G-formula. *Soc Sci Med,* 194**,** 142-150.

BINNS, C., LEE, M. K. & KAGAWA, M. 2017. Ethical Challenges in Infant Feeding Research. *Nutrients,* 9.

BLACK, R. E., ALLEN, L. H., BHUTTA, Z. A., CAULFIELD, L. E., DE ONIS, M., EZZATI, M., . . . RIVERA, J. 2008. Maternal and child undernutrition: global and regional exposures and health consequences. *The Lancet,* 371**,** 243-260.

BLACK, R. E., VICTORA, C. G., WALKER, S. P., BHUTTA, Z. A., CHRISTIAN, P., DE ONIS, M., . . . AND THE MATERNAL AND CHILD NUTRITION STUDY GROUP 2013. Maternal and child undernutrition and overweight in low-income and middle-income countries. *Lancet,* 382**,** 427-451.

BLUM, M. 2013. The influence of inequality on the standard of living: worldwide anthropometric evidence from the 19th and 20th centuries. *Econ Hum Biol,* 11**,** 436-52.

BOGIN, B. & VARELA-SILVA, M. I. 2010. Leg Length, Body Proportion, and Health: A Review with a Note on Beauty. *International Journal of Environmental Research and Public Health,* 7**,** 1047-1075.

BORK, K., CAMES, C., BARIGOU, S., COURNIL, A. & DIALLO, A. 2012. A summary index of feeding practices is positively associated with height-for-

age, but only marginally with linear growth, in rural senegalese infants and toddlers. *Journal of Nutrition,* 142**,** 1116-1122.

BORK, K. A. & DIALLO, A. 2017. Boys are more stunted than girls from early infancy to 3 years of age in rural senegal. *Journal of Nutrition,* 147**,** 940-947.

BOYD, A., GOLDING, J., MACLEOD, J., LAWLOR, D. A., FRASER, A., HENDERSON, J., . . . DAVEY SMITH, G. 2013. Cohort Profile: the 'children of the 90s'--the index offspring of the Avon Longitudinal Study of Parents and Children. *Int J Epidemiol,* 42**,** 111-27.

BRANCA, F., PIWOZ, E., SCHULTINK, W. & SULLIVAN, L. M. 2015. Nutrition and health in women, children, and adolescent girls. *BMJ,* 351**,** h4173.

BRAUN, K. V., ERLER, N. S., KIEFTE-DE JONG, J. C., JADDOE, V. W., VAN DEN HOOVEN, E. H., FRANCO, O. H. & VOORTMAN, T. 2016. Dietary Intake of Protein in Early Childhood Is Associated with Growth Trajectories between 1 and 9 Years of Age. *J Nutr,* 146**,** 2361-2367.

BRION, M. J., LAWLOR, D. A., MATIJASEVICH, A., HORTA, B., ANSELMI, L., ARAUJO, C. L., . . . SMITH, G. D. 2011. What are the causal effects of breastfeeding on IQ, obesity and blood pressure? Evidence from comparing high-income with middle-income cohorts. *Int J Epidemiol,* 40**,** 670-80.

BROERE-BROWN, Z. A., BAAN, E., SCHALEKAMP-TIMMERMANS, S., VERBURG, B. O., JADDOE, V. W. V. & STEEGERS, E. A. P. 2016. Sex-specific differences in fetal and infant growth patterns: A prospective population-based cohort study. *Biology of Sex Differences,* 7.

BROWN, K. H., DEWEY, K. G. & ALLEN, L. H. 1998a. Complementary Feeding of Young Children in Developing Countries: a Review of Current Scientific Knowledge. Geneva: WHO.

BROWN, K. H., DEWEY, K. G. & ALLEN, L. H. 1998b. Complementary Feeding of Young Children in Developing Countries: a Review of Current Scientific Knowledge. Geneva: WHO.

BUCHMANN, C. & HANNUM, E. 2001. Education and Stratification in Developing Countries: A Review of Theories and Research. *Annual Review of Sociology,* 27**,** 77-102.

BUDREE, S., GODDARD, E., BRITTAIN, K., CADER, S., MYER, L. & ZAR, H. J. 2017. Infant feeding practices in a South African birth cohort—A longitudinal study. *Maternal & Child Nutrition,* 13**,** e12371-n/a.

BUSERT, L. K., NEUMAN, M., REHFUESS, E. A., DULAL, S., HARTHAN, J., CHAUBE, S. S., . . . SAVILLE, N. M. 2016. Dietary diversity is positively associated with deviation from expected height in rural nepal. *Journal of Nutrition,* 146**,** 1387-1393.

CALEYACHETTY, A., KRISHNAVENI, G. V., VEENA, S. R., HILL, J., KARAT, S. C., FALL, C. H. & WILLS, A. K. 2013. Breastfeeding duration, age of starting solids and high BMI risk and adiposity in Indian children. *Matern Child Nutr,* 9**,** 199-216.

CAMERON, N. 2004. Measuring growth. *In:* HAUSPIE, R. C., CAMERON, N. & MOLINARI, L. (eds.) *Methods in Human Growth Research.* Cambridge: Cambridge University Press.

CASTILLO, H., SANTOS, I. S. & MATIJASEVICH, A. 2016. Maternal pre-pregnancy BMI, gestational weight gain and breastfeeding. *Eur J Clin Nutr,* 70**,** 431-6.

CHALMERS, I. & GLASZIOU, P. 2009. Avoidable waste in the production and reporting of research evidence. *Lancet,* 374**,** 86-9.

CHANDRAMOULI, C. 2011. Housing stock, amenities and assets in slums - Census 2011. Available from www.censusindia.gov.in. New Delhi: Office of the Registrar General and Census Commissioner,.

CHENG, T. S., KWOK, M. K., LEUNG, G. M. & SCHOOLING, C. M. 2018. The Associations of Breast Feeding with Infant Growth and Body Mass Index to 16 years: 'Children of 1997'. *Paediatric and Perinatal Epidemiology,* 32**,** 200-209.

CHOLA, L., FADNES, L. T., ENGEBRETSEN, I. M., TUMWINE, J. K., TYLLESKAR, T. & ROBBERSTAD, B. 2013. Infant feeding survival and Markov transition probabilities among children under age 6 months in Uganda. *Am J Epidemiol,* 177**,** 453-62.

CHRISTIAN, P., LEE, S. E., ANGEL, M. D., ADAIR, L. S., ARIFEEN, S. E., ASHORN, P., . . . BLACK, R. E. 2013. Risk of childhood undernutrition related to small-for-gestational age and preterm birth in low- and middle-income countries. *International Journal of Epidemiology,* 42**,** 1340-1355.

CLARKE, P. & HARDY, R. 2007. Methods for handling missing data. *In:* PICKLES, A., MAUGHAN, B. & WADSWORTH, M. (eds.) *Epidmiological Methods in Life Course Research.* Oxford: OUP.

CLEMENTE, D. B. P., CASAS, M., JANSSEN, B. G., LERTXUNDI, A., SANTA-MARINA, L., IÑIGUEZ, C., . . . VRIJHEID, M. 2017. Prenatal ambient air pollution exposure, infant growth and placental mitochondrial DNA content in the INMA birth cohort. *Environmental Research,* 157**,** 96-102.

COLE, T. J. 2000. Secular trends in growth. *Proc Nutr Soc,* 59**,** 317-24.

COLE, T. J. 2003. The secular trend in human physical growth: a biological view. *Econ Hum Biol,* 1**,** 161-8.

COLE, T. J., CORTINA-BORJA, M., SANDHU, J., KELLY, F. P. & PAN, H. 2008. Nonlinear growth generates age changes in the moments of the frequency distribution: the example of height in puberty. *Biostatistics,* 9**,** 159-71.

COLE, T. J., DONALDSON, M. D. & BEN-SHLOMO, Y. 2010. SITAR--a useful instrument for growth curve analysis. *Int J Epidemiol,* 39**,** 1558-66.

CONTRERAS, M., BLANDON, E. Z., PERSSON, L. A., HJERN, A. & EKSTROM, E. C. 2015. Socio-economic resources, young child feeding practices, consumption of highly processed snacks and sugar-sweetened beverages: a population-based survey in rural northwestern Nicaragua. *BMC Public Health,* 15**,** 25.

CORNELSEN, L., ALARCON, P., HÄSLER, B., AMENDAH, D. D., FERGUSON, E., FÈVRE, E. M., . . . RUSHTON, J. 2016. Cross-sectional study of drivers of animal-source food consumption in low-income urban areas of Nairobi, Kenya. *BMC Nutrition,* 2**,** 70.

COSTET, N., PELÉ, F., COMETS, E., ROUGET, F., MONFORT, C., BODEAU-LIVINEC, F., . . . CORDIER, S. 2015. Perinatal exposure to chlordecone and infant growth. *Environmental Research,* 142**,** 123-134.

COULTHARD, H., HARRIS, G. & EMMETT, P. 2009. Delayed introduction of lumpy foods to children during the complementary feeding period affects child's food acceptance and feeding at 7 years of age. *Matern Child Nutr,* 5**,** 75-85.

CUNNINGHAM, K., RUEL, M., FERGUSON, E. & UAUY, R. 2015. Women's empowerment and child nutritional status in South Asia: a synthesis of the literature. *Maternal & Child Nutrition,* 11**,** 1-19.

CUTTING, W. A. & KOTHARI, G. 1988. Children in Third World slums. *Br Med J (Clin Res Ed),* 296**,** 1683-4.

CZERWINSKI, S. A. & TOWNE, B. 2004. Methods for the study of the genetics of growth and development. *In:* HAUSPIE, R. C., CAMERON, N. & MOLINARI, L. (eds.) *Methods in Human Growth Research.* Cambridge: Cambridge University Press.

DANAEI, G., ANDREWS, K. G., SUDFELD, C. R., FINK, G., MCCOY, D. C., PEET, E., . . . FAWZI, W. W. 2016. Risk Factors for Childhood Stunting in 137 Developing Countries: A Comparative Risk Assessment Analysis at Global, Regional, and Country Levels. *PLoS Medicine,* 13.

DANIEL, R. M., DE STAVOLA, B. & COUSENS, S. 2011. gformula: Estimating causal effects in the presence of time-varying confounding or mediation using the g-computation formula. *The Stata Journal,* 11**,** 479-517.

DANIEL, R. M. & DE STAVOLA, B. L. 2019. Mediation analysis for life course studies. *In:* PLOUBIDIS, G. B., PONGIGLIONE, B., DE STAVOLA, B. L.,

DANIEL, R. M., BENOVA, L., GRUNDY, E. & REED, S. (eds.) *Pathways to Health.* The Netherlands: Springer.

DANIEL, R. M., DE STAVOLA, B. L. & VANSTEELANDT, S. 2016. Commentary: The formal approach to quantitative causal inference in epidemiology: misguided or misrepresented? *Int J Epidemiol,* 45**,** 1817-1829.

DANIEL, R. M., KENWARD, M. G., COUSENS, S. N. & DE STAVOLA, B. L. 2012. Using causal diagrams to guide analysis in missing data problems. *Stat Methods Med Res,* 21**,** 243-56.

DAPA MEASUREMENT TOOLKIT. 2018. Dietary assessment decision matrix. Available: https://www.measurement-toolkit.mrc.ac.uk/diet-individual-analysis-decision-matrix [Accessed 1 April 2018].

DAS, A., CHATTERJEE, R., KARTHICK, M., MAHAPATRA, T. & CHAUDHURI, I. 2016. The Influence of Seasonality and Community-Based Health Worker Provided Counselling on Exclusive Breastfeeding - Findings from a Cross-Sectional Survey in India. *PLoS One,* 11**,** e0161186.

DAS, S., BAPAT, U., MORE, N. S., ALCOCK, G., FERNANDEZ, A. & OSRIN, D. 2012. Nutritional status of young children in Mumbai slums: a follow-up anthropometric study. *Nutr J,* 11**,** 100.

DAVEY SMITH, G. 2012. Negative control exposures in epidemiologic studies. *Epidemiology,* 23**,** 350-1; author reply 351-2.

DAVEY SMITH, G. 2013. If data could talk back: Anzia Yezierska, Paul de Kruif and thousands of pages of 'research'. *Int J Epidemiol,* 42**,** 1-6.

DE BEAUDRAP, P., TURYAKIRA, E., NABASUMBA, C., TUMWEBAZE, B., PIOLA, P., BOUM II, Y. & MCGREADY, R. 2016. Timing of malaria in pregnancy and impact on infant growth and morbidity: A cohort study in Uganda. *Malaria Journal,* 15.

DE BEER, H. 2012. Dairy products and physical stature: a systematic review and meta-analysis of controlled trials. *Econ Hum Biol,* 10**,** 299-309.

DE BEER, M., VRIJKOTTE, T. G., FALL, C. H., VAN EIJSDEN, M., OSMOND, C. & GEMKE, R. J. 2016. Associations of Infant Feeding and Timing of Weight Gain and Linear Growth during Early Life with Childhood Blood Pressure: Findings from a Prospective Population Based Cohort Study. *PLoS One,* 11**,** e0166281.

DE BEER, M., VRIJKOTTE, T. G., VAN DER WAL, M. F., VAN WEISSENBRUCH, M. M. & GEMKE, R. J. 2010. Relation of maternal hypertension with infant growth in a prospective birth cohort: the ABCD study. *J Dev Orig Health Dis,* 1**,** 347-55.

DE BRUYN, J., BAGNOL, B., DARNTON-HILL, I., MAULAGA, W., THOMSON, P. C. & ALDERS, R. 2017. Characterising infant and young child feeding practices and the consumption of poultry products in rural Tanzania: A mixed methods approach. *Matern Child Nutr*.

DE HOOG, M. L. A., VAN EIJSDEN, M., STRONKS, K., GEMKE, R. J. B. J. & VRIJKOTTE, T. G. M. 2011. The role of infant feeding practices in the explanation for ethnic differences in infant growth: The Amsterdam Born Children and their Development study. *British Journal of Nutrition,* 106**,** 1592-1601.

DE ONIS, M., ONYANGO, A. W., VAN DEN BROECK, J., CHUMLEA, C. W. & MARTORELL, R. 2004. Measurement and Standardization Protocols for Anthropometry Used in the Construction of a New International Growth Reference. *Food and Nutrition Bulletin,* 25**,** S27-S36.

DE STAVOLA, B. L. & DANIEL, R. M. 2016. Commentary: Incorporating concepts and methods from causal inference into life course epidemiology. *International Journal of Epidemiology,* 45**,** 1006-1010.

DE STAVOLA, B. L., DANIEL, R. M., PLOUBIDIS, G. B. & MICALI, N. 2015. Mediation Analysis With Intermediate Confounding: Structural Equation Modeling Viewed Through the Causal Inference Lens. *American Journal of Epidemiology,* 181**,** 64-80.

DE STAVOLA, B. L., NITSCH, D., DOS SANTOS SILVA, I., MCCORMACK, V., HARDY, R., MANN, V., . . . LEON, D. A. 2006. Statistical issues in life course epidemiology. *Am J Epidemiol,* 163**,** 84-96.

DEATON, A. 2007. Height, health, and development. *Proc Natl Acad Sci U S A,* 104**,** 13232-7.

DEBES, A. K., KOHLI, A., WALKER, N., EDMOND, K. & MULLANY, L. C. 2013. Time to initiation of breastfeeding and neonatal mortality and morbidity: a systematic review. *BMC Public Health,* 13 Suppl 3**,** S19.

DEIERLEIN, A. L., SIEGA-RIZ, A. M., ADAIR, L. S. & HERRING, A. H. 2011. Effects of pre-pregnancy body mass index and gestational weight gain on infant anthropometric outcomes. *Journal of Pediatrics,* 158**,** 221-226.

DEMILEW, Y. M., TAFERE, T. E. & ABITEW, D. B. 2017. Infant and young child feeding practice among mothers with 0-24 months old children in Slum areas of Bahir Dar City, Ethiopia. *Int Breastfeed J,* 12**,** 26.

DEVAKUMAR, D., KULAR, D., SHRESTHA, B. P., GRIJALVA-ETERNOD, C., DANIEL, R. M., SAVILLE, N. M., . . . WELLS, J. C. K. 2018. Socioeconomic determinants of growth in a longitudinal study in Nepal. *Maternal and Child Nutrition,* 14.

DEWEY, K. G. 2013. The challenge of meeting nutrient needs of infants and young children during the period of complementary feeding: an evolutionary perspective. *J Nutr,* 143**,** 2050-4.

DIEZ ROUX, A. V. 2004. Estimating neighborhood health effects: the challenges of causal inference in a complex world. *Soc Sci Med,* 58**,** 1953-60.

DIEZ ROUX, A. V. 2019. The Unique Space of Epidemiology: Drawing on the Past to Project Into the Future. *Am J Epidemiol,* 188**,** 886-889.

DOMINGUEZ-SALAS, P., ALARCÓN, P., HÄSLER, B., DOHOO, I. R., COLVERSON, K., KIMANI-MURAGE, E. W., . . . GRACE, D. 2016. Nutritional characterisation of low-income households of Nairobi: socioeconomic, livestock and gender considerations and predictors of malnutrition from a cross-sectional survey. *BMC Nutrition,* 2**,** 47.

DOVEY, K. 2015. Sustainable Informal Settlements? *Procedia - Social and Behavioral Sciences,* 179**,** 5-13.

DOVEY, K. & KING, R. 2011. Forms of informality: morphology and visibility of informal settlements. *Built Environment,* 37**,** 11-29.

DOYAL, L. & PENNELL, I. 1979. *The Political Economy of Health*, Pluto Press.

DURMUS, B., ARENDS, L. R., AY, L., HOKKEN-KOELEGA, A. C., RAAT, H., HOFMAN, A., . . . JADDOE, V. W. V. 2013. Parental anthropometrics, early growth and the risk of overweight in pre-school children: The Generation R Study. *Pediatric Obesity,* 8**,** 339-350.

DURMUŞ, B., KRUITHOF, C. J., GILLMAN, M. H., WILLEMSEN, S. P., HOFMAN, A., RAAT, H., . . . JADDOE, V. W. V. 2011. Parental smoking during pregnancy, early growth, and risk of obesity in preschool children: The Generation R Study. *American Journal of Clinical Nutrition,* 94**,** 164-171.

EMMETT, P. 2009. Assessing diet in longitudinal birth cohort studies. *Paediatric and Perinatal Epidemiology,* 23**,** 154-173.

ENGLE, P., LHOTSKA, L. & ARMSTRONG, H. 1997a. The care initiative: guidelines for analysis, assessment, and action to improve nutrition. *New York: UNICEF*.

ENGLE, P. L., MENON, P., GARRETT, J. L. & SLACK, A. 1997b. Urbanization and caregiving: a framework for analysis and examples from southern and eastern Africa. *Environment and Urbanization,* 9**,** 253-270.

ERIKSEN, K. G., JOHNSON, W., SONKO, B., PRENTICE, A. M., DARBOE, M. K. & MOORE, S. E. 2017. Following the World Health Organization's Recommendation of Exclusive Breastfeeding to 6 Months of Age Does Not Impact the Growth of Rural Gambian Infants. *The Journal of Nutrition,* 147**,** 248-255.

ERTEL, K. A., KOENEN, K. C., RICH-EDWARDS, J. W. & GILLMAN, M. W. 2010. Maternal depressive symptoms not associated with reduced height in young children in a US prospective cohort study. *PLoS ONE,* 5.

EZEH, A., OYEBODE, O., SATTERTHWAITE, D., CHEN, Y.-F., NDUGWA, R., SARTORI, J., . . . LILFORD, R. J. 2017. The history, geography, and sociology of slums and the health problems of people who live in slums. *The Lancet,* 389**,** 547-558.

FAIRLEY, L., PETHERICK, E. S., HOWE, L. D., TILLING, K., CAMERON, N., LAWLOR, D. A., . . . WRIGHT, J. 2013. Describing differences in weight and length growth trajectories between white and Pakistani infants in the UK: Analysis of the Born in Bradford birth cohort study using multilevel linear spline models. *Archives of Disease in Childhood,* 98**,** 274-279.

FALKINGHAM, J. & NAMAZIE, C. 2002. Measuring health and poverty: a review of approaches to identifying the poor London: DFID Health Systems Resource Centre.

FALL, C. H., BORJA, J. B., OSMOND, C., RICHTER, L., BHARGAVA, S. K., MARTORELL, R., . . . VICTORA, C. G. 2011. Infant-feeding patterns and cardiovascular risk factors in young adulthood: data from five cohorts in low- and middle-income countries. *Int J Epidemiol,* 40**,** 47-62.

FERNANDEZ, N. A., BURGOS, J. C., ROBERTS, L. J. & ASENJO, C. F. 1968. Nutritional status in Puerto Rican slum area. *Am J Clin Nutr,* 21**,** 646-56.

FIERSTEIN, J. L., ELIASZIW, M., ROGERS, B. L. & FORRESTER, J. E. 2017. Nonnative Cattle Ownership, Diet, and Child Height-for-Age: Evidence from the 2011 Uganda Demographic and Health Survey. *Am J Trop Med Hyg,* 96**,** 74-82.

FILMER, D. & PRITCHETT, L. H. 2001. Estimating wealth effects without expenditure data--or tears: an application to educational enrollments in states of India. *Demography,* 38**,** 115-32.

FISHER, M. F. K. 1943. *The Gastronomical Me,* London, Daunt.

FLORESCU, A., FERRENCE, R., EINARSON, T., SELBY, P., SOLDIN, O. & KOREN, G. 2009. Methods for quantification of exposure to cigarette smoking and environmental tobacco smoke: focus on developmental toxicology. *Ther Drug Monit,* 31**,** 14-30.

FRASER, A., MACDONALD-WALLIS, C., TILLING, K., BOYD, A., GOLDING, J., DAVEY SMITH, G., . . . LAWLOR, D. A. 2013. Cohort Profile: the Avon Longitudinal Study of Parents and Children: ALSPAC mothers cohort. *Int J Epidemiol,* 42**,** 97-110.

FRONGILLO, E. A., LEROY, J. L. & LAPPING, K. 2019. Appropriate Use of Linear Growth Measures to Assess Impact of Interventions on Child Development and Catch-Up Growth. *Adv Nutr*.

FUEMMELER, B. F., WANG, L., IVERSEN, E. S., MAGUIRE, R., MURPHY, S. K. & HOYO, C. 2016. Association between Prepregnancy Body Mass Index and Gestational Weight Gain with Size, Tempo, and Velocity of Infant Growth: Analysis of the Newborn Epigenetic Study Cohort. *Child Obes,* 12**,** 210-8.

FULFORD, A. J., RAYCO-SOLON, P. & PRENTICE, A. M. 2006. Statistical modelling of the seasonality of preterm delivery and intrauterine growth restriction in rural Gambia. *Paediatr Perinat Epidemiol,* 20**,** 251-9.

FULLER, J. A., VILLAMOR, E., CEVALLOS, W., TROSTLE, J. & EISENBERG, J. N. S. 2016. I get height with a little help from my friends: herd protection from sanitation on child growth in rural Ecuador. *International Journal of Epidemiology,* 45**,** 460-469.

GALEA, S., FREUDENBERG, N. & VLAHOV, D. 2005. Cities and population health. *Social Science & Medicine,* 60**,** 1017-1033.

GALOBARDES, B., SHAW, M., LAWLOR, D. A., LYNCH, J. W. & DAVEY SMITH, G. 2006a. Indicators of socioeconomic position (part 1). *J Epidemiol Community Health,* 60**,** 7-12.

GALOBARDES, B., SHAW, M., LAWLOR, D. A., LYNCH, J. W. & DAVEY SMITH, G. 2006b. Indicators of socioeconomic position (part 2). *J Epidemiol Community Health,* 60**,** 95-101.

GARCED, S., TORRES-SÁNCHEZ, L., CEBRIÁN, M. E., CLAUDIO, L. & LÓPEZ-CARRILLO, L. 2012. Prenatal dichlorodiphenyldichloroethylene (DDE) exposure and child growth during the first year of life. *Environmental Research,* 113**,** 58-62.

GARZA, C., BORGHI, E., ONYANGO, A. W. & DE ONIS, M. 2013. Parental height and child growth from birth to 2 years in the WHO multicentre growth reference study. *Maternal and Child Nutrition,* 9**,** 58-68.

GARZÓN, M., PEREIRA-DA-SILVA, L., SEIXAS, J., PAPOILA, A. L. & ALVES, M. 2018. Subclinical enteric parasitic infections and growth faltering in infants in São Tomé, Africa: A birth cohort study. *International Journal of Environmental Research and Public Health,* 15.

GASSER, T., GERVINI, D. & MOLINARI, L. 2004. Kernal estimation, shape-invariant modelling and structural analysis. *In:* HAUSPIE, R. C., CAMERON, N. & MOLINARI, L. (eds.) *Methods in Human Growth Research.* Cambridge: Cambridge University Press.

GIUGLIANI, E. R., HORTA, B. L., LORET DE MOLA, C., LISBOA, B. O. & VICTORA, C. G. 2015. Effect of breastfeeding promotion interventions on

child growth: a systematic review and meta-analysis. *Acta Paediatr,* 104**,** 20-9.

GLASS, T. A., GOODMAN, S. N., HERNÁN, M. A. & SAMET, J. M. 2013. Causal Inference in Public Health. *Annual review of public health,* 34**,** 61-75.

GOLDING, J. 2009. Measuring outcomes in a longitudinal birth cohort. *Paediatr Perinat Epidemiol,* 23 Suppl 1**,** 185-200.

GOLDING, J. & BIRMINGHAM, K. 2009. Enrollment and response rates in a longitudinal birth cohort. *Paediatr Perinat Epidemiol,* 23 Suppl 1**,** 73-85.

GOLDING, J., JONES, R., PREECE, A., BRUNE, M. N. & PRONCZUK, J. 2009. Choice of environmental components for a longitudinal birth cohort study. *Paediatr Perinat Epidemiol,* 23 Suppl 1**,** 134-53.

GOLDING, J. & STEER, C. 2009. How many subjects are needed in a longitudinal birth cohort study? *Paediatric and Perinatal Epidemiology,* 23**,** 31-38.

GONZALEZ-CHICA, D. A., GONCALVES, H., NAZMI, A., SANTOS, I. S., BARROS, A. J., MATIJASEVICH, A. & VICTORA, C. G. 2012. Seasonality of infant feeding practices in three Brazilian birth cohorts. *Int J Epidemiol,* 41**,** 743-52.

GOUDET, S. M., GRIFFITHS, P. L., BOGIN, B. A. & SELIM, N. 2011. Impact of flooding on feeding practices of infants and young children in Dhaka, Bangladesh Slums: what are the coping strategies? *Matern Child Nutr,* 7**,** 198-214.

GOUGH, E. K., MOODIE, E. E. M., PRENDERGAST, A. J., NTOZINI, R., MOULTON, L. H., HUMPHREY, J. H. & MANGES, A. R. 2016. Linear growth trajectories in Zimbabwean infants. *American Journal of Clinical Nutrition,* 104**,** 1616-1627.

GREENLAND, S., DANIEL, R. & PEARCE, N. 2016a. Outcome modelling strategies in epidemiology: traditional methods and basic alternatives. *International Journal of Epidemiology,* 45**,** 565-575.

GREENLAND, S., MANSOURNIA, M. A. & ALTMAN, D. G. 2016b. Sparse data bias: a problem hiding in plain sight. *BMJ,* 352.

GREENLAND, S. & PEARCE, N. 2015. Statistical foundations for model-based adjustments. *Annu Rev Public Health,* 36**,** 89-108.

GREENLAND, S. & ROTHMAN, K. J. 2008. Measures of occurence. *In:* ROTHMAN, K. J., GREENLAND, S. & LASH, T. L. (eds.) *Modern Epidemiology.* Philadelphia: Lippincott Williams & Wilkins.

GREENLAND, S., ROTHMAN, K. J. & LASH, T. L. 2008. Measures of effect and measures of association. *In:* ROTHMAN, K. J., GREENLAND, S. & LASH, T. L. (eds.) *Modern Epidemiology.* Philadelphia: Lippincott Williams & Wilkins.

GREINER, T. 2014. Exclusive breastfeeding: measurement and indicators. *Int Breastfeed J,* 9**,** 18.

GRIFFITHS, L. J., DEZATEUX, C. & COLE, T. J. 2007. Differential parental weight and height contributions to offspring birthweight and weight gain in infancy. *International Journal of Epidemiology,* 36**,** 104-107.

GRIFFITHS, P. L., BALAKRISHNA, N., FERNANDEZ RAO, S. & JOHNSON, W. 2016. Do socio-economic inequalities in infant growth in rural India operate through maternal size and birth weight? *Annals of Human Biology,* 43**,** 154-163.

GUNDERSON, E. P. 2009. Childbearing and obesity in women: weight before, during, and after pregnancy. *Obstet Gynecol Clin North Am,* 36**,** 317-32, ix.

GUNDERSON, E. P. & ABRAMS, B. 2000. Epidemiology of gestational weight gain and body weight changes after pregnancy. *Epidemiol Rev,* 22**,** 261-74.

GUPTA, P. C. & RAY, C. S. 2003. Smokeless tobacco and health in India and South Asia. *Respirology,* 8**,** 419-31.

GUPTA, V., DOWNS, S. M., GHOSH-JERATH, S., LOCK, K. & SINGH, A. 2016. Unhealthy Fat in Street and Snack Foods in Low-Socioeconomic Settings in India: A Case Study of the Food Environments of Rural Villages and an Urban Slum. *Journal of Nutrition Education and Behavior,* 48**,** 269-279.e1.

HALL, J. A., BARRETT, G., COPAS, A. & STEPHENSON, J. 2017a. London Measure of Unplanned Pregnancy: guidance for its use as an outcome measure. *Patient Relat Outcome Meas,* 8**,** 43-56.

HALL, J. A., BENTON, L., COPAS, A. & STEPHENSON, J. 2017b. Pregnancy Intention and Pregnancy Outcome: Systematic Review and Meta-Analysis. *Matern Child Health J,* 21**,** 670-704.

HALLQVIST, J., LYNCH, J., BARTLEY, M., LANG, T. & BLANE, D. 2004. Can we disentangle life course processes of accumulation, critical period and social mobility? An analysis of disadvantaged socio-economic positions and myocardial infarction in the Stockholm Heart Epidemiology Program. *Social Science & Medicine,* 58**,** 1555-1562.

HAMBIDGE, K. M., MAZARIEGOS, M., KINDEM, M., WRIGHT, L. L., CRISTOBAL-PEREZ, C., JUÁREZ-GARCÍA, L., . . . KREBS, N. F. 2012. Infant stunting is associated with short maternal stature. *Journal of Pediatric Gastroenterology and Nutrition,* 54**,** 117-119.

HANIEH, S., HA, T. T., DE LIVERA, A. M., SIMPSON, J. A., THUY, T. T., KHUONG, N. C., . . . BIGGS, B. A. 2015. Antenatal and early infant predictors of postnatal growth in rural Vietnam: A prospective cohort study. *Archives of Disease in Childhood,* 100**,** 165-173.

HAUSPIE, R. C. & MOLINARI, L. 2004. Parametric models for postnatal growth. *In:* HAUSPIE, R. C., CAMERON, N. & MOLINARI, L. (eds.) *Methods in Human Growth Research.* Cambridge: Cambridge University Press.

HAUSPIE, R. C., WACHHOLDER, A., BARON, G., CANTRAINE, F., SUSANNE, C. & GRAFFAR, M. 1980. A comparative study of the fit of four different functions to longitudinal data of growth in height of Belgian girls. *Annals of Human Biology,* 7**,** 347-358.

HERMANUSSEN, M., STEC, K., ASSMANN, C., MEIGEN, C. & VAN BUUREN, S. 2016. Synthetic growth reference charts. *Am J Hum Biol,* 28**,** 98-111.

HERMANUSSEN, M. & WIT, J. M. 2017. How Much Nutrition for How Much Growth? *Horm Res Paediatr,* 88**,** 38-45.

HERNAN, M. A. 2016. Does water kill? A call for less casual causal inferences. *Ann Epidemiol,* 26**,** 674-680.

HERNAN, M. A., HERNANDEZ-DIAZ, S. & ROBINS, J. M. 2004. A structural approach to selection bias. *Epidemiology,* 15**,** 615-25.

HERNÁN, M. A., HSU, J. & HEALY, B. 2019. A Second Chance to Get Causal Inference Right: A Classification of Data Science Tasks. *CHANCE,* 32**,** 42-49.

HERNAN, M. A. & TAUBMAN, S. L. 2008. Does obesity shorten life? The importance of well-defined interventions to answer causal questions. *Int J Obes (Lond),* 32 Suppl 3**,** S8-14.

HILL, A. B. 1965. THE ENVIRONMENT AND DISEASE: ASSOCIATION OR CAUSATION? *Proc R Soc Med,* 58**,** 295-300.

HOLLIS, J. L., CROZIER, S. R., INSKIP, H. M., COOPER, C., GODFREY, K. M., HARVEY, N. C., . . . ROBINSON, S. M. 2017. Modifiable risk factors of maternal postpartum weight retention: an analysis of their combined impact and potential opportunities for prevention. *Int J Obes (Lond),* 41**,** 1091-1098.

HONG, J., LEE, H. A., PARK, E. A., KIM, Y. J., LEE, H., PARK, B. H., . . . PARK, H. 2014. Association of mid-pregnancy antioxidative vitamin and oxidative stress levels with infant growth during the first 3 years of life. *Food and Nutrition Research,* 58.

HOPPE, C., MOLGAARD, C. & MICHAELSEN, K. F. 2006. Cow's milk and linear growth in industrialized and developing countries. *Annu Rev Nutr,* 26**,** 131-73.

HOWE, C. J., COLE, S. R., LAU, B., NAPRAVNIK, S. & ERON, J. J., JR. 2016a. Selection Bias Due to Loss to Follow Up in Cohort Studies. *Epidemiology,* 27**,** 91-7.

HOWE, L. D., GALOBARDES, B., MATIJASEVICH, A., GORDON, D., JOHNSTON, D., ONWUJEKWE, O., . . . HARGREAVES, J. R. 2012a. Measuring socio-economic position for epidemiological studies in low- and middle-income countries: a methods of measurement in epidemiology paper. *Int J Epidemiol,* 41**,** 871-86.

HOWE, L. D., HARGREAVES, J. R., PLOUBIDIS, G. B., DE STAVOLA, B. L. & HUTTLY, S. R. 2011. Subjective measures of socio-economic position and the wealth index: a comparative analysis. *Health Policy Plan,* 26**,** 223-32.

HOWE, L. D., TILLING, K., GALOBARDES, B., SMITH, G. D., GUNNELL, D. & LAWLOR, D. A. 2012b. Socioeconomic differences in childhood growth trajectories: at what age do height inequalities emerge? *J Epidemiol Community Health,* 66**,** 143-8.

HOWE, L. D., TILLING, K., MATIJASEVICH, A., PETHERICK, E. S., SANTOS, A. C., FAIRLEY, L., . . . LAWLOR, D. A. 2016b. Linear spline multilevel models for summarising childhood growth trajectories: A guide to their application using examples from five birth cohorts. *Stat Methods Med Res,* 25**,** 1854-1874.

HUFFMAN, S. L., PIWOZ, E. G., VOSTI, S. A. & DEWEY, K. G. 2014. Babies, soft drinks and snacks: a concern in low- and middle-income countries? *Matern Child Nutr,* 10**,** 562-574.

HUMPHREY, J. H., MBUYA, M. N. N., NTOZINI, R., MOULTON, L. H., STOLTZFUS, R. J., TAVENGWA, N. V., . . . PRENDERGAST, A. J. 2019. Independent and combined effects of improved water, sanitation, and hygiene, and improved complementary feeding, on child stunting and anaemia in rural Zimbabwe: a cluster-randomised trial. *Lancet Glob Health,* 7**,** e132-e147.

HUSAIN, N., CRUICKSHANK, J. K., TOMENSON, B., KHAN, S. & RAHMAN, A. 2012. Maternal depression and infant growth and development in British Pakistani women: a cohort study. *BMJ Open,* 2**,** e000523.

HUSSEINI, M., DARBOE, M. K., MOORE, S. E., NABWERA, H. M. & PRENTICE, A. M. 2018. Thresholds of socio-economic and environmental conditions necessary to escape from childhood malnutrition: a natural experiment in rural Gambia. *BMC Med,* 16**,** 199.

IANNOTTI, L. L., LUTTER, C. K., STEWART, C. P., GALLEGOS RIOFRIO, C. A., MALO, C., REINHART, G., . . . WATERS, W. F. 2017. Eggs in Early Complementary Feeding and Child Growth: A Randomized Controlled Trial. *Pediatrics,* 140.

ISLAM, M. M., SANIN, K. I., MAHFUZ, M., AHMED, A. M. S., MONDAL, D., HAQUE, R. & AHMED, T. 2018. Risk factors of stunting among children living in an urban slum of Bangladesh: Findings of a prospective cohort study. *BMC Public Health,* 18.

JACKSON, J. W., VANDERWEELE, T. J., BLACKER, D. & SCHNEEWEISS, S. 2015. Mediators of First- Versus Second-generation Antipsychotic-related Mortality in Older Adults. *Epidemiology,* 26**,** 700-9.

JAGANATH, D., SAITO, M., GILMAN, R. H., QUEIROZ, D. M. M., ROCHA, G. A., CAMA, V., . . . CHECKLEY, W. 2014. First Detected Helicobacter pylori Infection in Infancy Modifies the Association Between Diarrheal Disease and Childhood Growth in Peru. *Helicobacter,* 19**,** 272-279.

JAYACHANDRAN, S. & KUZIEMKO, I. 2011. Why Do Mothers Breastfeed Girls Less than Boys? Evidence and Implications for Child Health in India. *The Quarterly Journal of Economics,* 126**,** 1485-1538.

JOHNSON, L., LLEWELLYN, C. H., VAN JAARSVELD, C. H., COLE, T. J. & WARDLE, J. 2011. Genetic and environmental influences on infant growth: prospective analysis of the Gemini twin birth cohort. *PLoS One,* 6**,** e19918.

JOHNSON, L., VAN JAARSVELD, C. H., LLEWELLYN, C. H., COLE, T. J. & WARDLE, J. 2014. Associations between infant feeding and the size, tempo and velocity of infant weight gain: SITAR analysis of the Gemini twin birth cohort. *Int J Obes (Lond),* 38**,** 980-7.

JOHNSON, W. 2015. Analytical strategies in human growth research. *Am J Hum Biol,* 27**,** 69-83.

JOHNSON, W., BALAKRISHNA, N. & GRIFFITHS, P. L. 2013. Modeling physical growth using mixed effects models. *Am J Phys Anthropol,* 150**,** 58-67.

JOHNSON, W., VAZIR, S., FERNANDEZ-RAO, S., KANKIPATI, V. R., BALAKRISHNA, N. & GRIFFITHS, P. L. 2012. Using the WHO 2006 child growth standard to assess the growth and nutritional status of rural south Indian infants. *Ann Hum Biol,* 39**,** 91-101.

JONES-SMITH, J. C., NEUFELD, L. M., LARAIA, B., RAMAKRISHNAN, U., GARCIA-GUERRA, A. & FERNALD, L. C. 2013. Early life growth trajectories and future risk for overweight. *Nutr Diabetes,* 3**,** e60.

JONSSON, U. 1981. The causes of hunger. *Food and Nutrition Bulletin,* 3**,** 1-9.

JONSSON, U. 1996. Nutrition and the convention on the rights of the child. *Food Policy,* 21**,** 41-55.

JOSHI, N., AGHO, K. E., DIBLEY, M. J., SENARATH, U. & TIWARI, K. 2012. Determinants of inappropriate complementary feeding practices in young

children in Nepal: secondary data analysis of Demographic and Health Survey 2006. *Matern Child Nutr,* 8 Suppl 1**,** 45-59.

JUDGE, T. A. & CABLE, D. M. 2004. The effect of physical height on workplace success and income: preliminary test of a theoretical model. *J Appl Psychol,* 89**,** 428-41.

KABIR, A. & MAITROT, M. R. 2017. Factors influencing feeding practices of extreme poor infants and young children in families of working mothers in Dhaka slums: A qualitative study. *PLoS One,* 12**,** e0172119.

KANG SIM, D. E., CAPPIELLO, M., CASTILLO, M., LOZOFF, B., MARTINEZ, S., BLANCO, E. & GAHAGAN, S. 2012. Postnatal Growth Patterns in a Chilean Cohort: The Role of SES and Family Environment. *Int J Pediatr,* 2012**,** 354060.

KATTULA, D., SARKAR, R., SIVARATHINASWAMY, P., VELUSAMY, V., VENUGOPAL, S., NAUMOVA, E. N., . . . KANG, G. 2014. The first 1000 days of life: prenatal and postnatal risk factors for morbidity and growth in a birth cohort in southern India. *BMJ Open,* 4**,** e005404.

KAVLE, J. A., FLAX, V. L., ABDELMEGEID, A., SALAH, F., HAFEZ, S., RAMZY, M., . . . GALLOWAY, R. 2016. Factors associated with early growth in Egyptian infants: Implications for addressing the dual burden of malnutrition. *Maternal and Child Nutrition,* 12**,** 139-151.

KAVLE, J. A., MEHANNA, S., SALEH, G., FOUAD, M. A., RAMZY, M., HAMED, D., . . . GALLOWAY, R. 2015. Exploring why junk foods are 'essential' foods and how culturally tailored recommendations improved feeding in Egyptian children. *Matern Child Nutr,* 11**,** 346-70.

KEYS, A., BROZEK, J., HENSCHEL, A., MICHELSEN, O., TAYLOR, H. L., SIMONSON, E., . . . WELLS, S. 1950. *The biology of human starvation. Volumes 1 and 2,* Minnesapolis, University of Minnesota Press.

KHANAL, V., LEE, A. H., SCOTT, J. A., KARKEE, R. & BINNS, C. W. 2016. Implications of methodological differences in measuring the rates of exclusive breastfeeding in Nepal: findings from literature review and cohort study. *BMC Pregnancy Childbirth,* 16**,** 389.

KIMANI-MURAGE, E. W., MADISE, N. J., FOTSO, J. C., KYOBUTUNGI, C., MUTUA, M. K., GITAU, T. M. & YATICH, N. 2011. Patterns and determinants of breastfeeding and complementary feeding practices in urban informal settlements, Nairobi Kenya. *BMC Public Health,* 11**,** 396.

KIMANI-MURAGE, E. W., MUTHURI, S. K., OTI, S. O., MUTUA, M. K., VAN DE VIJVER, S. & KYOBUTUNGI, C. 2015a. Evidence of a Double Burden of Malnutrition in Urban Poor Settings in Nairobi, Kenya. *PLoS One,* 10**,** e0129943.

KIMANI-MURAGE, E. W., WEKESAH, F., WANJOHI, M., KYOBUTUNGI, C., EZEH, A. C., MUSOKE, R. N., . . . GRIFFITHS, P. 2015b. Factors affecting actualisation of the WHO breastfeeding recommendations in urban poor settings in Kenya. *Matern Child Nutr,* 11**,** 314-32.

KIRKWOOD, B. R. & STERNE, J. A. 2009. *Essential Medical Statistics,* Oxford, Blackwell.

KLEINBAUM, D. G., MORGENSTERN, H. & KUPPER, L. L. 1981. Selection bias in epidemiologic studies. *Am J Epidemiol,* 113**,** 452-63.

KNOL, M. J. & VANDERWEELE, T. J. 2012. Recommendations for presenting analyses of effect modification and interaction. *Int J Epidemiol,* 41**,** 514-20.

KOHLI, D., SLIUZAS, R., KERLE, N. & STEIN, A. 2012. An ontology of slums for image-based classification. *Computers, Environment and Urban Systems,* 36**,** 154-163.

KOZUKI, N., KATZ, J., LEE, A. C., VOGEL, J. P., SILVEIRA, M. F., SANIA, A., . . . BLACK, R. E. 2015. Short Maternal Stature Increases Risk of Small-for-Gestational-Age and Preterm Births in Low- and Middle-Income Countries: Individual Participant Data Meta-Analysis and Population Attributable Fraction. *J Nutr,* 145**,** 2542-50.

KRAMER, K. L., VEILE, A. & OTÁROLA-CASTILLO, E. 2016. Sibling competition & growth tradeoffs. Biological vs. statistical significance. *PLoS ONE,* 11.

KRAMER, M. S., DAVIES, N., OKEN, E., MARTIN, R. M., DAHHOU, M., ZHANG, X. & YANG, S. 2018. Infant feeding and growth: putting the horse before the cart. *Am J Clin Nutr,* 107**,** 635-639.

KRASEVEC, J., AN, X., KUMAPLEY, R., BÉGIN, F. & FRONGILLO, E. A. 2017. Diet quality and risk of stunting among infants and young children in low- and middle-income countries. *Maternal & Child Nutrition,* 13**,** e:12430.

KRIEGER, N., WILLIAMS, D. R. & MOSS, N. E. 1997. Measuring social class in US public health research: concepts, methodologies, and guidelines. *Annu Rev Public Health,* 18**,** 341-78.

KRISTJANSSON, E., FRANCIS, D. K., LIBERATO, S., BENKHALTI JANDU, M., WELCH, V., BATAL, M., . . . PETTICREW, M. 2015. Food supplementation for improving the physical and psychosocial health of socio-economically disadvantaged children aged three months to five years. *Cochrane Database Syst Rev***,** Cd009924.

KRISTMAN, V., MANNO, M. & COTE, P. 2004. Loss to follow-up in cohort studies: how much is too much? *Eur J Epidemiol,* 19**,** 751-60.

KUH, D., BEN-SHLOMO, Y., LYNCH, J., HALLQVIST, J. & POWER, C. 2003. Life course epidemiology. *Journal of Epidemiology and Community Health,* 57**,** 778-783.

KULLER, L. H. 1999. Circular epidemiology. *Am J Epidemiol,* 150**,** 897-903.

KWOK, M. K., LEUNG, G. M., LAM, T. H., LEUNG, S. S. L. & SCHOOLING, C. M. 2013. Grandparental education, parental education and child height: Evidence from Hong Kong's "Children of 1997" birth cohort. *Annals of Epidemiology,* 23**,** 475-484.

LABEAUD, A. D., NAYAKWADI SINGER, M., MCKIBBEN, M., MUNGAI, P., MUCHIRI, E. M., MCKIBBEN, E., . . . MALHOTRA, I. 2015. Parasitism in Children Aged Three Years and Under: Relationship between Infection and Growth in Rural Coastal Kenya. *PLoS Negl Trop Dis,* 9**,** e0003721.

LAIRD, N. M. 1988. Missing data in longitudinal studies. *Statistics in Medicine,* 7**,** 305-315.

LAMPL, M. 2012. Perspectives on modelling human growth: Mathematical models and growth biology. *Annals of Human Biology,* 39**,** 342-351.

LAMPL, M. & MUMMERT, A. 2014. Historical approaches to human growth studies limit the present understanding of growth biology. *Annals of Nutrition and Metabolism,* 65**,** 114-120.

LAMPL, M. & SCHOEN, M. 2017. How long bones grow children: Mechanistic paths to variation in human height growth. *American Journal of Human Biology,* 29**,** n/a-n/a.

LANGO ALLEN, H., ESTRADA, K., LETTRE, G., BERNDT, S. I., WEEDON, M. N., RIVADENEIRA, F., . . . HIRSCHHORN, J. N. 2010. Hundreds of variants clustered in genomic loci and biological pathways affect human height. *Nature,* 467**,** 832-8.

LEE, G., PAN, W., PEÑATARO YORI, P., PAREDES OLORTEGUI, M., TILLEY, D., GREGORY, M., . . . KOSEK, M. 2013. Symptomatic and Asymptomatic Campylobacter Infections Associated with Reduced Growth in Peruvian Children. *PLoS Neglected Tropical Diseases,* 7.

LEROY, J. L. & FRONGILLO, E. A. 2019. Perspective: What Does Stunting Really Mean? A Critical Review of the Evidence. *Adv Nutr,* 10**,** 196-204.

LEROY, J. L., RUEL, M., HABICHT, J. P. & FRONGILLO, E. A. 2015. Using height-for-age differences (HAD) instead of height-for-age z-scores (HAZ) for the meaningful measurement of population-level catch-up in linear growth in children less than 5 years of age. *BMC Pediatr,* 15**,** 145.

LEUNG, M., PERUMAL, N., MESFIN, E., KRISHNA, A., YANG, S., JOHNSON, W., . . . ROTH, D. E. 2018. Metrics of early childhood growth in recent epidemiological research: A scoping review. *PLoS ONE,* 13.

LILFORD, R. J., OYEBODE, O., SATTERTHWAITE, D., MELENDEZ-TORRES, G. J., CHEN, Y.-F., MBERU, B., . . . EZEH, A. 2017. Improving the health and welfare of people who live in slums. *The Lancet,* 389**,** 559-570.

LIMA, A. A. M., SOARES, A. M., FILHO, J. Q. S., HAVT, A., LIMA, I. F. N., LIMA, N. L., . . . GUERRANT, R. L. 2018. Enteroaggregative Escherichia coli Subclinical Infection and Coinfections and Impaired Child Growth in the MAL-ED Cohort Study. *Journal of Pediatric Gastroenterology and Nutrition,* 66**,** 325-333.

LIN, S. H., YOUNG, J., LOGAN, R., TCHETGEN TCHETGEN, E. J. & VANDERWEELE, T. J. 2017. Parametric Mediational g-Formula Approach to Mediation Analysis with Time-varying Exposures, Mediators, and Confounders. *Epidemiology,* 28**,** 266-274.

LOK, K. Y. W., WANG, M. P., CHAN, V. H. S. & TARRANT, M. 2018. Effect of Secondary Cigarette Smoke from Household Members on Breastfeeding Duration: A Prospective Cohort Study. *Breastfeed Med,* 13**,** 412-417.

LOURENÇO, B. H., VILLAMOR, E., AUGUSTO, R. A. & CARDOSO, M. A. 2012. Determinants of linear growth from infancy to school-aged years: A population-based follow-up study in urban Amazonian children. *BMC Public Health,* 12.

LUNDEEN, E. A., STEIN, A. D., ADAIR, L. S., BEHRMAN, J. R., BHARGAVA, S. K., DEARDEN, K. A., . . . VICTORA, C. G. 2014. Height-for-age z scores increase despite increasing height deficits among children in 5 developing countries. *Am J Clin Nutr,* 100**,** 821-5.

MACHARIA, T. N., OCHOLA, S., MUTUA, M. K. & KIMANI-MURAGE, E. W. 2018. Association between household food security and infant feeding practices in urban informal settlements in Nairobi, Kenya. *J Dev Orig Health Dis***,** 1-10.

MACIEL, B., MORAES, M. L., SOARES, A. M., CRUZ, I., DE ANDRADE, M., FILHO, J. Q., . . . LIMA, A. 2018. Infant feeding practices and determinant variables for early complementary feeding in the first 8 months of life: results from the Brazilian MAL-ED cohort site. *Public Health Nutr,* 21**,** 2462-2470.

MAL-ED NETWORK INVESTIGATORS 2017. Relationship between growth and illness, enteropathogens and dietary intakes in the first 2 years of life: findings from the MAL-ED birth cohort study. *BMJ Glob Health,* 2**,** e000370.

MALHOTRA, R., NOHERIA, A., AMIR, O., ACKERSON, L. K. & SUBRAMANIAN, S. V. 2008. Determinants of termination of breastfeeding within the first 2 years of life in India: evidence from the National Family Health Survey-2. *Matern Child Nutr,* 4**,** 181-93.

MALLARD, S. R., HOUGHTON, L. A., FILTEAU, S., MULLEN, A., NIEUWELINK, J., CHISENGA, M., . . . GIBSON, R. S. 2014. Dietary diversity at 6 months of age is associated with subsequent growth and mediates the effect of maternal education on infant growth in Urban Zambia. *Journal of Nutrition,* 144**,** 1818-1825.

MANIKAM, L., PRASAD, A., DHARMARATNAM, A., MOEN, C., ROBINSON, A., LIGHT, A., . . . LAKHANPAUL, M. 2018. Systematic review of infant and young child complementary feeding practices in South Asian families: the India perspective. *Public Health Nutr,* 21**,** 637-654.

MANNAN, M., DOI, S. A. & MAMUN, A. A. 2013. Association between weight gain during pregnancy and postpartum weight retention and obesity: a bias-adjusted meta-analysis. *Nutr Rev,* 71**,** 343-52.

MARMOT, M. 2005. Social determinants of health inequalities. *Lancet,* 365**,** 1099-104.

MARQUIS, G. S., DIAZ, J., BARTOLINI, R., CREED DE KANASHIRO, H. & RASMUSSEN, K. M. 1998. Recognizing the reversible nature of child-feeding decisions: breastfeeding, weaning, and relactation patterns in a shanty town community of Lima, Peru. *Soc Sci Med,* 47**,** 645-56.

MARTIN, R. M., HOLLY, J. M. & GUNNELL, D. 2011. Milk and linear growth: programming of the igf-I axis and implication for health in adulthood. *Nestle Nutr Workshop Ser Pediatr Program,* 67**,** 79-97.

MARTÍNEZ-MESA, J., MENEZES, A. M. B., GONZÁLEZ, D. A., HORTA, B. L., MATIJASEVICH, A., GIGANTE, D. P. & HALLAL, P. C. 2012. Life course association of maternal smoking during pregnancy and offspring's height: Data from the 1993 Pelotas (Brazil) birth cohort. *Journal of Adolescent Health,* 51**,** S53-S57.

MARTORELL, R. 2017. Improved nutrition in the first 1000 days and adult human capital and health. *American Journal of Human Biology,* 29.

MATIJASEVICH, A., BRION, M. J., MENEZES, A. M., BARROS, A. J. D., SANTOS, I. S. & BARROS, F. C. 2011. Maternal smoking during pregnancy and offspring growth in childhood: 1993 and 2004 Pelotas cohort studies. *Archives of Disease in Childhood,* 96**,** 519-525.

MATIJASEVICH, A., HOWE, L. D., TILLING, K., SANTOS, I. S., BARROS, A. J. D. & LAWLOR, D. A. 2012. Maternal education inequalities in height growth rates in early childhood: 2004 Pelotas birth cohort study. *Paediatric and Perinatal Epidemiology,* 26**,** 236-249.

MATOS, S. M. A., AMORIM, L. D., CAMPOS, A. C. P., BARRETO, M. L., RODRIGUES, L. C., MOREJÓN, Y. A., . . . COOPER, P. J. 2017. Growth patterns in early childhood: Better trajectories in Afro-Ecuadorians independent of sex and socioeconomic factors. *Nutrition Research,* 44**,** 51-59.

MCEVOY, B. P. & VISSCHER, P. M. 2009. Genetics of human height. *Econ Hum Biol,* 7**,** 294-306.

MCKOEWN, T. 1976. *The modern rise of population,* London, Edward Arnold.

MENNELLA, J. A., JAGNOW, C. P. & BEAUCHAMP, G. K. 2001. Prenatal and postnatal flavor learning by human infants. *Pediatrics,* 107**,** E88.

MENNELLA, J. A. & TRABULSI, J. C. 2012. Complementary foods and flavor experiences: setting the foundation. *Ann Nutr Metab,* 60 Suppl 2**,** 40-50.

MENON, P., BAMEZAI, A., SUBANDORO, A., AYOYA, M. A. & AGUAYO, V. 2015. Age-appropriate infant and young child feeding practices are associated with child nutrition in India: insights from nationally representative data. *Maternal & Child Nutrition,* 11**,** 73-87.

MENON, P., NGUYEN, P. H., SAHA, K. K., KHALED, A., KENNEDY, A., TRAN, L. M., . . . RAWAT, R. 2016a. Impacts on Breastfeeding Practices of At-Scale Strategies That Combine Intensive Interpersonal Counseling, Mass Media, and Community Mobilization: Results of Cluster-Randomized Program Evaluations in Bangladesh and Viet Nam. *PLoS Med,* 13**,** e1002159.

MENON, P., NGUYEN, P. H., SAHA, K. K., KHALED, A., SANGHVI, T., BAKER, J., . . . RAWAT, R. 2016b. Combining Intensive Counseling by Frontline Workers with a Nationwide Mass Media Campaign Has Large Differential Impacts on Complementary Feeding Practices but Not on Child Growth: Results of a Cluster-Randomized Program Evaluation in Bangladesh. *J Nutr,* 146**,** 2075-2084.

MICHAELSEN, K. F. 2013. Effect of protein intake from 6 to 24 months on insulin-like growth factor 1 (IGF-1) levels, body composition, linear growth velocity, and linear growth acceleration: what are the implications for stunting and wasting? *Food Nutr Bull,* 34**,** 268-71.

MICHAELSEN, K. F., DEWEY, K. G., PEREZ-EXPOSITO, A. B., NURHASAN, M., LAURITZEN, L. & ROOS, N. 2011. Food sources and intake of n-6 and n-3 fatty acids in low-income countries with emphasis on infants, young children (6-24 months), and pregnant and lactating women. *Matern Child Nutr,* 7 Suppl 2**,** 124-40.

MICHAELSEN, K. F., GRUMMER-STRAWN, L. & BÉGIN, F. 2017. Emerging issues in complementary feeding: Global aspects. *Maternal & Child Nutrition,* 13**,** n/a-n/a.

MILLER, B. D. 1997. Social class, gender and intrahousehold food allocations to children in South Asia. *Social Science & Medicine,* 44**,** 1685-1695.

MISHRA, G., NITSCH, D., BLACK, S., DE STAVOLA, B., KUH, D. & HARDY, R. 2009. A structured approach to modelling the effects of binary exposure

variables over the life course. *International Journal of Epidemiology,* 38**,** 528-537.

MISHRA, G. A., KULKARNI, S. V., GUPTA, S. D. & SHASTRI, S. S. 2015. Smokeless tobacco use in Urban Indian women: Prevalence and predictors. *Indian J Med Paediatr Oncol,* 36**,** 176-82.

MISHRA, S., JOSEPH, R. A., GUPTA, P. C., PEZZACK, B., RAM, F., SINHA, D. N., . . . JHA, P. 2016. Trends in bidi and cigarette smoking in India from 1998 to 2015, by age, gender and education. *BMJ Glob Health,* 1**,** e000005.

MOFFAT, T. 2001. A biocultural investigation of the weanling's dilemma in Kathmandu, Nepal: do universal recommendations for weaning practices make sense? *J Biosoc Sci,* 33**,** 321-38.

MOLINARI, L. & GASSER, T. 2004. The human growth curve: distance, velocity and acceleration. *In:* HAUSPIE, R. C., CAMERON, N. & MOLINARI, L. (eds.) *Methods in Human Growth Research.* Cambridge: Cambridge University Press.

MONTERROSA, E. C., PELTO, G. H., FRONGILLO, E. A. & RASMUSSEN, K. M. 2012. Constructing maternal knowledge frameworks. How mothers conceptualize complementary feeding. *Appetite,* 59**,** 377-384.

MOORE, D. A., GOODWIN, T. L., BROCKLEHURST, P. R., ARMITAGE, C. J. & GLENNY, A. M. 2017. When Are Caregivers More Likely to Offer Sugary Drinks and Snacks to Infants? A Qualitative Thematic Synthesis. *Qual Health Res,* 27**,** 74-88.

MOORE, S. R., LIMA, N. L., SOARES, A. M., ORI, R. B., PINKERTON, R. C., BARRETT, L. J., . . . LIMA, A. A. M. 2010. Prolonged episodes of acute diarrhea reduce growth and increase risk of persistent diarrhea in children. *Gastroenterology,* 139**,** 1156-1164.

MORADI, M., MARACY, M. R., ESMAILLZADEH, A., SURKAN, P. J. & AZADBAKHT, L. 2018. Associations Between Dietary Energy Density in Mothers and Growth of Breastfeeding Infants During the First 4 Months of Life. *J Am Coll Nutr***,** 1-7.

MORENO-BETANCUR, M. & CARLIN, J. B. 2018. Understanding Interventional Effects: A More Natural Approach to Mediation Analysis? *Epidemiology,* 29**,** 614-617.

MUMM, R., IPSEN, M. J. & HERMANUSSEN, M. 2016. The association of weight, weight variability and socioeconomic situation among children. *Eur J Clin Nutr,* 70**,** 650-2.

MUNICIPAL CORPORATION OF GREATER MUMBAI 2010. Mumbai Human Development Report 2009. New Delhi: Oxford University Press.

MUNTALIF, B. S., NASTITI, A., ROOSMINI, D., SUDRADJAT, A., MEIJERINK, S. V. & SMITS, A. J. M. 2017. *Household Water Supply Strategies in Urban Bandung, Indonesia: Findings and Implications for Future Water Access Reporting*.

MURASKO, J. E. 2014. Associations between household income, height and BMI in contemporary US children: Infancy through early childhood. *Annals of Human Biology,* 41**,** 488-496.

MYRELID, A., GUSTAFSSON, J., OLLARS, B. & ANNEREN, G. 2002. Growth charts for Down's syndrome from birth to 18 years of age. *Arch Dis Child,* 87**,** 97-103.

NAGATA, J. M., GIPPETTI, J., WAGER, S., CHAVEZ, A. & WISE, P. H. 2016. Prevalence and predictors of malnutrition among guatemalan children at 2 years of age. *PLoS ONE,* 11.

NGURE, F. M., REID, B. M., HUMPHREY, J. H., MBUYA, M. N., PELTO, G. & STOLTZFUS, R. J. 2014. Water, sanitation, and hygiene (WASH), environmental enteropathy, nutrition, and early child development: making the links. *Ann N Y Acad Sci,* 1308**,** 118-28.

NGUYEN, P. H., AVULA, R., HEADEY, D., TRAN, L. M., RUEL, M. T. & MENON, P. 2018. Progress and inequalities in infant and young child feeding practices in India between 2006 and 2016. *Matern Child Nutr,* 14 Suppl 4**,** e12663.

O'CONNOR, R. J. 2012. Non-cigarette tobacco products: what have we learnt and where are we headed? *Tob Control,* 21**,** 181-90.

O'KEEFFE, L. M., KEARNEY, P. M., GREENE, R. A., ZUCCOLO, L., TILLING, K., LAWLOR, D. A. & HOWE, L. D. 2015. Maternal alcohol use during pregnancy and offspring trajectories of height and weight: A prospective cohort study. *Drug and Alcohol Dependence,* 153**,** 323-329.

OSRIN, D., DAS, S., BAPAT, U., ALCOCK, G. A., JOSHI, W. & SHAH MORE, N. 2011. A rapid assessment scorecard to identify informal settlements at higher maternal and child health risk in Mumbai. *J Urban Health,* 88**,** 919-932.

OWAIS, A., SCHWARTZ, B., KLEINBAUM, D. G., SUCHDEV, P. S., FARUQUE, A. S. G., DAS, S. K. & STEIN, A. D. 2016. Minimum acceptable diet at 9 months but not exclusive breastfeeding at 3 months or timely complementary feeding initiation is predictive of infant growth in rural Bangladesh. *PLoS ONE,* 11.

OZALTIN, E., HILL, K. & SUBRAMANIAN, S. V. 2010. Association of maternal stature with offspring mortality, underweight, and stunting in low- to middle-income countries. *Jama,* 303**,** 1507-16.

PADONOU, G., LE PORT, A., COTTRELL, G., GUERRA, J., CHOUDAT, I., RACHAS, A., . . . MARTIN-PREVEL, Y. 2014. Factors associated with growth patterns from birth to 18 months in a Beninese cohort of children. *Acta Tropica,* 135**,** 1-9.

PALWALA, M., SHARMA, S., UDIPI, S. A., GHUGRE, P. S., KOTHARI, G. & SAWARDEKAR, P. 2009. Nutritional quality of diets fed to young children in urban slums can be improved by intensive nutrition education. *Food Nutr Bull,* 30**,** 317-26.

PATEL, A., BANERJEE, A. & KALETWAD, A. 2013. Factors associated with prelacteal feeding and timely initiation of breastfeeding in hospital-delivered infants in India. *J Hum Lact,* 29**,** 572-8.

PATEL, A., PUSDEKAR, Y., BADHONIYA, N., BORKAR, J., AGHO, K. E. & DIBLEY, M. J. 2012. Determinants of inappropriate complementary feeding practices in young children in India: secondary analysis of National Family Health Survey 2005-2006. *Matern Child Nutr,* 8 Suppl 1**,** 28-44.

PATEL, R., TILLING, K., LAWLOR, D. A., HOWE, L. D., BOGDANOVICH, N., MATUSH, L., . . . MARTIN, R. M. 2014. Socioeconomic differences in childhood length/height trajectories in a middle-income country: A cohort study. *BMC Public Health,* 14.

PEARCE, A., SAWYER, A. C. P., CHITTLEBOROUGH, C. R., MITTINTY, M. N., LAW, C. & LYNCH, J. W. 2016. Do early life cognitive ability and self-regulation skills explain socio-economic inequalities in academic achievement? An effect decomposition analysis in UK and Australian cohorts. *Soc Sci Med,* 165**,** 108-118.

PEARL, J. Proceedings of the seventeenth conference on uncertainty in artificial intelligence. Morgan Kaufman, 2001 San Francisco.

PEARL, J. 2010. On the consistency rule in causal inference: axiom, definition, assumption, or theorem? *Epidemiology,* 21**,** 872-5.

PERKINS, J. M., SUBRAMANIAN, S. V., DAVEY SMITH, G. & OZALTIN, E. 2016. Adult height, nutrition, and population health. *Nutr Rev,* 74**,** 149-65.

PERUMAL, N., BASSANI, D. G. & ROTH, D. E. 2018. Use and Misuse of Stunting as a Measure of Child Health. *J Nutr,* 148**,** 311-315.

PETERSON, K. M., BUSS, J., EASLEY, R., YANG, Z., KORPE, P. S., NIU, F., . . . PETRI, W. A. 2013. REG1B as a predictor of childhood stunting in Bangladesh and Peru. *American Journal of Clinical Nutrition,* 97**,** 1129-1133.

PIZZI, C., COLE, T. J., RICHIARDI, L., DOS-SANTOS-SILVA, I., CORVALAN, C. & DE STAVOLA, B. 2014. Prenatal influences on size, velocity and tempo of infant growth: findings from three contemporary cohorts. *PLoS One,* 9**,** e90291.

PIZZI, C., DE STAVOLA, B., MERLETTI, F., BELLOCCO, R., DOS SANTOS SILVA, I., PEARCE, N. & RICHIARDI, L. 2011. Sample selection and validity of exposure–disease association estimates in cohort studies. *Journal of Epidemiology and Community Health,* 65**,** 407-411.

PIZZI, C., DE STAVOLA, B. L., PEARCE, N., LAZZARATO, F., GHIOTTI, P., MERLETTI, F. & RICHIARDI, L. 2012. Selection bias and patterns of confounding in cohort studies: the case of the NINFEA web-based birth cohort. *Journal of Epidemiology and Community Health,* 66**,** 976-981.

PLATTS-MILLS, J. A., LIU, J., ROGAWSKI, E. T., KABIR, F., LERTSETHTAKARN, P., SIGUAS, M., . . . HOUPT, E. R. 2018. Use of quantitative molecular diagnostic methods to assess the aetiology, burden, and clinical characteristics of diarrhoea in children in low-resource settings: a reanalysis of the MAL-ED cohort study. *Lancet Glob Health,* 6**,** e1309-e1318.

POOLE, C. 2010. On the origin of risk relativism. *Epidemiology,* 21**,** 3-9.

POPKIN, B. M., ADAIR, L. S. & NG, S. W. 2011. Global nutrition transition and the pandemic of obesity in developing countries. *Nutrition Reviews,* 70**,** 3-21.

POPOVIC, M., PIZZI, C., RUSCONI, F., GALASSI, C., GAGLIARDI, L., DE MARCO, L., . . . RICHIARDI, L. 2016. Infant weight trajectories and early childhood wheezing: the NINFEA birth cohort study. *Thorax,* 71**,** 1091-1096.

POTDAR, R. D., SAHARIAH, S. A., GANDHI, M., KEHOE, S. H., BROWN, N., SANE, H., . . . FALL, C. H. 2014. Improving women's diet quality preconceptionally and during gestation: effects on birth weight and prevalence of low birth weight--a randomized controlled efficacy trial in India (Mumbai Maternal Nutrition Project). *Am J Clin Nutr,* 100**,** 1257-68.

PRENDERGAST, A. J. & HUMPHREY, J. H. 2014. The stunting syndrome in developing countries. *Paediatrics and International Child Health,* 34**,** 250-265.

PRIES, A. M., HUFFMAN, S. L., ADHIKARY, I., UPRETI, S. R., DHUNGEL, S., CHAMPENY, M. & ZEHNER, E. 2016. High consumption of commercial food products among children less than 24 months of age and product promotion in Kathmandu Valley, Nepal. *Matern Child Nutr,* 12 Suppl 2**,** 22-37.

PRIES, A. M., HUFFMAN, S. L., CHAMPENY, M., ADHIKARY, I., BENJAMIN, M., COLY, A. N., . . . ZEHNER, E. 2017. Consumption of commercially produced snack foods and sugar-sweetened beverages during the complementary feeding period in four African and Asian urban contexts. *Maternal & Child Nutrition,* 13**,** n/a-n/a.

PSAKI, S., BHUTTA, Z. A., AHMED, T., AHMED, S., BESSONG, P., ISLAM, M., . . . CHECKLEY, W. 2012. Household food access and child malnutrition: results from the eight-country MAL-ED study. *Popul Health Metr,* 10**,** 24.

PSAKI, S. R., SEIDMAN, J. C., MILLER, M., GOTTLIEB, M., BHUTTA, Z. A., AHMED, T., . . . CHECKLEY, W. 2014. Measuring socioeconomic status in multicountry studies: results from the eight-country MAL-ED study. *Popul Health Metr,* 12**,** 8.

QUEIROZ, V. A. D. O., ASSIS, A. M. O., PINHEIRO, S. M. C. & RIBEIRO JR, H. D. C. 2012. Predictors of linear growth in the first year of life of a prospective cohort of full term children with normal birth weight. *Jornal de Pediatria,* 88**,** 79-86.

RABE-HESKETH, S. & SKRONDAL, A. 2012. *Multilevel and longitudinal modeling using Stata,* College Station, TX, Stata Press.

RAMANI, S., SHAIKH, N., DAS, S., PANTVAIDYA, S., FERNANDEZ, A. & JAYARAMAN, A. 2019. "Everybody breastfeeds if they have milk": factors that shape exclusive breastfeeding practices in informal settlements of Mumbai, India. *Int Breastfeed J,* 14**,** 10.

RAVINETTO, R. M., AFOLABI, M. O., OKEBE, J., VAN NUIL, J. I., LUTUMBA, P., MAVOKO, H. M., . . . GRIETENS, K. P. 2015. Participation in medical research as a resource-seeking strategy in socio-economically vulnerable communities: call for research and action. *Trop Med Int Health,* 20**,** 63-6.

RICHARD, S. A., BLACK, R. E., GILMAN, R. H., GUERRANT, R. L., KANG, G., LANATA, C. F., . . . CHECKLEY, W. 2014. Catch-up growth occurs after diarrhea in early childhood. *Journal of Nutrition,* 144**,** 965-971.

RICHARD, S. A., BLACK, R. E., GILMAN, R. H., GUERRANT, R. L., KANG, G., LANATA, C. F., . . . CHECKLEY, W. 2013. Diarrhea in early childhood: Short-Term association with weight and long-Term association with length. *American Journal of Epidemiology,* 178**,** 1129-1138.

RICHARD, S. A., BLACK, R. E., GILMAN, R. H., GUERRANT, R. L., KANG, G., LANATA, C. F., . . . VERASTEGUI, H. 2012. Wasting is associated with stunting in early childhood. *Journal of Nutrition,* 142**,** 1291-1296.

RICHTER, L., NORRIS, S., PETTIFOR, J., YACH, D. & CAMERON, N. 2007. Cohort Profile: Mandela's children: the 1990 Birth to Twenty study in South Africa. *Int J Epidemiol,* 36**,** 504-11.

ROBERTSON, D. 2015. Is bigger better, or do the best things really come in small packages? *BBC* [Online]. Available: http://www.bbc.com/future/story/20150928-tall-vs-small-which-is-it-better-to-be [Accessed 1 April 2019].

ROBINS, J. 1986. A new approach to causal inference in mortality studies with a sustained exposure period—application to control of the healthy worker survivor effect. *Mathematical Modelling,* 7**,** 1393-1512.

ROBINS, J. M. & GREENLAND, S. 1992. Identifiability and exchangeability for direct and indirect effects. *Epidemiology,* 3**,** 143-55.

ROCCA, C. H., KRISHNAN, S., BARRETT, G. & WILSON, M. 2010. Measuring pregnancy planning: An assessment of the London Measure of Unplanned Pregnancy among urban, south Indian women. *Demogr Res,* 23**,** 293-334.

ROGAWSKI, E. T., LIU, J., PLATTS-MILLS, J. A., KABIR, F., LERTSETHTAKARN, P., SIGUAS, M., . . . HOUPT, E. R. 2018. Use of quantitative molecular diagnostic methods to investigate the effect of enteropathogen infections on linear growth in children in low-resource settings: longitudinal analysis of results from the MAL-ED cohort study. *Lancet Glob Health,* 6**,** e1319-e1328.

ROGAWSKI, E. T., WESTREICH, D. J., ADAIR, L. S., BECKER-DREPS, S., SANDLER, R. S., SARKAR, R., . . . KANG, G. 2015. Early Life Antibiotic Exposure Is Not Associated with Growth in Young Children of Vellore, India. *Journal of Pediatrics,* 167**,** 1096-1102.

ROLLINS, N. C., BHANDARI, N., HAJEEBHOY, N., HORTON, S., LUTTER, C. K., MARTINES, J. C., . . . VICTORA, C. G. 2016. Why invest, and what it will take to improve breastfeeding practices? *Lancet,* 387**,** 491-504.

ROTH, D. E., KRISHNA, A., LEUNG, M., SHI, J., BASSANI, D. G. & BARROS, A. J. D. 2017. Early childhood linear growth faltering in low-income and middle-income countries as a whole-population condition: analysis of 179 Demographic and Health Surveys from 64 countries (1993-2015). *The Lancet Global Health,* 5**,** e1249-e1257.

ROTHMAN, K. J. & GREENLAND, S. 2008. Cohort studies. *In:* ROTHMAN, K. J., GREENLAND, S. & LASH, T. L. (eds.) *Modern Epidemiology.* Philadelphia: Lippincott Williams & Wilkins.

ROTHMAN, K. J., GREENLAND, S. & LASH, T. L. 2008a. Types of epidemiologic studies. *In:* ROTHMAN, K. J., GREENLAND, S. & LASH, T. L. (eds.) *Modern Epidemiology.* Philadelphia: Lippincott Williams & Wilkins.

ROTHMAN, K. J., GREENLAND, S. & LASH, T. L. 2008b. Validity in Epidemiologic Studies. *In:* ROTHMAN, K. J., GREENLAND, S. & LASH, T. L. (eds.) *Modern Epidemiology.* Philadelphia: Lippincott Williams & Wilkins.

ROWLAND, M. G. 1986. The Gambia and Bangladesh: the seasons and diarrhoea. *Dialogue Diarrhoea***,** 3.

RUBIN, D. B. 1976. Inference and Missing Data. *Biometrika,* 63**,** 581-592.

RUEL, M. T. 2017. Measuring Infant and Young Child Complementary Feeding Practices: Indicators, Current Practice, and Research Gaps. *Nestle Nutr Inst Workshop Ser,* 87**,** 73-87.

SAHA, K. K., ENGSTRÖM, A., HAMADANI, J. D., TOFAIL, F., RASMUSSEN, K. M. & VAHTER, M. 2012. Pre- and postnatal arsenic exposure and body size to 2 years of age: A cohort study in rural Bangladesh. *Environmental Health Perspectives,* 120**,** 1208-1214.

SAHA, K. K., FRONGILLO, E. A., ALAM, D. S., ARIFEEN, S. E., PERSSON, L. Å. & RASMUSSEN, K. M. 2008a. Appropriate infant feeding practices result in better growth of infants and young children in rural Bangladesh. *The American Journal of Clinical Nutrition,* 87**,** 1852-1859.

SAHA, K. K., FRONGILLO, E. A., ALAM, D. S., ARIFEEN, S. E., PERSSON, L. A. K. & RASMUSSEN, K. M. 2008b. Household Food Security Is Associated with Infant Feeding Practices in Rural Bangladesh. *The Journal of Nutrition,* 138**,** 1383-1390.

SAMUEL, T. M., THOMAS, T., BHAT, S. & KURPAD, A. V. 2012. Are infants born in baby-friendly hospitals being exclusively breastfed until 6 months of age? *Eur J Clin Nutr,* 66**,** 459-65.

SANIN, K. I., MUNIRUL ISLAM, M., MAHFUZ, M., SHAMSIR AHMED, A. M., MONDAL, D., HAQUE, R. & AHMED, T. 2018. Micronutrient adequacy is poor, but not associated with stunting between 12-24 months of age: A cohort study findings from a slum area of Bangladesh. *PLoS ONE,* 13.

SCHNEE, A. E., HAQUE, R., TANIUCHI, M., UDDIN, J., ALAM, M., LIU, J., . . . PLATTS-MILLS, J. A. 2018. Identification of etiology-specific diarrhea associated with linear growth faltering in Bangladeshi infants. *Am J Epidemiol*.

SEAMAN, S. R. & WHITE, I. R. 2013. Review of inverse probability weighting for dealing with missing data. *Stat Methods Med Res,* 22**,** 278-95.

SEAR, R. 2006. Height and reproductive success : How a Gambian population compares with the west. *Hum Nat,* 17**,** 405-18.

SGUASSERO, Y., DE ONIS, M., BONOTTI, A. M. & CARROLI, G. 2012. Community-based supplementary feeding for promoting the growth of children under five years of age in low and middle income countries. *Cochrane Database Syst Rev***,** Cd005039.

SHAH MORE, N., BAPAT, U., DAS, S., ALCOCK, G., PATIL, S., POREL, M., . . . OSRIN, D. 2012. Community mobilization in Mumbai slums to improve perinatal care and outcomes: a cluster randomized controlled trial. *PLoS Med,* 9**,** e1001257.

SHAH MORE, N., DAS, S., BAPAT, U., ALCOCK, G., MANJREKAR, S., KAMBLE, V., . . . OSRIN, D. 2017. Community resource centres to improve the health of women and children in informal settlements in Mumbai: a cluster-randomised, controlled trial. *Lancet Global Health,* 5**,** e335-e349.

SHAH MORE, N., DAS, S., BAPAT, U., RAJGURU, M., ALCOCK, G., JOSHI, W., . . . OSRIN, D. 2013. Community resource centres to improve the health of women and children in Mumbai slums: study protocol for a cluster randomized controlled trial. *Trials,* 14**,** 132.

SHAPIRO-MENDOZA, C., SELWYN, B. J., SMITH, D. P. & SANDERSON, M. 2005. Parental pregnancy intention and early childhood stunting: findings from Bolivia. *Int J Epidemiol,* 34**,** 387-96.

SILVA, L. M., VAN ROSSEM, L., JANSEN, P. W., HOKKEN-KOELEGA, A. C. S., MOLL, H. A., HOFMAN, A., . . . RAAT, H. 2012. Children of low socioeconomic status show accelerated linear growth in early childhood; results from the generation R study. *PLoS ONE,* 7.

SIMON, D. 2011. Situating slums. *City,* 15**,** 674-685.

SINHABABU, A., MUKHOPADHYAY, D. K., PANJA, T. K., SAREN, A. B., MANDAL, N. K. & BISWAS, A. B. 2010. Infant- and young child-feeding practices in Bankura district, West Bengal, India. *J Health Popul Nutr,* 28**,** 294-9.

SOLOMONS, N. W. & VOSSENAAR, M. 2013. Nutrient density in complementary feeding of infants and toddlers. *Eur J Clin Nutr,* 67**,** 501-6.

STEELE, F. 2011. Multilevel discrete-time event history models with applications to the analysis of recurrent employment transitions. *Australian & New Zealand Journal of Statistics,* 53**,** 1-20.

STEELE, F. 2014a. Multilevel Modelling for Binary Responses. LEMMA VLE Module 7, 1-21. Available: http://www.bristol.ac.uk/cmm/learning/course.html [Accessed 1 April 2018].

STEELE, F. 2014b. Multilevel Modelling of Repeated Measures Data. LEMMA VLE Module 15, 1-62. Available: http://www.bristol.ac.uk/cmm/learning/course.html [Accessed 1 April 2018].

STEINER, K. L., AHMED, S., GILCHRIST, C. A., BURKEY, C., COOK, H., MA, J. Z., . . . FARUQUE, A. S. G. 2018. Species of Cryptosporidia Causing Subclinical Infection Associated with Growth Faltering in Rural and Urban Bangladesh- a Birth Cohort Study. *Clin Infect Dis*.

STEWART, C. P., IANNOTTI, L., DEWEY, K. G., MICHAELSEN, K. F. & ONYANGO, A. W. 2013. Contextualising complementary feeding in a broader framework for stunting prevention. *Maternal & Child Nutrition,* 9 Suppl 2**,** 27-45.

STULP, G. & BARRETT, L. 2016. Evolutionary perspectives on human height variation. *Biol Rev Camb Philos Soc,* 91**,** 206-34.

SUBBARAMAN, R. & MURTHY, S. L. 2015. The right to water in the slums of Mumbai, India. *Bull World Health Organ,* 93**,** 815-6.

SUBBARAMAN, R., NOLAN, L., SAWANT, K., SHITOLE, S., SHITOLE, T., NANARKAR, M., . . . BLOOM, D. E. 2015. Multidimensional Measurement of Household Water Poverty in a Mumbai Slum: Looking Beyond Water Quality. *PLOS ONE,* 10**,** e0133241.

SUBBARAMAN, R., NOLAN, L., SHITOLE, T., SAWANT, K., SHITOLE, S., SOOD, K., . . . PATIL-DESHMUKH, A. 2014. The psychological toll of slum living in Mumbai, India: A mixed methods study. *Social Science & Medicine,* 119**,** 155-169.

SUBBARAMAN, R., O'BRIEN, J., SHITOLE, T., SHITOLE, S., SAWANT, K., BLOOM, D. E. & PATIL-DESHMUKH, A. 2012. Off the map: the health and social implications of being a non-notified slum in India. *Environment and Urbanization,* 24**,** 643-663.

SUBRAMANIAN, S. V., ACKERSON, L. K., DAVEY SMITH, G. & JOHN, N. A. 2009. Association of maternal height with child mortality, anthropometric failure, and anemia in India. *Jama,* 301**,** 1691-701.

SUBRAMANIAN, S. V., OZALTIN, E. & FINLAY, J. E. 2011. Height of nations: a socioeconomic analysis of cohort differences and patterns among women in 54 low- to middle-income countries. *PLoS One,* 6**,** e18962.

SVEFORS, P., RAHMAN, A., EKSTRÖM, E. C., KHAN, A. I., LINDSTRÖM, E., PERSSON, L. Å. & SELLING, K. E. 2016. Stunted at 10 years. Linear growth trajectories and stunting from birth to pre-adolescence in a rural Bangladeshi cohort. *PLoS ONE,* 11.

SWITKOWSKI, K. M., JACQUES, P. F., MUST, A., KLEINMAN, K. P., GILLMAN, M. W. & OKEN, E. 2016. Maternal protein intake during pregnancy and linear growth in the offspring. *American Journal of Clinical Nutrition,* 104**,** 1128-1136.

SYED, S., IQBAL, N. T., SADIQ, K., MA, J. Z., AKHUND, T., XIN, W., . . . ALI, S. A. 2018. Serum anti-flagellin and anti-lipopolysaccharide immunoglobulins as predictors of linear growth faltering in Pakistani infants at risk for environmental enteric dysfunction. *PLoS One,* 13**,** e0193768.

TADESSE, K., ZELENKO, O., MULUGETA, A. & GALLEGOS, D. 2018. Effectiveness of breastfeeding interventions delivered to fathers in low- and middle-income countries: A systematic review. *Matern Child Nutr,* 14**,** e12612.

TAKAHASHI, E. 1984. Secular trend in milk consumption and growth in Japan. *Hum Biol,* 56**,** 427-37.

TAN, F. E. S., JOLANI, S. & VERBEEK, H. 2018. Guidelines for multiple imputations in repeated measurements with time-dependent covariates: a case study. *J Clin Epidemiol,* 102**,** 107-114.

TU, Y. K., TILLING, K., STERNE, J. A. & GILTHORPE, M. S. 2013. A critical evaluation of statistical approaches to examining the role of growth trajectories in the developmental origins of health and disease. *Int J Epidemiol,* 42**,** 1327-39.

TWISK, J. W., SMIDT, N. & DE VENTE, W. 2005. Applied analysis of recurrent events: a practical overview. *J Epidemiol Community Health,* 59**,** 706-10.

UN-HABITAT 2003. The Challenge of Slums. Global Report on Human Settlements 2003. Kenya: UN Habitat.

UN-HABITAT 2016. World Cities Report 2016. Urbanization and development: Emerging futures. Nairobi: UN Habitat.

UNICEF 1990. Strategy for improved nutrition of children and women in developing countries. A UNICEF policy review. New York: UNICEF.

UPADHYAY, A. K. & SRIVASTAVA, S. 2016. Effect of pregnancy intention, postnatal depressive symptoms and social support on early childhood stunting: findings from India. *BMC Pregnancy Childbirth,* 16**,** 107.

VAIL, B., PRENTICE, P., DUNGER, D. B., HUGHES, I. A., ACERINI, C. L. & ONG, K. K. 2015. Age at Weaning and Infant Growth: Primary Analysis and Systematic Review. *Journal of Pediatrics,* 167**,** 317-324.e1.

VAN DEN BROECK, J., CUNNINGHAM, S. A., EECKELS, R. & HERBST, K. 2005. Data cleaning: detecting, diagnosing, and editing data abnormalities. *PLoS Med,* 2**,** e267.

VAN GAMEREN-OOSTEROM, H. B. M., VAN DOMMELEN, P., OUDESLUYS-MURPHY, A. M., BUITENDIJK, S. E., VAN BUUREN, S. & VAN WOUWE, J. P. 2012. Healthy Growth in Children with Down Syndrome. *PLoS ONE,* 7**,** e31079.

VANDENBROUCKE, J. P. 1991. Prospective or retrospective: what's in a name? *Bmj,* 302**,** 249-50.

VANDENBROUCKE, J. P., BROADBENT, A. & PEARCE, N. 2016. Causality and causal inference in epidemiology: the need for a pluralistic approach. *International Journal of Epidemiology,* 45**,** 1776-1786.

VANDERWEELE, T. J. 2009. On the distinction between interaction and effect modification. *Epidemiology,* 20**,** 863-71.

VANDERWEELE, T. J. 2016a. Commentary: On Causes, Causal Inference, and Potential Outcomes. *Int J Epidemiol,* 45**,** 1809-1816.

VANDERWEELE, T. J. 2016b. Mediation Analysis: A Practitioner's Guide. *Annual Review of Public Health,* 37**,** 17-32.

VANDERWEELE, T. J. & KNOL, M. J. 2014. A Tutorial on Interaction. *Epidemiologic Methods,* 3**,** 33.

VANDERWEELE, T. J. & ROBINS, J. M. 2007. Four types of effect modification: a classification based on directed acyclic graphs. *Epidemiology,* 18**,** 561-8.

VANDERWEELE, T. J. & TCHETGEN TCHETGEN, E. J. 2017. Mediation analysis with time varying exposures and mediators. *J R Stat Soc Series B Stat Methodol,* 79**,** 917-938.

VANSTEELANDT, S. & DANIEL, R. M. 2017. Interventional effects for mediation analysis with multiple mediators. *Epidemiology,* 28**,** 258-265.

VELUSAMY, V., PREMKUMAR, P. S. & KANG, G. 2017. Exclusive breastfeeding practices among mothers in urban slum settlements: pooled analysis from three prospective birth cohort studies in South India. *Int Breastfeed J,* 12**,** 35.

VICTORA, C. G., BAHL, R., BARROS, A. J., FRANCA, G. V., HORTON, S., KRASEVEC, J., . . . ROLLINS, N. C. 2016. Breastfeeding in the 21st century: epidemiology, mechanisms, and lifelong effect. *Lancet,* 387**,** 475-90.

VICTORA, C. G., DE ONIS, M., HALLAL, P. C., BLOSSNER, M. & SHRIMPTON, R. 2010. Worldwide timing of growth faltering: revisiting implications for interventions. *Pediatrics,* 125**,** e473-e480.

VITZTHUM, V. J., THORNBURG, J. & SPIELVOGEL, H. 2018. Impacts of nocturnal breastfeeding, photoperiod, and access to electricity on maternal sleep behaviors in a non-industrial rural Bolivian population. *Sleep Health,* 4**,** 535-542.

VYAS, S. & KUMARANAYAKE, L. 2006. Constructing socio-economic status indices: how to use principal components analysis. *Health Policy Plan,* 21**,** 459-468.

WANJOHI, M., GRIFFITHS, P., WEKESAH, F., MURIUKI, P., MUHIA, N., MUSOKE, R. N., . . . KIMANI-MURAGE, E. W. 2016. Sociocultural factors influencing breastfeeding practices in two slums in Nairobi, Kenya. *Int Breastfeed J,* 12**,** 5.

WELLS, G. & SHEA, B. 2018. Newcastle-Ottawa quality assessment scale cohort studies. Available: http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp [Accessed 1 March 2018].

WELLS, J. C. 2012. A critical appraisal of the predictive adaptive response hypothesis. *Int J Epidemiol,* 41**,** 229-35.

WELLS, J. C. K. 2017. Worldwide variability in growth and its association with health: Incorporating body composition, developmental plasticity, and intergenerational effects. *American Journal of Human Biology,* 29**,** n/a-n/a.

WELLS, J. C. K. 2018. Life history trade-offs and the partitioning of maternal investment: Implications for health of mothers and offspring. *Evolution, Medicine, and Public Health,* 2018**,** 153-166.

WEST, S., KING, V., CAREY, T. S., LOHR, K. N., MCKOY, N., SUTTON, S. F. & LUX, L. 2002. Systems to rate the strength of scientific evidence. *Evid Rep Technol Assess (Summ)***,** 1-11.

WHITE, J. M., BÉGIN, F., KUMAPLEY, R., MURRAY, C. & KRASEVEC, J. 2017. Complementary feeding practices: Current global and regional estimates. *Maternal & Child Nutrition,* 13**,** n/a-n/a.

WHO 2000. Obesity: Preventing and Managing the Global Epidemic. *WHO Technical Report Series: 894.* Geneva: World Health Organization.

WHO 2006. WHO Child Growth Standards. Length/height-for-age, weight-for-age, weight-for-length, weight-for-height and body mass index-for-age. Methods and development. Geneva: WHO.

WHO 2008a. *Indicators for assessing infant and young child feeding practices. Part 1: definitions,* Geneva, World Health Organization Department of Child and Adolescent Health and Development.

WHO 2008b. Training course on child growth assessment. Geneva: WHO.

WHO 2010. *Indicators for assessing infant and young child feeding practices. Part 2: measurement,* Geneva, World Health Organization Department of Child and Adolescent Health and Development.

WHO 2013. Essential Nutrition Actions: Improving maternal, newborn, infant and young child health and nutrition. Geneva: WHO.

WHO. 2016. Global Nutrition Targets 2025: Infographics. *Highlighting the key messages and recommended actions* [Online]. Available: http://www.who.int/nutrition/global-target-2025/infographics/en/.

WHO 2018a. Enabling women to breastfeed through better policies and programmes: Global breastfeeding scorecard 2018. Geneva: WHO.

WHO. 2018b. Guideline: counselling of women to improve breastfeeding practices. Available: https://apps.who.int/iris/bitstream/handle/10665/280133/9789241550468-eng.pdf?ua=1 [Accessed 1 April 2019].

WHO. 2019a. Simplified field tables. Length-for-age BOYS. Birth to 2 years (z-scores). Available: https://www.who.int/childgrowth/standards/sft_lhfa_boys_z_0_2.pdf?ua=1 [Accessed 1 January 2019].

WHO. 2019b. Simplified field tables. Length-for-age GIRLS. Birth to 2 years (z-scores). Available: https://www.who.int/childgrowth/standards/sft_lhfa_girls_z_0_2.pdf?ua=1 [Accessed 1 January 2019].

WHO / UNICEF 2017. Safely managed drinking water - thematic report on drinking water 2017. Geneva: WHO.

WHO / UNICEF 2018. JMP Methodology. 2017 Update & SDG Baselines. Geneva: WHO.

WHO EXPERT CONSULTATION 2004. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *The Lancet,* 363**,** 157-163.

WHO MULTICENTRE GROWTH REFERENCE STUDY GROUP 2006a. Assessment of differences in linear growth among populations in the WHO Multicentre Growth Reference Study. *Acta Paediatr Suppl,* 450**,** 56-65.

WHO MULTICENTRE GROWTH REFERENCE STUDY GROUP 2006b. Reliability of anthropometric measurements in the WHO Multicentre Growth Reference Study. *Acta Paediatr Suppl,* 450**,** 38-46.

WHO WORKING GROUP 1986. Use and interpretation of anthropometric indicators of nutritional status. WHO Working Group. *Bull World Health Organ,* 64**,** 929-41.

WHO/UNICEF. 2018. WHO Discussion Paper: The extension of the 2025 Maternal, Infant and Young Child nutrition targets to 2030. Available: https://www.who.int/nutrition/global-target-2025/discussion-paper-extension-targets-2030.pdf [Accessed 1 April 2019].

WILCOX, A. J. 2001. On the importance--and the unimportance--of birthweight. *Int J Epidemiol,* 30**,** 1233-41.

WIRTH, J. P., ROHNER, F., PETRY, N., ONYANGO, A. W., MATJI, J., BAILES, A., . . . WOODRUFF, B. A. 2017. Assessment of the WHO Stunting Framework using Ethiopia as a case study. *Matern Child Nutr,* 13.

WIT, J. M., HIMES, J. H., VAN BUUREN, S., DENNO, D. M. & SUCHDEV, P. S. 2017. Practical application of linear growth measurements in clinical research in low-and middle-income countries. *Hormone Research in Paediatrics,* 88**,** 79-90.

WOJCICKI, J. M., HEYMAN, M. B., ELWAN, D., LIN, J., BLACKBURN, E. & EPEL, E. 2016. Early exclusive breastfeeding is associated with longer telomeres in Latino preschool children. *Am J Clin Nutr,* 104**,** 397-405.

WOO, J. G., GUERRERO, M. L., RUIZ-PALACIOS, G. M., PENG, Y. M., HERBERS, P. M., YAO, W., . . . MORROW, A. L. 2013. Specific infant

feeding practices do not consistently explain variation in anthropometry at age 1 year in urban United States, Mexico, and China cohorts. *Journal of Nutrition,* 143**,** 166-174.

WRAY, J. D. 1986. Child health interventions in urban slums: are we neglecting the importance of nutrition? *Health Policy Plan,* 1**,** 299-308.

WRIGHT, M. J., BENTLEY, M. E., MENDEZ, M. A. & ADAIR, L. S. 2015. The interactive association of dietary diversity scores and breast-feeding status with weight and length in Filipino infants aged 6-24 months. *Public Health Nutrition,* 18**,** 1762-1773.

XU, F., BINNS, C., ZHANG, H., YANG, G. & ZHAO, Y. 2010. Paternal smoking and breastfeeding in Xinjiang, PR China. *J Hum Lact,* 26**,** 242-7.

ZEMEL, B. S. 2017. Influence of complex childhood diseases on variation in growth and skeletal development. *American Journal of Human Biology,* 29**,** n/a-n/a.

ZEMEL, B. S., PIPAN, M., STALLINGS, V. A., HALL, W., SCHADT, K., FREEDMAN, D. S. & THORPE, P. 2015. Growth Charts for Children With Down Syndrome in the United States. *Pediatrics,* 136**,** e1204-e1211.

ZHANG, Y., ZHOU, J., NIU, F., DONOWITZ, J. R., HAQUE, R., PETRI, W. A., JR. & MA, J. Z. 2017. Characterizing early child growth patterns of height-for-age in an urban slum cohort of Bangladesh with functional principal component analysis. *BMC Pediatrics,* 17.

ZHANG, Y. T., LARAIA, B. A., MUJAHID, M. S., TAMAYO, A., BLANCHARD, S. D., WARTON, E. M., . . . KARTER, A. J. 2015. Does food vendor density mediate the association between neighborhood deprivation and BMI?: a G-computation mediation analysis. *Epidemiology,* 26**,** 344-52.

ZHANG, Z. & SUN, J. 2010. Interval censoring. *Stat Methods Med Res,* 19**,** 53-70.

ZHAO, J., ZHAO, Y., DU, M., BINNS, C. W. & LEE, A. H. 2017. Maternal education and breastfeeding practices in China: A systematic review and meta-analysis. *Midwifery,* 50**,** 62-71.

ZULU, E. M., BEGUY, D., EZEH, A. C., BOCQUIER, P., MADISE, N. J., CLELAND, J. & FALKINGHAM, J. 2011. Overview of migration, poverty and health dynamics in Nairobi City's slum settlements. *J Urban Health,* 88 Suppl 2**,** S185-99.

Appendix 1

Appendix 1.1 Memorandum of understanding (signatures removed)

Memorandum of understanding

Use of data collected by SNEHA for research associated with graduate studentship: Komal Bhatia

This MOU describes a research collaboration between the SNEHA (Society for Nutrition, Education and Health Action) and Komal Bhatia, a PhD student at the Institute for Global Health, University College London. The MOU outlines expectations for the proposed collaboration.

As part of Komal Bhatia's research, SNEHA have suggested that she analyse data collected by the SNEHA Centres programme.

Presented here are conditions for the project:

i) Data generated from the project are owned by SNEHA.

ii) The results of any analysis of data generated from the project will not be presented or published without the express written consent of both SNEHA and Komal Bhatia.

iii) Access to the data will be provided to others only given the express written consent of both SNEHA and Komal Bhatia.

iv) Komal Bhatia will be the first author on any papers that she has taken the lead in conceptualisation and drafting. Others directly involved in the research will be considered for co-authorship in accordance with the conventions for authorship as set out by the International Committee of Medical Journal Editors (http://www.icmje.org/ethical_1author.html). Specifically, these guidelines state that authorship credit should be based upon:

1) Substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data.
2) Drafting the article or revising it critically for important intellectual content.
3) Final approval of the version to be published.

Authors should meet conditions 1, 2 and 3.

v) Komal Bhatia will be responsible for maintaining the security and confidentiality of any data generated. All personal identifiers will be removed from transcripts and any electronic files will be kept under electronic password-protection. Any written material arising from the data will similarly be kept securely.

vi) As part of the collaboration Komal Bhatia will develop an active involvement in the work of SNEHA as her PhD supervisor, David Osrin, and SNEHA project directors deem appropriate.

_____

Neena Shah More Date

Programme Director

SNEHA, Mumbai, India

_____

Komal Bhatia Date

PhD Student

Institute for Global Health

University College London, UK

Appendix 1.2 Ethics approval

UCL RESEARCH ETHICS COMMITTEE
ACADEMIC SERVICES

21 September 2015

Professor David Osrin
Institute for Global Health
UCL

Dear Professor Osrin

Notification of Ethical Approval
Project ID: 7403/001: The double burden of malnutrition in urban informal settlements in Mumbai: prevalence and determinants in a prospective observational cohort

Further to your satisfactory responses to the committee's comments, I am pleased to confirm in my capacity as Chair of the UCL Research Ethics Committee (REC) that your study has been approved by the UCL REC for the duration of the project i.e. until September 2016 on condition that local ethics approval from the Holy Family Hospital's Ethics Committee is secured before the commencement of the research.

Approval is also subject to the following conditions:

1. You must seek Chair's approval for proposed amendments to the research for which this approval has been given. Ethical approval is specific to this project and must not be treated as applicable to research of a similar nature. Each research project is reviewed separately and if there are significant changes to the research protocol you should seek confirmation of continued ethical approval by completing the 'Amendment Approval Request Form' :http://ethics.grad.ucl.ac.uk/responsibilities.php

2. It is your responsibility to report to the Committee any unanticipated problems or adverse events involving risks to participants or others. The Ethics Committee should be notified of all serious adverse events via the Ethics Committee Administrator (ethics@ucl.ac.uk) immediately the incident occurs. Where the adverse incident is unexpected and serious, the Chair or Vice-Chair will decide whether the study should be terminated pending the opinion of an independent expert. The adverse event will be considered at the next Committee meeting and a decision will be made on the need to change the information leaflet and/or study protocol.

   For non-serious adverse events the Chair or Vice-Chair of the Ethics Committee should again be notified via the Ethics Committee Administrator (ethics@ucl.ac.uk) within ten days of an adverse incident occurring and provide a full written report that should include any amendments to the participant information sheet and study protocol. The Chair or Vice-Chair will confirm that the incident is non-serious and report to the Committee at the next meeting. The final view of the Committee will be communicated to you.

On completion of the research you must submit a very brief report of your findings/concluding comments to the Committee, which includes in particular issues relating to the ethical implications of the research.

Academic Services, 1-19 Torrington Place (9th Floor),
University College London
Tel: +44 (0)20 3108 8216
Email: ethics@ucl.ac.uk
http://ethics.grad.ucl.ac.uk/

With best wishes for the research.

Yours sincerely

[redacted]

Professor John Foreman
Chair of the UCL Research Ethics Committee

Cc: Komal Bhatia, Applicant

Appendix 2

Appendix 2.1 Literature review search terms

Search	Concept	Search terms [wildcard entry] combined with Boolean operator OR
1	Infant linear growth	Infant length Infant height Infant linear growth Infant stature Infant length for age Infant height for age Infant growth faltering
2	Cause	Cause Causative Causal Factor Determinant Determine Risk factor Reason Contribute [contribut*] Predict [predict*] Prevent [prevent*] Explain Explanatory [explanat*] Influence [influenc*] Effect [effect*] Affect [affect*] Driver [drive*] Mediate [mediat*]
3	Population or disease state	Embryo Monkey Macaque Mouse Mice Murine Rodent Rat Rabbit Pig Porcine Pre term Premature Prematurity Fitness Adiposity Adolescent [adolescen*] Intensive care Twin Growth hormone Insulin Stillbirth HIV Acute renal failure Burn [burn*] Cancer

Search	Concept	Search terms [wildcard entry] combined with Boolean operator OR
		Oncology [oncol*] Dialysis Kidney Disease Puberty Dwarf* Dysplasia Mucopolysaccharidosis Achondroplasia Downs syndrome Chromosomal abnormality Hypothyroidism Telomere length Dystrophy Osteogenesis imperfecta Hearing loss Congenital Bradycardia Tachycardia Surgery Muscular atrophy [muscular atrophy*] Mitochondria [mitochondria*] Cystic fibrosis Motor neurone Sepsis Vocal [vocal*] Cochlea [cochlea*] Language Speech Vocabulary Cleft Myopia Parenteral Enteral Ultrasound Sudden infant death Sudden infant death syndrome

Search strategy

I first combined the search terms within each concept with the 'OR'. I then combined the first two concepts, infant linear growth and causes, with the Boolean operator 'AND' to search for studies that included terms related to both concepts, and then combined this search with the population group using 'NOT' in order to exclude studies that had any terms related to the population or diseases specified. In the databases, this search took the following form:

(1 AND 2) NOT 3

I also restricted my search to studies reported in English.

PubMed search

((((((((((((Infant length OR infant height OR infant linear growth OR infant stature OR growth faltering OR infant height for age OR infant length for age))) AND ((Cause OR causative OR causal OR factor OR determinant OR determine OR risk factor OR mediat* OR drive* OR reason OR contribut* OR predict* OR prevent* OR explain OR explanat* OR influenc* OR effect* OR affect*))) NOT (Embryo* OR monkey OR macaque OR mouse OR mice OR murine OR rodent OR rat OR rabbit OR pig OR porcine OR pre term OR premature OR prematurity OR fitness OR adiposity OR adolescen* OR intensive care OR twin OR growth hormone OR insulin OR stillbirth OR HIV OR acute renal failure OR burn* OR cancer OR oncol* OR dialysis OR kidney disease OR puberty OR dwarf* OR dysplasia OR mucopolysaccharidosis OR achondroplasia OR downs syndrome OR chromosomal abnormality OR hypothyroidism or telomere length OR dystrophy OR osteogenesis imperfecta OR hearing loss OR congenital OR bradycardia OR tachycardia OR surgery OR muscular atrophy* OR mitochondria* OR cystic fibrosis OR motor neurone OR sepsis OR vocal* OR cochlea* OR language OR speech OR vocabulary OR cleft OR myopia OR parenteral OR enteral OR ultrasound OR sudden infant death OR sudden infant death syndrome)) AND English[lang])) AND English[lang])) AND English[lang])))))) Sort by: Best Match

Scopus search

((infant AND length OR infant AND height OR infant AND linear AND growth OR infant AND stature OR growth AND faltering OR infant AND height AND for AND age OR infant AND length AND for AND age) AND (cause OR causative OR causal OR factor OR determinant OR determine OR risk AND factor OR mediat* OR drive* OR reason OR contribut* OR predict* OR prevent* OR explain OR explanat* OR influenc* OR effect* OR affect*)) AND NOT ((embryo* OR monkey OR macaque OR mouse OR mice OR murine OR rodent OR rat OR rabbit OR pig OR porcine OR pre AND term OR premature OR prematurity OR fitness OR adiposity OR adolescen* OR intensive AND care OR twin OR growth AND hormone OR insulin OR stillbirth

OR hiv OR acute AND renal AND failure OR burn* OR cancer OR oncol* OR dialysis OR kidney AND disease OR puberty OR dwarf* OR dysplasia OR mucopolysaccharidosis OR achondroplasia OR downs AND syndrome OR chromosomal AND abnormality OR hypothyroidism OR telomere AND length OR dystrophy OR osteogenesis AND imperfecta OR hearing AND loss OR congenital OR bradycardia OR tachycardia OR surgery OR muscular AND atrophy* OR mitochondria* OR cystic AND fibrosis OR motor AND neurone OR sepsis OR vocal* OR cochlea* OR language OR speech OR vocabulary OR cleft OR myopia OR parenteral OR enteral OR ultrasound OR sudden AND infant AND death OR sudden AND infant AND death AND syndrome)) AND (LIMIT-TO (LANGUAGE , "English "))

Appendix 2.2 Newcastle-Ottawa Scale

Domain	No.	Item	Options	Rating
Selection	1	Representativeness of the exposed cohort	Truly representative	1
			Somewhat representative	1
			Select group	0
			No description	0
	2	Selection of non-exposed cohort	Drawn from same community as exposed cohort	1
			Drawn from a different source	0
			No description	0
	3	Ascertainment of exposure	Secure record	1
			Structured interview	1
			Written self-report	0
			No description	0
	4	Demonstrate that outcome was not present at start of study	Yes	1
			No	0
Comparability of groups based on design or analysis	5	Controls for infant sex	Yes	1
			No	0
	6	Controls for additional confounders	Yes	1
			No	0
Outcome	7	Assessment of outcome	Independent blind assessment	1
			Record linkage	1
			Self-report	0
			No description	0
	8	Follow-up up to at least 1 year	Yes	1
			No	0
	9	Adequate follow-up	Complete follow-up	1
			Less than 10% attrition or missingness examined	1
			More than 10% attrition and missingness not examined	0
			No statement	0

Appendix 2.3 Data extraction framework template

Paper identifier
Number
First author, year
Year of publication
Study description & participants
Paper title
Aims
Cohort name
Study design
Nested in trial / intervention
Country
Urban/Rural
Participants
Timing of recruitment
Frequency of growth measurement
Age group for follow-up (months)
Expected number of growth measurements per participant
Analysis
Age range in analysis
Analysis sample
Growth used as
Standardization
Level of estimation
Metric type
Metric sub-type
Quantity of data to derive metric
Analytical approach to derive metric
Method(s) for determinants analysis
Main exposure(s) and timing of measurement
Definition(s) of outcome (effect estimate)
Covariates/confounders in final model
Mediators
Model selection methods
Results
Number of infants in the study
Number included in main analysis
Average number of length measurements per child
Exposure prevalence/incidence
Main findings
Conclusion
Critical appraisal checklist
Representativeness of the exposed cohort

Selection of non-exposed cohort
Ascertainment of exposure
Demonstrate that outcome was not present at start of study
Controls for infant sex
Control for confounders justified
Assessment of outcome
Follow-up spans full 1st year
Adequate follow-up
Additional comments

Appendix 2.4 Definitions of growth metrics

Selection	Description
1. **Standardization**	
Raw/Unstandardized	Anthropometric parameter was untransformed/unstandardized. Analysis was conducted on raw measures. For example, growth in length was analysed in cm, weight in g and BMI in kg/m^2.
Standardized	Anthropometric parameter is expressed in standard deviation score or percentile relative to the population mean. For example, the reference population used for standardization may be: 1) the study population (i.e., internal standardization), 2) a representative country population (i.e., country-specific standards), 3) a multi-ethnic population (i.e., the WHO-GS or INTERGROWTH-21st standards)
2. Level of analysis	
Group	Group-level analyses, based on comparing the average growth trajectories among 2 or more groups defined by an exposure or outcome other than size/growth (e.g., treatment group in a trial).
	Child-specific trajectories or velocities were not calculated, estimated or predicted in any analyses
	Children were not categorized on the basis of their individual trajectory
Individual	Child-specific trajectories were calculated, estimated or predicted for use in subsequent analyses.
3. Metric type	
Continuous	Group- or individual-level descriptor of growth is a continuous variable that quantitatively ranks children in terms of faster or slower growth relative to peers
	E.g., velocity, time to peak z-score, area-under-the-curve
Categorical	Group- or individual-level description of growth is based on assignment to a class or category of growth trajectories.
	This code should be selected even if the assignment to a category is based on an underlying continuous metric with a specified cut-off (e.g., 'fast growth' is based on a change in z-score of >0.67 z-scores). If the continuous metric is reported in both quantitative and categorical terms, these should be reported as distinct metrics.
4. **Quantity of data**	
1 data point	Growth was described based on only one anthropometric data point per child (cross-sectional analysis)
	For a group-average metric, this would be the mean of a group of children's values
	This code would apply even if the analysis involves serial cross-sectional analyses with only qualitative comments about changes over time.
2 data points	Each child or group trajectory or velocity was calculated, estimated, predicted or categorized based on a maximum of 2 size measurements, one at or near the beginning of the interval and one at or near the end of the interval
More than 2 points	Each child or group trajectory or velocity was calculated, estimated, predicted or categorized based on 3 or more size

Selection	Description
	measurements within an interval of interest.
	This code should be used if the intention of the investigators was to use >2 data points, even if some of the children in the dataset did not (or could not be confirmed to have had) at least 3 data points in each interval.
5. Metric sub-type	
Mean	This would be the mean of a group of children's values for a continuous variable
Proportion	Prevalence or incidence of a group of children's values for a categorical variable.
Incremental change	Arithmetic difference between size at the end and beginning of a specified age/time interval
	May be estimated at the individual or group level.
	For group-level metrics, this can be a change in the group's mean size, or the average of within-child changes.
Incremental rate of change	Arithmetic difference between size at the end and beginning of a specified age/time interval, expressed as a function of time (velocity or rate, e.g. cm/year)
	For metrics that were based on >2 data points per child, then incremental rate of change would be based on a slope representing the average rate of change of size over the specified interval.
	The slope could represent an individual child OR a group mean.
	Child-specific slopes may be expressed in absolute terms or relative to a group mean (e.g., a child-specific random slope, or best linear unbiased prediction, indicating a deviation from the group fixed effect).
Instantaneous rate of change	Expressed similarly to the incremental rate of change, but based on the first-derivative at a single specified point on the size-by-age slope.
	The slope from which the instantaneous rate of change is derived could represent an individual child OR a group mean trajectory.
Proportional change	Fractional change (%) in size from the beginning to end of interval, relative to the child's size at the beginning of the interval
	Note: the exponentiated difference on the log-scale is the same as a proportional difference
Proportional rate of change	Fractional change (%) in size from the beginning to end of interval, relative to the child's size at the beginning of the interval, expressed as a function of time (e.g., % per month).
	For metrics that were based on >2 data points per child, then proportional rate of change would be based on a child-specific linear slope representing the rate of change of *log-transformed* size over the specified interval.
	The slope could represent an individual child OR a group mean.
	Child-specific slopes may be expressed in absolute terms or relative to a group mean (e.g., a child-specific random slope as a deviation from the group fixed effect).
Conditional change (or conditional difference)	Difference between the observed and expected size at the end of the interval, where the expected value is based on the absolute size at the beginning of the interval and the overall correlation between size at the beginning and end of the interval (within the group as a whole).
	Usually this is an individual-level metric (therefore, be cautious about applying this descriptor to a group-level metric).

Selection	Description
	For studies in which growth is an exposure variable, conditional growth is typically estimated as the child-specific residual from a regression model in which size at a given age is regressed on size as an earlier age.
	In studies in which growth is the outcome variable, size at the beginning of the interval may be included as one of several covariates in a regression model designed to identify predictors of (or risk factors for) growth.
Age-scaling factor	Rate of growth is expressed in terms of a *proportional* expansion or contraction of the age scale
Tempo (time-to-event)	Growth is described quantitatively in terms of the duration of a specific interval of interest, for which the end marks a definable event.
	This metric may be derived directly from the size-by-age curve or from the velocity-by-age curve (e.g., time from birth to peak height velocity).
Maximum or minimum point on a trajectory	A child or group trajectory is described in terms of the highest or lowest value on either the size-by-age or velocity-by-age curve.
Velocity z-score	Use of an external reference or standard to assign a child-specific z-score to reflect *rate* of growth rather than size (e.g., the World Health Organization growth velocity z-scores)
Class	Group- or individual-level description of growth is based on assignment to a class or category of growth trajectories.
Other	Any other quantitative measure not mentioned above
6. Analytical approach	
Manual or simple calculation	Metric is based on the simple arithmetic operation for each child/group.
	Did not involve statistical modelling.
	Metric was derived empirically, rather than modelled or predicted.
	This could apply to either quantitative or categorical metrics, if the above criteria fit.
Threshold values or cut-points	Metric is based on categorizing an underlying continuous measure using threshold values or cut-points
	This analytical approach should only be used for ‘class’ metrics
	E.g., a catch-up growth ‘class’ is defined as
	≥0.67 increase in HAZ, where the underlying continuous measure from which this class metric is derived is change in HAZ over a specified interval
Child-specific regression model – pre-defined structural model	A regression model was built for each child, based on the repeated measures of size over time.
	This code is used for models for which the shape of the curve has a pre-set defined functional form (e.g., exponential).
	This code should only be used for analyses that involved classical parametric growth models (e.g., Jenss-Bayley)
	Indicate the specific model in the next item
Child-specific regression model – empirical, data-driven model	A regression model was built for each child, based on the repeated measures of size over time.
	However, this code is used for all child-specific models other than those considered to be pre-defined structural models, e.g., those for which the parameters were selected based on their fit to the observed data (e.g., natural cubic regression spline).
Modelling of group(s) data - fixed effect	Regression of size parameter as a linear function of age/time using data from all children in a group

Selection	Description
regression with linear splines	No random (child-specific) slopes
	Metrics are limited to group averages (e.g., group average slope)
Modelling of group(s) data - fixed effect regression with non-linear curve	Same as above, but the form of the regression function may be non-linear (e.g., cubic spline).
	No random (child-specific) slopes
	Metrics are limited to group averages (e.g., group average slope)
Modelling of group(s) data – random or mixed effect regression with or without linear splines	Regression of size parameter as a function of age/time using data from all children in a group
	Metrics are NOT limited to group averages, as child-specific metrics may be derived from child-specific random slopes
Modelling of group(s) data – random or mixed effect regression with non-linear functions	Same as above, but the model incorporates non-linear terms such as cubic splines or polynomials.
	Child-specific metrics may be derived from child-specific random slopes
Conditional regression	Regression modelling in which size at one age is regressed on size at a previous time point, thereby generating a conditional metric of growth (the model residual).
	The regression model includes data from multiple children (i.e., this is not a child-specific model).
	May be used as a 'step 1' model to generate child-specific growth metrics for use in subsequent modelling of the association between growth and a later outcome.
	Or, may be the primary analytical model in studies of predictors of (or risk factors for) growth; in such models, size at the previous time point may be one of several covariates.
SITAR model	Shape-invariant non-linear mixed effects model of size as a function of age/time
	Child-specific metrics may be derived from child-specific random effects: age-scaling factor, age intercepts and size intercepts.
	Group average curve is also fitted.
Growth Mixture Modelling	Structural equation modelling which identifies latent or unobservable subgroups within a given population
	If the model is linear, then child-specific metrics may be derived from child-specific random slopes and/or intercepts.
	If model is non-linear, child-specific metric is based on assignment to a 'latent class' or category of growth trajectories.
Latent Growth Curves	Same as above, but variance and covariance are set to zero (i.e., all individual growth trajectories within a 'latent class' are assumed to be homogeneous)
	Metrics are limited to random slope and/or intercept if the model is linear, and 'latent class' if the model is non-linear.
Machine Learning	Model uses algorithms to learn patterns from the data without explicitly being programmed (i.e., hierarchical clustering, Bayesian modelling)
	Child specific-metric is often but not limited to a class/category of growth trajectory, since machine learning is very flexible.
Other	Select this code if author describes another analytical approach that has not yet been described above.

Appendix 2.5 Data extraction framework

Results of the data extraction are presented as separate tables for study description and participants, analysis, results, and critical appraisal and comments.

ID		Study description & participants		
#	First author, year	Paper title	Aims	Cohort name
1	Syed, 2018	Serum anti-flagellin and anti-lipopolysaccharide immunoglobulins as predictors of linear growth faltering in Pakistani infants at risk for environmental enteric dysfunction	To determine whether levels of antibodies against bacterial components flagellin and lipopolysaccharide predict poor growth	N/A
2	Steiner, 2018	Species of Cryptosporidia Causing Subclinical Infection Associated with Growth Faltering in Rural and Urban Bangladesh- a Birth Cohort Study	Characterise the burden of cryptosporidiosis in the first two life, and estimate its impact on growth faltering	N/A
3	Schnee, 2018	Identification of aetiology-specific diarrhoea associated with linear growth faltering in Bangladeshi infants	To characterize diarrhoea aetiology and examine the association between aetiology-specific diarrhoea and linear growth and systemic inflammation	PROVIDE
4	Sanin, 2018	Micronutrient adequacy is poor, but not associated with stunting between 12-24 months of age: A cohort study findings from a slum area of Bangladesh	Prospectively examine determinants of stunting in a slum population in Bangladesh	MAL-ED
5	Moradi, 2018	Associations Between Dietary Energy Density in Mothers and Growth of Breastfeeding Infants During the First 4 Months of Life	To assess the influence of maternal dietary intake during lactation on infant growth	N/A
6	Lima, 2018	Enteroaggregative Escherichia coli Subclinical Infection and Coinfections and Impaired Child Growth in the MAL-ED Cohort Study	To evaluate the impact of subclinical enteroaggregative E.coli infection along with other pathogens between 0-6 months on child growth	MAL-ED
7	Kramer, 2018	Infant feeding and growth: putting the horse before the cart	Compare infant growth associated with 12+ months of BF vs shorter duration using different analytical approaches	PROBIT

ID		Study description & participants		
#	First author, year	Paper title	Aims	Cohort name
8	Islam, 2018	Risk factors of stunting among children living in an urban slum of Bangladesh: Findings of a prospective cohort study	Study the dietary practices of a cohort of children from birth to 24 months and identify predictors of stunting between 12 and 24 months in an urban slum	MAL-ED
9	Garzon, 2018	Subclinical enteric parasitic infections and growth faltering in infants in São Tomé, Africa: A birth cohort study	To explore the association between enteric pathogenic parasites and growth in infants during the first 24 months of life.	N/A
10	Devakumar, 2018	Socioeconomic determinants of growth in a longitudinal study in Nepal	To examine the potential associations between three components of SES (HH assess, maternal education, land ownership) measured before birth on child growth outcomes in early childhood.	N/A
11	Cheng, 2018	The Associations of Breast Feeding with Infant Growth and Body Mass Index to 16 years: 'Children of 1997'	To assess whether associations of breastfeeding with length from birth to 36 months differed by sex or by ag	Children of 1997
12	Admassu, 2018	Accretion of fat-free mass rather than fat mass in infancy is positively associated with linear growth in childhood	To examine the associations of early infancy fat mass (FM) and fat-free mass (FFM) with linear growth from 1 to 5 years of age	iABC
13	Zhang, 2017	Characterizing early child growth patterns of height-for-age in an urban slum cohort of Bangladesh with functional principal component analysis	To characterize early child growth patterns and quantify the change of growth curves from the WHO-GS in an urban slum cohort in Bangladesh	N/A
14	Matos, 2017	Growth patterns in early childhood: Better trajectories in Afro-Ecuadorians independent of sex and socioeconomic factors	Describe the growth patterns from 0 to 5 years of children living in rural, coastal Ecuador, testing the effects of ethnicity and sex.	ECUAVIDA
15	MAL-ED Network Investigators/ Caulfield, 2017	Relationship between growth and illness, enteropathogens and dietary intakes in the first 2 years of life: findings from the MAL-ED birth cohort study	To quantify the effects of enteropathogen infection, diarrhoea and diet on child growth	MAL-ED
16	Clemente, 2017	Prenatal ambient air pollution exposure, infant growth and placental mitochondrial DNA content in the INMA birth cohort	To describe the association between prenatal NO2 exposure and infant growth at 6 and 12 months, and whether growth at birth or placental mtDNA mediate that association.	INMA

ID		Study description & participants		
#	First author, year	Paper title	Aims	Cohort name
17	Bork, 2017	Boys are more stunted than girls from early infancy to 3 years of age in rural Senegal	To evaluate differences in height and complementary food intake between sexes from ages 2 to 39 months.	Alimfert cohort study
18	Bell, 2017	Associations of infant feeding with trajectories of body composition and growth	To assess associations of infant feeding with trajectories of growth and body composition from birth to 7 mo. in healthy infants	N/A
19	Swithowski, 2017	Maternal protein intake during pregnancy and linear growth in the offspring	to examine associations of maternal protein intake during pregnancy with offspring linear growth	Project Viva
20	Svefors, 2016	Stunted at 10 years. Linear growth trajectories and stunting from birth to pre-adolescence in a rural Bangladeshi cohort	To describe linear growth and stunting from birth to 10 years and examine influence of maternal and environmental determinants at conception.	MINIMat
21	Owais, 2016	Minimum acceptable diet at 9 months but not exclusive breastfeeding at 3 months or timely complementary feeding initiation is predictive of infant growth in rural Bangladesh	To prospectively assess the association between suboptimal infant feeding practices and growth faltering	Window of Opportunity
22	Nagata, 2016	Prevalence and predictors of malnutrition among Guatemalan children at 2 years of age	To identify the prevalence and predictors of malnutrition among 2-year old children in the Western Highlands of Guatemala	N/A
23	Kavle, 2016	Factors associated with early growth in Egyptian infants: Implications for addressing the dual burden of malnutrition	To determine if there were difference in growth patterns and in factors related to growth in Lower Egypt and Upper Egypt within the context of a USAID MCH programme	N/A
24	Griffiths, 2016	Do socio-economic inequalities in infant growth in rural India operate through maternal size and birth weight?	To establish whether SES inequalities in infant size at 12 months operate through maternal and early infant size measures.	Infant Feeding Study (IFS)
25	Gough, 2016	Linear growth trajectories in Zimbabwean infants	To identify the pattern and determinants of linear growth trajectories from birth through 24 months in a cohort of Zimbabwean infants	Zimbabwe Vitamin A for Mothers and Babies study
26	De Beaudrap, 2016	Timing of malaria in pregnancy and impact on infant growth and morbidity: A cohort study in Uganda	To describe the impact of malaria in pregnancy on infant growth, malaria, and morbidity.	N/A

ID		Study description & participants		
#	First author, year	Paper title	Aims	Cohort name
27	Busert, 2016	Dietary diversity is positively associated with deviation from expected height in rural Nepal	To examine the association between dietary diversity and conditional growth in children aged 0-89 months	N/A
28	Broere-Brown, 2016	Sex-specific differences in foetal and infant growth patterns: A prospective population-based cohort study	To assess whether sex-specific differences exist in foetal and infant growth	Generation R Study
29	Bhargava, 2016	Protein and micronutrient intakes are associated with child growth and morbidity from infancy to adulthood in the Philippines	To assess the effect of dietary intake on height during 2-24 months	Cebu Longitudinal Health and Nutrition Survey
30	Alkhalawi, 2016	Perfluoroalkyl acids (PFAAs) and anthropometric measures in the first year of life: Results from the Duisburg Birth Cohort	To examine the extent to which in utero exposure of these chemicals at background levels exerts an effect on new born and infant weight and length	Duisburg Birth Cohort Study
31	Wright, 2015	The interactive association of dietary diversity scores and breast-feeding status with weight and length in Filipino infants aged 6-24 months	To assess how BF and DD relate to infant LAZ	Cebu Longitudinal Health and Nutrition Survey
32	Vail, 2015	Age at Weaning and Infant Growth: Primary Analysis and Systematic Review	To test whether earlier age at weaning (3-6 months) may promote faster growth during infancy	Cambridge Baby Growth Study
33	Rogawski, 2015	Early Life Antibiotic Exposure Is Not Associated with Growth in Young Children of Vellore, India	To estimate the effects of antibiotic exposures in the first 6 months of life on short- and long-term growth.	(Vellore)
34	O'Keeffe, 2015	Maternal alcohol use during pregnancy and offspring trajectories of height and weight: A prospective cohort study	To examine the association of maternal alcohol use during pregnancy and offspring height trajectories.	ALSPAC
35	Hanieh, 2015	Antenatal and early infant predictors of postnatal growth in rural Vietnam: A prospective cohort study	To determine which antenatal and early-life factors were associated with infant postnatal growth in a resource-poor setting in Vietnam.	N/A
36	Costet, 2015	Perinatal exposure to chlorodecone and infant growth	To assess the impact of prenatal and postnatal exposure to chlorodecone on the growth of children from the TIMOUN mother-child cohort	TIMOUN
37	Richard, 2014	Catch-up growth occurs after diarrhoea in early childhood	To characterise catch-up growth in relation to diarrhoea burden	N/A

ID		Study description & participants		
#	First author, year	Paper title	Aims	Cohort name
38	Patel, 2014	Socioeconomic differences in childhood length/height trajectories in a middle-income country: A cohort study	To examine socioeconomic differences in stature from birth to childhood	PROBIT
39	Padanou, 2014	Factors associated with growth patterns from birth to 18 months in a Beninese cohort of children	To analyse factors influencing growth pattern of children from birth to 18 months	N/A
40	Murasko, 2014	Associations between household income, height and BMI in contemporary US children: Infancy through early childhood	To evaluate the association between household income and anthropometric development in early-life	Early Childhood Longitudinal Study - Birth Cohort (ECLS-B)
41	Mallard, 2014	Dietary diversity at 6 months of age is associated with subsequent growth and mediates the effect of maternal education on infant growth in Urban Zambia	To investigate whether meeting WHO IYCF indicators at 6 and 12 months of age is associated with growth at 18 months of age, and if DD mediates the relationship between household wealth, maternal education, and childhood growth	CIGNIS
42	Jaganath, 2014	First Detected Helicobacter pylori Infection in Infancy Modifies the Association Between Diarrheal Disease and Childhood Growth in Peru	To evaluate the role of H.pylori infection in infancy (6-11 months) vs early childhood (12-23 months) on height	N/A
43	Hong, 2014	Association of mid-pregnancy antioxidative vitamin and oxidative stress levels with infant growth during the first 3 years of life	To investigate the association between maternal micronutrient levels/oxidative stress in pregnancy and infant growth during the first 3 years of life.	Ewha Birth & Growth Cohort
44	Betoko, 2014	Determinants of infant formula use and relation with growth in the first 4 months	To determine the association between predominant type of formula used from birth to 4 months and growth	EDEN
45	Woo, 2013	Specific infant feeding practices do not consistently explain variation in anthropometry at age 1 year in urban United States, Mexico, and China cohorts	To examine the effect of differences in the timing of solid food introduction and the progression of specific foods on infant anthropometry at age 1 year.	Global Exploration of Human Milk (GEHM)
46	Richard, 2013	Diarrhoea in early childhood: Short-Term association with weight and long-Term association with length	To evaluate the lagged relationship between diarrhoea and growth in the first 2 years of life.	N/A
47	Peterson, 2013	REG1B as a predictor of childhood stunting in Bangladesh and Peru	To test whether the stool regenerating gene (REG1B) protein is a non-invasive biomarker of future childhood stunting	N/A

ID		Study description & participants		
#	First author, year	Paper title	Aims	Cohort name
48	Lee, 2013	Symptomatic and Asymptomatic Campylobacter Infections Associated with Reduced Growth in Peruvian Children	To examine the bi-directional relationship between Campylobacter infections and growth	N/A
49	Kwok, 2013	Grandparental education, parental education and child height: Evidence from Hong Kong's "Children of 1997" birth cohort	To elucidate socioeconomic influences on height at different growth phases	Children of 1997
50	Garza, 2013	Parental height and child growth from birth to 2 years in the WHO Multicentre Growth Reference Study	To estimate within-site variability in child length attributable to parental height in the WHO MGRS	WHO MGRS
51	Fairley, 2013	Describing differences in weight and length growth trajectories between white and Pakistani infants in the UK: Analysis of the Born in Bradford birth cohort study using multilevel linear spline models	To describe the growth pattern from birth to 2 years of UK-born white British and Pakistani infants	Born in Bradford
52	Durmus, 2013	Parental anthropometrics, early growth and the risk of overweight in pre-school children: The Generation R Study	To assess the associations of maternal and paternal anthropometrics with growth characteristics and the risk of overweight in pre-school children	Generation R Study
53	Addo, 2013	Maternal height and child growth patterns	To examine associations between maternal height and child growth in-utero, from birth to 2 years, 2 years to mid-childhood, and MC to adulthood	COHORTS
54	Silva, 2012	Children of low socioeconomic status show accelerated linear growth in early childhood; results from the generation R study	To examine the effect of maternal education as a marker of SES on linear growth in children aged 0-2 years	Generation R Study
55	Saha, 2012	Pre- and postnatal arsenic exposure and body size to 2 years of age: A cohort study in rural Bangladesh	To examine the effects of early-life arsenic exposure on weight and length of children from birth to 2 years of age	MINIMat
56	Richard, 2012	Wasting is associated with stunting in early childhood	To determine the effect of wasting in and variability in WLZ in the first 17 months on LAZ at 18-24 months, and change in WLZ in previous 6-month period on length at 18 and 24 months.	N/A
57	Queiroz, 2012	Predictors of linear growth in the first year of life of a prospective cohort of full term children with normal birth weight	To investigate determinants of the variation in mean LAZ in the first year of life of children born full term with normal birth	N/A

ID		Study description & participants		
#	First author, year	Paper title	Aims	Cohort name
58	Matijasevich, 2012	Maternal education inequalities in height growth rates in early childhood: 2004 Pelotas birth cohort study	To examine the associations of maternal education with birth length and trajectories of growth in length	Pelotas 2004
59	Martinez-Mesa, 2012	Life course association of maternal smoking during pregnancy and offspring's height: Data from the 1993 Pelotas (Brazil) birth cohort	To evaluate the effect of maternal smoking during pregnancy and partner smoking on offspring's height in infancy, childhood and adolescence	Pelotas 1993
60	Lourenco, 2012	Determinants of linear growth from infancy to school-aged years: A population-based follow-up study in urban Amazonian children	To investigate socioeconomic, maternal, and child determinants of linear growth	N/A
61	Kang Sim, 2012	Postnatal Growth Patterns in a Chilean Cohort: The Role of SES and Family Environment	To examine how family environmental characteristics serve as mediators in the relationship between socioeconomic conditions and infant growth	N/A
62	Husain, 2012	Maternal depression and infant growth and development in British Pakistani women: a cohort study	To examine if perinatal depression is a risk factor for poor child growth in first and second-generation British women of Pakistani origin	N/A
63	Hambridge, 2012	Infant stunting is associated with short maternal stature	To determine the range of maternal height associated with growth velocity of older infants and the magnitude of this relationship in an indigenous (Maya) population	N/A
64	Garced, 2012	Prenatal dichlorodiphenyldichloroethylene (DDE) exposure and child growth during the first year of life	To evaluate the association between prenatal dichlorodiphenyldichloroethylene (DDE) exposure and child growth at birth and the first year of life	N/A
65	Bork, 2012	A summary index of feeding practices is positively associated with height-for-age, but only marginally with linear growth, in rural Senegalese infants and toddlers	To study the relationship between ICFI, dietary diversity index, food variety index, meal frequency index, breastfeeding on HAZ and height growth over 6 months in rural Senegal	N/A
66	Matijasevich, 2011	Maternal smoking during pregnancy and offspring growth in childhood: 1993 and 2004 Pelotas cohort studies	To explore the effects of maternal smoking during pregnancy on offspring growth using (1) multiple adjustments for SES and parental factors (2) maternal-paternal comparisons as a test of putative intrauterine effects, (3) comparisons between two cohorts	Pelotas 1993 and 2004

ID		Study description & participants		
#	First author, year	Paper title	Aims	Cohort name
67	Durmus, 2011	Parental smoking during pregnancy, early growth, and risk of obesity in preschool children: The Generation R Study	To assess the associations of maternal and paternal smoking during pregnancy with early growth characteristics and risk of overweight and obesity in preschool children	Generation R Study
68	Deierlein, 2011	Effects of pre-pregnancy body mass index and gestational weight gain on infant anthropometric outcomes	To determine whether pre-pregnancy body mass index (BMI) and gestational weight gain (GWG) influence infant postnatal growth.	Pregnancy, Infection, and Nutrition (PIN) Postpartum Study
69	De Hoog, 2011	The role of infant feeding practices in the explanation for ethnic differences in infant growth: The Amsterdam Born Children and their Development study	To determine ethnic differences in growth rate 0-6 months and determine the role of infant feeding.	Amsterdam Born Children and their Development (ABCD)
70	Moore, 2010	Prolonged episodes of acute diarrhoea reduce growth and increased risk of persistent diarrhoea in children	To assess the relationship between acute diarrhoea and growth in children	N/A
71	Ertel, 2010	Maternal depressive symptoms not associated with reduced height in young children in a US prospective cohort study	To examine the relationships between antenatal and postpartum depressive symptoms and child linear growth from 0-3 years in a US sample.	Project Viva
72	de Beer, 2010	Relation of maternal hypertension with infant growth in a prospective birth cohort: the ABCD study	To investigate the assumed positive association of pre-existent and pregnancy-induced hypertension with the offspring's weight and length gain in the first 14 months of life	Amsterdam Born Children and their Development (ABCD)
73	Andersen, 2010	Prenatal exposures to perfluorinated chemicals and anthropometric measures in infancy	To estimate the associations between maternal plasma levels of perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) and infants' length during the first year of life	Danish National Birth Cohort
74	Le Beaud, 2015	Parasitism in Children Aged Three Years and Under: Relationship between Infection and Growth in Rural Coastal Kenya	To document the prevalence of parasitic infections and examine their association with growth during the first three years of life among children in coastal Kenya	N/A
75	Katulla, 2014	The first 1000 days of life: prenatal and postnatal risk factors for morbidity and growth in a birth cohort in southern India	To describe the effect of prenatal and postnatal factors on growth in the first 1000 days in a birth cohort in a semi-urban slum in Vellore, India	(Vellore)

ID		Study description & participants		
#	First author, year	Paper title	Aims	Cohort name
76	Howe, 2012	Socioeconomic differences in childhood growth trajectories: at what age do height inequalities emerge?	To examine the socioeconomic patterning of growth trajectories from birth to 10 years	ALSPAC
77	Johnson, 2012	Using the WHO 2006 child growth standard to assess the growth and nutritional status of rural south Indian infants	To assess the implications of using the WHO MGRS in India to investigate factors responsible for deviation from optimal growth	Infant Feeding Study (IFS)

ID		Study description & participants				
#	First author, year	Study design	Nested in trial / intervention	Country	Urban / Rural	Participants
1	Syed, 2018	Prospective birth cohort	Yes	Pakistan	Rural	Infants born in an RCT of RUTF, forming a cohort study of EED markers of growth
2	Steiner, 2018	Prospective birth cohort	No	Bangladesh	Both	Healthy infants born in two surveillance sites, one urban and one rural, to women over 18 years, without gestational illness (proteinuria, hypertension, oedema).
3	Schnee, 2018	Prospective birth cohort	Yes	Bangladesh	Urban	Infants born in an RCT of vaccination against rotavirus and polio
4	Sanin, 2018	Prospective birth cohort	No	Bangladesh	Urban slum	Live, singleton infants born to women > 16 years in a surveillance site in an urban slum in Dhaka, after screening for serious medical illness. Family intend to stay in area for at least 6 months, and willing to participate in monthly follow-up.
5	Moradi, 2018	Cross sectional study and retrospective record linkage study	No	Iran	Urban	Exclusively breastfed infants

ID		Study description & participants				
#	First author, year	Study design	Nested in trial / intervention	Country	Urban / Rural	Participants
6	Lima, 2018	Prospective birth cohort	No	Bangladesh, Brazil, India, Pakistan, South Africa, Tanzania	Both	Infants born in cohort sites between November 2009 and February 2012 to women >16 years, BW>1500g, singleton, and without any serious illness.
7	Kramer, 2018	Prospective study nested within a cluster-randomized trial of a breastfeeding promotion intervention	Yes	Belarus	Both	Infant-mother pairs enrolled in the PROBIT trial
8	Islam, 2018	Prospective birth cohort	No	Bangladesh	Urban slum	Live, singleton infants born to women > 16 years in a surveillance site in an urban slum in Dhaka, after screening for serious medical illness. Family intend to stay in area for at least 6 months, and willing to participate in monthly follow-up.
9	Garzon, 2018	Prospective birth cohort	No	Sao Tome and Principe	Both	Infants born AGA, birth weight > 2500g, gestation >37 weeks, no congenital malformation, recruited at mother infant health centres and local hospitals
10	Devakumar, 2018	Follow-up study of participants in an RCT of antenatal multiple micronutrient supplementation	Yes	Nepal	Both	Infants of all women recruited (intervention and control) into the trial (which showed no effect of MM on growth) attending antenatal appointments; singleton infants, no foetal abnormalities, no severe maternal illness
11	Cheng, 2018	Prospective birth cohort	No	Hong Kong	Urban	Infants born between 1 April to 31 May 1997 in Hong Kong
12	Admassu, 2018	Prospective birth cohort	No	Ethiopia	Urban	Apparently healthy children born (> 37 weeks gestation; BE > 1500g; no congenital malformation) in the maternity ward in a hospital in Jimma to parents residing in Jimma Town
13	Zhang, 2017	Prospective birth cohort	No	Bangladesh	Urban slum	Infants born in an urban slum in Dhaka's Mirpur Thana

ID		Study description & participants				
#	First author, year	Study design	Nested in trial / intervention	Country	Urban / Rural	Participants
14	Matos, 2017	Prospective birth cohort	No	Ecuador	Both	Infants born between November 2005 and December 2009 in a hospital in Quininde, in northern coastal Ecuador. Healthy infants born to women >17 years who had been in the District for at least 2 years and did not intend to move for 3 years, living in homes accessible from hospital.
15	MAL-ED Network Investigators / Caulfield, 2017	Prospective birth cohort	No	Bangladesh, Brazil, India, Nepal, Peru, South Africa, Tanzania	Both	Infants born in cohort sites between November 2009 and February 2012 to women >16 years, BW>1500g, singleton, and without any serious illness.
16	Clemente, 2017	Prospective birth cohort	No	Spain	Both	Infants of women in seven regions in Spain to women > 16 years of age, intention to deliver at particular hospital, no assisted conception.
17	Bork, 2017	Prospective cohort	Yes	Senegal	Rural	Infants in surveillance sites of two RCTs of pertussis vaccines between 1990 and 1997, and a subset born between Jan and Oct 1995.
18	Bell, 2017	Observational secondary analysis of an RCT	Yes	United States	Urban	Healthy, singleton infants born at >35 weeks gestation to women in a Vitamin D supplementation RCT in South Carolina and New York between January 2007 and December 2011
19	Swithowski, 2017	Prospective birth cohort	No	United States	Urban	Mother-child pairs enrolled between 1999 and 2002 in Boston, Massachusetts from Atrius Health <22 weeks gestation.
20	Svefors, 2016	Prospective birth cohort	Yes	Bangladesh	Rural	Infants born to mothers in the MINIMat factor RCT's intervention group in the ICDDR,B areas between April 2002 and June 2003
21	Owais, 2016	Prospective birth cohort	Yes	Bangladesh	Rural	Infants born to mothers in the Window of Opportunity IYCF programme's intervention and control areas

ID		Study description & participants				
#	First author, year	Study design	Nested in trial / intervention	Country	Urban / Rural	Participants
22	Nagata, 2016	Prospective birth cohort	Yes	Guatemala	Rural	Infants born in 20 communities in the Western Highlands of Guatemala between May 2008 and December 2013 in a community health surveillance setting. Children aged >6 months with WAZ< -2.5 were given a food supplement to treat malnutrition.
23	Kavle, 2016	Prospective birth cohort	Yes	Egypt	Both	Infants born in five semi-urban and five rural villages in USAID funded SMART implementation research projects, between February and March 2013 to women 18+ years old resident in SMART study sites.
24	Griffiths, 2016	Prospective study nested within a study to test the efficacy of an integrated feeding and care intervention among 3-16 month old infants in rural India	Yes	India	Rural	All infants born in three ICDS project areas covering 60 villages between September 2005 and April 2007.
25	Gough, 2016	Prospective birth cohort nested in a randomized, placebo-controlled trial of peripartum Vitamin A supplementation.	Yes	Zimbabwe	Urban	Subset of HIV-unexposed trial participants recruited between 1997 and 2001 were randomly selected for 24 months of follow up
26	De Beaudrap, 2016	Prospective birth cohort	Yes	Uganda	Rural	Live singleton infants born to pregnant women in between Oct 2006 and May 2009 in Mbarara district
27	Busert, 2016	Follow-up study of a cross-sectional survey	No	Nepal	Rural	Children 0-60 months in households in three villages in rural Nepal
28	Broere-Brown, 2016	Population-based prospective birth cohort	No	The Netherlands	Urban	Infants born to women in Rotterdam who gave birth between April 2002 and January 2006
29	Bhargava, 2016	Prospective birth cohort	No	Philippines	Both	Infants of pregnant women giving birth between May 1983 and April 1984 in 17 urban and 16 rural randomly selected areas; singleton births included

ID		Study description & participants				
#	First author, year	Study design	Nested in trial / intervention	Country	Urban / Rural	Participants
30	Alkhalawi, 2016	Prospective birth cohort	No	Germany	Urban	Highly motivated mother and infant pairs recruited between September 2002 and October 2002 in the North Rhine city of Duisburg
31	Wright, 2015	Prospective birth cohort	No	Philippines	Both	Infants of pregnant women giving birth between May 1983 and April 1984 in 17 urban and 16 rural randomly selected areas; singleton births included
32	Vail, 2015	Prospective birth cohort	No	United Kingdom	Urban	Women aged 16+ attending ultrasound clinics at Rosie Maternity Hospital in Cambridge between August 2001 and August 2009.
33	Rogawski, 2015	Prospective birth cohort	No	India	Urban slum	Pregnant women identified between March 2009 and May 2010 in four geographically adjacent semi urban slum areas of Vellore, Tamil Nadu, who gave birth to infants with birthweight >1500g and no major congenital malformations.
34	O'Keeffe, 2015	Prospective birth cohort	No	United Kingdom	Both	Infants of women who gave birth between April 1991 and December 1992 in one of three Bristol-based health districts
35	Hanieh, 2015	Prospective birth cohort	Yes	Vietnam	Rural	Infants born to women who had previously participated in a cluster RCT of micronutrient supplementation in Ha Nam province, Vietnam
36	Costet, 2015	Prospective birth cohort	No	Guadeloupe (French West Indies)	Urban	Infants born in Pointe a Pitre/Abymes or Basse-Terre public hospitals to pregnant women identified between November 2004 and December 2007.
37	Richard, 2014	Pooled analysis of 7 cohort studies	No	Peru, Brazil, Guinea-Bissau, and Bangladesh	Both	Infants in 7 cohort studies with diarrhoea surveillance, and anthropometry before 2 months and at least 4 measurements before 24 months in the original study
38	Patel, 2014	Prospective study nested within a cluster-randomized trial of a breastfeeding promotion intervention	Yes	Belarus	Both	Infant-mother pairs enrolled in the PROBIT trial who completed follow-up to 6.5 years

ID		Study description & participants				
#	First author, year	Study design	Nested in trial / intervention	Country	Urban / Rural	Participants
39	Padanou, 2014	Prospective birth cohort	No	Benin	Rural	Infants born to women recruited between July 2007 and July 2008 living permanently in nine villages in southern Benin's Tori Bossito district, and giving birth in a maternity ward in the catchment area
40	Murasko, 2014	Prospective birth cohort	No	United States	Both	Children born in the US during 2001, to women >15 years who survived to 9 months, with representative samples based on race/ethnicity, birthweight status, singleton/multiple births.
41	Mallard, 2014	Follow-up study of infants in an RCT of micronutrient fortified porridge to improve infant growth	Yes	Zambia	Urban	Infants in the CIGNIS trial's catchment areas in a middle income area in Lusaka
42	Jaganath, 2014	Prospective birth cohort	No	Peru	Urban slum	Singleton infants born with birthweight >1500g in two peri urban slums near Lima to pregnant women identified between May 2007 and February 2011 and randomly selected for participation.
43	Hong, 2014	Prospective birth cohort	No	Korea	Urban	Infants born to women (with no hypertension or diabetes) recruited at the Ehwa Women's University Hospital in Seoul between 24-28 weeks gestation
44	Betoko, 2014	Prospective birth cohort	No	France	Urban	Singleton infants born to women without a history of diabetes who visited Nancy and Poitiers University Hospitals for ANC between Feb-Sep 2003
45	Woo, 2013	Multi-country, prospective cohort study	No	United States, Mexico, China	Urban	Breastfeeding (for >75% of the time 0-2 months) mothers (18-49 years) and singleton infants born at 37+ weeks' gestation, without medical issues, and birthweight >2500grecruited between January 2007 and December 2008 from a single, large birth hospital and additional community based recruitment in each site (Shanghai, Cincinnati, Mexico City)

ID		Study description & participants				
#	First author, year	Study design	Nested in trial / intervention	Country	Urban / Rural	Participants
46	Richard, 2013	Pooled analysis of 7 cohort studies	No	Peru, Brazil, Guinea-Bissau, and Bangladesh	Both	Infants enrolled before 3 months of age with data on diarrhoea for at least 1 year (no gaps longer than 60 days) and at least 4 anthropometric measurements.
47	Peterson, 2013	Prospective birth cohort	No	Bangladesh, Peru	Urban	Children from impoverished communities in Dhaka (BG) and Iquitos (Peru)
48	Lee, 2013	Prospective cohort	No	Peru	Peri-urban	Children in an open-cohort community-based study of children aged 0-72 months in a semi-rural community of the Peruvian Amazon between 2002 and 2006.
49	Kwok, 2013	Prospective birth cohort	No	Hong Kong	Urban	Infants born between 1 April to 31 May 1997 in Hong Kong
50	Garza, 2013	Combined longitudinal study (0-24 months) and cross-sectional study (18-71 months)	No	United States, Oman, Norway, Brazil, Ghana, India	Urban	Infants born in six WHO MGRS sites to parents who did not experience any environmental and socioeconomic conditions likely to constrain growth
51	Fairley, 2013	Prospective birth cohort	No	United Kingdom	Urban	Infants born to women recruited between 2007 and 2010 in Bradford Royal Infirmary
52	Durmus, 2013	Population-based prospective birth cohort	No	The Netherlands	Urban	Infants born to women in Rotterdam who gave birth between April 2002 and January 2006
53	Addo, 2013	Pooled analysis of 5 cohort studies	Yes	Brazil, Guatemala (trial), India, the Philippines, South Africa	Both	Participants in 5 cohorts (Pelotas-Brazil 1982, INTCS-Guatemala 1969-77, New Delhi Birth Cohort-India 1969-72, CLHNS Cebu-Philippines 1983-4, Birth to Twenty-South Africa, 1990
54	Silva, 2012	Population-based prospective birth cohort	No	The Netherlands	Urban	Infants born to women in Rotterdam who gave birth between April 2002 and January 2006
55	Saha, 2012	Prospective birth cohort	Yes	Bangladesh	Rural	Infants born to mothers in the MINIMat factor RCT's intervention group in the ICDDR,B areas between April 2002 and June 2003

ID		Study description & participants				
#	First author, year	Study design	Nested in trial / intervention	Country	Urban / Rural	Participants
56	Richard, 2012	Pooled analysis of 8 cohort studies	Yes	Peru, Brazil, Guinea-Bissau, India, and Bangladesh	Both	Infants from 8 cohort studies, with at least one complete set of LAZ and WLZ measurements in the age groups 0-5, 6-11, 12-17, and 18-23 months.
57	Queiroz, 2012	Prospective birth cohort	No	Brazil	Rural	Normal birth weight (>2500g), term (>37 weeks GA), singleton infants born to women in Laje and Mutuipe municipalities in the Reconcavo Sul region, Bahia, between March 2005 and October 2006, at the two public maternity units in the areas
58	Matijasevich, 2012	Prospective birth cohort	No	Brazil	Urban	Live infants born to women in Pelotas, Brazil in five maternity hospitals in 2004
59	Martinez-Mesa, 2012	Prospective birth cohort	No	Brazil	Urban	Live infants born to women in Pelotas, Brazil in maternity hospitals in 1993
60	Lourenco, 2012	Follow-up study of a cross-sectional survey	No	Brazil	Peri-urban	Children aged 0-10 years in Acrelandia who were follow-up in 2007 and 2009 after participation in a cross-sectional survey of children <5 years in 2003
61	Kang Sim, 2012	Follow-up study of double-blind RCT of iron supplementation between 6-12 months	Yes	Chile	Urban	Infants with birthweights >3kg who completed the trial
62	Husain, 2012	Prospective birth cohort	No	United Kingdom	Urban	Women of Pakistani origin presenting to Central Manchester Hospital and East Lancashire Hospital for antenatal check-ups without any severe post-partum mental health disorders, and their live, term singleton infants without any severe diseases.
63	Hambridge, 2012	Observational secondary analysis of an RCT	Yes	Guatemala	Rural	Convenience sample of apparently healthy infants in San Juan Comalapa in the western highlands of predominantly Mayan descent who were still being breastfed at 6 months, in an RCT of low-phytate maize as a complementary food from 6-12 months

ID		Study description & participants				
#	First author, year	Study design	Nested in trial / intervention	Country	Urban / Rural	Participants
64	Garced, 2012	Prospective birth cohort	No	Mexico	Urban	Children born to women of reproductive age with no serious renal illness in four municipalities in Morelos, Mexico between January 2001 and June 2005.
65	Bork, 2012	Prospective open cohort	No	Senegal	Rural	Children 6-36 months of age in a sample of 615 extended families in a demographic surveillance site in the Sine region who were followed-up at 2 time points 6 months apart
66	Matijasevich, 2011	Prospective birth cohort	No	Brazil	Urban	Live infants born to women in Pelotas, Brazil in maternity hospitals in 1993 and five maternity hospitals in 2004
67	Durmus, 2011	Population-based prospective birth cohort	No	The Netherlands	Urban	Infants born to women in Rotterdam who gave birth between April 2002 and January 2006
68	Deierlein, 2011	Prospective birth cohort	No	United States	Urban	Women recruited at a maternity facility in North Carolina in 2003
69	De Hoog, 2011	Population-based prospective birth cohort	No	The Netherlands	Urban	Infants born to women who presented at an obstetric caregiver at the first ANC visit in Amsterdam between January 2003 and March 2004.
70	Moore, 2010	Prospective birth cohort	No	Brazil	Urban	Infants born in a 5-block area of a shantytown, Gonsalves Dias, in Fortaleza, Brazil
71	Ertel, 2010	Prospective birth cohort	No	United States	Urban	Mother-child pairs enrolled between 1999 and 2002 in Boston, Massachusetts from Atrius Health <22 weeks gestation.
72	de Beer, 2010	Population-based prospective birth cohort	No	The Netherlands	Urban	Infants born to women who presented at an obstetric caregiver at the first ANC visit in Amsterdam between January 2003 and March 2004.
73	Andersen, 2010	Population-based prospective birth cohort	No	Denmark	Both	Women recruited from all over Denmark at the first ANC visit to their GP between 1996 and 2002.
74	Le Beaud, 2015	Prospective birth cohort	No	Kenya	Rural	Offspring of healthy (no severe illness, disability or anaemia) women who gave birth to infants born at 37+ weeks gestation, and received prenatal or postnatal care at Msambweni District Hospital

ID		Study description & participants				
#	First author, year	Study design	Nested in trial / intervention	Country	Urban / Rural	Participants
75	Katulla, 2014	Prospective birth cohort	No	India	Urban slum	Pregnant women identified between March 2009 and May 2010 in four geographically adjacent semiurban slum areas of Vellore, Tamil Nadu, who gave birth to infants with birthweight >1500g and no major congenital malformations.
76	Howe, 2012	Prospective birth cohort	No	United Kingdom	Both	Infants of women who gave birth between April 1991 and December 1992 in one of three Bristol-based health districts
77	Johnson, 2012	Prospective study nested within a study to test the efficacy of an integrated feeding and care intervention among 3-16 month old infants in rural India	Yes	India	Rural	All infants born in three ICDS project areas covering 60 villages between September 2005 and April 2007.

ID		Study description & participants			
#	First author, year	Timing of recruitment	Frequency of growth measurement	Age group for follow-up (months)	Expected number of growth measurements per participant
1	Syed, 2018	Within 14 days of birth	Monthly	0-18	18
2	Steiner, 2018	Pregnant women in surveillance sites identified in second trimester of pregnancy, and infants enrolled within 7 days of birth after medical examination.	Every 3 months	0-24	9
3	Schnee, 2018	Within 7 days of birth	At enrolment, and 12, 24, 40, 52, and 104 weeks of age	0-24	6
4	Sanin, 2018	Within 17 days of birth	At enrollement, and monthly thereafter	0-24 months	24
5	Moradi, 2018	In late infancy	At birth, 2, 4 months	0-4 months	3

ID		Study description & participants			
#	First author, year	Timing of recruitment	Frequency of growth measurement	Age group for follow-up (months)	Expected number of growth measurements per participant
6	Lima, 2018	Within 17 days of birth	Monthly	0-24months	24
7	Kramer, 2018	At birth	Birth, 1, 2, 3, 6, 9, 12 months	0-12 months	7
8	Islam, 2018	Within 17 days of birth	Monthly	0-24 months	24
9	Garzon, 2018	With 28 days of birth	At enrolment, and 3, 6, 9, 12, 16, 18, and 24 months.	0-24 months	8
10	Devakumar, 2018	Antenatal, before 20 weeks of gestation	At birth, and then 2.5 years.	0 to 8.5 years	2
11	Cheng, 2018	At first Maternal and Child Health Centre visit, shortly after birth	3, 9 and 36 months	0 to 16 years	3
12	Admassu, 2018	Within 48 hours of birth	At birth, and 1.5, 2.5, 3.5, 4.5, 6, 12, 18, 24, 36, 48, 60 months.	0-60 months	12
13	Zhang, 2017	Within 72 hours of birth	Every 3 months	0-24 months	8
14	Matos, 2017	Within 14 days of birth	At birth/14 days, and 7, 13, 24, 36, and 60 months	0-60 months	6
15	MAL-ED Network Investigators/ Caulfield, 2017	Within 17 days of birth	At enrolment, and monthly thereafter	0-24 months	24
16	Clemente, 2017	First trimester of pregnancy	At birth, 6 months and 12 months	Non-specific	3
17	Bork, 2017	At 2 months	At 2-3, 4-5, 6-7, 9-10, 13-23, 18-28, 23-33, 29-39 months	2-39 months	8
18	Bell, 2017	<6 weeks	At 1, 4 and 7 months	1-7 months	3
19	Swithowski, 2017	<22 weeks gestation	At birth, infancy, early childhood, or mid-childhood.	0-120 months	4
20	Svefors, 2016	Pregnancy	At birth, monthly up 12 months, every 3 months up to 2 years, then at 4.5 and 10 years.	0-120 months	19
21	Owais, 2016	In the 7th month of pregnancy	At 3, 9, 16 and 24 months	0-24 months	4

ID		Study description & participants			
#	First author, year	Timing of recruitment	Frequency of growth measurement	Age group for follow-up (months)	Expected number of growth measurements per participant
22	Nagata, 2016	At birth	Every 2 months	0-24 months	12
23	Kavle, 2016	Last trimester of pregnancy	At 0, 4, 6, 8, 12 months	0-12 months	13
24	Griffiths, 2016	At 2-3 months	Monthly	3-12 months	9
25	Gough, 2016	Within 96 hours after birth	At birth, 6 weeks, and every three months after	0-24 months	9
26	De Beaudrap, 2016	at 13+ weeks of gestation	Monthly	0-12 months	12
27	Busert, 2016	Before 5th birthday	At baseline (0-59 months), and after 9 months of follow up (9-69 months), and after 29 months (29-89 months)	0-89 months	3
28	Broere-Brown, 2016	In early pregnancy (<18 weeks gestation)	At 1.1, 2.2, 3.3, 4.4, 6.2, 11.1, 14.3, 18.3, and 24.8 months	0-28 months	9
29	Bhargava, 2016	In pregnancy	At birth, then every 2 months until 24 months	0-24 months	13
30	Alkhalawi, 2016	In pregnancy	At birth, then at four mean ages of 1.2, 3.6, 6.6, and 11.88 months.	0-12 months	5
31	Wright, 2015	In pregnancy	At birth, then every 2 months until 24 months	0-24 months	13
32	Vail, 2015	In pregnancy	Birth, 3 months, and 12 months	0-12 months	3
33	Rogawski, 2015	In pregnancy	At birth, and then every month up to 36 months.	0-36 months	37
34	O'Keeffe, 2015	In pregnancy	At birth, 6 weeks, 10, 21, and 48 months of age, and annually from 7-10 years	Birth onwards	9
35	Hanieh, 2015	In pregnancy	At birth, 6 weeks, and 6 months	0-6 months	3
36	Costet, 2015	In pregnancy	At 3, 7, and 18 months. Additional record linkage to health data between 7 and 18 months	0-18 months	3
37	Richard, 2014	At birth	Before 2 months, and at least 4 times between 2-24 months.	0-24 months	5

ID		Study description & participants			
#	First author, year	Timing of recruitment	Frequency of growth measurement	Age group for follow-up (months)	Expected number of growth measurements per participant
38	Patel, 2014	At birth	Birth, 1, 2, 3, 6, 9, 12 months; then at 6.5 years (with record linkage in between 12 mo. and 6.5 years)	0-84 months	8
39	Padanou, 2014	In pregnancy	At birth, 1, 2, 3, 4, 5, 6, 9, 12, 15, 18 months	0-18 months	11
40	Murasko, 2014	At 9 months	At 9 months, 24 months, and 4 and 5 years	9-60 months	4
41	Mallard, 2014	At 6 months	At 6, 9, 12, 15, 18 months	6-18 months	5
42	Jaganath, 2014	In pregnancy	At birth, weekly until 3 months, twice per month until 1 year, then monthly up to 24 months	0-24 months	43
43	Hong, 2014	In pregnancy	At birth, 6, 12, 18, 24, and 36 months	0-36 months	6
44	Betoko, 2014	<24 weeks gestation	At birth, 1, 2, 3, 4 months	0-4 months	5
45	Woo, 2013	2 weeks after birth	Five times between enrolment and 12 months	0-12 months	5
46	Richard, 2013	At birth	Before 3 months, and at least 4 times between 2-24 months.	0-24 months	5
47	Peterson, 2013	At birth	Every three months	0-24 months	8
48	Lee, 2013	< 70 months	Every month	0-72 months	Variable
49	Kwok, 2013	At first Maternal and Child Health Centre visit, shortly after birth	3, 9 and 36 months	0 to 16 years	3
50	Garza, 2013	At birth	At birth, at weeks 1,2,4 and 6; monthly from 2 to 12 months, and bi-monthly from 12-24 months.	0-71 months	21
51	Fairley, 2013	26-28 weeks gestation	At birth, 6, 12, 18 and 24 months of age	Birth onwards	5
52	Durmus, 2013	In early pregnancy (<18 weeks gestation)	At 3, 6, 12, 24, 36, 48 months	Birth onwards	6
53	Addo, 2013	Pregnancy or birth (site-specific)	At birth, 2 years, mid childhood, and adulthood	0-19 years	4

ID		Study description & participants			
#	First author, year	Timing of recruitment	Frequency of growth measurement	Age group for follow-up (months)	Expected number of growth measurements per participant
54	Silva, 2012	In early pregnancy (<18 weeks gestation)	At birth, 1, 2, 3, 4, 6, 11, 18 and 24 months	Birth onwards	9
55	Saha, 2012	Pregnancy	At birth, monthly up 12 months, every 3 months up to 2 years, then at 4.5 and 10 years.	0-120 months	19
56	Richard, 2012	At birth	Variable across four periods: 0-5, 6-11, 12-17 and 18-23 months	0-24 months	4
57	Queiroz, 2012	At birth	At birth, 1, 2, 3, 4, 5, 6, 12 months.	0-12 months	8
58	Matijasevich, 2012	At birth	At birth, 3, 12, 24, and 48 months	Birth onwards	5
59	Martinez-Mesa, 2012	At birth	At birth, 1, 3, 6 months, 1, 4, 11, 15 years	Birth onwards	8
60	Lourenco, 2012	Before 5th birthday	At baseline (0-59 months), and after 4 years and 6 years of follow-up	0-120 months	3
61	Kang Sim, 2012	At birth	At birth, and monthly until 12 months.	0-120 months	13
62	Husain, 2012	In pregnancy	At birth, 3, and 6 months	0-6 months	3
63	Hambridge, 2012	At 6 months	At 6 months and 12 months	6-12 months	2
64	Garced, 2012	Before pregnancy	At birth, 1, 3,6, and 12 months	0-12 months	5
65	Bork, 2012	Between 6-36 months	At recruitment, and after 6 months	6-36 months	2
66	Matijasevich, 2011	At birth	At birth, 3, 12, 48 months (1993 cohort); at birth, 3, 12, 24, 48 months (2004 cohort)	Birth onwards	4
67	Durmus, 2011	In early pregnancy (<18 weeks gestation)	At 3, 6, 12, 24, 36, 48 months	Birth onwards	6
68	Deierlein, 2011	In pregnancy	At 6 months	0-6	1
69	De Hoog, 2011	First trimester of pregnancy	At 1 month and 6 months	Birth onwards	2

ID		Study description & participants			
#	First author, year	Timing of recruitment	Frequency of growth measurement	Age group for follow-up (months)	Expected number of growth measurements per participant
70	Moore, 2010	In pregnancy	Every three months	0-120 months	Unclear
71	Ertel, 2010	<22 weeks gestation	At birth, 6 months, 1, 2, and 3 years	0-36 months	5
72	de Beer, 2010	First trimester of pregnancy	At 1 month and 14 months	Birth onwards	2
73	Andersen, 2010	In early pregnancy	At 5 months and 12 months	Birth onwards	2
74	Le Beaud, 2015	In pregnancy	Every three months	0-36 months	12
75	Katulla, 2014	In pregnancy	At birth, and then every month up to 24 months.	0-24 months	25
76	Howe, 2012	In pregnancy	At birth, 6 weeks, 10, 21, and 48 months of age, and annually from 7-10 years	Birth onwards	9
77	Johnson, 2012	At 2-3 months	Monthly	3-12 months	9

ID		Analysis					
#	First author, year	Age range in analysis	Analysis sample	Growth used as	Standardization	Level of estimation	Metric type
1	Syed, 2018	0-18 months	All children with exposure data	Outcome	Standardized	Group	Continuous
2	Steiner, 2018	0-24 months	(1) Urban site, all children irrespective of LAZ at baseline (2) Rural site, all children irrespective of LAZ at baseline	Outcome	Standardized	Group	Continuous
3	Schnee, 2018	0-24 months	All children with valid exposure and outcome data	Both	Standardized	Group	Continuous
4	Sanin, 2018	9-24 months	All children with complete information	Outcome	Standardized	Group	Categorical
5	Moradi, 2018	0-4 months	Singleton infants, no maternal smoking, no maternal or infant disease, no missing data, maternal calorie intake between 800-4200 kcal	Outcome	Raw	Group	Continuous

ID		Analysis					
#	First author, year	Age range in analysis	Analysis sample	Growth used as	Standardization	Level of estimation	Metric type
6	Lima, 2018	0-6 months	All children with 90% or more active surveillance between 0-6 months, and complete data	Outcome	Standardized	Group	Continuous
7	Kramer, 2018	0-12 months	All infants with valid data.	Outcome	Standardized	Group	Continuous
8	Islam, 2018	9-24 months	All children with complete information	Outcome	Standardized	Group	Categorical
9	Garzon, 2018	0-24 months	All children followed up until the end of the study	Outcome	Standardized	Group	Both
10	Devakumar, 2018	0-30 months	All children successfully followed up at 2.5 years	Outcome	Standardized	Individual	Both
11	Cheng, 2018	0-36 months	All singleton infants with breastfeeding status and at least one LAZ measurement and BF status. (Missing confounders imputed)	Outcome	Standardized	Group	Continuous
12	Admassu, 2018	0-60 months	All children with 2+ body composition measurements between 0-6 months, height measured at least once, and no missing data for covariates.	Outcome	Raw	Group	Continuous
13	Zhang, 2017	0-24 months	All children with 5+ growth measurements	Outcome	Standardized	Group	Continuous
14	Matos, 2017	0-60 months	All children with complete covariate data	Outcome	Standardized	Group	Continuous
15	MAL-ED Network Investigators / Caulfield, 2017	0-24 months	All children with at least one growth measurement, dietary assessment, and stool sample in each time period (0-2, 3-5, 6-8, 9-11, 12-17, 18-24 months).	Outcome	Raw	Group	Continuous
16	Clemente, 2017	0-12 months	All singleton live-born infants from 3 of 7 regions	Outcome	Standardized	Group	Continuous

ID		Analysis					
#	First author, year	Age range in analysis	Analysis sample	Growth used as	Standardization	Level of estimation	Metric type
17	Bork, 2017	2-39 months	For anthropometry, all children in the larger study nested in the trial between 1990 and 1997, for IYCF, a subset born between Jan to Oct 1995 followed up at home.	Outcome	Standardized	Group	Continuous
18	Bell, 2017	1-7 months	Length data at 7 months and at least 2 data points recording feeding	Outcome	Standardized	Group	Continuous
19	Swithowski, 2017	0-120 months	Children with at least one post-baseline follow-up measurement.	Outcome	Raw	Both	Continuous
20	Svefors, 2016	0-120 months	Children who completed follow up from birth to 10 years	Outcome	Standardized	Group	Continuous
21	Owais, 2016	0-24 months	Children with complete data	Outcome	Standardized	Group	Continuous
22	Nagata, 2016	0-24 months	All children with complete data	Outcome	Standardized	Group	Continuous
23	Kavle, 2016	0-12 months	All infants with complete anthropometric data	Outcome	Standardized	Group	Both
24	Griffiths, 2016	0-12 months	All infants with complete anthropometric data	Outcome	Standardized	Group	Continuous
25	Gough, 2016	0-24 months	Infants who were not administratively censored at 12 months	Outcome	Standardized	Group	Continuous
26	De Beaudrap, 2016	0-12 months	Live-born singletons with a valid ultrasound assessment of gestational age, and data on anthropometry and gender	Outcome	Raw	Group	Continuous
27	Busert, 2016	0-89 months	All children who had complete data for the second follow-up visit	Outcome	Raw	Group	Continuous
28	Broere-Brown, 2016	0-28 months	Live singleton births with biometrical data from pregnancy	Outcome	Standardized	Group	Continuous
29	Bhargava, 2016	2-24 months	Participants followed up in 11 periods	Outcome	Raw	Group	Continuous

ID		Analysis					
#	First author, year	Age range in analysis	Analysis sample	Growth used as	Standardization	Level of estimation	Metric type
30	Alkhalawi, 2016	0-12 months	Live singleton births at 37+ weeks gestation, with no pregnancy or birth complications, and <4th pregnancy of the mother, and an APGAR score of 8+.	Outcome	Raw	Group	Continuous
31	Wright, 2015	6-24 months	Participants with complete and plausible data for at least one of the 10 survey points.	Outcome	Standardized	Group	Continuous
32	Vail, 2015	0-12 months	Singleton infants born at 36+ weeks' gestation, with data on age at weaning between 3-7 months, and complete anthropometric data.	Outcome	Standardized	Group	Continuous
33	Rogawski, 2015	0-36 months	All participants with valid data	Outcome	Standardized	Group	Continuous
34	O'Keeffe, 2015	0-120 months	Participants with complete information on exposures at both time points, and infants born after 23 weeks gestation, and complete birth anthropometry and confounder data	Outcome	Raw	Individual	Continuous
35	Hanieh, 2015	0-6 months	Infants with LAZ data available	Outcome	Standardized	Group	Continuous
36	Costet, 2015	0-18 months	Life, singleton, term, healthy infants born to women who agreed to additional home visits.	Outcome	Raw	Both	Continuous
37	Richard, 2014	0-24 months	Children with complete data as per inclusion criteria	Outcome	Raw	Both	Continuous
38	Patel, 2014	0-84 months	Children who completed follow up at 6.5 years	Outcome	Raw	Individual	Continuous
39	Padanou, 2014	0-18 months	All children who completed follow-up	Outcome	Standardized	Group	Continuous
40	Murasko, 2014	0-24 months	Children with available data	Outcome	Raw	Individual	Continuous

ID		Analysis					
#	First author, year	Age range in analysis	Analysis sample	Growth used as	Standardization	Level of estimation	Metric type
41	Mallard, 2014	6-18 months	Children with complete data	Outcome	Standardized	Group	Continuous
42	Jaganath, 2014	0-24 months	Children with >500 days follow-up, and any report of h. pylori infection before 24 months, and available anthropometry.	Outcome	Raw	Group	Continuous
43	Hong, 2014	0-36 months	Live, singleton, term infants born without any congenital malformations	Outcome	Raw	Group	Continuous
44	Betoko, 2014	0-12 months	Infants with complete data	Outcome	Raw	Individual	Continuous
45	Woo, 2013	0-12 months	Participants with complete data, who restricted introduction of certain foods.	Outcome	Standardized	Group	Continuous
46	Richard, 2013	0-24 months	Children with complete data as per inclusion criteria and plausible length measurements (<2.5cm difference between adjacent measurements)	Outcome	Standardized	Individual	Continuous
47	Peterson, 2013	0-24 months	Children with REG1B data available	Outcome	Standardized	Group	Continuous
48	Lee, 2013	0-72 months	All participants with valid data	Outcome	Standardized	Individual	Continuous
49	Kwok, 2013	3-9 months	Participants with data during the scheduled visits at 3 and 9 months	Outcome	Standardized	Group	Continuous
50	Garza, 2013	0-24 months	Children in the longitudinal component of the MGRS	Outcome	Raw	Both	Continuous
51	Fairley, 2013	0-24 months	Live, singleton births among those who participated in sub-study for follow-up in infancy.	Outcome	Raw	Individual	Continuous
52	Durmus, 2013	0-48 months	Live, singleton births with complete data	Outcome	Standardized	Group	Continuous
53	Addo, 2013	0-24 months	All participants with valid data	Outcome	Standardized	Individual	Continuous
54	Silva, 2012	0-24 months	Live, singleton infants born to women of Dutch ethnicity, with complete data	Outcome	Standardized	Group	Continuous

ID		Analysis					
#	First author, year	Age range in analysis	Analysis sample	Growth used as	Standardization	Level of estimation	Metric type
55	Saha, 2012	0-24 months	All infants born before the end of December 2003 with Urinary Arsenic (U-A) measurements at 18 months	Outcome	Standardized	Group	Both
56	Richard, 2012	0-24 months	Children with complete data as per inclusion criteria	Outcome	Standardized	Group	Both
57	Queiroz, 2012	0-12 months	Children who completed follow-up to 1 year	Outcome	Standardized	Individual	Continuous
58	Matijasevich, 2012	0-48 months	Children with data on maternal education and at least two length measurements	Outcome	Raw	Individual	Continuous
59	Martinez-Mesa, 2012	0-12 months	Children with available data on maternal and paternal smoking and anthropometry at 1 year	Outcome	Standardized	Group	Continuous
60	Lourenco, 2012	0-120 months	All children who had complete data for the first visit and at least one of two follow-up visits	Outcome	Standardized	Individual	Continuous
61	Kang Sim, 2012	0-12 months	All children with complete data	Outcome	Raw	Individual	Continuous
62	Husain, 2012	0-6 months	All children with complete data	Outcome	Standardized	Group	Continuous
63	Hambridge, 2012	6-12 months	All children with complete data	Outcome	Standardized	Individual	Both
64	Garced, 2012	0-12 months	Children born to women aged 15+ with no birth complications	Outcome	Standardized	Individual	Continuous
65	Bork, 2012	6-36 months	All children with data for both follow up visits	Outcome	Raw	Individual	Continuous
66	Matijasevich, 2011	0-48 months	Children with data available at each follow-up point	Outcome	Standardized	Group	Continuous
67	Durmus, 2011	0-48 months	Live, singleton infants with data on maternal smoking during pregnancy and at least one postnatal growth characteristic.	Outcome	Standardized	Group	Continuous

ID		Analysis					
#	First author, year	Age range in analysis	Analysis sample	Growth used as	Standardization	Level of estimation	Metric type
68	Deierlein, 2011	0-6 months	Live, singleton, term infants born without any congenital malformations	Outcome	Standardized	Group	Continuous
69	De Hoog, 2011	0-6 months	Live, singleton infants with data on growth and infant feeding	Outcome	Standardized	Group	Continuous
70	Moore, 2010	0-72 months	Children with any anthropometry data and who did not develop persistent diarrhoea before an episode prolonged diarrhoea.	Outcome	Standardized	Group	Continuous
71	Ertel, 2010	0-36 months	Children with data on perinatal and postnatal depression and anthropometry data at 3 years.	Outcome	Standardized	Group	Continuous
72	de Beer, 2010	1-14 months	Live, singleton infants with data on growth and infant feeding	Outcome	Standardized	Group	Categorical
73	Andersen, 2010	0-12 months	Children with anthropometry data at 5 and 12 months	Outcome	Standardized	Group	Continuous
74	Le Beaud, 2015	0-36 months	All children with any data available (0-36 months); and a subset with all data available (0-24 months)	Outcome	Standardized	Group	Continuous
75	Katulla, 2014	0-24 months	Children followed up for two years	Outcome	Standardized	Individual	Continuous
76	Howe, 2012	0-120 months	Participants with maternal education and growth data (at least one measurement in each time period)	Outcome	Raw	Individual	Continuous
77	Johnson, 2012	3-15 months	All infants born with gestational age between 37-41 weeks, with complete data on IYCF between 9-12 months.	Outcome	Raw	Individual	Continuous

ID		Analysis			
#	First author, year	Metric sub-type	Quantity of data to derive metric	Analytical approach to derive metric	Method(s) for determinants analysis
1	Syed, 2018	Incremental rate of change	>2 data points	Linear mixed effects model	Linear mixed effects model; Cox proportional hazards model
2	Steiner, 2018	Incremental change	2 data points	Manual	Step-wise linear regression
3	Schnee, 2018	Incremental change	1 data point	Manual	Multivariable linear regression, Generalized estimating equations (logistic) regression
4	Sanin, 2018	Proportion	1 data point	Manual	Generalized estimating equations (logistic) regression
5	Moradi, 2018	Mean	1 data point	Manual	Logistic regression
6	Lima, 2018	Incremental change	2 data points	Manual	Linear mixed effects model
7	Kramer, 2018	Other	1 data point	Manual	Linear mixed effects regression to conduct intention to treat analysis and observational analysis; instrumental variable analysis
8	Islam, 2018	Proportion	1 data point	Manual	Generalized estimating equations (logistic) regression
9	Garzon, 2018	Velocity z-score	2 data points	Manual	Linear mixed effects regression model
10	Devakumar, 2018	Conditional difference	2 data points	Conditional regression	Linear regression
11	Cheng, 2018	Incremental change	2 data points	Manual	Linear regression; linear mixed effect modelling
12	Admassu, 2018	Mean	>2 data points	Linear mixed effects model	Linear mixed effects models
13	Zhang, 2017	Proportional rate of change	>2 data points	Other	Linear regression
14	Matos, 2017	Mean	>2 data points	Linear mixed effects model	Linear mixed effects model
15	MAL-ED Network Investigators / Caulfield, 2017	Mean	>2 data points	Linear mixed effects model	Linear mixed effects model
16	Clemente, 2017	Proportional change	2 data points	Manual	Linear regression; mediation analysis

ID		Analysis			
#	First author, year	Metric sub-type	Quantity of data to derive metric	Analytical approach to derive metric	Method(s) for determinants analysis
17	Bork, 2017	Instantaneous rate of change	>2 data points	Linear mixed effects model	Linear mixed effects model (Sex differences in growth over 2-39 months); general linear models (for sex differences in mean HAZ by IYCF type)
18	Bell, 2017	Mean	1 data point	Manual	Linear regression
19	Swithowski, 2017	Incremental rate of change	>2 data points	Linear mixed effects model	Linear regression
20	Svefors, 2016	Mean	>2 data points	Linear mixed effects model	Linear mixed effects model; logistic regression
21	Owais, 2016	Mean	>2 data points	Generalized estimating equations	Generalized estimating equations models
22	Nagata, 2016	Mean	1 data point	Manual	Multivariable linear regression
23	Kavle, 2016	Mean	>2 data points	Linear mixed effects model	Linear mixed effects regression; logistic regression
24	Griffiths, 2016	Mean	1 data point	Manual	Structural equation modelling (SEM)
25	Gough, 2016	Mean	>2 data points	Other	Multivariable multinomial logistic regression, corrected for multiple testing with the Bonferroni method.
26	De Beaudrap, 2016	Incremental change	2 data points	Manual	Multivariable linear regression
27	Busert, 2016	Conditional difference	2 data points	Conditional regression	Ordinary least squares regression
28	Broere-Brown, 2016	Mean	>2 data points	Linear mixed effects model	Linear mixed effects regression
29	Bhargava, 2016	Mean	>2 data points	Linear mixed effects model	Dynamic random effects models
30	Alkhalawi, 2016	Mean	>2 data points	Generalized estimating equations	Multivariable and GEE linear regression
31	Wright, 2015	Mean	>2 data points	Linear fixed-effects model	Fixed effects longitudinal regression

ID		Analysis			
#	First author, year	Metric sub-type	Quantity of data to derive metric	Analytical approach to derive metric	Method(s) for determinants analysis
32	Vail, 2015	Mean	1 data point	Manual	Multiple linear regression
33	Rogawski, 2015	Mean	>2 data points	Generalized estimating equations	Generalized estimating equations (GEE)
34	O'Keeffe, 2015	Mean	>2 data points	Linear mixed effects model	Multilevel model
35	Hanieh, 2015	Mean	1 data point	Manual	Structural equation modelling (SEM)
36	Costet, 2015	Instantaneous rate of change	>2 data points	Pre-designed structural model	Generalized linear models (GLM)
37	Richard, 2014	Incremental rate of change	>2 data points	Non-linear mixed effects model	Non-linear mixed effects models
38	Patel, 2014	Incremental rate of change	>2 data points	Linear mixed effects model	Multilevel regression model
39	Padanou, 2014	Mean	>2 data points	Linear mixed effects model	Multivariable linear mixed effects regression model
40	Murasko, 2014	Incremental rate of change	>2 data points	Linear mixed effects model	Multivariable linear mixed effects regression model
41	Mallard, 2014	Mean	1 data point	Manual	Multiple linear regression; seemingly unrelated regression
42	Jaganath, 2014	Mean	>2 data points	Linear mixed effects model	Multivariable linear mixed effects regression model
43	Hong, 2014	Mean	1 data point	Manual	Mixed models (unclear)
44	Betoko, 2014	Incremental change	>2 data points	Pre-designed structural model	Multiple linear regression
45	Woo, 2013	Mean	1 data point	Manual	Multiple linear regression
46	Richard, 2013	Mean	>2 data points	Non-linear mixed effects model	Non-linear mixed effects models
47	Peterson, 2013	Mean	>2 data points	Linear mixed effects model	Linear mixed effects regression

ID		Analysis			
#	First author, year	Metric sub-type	Quantity of data to derive metric	Analytical approach to derive metric	Method(s) for determinants analysis
48	Lee, 2013	Incremental change	>2 data points	Linear mixed effects model	Linear mixed effects regression
49	Kwok, 2013	Incremental change	>2 data points	Generalized estimating equations	Generalized estimating equations
50	Garza, 2013	Mean	>2 data points	Other	Repeated measures analysis of variance using generalized linear models
51	Fairley, 2013	Incremental rate of change	>2 data points	Linear mixed effects model	Linear mixed effects model
52	Durmus, 2013	Mean	1 data point	Manual	Linear regression
53	Addo, 2013	Conditional difference	2 data points	Conditional regression	Linear regression; GEE with robust error variances
54	Silva, 2012	Incremental rate of change	>2 data points	Linear mixed effects model	Linear mixed effects model
55	Saha, 2012	Mean	1 data point	Manual	Linear regression model
56	Richard, 2012	Mean	1 data point	Manual	Mixed effects model to include random effect for study site
57	Queiroz, 2012	Mean	>2 data points	Linear mixed effects model	Multivariable mixed effects analysis
58	Matijasevich, 2012	Incremental rate of change	>2 data points	Linear mixed effects model	Linear mixed effects model
59	Martinez-Mesa, 2012	Mean	1 data point	Manual	Multivariable linear regression
60	Lourenco, 2012	Mean	>2 data points	Non-linear mixed effects model	Non-linear mixed effects models
61	Kang Sim, 2012	Incremental rate of change	2 data points	Manual	Path analysis
62	Husain, 2012	Mean	1 data point	Manual	Multiple regression analysis
63	Hambridge, 2012	Incremental rate of change	2 data points	Manual	Linear and logistic regression analyses

ID		Analysis			
#	First author, year	Metric sub-type	Quantity of data to derive metric	Analytical approach to derive metric	Method(s) for determinants analysis
64	Garced, 2012	Mean	>2 data points	Linear mixed effects model	Linear mixed effects model
65	Bork, 2012	Incremental rate of change	2 data points	Linear mixed effects model	Linear mixed effects model
66	Matijasevich, 2011	Mean	1 data point	Manual	Multiple linear regression
67	Durmus, 2011	Mean	>2 data points	Linear mixed effects model	Linear mixed effects model
68	Deierlein, 2011	Mean	1 data point	Manual	Multivariable linear regression
69	De Hoog, 2011	Incremental change	2 data points	Manual	Linear regression model
70	Moore, 2010	Mean	1 data point	Manual	Paired t test
71	Ertel, 2010	Incremental change	2 data points	Linear mixed effects model	Multivariable linear regression, linear mixed effects model
72	de Beer, 2010	Class	2 data points	Threshold/ cut-off	Logistic regression
73	Andersen, 2010	Mean	1 data point	Manual	Multivariable linear regression
74	Le Beaud, 2015	Mean	>2 data points	Generalized estimating equations	Generalized estimating equations
75	Katulla, 2014	Incremental rate of change	>2 data points	Manual	Multivariable linear regression
76	Howe, 2012	Incremental rate of change	>2 data points	Linear mixed effects model	Linear mixed effects model
77	Johnson, 2012	Mean	>2 data points	Pre-designed structural model	Mixed effects general regression models

ID		Analysis	
#	First author, year	Main exposure(s) and timing of measurement	Definition(s) of outcome (effect estimate)
1	Syed, 2018	Flagellin-specific and bacterial lipopolysaccharide (LPS)-specific immunoglobulins A and G at 6 months and 9 months (4 exposures at each time), using quartiles as well as continuous measures of biomarkers	(1) Rate of growth (LAZ) change per year derived from a linear mixed effect model of growth from 0-18 months (Beta and SD) (2) Risk of stunting (event) between 6-18 months, as Hazard Ratio (95% CI)
2	Steiner, 2018	Cryptosporidium in routine stool sample (monthly) and during episodes of diarrhoea (incident) at least 65 days apart. Cryptosporidium infection phenotype (diarrhoeal or subclinical) from a positive stool categorized according to whether stool sample was obtained routinely or for a diarrhoeal episode. Groups compared: 0, 1 and 2+ detected infections between 0-24 months.	Change in LAZ (Delta LAZ) between enrolment and 24 months.
3	Schnee, 2018	(1) Number of total diarrhoeal episodes or days of diarrhoea between birth and 12 months (2) LAZ at the beginning of a time window	(1) LAZ at 12 months (LAZ) (2) Any pathogen-attributable diarrhoea in the window between length measurements
4	Sanin, 2018	Micronutrient Adequacy Ratio (%MAR) based on 13 micronutrients at 9-12, 15-18, and 21-24 months.	Development of stunting between 12-24 months (summarized at 12, 15, and 24 months), as an adjusted OR (95% CI)
5	Moradi, 2018	Dietary energy density measured in late infancy (quartiles)	Infant length at birth, 2 and 4 months.
6	Lima, 2018	Cumulative infection between 0-6 months, in 7 categories: no infection; any E.coli; E.coli + 1 co-infection; E.coli + 2 co-infections; E.coli + 3 co-infections; < 3 non-E.coli infections, 3+ non-E.coli infections	Change in LAZ (cumulative delta LAZ) between enrolment and 6 months
7	Kramer, 2018	Breastfeeding at each follow-up time (0, 1, 2, 3, 6, 9, 12 months) measured as (1) randomized allocation to intervention vs control, (2) as fed, based on observed duration (3) predicted probability of breastfeeding using randomization an instrumental variable	Group differences in LAZ of infants at birth, 1, 2, 3, 6, 9, 12 months in cluster-adjusted and fully adjusted models. Same outcome definition applied in all 3 analytical approaches.
8	Islam, 2018	Dietary Diversity Score (DDS) and Minimum Dietary Diversity (MDD) from 24-hr recall combined within age ranges, representing intake at 9-12, 15-18, and 21-24 months.	Stunting at 12, 18, and 24 months of age (adjusted OR and 95% CI)

ID		Analysis	
#	**First author, year**	**Main exposure(s) and timing of measurement**	**Definition(s) of outcome (effect estimate)**
9	Garzon, 2018	Enteric pathogenic parasites (Giardia lamblia, Cryptosporidium spp, soil-transmitted helminth (STH) infections)	Attained LAZ at 24 months (LAZ); age and sex-specific Length-for-Age Velocity Z-scores (LAVZ) for two-month intervals using methods suggested by WHO.
10	Devakumar, 2018	SES (measured as asset score, maternal education, land ownership) measured before birth	At 2.5 years (1) HAZ; (2) Stunting (OR, 95% CI) (3) conditional height measured by change in growth from that expected for the child based on previous measures, after accounting for regression to the mean (+ve = faster than expected).
11	Cheng, 2018	EBF for 3+ months; mixed or partial BF; never breastfed. Most recent response used when multiple available or missing data.	Change in LAZ between 3-9 months and 9-36 months.
12	Admassu, 2018	Standardized FFM accretion (0-6 months) and FM accretion (0-4 months) rates	Length at 1 year (cm); length gain accumulated from age 1 to 5 years (cm)
13	Zhang, 2017	Non-specific, but most measured at birth or up to 6 months.	Growth faltering (score) calculated as a deviation of infant HAZ from the WHO Growth Curves, derived using a Functional Principal Components score showing modes of temporal variation to fit study data 'curves' as well as reference 'curves' for WHO standards. 'Strata' of FPC scores were also derived.
14	Matos, 2017	Infant sex and ethnicity (birth)	Differences in HAZ trajectories between groups (mean, SD)
15	MAL-ED Network Investigators / Caulfield, 2017	Breastfeeding (% days of full (EBF or PBF) breastfeeding from 0 to 5 months) in 10th (Low) and 90th (High) groups; Complementary feeding (% days fed animal milks and dairy between 2-8 months, and energy and energy-adjusted protein (by regressing protein against energy in a linear mixed model and using residuals as energy adjusted protein) intakes from non-breastmilk foods from 9-24 months) in 10th (Low) and 90th (High) groups; pathogens in non-diarrhoeal stool in each period; number of untreated (by antibiotics) episodes of diarrhoea in each age period.	Differences from average estimated LAZ for children in the cohort (cm); linear growth velocity (cm/month) rom
16	Clemente, 2017	Prenatal NO2 exposure using ambient concentrations from passive samples residential areas, at any point in the pregnancy, and in each trimester	Change in LAZ between 0-6 months and 0-12 months (%).

ID		Analysis	
#	First author, year	Main exposure(s) and timing of measurement	Definition(s) of outcome (effect estimate)
17	Bork, 2017	Sex (at birth) for growth 2-39 months; IYCF Complementary Feeding at 2-3, 4-5, 6-7, 9-10 months (Meal frequency grouped as 0, 1, 2, 3+ in the last 24 hours, combined with perceived appetite for CF) with for HAZ in each time period.	For exposure = infant sex: Estimated change in HAZ per month between 2-39 months (HAZ) and Height-for-Age Difference (HAD) from WHO age-sex-specific median (cm) at baseline (2 mo.) and overall (2-39 months); For exposure = meal frequency: HAZ in each time period
18	Bell, 2017	BF to 6 months (predominantly BF or EFF)	Infant length at 7 months (LAZ); LAZ trajectories between 1-7 months.
19	Swithowski, 2017	Maternal protein intake <22 weeks gestation from an FFQ	(1) Length at birth, 6 months, 2 years, and mid childhood. (2) Growth trajectories (rate of growth) from birth to mid-childhood
20	Svefors, 2016	Maternal height (tertiles), season of conception (Nov-Feb; Mar-May; Jun-Oct); maternal education level at enrolment (>5 years, 1-5 years, no education)	Mean HAZ over 0-120 months (z-score, 95% CI); odds of stunting at 10 years (OR< 95%CI)
21	Owais, 2016	EBF at 3 months; MAD at 9 months	LAZ over the 9-24 months (Beta coefficient, 95%) using a GEE model with an autoregressive covariance matrix
22	Nagata, 2016	Sociodemographic factors (children <5 years; HH size); HAZ at 1 year; health symptoms (diarrhoea, vomiting, cough, fever in the past week) at 1 year	HAZ at 2 years
23	Kavle, 2016	At 2,4, 6,8,10, 12 months: diarrhoea for 7+ days, fever, exposure to programme; At 4,6,8 and 12 months: MDD. For stunting: decrease in WLZ between two adjacent study visits.	LAZ over 4-12 months (B, 95%CI); stunting at 12 months (OR 95%CI)
24	Griffiths, 2016	SES measured using Standard of Living Index at enrolment	LAZ at 12 months (LAZ)
25	Gough, 2016	maternal age, education (years), MUAC, height; birth length and weight; gestational age; season of birth (month); infant sex;	Four groups describing longitudinal growth trajectories (LAZ), extracted using k-means clustering. Mean LAZ compared across groups; predicted probability of group membership in multivariable logistic regression.
26	De Beaudrap, 2016	Malaria in pregnancy (placental malaria, any, >1 episode), malaria by gestational age (<15 weeks, 15-20 weeks, 20-24 weeks, 24+ weeks).	Change in length (cm) between 0-12 months
27	Busert, 2016	Dietary diversity score (DDS) in the past 7 days before first and second follow-up.	HAD (cm) between baseline and first follow-up, and first and second follow-up.
28	Broere-Brown, 2016	Infant sex	Length (SDS, 95%CI) from 3-24 months

ID		Analysis	
#	First author, year	Main exposure(s) and timing of measurement	Definition(s) of outcome (effect estimate)
29	Bhargava, 2016	Lagged protein/energy intake (g/kcal-day) and calcium/energy intake (mg/kcal-day)	Length (effect in cm, SE) between 2-24 months
30	Alkhalawi, 2016	Perfluorooctanoic acid (PFOA), perfluorooctanesulfonic acid (PFOS), and perfluorohexanesulfonic acid (PFHxS) measured in maternal blood samples at 32 weeks of pregnancy, converted to quartiles of exposure.	Infant length in cm at t0, t1, t2, t3, t4 (B, 95%CI)
31	Wright, 2015	BF in the last 24h (0/1) and CF in the last 24h (0/1) as high or low (4+ groups, <4 groups) at each bimonthly visit from 6-24 months; formed into 4 groups (00, 01, 10, 11) as time-varying covariates	LAZ (B, 95%CI) over 6-24 months, and predicted sex-specific LAZ based on B for LAZ.
32	Vail, 2015	Feeding at 3 months (EBF, EFF, MF); age at weaning (reported at 12 months) in 4 groups (3-3.99, 4-4.99, 5-5.99, 6-6.99).	LAZ at birth, 3 months, and 12 months in separate models.
33	Rogawski, 2015	Antibiotic exposure between 0-6 months (recorded monthly); number of antibiotic courses between 0-6 months	Effect on LAZ between 0-6 months (B, CI) as short term effects; average LAZ (SD) of children each month after 6 months in groups by number of antibiotic courses as long term effects; stunting (RR)
34	O'Keeffe, 2015	Maternal alcohol consumption in pregnancy (index based on measurements in early, mid, and late pregnancy)	Predicted height (cm) at birth and 2 years (mean, SD), and mean difference compared with offspring pregnancy abstainers
35	Hanieh, 2015	Maternal antenatal factors (BMI at enrolment, weight gain during pregnancy, Vitamin D status in late pregnancy, iodine, haemoglobin, ferritin)	LAZ at 6 months (zscore SD, 95%CI)
36	Costet, 2015	Chlorodecone (insecticide) exposure prenatally (at birth) in cord blood, and postnatally in breastmilk (at 3 months), and food (at 7 and 18 months) using environmental food contamination data to estimate exposure.	(1) Four growth parameters of the Jens-Bayley model for 0-18 months (2) Predicted instantaneous growth velocity from JB model, and length (cm) at birth, 3, 8 and 18 months.
37	Richard, 2014	Diarrhoea (3+ liquid or semiliquid stools in a 24 hour period), diarrhoea episodes and duration (0-3, >3-6, >6-12, >12-18, >18-24)	(1) Linear growth (cm, SE) between 0-24 months (mm/month)(2) Length velocity (3) Predicted length (cm) and HAZ at 24 months

ID		Analysis	
#	First author, year	Main exposure(s) and timing of measurement	Definition(s) of outcome (effect estimate)
38	Patel, 2014	Parental education (up to secondary, advanced secondary, university) and occupation (manual, non-manual)	(1) Predicted height at birth, 3, 6, 9 months, 1, 2, 3, 4, 5, 6,6.5, 7 years. (2) Growth velocity between 0-3, 3-12, 12-34, 34-84 months
39	Padanou, 2014	(1) Birth characteristics (LBW, prematurity, IUGR); (2) maternal nutritional status (short stature, low BMI); (3) IYCF practices (BF 0-6 months; MDD, MFF 6-18 months)	HAZ 0-18 months (B, SE)
40	Murasko, 2014	Household income (average real family income over the study period)	Height velocity (cm/year)
41	Mallard, 2014	WHO IYCF indicators at 6 and 12 months (MDD, MMF, MAD, iron-rich)	HAZ at 18 months
42	Jaganath, 2014	(1) Timing of first diarrhoeal infection due to H.pylori (between 6-11 months vs 12-23 months), and (2) average number of episodes and timing of first episode (6-11 months vs 12-23 months).	Height at 24 months (B, 95%CI)
43	Hong, 2014	Maternal serum vitamin (A, C, E, MDA (creatinine), 8-OHdG (creatinine) and urinary oxidative stress levels between 24-38 weeks gestation.	Infant length in cm at birth, 6, 12, 18, 24, and 36 months
44	Betoko, 2014	Formula type (regular or partially hydrolysed)	Change in infant LAZ between birth and 4 months (Delta z-score) as predicted by Jenss-Bayley model fitted to all growth data between 0-12 months.
45	Woo, 2013	Introduction of complementary foods (Month of introduction) and ever/never (0/1)introduction of food groups within the first year)	LAZ at 12 months (LAZ)
46	Richard, 2013	Diarrhoea (3+ liquid or semiliquid stools in a 24 hour period) prevalence (ratio of diarrhoea days to the number of days under surveillance), and incidence (ratio of the number of new episodes to the number of days at risk for a diarrhoeal episode)	Length at 24 months
47	Peterson, 2013	REG1B concentration in stool sample at 3 months	Group difference in LAZ between 6-24 months (B, SE)

ID		Analysis	
#	First author, year	Main exposure(s) and timing of measurement	Definition(s) of outcome (effect estimate)
48	Lee, 2013	Campylobacter infection timing (categorical observed in 1st, 2nd, or 3rd quarter of 9 month follow-up periods) and total incidence of diarrhoea (continuous)	Change in height (cm) over 9 month period
49	Kwok, 2013	(1) Parental education (at recruitment) and grandparent's education as indicators of SEP. (2) Mid-parental height.	Length gain z-score (Mean, 95%CI) between 3-9 months
50	Garza, 2013	Maternal, paternal, and mid-parental height	Proportion of within variability in length at 6, 9, 12, 18, and 24 months and between-child variability at 24 months in repeated measures analysis of variance general linear models (with orthogonal polynomial transformation for repeated measures); children's predicted adult heights.
51	Fairley, 2013	Interaction between sex and ethnicity (White British, Pakistani origin) (4 groups)	Average growth trajectory with four parameters describing birth length and linear growth in three time periods (0-4, 4-9, and 9-24 months)
52	Durmus, 2013	Maternal pre-pregnancy and paternal height, weight and BMI (SD)	Infant length at birth, 3, 6, 12, 24, 36, 48 months.
53	Addo, 2013	Maternal height (cm); maternal short stature (height <150.1cm)	Birth length z-score, attained height at 2 years (z-score), conditional growth 0-2 years (z-scores), stunting at 2 years (prevalence ration, 95%CI)
54	Silva, 2012	Maternal education (as a marker of SES) measured at enrolment (High=university, Mid-high=higher vocational training, Mid-low= 3+ years of secondary school, Low= <3 years of secondary school)	(1) Height SDS at 2, 6, 14, and 25 months (2) Length velocity from 0-2 years (cm/month)
55	Saha, 2012	(1) Maternal urinary arsenic (8 and 30 weeks gestation) and (2) child urinary arsenic (18 months).	(1) Attained length in cm (mean, SE) at 3, 6, 9, 12, 18 and 24 months of age (2) Stunting (OR, 95%CI) at 24 months
56	Richard, 2012	(1) WLZ variability in 0-17 months (SD), (2) Wasting during 0-5, 6-11, 12-17 month age bands	(1) LAZ at 18-24 months (2) LAZ at 18 and 24 months
57	Queiroz, 2012	(1) Environmental variables (WASH index), (2) maternal characteristics (education, age, height, Vitamin A supplementation post-partum), (3) infant characteristics (sex, birthweight >3000g, EBF, diarrhoea, newborn anaemia)	Mean LAZ over 0-12 months (z-score, SE)

ID		Analysis	
#	First author, year	Main exposure(s) and timing of measurement	Definition(s) of outcome (effect estimate)
58	Matijasevich, 2012	Maternal education (as a marker of socioeconomic inequality) measured perinatally (0-4, 5-8, and 9+ years of formal education)	Average growth trajectory with four parameters describing birth length and linear growth in three time periods (0-3, 3-12, 12-32, 32-48 months for girls, and 0-3, 3-12, 12-29, 29-48 months for boys)
59	Martinez-Mesa, 2012	Maternal smoking during pregnancy (never, <10/day, 10-19/day, 20+/day) and partner smoking (Yes/No)	LAZ at birth, 1 year (B, 95%CI)
60	Lourenco, 2012	(1) Socioeconomic characteristics, (2) access to public services, (3) pre-pregnancy maternal characteristics, (4) maternal characteristics during pregnancy, (5) child characteristics at birth, (6) IYCF (7) morbidity	(1) Mean HAZ curve for the population 0-10 years using restricted cubic splines. (2)Mean HAZ values at 6 months, 1, 2, 5,7, 10 years
61	Kang Sim, 2012	(1) SES (low/high) using Graffar Index	Rate of infant length gain 0-12 months (cm/month)
62	Husain, 2012	Maternal depression (perinatally using EPDS and SCAN)	LAZ at 6 months (zscore SD, 95%CI)
63	Hambridge, 2012	Maternal height (cm)	(1) LAZ at 6 and 12 months; (2) Stunting LAZ<-2 at 6 and 12 months, (3) linear growth velocity between 6-12 months (mm/month)
64	Garced, 2012	Prenatal DDE (pesticide) exposure (in serum) in 1st, 2nd, or 3rd trimester	LAZ over 0-12 months (B, 95%CI)
65	Bork, 2012	(1) Infant and Child Feeding Index (0-7) using Food Variety Index (BF, MF, DD, FV), Dietary Diversity Index (0-7), Meal Frequency Index (0-5), with an average of each component across both visits.	(1) Mean HAZ between 6-12, 12-18, 18-24, 24-30, 30-36 months (2) Length/height increase over 6 month period (mean cm, SE)
66	Matijasevich, 2011	Maternal and paternal smoking (1+ cigarette per day) during any trimester of pregnancy	LAZ score (B, 95%CI) at birth, 3, 12, 24 (2004 cohort only) and 48 months
67	Durmus, 2011	Maternal smoking during pregnancy (None, 1st trimester only, continued, 0-4 cigarettes/day, 5+ cigarettes/day)	Length SDS (B, 95%CI) at birth, 3, 6, 12, 24, 36, 48 months
68	Deierlein, 2011	Maternal (1) pre-pregnancy BMI (2) Gestational weight gain (adequacy)	LAZ at 6 months (B, 95%CI)
69	De Hoog, 2011	(1) Duration of breastfeeding (2) age at introduction of formula feeding (none, <1, 1-3, 4-6, and >6 months) (3) age at introduction of complementary food (< 4 months, 4 months, 5 months, >5 months)	Delta SDS between 1 month and 6 months

ID		Analysis	
#	First author, year	Main exposure(s) and timing of measurement	Definition(s) of outcome (effect estimate)
70	Moore, 2010	(1) Acute diarrhoea <7 days, (2) Prolonged diarrhoea 7-14 days, (3) persistent diarrhoea (14+ days)	HAZ 3 months before and 3 months after diarrhoeal episode
71	Ertel, 2010	Maternal antenatal (mean of 28 weeks gestation) and postnatal (approx. 6 months after birth) depression.	(1) HAZ at 3 years (B, 95%CI) (2) change in HAZ from 0-3 years (B, 95%CI)
72	de Beer, 2010	(1) Pre-existing hypertension (2) pregnancy-induced hypertension	Normal or accelerated growth up to 14 months (normal: ΔSDS ⩽ 0.67 v. growth acceleration: ΔSDS > 0.67)
73	Andersen, 2010	First trimester plasma concentrations of PFOS and PFOA	Length at 5 and 12 months
74	Le Beaud, 2015	Cumulative infant parasitic infection (0/1) before the point of length measurement	(1) Cumulative LAZ over 0-36 months (2) Cumulative LAZ at 6, 12, 18, 24, and 36 months
75	Katulla, 2014	(1) Sociodemographic (religion, maternal education, type of family, SES), (2) birth and postnatal characteristics (maternal anaemia in pregnancy, hypertension, diabetes, preterm birth, parity, history of abortion/still birth, duration of BF)	(1) length velocity (cm/month)
76	Howe, 2012	Maternal education (measured at 32 weeks gestation) as a proxy for SES (< O-level, O-level, A-level, or university degree)	Average sex-specific growth trajectory describing birth length, and growth velocity (cm/month) in four time periods (0-3 months, 3-10 months, 10-29 months, and 29-120 months)
77	Johnson, 2012	(1) Maternal education (none, primary, secondary/college) (2) EBF at 3 months (3) Standard of Living Index tertile (4) Energy from complementary food at 9 or 12 months (5) morbidity in past week (time varying covariate (* with age))	Length (cm) (B, SE)

ID		Analysis		
#	First author, year	Covariates/confounders in final model	Mediators	Model selection methods
1	Syed, 2018	Child sex, preterm birth, maternal age, maternal literacy, antibiotic use at 6 or 9 months, and RUTF use (RCT intervention)	N/A	Conceptual diagram of EED pathways, traditional risk factors for growth, or p < 0.10 in univariable analysis
2	Steiner, 2018	Enrolment LAZ, maternal BMI, household income, water source, water treatment, EBF (days).	N/A	P value < 0.1 'at entry'. Also considered maternal education, maternal age, HH size, gestational age, infant sex, open drain near home.
3	Schnee, 2018	(1) Enrolment LAZ, child sex; maternal age, height, education; household monthly income; household crowding (5+), trial arm; presence of flush toilet; routine treatment of drinking water; cement floor in the home; kitchen in the home; duration of EBF; antibiotic treatment of each episode; seasonality (Fourier series) (2) Any pathogen-attributable diarrhoea in the prior window, enrolment LAZ, child sex; maternal age, height, education; household monthly income; household crowding (5+), trial arm; presence of flush toilet; routine treatment of drinking water; cement floor in the home; kitchen in the home; duration of EBF; antibiotic treatment of each episode; seasonality (Fourier series)	N/A	Not mentioned
4	Sanin, 2018	Age; Sex; At 12, 18 and 24 months - current BF, diarrhoea in last 15 days; LBW, birth order; at baseline - toilet with flush, drinking water source, SES from asset score PCA; maternal age, education	N/A	Conceptual diagram of determinants of stunting in LMICs based on published literature; univariable models investigated, all predictors entered simultaneously in final model.
5	Moradi, 2018	Smoking, physical activity, SES, use of iron, folate, multivitamins, shirafza use, duration of BF, maternal pre-pregnancy BMI.	N/A	p-values from univariate analyses
6	Lima, 2018	Child sex, weight at enrolment; household food insecurity; % days EBF; symptoms of ALRI; antibiotic use; Random intercept for study site.	N/A	Biological plausibility

ID		Analysis		
#	First author, year	Covariates/confounders in final model	Mediators	Model selection methods
7	Kramer, 2018	Maternal education, infant sex, region, urban, maternal and paternal height and BMI. Random effect for clustering by polyclinic.	N/A	None - analysis compares three analytical approaches (two experimental - ITT and VI) and one observational. Intention to treat analysis (exp 1), as observed (exp 2), IV analysis (exp 3); all included random effect for cluster.
8	Islam, 2018	Number of days of diarrhoea per month in each interval (9-12, 15-18, 21-24); maternal education (3 groups: none, 5 years, >5 years); improved toilet (flush and pit latrine); household asset index (PCA); maternal age; LAZ at birth; WAZ at birth; drinking water source; proportion of calories from complementary feeding; total days of EBF during first 6 months; average DDS score in each time interval.	N/A	Checking for multicollinearity using VIF; all hypothesised covariates included in final model, unless highly collinear with another factor.
9	Garzon, 2018	Poverty (MPI score); feeding practices (EBF 0/1, BF at 12 and 24 months 0/1, age at CF in months); maternal height; acute diarrhoea; acute respiratory infection; malaria; any infection single infections.	N/A	No mention of conceptual diagram; variables in final models selected based on p-value <0.25 in univariable models.
10	Devakumar, 2018	Trial allocation.	N/A	None. SES variables most distal in hypothesized conceptual model.
11	Cheng, 2018	Maternal active and passive smoking during pregnancy; maternal birthplace; maternal education; maternal age, parity; household income; gestational age; LAZ at 3 months.	N/A	A priori selection of confounders;
12	Admassu, 2018	FM and FFM (kg) at birth; standardized length accretion (0-4 or 0-6 months), sex, birth order, maternal age at delivery, maternal education, HH wealth index at birth; breastfeeding status at 2.5 months; maternal BMI at 2.5 mo. post-partum	N/A	A priori selection of confounders;
13	Zhang, 2017	HAZ at birth, maternal height and weight (at birth), mother with any formal education, family size, monthly family income, duration of EBF, number of diarrhoeal episodes from birth to 6 months, source of drinking water, food coverage practice, strata of FPC score.	N/A	Not mentioned
14	Matos, 2017	Number of children <15 years in the house; area of residence (urban/rural); maternal marital status and ethnicity; number of persons in the house; number of natural children of the mother; monthly family income	N/A	P value <0.2 in multivariable models

ID		Analysis		
#	First author, year	Covariates/confounders in final model	Mediators	Model selection methods
15	MAL-ED Network Investigators / Caulfield, 2017	Study site, sex, length-for-age and weight-for-age at enrolment and WAMI (Water, Assets, Maternal education, and household Income) Index using mean values from data collected at 6, 12, 18 and 24 months.	N/A	(1) clear biological rationale; (2) reduction in the random effects between children; (3) improvement in model fit (based on AIC)
16	Clemente, 2017	Maternal age, ethnicity, education, smoking status, place of residence, pre-pregnancy BMI, parity. Child sex, gestational age, season of birth (4 groups of 3 months each starting Jan), and region.	Birth length; placental mitochondrial DNA (mtDNA)	A priori selection of confounders
17	Bork, 2017	Meal frequency (in IYCF and HAZ in each age interval)	N/A	Not mentioned
18	Bell, 2017	Gestational age, sex, race/ethnicity, maternal BMI at 7 months, insurance type, maternal education (SES), study site, LAZ at birth	N/A	A priori selection of confounders
19	Swithowski, 2017	Maternal age, education, race/ethnicity, parity, height, prepregnancy weight and smoking, household income, paternal height and weight, child sex.	N/A	A priori selection of confounders
20	Svefors, 2016	Maternal parity, age, SES at birth	N/A	A priori selection of confounders based on literature review
21	Owais, 2016	Household SES, maternal age, literacy, and parity, maternal height, infant sex, infant illness (in the 2 weeks prior to each visit)	N/A	Not mentioned
22	Nagata, 2016	Infant sex, WAZ at 1 year	N/A	A priori selection based on literature review; Benjamini-Hochberg procedure to adjust for multiple testing
23	Kavle, 2016	Sex, maternal height, parity, maternal education, birth LAZ.	N/A	A priori selection of exposures and covariates based on literature review and programme objectives

ID		Analysis		
#	First author, year	Covariates/confounders in final model	Mediators	Model selection methods
24	Griffiths, 2016	None	Maternal BMI and height at 2-3 months; infant birth weight; infant LAZ at 6 months	A priori selection of exposure and mediators; covariates excluded if they did not show statistical significance ($p< 0.05$) in any of the paths;
25	Gough, 2016	All exposures.	N/A	Variables retained in final model based on p-value of univariable regression
26	De Beaudrap, 2016	Maternal education, age, gravidity, residential area, season, maternal HIV status, use of a bed net, gestational age.	N/A	A priori selection of exposures and covariates for inclusion in multivariable analyses
27	Busert, 2016	HH wealth, HH food insecurity, crowding, maternal height, maternal education, child care during illness, continued breastfeeding, child infections, general care, child age, sex.	N/A	A priori selection of exposure and covariates
28	Broere-Brown, 2016	None	N/A	A priori specification of no confounders due to random assignment of infant sex.
29	Bhargava, 2016	SES, birth order, number of people in the household, mother's education, BMI, energy intake; immunization; morbidity; diarrhoea (2-24 months)	N/A	A priori selection of confounders
30	Alkhalawi, 2016	Pregnancy duration, pre-pregnancy maternal BMI, maternal height, lead in maternal blood, newborn sex, mode of delivery, maternal place of birth (in/outside Germany), smoking during pregnancy, infant age at examination, duration of EBF.	N/A	A priori selection of confounders from the literature, and by p-value <0.1 in univariable analysis.
31	Wright, 2015	age, non-BM energy intake, non-BM energy intake x age	N/A	A priori selection of confounders; inclusion in final model based on p-values
32	Vail, 2015	Infant age and sex, maternal age, parity and deprivation score, and type of feeding at 3 months.	N/A	Not mentioned
33	Rogawski, 2015	Child sex, SES using Kuppuswamy scale, maternal education, HH hygiene, household crowding, LBW, preterm birth, C-section delivery, and growth z-score at the beginning of the month, EBF, days of diarrhoea, n episodes of diarrhoea, dehydration, ORS, hospitalization, and days with diarrhoea in previous month.	N/A	A priori selection of confounders

ID		Analysis		
#	First author, year	Covariates/confounders in final model	Mediators	Model selection methods
34	O'Keeffe, 2015	Maternal education, parity, smoking, age, height, BMI, partner drinking, infant gender.	N/A	A priori selection of confounders
35	Hanieh, 2015	Maternal age, gravidity, gestational age at enrolment, infant sex, trail arm.	Birth weight	Backward elimination stepwise regression to select a subset of variables from a list of measured factors identified a priori
36	Costet, 2015	Duration of gestation, maternal place of birth, maternal age, parity, maternal pre-pregnancy height and weight, maternal weight gain during pregnancy, education, marital status, smoking and alcohol consumption during pregnancy, and cord lipid concentration.	N/A	A priori selection of confounders
37	Richard, 2014	Sex	N/A	A priori model specification
38	Patel, 2014	Rural/urban location, mid-parental height, trial intervention (proxy for prolonged BF), maternal smoking, number of older siblings.	N/A	A priori model specification
39	Padanou, 2014	LBW, prematurity, IUGR, IYCF score, n malarial episodes, length at birth, maternal short stature, maternal low weight, parity, and ANC visits, infant sex, infant age	N/A	p<0.2 in univariate models
40	Murasko, 2014	Sex, race/ethnicity	N/A	A priori selection of variables
41	Mallard, 2014	Haemoglobin at 6 or 12 months; birth weight, maternal height, sex, HIV exposure, diarrhoea in past 3 months (6mo model) or between 6-12 months (12mo model), hospital admission between 6-12 months, current HAZ (6 or 12 month exposure models), treatment group, HH wealth, maternal education.	Dietary Diversity (continuous)	A priori selection of confounders and mediators
42	Jaganath, 2014	Sex, EBF 6 months, antibiotic use in 2 weeks before first infection, total number of diarrhoeal episode between 0-24 months, SES tertile	N/A	A priori selection of confounders
43	Hong, 2014	First weaning month, breastfeeding, mother's employment, second hand smoke exposure, caregiver, infant dietary supplementation, hospital admission history, parental education, HH income.	N/A	Confounders included based on t-tests and chi-squared tests.
44	Betoko, 2014	Related to growth and formula (centre, education, family income, mother's return to employment, EBF duration, type of physician), to growth (parental height and BMI, infant sex, gestational age), and type of formula used and growth (diarrhoea and regurgitations), average z-score between 0-4 months.	N/A	p values in univariable analyses

ID		Analysis		
#	First author, year	Covariates/confounders in final model	Mediators	Model selection methods
45	Woo, 2013	Maternal age, maternal gestational weight gain, infant sex, birth weight, model of delivery, cohort site	N/A	Backward elimination stepwise regression to select (p<0.2) a subset of variables from a list of measured factors identified a priori
46	Richard, 2013	Sex	N/A	A priori model specification
47	Peterson, 2013	Sex, family income, LAZ at 3 months	N/A	Not mentioned
48	Lee, 2013	Stunting at start of interval, WHZ at start of interval, season (Fourier's), age, birth weight, per capita income	N/A	Variables retained in final model based on model fit
49	Kwok, 2013	Sex, birthweight, parity, parents' age and parents' birthplace.	N/A	Based on model fit compared between specifications
50	Garza, 2013	Child sex, birthweight, maternal age and education, parity, household income, duration of breastfeeding, dietary diversity score 6-24 months, child morbidity due to diarrhoeal episodes, nutrient supplementation.	N/A	Not applicable
51	Fairley, 2013	Gestational age, smoking during pregnancy, maternal height.	N/A	Pre-specified model of interest
52	Durmus, 2013	Child's age at visit and sex	N/A	A priori model specification
53	Addo, 2013	Sex, site, SES quintile, birth order, nutrition supplementation	N/A	A priori model specification
54	Silva, 2012	Child age at measurement, smoking in pregnancy, birth weight, gestational age, maternal and paternal height, breastfeeding duration, day-care attendance at 24 months	(stated, but mediation analyses not conducted)	Variables retained in full models based on their (statistically significant) independent relationship with height.
55	Saha, 2012	Maternal or child U-As as relevant; age, sex, maternal BMI, SES quintile.	N/A	Not mentioned
56	Richard, 2012	None	N/A	Not mentioned
57	Queiroz, 2012	Maternal education, inadequate environmental conditions (WASH), birthweight <3000g, maternal height <150cm, new born anaemia.	N/A	p<0.2 in bivariable models
58	Matijasevich, 2012	Family income, marital status, maternal age, parity and skin colour maternal height, maternal smoking during pregnancy, gestational age, duration of breastfeeding. Separate model for all significant confounders p<0.2 (family income, marital status, maternal skin colour, maternal height)	N/A	A priori selection of confounders. Separate model fitted including only significant (univariable p<0.2) confounders

ID		Analysis		
#	First author, year	Covariates/confounders in final model	Mediators	Model selection methods
59	Martinez-Mesa, 2012	Paternal smoking, family income quintile, maternal height, maternal age, skin colour, LAZ at birth	N/A	A priori selection of confounders
60	Lourenco, 2012	Wealth index, land ownership, mother's height, child's birth weight, age at introduction of cow's milk, morbidities in the past 15 days	N/A	Variables retained in multivariable analyses based on p-values of univariable analyses (p<0.1) or a priori conceptual interest
61	Kang Sim, 2012	Maternal life stress (at 1 year) using Life Experiences Survey, maternal depression risk (CES-D), income earning-adult-to-child ratio (-1, 0, +1). Also trial arm, gestational age, breastfeeding (bottle at 6 weeks, still BF at 6 months)	Family environment for nurturing (HOME) - maternal warmth and emotional support, sibling participation in care, physical environment, father-infant interaction, cognitive stimulation.	A priori selection of confounders and mediators
62	Husain, 2012	First generation Pakistani, educated to A-level+, married, planned pregnancy, prim gravida, 2+ children, marked health difficulty, age.	N/A	A priori selection of confounders
63	Hambridge, 2012	Sex, maternal weight, infant BMI	N/A	A priori selection of confounders
64	Garced, 2012	Age at evaluation, maternal age, height and parity.	N/A	Backward elimination using change in estimate (10% change in B co-efficient)
65	Bork, 2012	Child age, wealth index, maternal education and occupation.	N/A	p<0.2 in univariable models
66	Matijasevich, 2011	Family income, marital status, schooling, age, skin colour, parity, height, body mass index (BMI), pregnancy duration and paternal smoking.	N/A	Not mentioned

ID		Analysis		
#	First author, year	Covariates/confounders in final model	Mediators	Model selection methods
67	Durmus, 2011	Child age at visit, sex, maternal ethnicity, education, height and weight, breastfeeding (0/1)	N/A	Change in estimate (>10%) or a priori importance of confounder
68	Deierlein, 2011	Gestational age, maternal height, maternal ethnicity, marital status, prenatal smoking, household income, and education, pre-existing diabetes mellitus.	N/A	A priori identification of confounders
69	De Hoog, 2011	Prenatal factors (maternal age, years of education, smoking during pregnancy, hypertension, diabetes, pre-pregnancy BMI, parity, maternal and paternal height); birth outcome (birthweight and gestational age).	N/A	A priori identification of confounders
70	Moore, 2010	None	N/A	N/A
71	Ertel, 2010	Maternal age, race/ethnicity, household income, gestational weight gain; child sex, gestational age, and birthweight for gestational age z-score	Gestational age at birth, birthweight for gestational age, BF duration, age of introduction of solid foods, and postnatal depression at 6 months or 1 year	Change in estimate (substantial change) in multivariable models.
72	de Beer, 2010	Maternal age, pre-pregnancy BMI, parity, education level, cohabitant status, ethnicity, duration of breastfeeding, standardized birth weight and pregnancy duration (linear and quadratic term)	N/A	Not mentioned
73	Andersen, 2010	Maternal age, parity, prepregnancy BMI, smoking during pregnancy, SES, gestational age at exposure measurement, duration of breastfeeding, child's age at measurement.	N/A	A priori selection of covariates.

ID		Analysis		
#	First author, year	Covariates/confounders in final model	Mediators	Model selection methods
74	Le Beaud, 2015	Sex, birthweight, birth length, birth head circumference, maternal education (SES proxy).	N/A	Not mentioned
75	Katulla, 2014	SES (forced variable)	N/A	Variables retained in multivariable analyses based on p-values of univariable analyses (p<0.3) or a priori conceptual interest
76	Howe, 2012	Source of measurement (routine/research)	N/A	A priori model specification
77	Johnson, 2012	Sex	N/A	A priori specification of covariates

ID		Results			
#	First author, year	Number of infants in the study	Number included in main analysis	Average number of length measurements per child	Exposure prevalence/incidence
1	Syed, 2018	380	376 at 6 months; 322 at 9 months	Not mentioned	Not reported
2	Steiner, 2018	512	(1) 212 (2) 254	2	(1) 36% with no crypto infection; 38% with 1 crypto infection, 26% with 2 or more crypto infections. 93% infections due to C.hominis species of crypto. (2) 56% with no crypto infection; 35% with 1 crypto infection, 9% with 2 or more crypto infections. 90% of infections dur to C.meleagridis species of crypto.
3	Schnee, 2018	700	(1) 603 (2) 575	Not mentioned	(1) 2559 episodes of diarrhoea between 0-12 months; 86.4% 603 children had at least 1 episode of diarrhoea. (2)

ID		Results			
#	First author, year	Number of infants in the study	Number included in main analysis	Average number of length measurements per child	Exposure prevalence/incidence
4	Sanin, 2018	265	234 at 9-12 months; 225 at 15-18 months; 214 at 21-24 months	Not mentioned	Mean MAR 0.24 at 12 months, 0.35 at 18 months, 0.48 at 24 months; overall 0.39 between 12-24 months.
5	Moradi, 2018	350	301	Total 3	Range of Dietary Energy Density Quartiles 1 to 4: <1613.4, 1613.4-2197.2, 2197.2-2699, >2699.
6	Lima, 2018	2145	1684	Not mentioned	10% had no pathogens in any stool samples.
7	Kramer, 2018	17046	Between 16089 and 17046	Not mentioned; length measured 7 times in 1st year	In intervention and control groups, BF rates were 73% and 60% at 3 months, 50% and 36% at 6 months, 36% and 24% at 9 months, and 20% and 11% at 12 months.
8	Islam, 2018	265	265 at birth, 229 at 12 months, 218 at 18 months, 211 at 24 months.	Not mentioned	MDD prevalence was 33% at 12 months, 60% at 18 months, and 79% at 24 months.
9	Garzon, 2018	475	282	Not mentioned	Presence of pathogens in at least one stool sample: Giardia lamblia 35% infants; Cryptosporidium spp. 15%, helminth 30%
10	Devakumar, 2018	Not mentioned (~1000)	793	Not mentioned	Maternal education (none = 50%; primary = 8%, secondary =41%), land (none = 11%, < 30 dhur =69%, > 30 dhur = 20%), assests (none = 15%, small = 33%, large = 52%)
11	Cheng, 2018	8327	7367	Not mentioned	Breastfeeding = 6%, mixed feeding = 37%, formula feeding = 57%
12	Admassu, 2018	634	354	Not mentioned	From 0-6 months, mean FM was 316 SD 97), FFM was 429 (SD 59)
13	Zhang, 2017	626	495	Minimum 5 measurements	37% mothers had no formal education, 35% had access to toilets, EBF mean 4 months; 51% had 2+ diarrhoeal episodes between 0-6 months.
14	Matos, 2017	2404	1907	Minimum 2 measurements	25% Afro-Ecuadorian ethnicity; 70% in urban or peri-urban areas; 54% in HH with 2+ children; 90% were EBF, and 47% for 3+ months.

ID		Results			
#	First author, year	Number of infants in the study	Number included in main analysis	Average number of length measurements per child	Exposure prevalence/incidence
15	MAL-ED Network Investigators / Caulfield, 2017	1868	1291	Not mentioned	BF at 0-2 (66% EBF, 12% PBF) and 3-5 months (28% EBF, 12% PBF); protein g/day at 9-11, 12-17, and 18-24 months (15, 19, and 26g); Diarrhoea episodes in each period (0.2, 0.4, 0.4, 0.4, 0.7, 0.5)
16	Clemente, 2017	502	336	Total 3	Daily outdoor NO2 exposure = 26.2 mg/m2 in entire pregnancy; mean birth length = 49cm, SD 2.1; Placental mtDNA content = 1.5
17	Bork, 2017	8019	512	Not mentioned	50.5% Male infants; in each age interval, proportions fed 2+ meals in last 24h (boys, girls) were (13, 8; 19, 17; 32, 32; 48; 52) with sex differences only at 2-3 months.
18	Bell, 2017	460	276	Not mentioned	77% PBF
19	Swithowski, 2017	2128	1961	Not mentioned	Mean protein intake in second trimester = 1.4g per kg pre-pregnancy body weight per day
20	Svefors, 2016	1663	1054	Not mentioned	Maternal height (34% <147.5cm; 34% >147.5 <152cm; 32% >152cm); education (39% None; 23% 1-5 years; 38% >5 years); season of conception (34% Winter, 30% Pre-monsoon; 37% Monsoon)
21	Owais, 2016	2400	2189 at 3 months; 2074 at 9 months; 1969 at 16 months; and 1885 at 24 months	Not mentioned	45% EBF at 3 months; 16% MAD at 9 months
22	Nagata, 2016	852	842	Not mentioned	53% Female; Mean n of children <5 in the household = 1.7 (0.69); mean total HH size = 6.12 (2.79); mean HAZ at 1 year -1.88(1.19); mean WAZ at 1 year -0.67 (1.01); mean days of illness in past week - diarrhoea 0.82, vomiting 0.12, cough 0.99, fever 0.54
23	Kavle, 2016	300	277	Not mentioned	Range of prevalence (%) between 4 to 12 months: diarrhoea (10%-21%); fever (39%-53%); breastfed (88%-97%); MDD (9%-59%)
24	Griffiths, 2016	600	511	Not mentioned	4% low SES, 37% medium SES, 59% high SES; mean maternal BMI =19.83 and height = 151.48cm

ID		Results			
#	First author, year	Number of infants in the study	Number included in main analysis	Average number of length measurements per child	Exposure prevalence/incidence
25	Gough, 2016	4526	3338	6	Mean maternal age (24.2), parity (2); gestational age (39.3); male infants (50%); at 3 months MF 43%, EBF 2.6%
26	De Beaudrap, 2016	1218	832	Not mentioned	No malaria (76%), 1 episode (19%), >1 episode (4%)
27	Busert, 2016	689	529, and 515	2 or 3 (depending on analysis)	Mean DDS at baseline (3.6 out of 7), first follow-up (3.9), second follow-up (3.4)
28	Broere-Brown, 2016	9778	8556	Not mentioned	49.4% female
29	Bhargava, 2016	3080	2076	11	Mean protein intake g/d (12.3 at 12 months, 21.6 at 24 months; calcium intake mg/d (256 at 12 mo. and 282 at 24 months)
30	Alkhalawi, 2016	232	148	(5 measurements; complete cases only)	Mean cord and maternal plasma for PFOA (1.75 *m*g/L and 2.44), for PFOS (2.83 and 9.04), and for PFHxS (0.4 and 0.74)
31	Wright, 2015	3080	2822	Not mentioned	At all time points, the recommended feeding pattern (BF+high DDS) did not exceed 5%
32	Vail, 2015	1121	571	(3 measurements; cc analysis)	Proportions weaned at each group were 7.7%, 25.6%, 38.6%, 27.1%.
33	Rogawski, 2015	497	456	85% had at least 29 measurements before 3 years of age	57.5% exposed to antibiotics by 6 months of age, and 28.1% received >1 dose. Highest exposure occurred during 3-5 months, average prevalence of 20.3%.
34	O'Keeffe, 2015	13761	7957	Not mentioned	57% of women and 96% of their partners consumed alcohol during pregnancy.
35	Hanieh, 2015	1171	1046	Not mentioned	Maternal BMI at enrolment = 19.9, weight gain during pregnancy = 8.19kg; prim gravida=31%,
36	Costet, 2015	1068	222	7	Prenatal exposure to chlorodecone = 59%, postnatal in maternal milk =79%
37	Richard, 2014	1007	1007	Not mentioned	Mean diarrhoea prevalence per year = 25 days; 84% were acute episodes. Prevalence was highest between 6 to 18 months.

ID		Results			
#	First author, year	Number of infants in the study	Number included in main analysis	Average number of length measurements per child	Exposure prevalence/incidence
38	Patel, 2014	17046	12463	12	Secondary (36%), advanced secondary (50%), university (14%) - for boys and girls in full sample
39	Padanou, 2014	656	520	Not mentioned	46% never experienced malaria, 18.4% had 1 episode, and 9.6% had more than 5.
40	Murasko, 2014	14000	~6950	Not mentioned	Average income at 9 months ($67.6k) at 24 month ($68.7K)
41	Mallard, 2014	811	631	Not mentioned	At 6 months, MMF=91%, MDD=12% and MAD=10%, and 61% consumed iron rich food.
42	Jaganath, 2014	304	183	Range (22-44)	77% had first h.pylori infection in infancy.
43	Hong, 2014	593	383	Not mentioned	Mean Vitamin A, C, E levels (100, 7.49, 1654), MDA 2.33, 8-Ohdg 0.12
44	Betoko, 2014	2002	1239	4	One third were formula fed predominantly.
45	Woo, 2013	365	285	Only 1 value used	Introduction of cereals, and fruit/veg occurred between 4.3 and 6.7 months; high-protein foods earlier in Shanghai (4.8 months) than Mexico City (7 months) or Cincinnati (9.3 months). Salty snack foods in 1st year: Cincinnati 34%, Mexico 78%, Shanghai 3%.
46	Richard, 2013	1007	1007	Not mentioned	Mean diarrhoea burden (prevalence range 8.9 to 47.3 days per child-year, incidence range 3.5 to 10.4 episodes per child-year).
47	Peterson, 2013	319	319	Not mentioned	Median (SE) REG1B concentration in stool samples for Bangladesh and Peru (30.8, SE 26.3 and 16.5, SE 17.7 mg/mL)
48	Lee, 2013	442	433	Not mentioned	Crude incidence of Campylobacter = 0.37 episodes per year
49	Kwok, 2013	8327	6510	Total 2	26% parents educated beyond 12th grade; 47% grandparents attained secondary education; mid-parental height = 164cm
50	Garza, 2013	1542	1542	5	Mid-parental height range across sites (163.6cm to 175.7cm)
51	Fairley, 2013	1707	1434	4	21% White British boys, 27% Pakistani boys, 23% White British girls, 29% Pakistani girls
52	Durmus, 2013	6969	4116	Not mentioned	Among mothers and fathers, 18.1% (n = 946) and 31.6% (n = 1652) were overweight, and 7.3% (n = 382) and 6.0% (n = 314) were obese
53	Addo, 2013	7630	7630	Not mentioned	Mean maternal height varied across sites (148.6cm in Guatemala to 158.3cm in South Africa)

ID		Results			
#	First author, year	Number of infants in the study	Number included in main analysis	Average number of length measurements per child	Exposure prevalence/incidence
54	Silva, 2012	6969	1972	Not mentioned	34.6% mothers had high education, 26.7% had mid-high level, 24.7% had mid-low, and 14% had low education.
55	Saha, 2012	2853	2372	Not mentioned	Median UA was 80 Mg/L (25-400) in mothers and 34 Mg/L (12-159) in children.
56	Richard, 2012	1604	1599	Not mentioned	Proportions wasted before 18 months ranged between 3% and 59% across sites
57	Queiroz, 2012	489	373	Not mentioned	Inadequate environment (WASH) = 35%, mother not cohabiting with partner = 30%, low maternal height = 10%, birthweight <3000g = 31%
58	Matijasevich, 2012	4231	4053	86% had complete data	Maternal education 0-4 years = 15%, 5-8 years = 41%, 9+ years = 44%
59	Martinez-Mesa, 2012	5249	1362	Not mentioned	Maternal smoking during pregnancy=33.5%, partner smoking 49.5%
60	Lourenco, 2012	256	210	3	Not reported
61	Kang Sim, 2012	1657	999	Not mentioned	Low SES (47%), Middle SES (53%). Middle SES had better optimal nurturing environment (p<0.05)
62	Husain, 2012	237	186	Not mentioned	36% mothers were perinatally depressed
63	Hambridge, 2012	412	388	Not mentioned	Mean maternal height 144.3cm (range 131.5cm to 164cm)
64	Garced, 2012	442	253	Not mentioned	Mean maternal serum DDE levels ranged from 6.3 ng/mL to 7.6 ng/mL during pregnancy.
65	Bork, 2012	1130	879	Not mentioned	96% of 9-12 month olds had eaten 1+ meal in last 24 hours
66	Matijasevich, 2011	5304 and 4287	1993 cohort (655, 1460, 1450); 2004 cohort (3985, 3907, 3869, 3799)	Not mentioned	1993 cohort: 33.5% of mothers and 44.8% of fathers smoked during pregnancy. 2004 cohort: 28% mothers, and 31% fathers.
67	Durmus, 2011	6969	5342	Not mentioned	9% of mothers smoked in 1st trimester, 15% continued.

ID		Results			
#	First author, year	Number of infants in the study	Number included in main analysis	Average number of length measurements per child	Exposure prevalence/incidence
68	Deierlein, 2011	1169	355	1	Mean pre-pregnancy BMI was 24.2; mean GWG was 16kg, and 59% gained excessive weight gain.
69	De Hoog, 2011	6575	2998	2	Duration of BF for 4-6 months ranged from 20% to 29%, and for 6+ months ranged from 24% to 46% across ethnic groups.
70	Moore, 2010	414	414	Not mentioned	Between 6-12 months of age, any diarrhoea = 5.15 episodes per child-year, acute = 4.22 episodes per child-year, persistent diarrhoea = 0.68 per child-year. Prolonged diarrhoea peaked between 12-24 months (0.26 episodes per child-year).
71	Ertel, 2010	2128	872	Not mentioned	Antenatal depression = 8% and postpartum depression = 7.3%
72	de Beer, 2010	6575	3994	2	No hypertension = 88%, pre-existing hypertension = 3%, pregnancy-induced hypertension = 9%
73	Andersen, 2010	1400	1010	2	Mean PFOS ranged from 6.4 to 106.7 ng/mL and PFOA max was 21.9 ng/mL.
74	Le Beaud, 2015	545	545	Not mentioned	32% experienced at least one parasitic infection and 7% experienced multiple infection.
75	Katulla, 2014	497	420	Not mentioned	Primaparity = 39%, maternal height (mean, SD) = 153.03 cm, 6.4; median (IQR) duration of BF = 4.09 (2.36, 5.24)
76	Howe, 2012	14541	12366	7	Maternal education < O-level (30%), O-level (35%), A-level (22.4%), degree (13%)
77	Johnson, 2012	600	384	Not mentioned	EBF at 3 month = 75%, maternal education (primary = 33.6%, none= 39%)

ID		Results	
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1	Syed, 2018	(1) At 6 months, highest quartile of anti-LPS IgA was associated with linear growth (LAZ change/year -0.33 (0.12), p=0.008)). At 9 months, two highest quartiles of anti-flic IgA (LAZ change/year -0.29 (0.13) p=0.02 and -0.32 (0.13) p=0.01, resp) (2) At 6 months, second and third quartiles of anti-LPS IgA had increased risk of subsequent stunting (HR 1.57 (0.81, 3.05) and HR 2.23 (1.15, 4.33))	High concentrations of anti-flagellin specific and anti-LPS IgA antibodies at 6 and 9 months predict declines in linear growth. Correlation between systemic/enteric inflammation and anti-LPS IgA points to mechanistic considerations for future studies.
2	Steiner, 2018	Overall: in adjusted models, any crypto infection associated with Delta-LAZ of -0.215 (p=0.0088). Those with 2+ crypto had greater declines (D-LAZ -0.2385, p=0.039) than those with 1 crypto (D-LAZ -0.2056, p=0.020) (1) In urban site, any crypto led to Delta-LAZ of -0.253, p=0.13 (2) In the rural site, any crypto associated with Delta-LAZ of -0.253 (p=0.011) in adjusted models. 1	Cryptosporidiosis in early childhood is associated with early growth faltering in Bangladeshi children. Differences in crypto species between urban and rural sites suggest different exposures or modes of transmission have similar outcomes for child growth
3	Schnee, 2018	(1) For each episode of diarrhoea, LAZ at 12 months changed by -0.01, (-0.06, 0.03), and for total number of days of diarrhoea -0.02, (-0.07, 0.03); for each additional episode of diarrhoea attributable to bacteria -0.09 (-0.16, -0.01), protozoa -0.24 (-0.49, 0.01), and viruses -0.01 (-0.11, 0.08); for each additional episode of diarrhoea attributable to Cryptosporidium -0.23 (-0.50, 0.03), Campylobacter -0.16 (-0.32, -0.01), Shigella -0.12 (-0.26, 0.03). (2) Cryptosporidium and Campylobacter attributable diarrhoea in the first year of life and subsequent growth at 24 months (LAZ at 24 months was lower by -0.72, (-1.83, 0.38) and -0.51 (-0.92, -0.10), respectively). Per 1 unit change in baseline LAZ associated with subsequent Cryptosporidium-attributable diarrhoea Risk Ration = 1.14 (0.91, 1.44)	(1) No relationship between all-cause diarrhoea and length at 12 months; but specific species (crypto, campylobacter and Shigella) associated with linear growth deficits. Link between diarrhoea and linear growth is pathogen-specific. (2) No evidence that chronic malnutrition was associated with subsequent specific enterpathogens, but lower LAZ was related to subsequent Cryptosporidium diarrhoea.
4	Sanin, 2018	Determinants of stunting between 12-24 months, in adjusted model, were child age 18 months 1.97 (1.49, 2.59) and 24 months 2.12 (1.45, 3.11) compared to 12 months; sex (male OR 1.98 (1.17, 3.33)); LBW OR=3.03 (1.69, 5.44);	Micronutrient Adequacy Ratio from 9 months is not associated with stunting between 12-24 months. LBW, age and sex were strongest predictors of stunting.

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5	Moradi, 2018	No difference in length at birth, 2 or 4 months by quartiles of dietary energy density (p=0.7, 0.1, 0.2)	No association between dietary energy density of lactating mothes and infant growth
6	Lima, 2018	No significant impact of infection on Delta LAZ	Subclinical enteroagregative E.coli did not alter growth in the first six months of life.
7	Kramer, 2018	Important differences between experimental and observational approaches; in ITT and IV, faster growth in the intervention group and in the group breastfed for 12+ months than in the control and <12 month BF group, during 2-3 months, and then decreasing difference and near equivalence by 12 months of age. Observational analysis showed different direction in second half of infancy, with intervention arm having lower LAZ at 6, 9 and 12 months.	The differences in experimental compared to observational analytical approaches using data from the same population, with effects from observational analysis in the opposite direction, lead to contrasting causal inference about link between breastfeeding and growth. Observational analyses were not able to rule out temporality and reverse causality i this instance.
8	Islam, 2018	Determinants of stunting between 12-24 months, in adjusted model, were male sex (aOR 1.75, CI 1.04, 2.95), LAZ at birth (aOR 0.40, CI 0.26, 0.61), and low asset index of poor HHs (aOR 2.81, CI 1.43, 5.52), and infant age at 18 months (aOR 2.13, CI 1.55, 2.92) and 24 months (aOR 2.34, CI 1.56, 3.52).	Stunting mechanism begins before a child is born, therefor a life course approach is important to target pregnancy-related determinants of infant stunting.
9	Garzon, 2018	Giardia lamblia and helminth infections associated with mean decreases of 0.10 in LAZ and 0.32 in LAD, and of 0.16 in LAZ and 0.48 in LAD, respectively. No relationship between Cryptosporidium and linear growth.	Subclinical parasitic enteric infections lead to mild growth faltering in infants and should be addressed in public health policies.
10	Devakumar, 2018	HAZ increased positively with maternal education and assets score; but only with highest category of land ownership. Maternal education and assets score protected against stunting at 2.5 years. Only highest level of all 3 SES indicators showed positive increase in conditional height gain between 0 to 2.5 years.	SES at birth is important for growth of children; influence of maternal education is strongest.
11	Cheng, 2018	Associations between breastfeeding and infant growth (LAZ) did not vary by sex; mixed or formula feeding had very small effects on LAZ (0.09 (-0.02, 0.20).	Breastfeeding in the first three months was not associated with infant length gain by sex

ID		Results	
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12	Admassu, 2018	FFM accretion from 0-6 months led to higher length at 1 year by 0.64cm (0.19, 1.09) and linear growth from 1 to 5 years by 0.63 cm (0.19, 1.07). Linear growth effect from 1 to 5 was due to influence at 1 year. FM accretion from 0 to 4 months led to greater linear growth from 1 to 5 years by 0.45cm (0.02, 0.88) in fully adjusted model.	FFM accretion was associated with linear growth at 1 year, with no additional longitudinal effect from 1 to 5 years. FM accretion had a weak association with linear growth from 1 to 5 years.
13	Zhang, 2017	Children with poor overall growth (low FPC strata) were more likely to experience growth faltering. Family income, family size, having an animal in the house, EBF <6 months (for boys), maternal weight, access to municipal water (for girls) were associated ($p<0.05$) with poor growth.	Understanding faltering patterns and associated risk factors are important in the development of effective intervention strategies to improve childhood growth globally
14	Matos, 2017	Native children had greater z-score deficits than Afro-Ecuadorian children, who were taller at each time point (boys and girls). In adjusted models, Afro-Ecuadorians had higher HAZ scores (0.25, SE 0.04) than native children in the first five years of life. Number of children in the household was also associated with lower HAZ (-0.13, SE 0.04) in the adjusted model.	Ethnicity is a determinant of growth trajectories during the first 5 years of life independent of socioeconomic factors
15	MAL-ED Network Investigators / Caulfield, 2017	WAMI was associated with 0.018 (SE 0.003) cm increase in length per month for 10% increase in WAMI; boys grew faster (0.26cm/month (SE 0.04) from 0-3 months), (0.14 (0.02) from 3-5 months), and 0.03 (0.007) from 6-24 months. No significant effect of diarrhoea on growth. High enteropathogen exposure led to shorter length (by 1.21 (0.33)cm, 0.39 LAZ) at 24 months. Campylobacter and E.coli led to length deficits (0.83cm and 0.85cm). Lower energy intakes and protein density led to shorter length (by 1.39cm (0.33), 0.42 LAZ) at 24 months.	Reducing enteropathogen burden and improving energy and protein density of complementary feeding could reduce stunting.

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16	Clemente, 2017	Inverse association between prenatal NO2 exposure and infant growth. Length was lower for a 10 mg/m3 increase in NO2 levels during trimester 1 at 6 months (-6.6% (-11.4, -1.9)). Birth length mediated 31.7% (34.5, 14.3), and mtDNA mediated 5.5% of the association between prenatal NO2 and length at 6 months.	Impaired foetal growth due to prenatal exposure to air pollution can lead to impaired growth in infancy. Molecular adaptations in placental mtDNA are associated with air pollution-induced alterations in postnatal growth.
17	Bork, 2017	Sex differences in growth: Boys had lower HAZ at 2 months (-0.191; p<0.035) but not HAD (-0.239cm, p=0.32); from 2-39 months, boys had lower HAZ per month (-0.007, p<0.001) and HAD per month (-0.025cm; p<0.001). Complementary feeding effects on HAZ: at 2-3 and 4-5 months, mean WHO HAZ was lower for infants with 2 or 3+ meals in last 24 hours compared to those with 0-1 meals. No difference at older ages (6-7, 9-10 months). There were sex differences in mean HAZ adjusted for meal frequency at older ages (6-8 and 9-10 months), p<0.001 and p=0.005.	Boys were more likely to consume CF; boys had lower HAZs than girls during infancy and up to 39 months. Importance of sex in complementary feeding and growth needs more attention in LMICs.
18	Bell, 2017	No difference in LAZ at 7 months between PBF and EFF infants (0.05 (-0.24, 0.34)); no difference in LAZ trajectories (p=0.16). Differences in weight were attributable to greater lean body mass accretion.	Formula fed infants did not differ in linear growth to PBF infants, and gained weight more rapidly and out of proportion to linear growth
19	Swithowski, 2017	Each 1 SD (0.36g/kg/day) increase in second-trimester protein intake led to a -0.1 LAZ at birth; -0.03 cm/mo. growth velocity from 0-6 months; and -0.09 cm/year from 6mo to mid-childhood.	In a population with relatively high protein intake during pregnancy, higher protein intake was associated with shorter offspring birth length and slower linear growth into midchildhood.
20	Svefors, 2016	In fully adjusted models, having a mother in the shortest group (-0.59HAZ, 95%CI -0.71, -0.466), no education (-0.25HAZ, 95%CI -0.39, -0.11), and pre-monsoon (-0.21HAZ, 95% CI -0.33, -0.09) were associated with HAZ scores over time.	Height growth trajectories from birth to pre-adolescence show strong intergenerational associations, social differentials, and environmental influences.

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21	Owais, 2016	In GEE models,EBF at 3 months did not predict linear growth between 9-24 months; infants with MAD at 9 months had higher LAZ (B=0.25; 95%CI 0.13, 0.37); female children were taller (B=0.13; CI 0.05, 0.21); each additional cm of maternal height led to 0.07SD (0.06, 0.08) gain in LAZ.	IYCF practices are linked to subsequent linear growth in childhood.
22	Nagata, 2016	In adjusted models corrected for multiple testing, HH size, number of children and diarrhoea in past week were most significant predictors of HAZ at 2 years.	Number of children <5, diarrhoea most strongly predict nutritional status at 2 years.
23	Kavle, 2016	LAZ decreased from 6 to 12 months. In adjusted models controlling for infant sex and birth z-score, maternal height, parity and education, there was no effect of diarrhoea, fever, or MDD on LAZ over 4-12 months of age. Visits at 6, 8, 12 months (ref 4 months) were associated with lower LAZ (-0.37, -0.61, -0.98, all p<0.001).	In Egypt, stunting and overweight both begin in the first year of life and interventions should address both
24	Griffiths, 2016	A 1 SD increase in SLI had a total effect of 0.17SD increase in LAZ at 12 months. Direct effect of SLI on LAZ = 0.08SD; indirect effect through maternal height = 0.02SD; indirect effect through maternal height and through LAZ at 6 months = 0.06SD; through maternal height and birthweight and LAZ at 6 months =0.01SD. This corresponds to a 0.09SD increase in LAZ for a 1SD increase in SLI, which is about 53% of the total effect of SES. Path through birthweight, LAZ at 6 months, and maternal BMI to LAZ at 12 months was not statistically significant.	SES inequalities in infant growth partly operate through maternal height and to a lesser extent through birth weight's relationship with maternal height.
25	Gough, 2016	LAZ declined from -0.6 to -1.4 between 0-24 months; probability of group membership was predicted by: maternal height and education, infant sex, birth length, birth weight.	Differences in magnitude of LAZ were influenced by factors that are already established by the time of birth.

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26	De Beaudrap, 2016	In multivariable models without the exposure term, length gain was greater in boys, preterm infants, and those born to mothers with higher education level. In adjusted models, infants born to mothers exposed to >1 episode of malaria in pregnancy had lower length (-2.71cm, 95%CI -4.17, -1.25). Risk of length restriction was higher for malaria in the 12 weeks before delivery (RR -1.39, 95%CI -2.76, -0.03).	Late pregnancy malarial infection was associated with impaired infant growth in the first year of life.
27	Busert, 2016	In adjusted analysis, an increase in dietary diversity by 1 food group/week was associated with a 0.09cm (0.00, 0.17) higher conditional growth in the second growth period (between 9-69 months and 29-89 months).	Increasing dietary diversity reduces risk of stunting and improves growth after growth faltering.
28	Broere-Brown, 2016	Males had greater body length than females, which was statistically significant from 9 months onwards, but the overall difference in pattern between sexes was not statistically significant (p=0.38).	There are sex differences in infant growth
29	Bhargava, 2016	Positive effect of calcium on length between 2-24 months (0.577cm, p<0.05); SES, maternal education, BMI, energy, child immunization, +ve effect on length; birth order, maternal morbidity, child diarrhoea and morbidity, female sex, and n persons in HH had a -ve effect	Food and health-related factors influence children's linear growth
30	Alkhalawi, 2016	In the GEE model, PFHxS was significantly related to infant length across all 5 time points (first year of life) in a sample of 37 infants (co-eff 4.516, 95%CI 1.368, 7.664)	The observed positive relationship between PFHxS and infant length may be due to the limited sample size rather than a real effect.

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31	Wright, 2015	In sex-stratified analyses, BF (regardless of DDS) was associated with LAZ (LAZ SD coeff increased by 0.16 for BF+lowDDS and 0.14 for BF+highDDS) between 6-24 months; predicted LAZ at all ages were higher among BF+lowDDS (at six months: boys were 0.25, (CI 0.19, 0.30) longer and girls were 0.2 (0.12, 0.28)) than the noBF+lowDDS group, but the magnitude of the benefit decreased over time. Recommended feeding pattern (BF+highDDS) did not confer benefit, neither did noBF+highDDS.	Results demonstrate the importance of prolonged breastfeeding up to 24 months, but DDS did not confer an advantage in LAZ.
32	Vail, 2015	Earlier weaning was associated with higher LAZ at 12 months (0.14 (0.05, 0.24) p<0.1), but the effect was attenuated after adjusting for feeding type at 3 months (0.08 (-0.02, 0.17) and earlier LAZ (0.04 (-0.02, 0.11).	In HICs weaning between 3-6 months has a neutral effect on infant length. Inverse associations are likely due to reverse causality.
33	Rogawski, 2015	In adjusted analysis for short term effects (0-6 months), there was no effect of antibiotic use on HAZ among boys or girls, corresponding to a difference of -0.1mm in boys and -1.2mm in girls. Girls who received antibiotics in a given month had a higher risk of stunting in the following month (RR 1.27 (1.04-1.56). Long term effects: Children with 2+ courses of antibiotics in 0-6 months were shorter during 6-36 months (-3.1mm from 2-3years, -1.5mm overall), but effects were not statistically significant.	Antibiotic exposures early in life were not associated with increased or decreased growth
34	O'Keeffe, 2015	Maternal occasional or light drinking during pregnancy was not associated with reduced birth length or growth at 2 years; infants of heavy drinking mothers had 0.78cm (-1.34, -0.22) birth length but did not differ in height at 2 years (0.11 (-0.56, 0.78)).	Maternal occasional or light drinking is not associated with birth length or postnatal growth, but residual confounding may persist. The adverse effect of maternal heavy drinking on birth length is overcome in childhood.

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35	Hanieh, 2015	In SEM, maternal BMI (0.04, CI 0.01-0.07) and weight gain during pregnancy (0.04 CI 0.01 to 0.06) on LAZ, maternal ferritin at 32 weeks gestation was indirectly associated with LAZ at 6 months through birth weight (-66g per two fold increase in ferritin (-104g, -29g). Direct association between Vitamin D in late pregnancy and LAZ at 6 months (-0.06 per 20nmol/L (-0.11 to -0.001)).	Maternal nutritional status is an important predictor of early infant growth.
36	Costet, 2015	(1) In the JB model, no effect of prenatal or postnatal cholorodecone on linear height growth (2) Boys with high postnatal exposure to chlorodecone had lower height at 3 and 18 months. Girls with high prenatal exposure had lower height at 8 and 18 months, and lower instantaneous height growth velocity at 8 and 18 months.	Chlorodecone exposure may affect growth trajectories in children aged 0-18 months
37	Richard, 2014	(1) Diarrhoea during the current period was associated with lower length velocity at all ages. For boys, diarrhoea was associated with lower length velocity during 3-6, 6-12, and 18-24 months. (2) Diarrhoea in previous time period followed by no diarrhoea in the current time period was associated with higher length velocity (3) No diarrhoea 0-2 years was associated with greater length at 24 months than those who had average or 2x more than average diarrhoea. Among boys, compared to those with no diarrhoea, those with average diarrhoea were 0.7cm shorter and those with 2x average were 1.4cm shorter at 24 months.	When diarrheal episodes are followed by diarrhoea-free periods in the first 2 y of life, catch-up growth is observed that may allow children to regain their original trajectories.

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38	Patel, 2014	(1) Children of most vs least educated mothers were longest at birth (girls = 0.43cm (0.28, 0.58) and boys = 0.30cm (0.15, 0.46); and at 2 years (girls=1.30cm (1.00, 1.61) and boys = 1.30 (1.01, 1.59), and 3 years (girls = 1.82 (1.35, 2.28) and boys =1.70 (1.25, 2.14)). Continued up to 7 years. But attenuated by mid-parental height. (2) In urban/rural adjusted models: Change in length gain rate per education category was (0-3 months) 0.31 cm/year (-0.03, -.65, trend p=0.08) for girls and 0.83cm/year (0.48, 1.17, p<0.001for boys. Did not persist from 3-12 or 34-84 months. But between 12-34 months strong evidence of a difference for girls (0.31 cm/yr. (0.18, 0.43) trend p<0.001) and boys (0.23cm/year (0.11, 0.35) trend p<0.001)	Socioeconomic difference in offspring growth commence in the early pre-natal period, and are partly explained by genetic or other factors influencing parental stature
39	Padanou, 2014	In multivariable analysis, LBW, IUGR, IYCF score, maternal short stature, maternal low BMI were -vely associated with HAZ, length at birth was +vely associated.	IUGR and LBW are persistent factors that influence linear growth
40	Murasko, 2014	A doubling of permanent income is associated with a ~0.26 cm height advantage between 9-60 months, and a ~0.11cm/year faster velocity between 9-24 months.	Associations between income and anthropometric development in US children originate in early life.
41	Mallard, 2014	Iron rich food was related to greater HAZ at 18 months (0.16, CI 0.03, 0.29, p=0.016). No effect of ASF at 6 months on HAZ at 18 months. IYCF indicators at 12 months were not related to HAZ at 18 months. Dietary diversity at 6months was positively associated with HAZ at 18mo (both P <0.001) and mediated 13.4% of the total effect of maternal education on HAZ at 18 mo.	IYCF programs should target the early period of complementary feeding; improving formal education for women may improve child growth through better IYCF

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42	Jaganath, 2014	(1) HR=1.59 (1.16, 2.19, p=0.04) for low SES and HR=0.63 (0.40, 0.98, p=0.04) for EBF to 6 months in Cox regression model. (2) H. pylori infection in infancy was not independently associated with growth deficits (0.19cm, (-0.40, 5.06) p=0.58), children who had their first detected H. pylori infection in infancy (6–11 months) versus early childhood (12–23 months) and who had an average number of diarrhoea episodes per year (3.4) were significantly shorter at 24 months (0.37 cm, 95% CI, 0.60, 0.15 cm; p = .001).	Lower SES associated with higher risk of H.pylori in infancy, which increased the adverse effect of diarrhoea on linear growth
43	Hong, 2014	Maternal antioxidant vitamin and oxidative stress levels were not associated with infant length in adjusted models, but had an impact on head circumference and weight.	Antioxidative vitamins supplementation during pregnancy can improve child growth (weight, head circumference) in the first 3 years of life.
44	Betoko, 2014	No relationship between infant growth and type of formula (0.08 (-0.07, 0.24)). Infants breastfed for shorter duration showed higher LAZ change between 0-4 months (-0.08 (-0.12, -0.03))	Infant growth in the first 4 months is related to other factors than to type of formula used.
45	Woo, 2013	In adjusted analysis, there was no effect of IYCF on LAZ at 12 months. But there were differences by cohort site, and birthweight (0.65, SE 0.14, p<0.005)	In these urban, international cohorts of breast-fed babies, the impact of feeding on anthropometry was not consistent across sites, and did not explain variation in anthropometry between settings.
46	Richard, 2013	The cumulative association between the average diarrhoea burden (23 days per year vs no diarrhoea) and length at age 24 months was −0.38 cm (95% CI: −0.59, −0.17).	Diarrhoea prevention is an important public health strategy for improving child health and nutrition
47	Peterson, 2013	Higher stool REG1B concentrations (every 20 Mg/mL) at 3 months were related to lower future LAZ between 6-24 months in Bangladesh (-0.1, SE 0.036, p=0.0061) and Peru (-0.129, SE 0.068, p=0.0588)	REG1B predicts growth deficits in independent cohorts of poor children in developing countries and supports the role of environmental enteropathy in pathogenesis of growth faltering.
48	Lee, 2013	Symptomatic Campylobacter infections were associated with reduced linear growth over a nine month period (-0.059 cm per episode (-0.118, 0.001, p=0.054); severe episodes were associated with reduced linear growth (-0.169 cm per episode (-0.310, -0.028, p=0.019)	Campylobacter is not as benign as commonly assumed; there is evidence to support antibiotic therapy for campylobacteriosis in children

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49	Kwok, 2013	In fully adjusted models, parents' education (0.11, (0.05, 0.18, p<0.05) and parents' height (0.04 (0.04, 0.05) p<0.05) were associated with infant length gain. No effect on later childhood growth	Parental education positively affects growth in infancy, suggesting that the mechanism underlying socioeconomic influences on height at different growth stages in life may be contextually specific
50	Garza, 2013	Across sites, mid-parental height accounted for greater proportions of observed variability in attained child length than paternal or maternal height alone (Mean 16%, range 11% in Ghana, 21% India), and 6% of the within child variability. Except in Norway and US, predicted adult heights exceeded mid-parental heights by 6.2-7.8cm.	The MGRS infants showed similar growth patterns despite large differences in parental heights across the six sites. The link between mid-parental height and predicted height are indicative of the expectation that favourable conditions in care and nutrition can translate into community-wide height gains.
51	Fairley, 2013	At birth, Pakistani boys and girls were 0.5cm shorter than White British boys and girls. In unadjusted models, length velocity among Pakistani boys and girls compared to British counterparts was faster at 0-4 months (0.3cm/mo. (0.1, 0.5) and 0.4cm/mo. (0.2, 0.6)). Adjusted models were similar. By 2 year, Pakistani boys and girls were 0.6cm (0.02, 1.21) and 1.1cm (0.48,1.64) taller than white British boys and girls	Pakistani infants were shorter at birth than white British children, but gained length quicker in infancy.
52	Durmus, 2013	Maternal and paternal anthropometry (height, weight, BMI SD) were strongly related to offspring length at all ages, with increasing strength with age (range 0.24 at birth to 0.36 at 48 months for mothers and 0.21 to 0.33 for fathers, all p=<0.05). Combined maternal and paternal heights explained 5.9%, 9.8%, 11.6%, 15.3% 16.7% of the variance in child height measurements at birth, 1, 2, 3 and 4 years.	Maternal and paternal anthropometrics affect early growth in pre-school children differently.
53	Addo, 2013	A 1cm mean increase in maternal height was associated with an increase in offspring length SD at 2 years (0.078 (0.074-0.083), and conditional increase from 0-2 of 0.037(0.033-0.040). Maternal height was related to child stunting at 2 years (PR=0.88, CI 0.87-0.89), short mothers more likely to have stunted children at 2 years (PR=3.20, CI 2.80-3.60).	Maternal height influences infant linear growth through genetic and non-genetic factors

ID		Results	
#	First author, year	Main findings	Conclusion
54	Silva, 2012	At 2 months, infants of low education mothers were shorter than those of highest education (-0.8cm, CI: -1.16, -0.58), but their growth velocity was higher between 1-18 months, and they were taller by 14 months of age (0.4cm, CI: 0.08, 0.72). Adjustment for other factors increased the difference (0.61cm, CI: 0.26, 0.95 at 14 months, and 1.0cm, CI: 0.57, 1.43 by 25 months).	Children of low SES show accelerated linear growth until 18 months postnatally, overcompensating their initial height deficit.
55	Saha, 2012	In adjusted models, the inverse relationship between U-As and length was attenuated for children aged 3-24 months, but was robust for children aged 18-24 months, particularly girls. At 21 months, those with the highest quintile of U-A were 0.7cm shorter and had increased odds of stunting (OR 1.58, CI: 1.05-2.37).	Postnatal arsenic exposure was associated with lower length among girls, but not boys.
56	Richard, 2012	Wasting at 6–11 or 12–17 months was associated with decreased LAZ. Children who experienced wasting only at 0–5 mo. did not suffer any long-term growth deficits compared with children with no wasting during any period. Children with greater WLZ variability (<= 0.5 SD) in the first 17 months of life were shorter [LAZ = 20.51 SD (95% CI: 20.67, 20.36 SD)] at 18–24 months of age than children with WLZ variability <0.5. Change in WLZ in the previous 6-month period was directly associated with greater attained length at 18 mo. [0.33 cm (95% CI: 0.11, 0.54 cm)] and 24 mo. [0.72 cm (95% CI: 0.52, 0.92 cm)].	Children with wasting, highly variable WLZ, or negative changes in WLZ are at a higher risk for linear growth retardation, although instances of wasting may not be the primary cause of stunting in developing countries.
57	Queiroz, 2012	Mother not cohabiting with partner (0.2347, p=0.004), greater EBF duration (0.0031, p<0.001), low maternal height (-0.4393, p<0.001), birthweight<3000g (-0.8084, p<0.001), and newborn anaemia (-0.0875, p<0.001) had an effect on estimated mean LAZ.	Short maternal stature, birthweight <3000g, and newborn anaemia had a negative effect on linear growth in the 1st year, but longer EBF duration and mothers who did not cohabit with partners had a protective effect on linear growth.

ID		Results	
#	First author, year	Main findings	Conclusion
58	Matijasevich, 2012	Linear and positive associations with birth length and length growth rates at 0-3, and 12-29/32 months for boys and girls, but little association between 3-12 months. By age 4, there was a mean difference of 3cm between extreme education categories, which persisted after adjustment for maternal height.	The data demonstrate that height inequality, which was already present after birth, widens through differential growth rates 0-2 years.
59	Martinez-Mesa, 2012	In crude analysis, there was a negative dose response relationship between number of cigarettes/day (<10, 10-19, 20+) and infant LAZ at 1 year [-0.39(-0.56, -0.22), -0.70 (-0.98, -0.42), -0.67 (-0.97, -0.37)]. In adjusted models, the negative association was attenuated. Paternal smoking was not associated with offspring height in adjusted models.	Maternal smoking during pregnancy negatively affects offspring height
60	Lourenco, 2012	In adjusted analysis, maternal height (third tallest vs shortest) [0.60 (0.17, 1.03)], and child's birth weight (LBW vs normal (2.5-4.0kg)) [-0.78 (-1.39, -0.16)] were related to LAZ at 2 years.	Maternal height and child anthropometry at birth are associated with linear growth in childhood
61	Kang Sim, 2012	Higher SES was directly related to length gain in infancy (B=0.06, p=0.05) and number of children was inversely related to length gain (B=-0.07, p<0.05)	A direct relationship between SES and length gain developed during infancy
62	Husain, 2012	In crude analysis there was no difference in LAZ at 6 months between infants of depressed and non-depressed mothers [0.03 (-0.30, 0.37) p=0.85]. Association remained unchanged after adjustment for covariates. No relationship between EPDS score and HAZ in sex-stratified analysis (girls= -0.15, p=0.15, boys = -0.11, 0.37)	Perinatal depression is not associated with impaired growth in this sample of British Pakistani women.

ID		Results	
#	First author, year	Main findings	Conclusion
63	Hambridge, 2012	(1) In adjusted analysis, maternal height was positively associated with LAZ at 6 and 12 months (0.06 and 0.07). (2) Average maternal height for stunted children was lower than that for non-stunted children (143.2cm vs 145.5cm, p<0.0001). OR for stunting at 6 and 12 months was 0.91 (0.87, 0.96) and 0.88 (0.83, 0.93). (3) Linear growth velocity between 6-12 months was positively associated with maternal height (0.072 mm/month, p=0.0012)	
64	Garced, 2012	There was no effect of DDE exposure in any trimester on infant length growth from 0-12 months [1st trimester = 0.04 (-0.05, 0.12), 2nd trimester = 0.01 (-0.08, 0.09), 3rd trimester = 0.08 (-0.004, 0.16)]	There is no evidence of an association between prenatal DDE exposure and child growth during the first year of life
65	Bork, 2012	Length increment between visits was associated with MFI and CFI for those aged 18-24 months (0<0.001, 0<0.05) but not DDI, FVI or BF at any age.	No ICFI indicator was associated with linear growth in children aged 6-36 months
66	Matijasevich, 2011	In adjusted analyses for the 1993 and 2004 Pelotas cohorts, maternal smoking during pregnancy was associated with lower LAZ score at birth [-0.34 (-0.40, -0.27) and -0.24 (-0.33, -0.16], 3 months [-0.35 (-0.56, -0.15) and -0.24 (-0.32, -0.15)], 12 months [-0.20 (-0.35, -0.05) and -0.20 (-0.28, -0.11)], 24 months [-0.20 (-0.28, -0.12)]. Paternal smoking had a negative effect on LAZ in crude analyses only.	The results support the hypothesis that maternal smoking during pregnancy impairs linear growth in childhood
67	Durmus, 2011	Compared to no smoking, maternal smoking in the first trimester only did not have an effect on length SDS at any age. Continued smoking had a negative effect across all age groups: birth, 3, 6, 12, 24, 36, 48 months [-0.4 (-0.49, -0.31), -0.30 (-0.38, -0.23), -0.14 (-0.21, -0.06), -0.14 (-0.21, -0.06), -0.13 (-0.21, -0.05), -0.11 (-0.20, -0.03) and -0.10 (-0.19, -0.01)]. The largest effect was for mothers who smoked 5+ cigarettes/day [at 3 months -0.45 (-0.59, -0.37), p<0.01, and at 48 months, -0.23, (-0.35, -0.10), p<0.01]	Direct intrauterine exposure to smoke until late pregnancy leads to different height and weight adaptations and increased risks of overweight and obesity in preschool children.

ID		Results	
#	First author, year	Main findings	Conclusion
68	Deierlein, 2011	Pre-pregnancy BMI was not associated with LAZ at 6 months; excessive GWG was associated with higher LAZ [0.34 (0.12, 0.56)], and excessive GWG over 200% was associated with higher LAZ [0.45 (0.06, 0.83)].	Pre-pregnancy BMI and GWG are modifiable intrauterine exposures that influence infant postnatal growth
69	De Hoog, 2011	Shorter duration of EBF is associated with higher length gain from 0-6 months ($p<0.01$). EBF for at least 4 months was associated with the slowest growth in length (Delta SDS 0.16 (0.09, 0.24) compared to 0.46 (0.36, 0.55) for no breastfeeding). After adjusting for confounding and infant feeding, length Delta SDS was higher in all minority ethnic groups compared to ethnic Dutch infants.	Feeding factors explained, to a small degree, the higher weight and length gain in African descent infants, but not the higher DSDS weight-for-length in the Moroccan population
70	Moore, 2010	Decreases in HAZ after first episode of prolonged diarrhoea [-0.81 before, -1.40 after, $p=0.002$), acute diarrhoea (-0.51 before, -0.82 after, $p<0.0001$).	Prolonged diarrhoea is a risk factor malnutrition in resource-limited settings
71	Ertel, 2010	In adjusted models, postpartum depression was associated with greater HAZ (0.37 (0.16, 0.58) at 3 years. The longitudinal association between postpartum depression and child HAZ was 0.29 (0.11, 0.47). Antenatal depression was not associated with HAZ from birth to 3 years [-0.01 (-0.19, 0.17)]	Findings do not support the hypothesis that maternal depression is associated with reduced height in children in a relatively advantaged sample in a high income country
72	de Beer, 2010	In unadjusted models, pre-existing hypertension was associated with accelerated linear growth [OR 1.77 (1.19, 2.63)], but the effect did not remain in fully adjusted models [OR 1.58, (0.99, 2.51)]. Pregnancy-induced hypertension showed a similar relationship. Weight acceleration was related to pre-existing hypertension in adjusted models.	Infants of women with pre-existent hypertension during pregnancy more frequently have growth acceleration in weight and length, and yet the mechanisms acting on postnatal growth appear to be different.
73	Andersen, 2010	There was no effect of PFOA and PFOS on infant length at 5 or 12 months of age (all Cis crossed 0).	PFOA and PFOS levels did not affect length growth in this population

ID		Results	
#	First author, year	Main findings	Conclusion
74	Le Beaud, 2015	In the full cohort (n=545), parasitic infections were associated with reduced longitudinal growth in length up to 24 months for hookworm (-0.36, p=0.001), ascaris (-0.93 p<0.01), and all types (-0.35, p=0.01), and up to 18 months for malaria (-0.46, p=0.003). In the complete follow-up group (n=180), ascaris (-0.86, p=0.02) and E.histolytica (-0.51, p=0.03) at 24 months, Giardia (-0.05, p=0.03) at 12 months, and malaria (-0.64, p=0.003) at 18 months were associated with reduced linear growth. Infection with any parasite by 24 months led to a lower relative change in z-score (-0.33, p=0.022). Children with polyparasitism at any time point had lower length gain (p=0.004) than those who had not experienced any parasitic infection by 36 months.	Parasitic infection and polyparasitism were common among children <3 years in rural Kenya, and associated with growth impairments in length.
75	Katulla, 2014	In multivariable analysis, the mean length velocity was 1.37 cm/month, and factors associated with monthly height gain were female sex [-0.05 cm/month (-0.08, -0.02)], maternal height [0.006 cm/month], primaparity [0.03 (0.002, 0.06)], EBF 6 month [-0.06 (-0.10, -0.01)], and birth height [-0.03 (-0.04, -0.03)].	
76	Howe, 2012	Gradient in birth length across maternal education levels (mean difference between lowest and highest categories = 0.41 cm for boys and 0.65cm for girls). SES differentials in some growth periods (10-29 months, 29-120 months for boys, and 2-11 months, 11-32 months, and 32-120 months for girls).	Socioeconomic differentials in length in contemporary high-income populations are present at birth, with some widening of disparities in later childhood.
77	Johnson, 2012	No difference in length 3-15 months by EBF status to 3 months (-0.165cm, SE=0.239). Girls were shorter than boys (-1.547cm, SE=0.211). Maternal education (none vs secondary) led to shorter length (-0.791cm, SE=0.269). Compared to those of highest Standard of Living Index, length was lower in those of middle (-0.7cm, SE=0.3) and lowest (-0.8, SE=0.3) tertiles.	Not adhering to WHO breastfeeding regime at 3 months was not responsible for reduced linear growth, but low SES and morbidity were more strongly related to child growth.

ID		Critical appraisal: Selection				Critical appraisal: Comparability of groups based on design or analysis	
#	First author, year	Representativeness of the exposed cohort	Selection of non-exposed cohort	Ascertainment of exposure	Demonstrate that outcome was not present at start of study	Controls for infant sex	Control for confounders justified
1	Syed, 2018	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	Yes	Yes
2	Steiner, 2018	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	No	Yes
3	Schnee, 2018	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	Yes	No
4	Sanin, 2018	Somewhat representative	Drawn from same community as exposed cohort	Secure record	No	Yes	Yes
5	Moradi, 2018	Select group	Drawn from same community as exposed cohort	Written self-report	No	No	No
6	Lima, 2018	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	Yes	Yes
7	Kramer, 2018	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
8	Islam, 2018	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	No	Yes	Yes

ID		Critical appraisal: Selection				Critical appraisal: Comparability of groups based on design or analysis	
#	First author, year	Representativeness of the exposed cohort	Selection of non-exposed cohort	Ascertainment of exposure	Demonstrate that outcome was not present at start of study	Controls for infant sex	Control for confounders justified
9	Garzon, 2018	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	Yes	No
10	Devakumar, 2018	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
11	Cheng, 2018	Truly representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
12	Admassu, 2018	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	Yes	Yes
13	Zhang, 2017	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	No
14	Matos, 2017	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	No
15	MAL-ED Network Investigators / Caulfield, 2017	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	Yes	Yes
16	Clemente, 2017	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	Yes	Yes
17	Bork, 2017	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	No	Yes	No

ID		Critical appraisal: Selection				Critical appraisal: Comparability of groups based on design or analysis	
#	First author, year	Representativeness of the exposed cohort	Selection of non-exposed cohort	Ascertainment of exposure	Demonstrate that outcome was not present at start of study	Controls for infant sex	Control for confounders justified
18	Bell, 2017	Select group	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
19	Swithowski, 2017	Somewhat representative	Drawn from same community as exposed cohort	Written self-report	Yes	Yes	Yes
20	Svefors, 2016	Select group	Drawn from same community as exposed cohort	Secure record	Yes	Yes	Yes
21	Owais, 2016	Select group	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	No
22	Nagata, 2016	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
23	Kavle, 2016	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
24	Griffiths, 2016	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	No	Yes	Yes
25	Gough, 2016	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
26	De Beaudrap, 2016	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	No	Yes

ID		Critical appraisal: Selection				Critical appraisal: Comparability of groups based on design or analysis	
#	First author, year	Representativeness of the exposed cohort	Selection of non-exposed cohort	Ascertainment of exposure	Demonstrate that outcome was not present at start of study	Controls for infant sex	Control for confounders justified
27	Busert, 2016	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	No	Yes	Yes
28	Broere-Brown, 2016	Truly representative	Drawn from same community as exposed cohort	Secure record	Yes	Yes	Yes
29	Bhargava, 2016	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	No	Yes	Yes
30	Alkhalawi, 2016	Select group	Drawn from same community as exposed cohort	Secure record	Yes	Yes	Yes
31	Wright, 2015	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	No	Yes	Yes
32	Vail, 2015	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	No
33	Rogawski, 2015	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
34	O'Keeffe, 2015	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
35	Hanieh, 2015	Select group	Drawn from same community as exposed cohort	Secure record	Yes	Yes	Yes

ID		Critical appraisal: Selection				Critical appraisal: Comparability of groups based on design or analysis	
#	First author, year	Representativeness of the exposed cohort	Selection of non-exposed cohort	Ascertainment of exposure	Demonstrate that outcome was not present at start of study	Controls for infant sex	Control for confounders justified
36	Costet, 2015	Select group	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
37	Richard, 2014	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	No
38	Patel, 2014	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
39	Padanou, 2014	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	No
40	Murasko, 2014	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	No	Yes	Yes
41	Mallard, 2014	Select group	Drawn from same community as exposed cohort	Structured interview	No	Yes	Yes
42	Jaganath, 2014	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	Yes	Yes
43	Hong, 2014	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	Yes	No
44	Betoko, 2014	Somewhat representative	Drawn from same community as exposed cohort	Written self-report	Yes	Yes	No

ID		Critical appraisal: Selection				Critical appraisal: Comparability of groups based on design or analysis	
#	First author, year	Representativeness of the exposed cohort	Selection of non-exposed cohort	Ascertainment of exposure	Demonstrate that outcome was not present at start of study	Controls for infant sex	Control for confounders justified
45	Woo, 2013	Select group	Drawn from same community as exposed cohort	Structured interview	Yes	No	Yes
46	Richard, 2013	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	No
47	Peterson, 2013	Select group	Drawn from same community as exposed cohort	Secure record	Yes	Yes	No
48	Lee, 2013	Somewhat representative	Drawn from same community as exposed cohort	Secure record	No	Yes	Yes
49	Kwok, 2013	Truly representative	Drawn from same community as exposed cohort	Structured interview	No	Yes	Yes
50	Garza, 2013	Select group	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
51	Fairley, 2013	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
52	Durmus, 2013	Truly representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
53	Addo, 2013	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes

ID		Critical appraisal: Selection				Critical appraisal: Comparability of groups based on design or analysis	
#	First author, year	Representativeness of the exposed cohort	Selection of non-exposed cohort	Ascertainment of exposure	Demonstrate that outcome was not present at start of study	Controls for infant sex	Control for confounders justified
54	Silva, 2012	Select group	Drawn from same community as exposed cohort	Structured interview	Yes	No	Yes
55	Saha, 2012	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	Yes	No
56	Richard, 2012	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	No	No
57	Queiroz, 2012	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	No	No
58	Matijasevich, 2012	Truly representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
59	Martinez-Mesa, 2012	Truly representative	Drawn from same community as exposed cohort	Written self-report	Yes	No	No
60	Lourenco, 2012	Select group	Drawn from same community as exposed cohort	Structured interview	No	No	Yes
61	Kang Sim, 2012	Select group	Drawn from same community as exposed cohort	Structured interview	Yes	No	Yes
62	Husain, 2012	Select group	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes

ID		Critical appraisal: Selection				Critical appraisal: Comparability of groups based on design or analysis	
#	First author, year	Representativeness of the exposed cohort	Selection of non-exposed cohort	Ascertainment of exposure	Demonstrate that outcome was not present at start of study	Controls for infant sex	Control for confounders justified
63	Hambridge, 2012	Select group	Drawn from same community as exposed cohort	Secure record	No	Yes	No
64	Garced, 2012	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	No	Yes
65	Bork, 2012	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	No	No	Yes
66	Matijasevich, 2011	Truly representative	Drawn from same community as exposed cohort	Written self-report	Yes	No	No
67	Durmus, 2011	Truly representative	Drawn from same community as exposed cohort	Written self-report	Yes	No	Yes
68	Deierlein, 2011	Somewhat representative	Drawn from same community as exposed cohort	Written self-report	Yes	Yes	Yes
69	De Hoog, 2011	Truly representative	Drawn from same community as exposed cohort	Written self-report	Yes	Yes	Yes
70	Moore, 2010	Select group	Drawn from same community as exposed cohort	Secure record	Yes	No	No
71	Ertel, 2010	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes

ID		Critical appraisal: Selection				Critical appraisal: Comparability of groups based on design or analysis	
#	First author, year	Representativeness of the exposed cohort	Selection of non-exposed cohort	Ascertainment of exposure	Demonstrate that outcome was not present at start of study	Controls for infant sex	Control for confounders justified
72	de Beer, 2010	Truly representative	Drawn from same community as exposed cohort	Secure record	Yes	Yes	No
73	Andersen, 2010	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	Yes	Yes
74	Le Beaud, 2015	Somewhat representative	Drawn from same community as exposed cohort	Secure record	Yes	Yes	No
75	Katulla, 2014	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	Yes
76	Howe, 2012	Truly representative	Drawn from same community as exposed cohort	Structured interview	Yes	Yes	No
77	Johnson, 2012	Somewhat representative	Drawn from same community as exposed cohort	Structured interview	No	Yes	Yes

ID		Critical appraisal: Outcome			Overall
#	First author, year	Assessment of outcome	Follow-up spans full 1st year	Adequate follow-up	Comments
1	Syed, 2018	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
2	Steiner, 2018	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	

ID		Critical appraisal: Outcome			Overall
#	First author, year	Assessment of outcome	Follow-up spans full 1st year	Adequate follow-up	Comments
3	Schnee, 2018	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
4	Sanin, 2018	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
5	Moradi, 2018	Record linkage	No	No statement	
6	Lima, 2018	Independent blind assessment	No	More than 10% attrition and missingness not examined	
7	Kramer, 2018	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
8	Islam, 2018	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
9	Garzon, 2018	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
10	Devakumar, 2018	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
11	Cheng, 2018	Record linkage	Yes	Less than 10% attrition or missingness examined	
12	Admassu, 2018	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
13	Zhang, 2017	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
14	Matos, 2017	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
15	MAL-ED Network Investigators / Caulfield, 2017	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
16	Clemente, 2017	Record linkage	Yes	More than 10% attrition and missingness not examined	

ID		Critical appraisal: Outcome			Overall
#	First author, year	Assessment of outcome	Follow-up spans full 1st year	Adequate follow-up	Comments
17	Bork, 2017	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
18	Bell, 2017	Independent blind assessment	No	More than 10% attrition and missingness not examined	
19	Swithowski, 2017	Record linkage	Yes	Less than 10% attrition or missingness examined	
20	Svefors, 2016	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
21	Owais, 2016	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
22	Nagata, 2016	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
23	Kavle, 2016	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
24	Griffiths, 2016	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
25	Gough, 2016	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
26	De Beaudrap, 2016	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
27	Busert, 2016	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
28	Broere-Brown, 2016	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
29	Bhargava, 2016	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
30	Alkhalawi, 2016	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
31	Wright, 2015	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	Good IYCF analysis methods.

ID		Critical appraisal: Outcome			Overall
#	First author, year	Assessment of outcome	Follow-up spans full 1st year	Adequate follow-up	Comments
32	Vail, 2015	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
33	Rogawski, 2015	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	India-slum
34	O'Keeffe, 2015	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
35	Hanieh, 2015	Independent blind assessment	No	Less than 10% attrition or missingness examined	
36	Costet, 2015	Record linkage	Yes	More than 10% attrition and missingness not examined	
37	Richard, 2014	Independent blind assessment	Yes	No statement	
38	Patel, 2014	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
39	Padanou, 2014	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
40	Murasko, 2014	No description	Yes	More than 10% attrition and missingness not examined	
41	Mallard, 2014	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	IYCF analysis…
42	Jaganath, 2014	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	Good use of Cox regression to examine determinants of exposure (time to event)
43	Hong, 2014	Record linkage	Yes	Less than 10% attrition or missingness examined	
44	Betoko, 2014	Record linkage	Yes	Less than 10% attrition or missingness examined	Good use of Jenss Bayley
45	Woo, 2013	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	IYCF as exposure, KM curves, and reporting of snack foods.
46	Richard, 2013	Independent blind assessment	Yes	No statement	Diarrhoea - Good longitudinal exposure specification

ID		Critical appraisal: Outcome			Overall
#	First author, year	Assessment of outcome	Follow-up spans full 1st year	Adequate follow-up	Comments
47	Peterson, 2013	Independent blind assessment	Yes	No statement	
48	Lee, 2013	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	Good longitudinal Poisson analysis of diarrhoea
49	Kwok, 2013	Independent blind assessment	No	More than 10% attrition and missingness not examined	
50	Garza, 2013	Independent blind assessment	Yes	Complete follow-up	MGRS data showing mid-parental height explains 16% of between-child and 6% within child variability in length 0-24 months
51	Fairley, 2013	Record linkage	Yes	More than 10% attrition and missingness not examined	
52	Durmus, 2013	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
53	Addo, 2013	Independent blind assessment	Yes	No statement	
54	Silva, 2012	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	See reverse SES and height association
55	Saha, 2012	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
56	Richard, 2012	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
57	Queiroz, 2012	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
58	Matijasevich, 2012	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	Good model selection, methods write up, discussion section
59	Martinez-Mesa, 2012	Independent blind assessment	Yes	No statement	
60	Lourenco, 2012	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	

ID		Critical appraisal: Outcome			Overall
#	First author, year	Assessment of outcome	Follow-up spans full 1st year	Adequate follow-up	Comments
61	Kang Sim, 2012	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	Good measurement of home environment using a scale (Home Observation for Measurement of the Environment). Mediation analysis
62	Husain, 2012	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
63	Hambridge, 2012	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
64	Garced, 2012	Independent blind assessment	Yes	More than 10% attrition and missingness not examined	
65	Bork, 2012	Independent blind assessment	No	More than 10% attrition and missingness not examined	See coding of IYCF based on ICFI, and also note on increased snacking adding to meal frequency index
66	Matijasevich, 2011	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	Negative effect of maternal smoking on LAZ, but note association with higher WAZ and future obesity.
67	Durmus, 2011	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
68	Deierlein, 2011	Independent blind assessment	No	Less than 10% attrition or missingness examined	Excess GWG --> increased LAZ at 6 months
69	De Hoog, 2011	Independent blind assessment	No	Less than 10% attrition or missingness examined	
70	Moore, 2010	Independent blind assessment	Yes	No statement	
71	Ertel, 2010	Record linkage	Yes	Less than 10% attrition or missingness examined	
72	de Beer, 2010	Independent blind assessment	Yes	No statement	
73	Andersen, 2010	Record linkage	Yes	No statement	

ID		Critical appraisal: Outcome			Overall
#	First author, year	Assessment of outcome	Follow-up spans full 1st year	Adequate follow-up	Comments
74	Le Beaud, 2015	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	Note that they used GEE, which assumes data are MCAR, but they have shown that data were not MCAR, because complete follow-up to 36 months was associated with higher maternal education (confounder) and higher parasitic infection (exposure) between 0-24 months.
75	Katulla, 2014	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	India-slum. Good use of longitudinal data analysis methods. Bi-weekly morbidity surveillance. Note that SES doesn't seem to impact growth in an urban slum
76	Howe, 2012	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	
77	Johnson, 2012	Independent blind assessment	Yes	Less than 10% attrition or missingness examined	

Appendix 2.6 Frequency distribution of content signatures

Label	Metric name	Number of articles	%	Cumulative %
1	1 data point_Raw_Mean_Manual	2	2.6	2.6
2	1 data point_Standardized_Incremental change_Manual	1	1.3	3.9
3	1 data point_Standardized_Mean_Manual	16	20.78	24.68
4	1 data point_Standardized_Other_Manual	1	1.3	25.97
5	1 data point_Standardized_Proportion_Manual	2	2.6	28.57
6	2 data points_Raw_Conditional difference_Conditional regression	1	1.3	29.87
7	2 data points_Raw_Incremental change_Manual	1	1.3	31.17
8	2 data points_Raw_Incremental rate of change_Linear mixed effects model	1	1.3	32.47
9	2 data points_Raw_Incremental rate of change_Manual	1	1.3	33.77
10	2 data points_Standardized_Class_Threshold/ cut-off	1	1.3	35.06
11	2 data points_Standardized_Conditional difference_Conditional regression	2	2.6	37.66
12	2 data points_Standardized_Incremental change_Linear mixed effects model	1	1.3	38.96
13	2 data points_Standardized_Incremental change_Manual	4	5.19	44.16
14	2 data points_Standardized_Incremental rate of change_Manual	1	1.3	45.45
15	2 data points_Standardized_Proportional change_Manual	1	1.3	46.75
16	2 data points_Standardized_Velocity z-score_Manual	1	1.3	48.05
17	>2 data points_Raw_Incremental change_Pre-designed structural model	1	1.3	49.35
18	>2 data points_Raw_Incremental rate of change_Linear mixed effects model	5	6.49	55.84
19	>2 data points_Raw_Incremental rate of change_Non-linear mixed effects model	1	1.3	57.14
20	>2 data points_Raw_Instantaneous rate of change_Pre-designed structural model	1	1.3	58.44

Label	Metric name	Number of articles	%	Cumulative %
21	>2 data points_Raw_Mean_Generalized estimating equations	1	1.3	59.74
22	>2 data points_Raw_Mean_Linear mixed effects model	6	7.79	67.53
23	>2 data points_Raw_Mean_Other	1	1.3	68.83
24	>2 data points_Raw_Mean_Pre-designed structural model	1	1.3	70.13
25	>2 data points_Standardized_Incremental change_Generalized estimating equations	1	1.3	71.43
26	>2 data points_Standardized_Incremental change_Linear mixed effects model	1	1.3	72.73
27	>2 data points_Standardized_Incremental rate of change_Linear mixed effects model	2	2.6	75.32
28	>2 data points_Standardized_Incremental rate of change_Manual	1	1.3	76.62
29	>2 data points_Standardized_Instantaneous rate of change_Linear mixed effects model	1	1.3	77.92
30	>2 data points_Standardized_Mean_Generalized estimating equations	3	3.9	81.82
31	>2 data points_Standardized_Mean_Linear fixed-effects model	1	1.3	83.12
32	>2 data points_Standardized_Mean_Linear mixed effects model	9	11.69	94.81
33	>2 data points_Standardized_Mean_Non-linear mixed effects model	2	2.6	97.4
34	>2 data points_Standardized_Mean_Other	1	1.3	98.7
35	>2 data points_Standardized_Proportional rate of change_Other	1	1.3	100

Appendix 3

Appendix 3.1 Cohort protocol

Protocol

SNEHA Centres infant nutrition study

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The SNEHA Centres infant nutrition study is a prospective observational cohort study nested in an ongoing intervention program.

Background

Severe acute malnutrition in childhood has become steadily less common in India [1]. Malnutrition remains ubiquitous, however, with worrying implications for both short-term survival and longer-term wellbeing, economic growth, and socioeconomic inequalities [2]. An estimated 52 million children are stunted (height for age standard deviation [z] score <-2) [3]. Urban levels of childhood malnutrition are lower than rural, but the most recent National Family Health Survey (NFHS-3: 2005-6) described stunting in 40%, wasting (weight for height z score <-2 SD) in 17%, and low weight for age (<-2 SD) in 33% of urban children under 5 [4]. In the same survey, 47% of children from Mumbai slum areas were stunted, 16% wasted, and 36% had low weight for age [5]. Why this should be remains unresolved [6], although trans-generational, environmental, and dietary factors probably all play a part. There are questions about the underlying dynamics of nutrition in the face of substantial increases in gross national income per capita [1], and concerns about inequalities [7].

Ideas about the development of childhood malnutrition are also changing. Of critical importance is the window of vulnerability within which interventions may be effective. It has long been known that growth trajectories are set early in life, but recent work on the developmental origins of health and disease has focused attention on gestation and the first two years ('the 1000 days': see, for example, www.thousanddays.org). A second stimulus to rethinking has been the switch to classification using the World Health Organization (WHO) standards of 2006 (www.who.int/childgrowth/standards/en/). In a 2001 analysis of 39 national samples against National Center for Health Statistics (NCHS) standards, height for age declined until 24 months and then stabilized, weight for height declined until 15 months, and weight for age faltered rapidly from three to 12 months, followed by some catch-up [8]. However, a recent analysis of data from 54 countries, using the WHO standards, suggested that early growth faltering was more pronounced and that the window of opportunity included pregnancy and the first 24 months [9]. The authors pointed to "… a much greater problem of undernutrition during the first 6 months of life than previously believed, bringing coherence between the rates of undernutrition observed in young infants and the prevalence of low birth weight and early abandonment of exclusive breastfeeding" [9].

Rationale

During our previous trial of community mobilization to improve perinatal outcomes,[10] we collected anthropometric data from a sample of children followed up from birth. We described the proportions of underweight, stunting, and wasting in young children, and examined their relationships with age. We used two linked datasets: one based on institutional birth weight records for 17 318 infants, collected prospectively, and one based on follow-up of a subsample of 1941 children under five. 21% of infants had low weight for age z scores at birth (<-2 SD). At follow-up, 35% of young children had low weight for age, 17% low weight for height, and 47% low height for age. Downward change in weight for age was greater in children who had been born with higher z scores. At this stage, our data support the idea that much of growth faltering was explained by faltering in height for age, rather than by wasting. Stunting appeared to be established early and the subsequent decline in height for age was limited. Our findings suggest a focus on a younger age-group than the children over the age of three who are prioritized by existing support systems.

The study had shortcomings, however. We were unable to identify a downturn in growth during the first year. Either there was no downturn (which would conflict with the literature), or our reliance on two measures (birth weight and one follow-up weight) was too crude. In discussion with experts, the need is for more detailed information that helps us to track the growth of infants and locate them within their home context.

The current SNEHA Centres trial is a tremendous opportunity to understand the factors that influence child malnutrition in Mumbai's poorer groups. The SNEHA Centres program already includes nutritional intervention: all the children under 5 in 20 urban clusters are checked monthly and any with acute malnutrition are offered support through a daycare or home-based nutritional program.

Aims

General

To understand infant growth in Mumbai's slums, in detail and in context.

Specific

To enrol a cohort of infants for whom we have a detailed understanding of growth in the first year of life, and of contextual factors such as socioeconomic status, infant feeding, gender, parental anthropometry, water and sanitation.

Research questions

Primary

1. At what point does growth faltering begin in slum-dwelling children?
2. How does their growth relate to parental body size?

Secondary questions

1. What sort of diet do infants and young children have?
2. How does growth faltering relate to morbidity?
3. Is there a gender dimension to growth faltering, diet, morbidity and careseeking?

Methods/Design

Setting

The study will be done in 20 urban slum clusters of ~600 households in M/E and L wards, Mumbai. These clusters are already involved in the intervention arm of the SNEHA Centres cluster randomized controlled trial. Support for women's and children's health is provided by community organizers based at neighbourhood centres. Specifically, the SNEHA Centres offer dietary advice and intervention (at daycare centres and at home) for children under 5. Trained community investigators have already conducted a baseline census of all households in the trial clusters, and are familiar with the areas, their residents and the use of electronic data capture.

Design

A prospective observational cohort study nested within an ongoing intervention program. The requirements of the study are that investigators identify infants within each cluster as close to birth as possible, visit them at home, record their anthropometry, and collect questionnaire information from their mothers. Subsequently, investigators will visit monthly to record anthropometry, and to collect other information not covered in the initial questionnaire, including parental anthropometry.

Participants

Infants born to mothers in any of 20 SNEHA Centre slum clusters, over one year from 1st March 2013.

Inclusion criteria

- Family live in a SNEHA Centre cluster.
- Family say that they intend to stay in SNEHA Centres intervention cluster for at least 6 months.
- If weight not measured by Investigators within 72 hours of birth, possession of an institutional birth weight record.
- If birth outside the cluster, institutional birth weight record available and infant visited and weighed within 2 months.
- Live singleton infant born at 8 months gestation or greater.
- Infant born from 1st March 2013.

Data collection

The plan for data collection is summarized in **Figure 1**. Pregnancies will be identified, if possible, by SNEHA community organizers and investigators working in each cluster. Families and other community members will be encouraged to inform them when a baby is born. As soon after identification as possible, Investigators will

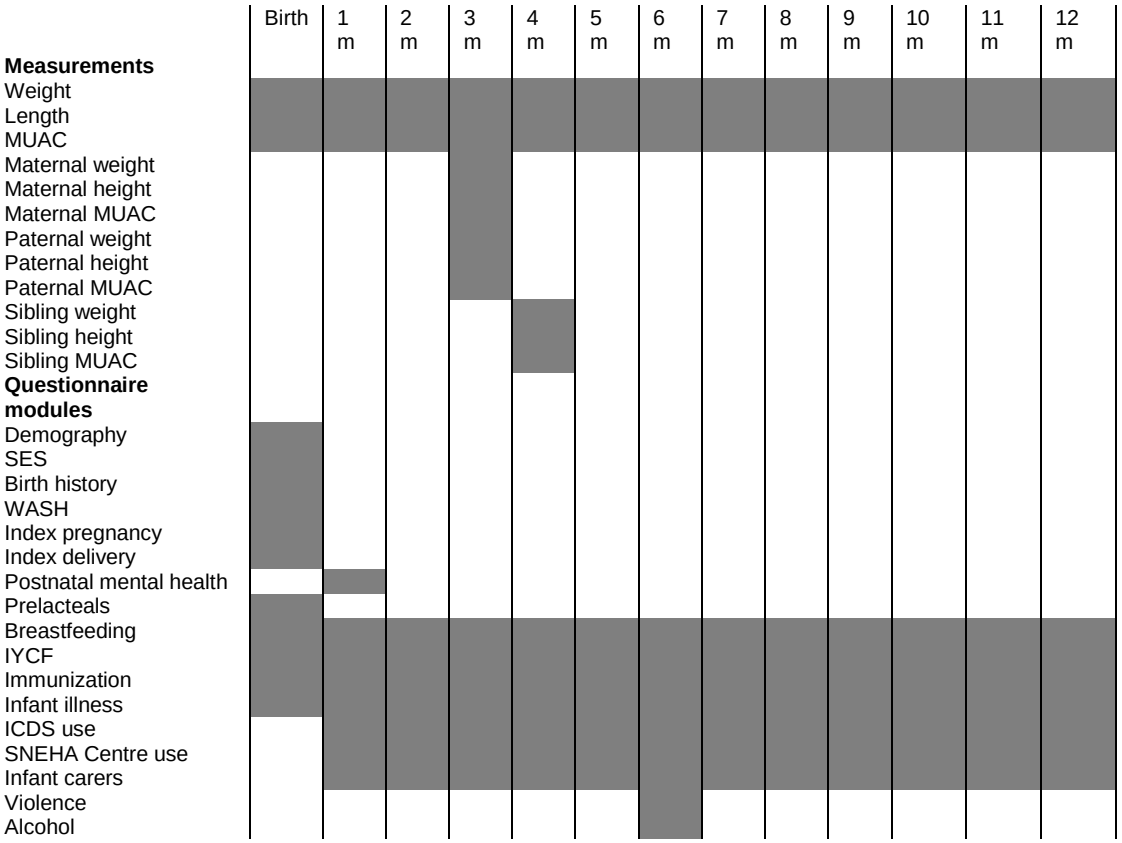
visit families at home and explain the idea of the study. If they meet the inclusion criteria, they will be given a participant information sheet and the parameters of the study will be described. Signed consent for participation will be taken.

Infant anthropometry will be taken in duplicate using electronic scales and length boards, with regular calibration and estimation of Technical Error of Measurement (TEM). Investigators have already been trained in the methods and have been using them for 18 months; they will have refresher training. Parental and sibling anthropometry will be done at families' convenience, although set at 3 months in the study plan as a reminder.

Questionnaire modules will follow existing validated formats as far as possible. Investigators have been administering questionnaire interviews for 18 months and will attend training sessions on the new questionnaires. Sensitive issues (domestic violence, alcohol use, which we think may be important with respect to infant growth) will be reserved for later visits, by which time mothers will be familiar with the process and team. If at the first visit a mother is unable to answer questions, investigators will measure her infant and will return within the next few weeks to collect the initial questionnaire modules.

Data will be entered electronically on site, using a system implemented in CommCare (Dimagi, Inc. Cambridge MA, USA) on Samsung smartphones. The system will include validation constraints within fields and between tables. SNEHA data managers will program and troubleshoot the system, which is already being used in other projects. Quality control will also be ensured through peer-review in work groups, supervision by project officers, and regular review of data.

Figure 1. Study diagram



Community Organizers working at SNEHA Centres, and field investigators working on the ongoing trial evaluation, will identify births within their catchment areas. We will try to measure weight and length of babies as soon as possible after birth, and then monthly. Any evidence of malnutrition will trigger support from the SNEHA Centres program. During monthly visits, information will be collected about demography, socioeconomic status, obstetric history, infant and young child feeding, maternal and paternal anthropometry, water and sanitation, and immunization.

Anthropometric data collected in the study will be fed into the process of community screening run by the SNEHA Centres in collaboration with the ICDS, in order to avoid duplication of data collection.

Outcomes

Sample size

The sample size (800) is predicated on the number of births expected annually (1000), and a loss to follow-up of 20%. Broadly, it will have a power of over 80% to describe a proportion of stunting of 45% within a two-sided precision of 5%. It compares favourably with the sizes of other cohorts.

Data management

After on-site entry on smartphones, data will be transferred electronically to a secure

cloud repository. Data will be checked online and after download for duplication and errors in key fields, and monitoring summaries will be produced through do-files written in Stata 12 (StataCorp, College Station, Tx: www.stata.com). The definitive dataset will contain numerical identifiers for cluster, household number, mother and infant. The names of heads of household and participants are collected during the interviews, and need to be retained so that Investigators can follow infants up. Identifying fields will be removed from the analytical dataset. Access to data will be restricted to the data manager and analysts. Datasets will be backed up weekly on a server and compact discs.

Analysis plan

Normally distributed continuous variables will be reported using mean and SD. Non-normally distributed variables will be reported using median and interquartile range, and transformed for inclusion in subsequent analyses. Categorical variables will be reported using frequencies and percentages. The collection of cohort data means that the resulting dataset will be rich and amenable to a range of analyses. An obvious first step will be to plot infant growth as twoway lines of weight, length, and z scores against month. This is likely to suggest a quadratic model. We will approach the analyses for our primary research questions as follows.

At what point does growth faltering begin in slum-dwelling children? We will plot infant weight, height, MUAC, and z scores against age in months, and generate smoothed growth curves. We will fit polynomial regression lines with confidence bands. Visual inspection of the resulting graphs will give an impression of when growth faltering begins. If there seems to be a breakpoint, we will consider regression discontinuity analysis.

How does infant growth relate to parental body size? We will compare infant size (z scores) at selected timepoints with parental body size using stratified summaries and regression models including terms describing parental body size as independent variables.

What sort of diet do infants and young children have? We will describe proportions of exclusive breastfeeding and IYCF core indicators. We will use these as dependent variables in logistic regression models (adjusted for clustering with a random effect) that examine the influence of factors such as sex and socioeconomic status.

How does growth faltering relate to morbidity? We will use a binomial variable describing growth faltering as the dependent variable in regression models that examine the influence of periods of illness. It will probably be best to express these in terms of infant-time, and therefore to use time-series models. We will also need to

adjust for repeated measures within each infant, probably through mixed models, but we will take expert advice on this.

Is there a gender dimension to growth faltering, diet, morbidity and careseeking? We will use dimensions of growth faltering, IYCF indicators, morbidity and careseeking as dependent variables in adjusted regression models that include sex as an independent variable.

Ethical considerations

Consent

Signed consent will be taken from mothers after explanation of the study and provision of a participant information sheet.

Risk

All the infants participating in the study will be eligible for support through the SNEHA Centres program. If an infant is observed to be faltering in growth, s/he will be supported through the system by the SNEHA Community Organizer who visits the household regularly. We also collaborate with the ICDS, to whom children can be referred.

We do not anticipate any risks associated with questionnaire data collection or anthropometry.

The only risk is that infants find the anthropometry upsetting, and that their parents become tired of the monthly follow-up. For this reason, we do not intend to measure skinfolds or take any biological samples.

A primary ethical issue in descriptive research of this nature is intervention to improve the situation. We have conceived this study as nested within an intervention. Infants identified during the study as having problems will be referred to the integrated SNEHA Centres program for nutritional intervention. Investigators will have daily contact with community organizers, and access to both day-care and home-based nutritional support. It is true that this should lead to improvement – making the study unrepresentative of communities in which intervention is not being undertaken – but we think that this is of secondary importance. Our main objective is to understand the development and dynamics of malnutrition in slum-dwelling infants, which we believe the study will do, and not to track malnutrition once it is manifest.

Approval

We will seek approval from the Multi-Institutional Ethics Committee hosted by the Anusandhan Trust, Mumbai.

Communication

We will share the findings with the community team within SNEHA and with representatives of the ICDS and MCGM, with whom we have regular contact.

We will present the findings at local, national and international meetings.

We will publish the findings in an open access peer-reviewed form.

We will share the findings with the participants and community members through regular community groups facilitated by SNEHA Community Organizers.

Abbreviations

ICDS: Integrated Child Development Services

IYCF: Infant and Young Child Feeding

NGO: non-government organization

SD: standard deviation

SNEHA: Society for Nutrition, Education and Health Action

UCL: University College London

References

1. Deaton A, Dreze J: Food and nutrition in India: facts and interpretations. *Economic & Political Weekly* 2009, 44:42-65.
2. Subramanyam MA, Kawachi I, Berkman LF, Subramaniam SV: Socioeconomic inequalities in childhood undernutrition in India: analyzing trends between 1992 and 2005. *PLoS One* 2010, 5:e11392.
3. Paul VK, Sachdev HS, Mavalankar D, Ramachandran P, Sankar MJ, Bhandari N, Sreenivas V, Sundararaman T, Govil D, Osrin D, Kirkwood B: Reproductive health, and child health and nutrition in India: meeting the challenge. *Lancet* 2011, 377:332-349.
4. Government of India Ministry of Health and Family Welfare: National Family Health Survey, India (NFHS-3 2005-06). Mumbai: International Institute for Population Sciences; 2007.
5. IIPS and Macro International: National Family Health Survey (NFHS-3), India, 2005-06: Maharashtra. Mumbai: International Institute for Population Sciences and Macro International; 2008.
6. Walton M: The political economy of India's malnutrition puzzle. *IDS Bull* 2009, 40:16-24.
7. Kanjilal B, Guha Mazumdar P, Mukherjee M, Rahman MH: Nutritional status of children in India: household socio-economic condition as the contextual determinant. *Int J Equity Health* 2010, 9.
8. Shrimpton R, Victora CG, de Onis M, Lima RC, Blossner M, Clugston G: Worldwide timing of growth faltering: implications for nutritional interventions. *Pediatrics* 2001, 107:E75.
9. Victora CG, de Onis M, Hallal PC, Blossner M, Shrimpton R: Worldwide timing of growth faltering: revisiting implications for interventions. *Pediatrics* 2010, 125:e473-e480.
10. Shah More N, Bapat U, Das S, Alcock G, Patil S, Porel M, Vaidya L, Fernandez A, Joshi W, Osrin D: Community mobilization in Mumbai slums to improve perinatal care and outcomes: a cluster randomized controlled trial. *PLoS Med* 2012, 9:e1001257.

Appendix 3.2 Participant information sheet and consent form in English

SNEHA Society for Nutrition, Education and Health Action
[REG. NO. E-17858(MUM)] every woman and child counts.

Participant Information Sheet and Consent Form for SNEHA Centres infant nutrition study

Introduction
Namaste.
I am ……..(write name). I work with the Society for Nutrition, Education and Health Action(SNEHA). SNEHA is an NGO working for the health of women and children in the slums of Mumbai. I have come to meet you regarding a SNEHA Centres study on infant health and nutrition.

Purpose of study
We are starting a study to understand the factors which affect the growth of babies in their first year of life. The study will be for a period of two years. Participants will be babies and their mothers, who will be followed up for one year from 1st April 2013 in areas covered by 20 SNEHA Centres in M East and L ward.

We will identify babies as soon as possible after they are born, measure and weigh them at home, and collect some information from the mother about her household and the people who live there, her previous pregnancies and births, and water and sanitation in the community. Subsequently, we will visit every month to collect information on child feeding, immunization and illnesses. We will measure the height and weight of the baby. We will also collect information on postnatal depression and domestic violence once mothers are familiar with the process and the team. It will take about half an hour to do the measuring and answer the questions every month.

We invite you to participate in this study. It is important that you thoroughly understand your role in it and also the benefits and risks to you for participating. Please give your consent to participate only if you have completely understood the nature of this survey and if you are aware of your rights as a participant.

Benefits and risks
There will be no harm to you for participating in this survey. You will get updated information about your child's growth every month. In case of illness, we will refer you to the nearest health post or SNEHA Centre for medical advice and treatment.

You will receive help and support from SNEHA's Community Organizers during their regular home visits. You will spend some time in giving us information, which will help us to understand the health needs of children better so that we can help improve the health of children in your community.

Participation
Participation in this study is totally voluntary. You can refuse to participate or to leave the process at any point. If you do this, you will not pay any penalty or be denied any service by the SNEHA Centre or any other agency. We will not be paying anyone for their consent or participation in the study.

Confidentiality
All information you give will be kept confidential. We will collect the information on mobile phones and it will be stored in a computer. Your name will be removed so that nobody knows what you said.

The'SNEHA Centre' project has received ethical approvals from the Multi Institutional Ethics Committee of the Anusandhan Trust and University College of London.

We request your written consent to participate in the study. If you have any questions about the project or need any kind of help, you can contact me or Sushmita Das at SNEHA's office (Ground floor, Gurunanak Kutia, Plot No. 85, Sindhi Society, Chembur, Mumbai 400 074) or on telephone number 25220268.

INFORMED CONSENT FORM

Please read and place a ✓or X against the following statements before giving consent:

1	I have been informed by the investigators about the study including the nature, period, objective and benefits and risks and I have understood them.	
2	I have been given the opportunity to ask questions and have been given satisfactory responses.	
3	I understand that I am free to participate or not to participate in the study.	
4	I understand that, I can withdraw at any point in the interview and the study and if I withdraw from the study, I can decide whether the information I have shared may be used in the final study.	
5	I understand that I will not be penalized for refusing to participate in this interview or for withdrawing from the study.	
6	I understand that the information that I share will be only used for the purposes of SNEHA's project.	
7	I understand that my personal information will be treated as strictly confidential.	
8	I understand that the information I provide may be published in a report or study and that confidentiality and anonymity will be maintained so that it will not be possible to identify me or any individual in the community.	

Do you agree to participate in this survey? Yes ☐ No ☐

(If No, document the reasons and leave after thanking the person).

Signature: Thumb Impression (Left):

Name:

Address:

Date:

Appendix 3.3 Cohort study questionnaire

SNEHA Centre Nutrition Study Questionnaire

Module 1: Household registration				
Question	**Field**	**Type**	**Constraint/options**	**Skip**
Enter your ID number	interviewerid	Number	1 to 12	
Enter the cluster number	clusterid	Number	1 to 40	
Enter the household number	hhid	Number	1 to 1200	
What is the name of the head of household?	hhname	Text	2-50 chars	
No child found after 3 visits	noinfant	Select 1	1 Infant found 0 Infant not found after 3 visits	 End registration
Interviewer: Read the participant information sheet to the respondent	studyinfo	Text	Read only	
Do you agree to participate in the study?	respconsent	Select 1	1 Yes: Take signature 0 No	 End registration
Enter the respondent ID	respid	Number	1 to 9	
Ideally respondent should always be mother. Enter 9 if mother is not alive				
Respondent's name	respname	Text		

Module 1: Household registration				
Question	**Field**	**Type**	**Constraint/options**	**Skip**
Index child date of birth	cdob1	Date		
Index child sex	csex1	Select 1	1 male 2 female	
Index child's ID number	childid1	Number	1 to 2	
Click the button to record the location of the household	gps	Location		

Module 2: Household information				
Question	**Field**	**Type**	**Constraint/options**	**Skip**
Date of interview (HIDDEN)	datestamp	Date		
Interview start time (HIDDEN)	timestart	Time		
Respondent's relation to the child *ideally interview the mother*	resprel	Select 1	1 Mother	
			2 Grandmother	
			3 Aunt	
			4 Sibling	
			5 Father	
			6 Other caretaker	
Say to respondent: I am going to ask you about the people who live in your house.	hhintro	Text	Read only	

The household				
Mother of the index child available	mother	Select 1	1 Yes	Go to father
			0 No	
Why not?	mstayreason	Select 1	1 Not alive	
			2 Separated	
			3 Works elsewhere	
Father is available?	father	Select 1	1 Yes	Go to ownkids
			0 No	
Why not?	fstayreason	Select 1	1 Not alive	
			2 Separated	
			3 Works elsewhere	
How many of her own children live here? *Including the index one*	ownkids	Number	1 to 15	
How many other men over 18 live here?	othermales	Number	0 to 8	To be asked if respid is 1 or 9
How many other women over 18 live here?	otherfemales	Number	0 to 8	To be asked if respid is 1 or 9
How many other children (not her own) live here?	otherchildren	Number	0 to 15	To be asked if respid is 1 or 9
Father's age	fage	Number	18 to 55	fedu to fsmoke will be asked even if fstayreason is 1 or 2
Father's highest school class	fedu	Number	0 to 17	
Father's livelihood	focc	Select 1	77 Does not work	
			88 Student	
			1 Unskilled work, like pheriwalla, domestic servant, watchman, labourer	

The household				
			2 Plant or machine operator or assembler , or driver	
			3 Skilled craftsperson like potter, tailor, plumber, electrician, jewellery maker	
			4 Agriculture or fishery worker	
			5 Service worker, shop or market sales worker, Ayah, caterer, bus conductor	
			6 Clerk in an office, computer operator, typist	
			7 Technician, KG or tprimary school teacher, nurse, dai	
			8 Professional (Doctor, lawyer, engineer, school or college teacher, pandit, moulvi)	
			9 High level government job (Legislator, senior official or manager, corporator)	
Father smokes	fsmoke	Select 1	1 Yes	
			0 No	
Mother's age	mage	Number	15 to 49	mage to msmoke will be asked even if mstayreason is 1 or 2
Mother's age at marriage	magemarriage	Number	10 to 40	
Mother's age at first pregnancy	magepreg	Number	10 to 49	
Mother's age at index pregnancy	mageindex	Number	10 to 49	
Mother’s highest school class	medu	Number	0 to 17	
Mother’s livelihood	mocc	Select 1	77 Does not work	

The household				
			88 Student	
			1 Unskilled work, like pheriwalla, domestic servant, watchman, labourer	
			2 Plant or machine operator or assembler , or driver	
			3 Skilled craftsperson like potter, tailor, plumber, electrician, jewellery maker	
			4 Agriculture or fishery worker	
			5 Service worker, shop or market sales worker, Ayah, caterer, bus conductor	
			6 Clerk in an office, computer operator, typist	
			7 Technician, KG or tprimary school teacher, nurse, dai	
			8 Professional (Doctor, lawyer, engineer, school or college teacher, pandit, moulvi)	
			9 High level government job (Legislator, senior official or manager, corporator)	
Mother smokes	msmoke	Number	1 Yes	
			0 No	
Have you had any children who died?	dchild	number	0 to10	If 0, go to Misctopyn
Enter number of children who died. If none, enter 0				
Child 1 sex	Dcsex1	number	1 male	
			2 female	

The household				
Child 1 date of birth	Dcdob1	Date		
Child 1 how long ago did s/he die (years)?	Dcyago1	number	Years	
Child 1 was she born alive or had she already died when she was born?	Dcstill1	number	1 stillbirth 0 born alive	
Child 1 how old was s/he when she died (years)?	Dcdage1	number	Years	
Child 2 sex	Dcsex2	number	1 male 2 female	
Child 2 date of birth	Dcdob2	Date		
Child 2 how long ago did s/he die?	Dcyago2	number	0 to 15	
Child 2 was she born alive or had she already died when she was born?	Dcstill2	number	1 stillbirth 0 born alive	
Child 2 how old was s/he when she died (years)?	Dcdage2	number	Years	
Child 3 sex	Dcsex3	number	1 male 2 female	
Child 3 date of birth	Dcdob3	Date		
Child 3 how long ago did s/he die?	Dcyago3	number	Years	
Child 3 was she born alive or had she already died when she was born?	Dcstill3	number	1 stillbirth 0 born alive	

The household				
Child 3 how old was s/he when she died (years)?	Dcdage3	number	Years	
Have you had any miscarriages or terminations?	Misctopyn	number	0 to 10	Go to religion if Misctopyn==0
Enter number of miscarriages or terminations. If none, enter 0				
How long ago was your 1st miscarriage or termination	Misctopago1	number	Years	
How long ago was your 2nd miscarriage or termination	Misctopago2	number	Years	
How long ago was your 3rd miscarriage or termination	Misctopago3	number	Years	
How long ago was your 4th miscarriage or termination	Misctopago4	number	Years	
How long ago was your 5th miscarriage or termination	Misctopago5	number	Years	religion to stoiletsharenwill be skipped if respid>=2 but will be asked if respid==9

Socioeconomic status of the household				
What is the main religion in the household?	religion	number	1 Muslim	
			2 Hindu	
			99 Other	

Socioeconomic status of the household				
How long has the family been living in Mumbai? *Enter number of years. If <1 year, enter 0*	mumdur	number	Years	
Do your family own or rent your home?	hown	Select 1	1 Yes	
			0 No	Go to house
Do you have a ration card?	rationcard	Select 1	1 Yes	
			0 No	
What colour is your ration card?	rationcardcolour	Select 1	1 White	
			2 Yellow	
			3 Orange	
Interviewer: select the type of house the respondent lives in	house	Select 1	1 Pucca	
			2 Semi-pucca	
			3 Kaccha	
Do you own any of the following household items?	assetlist	Text	Read only	
	assetlist-mattress	Multi-Select Question	1 Mattress	
	assetlist-presscook		2 Pressure cooker	
	assetlist-gascylinder		3 Gas cylinder	
	assetlist-stove		4 Stove	
	assetlist-chair		5 Chair	
	assetlist-bed		6 Bed	

Socioeconomic status of the household				
	assetlist-table		7 Table	
	assetlist-clock		8 Clock	
	assetlist-elecfan		9 Electric fan	
	assetlist-mixer		10 Mixer	
	assetlist-radio		11 Radio	
	assetlist-phone		12 Phone (landline or mobile)	
	assetlist-fridge		13 Fridge	
	assetlist-tv		14 TV	
	assetlist-bicycle		15 Bicycle	
	assetlist-twowheeler		16 Two-wheeler	
	assetlist-car		17 Car	
What type of electricity supply does your home have?	elec	Select 1	0 None	
			1 Metered, family pay bill	
			2 Pay landlord	
			3 Other	
Interviewer: select the type of flooring in the home	floor	Select 1	0 Dirt, sand, mud	
			1 Concrete, brick, mud, tiled	
What fuel do you mainly use for cooking?	fuel	Select 1	0 Wood, charcoal, dung	
			1 Kerosene, LPG	
			2 Electricity	
			3 does not cook at home	

Questions about drinking-water				
What is the main source of drinking-water for members of your household?	dwater	Select 1	1. Piped water into dwelling	
			2. Piped water to yard/plot	
			3. Public tap/standpipe	
			4. Tubewell/borehole	
			5. Protected dug well	
			6. Unprotected dug well	
			7. Protected spring	
			8. Unprotected spring	
			9. Rainwater collection	
			10. Bottled water	
			11. Cart with small tank/drum	
			12. Tanker-truck	
			13. Surface water (river, dam, lake, pond, stream, canal, irrigation channel)	go to dwtreat if 1 or 2
			14. Other (specify)	
If main source is bottledwater	dwbottle	Select 1	1. Piped water into dwelling	
What is the main source of water used by your household for other purposes, such as cooking and handwashing?			2. Piped water to yard/plot	
			3. Public tap/standpipe	
			4. Tubewell/borehole	
			5. Protected dug well	
			6. Unprotected dug well	
			7. Protected spring	
			8. Unprotected spring	

Questions about drinking-water				
			9. Rainwater collection	
			11. Cart with small tank/drum	
			12. Tanker-truck	
			13. Surface water (river, dam, lake, pond, stream, canal, irrigation channel)	
			14. Other (specify)	go to dwtreat
How long does it take to go there, get water, and come back?	dwtrip	number	1. Number of minutes	
For one trip				
Excludes socializing			99. Don't know	
Who usually goes to this source to fetch water for your household?	dwtripwho	Select 1	1. Adult woman 15+	
Probe: is the person under 15? What sex?			2. Adult man 15+	
			3. Female child <15 y	
			4. Male child <15 y	
			99. Don't know	
Do you treat your water in any way to make it safer to drink?	dwtreat	Select 1	1. Yes	
			2. No	go to stoilet
			99. Don't know	go to stoilet

Questions about drinking-water				
What do you usually do to the water to make it safer to drink?	dwtreathow-boil	Multi-Select Question	1. Boil	
Probe: anything else?	dwtreathow-bleach		2. Add bleach/chlorine	
Can tick more than one	dwtreathow-strain		3. Strain it through a cloth	
	dwtreathow-filter		4. Use a water filter (ceramic, sand, composite)	
	dwtreathow-solar		5. Solar disinfection	
	dwtreathow-stand		6. Let it stand and settle	
	dwtreathow-other		7. Other (specify)	
	dwtreathow-dknow		99. Don't know	

Questions about sanitation				
What kind of toilet facility do members of your household usually use?	stoilet	Select 1	1Flush/pour flush to:	
If flush or pour flush, ask where does it flush to?			1.1 Piped sewer system	
			1.2. Septic tank	
			1.3. Pit latrine	
			1.4. Elsewhere	

Questions about sanitation				
			1.5. Don't know	
			6. Ventilated improved pit latrine (VIP)	
			7. Pit latrine with slab	
			8. Pit latrine without slab/open pit	
			9. Composting toilet	
			10. Bucket	
			11. Hanging toilet/hanging latrine	
			12. No facilities or field or road	go to faeces
			13. Other (specify)	
Do you share this facility with other households?	stoiletshare	Select 1	Yes	
			No	
How many households use this toilet facility?	stoiletsharen	number		
			99. Don't know	
Disposal of children's faeces				
The last time [the baby] passed stools, what was done to dispose of the stools?	faeces	Select 1	1. Child used toilet/latrine (= sanitary)	
Ask for EACH child under 3 y			2. Put/rinsed into toilet or latrine (= sanitary)	
			3. Put/rinsed into drain or ditch	
			4. Thrown into garbage	
			5. Buried (= sanitary)	
			6. Left in the open	

Questions about sanitation				
			7. Other (specify)	
			8. Don't know	

Index infant maternity				
Please tick the statement which most applies to you:				
In the month that I became pregnant.....	unplannedcontra	Select 1	1 I/we were not using contraception	
			2 I/we were using contraception, but not on every occasion	
			3 I/we always used contraception, but knew that the method had failed (i.e. broke, moved, came off, came out, not worked etc) at least once	
			4 I/we always used contraception	
In term s of becoming a mother (first time or again), I feel that my pregnancy happened at the.....	unplannedtime	Select 1	1 right time	
			2 ok, but not quite right time	
			3 wrong time	
Just before I became pregnant.....	unplannedwant	Select 1	1 I intended to get pregnant	
			2 my intentions kept changing	

Index infant maternity				
			3 I did not intend to get pregnant	
Just before I became pregnant....	unplannedbaby	Select 1	1 I wanted to have a baby	
			2 I had mixed feelings about having a baby	
			3 I did not want to have a baby	
In the next question, we ask about your partner:				
Before I became pregnant....	unplannedagree	Select 1	1 My partner and I had agreed that we would like me to be pregnant	
			2 My partner and I had discussed having children together, but hadn't agreed for me to get pregnant	
			3 We never discussed having children together	
Before you became pregnant, did you do anything to improve your health in preparation for pregnancy? *Please tick all that apply*	unplanned-folicacid	Multi select question	1 took folic acid	
	unplanned-smoke		2 stopped or cut down smoking	
	unplanned-alcohol		3 stopped or cut down drinking alcohol	
	unplanned-atehealthy		4 ate more healthily	

Index infant maternity				
	unplanned-consult		5 sought medical/health advice	
	unplanned-other		6 took some other action Specify	
	unplanned-none		7 I did not do any of the above before my pregnancy	
Did you have antenatal care at a health facility?	ancinst	Select 1	1 Yes	
			0 No	Go to delmumbai
At which facility did you have antenatal care?	ancsite	Select 1	1 BMC health post	
			2 BMC maternity home	
			3 BMC hospital	
			4 Private practitioner	
			5 Private hospital	
			6 Government hospital	
			7 Urban health centre	
Did you have the baby in Mumbai or outside?	delmumbai	Select 1	1 Mumbai	
			2 Outside Mumbai	
Did you give birth in a health facility or at home?	delsite	Select 1	1 Facility	
			0 Home	Go to delgest
At which facility did you give birth?	delfac	Select 1	2 BMC maternity home	
			3 BMC hospital	
			4 Private practitioner	
			5 Private hospital	

Index infant maternity				
			6 Government hospital	
			7 Urban health centre	
At what gestation was your baby born? *In months (if less than 8 months, end questionnaire)*	delgest	Select 1	6 to 9	End questionnaire

Prelacteal and breastfeeding initiation				
Have you ever breastfed (NAME)?	bfever	Number	1 Yes	
			0 No	Go to bfprelac
How long after birth did you first put (NAME) to the breast?	bfstart	Number	Number must be <24 or 88 or 99	Go to bfstarted if 88
Enter number in hours. If <1hr, enter 0. If >24hrs, enter 88. If unknown, enter 99				
Enter the number of days	bfstartd	Number		
In the first three days after delivery, was (NAME) given anything to drink other than breast milk?	bfprelac	Number	1 Yes	
			0 No	Go to qcomplete
Which of the following was (NAME) given to drink?	bfprelactype	Text	Read only	

Prelacteal and breastfeeding initiation				
Milk other than breast milk	bfprelacmilk	Select 1	1 Yes	
			0 No	
Plain water	bfprelacwater	Select 1	1 Yes	
			0 No	
Sugar or glucose water	bfprelacsugar	Select 1	1 Yes	
			0 No	
Gripe water	bfprelacgripe	Select 1	1 Yes	
			0 No	
Sugar-salt-water solution	bfprelacsugsalt	Select 1	1 Yes	
			0 No	
Fruit juice	bfprelacjuice	Select 1	1 Yes	
			0 No	
Infant formula	bfprelacformula	Select 1	1 Yes	
			0 No	
Tea	bfprelactea	Select 1	1 Yes	
			0 No	
Honey	bfprelachoney	Select 1	1 Yes	
			0 No	
JanamGhutti	bfprelacjanam	Select 1	1 Yes	
			0 No	
Other	bfprelacother	Select 1	1 Yes	
			0 No	
Questionnaire complete	qcomplete	Select 1	0 Not complete	

Prelacteal and breastfeeding initiation				
(HIDDEN)			1 Complete	
Questionnaire end time (HIDDEN)	timestamp	Time	Current time	

Module 3: IYCF module for subsequent interviews (to be filled every month)				
Question	**Field**	**Type**	**Constraint**	**Skips**
Does anyone else look after the baby for the mother?	caretaker	Select 1	1 Yes	
			0 No	Go to feed
Who looks after the baby?	caretakerrel	Select 1	1 Grandmother	
			2 Aunt	
			3 Sibling	
			4 Father	
			5 Other caretaker	
How many hours a day?	caretakerhhs	Number		
How do you/they feed the baby?	feedmethod	Select 1	1 Only breastfeeding	
			2 Bottle	
			3 Spoon	

Module 3: IYCF module for subsequent interviews (to be filled every month)				
Question	**Field**	**Type**	**Constraint**	**Skips**
			4 Finger /hand	
			5 Cotton wick	
			6 Other	
Are you still breastfeeding (NAME)?	bfnow	Select 1	1 Yes	
			0 No	Go to bmeds
How many times did you breastfeed (NAME) last night between sunset and sunrise?	bfnightx	Number	0-10	
How many times did you breastfeed (NAME) yesterday during the daylight hours?	bfdayx	Number	0-10	
Was (NAME) given any vitamin drops or other medicine as drops yesterday during the day or at night?	bmeds	Select 1	1 Yes	
			0 No	
Next I would like to ask you about some liquids that (NAME) may have had yesterday during the day or at night. Did (NAME) have any of the following?	bfluid	Text	Read only	
Plain water	bfluidwater	Select 1	1 Yes	
			0 No	
			99 Don't know	
Infant formula such as Lactogen	bfluidformula	Select 1	1 Yes	
			0 No	

Module 3: IYCF module for subsequent interviews (to be filled every month)				
Question	**Field**	**Type**	**Constraint**	**Skips**
			99 Don't know	
Other milk such as tinned, powdered or fresh animal milk	bfluidmilk	Select 1	1 Yes	
			0 No	
			99 Don't know	
Lassi, chaas or other yoghurt drinks	bfluidyoghurt	Select 1	1 Yes	
			0 No	
			99 Don't know	
How many times did (NAME) have milk of any kind yesterday during the day or at night? *If 7 or more times, record 7*	bfluidmilkx	Number	0-7	if bfluidformula=1 or bfluidmilk=1
Fruit juice	bfluidjuice	Select 1	1 Yes	
			0 No	
			99 Don't know	
Clear broth	bfluidbroth	Select 1	1 Yes	
			0 No	
			99 Don't know	
Tea or coffee	bfluidtea	Select 1	1 Yes	
			0 No	
			99 Don't know	
Cold drinks such as Pepsi, Coke and Frootie	bfluidcolddrink	Select 1	1 Yes	
			0 No	

Module 3: IYCF module for subsequent interviews (to be filled every month)				
Question	**Field**	**Type**	**Constraint**	**Skips**
			99 Don't know	
Any other liquids	bfluidother	Select 1	1 Yes	
			0 No	
			99 Don't know	
Have you ever given (NAME) any kind of solid foods, or semi-solid food?	food	Select 1	1 Yes	
			0 No	Go to vcard
I would like to ask you about the food (NAME) ate yesterday during the day or at night, either separately or combined with other foods. Did (NAME) eat any of the following?	foodcomb	Text	Read only	
Commercial baby food like Cerelac or Farex	foodbabyfood	Select 1	1 Yes	
			0 No	
			99 Don't know	
Porridge, bread, roti, chapatti, rice, noodles, idli, or any other foods made from grains	foodgrain	Select 1	1 Yes	
			0 No	
			99 Don't know	
Pumpkin, carrots, sweet potatoes that are yellow or orange inside	foodorange	Select 1	1 Yes	
			0 No	
			99 Don't know	
White potatoes, white yams, cassava, or any other foods made from roots	foodwhite	Select 1	1 Yes	
			0 No	
			99 Don't know	

Module 3: IYCF module for subsequent interviews (to be filled every month)				
Question	**Field**	**Type**	**Constraint**	**Skips**
Dark green leafy vegetables	foodglv	Select 1	1 Yes	
			0 No	
			99 Don't know	
Ripe mangoes, papayas, cantaloupe or jackfruit	foodredfruit	Select 1	1 Yes	
			0 No	
			99 Don't know	
Other fruits or vegetables	foodotherfruitveg	Select 1	1 Yes	
			0 No	
			99 Don't know	
Liver, kidney, heart or other organ meats	foodorgan	Select 1	1 Yes	
			0 No	
			99 Don't know	
Chicken, duck or other birds	foodpoultry	Select 1	1 Yes	
			0 No	
			99 Don't know	
Other meat	foodothermeat	Select 1	1 Yes	
			0 No	
			99 Don't know	
Eggs	foodegg	Select 1	1 Yes	
			0 No	
			99 Don't know	
Fresh or dried fish or shellfish	foodfish	Select 1	1 Yes	

Module 3: IYCF module for subsequent interviews (to be filled every month)				
Question	**Field**	**Type**	**Constraint**	**Skips**
			0 No	
			99 Don't know	
Foods made from beans, peas or lentils?	foodpulse	Select 1	1 Yes	
			0 No	
			99 Don't know	
Nuts	foodnuts	Select 1	1 Yes	
			0 No	
			99 Don't know	
Cheese, yoghurt or other milk products	fooddairy	Select 1	1 Yes	
			0 No	
			99 Don't know	
Food made with oil, fat, ghee or butter	foodfat	Select 1	1 Yes	
			0 No	
			99 Don't know	
Sugary foods such as chocolates, sweets, candies, pastries, cakes or biscuits	foodsugary	Select 1	1 Yes	
			0 No	
			99 Don't know	
Nalli or wafers such as Lays, Kurkure, and Pogo	foodwafer	Select 1	1 Yes	
			0 No	
			99 Don't know	
VadaPav	foodvadapav	Select 1	1 Yes	
			0 No	

Module 3: IYCF module for subsequent interviews (to be filled every month)				
Question	**Field**	**Type**	**Constraint**	**Skips**
			99 Don't know	
Maggi noodles	foodmaggi	Select 1	1 Yes	
			0 No	
			99 Don't know	
Any other solid or semi-solid food	foodother	Select 1	1 Yes	
			0 No	
			99 Don't know	
How many times did (NAME) eat solid, semi-solid, or soft foods other than liquids yesterday during the day or at night? *If 7 or more times, record 7*	foodx	Number	0-7	

Immunization				
Do you have the vaccination card?	vcard	Select 1	1 Yes	
			0 No	
BCG (after birth)	bcg	Select 1	1 Yes	
			0 No	
			99 Don't know	
Polio 0 (after birth)	polio0	Select 1	1 Yes	
			0 No	
			99 Don't know	

Immunization				
Hepatitis B (after birth)	Hepb0	Select 1	1 Yes	
			0 No	
			99 Don’t know	
Polio -1 (after 6 week)	polio1	Select 1	1 Yes	
			0 No	
			99 Don’t know	
DPT-1 (after 6 week)	dpt1	Select 1	1 Yes	
			0 No	
			99 Don’t know	
Hepatitis(B-1) (after 6 week)	hepb1	Select 1	1 Yes	
			0 No	
			99 Don’t know	
Polio-2 (after 10 week)	polio2	Select 1	1 Yes	
			0 No	
			99 Don’t know	
DPT-2 (after 10 week)	dpt2	Select 1	1 Yes	
			0 No	
			99 Don’t know	
Hepatitis(B-2) (after 10 week)	hepb2	Select 1	1 Yes	
			0 No	
			99 Don’t know	
Polio-3 (after 14 week)	polio3	Select 1	1 Yes	
			0 No	
			99 Don’t know	

Immunization				
DPT 3 (after 14 week)	dpt3	Select 1	1 Yes	
			0 No	
			99 Don’t know	
Hepatitis(B-3) (after 14 week)	hepb3	Select 1	1 Yes	
			0 No	
			99 Don’t know	
Measles (After 9 month)	measles	Select 1	1 Yes	
			0 No	
			99 Don’t know	
Vitamin A First dose (9 Month)	vitamina1	Select 1	1 Yes	
			0 No	
			99 Don’t know	
MMR (after 15 month)	mmr	Select 1	1 Yes	
			0 No	
			99 Don’t know	
Vitamin A second dose (after18 month)	vitamina2	Select 1	1 Yes	
			0 No	
			99 Don’t know	
Booster Polio (after 18 month)	bpolio	Select 1	1 Yes	
			0 No	
			99 Don’t know	
Booster DPT-1 (after 18 month)	boosterdpt1	Select 1	1 Yes	
			0 No	
			99 Don’t know	

Immunization				
Booster DPT-2 (after 18 month)	boosterdpt2	Select 1	1 Yes	
			0 No	
			99 Don’t know	
Vitamin A third dose (after 24 months)	vitamina3	Select 1	1 Yes	
			0 No	
			99 Don’t know	
Vitamin A forth dose (after 30 month)	vitamina4	Select 1	1 Yes	
			0 No	
			99 Don’t know	
Deworming	dworm	Select 1	1 Yes	
			0 No	Go to diarr
			99 Don’t know	Go to diarr
When deworming given (If less than 1 month enter 0, don't know enter 99))	dwormtimes	Integer Number		

Infant illness and treatment				
Has [Name] had diarrhea in the last month?	diarr	Select 1	1 Yes	
			0 No	
How many days did the diarrhea last?	diarrdays	Number		
Is the diarrhoea better or still going?	diarrcondition		1 Better	
			2 Still going	

Infant illness and treatment				
How much was [name] given to drink during the diarrhea? Was s/he given less than usual to drink, about the same amount, or more than usual to drink?	diarrfluid		1 Less	
			2 Usual	
			3 More	
Did you seek advice or treatment for the diarrhea from any source?	diarrconsult		1 Yes	
			0 No	Go to diarrors
Where did you seek advice or treatment?	diarrfacility	Select 1	1 BMC health post	
			3 BMC hospital	
			4 Private practitioner	
			5 Private hospital	
			6 Government hospital	
			7 Urban health centre	
Did [name] have to stay in hospital?	diarrstay	Select 1	1 Yes	
			0 No	
For how many days?	diarrstaydays	Number	days	
Has [name] been given any fluid from a special packet called ORS (local name)?	diarrors	Select 1	1 Yes	
			0 No	

Infant illness and treatment				
Has [name] been given any gruel made from rice or other grain?	diarrgruel	Select 1	1 Yes	
			0 No	
Has [name] been given anything else to treat the diarrhea?	diarrmed	Select 1	1 Yes	
			0 No	Go to fever
What else?	diarrmed-santibiotic	Multi- select question	1 Syrup: antibiotic	
	diarrmed-santimotility		2 Syrup: antimotility	
	diarrmed-szinc		3 Syrup: zinc	
	diarrmed-sother		4 Syrup: other	
	diarrmed-sunknown		5 Syrup: unknown	
	diarrmed-iantibiotic		6 Injection: antibiotic	
	diarrmed-inonantibiotic		7 Injection: non-antibiotic	
	diarrmed-iunknown		8 Injection: unknown	
	diarrmed-iv		9 Intravenous (IV)	
	diarrmed-herbal		10 Home remedy/herbal medicine	
Has [name] been ill with a fever in the last month?	fever	Select 1	1 Yes	
			0 No	
Has [name] been ill with a cough in the last month?	cough	Select 1	1 Yes	

Infant illness and treatment				
			0 No	
When [name] had the illness with a cough, did s/he breathe faster than usual with short, rapid breaths or have difficulty breathing?	diffbreath	Select 1	1 Yes	
			0 No	
When [name] had the illness, did s/he have a problem in the chest or a blocked or runny nose?	probchest	Select 1	1 Chest only	
			2 Nose only	
			3 Both	
			99 Don't know	
How many days did the cough or breathing problem last?	coughdays	Number		
Is the cough or breathing problem better or still going?	coughcondition	Select 1	1 Better	
			2 Still going	
How much was [name] given to drink during the illness? Was s/he given less than usual to drink, about the same amount, or more than usual to drink?	coughfluid	Select 1	1 Less	
			2 Usual	
			3 More	

Infant illness and treatment				
Did you seek advice or treatment for the illness from any source?	coughconsult	Select 1	1 Yes	
			0 No	Go to illmed
Where did you seek advice or treatment?	coughfacility	Select 1	1 BMC health post	
			3 BMC hospital	
			4 Private practitioner	
			5 Private hospital	
			6 Government hospital	
			7 Urban health centre	
Did [name] have to stay in hospital?	coughstay	Select 1	1 Yes	
			0 No	
For how many days?	coughstaydays		days	
Has [name] been given anything to treat the illness?	coughmed	Select 1	1 Yes	
			0 No	Go to illother
What else?	coughmed-santibiotic		1 Syrup: antibiotic	
	coughmed-santimalerial		2 Syrup: antimalarial	
	coughmed-sother		3 Syrup: other	
	coughmed-unknown		4 Syrup: unknown	
	coughmed-iantibiotic		5 Injection: antibiotic	
	coughmed-inonantibiotic		6 Injection: non-antibiotic	
	coughmed-iunknown		7 Injection: unknown	

Infant illness and treatment				
	coughmed-iv		8 Intravenous (IV)	
	coughmed-herbal		9 Home remedy/herbal medicine	
Has [name] had any other illness in the last month?	illother	Select 1	1 Yes	
			0 No	Go to icds
What sort of illness?	illtype-rash	Multi-Select Question	1 Rash	
	illtype-vomiting		2 Vomiting	
	illtype-skin		3 Skin infection	
	illtype-ear		4 Ear infection	
	illtype-jaundice		5 Jaundice	
	illtype-stomach		6 Stomach problem	
	illtype-urine		7 Urine problem	
	illtype-fits		8 Fits or seizures	
	illtype-injury		9 Injury	
In the last one month, has (NAME) got any benefits from the anganwadi or ICDS centre?	icds	Select 1	1 Yes	
			0 No	Go to sneha
What were the services received?	icds-growth		1 Growth monitoring	
	icds-food		2 Food supplement	
	icds-medscreening		3 Medical screening	
	icds-imm		4 Immunization	
In the last one month, has (NAME) got any services from SNEHA centre?	sneha	Select 1	1 Yes	

Infant illness and treatment				
			0 No	End questionnaire
What were the services received?	sneha-growth		1 Growth monitoring	
	sneha-food		2 Food supplement	
	sneha-medscreening		3 Medical screening	
	sneha-imm		4 Immunization	

Module 4: Anthropometry (to be filled every month)				
Show clusterid, hhid, respid, childid1				
Question	Field	Type	Constraint/Options	Skips
Date of anthropometry	canthrodate	Date		
Weights to be taken twice		Text	Read only	
Enter the exact weight in kg (e.g. 12.55)				
Enter first weighing	cweight1	Number		
Enter second weighing	cweight2	Number		
Measure height twice		Text	Read only	
Enter the exact height in cm (e.g. 121.45)				
Enter first measurement	cheight1	Number		
Enter second measurement	cheight2	Number		
MUAC	cmuac	Number		
Head circumference *(not yet decided)*	cheadcir	Number		
Abdominal circumference? *(not yet decided)*	cabdocir	Number		

Module 5: Anthropometry module (to be filled once for parents and siblings)				
Show clusterid, hhid, respid				
Question	**Field**	**Type**	**Constraint/Options**	**Skips**
Date of anthropometry	anthrodate	Date		
Who is being measured?	anthrowho	Select 1	1 Mother	
			2 Father	
			3 Sibling	Go to sibid
Enter father ID number	fatherid	Number		Go to weight
Enter sibling ID number	sibid	Number		
Sibling sex	sibsex	Number	1 Male	
			2 Female	
Sibling date of birth	sibdob	Date		
			99 Don't know	
Sibling age *If dob not known*	sibage	Number		
Enter the exact weight in kg (e.g. 12.55)				
Enter weighing	weight	Number		
Enter the exact height in cm (e.g. 121.45)				
Enter height	height	Number		
MUAC	muac	Number		
Head circumference?	headcir	Number		
Abdominal circumference?	abdocir	Number		
Enter waist measurement	waist	Number		
Enter hip measurement	hip	Number		

Module 6: Edinburgh Postnatal Depression Scale (EPDS) T*o be filled after 4 weeks of birth*				
Show clusterid, hhid, respid				
Question	**Field**	**Type**	**Constraint/Options**	**Skips**
As you are pregnant or have recently had a baby, we would like to know how you are feeling. Please check the answer that comes closest to how you have felt IN THE PAST 7 DAYS, not just how you feel today.				
In the past 7 days:				
I have been able to laugh and see the funny side of things	epds1	Select 1	0 As much as I always could	
			1 Not quite so much now	
			2 Definitely not so much now	
			3 Not at all	
I have looked forward with enjoyment to things	epds2	Select 1	0 As much as I ever did	
			1 Rather less than I used to	
			2 Definitely less than I used to	
			3 Hardly at all	

Module 6: Edinburgh Postnatal Depression Scale (EPDS) T*o be filled after 4 weeks of birth*				
Show clusterid, hhid, respid				
Question	**Field**	**Type**	**Constraint/Options**	**Skips**
I have blamed myself unnecessarily when things went wrong	epds3	Select 1	3 Yes, most of the time	
			2 Yes, some of the time	
			1 Not very often	
			0 No, never	
I have been anxious or worried for no good reason	epds4	Select 1	0 No, not at all	
			1 Hardly ever	
			2 Yes, sometimes	
			3 Yes, very often	
I have felt scared or panicky for no very good reason	epds5	Select 1	3 Yes, quite a lot	
			2 Yes, sometimes	
			1 No, not much	
			0 No, not at all	
Things have been getting on top of me	epds6	Select 1	3 Yes, most of the time I haven't been able to cope at all	
			2 Yes sometimes I haven't been coping as well as usual	
			1 No, most of the time I have coped quite well	
			0 No, I have been coping as well as ever	
I have been so unhappy that I have had difficulty sleeping	epds7	Select 1	3 Yes, most of the time	

Module 6: Edinburgh Postnatal Depression Scale (EPDS) T*o be filled after 4 weeks of birth*				
Show clusterid, hhid, respid				
Question	**Field**	**Type**	**Constraint/Options**	**Skips**
			2 Yes, sometimes	
			1 Not very often	
			0 No, not at all	
I have felt sad or miserable	epds8	Select 1	3 Yes, most of the time	
			2 Yes, quite often	
			1 Not very often	
			0 No, not at all	
I have been so unhappy that I have been crying	epds9	Select 1	3 Yes, most of the time	
			2 Yes, quite often	
			1 Only occasionally	
			0 No, never	
The thought of harming myself has occurred to me	epds10	Select 1	3 Yes, quite often	
			2 Sometimes	
			1 Hardly ever	
			0 Never	

Module 7: Domestic violence (To be filled once from the mother only)				
Show clusterid, hhid, respid				
Question	**Field**	**Type**	**Constraint/Options**	**Skips**

Module 7: Domestic violence (To be filled once from the mother only)				
Show clusterid, hhid, respid				
Question	**Field**	**Type**	**Constraint/Options**	**Skips**
I am going to ask you about some situations that are true for many women. Thinking about your husband, would you say it is generally true that he:				
Tries to keep you from seeing your friends	dvfriend	Select1	1 Yes	
			0 No	
Tries to restrict contact with your marital family	dvfamily	Select1	1 Yes	
			0 No	
Insist on knowing where you are at all the times		Select1	1 Yes	
			0 No	
Ignores you and treats you indifferently	dvindifferent	Select1	1 Yes	
			0 No	
Gets angry if you speak with another man	dvangry	Select1	1 Yes	
			0 No	
Is often suspicious that you are unfaithful	dvsuspicion	Select1	1 Yes	
			0 No	
Expects you to ask for his permission before seeking healthcare for yourself	dvhealth	Select1	1 Yes	
			0 No	

Module 7: Domestic violence (To be filled once from the mother only)				
Show clusterid, hhid, respid				
Question	**Field**	**Type**	**Constraint/Options**	**Skips**
Next questions are about things that happens to many women and that your husband may have done to you. I want you to tell me if your husband has done following things to you in last 12 months:				
Did things to scare or intimidate you on purpose	dvintimidate	Select1	1 Yes	
			0 No	
Said or did something to humiliate you in front of others	dvhumiliate	Select1	1 Yes	
			0 No	
Threatened to hurt or harm you or someone you care about	dvharm	Select1	1 Yes	
			0 No	
Insulted you and makes you feel bad about yourself	dvinsult	Select1	1 Yes	
			0 No	
In last 12 months, has he ever:				
Slapped you or thrown something at you that could hurt you	dvslap	Select1	0 Never	
			1 Once	
			2 Few	
			3 Many	
Pushed you or shoved you	dvpush	Select1	0 Never	
			1 Once	
			2 Few	

Module 7: Domestic violence (To be filled once from the mother only)				
Show clusterid, hhid, respid				
Question	**Field**	**Type**	**Constraint/Options**	**Skips**
			3 Many	
Hit you with his fist or something else that could hurt you?	dvhit	Select1	0 Never	
			1 Once	
			2 Few	
			3 Many	
Kicked you, dragged you or beaten you up?	dvkick	Select1	0 Never	
			1 Once	
			2 Few	
			3 Many	
Choked or burnt you on purpose?	dvchoke	Select1	0 Never	
			1 Once	
			2 Few	
			3 Many	
Threatened you with, or actually used a gun, knife or other weapon against you?	dvweapon	Select1	0 Never	
			1 Once	
			2 Few	
			3 Many	
In last 12 months, has he ever:				
Physically forced you to have sexual intercourse with him even when you did not want to?	dvsexforce	Select1	0 Never	
			1 Once	
			2 Few	
			3 Many	

Module 7: Domestic violence (To be filled once from the mother only)				
Show clusterid, hhid, respid				
Question	**Field**	**Type**	**Constraint/Options**	**Skips**
Did you ever have sexual intercourse you did not want because you were afraid of what he might do?	dvsexafraid	Select1	0 Never	
			1 Once	
			2 Few	
			3 Many	
Did he ever force you to do something sexual that you found degrading or humiliating?	dvsexhumiliate	Select1	0 Never	
			1 Once	
			2 Few	
			3 Many	
Alcohol abuse				
Does your husband drink alchohol?	alcodrink	Select1	0 Never	End questionnaire
			1 Sometimes	
			2 Often	
Has his alcohol use affected [Name's] care taking?	alcocare	Select 1	1 Yes	
			0 No	
Has he ever manhandled [Name] under the influence of alcohol?	alcomanhandle	Select 1	1 Yes	
			0 No	

Appendix 3.4 TEM exercise results

TEM calculator

	Measurers	6	Adults	10		
Adult number	**1**	**2**	**3**	**4**	**5**	**6**
1	169.5	169.4	169.9	169.6	169.2	170.0
2	155.3	155.4	155.6	155.3	155.5	155.3
3	157.8	157.8	157.9	157.8	158.0	158.1
4	148.7	148.6	148.8	148.8	148.7	148.9
5	174.3	174.3	174.3	174.4	174.6	174.5
6	157.8	156.7	156.9	157.8	157.5	158.0
7	178.4	178.4	178.2	178.4	177.9	178.2
8	158.4	158.4	158.2	158.9	158.6	158.4
9	149.1	149.4	149.1	149.4	149.0	149.1
10	166.8	166.7	166.9	166.9	167.0	167.0

TEM	**0.2305**
%TEM	**0.143**

Appendix 4

Appendix 4.1 Variable coding

```
 *asset score
tab female sesquintile if bl==1, row chi //p=0.045
logistic female sescore if bl==1 // OR=0.85 (0.75,0.97) p=0.018
logistic female ib5.sesquintile if bl==1 // compared to highest, lowest OR=1.67 (1.11, 2.52) p=0.014
contrast p.sesquintile //chi2 test for trend=6.84, p=0.0089

tab female ses2 if bl==1, row chi //p=0.189
logistic female sescore2 if bl==1 // OR=0.86 (0.76,0.98) p=0.026
logistic female ib5.ses2 if bl==1 // compared to highest, lowest OR=1.52 (1.01, 2.27) p=0.043
contrast p.ses2 //chi2 test for trend=4.95, p=0.0261


        *without wash services
factor hown pucca_house mattress pressurecooker stove chair bed table fan clock mixer fridge tv floor
if bl==1, factors(1) pcf
predict sescore2
xtile sesquintile2 = sescore2, nq(5)
rename sesquintile2 ses2
label var ses2 "SES2"
label define SESQUINTILE 1 "Lowest" 2 "Secondlowest" 3 "Middle" 4 "Secondhighest" 5 "Highest",
replace
label values ses2 SESQUINTILE

tab female ses2 if bl==1, row chi //p=0.189
logistic female sescore2 if bl==1 // OR=0.86 (0.76,0.98) p=0.026
logistic female ib5.ses2 if bl==1 // compared to highest, lowest OR=1.52 (1.01, 2.27) p=0.043
contrast p.ses2 //chi2 test for trend=4.95, p=0.0261

*education
        *in number of years
tab1 medu fedu if bl==1
sum medu fedu if bl==1, detail          //mean, median: medu n=971, 5.9 yrs, 7 yrs; fedu n=970, 6.7 yrs, 7
years
graph box medu fedu if bl==1, ytitle(Years of education) over(childsex)

twoway (hist medu if bl==1, frac lcolor(gs12) fcolor(gs12)) (hist fedu if bl==1, frac fcolor(none)
lcolor(black)), legend(off) xtitle("Mother's education (Black: Father's education)")
graph save Graph "N:\Documents\IGH\Analysis\Cohort\Profile\medu fedu.gph"

ttest medu if bl==1, by(female) //diff= 0.58 yrs, p=0.0300
ttest fedu if bl==1, by(female) //diff= 0.26 yrs, p=0.3069
regress medu fedu if  bl==1 // co-eff 0.46 (0.41-0.52, p<0.001)
        *using a cut-off
recode medu (0/5 = 0 "Below6th") (6/17 = 1 "6thStd"), gen(med)
recode fedu (0/5 = 0 "Below6th") (6/17 = 1 "6thStd"), gen(fed)

tab1 med fed if bl==1
tab med fed if bl==1, row chi
logistic fed med if bl==1
logistic female med if bl==1
logistic female fed if bl==1 //OR=0.92 (0.71, 1.19; p=0.554)
logistic female c.fedu if bl==1
logistic female med fed if bl==1 //OR= 0.70 (0.54, 0.93; p=0.013)
logistic female c.fedu c.medu if bl==1
logistic female c.medu if bl==1

        *check if linear relationship holds with more categories
recode medu (0=0) (1/5 = 1) (6/9 = 2) (10/17=3), gen(med4)
recode fedu (0=0) (1/5 = 1) (6/9 = 2) (10/17=3), gen(fed4)
```

```
label define edu4 0 "None" 1 "1st -5th" 2 "6th-9th" 3 "10th plus"
label values med4 edu4
label values fed4 edu4
logistic female i.med4 if bl==1
contrast p.med4
logistic female i.fed4 if bl==1
contrast p.fed4
logistic female i.fed4 i.med4 if bl==1 // only for 10th vs none.

*and occupation
tab1 focc mocc if bl==1, sort //nothing interesting

* WASH
*water var
recode dwater (1 2 = 1 "Piped" ) (3/14 = 0 "Not piped"), gen(water)
tab1 dwater water stoiletshare stoilet if bl==1
tab water stoiletshare if bl==1, row chi
logistic water stoiletshare if bl==1
recode water (0=1 "Not piped") (1=0 "Piped"), gen(notpiped)
logistic notpiped stoiletshare if bl==1
                *linked to infant sex?
logistic female water if bl==1
logistic female stoiletshare if bl==1
logistic female water stoiletshare if bl==1


*hh composition
        *other children in the house, including siblings <18yrs and other children
egen hhkids=rowtotal(ownkidsunder18 otherchildren)

        *other adults in the house who are not the infant's parents. Includes siblings>18yrs and other
male/female adults
egen hhadults = rowtotal(ownkidsabove18 othermales otherfemales)

        *also check constituent vars
tab1 ownkidsunder18 otherchildren ownkidsabove18 othermales otherfemales if bl==1
sum ownkidsunder18 otherchildren ownkidsabove18 othermales otherfemales if bl==1, detail
graph box ownkidsunder18 otherchildren ownkidsabove18 othermales otherfemales if bl==1
graph save Graph "N:\Documents\IGH\Analysis\Cohort\Profile\medu fedu.gph", replace
swilk ownkidsunder18 otherchildren ownkidsabove18 othermales otherfemales if bl==1
        //all non-normally distributed p<0.001

        *tab hh vars
sum hhkids hhadults if bl==1, detail
hist hhkids if bl==1 //skewed. don't save
hist hhadults if bl==1 //skewed. don't save

recode hhkids (0/3 = 0 "Less than 4") (4/14 = 1 "4+"), gen(kids)
recode hhadults (0/1 = 0 "Less than 2") (2/27 = 1 "2+"), gen(adults)

logistic kids adults if bl==1
logistic female kids if bl==1
logistic female adults if bl==1
logistic female adults kids if bl==1
```

Appendix 5

Appendix 5.1 Stata .do file for cohort profile

```
*univariable chi-squared and logistic for binary vars, and trend for ses as exposure
tab2 med fed water stoiletshare kids adults if bl==1, row chi

foreach var of varlist mage25 fage30 med fed ses2 water stoiletshare adults kids fsmoke msmoke
lmup2{
logistic female i.`var' if bl==1
}
logistic mage25 fage30 if bl==1

foreach var of varlist med fed ses2 water stoiletshare adults kids fsmoke msmoke lmup2{
logistic mage25 i.`var' if bl==1
logistic fage30 i.`var' if bl==1
}
logistic med fed if bl==1
foreach var of varlist ses2 water stoiletshare adults kids fsmoke msmoke lmup2{
logistic med i.`var' if bl==1
logistic fed i.`var' if bl==1
}
foreach var of varlist water stoiletshare kids adults fsmoke msmoke lmup2 {
logistic `var' i.ses2 if bl==1
}
logistic water stoiletshare if bl==1

foreach var of varlist adults kids {
logistic `var' water
logistic `var' stoiletshare
}
                                                    // p for trend <0.001 for all except kids (p=0.7561)

*Univariables ORs matrix
foreach var of varlist mage25 fage30 med fed water stoiletshare adults kids fsmoke msmoke lmup2
ses2 {
logistic female i.`var' if bl==1
}
foreach var of varlist fage30 med fed water stoiletshare adults kids fsmoke msmoke lmup2 ses2 {
logistic mage25 i.`var' if bl==1
}
foreach var of varlist med fed water stoiletshare adults kids fsmoke msmoke lmup2 ses2 {
logistic fage30 i.`var' if bl==1
}
foreach var of varlist fed water stoiletshare adults kids fsmoke msmoke lmup2 ses2 {
logistic med i.`var' if bl==1
}
foreach var of varlist water stoiletshare adults kids fsmoke msmoke lmup2 ses2 {
logistic fed i.`var' if bl==1
}
foreach var of varlist stoiletshare adults kids fsmoke msmoke lmup2 ses2 {
logistic water i.`var' if bl==1
}
foreach var of varlist adults kids fsmoke msmoke lmup2 ses2 {
logistic stoiletshare i.`var' if bl==1
}
foreach var of varlist kids fsmoke msmoke lmup2 ses2 {
logistic adults i.`var' if bl==1
}
foreach var of varlist fsmoke msmoke lmup2 ses2 {
logistic kids i.`var' if bl==1
}
foreach var of varlist msmoke lmup2 ses2 {
```

```
logistic fsmoke i.`var' if bl==1
}
foreach var of varlist lmup2 ses2 {
logistic msmoke i.`var' if bl==1
}
logistic lmup2 i.ses2 if bl==1

        *are the relationships between SEP variables the same when stratified by sex?
mhodds med fed if bl==1, by(female)
mhodds med water if bl==1, by(female)
mhodds med stoiletshare if bl==1, by(female)
mhodds med kids if bl==1, by(female)
mhodds med adults if bl==1, by(female)
mhodds fed water if bl==1, by(female)
mhodds fed stoiletshare if bl==1, by(female)
mhodds fed kids if bl==1, by(female)
mhodds fed adults if bl==1, by(female)
mhodds water stoiletshare if bl==1, by(female)
mhodds water kids if bl==1, by(female)
mhodds water adults if bl==1, by(female)
mhodds stoiletshare kids if bl==1, by(female)
mhodds stoiletshare adults if bl==1, by(female)
mhodds kids adults if bl==1, by(female)
foreach var of varlist med fed water stoiletshare kids adults {
mhodds `var' ses2 if bl==1, by(female)
}
                                        //yes. all test of homogeneity p>0.05

        *are the relationships between SEP variables the same when stratified by SES quintile?
mhodds med fed if bl==1, by(ses2)
mhodds med water if bl==1, by(ses2)
mhodds med stoiletshare if bl==1, by(ses2)
mhodds med kids if bl==1, by(ses2)
mhodds med adults if bl==1, by(ses2)
mhodds fed water if bl==1, by(ses2)
mhodds fed stoiletshare if bl==1, by(ses2)
mhodds fed kids if bl==1, by(ses2)
mhodds fed adults if bl==1, by(ses2)
mhodds water stoiletshare if bl==1, by(ses2)
mhodds water kids if bl==1, by(ses2)
mhodds water adults if bl==1, by(ses2)
mhodds stoiletshare kids if bl==1, by(ses2)
mhodds stoiletshare adults if bl==1, by(ses2)
mhodds kids adults if bl==1, by(ses2)
                                                //yes.

        *related to sex?
logistic female med fed water stoiletshare kids adults ib5.ses2 if bl==1

*related to parents' ages?
tab med fed water stoiletshare kids adults if bl==1, row chi

foreach var of varlist med fed water stoiletshare kids adults {
tab fage30 `var' if bl==1, row chi
tab mage25 `var' if bl==1, row chi
}
tabodds fage30 ses2 if bl==1, or
tabodds mage25 ses2 if bl==1, or

mhodds med fed if bl==1, by(mage25)
mhodds med fed if bl==1, by(fage30)

        *are the relationships between SEP variables the same when stratified by parental ages?


foreach var of varlist mage25 fage30 {
mhodds med fed if bl==1, by(`var')
```

```
mhodds med water if bl==1, by(`var')
mhodds med stoiletshare if bl==1, by(`var')
mhodds med kids if bl==1, by(`var')
mhodds med adults if bl==1, by(`var')
mhodds fed water if bl==1, by(`var')
mhodds fed stoiletshare if bl==1, by(`var')
mhodds fed kids if bl==1, by(`var')
mhodds fed adults if bl==1, by(`var')
mhodds water stoiletshare if bl==1, by(`var')
mhodds water kids if bl==1, by(`var')
mhodds water adults if bl==1, by(`var')
mhodds stoiletshare kids if bl==1, by(`var')
mhodds stoiletshare adults if bl==1, by(`var')
mhodds kids adults if bl==1, by(`var')
foreach var2 of varlist med fed water stoiletshare kids adults {
mhodds `var2' ses2 if bl==1, by(`var')
}
}
        //No.
        //Mage25: for med&kids p=0.0002 ; water& adults p=0.0328; kids& adults p<0.001
                //kids & ses p = 0.0002; adults & ses p =0.0099
        //Fage30: med&kids p=0.0098 ;       water&kids p=0.0035 ; kids&adults <0.001
                //kids& ses p=0.0003 ; adults&ses p=0.0163

***** (2) Health

*Smoking
tab1 fsmoke msmoke if bl==1
tab fsmoke msmoke if bl==1, row chi
logistic fsmoke msmoke if bl==1
        *related to age, infant sex, or any SEP?
foreach var of varlist female mage25 fage30 med fed water stoiletshare kids adults {
tab fsmoke `var' if bl==1, row chi
tab msmoke `var' if bl==1, row chi
}
tabodds fsmoke ses2 if bl==1, or
tabodds msmoke ses2 if bl==1, or

foreach var of varlist female mage25 fage30 med fed ses2 water stoiletshare kids adults {
logistic fsmoke i.`var' if bl==1
logistic msmoke i.`var' if bl==1
}
                *link with SEP and age variables differs by sex?
foreach var of varlist mage25 fage30 med fed water stoiletshare kids adults {
mhodds fsmoke `var' if bl==1, by(female)
mhodds msmoke `var' if bl==1, by(female)
}
//No. all p>0.05

                *link with SEP and age variables differs by parental ages?
foreach var of varlist med fed water stoiletshare kids adults {
foreach strat of varlist mage25 fage30{
mhodds fsmoke `var' if bl==1, by(`strat')
mhodds msmoke `var' if bl==1, by(`strat')
}
}
                //Yes.
                //MAGE25: fsmoke-med p=0.0343; msmoke-adults p=0.0185

logistic fsmoke msmoke female med fed water stoiletshare kids adults mage25 fage30 c.sescore2 if
bl==1
logistic msmoke fsmoke female med fed water stoiletshare kids adults mage25 fage30 c.sescore2 if
bl==1

* LMUP
        *continuous
sum unplantotal if bl==1, detail
```

```
graph box unplantotal if bl==1, over(childsex) //did not save
swilk unplantotal if bl==1 //not normally distributed p<0.001
graph bar (count) if bl==1, over(unplantotal) blabel(bar) ytitle(N) title(Total LMUP score)
graph save Graph "N:\Documents\IGH\Analysis\Cohort\Profile\lmup hist.gph"
tab unplantotal if bl==1
kwallis unplantotal if bl==1, by(female) /* p=0.5653 no evidence of sex diff*/

                *assocaition with all sep
foreach var of varlist female mage25 fage30 med fed water stoiletshare kids adults fsmoke msmoke {
kwallis unplantotal if bl==1, by(`var')
}
//mage25, fage30, kids4, p=0.0001; fed p=0.0141; fsmoke p=0.0006,
        *binary
recode lmup (1 2 = 0 "NotPlanned") (3 = 1 "Planned"), gen(lmup2)
tab lmup2 if bl==1
                *related to age, infant sex, any SEP, or smoking?
foreach var of varlist female mage25 fage30 med fed ses2 water stoiletshare kids adults fsmoke
msmoke {
tab lmup2 `var' if bl==1, row chi
}
foreach var of varlist female mage25 fage30 med fed water stoiletshare kids adults fsmoke msmoke {
logistic lmup2 `var' if bl==1
}
logistic lmup2 i.ses2 if bl==1
//lower odds: fsmoke, kids, fage30, mage25

tabodds lmup2 ses2 if bl==1, or //not related to any quintile. NO evidence of trend p=0.4026

                *link with SEP, age and smoking variables differs by sex?
foreach var of varlist mage25 fage30 ses2 med fed water stoiletshare kids adults fsmoke msmoke {
mhodds lmup2 `var' if bl==1, by(female)
}
                //none whatsoever. all p>0.05

                *logistic regression with all predictors
logistic lmup2 female med fed water stoiletshare kids adults mage25 fage30 fsmoke msmoke
c.sescore2 if bl==1
logistic lmup2 female med fed water stoiletshare kids adults mage25 fage30 fsmoke msmoke ib5.ses2
if bl==1
                *with sig predictors
logistic lmup2 kids mage25 fage30 fsmoke  if bl==1
                        //same either way: mage attenuated; fage, fsmoke, kids - still remained


*Variables related to being female
        *background
logistic female mage25 fage30 if bl==1          //none

        *SEP only
logistic female med fed water stoiletshare kids adults c.sescore2 if bl==1
                //med 0.68, 0.51, 0.92 || kids 0.75 0.56-0.99
logistic female med fed water stoiletshare kids adults ib5.ses2 if bl==1

        *background + SEP
logistic female med fed water stoiletshare kids adults mage25 fage30 c.sescore2 if bl==1
                        //med only OR 0.65, 0.48 - 0.88
logistic female med fed water stoiletshare kids adults mage25 fage30 ib5.ses2 if bl==1


        *background + SEP + health
logistic female med fed water stoiletshare kids adults mage25 fage30 fsmoke msmoke lmup2
c.sescore2 if bl==1
                //med only OR0.66 0.49 - 0.89
logistic female med fed water stoiletshare kids adults mage25 fage30 fsmoke msmoke lmup2 ib5.ses2
if bl==1
                //med only OR 0.66 (0.49, 0.90)
```

```
	*health only
logistic female fsmoke msmoke lmup2 if bl==1 //none

***** (3) Parental anthro

*sum
sum f_height m_height f_weight m_weight f_bmi m_bmi if bl==1, detail
sum f_height m_height f_weight m_weight f_bmi m_bmi if bl==1
count if f_height!=. & f_weight!=. & bl==1
count if m_height!=. & m_weight!=. & bl==1

	*re-clean
	{
* (!!!!) scatter of 1st and 2nd readings to identify those that are off... re-clean..
gen mhtdiff=m_height2-m_height1
gen fhtdiff=f_height2-f_height1
gen mwtdiff=m_weight2-m_weight1
gen fwtdiff=f_weight2-f_weight1

sum mhtdiff mwtdiff fhtdiff fwtdiff if bl==1 , detail
list id if mhtdiff <-1 & mhtdiff!=. & bl==1 //25, 1003, 800
list id if mwtdiff <=-1 & mwtdiff!=. & bl==1 //806, 306
list id if fhtdiff >1 & fhtdiff!=. & bl==1 // 636
list id if fwtdiff >1 & fwtdiff!=. & bl==1 //109


*id=25 mht2 should be 147.2 like ht1, currently 14.2
replace m_height2=147.2 if id==25
*id=1003 mht1=173, mht2=143. change 1 to 143
replace m_height1=143 if id==1003
*id=800. 2cm diff, it's ok, don't change
*id=806. 1 kg diff, it's ok.
*id=306. wt2=20, wt1=50. change wt2 to 50
replace m_weight2=50 if id==306
*id=636 ht1=160.9, ht2=170. change ht2 to ht1
replace f_height2=160.9 if id==636
*id=109 wt1=52.7, wt2=57.2. change wt1 to 57.2
replace f_weight1=57.2 if id==109

	//extreme maternal low BMI
list id m_height1 m_height2 m_weight1 m_weight2 m_bmi if m_bmi<15 & bl==1
		//change only 1 which is a BMI of 9
		//id=727, hts are 199.2 and 199.3 and wt is 38.2. likely digit error
			//change ht to 149.2 and 149.3. would then make sense with WC
replace m_height1=149.2 if id==727
replace m_height2=149.3 if id==727

	*very short mothers
list id if m_height<60 // 45, 31, 468, 22
	//hts are 55cm, 48.2cm, 56.7cm, and 58.7cm. likely missing the 1 in 155 etc
replace m_height1=155 if id==45
replace m_height2=155 if id==45
replace m_height1=148.2 if id==31
replace m_height2=148.2 if id==31
replace m_height1=156.7 if id==468
replace m_height2=156.8 if id==468
replace m_height1=158.7 if id==22
replace m_height2=158.8 if id==22

	*very obese fathers.. what's off?
list id f_height1 f_height2 f_sitting_ht1 f_sitting_ht2 f_weight1 f_weight2 f_WC f_bmi if f_bmi>40 &
bl==1 &f_bmi!=.
	//id= 441, 439, 616. first two have swapped ht and sitting ht, third is large.
replace f_height1=165.1 if id==441
replace f_height2=165.2 if id==441
replace f_sitting_ht1=136.2 if id==441
replace f_sitting_ht2=136.3 if id==441
```

```
replace f_height1=164.4 if id==439
replace f_height2=164.5 if id==439
replace f_sitting_ht1=129.3 if id==439
replace f_sitting_ht2=129.4 if id==439


	*very obese mothers
list id m_height1 m_height2 m_weight1 m_weight2 m_bmi if m_bmi>40 & bl==1 & m_bmi!=.
	//id 521, 733, 1011, 227 but all seem legit and large..

	*very short mothers <140cm
list id m_height1 m_height2 m_sitting_ht1 m_sitting_ht2 m_weight1 m_weight2 m_bmi if m_height<140
& bl==1 & m_height!=.
		//digit swap error: 988 written as 124cm instead of 142.
replace m_height1=142 if id==988
replace m_height2=142 if id==988

		//swapped with sitting ht: 740, 957, 731
replace m_height1=145 if id==740
replace m_height2=145 if id==740
replace m_sitting_ht1=121.6 if id==740
replace m_sitting_ht1=121.6 if id==740

replace m_height1=160 if id==957
replace m_height2=160 if id==957
replace m_sitting_ht1=127 if id==957
replace m_sitting_ht1=127 if id==957

replace m_height1=150.7 if id==731
replace m_height2=150.8 if id==731
replace m_sitting_ht1=119.4 if id==731
replace m_sitting_ht1=119.5 if id==731

		*very short fathers
list id f_height1 f_height2 f_sitting_ht1 f_sitting_ht2 f_weight1 f_weight2 f_WC f_bmi if f_height<140 &
bl==1 &f_height!=.
	//487. swapped with sitting height
replace f_height1=168.6 if id==487
replace f_height2=168.7 if id==487
replace f_sitting_ht1=131.6 if id==487
replace f_sitting_ht2=131.7 if id==487

		*very thin fathers
list id f_height1 f_height2 f_sitting_ht1 f_sitting_ht2 f_weight1 f_weight2 f_WC f_bmi if f_bmi<16 &
bl==1 &f_bmi!=.
		//most >15 seem plausible.
		//2 are below 15: 14.4 seems plausible. 12.9 doesn't, but no way to fix.


*keep old mean vars
foreach v of varlist f_height m_height f_weight m_weight f_bmi m_bmi f_bmicat m_bmicat{
rename `v' `v'_old
}
				*drop extra vars
drop mhtdiff fhtdiff mwtdiff fwtdiff


	*re-gen vars for mean measurements
egen f_weight=rowmean(f_weight1 f_weight2)
egen f_height=rowmean(f_height1 f_height2)
egen m_weight=rowmean(m_weight1 m_weight2)
egen m_height=rowmean(m_height1 m_height2)

	*gen vars for BMI vars
gen f_bmi= f_weight/(f_height/100)^2
gen m_bmi= m_weight/(m_height/100)^2
```

```
		*egen vars for BMI cat (Asian)
		*Fathers
gen f_bmicat = 1 if f_bmi<18.5 & f_bmi!=.
replace f_bmicat=2 if f_bmi>=18.5 & f_bmi<23.5
replace f_bmicat=3 if f_bmi>=23.5 & f_bmi<27.5
replace f_bmicat=4 if f_bmi>=27.5 & f_bmi!=.
		*label variable
label var f_bmicat "Father's BMI Category (Asian Cut-off)"
label values f_bmicat bmicat

		*Mothers
gen m_bmicat = 1 if m_bmi<18.5 & m_bmi!=.
replace m_bmicat=2 if m_bmi>=18.5 & m_bmi<23.5
replace m_bmicat=3 if m_bmi>=23.5 & m_bmi<27.5
replace m_bmicat=4 if m_bmi>=27.5 & m_bmi!=.
		*label variable
label var m_bmicat "Mother's BMI Category (Asian Cut-off)"
label values m_bmicat bmicat

order f_bmi f_bmicat, after (f_height)
order m_bmi m_bmicat, after (m_height)

label var f_height "Father's height (cm)"
label variable m_height "Mother's height (cm)"
label variable f_weight "Father's weight (kg)"
label variable m_weight "Mother's weight (kg)"
label variable f_bmi "Father's BMI"
label variable m_bmi "Mother's BMI"
label variable f_bmicat "Father's BMI Category (Asian Cut-off)"
label variable m_bmicat "Mother's BMI Category (Asian Cut-off)"

sum f_height m_height f_weight m_weight f_bmi m_bmi if bl==1, detail
sum f_height m_height f_weight m_weight f_bmi m_bmi if bl==1
}

*normally distributed
swilk f_height m_height f_weight m_weight f_bmi m_bmi if bl==1 // suggests none are
sktest f_height m_height f_weight m_weight f_bmi m_bmi if bl==1 // suggests none are

/* swilk
    Variable |    Obs       W          V        z       Prob>z
-------------+-------------------------------------------------
    f_height |    537    0.98916      3.890     3.276    0.00053
    m_height |    690    0.99524      2.142     1.857    0.03165
    f_weight |    537    0.97781      7.959     5.002    0.00000
    m_weight |    690    0.93913     27.399     8.072    0.00000
      f_bmi |    537    0.98757      4.460     3.605    0.00016
      m_bmi |    690    0.94003     26.991     8.035    0.00000
*/
* format vars
format %3.1f f_height m_height f_weight m_weight f_bmi m_bmi

*plot departures from normal distribution
		*quantile normal plots to check for normality in the tails of distribution
		* & histograms for frequency distribution plots
cd "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile"

			// (!!!) set more on so you can inspect each one as they are generated
				*Quantile Normal with grid so you can inspect 5th and 95th percentiles
set more on
foreach v of varlist f_height m_height f_weight m_weight f_bmi m_bmi {
qnorm `v' if bl==1, grid
graph save "`v'_qnorm.gph", replace
more
}
				*Histogram
```

```
foreach v of varlist f_height m_height f_weight m_weight f_bmi m_bmi {
hist `v' if bl==1, freq normal normopts(lcolor(red))
graph save "`v'_hist.gph", replace
more
}
set more off

	*Combined q normal plots
format %3.0f f_height m_height f_weight m_weight f_bmi m_bmi
foreach v of varlist f_height m_height f_weight m_weight f_bmi m_bmi {
qnorm `v' if bl==1
graph save "`v'_qnorm.gph", replace
more
}
graph combine "m_height_qnorm.gph" "m_weight_qnorm.gph" "m_bmi_qnorm.gph"
"f_height_qnorm.gph" "f_weight_qnorm.gph" "f_bmi_qnorm.gph"
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\combine qnorm p ht
wt bmi.gph"
format %3.1f f_height m_height f_weight m_weight f_bmi m_bmi

	*Combined hist plots
graph combine "m_height_hist.gph" "m_weight_hist.gph" "m_bmi_hist.gph" "f_height_hist.gph"
"f_weight_hist.gph" "f_bmi_hist.gph"

	*superimposed maternal and paternal data in histograms
twoway (hist m_height if bl==1, freq lcolor(gs12) fcolor(gs12)) (hist f_height if bl==1, freq fcolor(none)
lcolor(black)), legend(off) xtitle("Mother's height (Black: Father's height)") title(Parental heights)
graph save Graph "N:\Documents\IGH\Analysis\Cohort\Profile\f m ht hist.gph"      , replace

twoway (hist m_weight if bl==1, freq lcolor(gs12) fcolor(gs12)) (hist f_weight if bl==1, freq fcolor(none)
lcolor(black)), legend(off) xtitle("Mother's weight (Black: Father's weight)") title(Parental weights)
graph save Graph "N:\Documents\IGH\Analysis\Cohort\Profile\f m wt hist.gph", replace

twoway (hist m_bmi if bl==1, freq lcolor(gs12) fcolor(gs12)) (hist f_bmi if bl==1, freq fcolor(none)
lcolor(black)), legend(off) xtitle("Mother's BMI (Black: Father's BMI)") title(Parental BMI)
graph save Graph "N:\Documents\IGH\Analysis\Cohort\Profile\f m bmi hist.gph", replace

graph combine "f m ht hist.gph" "f m wt hist.gph" "f m bmi hist.gph", ycommon cols(1)
	//change siz of graph to y=7, x=3.5
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\combine hist p ht wt
bmi.gph", replace

	*Generate binary variables for overweight
recode m_bmicat (1/2 = 0 "No") (3/4 = 1 "Yes"), gen(mow)
label var mow "Mother overweight"
recode f_bmicat (1/2 = 0 "No") (3/4 = 1 "Yes"), gen(fow)
label var fow "Father overweight"

tab1 m_bmicat f_bmicat mow fow if bl==1

	* combined graphs of bmi category (%)
graph bar if bl==1, over(m_bmicat) blabel(bar, format(%2.0f)) ytitle(%) title(Maternal BMI Category)
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\mbmicat.gph",
replace

graph bar if bl==1, over(f_bmicat) blabel(bar, format(%2.0f)) ytitle(%) title(Paternal BMI Category)
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\fbmicat.gph",
replace

graph combine mbmicat.gph fbmicat.gph, ycommon
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\combine bar
mbmicat fbmicat.gph"

	*scatter plots of parental anthro
scatter m_height f_height if bl==1, ytitle(Mother's height (cm)) legend(off) || lfit m_height f_height if
bl==1
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\f m ht scatter.gph"
```

```
scatter m_weight f_weight if bl==1, ytitle(Mother's weight (kg)) legend(off)  || lfit m_weight f_weight if
bl==1
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\f m wt scatter.gph"
scatter m_bmi f_bmi if bl==1, ytitle(Mother's BMI) legend(off)  || lfit m_bmi f_bmi if bl==1
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\f m bmi scatter.gph"

graph combine "f m ht scatter.gph" "f m wt scatter.gph" "f m bmi scatter.gph", cols(1)
        //change graph size to y=7, x=3.5
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\combine scatter p
ht wt bmi.gph"

        * Wilcoxon signed rank test for whether median difference between paired values = 0.

signrank m_height= f_height if bl==1             // p<0.001
signrank m_weight= f_weight if bl==1 // p<0.0001
signrank m_bmi= f_bmi if bl==1       //p=0.0559

        *Kendall's tau to check independence of m and f anthro
ktau m_height f_height if bl==1 //p==0.001. data are correlated
ktau m_weight f_weight if bl==1 //p==0.001
ktau m_bmi f_bmi if bl==1 //p==0.001

        *Maternal and paternal BMI cat are associated?
tab m_bmicat f_bmicat if bl==1 //small n in fUW, mOb. try fishers exact
tab m_bmicat f_bmicat if bl==1, exact(2) //doesn't work. must collapse categories.

                *3 categories
recode m_bmicat (1 = 1 "Underweight") (2 = 2 "Normal") (3/4 = 3 "Overweight"), gen(mow3)
label var mow3 "Mother's BMI Category"
recode f_bmicat (1 = 1 "Underweight") (2 = 2 "Normal") (3/4 = 3 "Overweight"), gen(fow3)
label var fow3 "Father's BMI Category"

tab mow3 fow3 if bl==1, row chi //p=0.01

*binary O/W variable
tab mow fow if bl==1, row chi //p=0.009
logistic mow fow if bl==1 //OR 1.59 (1.12, 2.27) p=0.009

*groups of parental o/w
egen pow = group(mow fow)
label define pow 1 "Neither" 2  "Father only" 3  "Mother only" 4  "Both parents", modify
label values pow pow
label var pow "Overweight parents"

tab pow if bl==1

tab pow childsex if bl==1, row chi

*Z scores
*internal z-scores for parental heights and weights. = (ht-mean)/sd
        *use all available parental data.

                *get mean and sd values for maternal and paternal heights and weights,
                        *

sum m_height if bl==1, detail
//mean= 150.9725 sd= 5.62751
sum f_height if bl==1, detail
//mean=163.9705 sd=6.60056

sum m_weight if bl==1, detail
//mean= 52.11993 sd= 11.82088
sum f_weight if bl==1, detail
//mean= 62.66993 sd= 11.33759


                *gen z-scores for non-missing height and weight values
```

```
gen mhtz=(m_height-150.9725)/5.62751 if m_height!=.
gen fhtz=(f_height-163.9705)/6.60056 if f_height!=.
label var mhtz "Maternal height internal z-score"
label var fhtz "Paternal height internal z-score"


gen mwtz=(m_weight-52.11993)/11.82088 if m_weight!=.
gen fwtz=(f_weight-62.66993)/11.33759 if f_weight!=.
label var mwtz "Maternal weight internal z-score"
label var fwtz "Paternal weight internal z-score"

                        * gen sum of parental z-scores and half difference
gen phtz =mhtz+fhtz
gen pwtz=mwtz+fwtz

label var phtz "Sum of parental height z-scores"
label var pwtz "Sum of parental weight z-scores"

gen phtzdiff=(mhtz-fhtz)/2
gen pwtzdiff=(mwtz-fwtz)/2

label var phtzdiff "Half-diff of parental height z-scores"
label var pwtzdiff "Half-diff of parental weight z-scores"



            *box and whiskers plot: height, weight, bmi by CHILDSEX
graph box f_height m_height if bl==1, ytitle(Height (cm))by(childsex)
graph save "p height childsex.gph"
graph box f_weight m_weight if bl==1, ytitle(Weight (kg)) by(childsex)
graph save "p weight childsex.gph"
graph box f_bmi m_bmi if bl==1, ytitle(BMI) by(childsex)
graph save "p bmi childsex.gph"

graph combine "p height childsex.gph" "p weight childsex.gph" "p bmi childsex.gph", ycommon cols(1)
            //change graph size to y=7, x=3.5
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\combine box p
anthro sex.gph"

 *diff in p.anthro by sex. Wilcoxon rank sum test, and Kruksal-Wallist for ordinal data
foreach v of varlist f_height m_height f_weight m_weight f_bmi m_bmi {
ranksum `v' if bl==1, by(female)
}
//in order, p-values are: 0.3287, 0.4339, 0.1252, 0.5781, 0.1713, 0.8019
//no evidence of a difference.
kwallis m_bmicat if bl==1, by(female) //p for trend = 0.5005
kwallis f_bmicat if bl==1, by(female) //p for trend = 0.1637

tab mow female if bl==1, row chi //0.519
tab fow female if bl==1, row chi //0.077

*associations with parental age
                        *Mothers
foreach v of varlist  m_height m_weight m_bmi {
ranksum `v' if bl==1, by(mage25)
}
//p=0.0058, <0.001, <0.001
kwallis m_bmicat if bl==1, by(mage25) //p for trend = 0.0001
tab mow mage25 if bl==1, row chi //p<0.000
                        *Fathers
foreach v of varlist f_height f_weight f_bmi {
ranksum `v' if bl==1, by(fage30)
}
//p=0.4684, 0.0880, 0.2471
kwallis f_bmicat if bl==1, by(fage30) //p for trend = 0.1904
tab fow fage30 if bl==1, row chi //p=0.624
```

```
*ASSOCIATIONS BETWEEN ANTHRO AND SEP AND HEALTH VARIABLES

** CONTINUOUS ANTHRO and  SEP /HEALTH
	*Maternal
foreach anthro of varlist m_height m_weight m_bmi {
	foreach sep of varlist med fed water stoiletshare kids adults lmup2 msmoke fsmoke{
ranksum `anthro' if bl==1, by(`sep')
}
kwallis `anthro' if bl==1, by(ses2)
}
	*Paternal
foreach anthro of varlist f_height f_weight f_bmi {
	foreach sep of varlist med fed water stoiletshare kids adults lmup2 msmoke fsmoke{
ranksum `anthro' if bl==1, by(`sep')
}
kwallis `anthro' if bl==1, by(ses2)
}
**CATEGORICAL ANTHRO AND ALL SEP / HEALTH
foreach anthcat of varlist m_bmicat f_bmicat{
	foreach sep of varlist med fed ses2 water stoiletshare kids adults lmup2 msmoke fsmoke{
kwallis `anthcat' if bl==1, by(`sep')
}
}
**BINARY ANTHRO AND BINARY SEP / HEALTH
foreach ow of varlist mow fow{
	foreach sep of varlist med fed ses2 water stoiletshare kids adults lmup2 msmoke fsmoke{
tab `ow' `sep' if bl==1, row chi
}
}
tabodds mow ses2 if bl==1, or //p=0.0007
tabodds fow ses2 if bl==1, or //p=0.0195

	***POW and all sep / health
foreach sep of varlist mage25 fage30 med fed ses2 water stoiletshare kids adults lmup2 msmoke
fsmoke {
tab pow `sep' if bl==1, row chi
}
foreach sep of varlist mage25 fage30 med fed ses2 water stoiletshare kids adults lmup2 msmoke
fsmoke {
kwallis pow if bl==1, by(`sep' )
}
		//same in both: water, toilet, ses, fage, mage,
****** (4) Are Parental anthro variables related to SEP / health variables
	*Univariable
	*mhtz, fhtz, mbmi, fbmi
foreach y of varlist mhtz fhtz m_bmi f_bmi {
foreach x of varlist mage25 fage30 med fed water stoiletshare kids adults lmup2 msmoke fsmoke {
regress `y' `x' if bl==1
}
regress `y' i.ses2 if bl==1
}
	*mow
foreach sep of varlist mage25 fage30 med fed water stoiletshare kids adults lmup2 msmoke fsmoke {
logistic `sep' i.mow if bl==1
}
regress sescore2 mow if bl==1
//mage25 OR2.84 (2.07, 3.91); fage30 OR 2.07 (1.51, 2.83); water OR 1.45 (1.05, 2.01)
//toilet OR 0.58 (0.39, 0.88); sescore 0.26 (0.11, 0.41)

	*fow
foreach sep of varlist mage25 fage30 med fed water stoiletshare kids adults lmup2 msmoke fsmoke {
logistic `sep' i.fow if bl==1
}
regress sescore2 i.fow if bl==1
//water OR 1.75 (1.22, 2.51); fsmoke OR 0.67 (0.47, 0.95); sescore 0.2 (0.03, 0.37)
logistic fow water female female##water if bl==1
	*pow
```

```
foreach sep of varlist mage25 fage30 med fed water stoiletshare kids adults lmup2 msmoke fsmoke {
logistic `sep' i.pow if bl==1
}
mlogit ses2 i.pow if bl==1, base(1) rrr
regress sescore2 i.pow if bl==1
//mage25 m1f0 OR 2.75 (1.65,4.59) m1f1 OR 3.46 (2.11, 5.68)
//fage30 m1f0 OR1.78 (1.07, 2.94) m1f1 OR 2.10 (1.30, 3.40)
//toilet m1f0 OR 0.42 (0.21, 0.85) m1f1 OR 0.41 (0.21, 0.81)
//water m0f1 OR 1.91 (1.19, 3.05) m1f1 OR 2.41 (1.45, 4.00)
///Asset quintile (REF: Lowest): Middle quintile, m1f1 RRR 2.67 (1.12, 6.39)
        // Second highest, m1f1 RRR 4.08 (1.78, 9.37)
        // Highest, m1f1 RRR 3.41 (1.49, 7.78)

        *oops! forgot child sex!
logistic female mow if bl==1 //nope. CIs cross 1
logistic female fow if bl==1 //nope. CIs cross 1
logistic female i.pow if bl==1 //nope. CIs cross 1

        *background + health
**mhtz, fhtz, mbmi, fbmi
foreach y of varlist mhtz fhtz m_bmi f_bmi {
regress `y' female mage25 fage30 lmup fsmoke msmoke if bl==1
}
*mow
logistic mow female mage25 fage30 c.mhtz c.fhtz lmup fsmoke msmoke if bl==1
                //mage25 OR 2.74 (1.74, 4.34)
*fow
logistic fow female mage25 fage30 c.mhtz c.fhtz lmup fsmoke msmoke if bl==1
                //mage25 OR 1.58 (1.01, 2.47) fhtz OR 0.74 (0.62, 0.93) fsmoke OR 0.65 (0.45,
0.93)
*pow
mlogit pow female mage25 fage30 c.mhtz c.fhtz lmup fsmoke msmoke if bl==1, rrr
        //m1f0 mage25 RRR 2.53 (1.32, 4.87); fhtz RRR 1.42 (1.09, 1.85);
        //m1f1 mage25 RRR 3.85 (2.05, 7.22); mhtz RRR 1.39 (1.07, 1.80); fhtz RRR 0.70 (0.54,
0.92); fsmoke RRR (0.32, 0.91)


                *background + SEP
*mhtz, fhtz, mbmi, fbmi
foreach y of varlist mhtz fhtz m_bmi f_bmi {
regress `y' female mage25 fage30 med fed i.ses2 water stoiletshare kids adults if bl==1
}
*mow
logistic mow female mage25 fage30 c.mhtz c.fhtz med fed i.ses2 water stoiletshare kids adults if bl==1
                //higher odds: female, mage25, ses (mid, sh, highest),
                //lower odds: fed
*fow
logistic fow female mage25 fage30 c.mhtz c.fhtz med fed i.ses2 water stoiletshare kids adults if bl==1
                //higher odds: water
                //lower odds: fhtz
*pow
mlogit pow female mage25 fage30 c.mhtz c.fhtz med fed i.ses2 water stoiletshare kids adults if bl==1,
rrr
                //m0f1 water RRR 1.92 (1.15, 3.18)
                //m1f0 mage25 RRR 2.45 (1.22, 4.89); fhtz RRR 1.38 (1.05, 1.82)
                //m1f1 mage25 RRR 3.41 (1.76m 6.59); mhtz 1.35 (1.04, 1.76); fhtz 0.65 (0.48, 0.86)
                        //SES (middle, second highest, highest) all RRR>3; water 2.14 (1.21, 3.79)


        *background + SEP + health
*mhtz, fhtz, mbmi, fbmi
foreach y of varlist mhtz fhtz m_bmi f_bmi {
regress `y' female mage25 fage30 med fed i.ses2 water stoiletshare kids adults lmup fsmoke msmoke
if bl==1
}
*mow
```

```
logistic mow female mage25 fage30  c.mhtz c.fhtz med fed i.ses2 water stoiletshare kids adults lmup
fsmoke msmoke if bl==1
	//higher odds: female, mage25, ses(middle, sh, highest),
	//lower odds: fed
*fow
logistic fow female mage25 fage30  c.mhtz c.fhtz med fed i.ses2 water stoiletshare kids adults  lmup
fsmoke msmoke if bl==1
	//higher odds: water
	//lower odds: fhtz, fsmoke
*pow
mlogit pow female mage25 fage30  c.mhtz c.fhtz med fed i.ses2 water stoiletshare kids adults lmup
fsmoke msmoke if bl==1, rrr
	//m0f1: higher RRR water
	//m1f0: higher mage25, fhtz,
	//m1f1: higher RRR -  mage25, mhtz, ses (top3), water,
	//m1f1 lower RRR - fhtz

	* holds when m1f1 is binary?
recode pow (1/3=0 "No") (4=1 "Yes"), gen(pow2)
label var pow2 "Both parents o/w"
logistic pow2 female mage25 fage30 c.mhtz c.fhtz med fed i.ses2 water stoiletshare kids adults  lmup
fsmoke msmoke if bl==1
		//higher OR: mage25, mhtz, ses (all)
		//lower OR: fhtz, fsmoke
			//m1f1 couples more likely to  be older and taller women, higher SES,
				//less likely to be taller fathers or those who smoke.

** Collinearity among background variables
regress clusterid female mage25 fage30 med fed i.ses2 water stoiletshare kids adults lmup fsmoke
msmoke if bl==1
vif //all are >0.1 and <10. So we're OK! Phew!


**********************************************************
**********************************************************
*****follow-up time, using obs where length was observed

*Criteria: either IYCF or length was recorded
count if lt!=. | iycfdate!=. //17929 obs
gen follow=1 if lt!=. | iycfdate!=. //5205 miss val gen
gen id_follow=id if follow==1
gen id_follow24=id if follow==1 & agemonths<25
gen age_follow=agemonths if follow==1
gen age_follow24=agemonths if follow==1 & agemonths<25

*up to April 2016: study-time
sort id_follow
by id_follow: egen last=max(age_follow)
replace last=. if last!=age_follow
replace last=1 if last!=.

stset age_follow, id(id_follow) fail(last==1)
stdes //median duration of follow-up: 26.1

stci //median 26.2
foreach var of varlist sex mage25 fage30 ses2 med fed water stoiletshare kids adults fsmoke msmoke
lmup2 {
stci, by(`var')
stci, p(25) by(`var')
sts test `var', noshow notitle
}
sts test ses2, trend noshow notitle
	*graph of those that indicate difference
sts graph, by(ses2) title("Household wealth quintile")
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_follow_ses.gph"
sts graph, by(mage25) title("Maternal age")
```

```
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\thesis_follow_mage.gph"
sts graph, by(lmup2) title ("Pregnancy intention")
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\thesis_follow_lmup.gph"
graph combine "thesis_follow_ses.gph" "thesis_follow_lmup.gph" "thesis_follow_mage.gph", colfirst
ycommon xcommon xsize(3.5) ysize(7)
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\combine_thesis_follow_ses_mage_lm
up.gph"


*up to 24 months: person-time
sort id_follow24
bys id_follow24: egen last24=max(age_follow24)
replace last24=. if last24!=age_follow24
replace last24=1 if last24!=.

stset age_follow24, id(id_follow24) fail(last24==1)

stdes // median n of records per participant =
stci
foreach var of varlist sex mage25 fage30 ses2 med fed water stoiletshare kids adults fsmoke msmoke
lmup2 {
stci, by(`var')
stci, p(25) by(`var')
sts test `var'
}
sts test ses2, trend

        *graph
sts graph, by(mage25) ytitle(Proportion still in study) xtitle(Person-time (age in months)) title(Maternal
age) legend(on)
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\thesis_follow24_mage.gph"
sts graph, by(fage30) ytitle(Proportion still in study) xtitle(Person-time (age in months)) title(Paternal
age) legend(on)
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\thesis_follow24_fage.gph"
sts graph, by(ses2) ytitle(Proportion still in study) xtitle(Person-time (age in months)) title(Household
wealth quintile) legend(on)
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\thesis_follow24_ses.gph"
sts graph, by(stoiletshare) ytitle(Proportion still in study) xtitle(Person-time (age in months)) title(Use of
shared toilet) legend(on)
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\thesis_follow24_toilet.gph"

graph combine "thesis_follow24_mage.gph" "thesis_follow24_fage.gph" "thesis_follow24_ses.gph"
"thesis_follow24_toilet.gph" , colfirst ycommon xcommon
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\Profile\combine_thesis_follow24_mage_fage
_ses_toilet.gph"

******************
*                               DETERMINANTS OF CASE CLOSURE

        *use binary variable for highest asset quintile
recode ses2 (5=1 "Yes") (1/4 = 0 "No"), gen(highest) label(high)

tab remove_reason if bl==1, miss
recode remove_case_confirm (1=1) (.=0) if bl==1 , gen(closed)
foreach var of varlist sex mage25 fage30 highest med fed water stoiletshare kids adults fsmoke
msmoke lmup2 {
logistic closed i.`var'
}
        *all
```

```
logistic closed sex mage25 fage30  med fed highest water stoiletshare adults kids fsmoke msmoke
lmup2
        *those with p< 0.1
logistic closed mage25 fage30 highest water stoiletshare kids lmup2


*                   DETERMINANTS OF DROPOUT

        *joinby dropout variable created in the dropout.dta file in missingness.do
                *but first joinby newid from id_newid.dta because that's where both id and newid are
joinby id using id_newid.dta, unmatched(both)
joinby newid using dropout.dta, unmatched(both) _merge(_mergenew)

tab closed
tab dropout if bl==1
tab dropout closed, row chi

foreach var of varlist sex mage25 fage30  med fed highest water stoiletshare adults kids fsmoke
msmoke lmup2 {
logistic dropout i.`var' if bl==1
}
        *all
logistic dropout sex mage25 fage30 med fed highest  water stoiletshare adults kids fsmoke msmoke
lmup2 if bl==1
        *those with p< 0.1
logistic dropout  mage25 fage30  med highest water kids lmup2 if bl==1


************************************
*******************

*                   WAVE NON-RESPONSE
        *joinby wave `var'_n variables created for diarr, iycf, length from 0-24 in Rmisswide.dta
                *but first joinby newid from id_newid.dta
joinby id using id_newid.dta, unmatched(both)
joinby newid using Rmisswide.dta, unmatched(both) _merge(_mergewave)

*recode serial data 0/1 for Missing (in Rmisswide it is coded 0=observed , .= Missing
recode diarr_0-iycf_24 (.=1)
label define missing 0 "Observed" 1 "Missing"
label values diarr_0-iycf_24 missing

*label wave vars
forvalues i=0/24 {
label var diarr_`i' "Diarrhoea `i' months"
label var ht_`i' "Length `i' months"
label var iycf_`i' "IYCF `i' months"
}
        ***!! Use only one obs per child, ie. bl==1

                *DETERMINANTS OF WAVE NON-RESPONSE

        *use binary variable for highest asset quintile
recode ses2 (5=1 "Yes") (1/4 = 0 "No"), gen(highest) label(high)

        *ensure covariates are labelled correctly
d sex mage25 fage30 med fed highest water stoiletshare adults kids fsmoke msmoke lmup2
label var sex "Sex"
label var mage25 "Maternal age 25+"
label var fage30 "Paternal age 30+"
label var med "Maternal education 6+"
label var fed "Paternal education 6+"
label var highest "Highest SEP"
label var water "Piped water"
label var stoiletshare "Shared toilet"
label var adults "2+ adults"
label var kids "4+ kids"
```

```
label var lmup2 "Planned pregnancy"

*Frequency distribution of BL variables at each wave
foreach wave of varlist diarr_0-iycf_24 {
foreach blcov of varlist sex mage25 fage30 med fed highest water stoiletshare adults kids fsmoke
msmoke lmup2 {
tab `wave' `blcov' if bl==1, row chi
}
}
* Crude Effect of each BL var on participation in each wave
putexcel set "wave_thesis.xls", sheet("crude")
putexcel C1=("Odds Ratio") D1=("Std Error") F1=("p-value") G1=("95% CI") H1=("95% CI") I1=("N") ///
using "wave_thesis.xlsx", modify keepcellformat sheet("crude")
local row=2
foreach outcome of varlist diarr_0-iycf_24 {
        foreach covariate of varlist sex mage25 fage30 med fed highest water stoiletshare adults kids
fsmoke msmoke lmup2 {
        qui logistic `outcome' `covariate' if bl==1
matrix a = r(table)'
matrix a = a[.,1..6]
putexcel A`row'=matrix(a, names) using "wave_thesis.xlsx", modify keepcellformat sheet("crude")
local rowplus = `row'+1
putexcel I`rowplus' =(e(N)) using "wave_thesis.xlsx", modify keepcellformat sheet("crude")
local outlabel: variable label `outcome'
putexcel A`rowplus'=("`outlabel'") using "wave_thesis.xlsx", modify keepcellformat sheet("crude")
local covlbl : variable label `covariate'
putexcel B`rowplus' =("`covlbl'") using "wave_thesis.xlsx", modify keepcellformat sheet("crude")
local row = `row' +3
        }
        }
        * Adjusted Effect of all BL variables on participation in each wave
putexcel set "wave_thesis.xls", sheet("adjusted")
putexcel A1=("Wave") B1=("Covariate") C1=("Odds Ratio") D1=("Std Error") F1=("p-value") G1=("95%
CI") H1=("95% CI") I1=("N") ///
 using "wave_thesis.xlsx", modify keepcellformat sheet("adjusted")

local row=2
foreach outcome of varlist diarr_0-iycf_24{
qui logistic `outcome' sex mage25 fage30 med fed highest water stoiletshare adults kids fsmoke
msmoke lmup2 if bl==1
matrix a = r(table)'
matrix a = a[.,1..6]
putexcel A`row' = matrix(a, names) using "wave_thesis.xlsx", modify keepcellformat sheet("adjusted")
local rowplus = `row'+1
putexcel I`rowplus' =(e(N)) using "wave_thesis.xlsx", modify keepcellformat sheet("adjusted")
local row = `row' +15
}
*

                * DETERMINANTS OF Parental anthropometry NON-RESPONSE
*gen missing data vars, using BMI cat since it implies ht and wt were both recorded
recode m_bmicat (1/4=0 "No") (. = 1 "Yes"), gen(m_anthromiss) label(manthromiss)
recode f_bmicat (1/4=0 "No") (. = 1 "Yes"), gen(f_anthromiss) label(fanthromiss)
egen p_anthromiss=group( m_anthromiss f_anthromiss)
tab m_anthromiss f_anthromiss
tab p_anthromiss // 1=both observed
recode p_anthromiss (1=0) (2/4=1)
label values p_anthromiss manthromiss

label var m_anthromiss m_anthro
label var f_anthromiss f_anthro
label var p_anthromiss p_anthro

* Crude Effect of each BL var on participation in parental anthropometry
putexcel set "panthro_thesis.xls", sheet("crude")
putexcel C1=("Odds Ratio") D1=("Std Error") F1=("p-value") G1=("95% CI") H1=("95% CI") I1=("N") ///
using "panthro_thesis.xlsx", modify keepcellformat sheet("crude")
```

```
local row=2
foreach outcome of varlist m_anthromiss f_anthromiss p_anthromiss {
	foreach covariate of varlist sex mage25 fage30 med fed highest water stoiletshare adults kids
fsmoke msmoke lmup2 {
	qui logistic `outcome' `covariate' if bl==1
matrix a = r(table)'
matrix a = a[.,1..6]
putexcel A`row'=matrix(a, names) using "panthro_thesis.xlsx", modify keepcellformat sheet("crude")
local rowplus = `row'+1
putexcel I`rowplus' =(e(N)) using "panthro_thesis.xlsx", modify keepcellformat sheet("crude")
local outlabel: variable label `outcome'
putexcel A`rowplus'=("`outlabel'") using "panthro_thesis.xlsx", modify keepcellformat sheet("crude")
local covlbl : variable label `covariate'
putexcel B`rowplus' =("`covlbl'") using "panthro_thesis.xlsx", modify keepcellformat sheet("crude")
local row = `row' +3
	}
	}
	* Adjusted Effect of all BL variables on participation in parental anhtropmetry
putexcel set "panthro_thesis.xls", sheet("adjusted")
putexcel A1=("Outcome") B1=("Covariate") C1=("Odds Ratio") D1=("Std Error") F1=("p-value")
G1=("95% CI") H1=("95% CI") I1=("N") ///
 using "panthro_thesis.xlsx", modify keepcellformat sheet("adjusted")

local row=2
foreach outcome of varlist m_anthromiss f_anthromiss p_anthromiss{
qui logistic `outcome' sex mage25 fage30 med fed highest water stoiletshare adults kids fsmoke
msmoke lmup2 if bl==1
matrix a = r(table)'
matrix a = a[.,1..6]
putexcel A`row' = matrix(a, names) using "panthro_thesis.xlsx", modify keepcellformat
sheet("adjusted")
local rowplus = `row'+1
putexcel I`rowplus' =(e(N)) using "panthro_thesis.xlsx", modify keepcellformat sheet("adjusted")
local row = `row' +15
}
//none with OR <0.5 or >2 in univariable or multivariable analyses. Yipee!

		*** ANALYSIS PATTERNS OF MISSINGNESS

		*use iycf_long24.dta in the IYCF folder
use "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\iycf_long24.dta", clear

*for EBF /PBF,
	*after fitting discrete model, I predicted hazard
		*this generated a var called haz_e. note, it is not tagged to bl==1
egen ebf_miss = tag(id) if haz_e!=.
by id: egen ebf_miss2 = max(ebf_miss)
recode ebf_miss2 (0=1) (1=0)
tab ebf_miss2
codebook id if ebf_miss2==1 //445. correct, since 533 were in analysis
drop ebf_miss
gen ebf_miss = ebf_miss2 if bl==1
tab ebf_miss //978, with 445 obs==1. correct.

	*crude & adjusted ORs
foreach var of varlist sex mage25 fage30 med fed highest water stoiletshare adults kids fsmoke
msmoke lmup2{
logistic ebf_miss `var'
}
	//msmoke OR=0.47
logistic ebf_miss sex mage25 fage30 med fed highest water stoiletshare adults kids fsmoke msmoke
lmup2
	//no OR was <0.5 or >2.0

*for SOLIDS
	* the predicted var in discrete st model is called ff
codebook id if ff!=. //550. correct, since these many are in the analysis
```

```
egen ff_miss = tag (id) if ff!=.
by id: egen ff_miss2=max(ff_miss)
recode ff_miss2 (0=1) (1=0)
tab ff_miss2
codebook id if ff_miss2==0 //428. correct, since these were excluded
drop ff_miss
gen ff_miss=ff_miss2 if bl==1
tab ff_miss //978, with 428==1

	*crude & adjusted ORs
foreach var of varlist sex mage25 fage30 med fed highest water stoiletshare adults kids fsmoke
msmoke lmup2{
logistic ff_miss `var'
}
	//no OR was <0.5 or >2.0
logistic ff_miss sex mage25 fage30 med fed highest water stoiletshare adults kids fsmoke msmoke
lmup2
	//no OR was <0.5 or >2.0


*for MDD, ASF, and SNACKS (same subset)
	*tagging var is _est_mdd3f==1 (or replace `mdd' with asf or snk). not tagged to bl
by id: egen cf_miss=max( _est_mdd3f)
codebook id if cf_miss==1 //746
codebook id if cf_miss==0 //232
gen cf_miss2 = cf_miss if bl==1
tab cf_miss2 //978, with 746==1
recode cf_miss2 (0=1) (1=0)
tab cf_miss2 //978, with 746==0

foreach var of varlist sex mage25 fage30 med fed highest water stoiletshare adults kids fsmoke
msmoke lmup2{
logistic cf_miss2 `var'
}
	//no OR <0.5 or >2.0
logistic cf_miss2 sex mage25 fage30 med fed highest water stoiletshare adults kids fsmoke msmoke
lmup2
	//no OR <0.5 or >2.0

	*MEDIATION ANALYSIS
*use mediation analysis dataset.
use "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Mediation\komal_med_analysis.dta", clear

*tag one obs per person
gen med_miss=complete
replace med_miss=0 if med_miss==.
recode med_miss (0=1) (1=0)
egen tagged=tag(id)
gen miss=med_miss if tagged==1 //978, with 438=0 and 540==1

*recode SES2, c3
recode c3 (5=1 "Yes") (1/4 = 0 "No"), gen(c15) label(high)
*crude missingness
foreach n of numlist 14 15 8 11 1 2 4 5 6 10 12 7 13{
logistic miss c`n'
}
// mage OR=0.45 (0.3, 0.6); fage OR 0.51 (0.4, 0.7)
logistic miss c14 c15 c8 c11 c1 c2 c4 c5 c6 c10 c12 c7 c13
//none <0.5 or >2.0
```

Appendix 5.2 R code for missingness analysis

```
#Missingness map for cohort dataset, showing observed and missing data for each infant over the two years...

#X axis is time or variable of interest, all coded 0/1 for observed, missing.
#Y axis is infant ID

#data in Wide form, with 1 row per infant, and vars corresponding to ID, sex, age, set of BL vars, follow-up at each wave.
#Missingness mapped by missmap() function of package "Amelia"

#load package
library(Amelia)
library(readstata13)

setwd("N:/Documents/IGH/Analysis/Cohort")
df <- read.dta13('Rmiss.dta')
names(df)
#prepare and use 3 subsets/frames for map
  #(1) Baseline
  #(2) parents anthro
  #(3) Follow-up

#(1) All baseline
bl <- subset(df, bl==1, c(1, 5:85))
missmap(bl, legend=TRUE, col=c("grey", "black"))

#Baseline - condensed. Save as Missmap_baseline.png
blc <- subset(df, bl==1, c(7, 8,9,11, 12, 15, 18, 21, 23, 26: 28, 29, 33, 34, 53, 54, 55, 59, 60, 68, 70, 72, 73, 74, 75, 76))
missmap(blc, col=c("grey", "black"), legend=TRUE, x.cex=0.75, y.labels=NA, y.at=0, main="Selected baseline variables: Missingness Map")

#(1a) Demography (mother to mum dur)
df <- read.dta13('Rmiss.dta')
dem <- subset(df, bl==1, c(7:27))
missmap(dem, col=c("red", "grey"))

#(1b) SES
ses <-subset(df, bl==1, c(28:52))
missmap(ses, col=c("red", "grey"))

# (1c) WASH
wash <- subset(df, bl==1, c(53, 54, 55))
missmap(wash, col=c("red", "grey"))

#(1d) LMUP
lmup <- subset(df, bl==1, c(56:67))
missmap(lmup, col=c("red", "grey"))

# (1e) ANC, del
anc <- subset(df, bl==1, c(68:75))
missmap(anc, col=c("red", "grey"))

#(1f) EPDS
epds <- subset(df, bl==1, c(76:85))
missmap(epds, col=c("red", "grey"))

#(2) Parents anthro
df <- read.dta13('Rmiss.dta')
parents <- subset(df, bl==1, c(86:95))
```

```
   #map, saved as Missmap_parentanthro.png
missmap(parents, col=c("grey", "black"), legend=TRUE, y.labels=NA, y.at=0, main="Parental anthropometry")
missmap(parents, col=c("grey", "black"), legend=TRUE, rank.order=FALSE, y.labels=NA, y.at=0, main="Parental anthropometry")

  #leaving out abdominal anthro
parents2 <- subset(df, bl==1, c(86, 87, 91, 92))
missmap(parents2, col=c("grey", "black"), legend=TRUE, y.labels=NA, y.at=0, main="Parental anthropometry")


#(3) Follow-up
df <- read.dta13('Rmisswide.dta')
names(df)
diarr <- subset(df, newid>0, c(3:26))  #leave out month 0
ht <- subset(df, newid>0, c(27:51))
iycf <- subset(df, newid>0, c(53:76)) #leave out month 0

  #map, sorted by % of missingness
missmap(diarr, col=c("red", "grey"))
missmap(ht, col=c("red", "grey"))
missmap(iycf, col=c("red", "grey"))

  #map, in time seq. save as (1) Missmap_length.png (2) Missmap_diarr.png, (3) Missmap_iycf.png
missmap(ht, col=c("grey", "black"), rank.order=FALSE, legend=TRUE, y.labels=NA, y.at=0, main="Length/height: Missingness Map")
missmap(diarr, col=c("grey", "black"), rank.order=FALSE, legend=TRUE, y.labels=NA, y.at=0, main="Diarrhoea: Missingness Map")
missmap(iycf, col=c("grey", "black"), rank.order=FALSE, legend=TRUE, y.labels=NA, y.at=0, main="IYCF: Missingness Map")

#map, sorted by number of ht measurements, #(1)Ht_sort_totht.png
sortnht <- subset(df, newid>0, c(27:51))
missmap(sortnht, col=c("grey", "black"), rank.order=FALSE, legend=TRUE, y.labels=NA, y.at=0, main="Lenght/height, infants sorted by number of measurements")

#map, for those who dropped out
df <- read.dta13('Rmisswide.dta')
df<-df[order(df$dropout, df$totht),]
df<-df[order(-df$dropout, df$totht),]

dropout <- subset(df, newid>0, c(27:51))
dropout0 <- subset(df, dropout==0, c(27:51))
dropout1 <- subset(df, dropout==1, c(27:51))
dropout17m <-subset(df, dropout==1, c(27:44))

missmap(dropout, col=c("grey", "black"), rank.order=FALSE, legend=TRUE, y.labels=NA, y.at=0, main="Lenght/height, sorted by dropout")
missmap(dropout0, col=c("grey", "black"), rank.order=FALSE, legend=TRUE, y.labels=NA, y.at=0, main="Lenght/height, 607 infants who did not dropout by 18 months")
  # (1) Ht_nodropout.png
missmap(dropout1, col=c("grey", "black"), rank.order=FALSE, legend=TRUE, y.labels=NA, y.at=0, main="Lenght/height, dropped out")
missmap(dropout17m, col=c("grey", "black"), rank.order=FALSE, legend=TRUE, y.labels=NA, y.at=0, main="Lenght/height, 371 infants who dropped out by 18 months")
  #(1) Ht_dropout.png
```

Appendix 6

Appendix 6.1 R code file for SITAR

```
# CONTENTS: Re-run analyses for thesis

#PART 1. ENVIRONMENTAL DETERMINANTS OF INFANT GROWTH -- 'e' Models
   # SIMPLE MODEL
      #ALL AVAILABLE DATA
      #DATA UP TO 24 MONTHS ONLY
      #SEASONALITY ETC
   # UNIVARIABLE MODELS
      #ALL AVAILABLE DATA
      #DATA UP TO 24 MONTHS ONLY
   # 'FORCED' UNIVARIABLE MODELS
      #ALL AVAILABLE DATA
      #DATA UP TO 24 MONTHS ONLY
   # MULTIVARIABLE MODELS
      #ALL AVAILABLE DATA
      #DATA UP TO 24 MONTHS ONLY

#PART 2. EFFECT OF PARENTAL ANTHROPOMETRY ON INFANT GROWTH –
# 'FORCED' UNIVARIABLE MODELS
  # MULTIVARIABLE MODELS

##############################################################################

#LOAD PACKAGES
library(sitar)
library(foreign)
library(readstata13)

#LOAD DATASET
df <- read.dta13('cohort_anon_all.dta')
names(df)
#SELECT VARIABLES & CREATE dfs
#          2          id
#10 cdob
#          16         age
#          17         agemonths
#          21         ht
#          29         sex
#          43         fsmoke
#          55         msmoke
#          122        stoiletshre
#          556        ageint
#          558        sescore2
#          559        ses2
#          560        med
#          561        fed
#          562        water
#          563        mage25
#          564        fage30
#          567        kids
#          568        adults
#          570        lmup2
# 573, 574, 577:592 - p. anthro vars
#599 lt (Length corrected by 0.7m after 730 days)

envt <- c(2, 10,    16,       17,       21,       29,       43,       55,       122,      556,      558,
          559,      560,      561,      562,      563,      564,      567,      568,      570, 599)
df <- df[,envt]
```

```
write.dta(df, "envt.dta", convert.dates = TRUE, convert.factors = c("labels", "string", "numeric", "codes"))

df <- read.dta13('cohort_anon_all.dta')
parents <- c(2, 10, 16, 17, 21, 29, 43, 55, 122, 556, 558,
          559, 560, 561, 562, 563, 564, 567, 568, 570, 573, 574, 577:592, 599)
df <- df[,parents]
write.dta(df, "parents.dta", convert.dates = TRUE, convert.factors = c("labels", "string", "numeric", "codes"))

###(1) Environmental determinants of growth
df <- read.dta("envt.dta")
summary(df)
#RECODE ENVIRONMENTAL VARIABLES
df$sex <- factor(df$sex)
df$lmup2 <- factor(df$lmup2)
df$water <- factor(df$water)
df$mage25 <- factor(df$mage25)
df$fage30 <- factor(df$fage30)
df$kids <- factor(df$kids)
df$adults <- factor(df$adults)

#rename water
names(df) [names(df)=="ses2"] <- "ses"
#change  labels so that they do not have any spaces between them
levels(df$water)[levels(df$water)=="Not piped"] <- "NotPiped"


#value labels
levels(df$kids) [levels(df$kids) =="0"] <- "less4"
levels(df$kids) [levels(df$kids) =="1"] <- "4plus"
levels(df$adults) [levels(df$adults) =="0"] <- "less2"
levels(df$adults) [levels(df$adults) =="1"] <- "2plus"
levels(df$lmup2) [levels(df$lmup2)=="0"] <- "NotPlanned"
levels(df$lmup2) [levels(df$lmup2)=="1"] <- "Planned"
levels(df$mage25) [levels(df$mage25)=="0"] <- "below25"
levels(df$mage25) [levels(df$mage25)=="1"] <- "25plus"
levels(df$fage30) [levels(df$fage30)=="0"] <- "below30"
levels(df$fage30) [levels(df$fage30)=="1"] <- "30plus"


#drop NAs (change from 23134 to 16753)
df <- na.omit(df)

#GENERATE SEASONALITY VARIABLES

  #Fourier's
df$sint <- sinpi(df$agemonths/12 * 2)
df$cost <- cospi(df$agemonths/12 * 2)

  #Season of birth
df$mnth <- months(df$cdob)
df$mnth <- factor(df$mnth)
df$mnth <- factor(df$mnth,
                  levels = c('January', 'February', 'March', 'April', 'May', 'June', 'July', 'August', 'September', 'October', 'November', 'December'),
                  labels = c(1,2,3,4,5,6,7,8,9,10,11,12))
df$mnth <- as.numeric(df$mnth)
df$season[df$mnth<7] <- "0"
df$season[df$mnth>=7] <- "1"
df$season <- factor(df$season)
levels(df$season) [levels(df$season) =="0"] <- "Jan-Jun"
levels(df$season) [levels(df$season) =="1"] <- "Jul-Dec"

  #basic models
     #compare original vars with and without lenght correction (0.7cm)
```

```
e0 <- sitar(agemonths, ht, id, na.omit(df), 4)
summary(e0)
l0 <- sitar(agemonths, lt, id, na.omit(df), 4)
summary(l0)
  #l0 has slightly lower BIc, slightly smaller a, b,c, but slightly higher residual (1.10 vs 1.09)

#use lt instead of ht variable. refit e0 with lt var
sink(file="envt_run_e.txt")
e0 <- sitar(agemonths, lt, id, na.omit(df), 4)
summary(e0)
e2 <- update(e0, a.formula=~ sex , b.formula=~ sex , c.formula=~ sex)
summary( e2 )

e3 <- update(e0, a.formula=~ sex+mage25 , b.formula=~ sex+mage25 , c.formula=~ sex+ mage25 )
summary( e3 )

e4 <- update(e0, a.formula=~ sex+ fage30 , b.formula=~ sex+ fage30 , c.formula=~ sex+ fage30 )
summary( e4 )

e5 <- update(e0, a.formula=~ sex+ sescore2 , b.formula=~ sex+ sescore2 , c.formula=~ sex+ sescore2 )
summary( e5 )

e6 <- update(e0, a.formula=~ sex+ ses , b.formula=~ sex+ ses , c.formula=~ sex+ ses )
summary( e6 )

e7 <- update(e0, a.formula=~ sex+ med , b.formula=~ sex+ med , c.formula=~ sex+ med )
summary( e7 )

e8 <- update(e0, a.formula=~ sex+ fed , b.formula=~ sex+ fed , c.formula=~ sex+ fed )
summary( e8 )

e9 <- update(e0, a.formula=~ sex+ water , b.formula=~ sex+ water , c.formula=~ sex+ water )
summary( e9 )

e10 <- update(e0, a.formula=~ sex+ stoiletshare , b.formula=~ sex+ stoiletshare , c.formula=~ sex+ stoiletshare )
summary( e10 )

e11 <- update(e0, a.formula=~ sex+ kids , b.formula=~ sex+ kids , c.formula=~ sex+ kids )
summary( e11 )

e12 <- update(e0, a.formula=~ sex+ adults , b.formula=~ sex+ adults , c.formula=~ sex+ adults )
summary( e12 )

e13 <- update(e0, a.formula=~ sex+ lmup2 , b.formula=~ sex+ lmup2 , c.formula=~ sex+ lmup2 )
summary( e13 )

e14 <- update(e0, a.formula=~ sex+ fsmoke , b.formula=~ sex+ fsmoke , c.formula=~ sex+ fsmoke )
summary( e14 )

e15 <- update(e0, a.formula=~ sex+ msmoke , b.formula=~ sex+ msmoke , c.formula=~ sex+ msmoke )
summary( e15 )
```

```
e16 <- update(e0, a.formula=~ sex+
mage25+fage30+ses+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke+msmoke ,
b.formula=~ sex+
mage25+fage30+ses+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke+msmoke ,
c.formula=~ sex+
mage25+fage30+ses+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke+msmoke )
summary( e16 )

e17 <- update(e0, a.formula=~ sex+
mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke+msmo
ke , b.formula=~ sex+
mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke+msmo
ke , c.formula=~ sex+
mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke+msmo
ke )
summary( e17 )


sink()

#save fitted models
save(e0,file="e0")
save(e2,file="e2")
save(e3,file="e3")
save(e4,file="e4")
save(e5,file="e5")
save(e6,file="e6")
save(e7,file="e7")
save(e8,file="e8")
save(e9,file="e9")
save(e10,file="e10")
save(e11,file="e11")
save(e12,file="e12")
save(e13,file="e13")
save(e14,file="e14")
save(e15,file="e15")
save(e16,file="e16")
save(e17,file="e17")

#sink and save es models output
sink(file="envt.txt")
model_envt<-list(e0, e2, e3, e4, e5, e6, e7, e8, e9, e10, e11, e12, e13, e14, e15, e16, e17)
lapply(model_envt, summary)
lapply(model_envt, varexp)
BICadj(pattern='^e')
sink()
   #ES models
sink(file="envt_run_es.txt")
es1 <- update(e0, a.formula=~ sex , b.formula=~ sex , c.formula=~ sex
)
summary( es1 )

es2 <- update(e0, a.formula=~ sex+sint+cost , b.formula=~ sex+sint+cost ,
c.formula=~ sex+sint+cost )
summary( es2 )

es3 <- update(e0, a.formula=~ sex+sint+cost+ mage25 , b.formula=~
sex+sint+cost+ mage25 , c.formula=~ sex+sint+cost+ mage25 )
summary( es3 )

es4 <- update(e0, a.formula=~ sex+sint+cost+ fage30 , b.formula=~
sex+sint+cost+ fage30 , c.formula=~ sex+sint+cost+ fage30 )
summary( es4 )

es5 <- update(e0, a.formula=~ sex+sint+cost+ sescore2 , b.formula=~
sex+sint+cost+ sescore2 , c.formula=~ sex+sint+cost+ sescore2 )
```

```
summary(	es5	)

es6	<-	update(e0, a.formula=~	sex+sint+cost+	ses	,	b.formula=~
	sex+sint+cost+	ses	, c.formula=~	sex+sint+cost+	ses	)
summary(	es6	)

es7	<-	update(e0, a.formula=~	sex+sint+cost+	med	,	b.formula=~
	sex+sint+cost+	med	, c.formula=~	sex+sint+cost+	med	)
 #did not converge
summary(	es7	)

#leave to run o/n 28nov18
es8	<-	update(e0, a.formula=~	sex+sint+cost+	fed	,	b.formula=~
	sex+sint+cost+	fed	, c.formula=~	sex+sint+cost+	fed	)
summary(	es8	)

es9	<-	update(e0, a.formula=~	sex+sint+cost+	water	,	b.formula=~
	sex+sint+cost+	water	, c.formula=~	sex+sint+cost+	water	)
summary(	es9	)

es10	<-	update(e0, a.formula=~	sex+sint+cost+	stoiletshare	,	b.formula=~
	sex+sint+cost+	stoiletshare	, c.formula=~	sex+sint+cost+	stoiletshare	)
summary(	es10	)

es11	<-	update(e0, a.formula=~	sex+sint+cost+	kids	,	b.formula=~
	sex+sint+cost+	kids	, c.formula=~	sex+sint+cost+	kids	)
summary(	es11	)

es12	<-	update(e0, a.formula=~	sex+sint+cost+	adults	,	b.formula=~
	sex+sint+cost+	adults	, c.formula=~	sex+sint+cost+	adults	)
summary(	es12	)

es13	<-	update(e0, a.formula=~	sex+sint+cost+	lmup2	,	b.formula=~
	sex+sint+cost+	lmup2	, c.formula=~	sex+sint+cost+	lmup2	)
summary(	es13	)
#es13 did not converge
es14	<-	update(e0, a.formula=~	sex+sint+cost+	fsmoke	,	b.formula=~
	sex+sint+cost+	fsmoke	, c.formula=~	sex+sint+cost+	fsmoke	)
summary(	es14	)

es15	<-	update(e0, a.formula=~	sex+sint+cost+	msmoke	,	b.formula=~
	sex+sint+cost+	msmoke	, c.formula=~	sex+sint+cost+	msmoke	)
summary(	es15	)

es16	<-	update(e0,	a.formula=~
	sex+sint+cost+mage25+fage30+ses+med+fed+water+stoiletshare+kids+adults+lmup2+fsmok
e+msmoke	,	b.formula=~
	sex+sint+cost+mage25+fage30+ses+med+fed+water+stoiletshare+kids+adults+lmup2+fsmok
e+msmoke	,	c.formula=~
	sex+sint+cost+mage25+fage30+ses+med+fed+water+stoiletshare+kids+adults+lmup2+fsmok
e+msmoke)
summary(	es16	)
es17	<-	update(e0,	a.formula=~
	sex+sint+cost+mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+f
smoke+msmoke	,	b.formula=~
	sex+sint+cost+mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+f
smoke+msmoke	,	c.formula=~
	sex+sint+cost+mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+f
smoke+msmoke)
summary(	es17	)
es18	<-	update(e0,
a.formula=~sex+sint+cost+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+msmoke,
b.formula=~	sex+sint+cost+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+msmoke,
c.formula=~sex+sint+cost+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+msmoke)
summary(	es18)
es19	<-	update(e0,
a.formula=~sex+sint+cost+ses+med+fed+water+stoiletshare+kids+adults+lmup2+msmoke,
```

```
b.formula=~ sex+sint+cost+ses+med+fed+water+stoiletshare+kids+adults+lmup2+msmoke,
c.formula=~sex+sint+cost+ses+med+fed+water+stoiletshare+kids+adults+lmup2+msmoke)
summary( es19)
save(es8,file="es8")
save(es9,file="es9")
save(es10,file="es10")
save(es11,file="es11")
save(es12,file="es12")
save(es13,file="es13")
save(es14,file="es14")
save(es15,file="es15")
save(es17,file="es17")
save(es18,file="es18")
save(es19,file="es19")


e16 <- update(e0, a.formula=~ sex+
mage25+fage30+ses+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke+msmoke ,
b.formula=~ sex+
mage25+fage30+ses+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke+msmoke ,
c.formula=~ sex+
mage25+fage30+ses+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke+msmoke )
summary( e16 )

e17 <- update(e0, a.formula=~ sex+
mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke+msmo
ke , b.formula=~ sex+
mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke+msmo
ke , c.formula=~ sex+
mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke+msmo
ke )
summary( e17 )

e18 <- update(e0,
a.formula=~sex+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke, b.formula=~
sex+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke,
c.formula=~sex+sescore2+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke)
summary(e18)
e19 <- update(e0,
a.formula=~sex+ses+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke,
b.formula=~sex+ses+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke,
c.formula=~sex+ses+med+fed+water+stoiletshare+kids+adults+lmup2+fsmoke)
summary(e19)
save(e16,file="e16")
save(e17,file="e17")
save(e18,file="e18")
save(e19,file="e19")


#sink models
sink(file="envt_es.txt")
model_envtes<-list(e0, es2, es3, es4, es5, es6, es7, es8, es9, es10, es11, es12, es13, es14, es15,
e17, e18, e19)
lapply(model_envtes, summary)
lapply(model_envtes, varexp)
BICadj(pattern='^es')
sink()

sink(file="envt.txt")
model_envt<-list(e0, e2, e3, e4, e5, e6, e7, e8, e9, e10, e11, e12, e13, e14, e15, e16, e17, e18, e19)
lapply(model_envt, summary)
lapply(model_envt, varexp)
BICadj(pattern='^e')
sink()

 #end of o/n
#save es models
```

```
save(es2,file="es2")
save(es3,file="es3")
save(es4,file="es4")
save(es5,file="es5")
save(es6,file="es6")
save(es7,file="es7")
save(es8,file="es8")
save(es9,file="es9")
save(es10,file="es10")
save(es11,file="es11")
save(es12,file="es12")
save(es13,file="es13")
save(es14,file="es14")
save(es15,file="es15")
save(es17,file="es17")

#PLOT ES models

plot(es6, 'dv', col=3, y2par=list(col=3), subset=ses=='Lowest', xlegend=NULL, apv=TRUE)
lines(es6, col=2, subset=ses=='Secondlowest', y2par=list(col=2), apv=TRUE)
lines(es6, col=4, subset=ses=='Middle', y2par=list(col=4), apv=TRUE)

plot(es6, 'dv', xlegend=NULL, apv=TRUE)
lines(es6, col=2, subset=sescore2<=-1, y2par=list(col=2), apv=TRUE) #red
lines(es6, col=4, subset=sescore2>= 1, y2par=list(col=4), apv=TRUE) #blue

  #full - asset +1SD -1SD
plot(es17, 'dv', subset=sescore2<= -1, xlegend=NULL, apv=TRUE)
lines(es17, col=6, subset=sescore2>= 1, y2par=list(col=6), apv=TRUE)

  #full - toilet
plot(es17, 'dv', subset=stoiletshare=="Yes", xlegend=NULL, apv=TRUE)
lines(es17, col=6, subset=stoiletshare=="No", y2par=list(col=6), apv=TRUE)


    #ALL AVAILABLE DATA


#checking data etc
  #identify and list outliers
  if (interactive()) plotclean(agemonths, lt, id, df)

  #plot residuals, with and without outliers labelled
  plot.lme(e0, pch=20, idLabels=~id, id=0.0001)
  plot.lme(e0, pch=20)
  #inspect residuals for departure from normality
qqnorm(resid(e0), col="blue")
qqline(resid(e0), col="red")

#plots
  #simple
plot(e0, xlegend=NULL, apv=TRUE, xlab="Age (months)", ylab="Length (cm)", y2lab="Length velocity
(cm/month)")

 #distance curves for tempo -1 and +1, mean velocity
plot(e0, 'd', xlab="Age (months)", ylab="Length (cm)", abc=c(c=0, b=-1), col="blue")
lines(e0, 'd', abc=c(c=0, b=1), col="red")
legend(1, 90, legend=c("Early tempo", "Late tempo"), col=c("blue", "red"), bty="n", cex=0.8, lwd=1)

# distance curve for velocity -0.1 and +0.1, mean tempo
plot(e0, 'd', xlab="Age (months)", ylab="Length (cm)", abc=c(b=0, c=-0.1), col="navy")
lines(e0, 'd', abc=c(b=0, c=0.1), col="maroon")
legend(1, 85, legend=c("Low velocity", "High velocity"), col=c("navy", "maroon"), bty="n", cex=0.8,
lwd=1)

#velocity curve for early or late tempo, mean size
plot(e0, 'v', xlab="Age (months)", ylab="Length (cm)", abc=c(a=0, b=-1), col="blue")
```

```
lines(e0, 'v', abc=c(a=0, b=1), col="red")
legend('topright', legend=c("Early tempo", "Late tempo"), col=c("blue", "red"), bty="n", cex=0.8, lwd=1)

#velocity curve for more/late vs less/early
plot(e0, 'v', xlab="Age (months)", ylab="Length (cm)", abc=c(a=1, b=-1), col="blue")
lines(e0, 'v', abc=c(a=-1, b=1), col="red")
legend('topright', legend=c("Less/early", "More/late"), col=c("blue", "red"), bty="n", cex=0.8, lwd=1)

# distance curves for later tempo, slow velocity vs early tempo, fast velocity
plot(e0, 'd', xlab="Age (months)", ylab="Length (cm)", abc=c(b=1, c=-0.1), col="blue")
lines(e0, 'd', abc=c(b=-1, c=0.1), col="red")
legend(1,90, legend=c("Late tempo, low velocity", "Early tempo, high velocity"), col=c("blue", "red"),
bty="n", cex=0.8, lwd=1)

#distance curves for less/late/slow vs more/early/fast - ideally
plot(e0, 'd', xlab="Age (months)", ylab="Length (cm)", abc=c(a=-1, b=1, c=-0.1), col="navy", lwd=2,
ylim=c(40,95))
lines(e0, 'd', abc=c(a=1, b=-1, c=0.1), col="maroon", lwd=2)
legend('topleft', legend=c("More/early/fast", "Less/late/slow"), col=c("maroon", "navy"), bty="n",
cex=0.8, lwd=2)

#distance curves for more/late/slow vs less/early/fast - what the model predicts
plot(e0, 'd', xlab="Age (months)", ylab="Length (cm)", abc=c(a=1, b=1, c=-0.1), col="blue", lwd=2)
lines(e0, 'd', abc=c(a=-1, b=-1, c=0.1), col="red", lwd=2)
legend(1,90, legend=c("More/late/slow", "Less/early/fast"), col=c("blue", "red"), bty="n", cex=0.8,
lwd=2)

#velocity curve for those with more size gain or less size gain, mean tempo

# mean curve for a, b,c=-1SD (did not work)
plot(e0, 'd', xlab="Age (months)", ylab="Length (cm)")
lines(e0, 'd', lwd=2, abc=-sqrt(diag(getVarCov(e0))))

#predicted lengths and velocities
predict(e0,  newdata=data.frame(agemonths=0:37), level=0)
predict(e0, deriv=1, newdata=data.frame(agemonths=0:37), level=0)

      #length
# [1] 45.67855 50.46756 54.42044 57.63560 60.21145 62.24639 63.83883 65.08719 66.08987
66.94528 67.75150
#[12] 68.57356 69.41522 70.27362 71.14590 72.02918 72.92060 73.81730 74.71640 75.61504
76.51035 77.39947
#[23] 78.27953 79.14766 80.00099 80.83667 81.65182 82.44357 83.20906 83.94543 84.64980
85.31930 85.95108
#[34] 86.54227 87.09067 87.59658 88.06089 88.48449


   # velocity. deriv=1
#[1] 4.9700925 4.4268309 3.5482477 2.8842912 2.2875997 1.7976716 1.4038750 1.1092390
0.9122791 0.8159046
#[11] 0.8089952 0.8329553 0.8503458 0.8658617 0.8782451 0.8878330 0.8945351 0.8983756
0.8993480 0.8974540
#[21] 0.8926933 0.8850659 0.8745717 0.8612109 0.8449833 0.8258890 0.8039280 0.7791003
0.7514058 0.7208451
#[31] 0.6874155 0.6511273 0.6119424 0.5700025 0.5267912 0.4857478 0.4408829 0.4144625


#predict lengths of those with early and late apv (-ve and +ve b)
predict(e0, newdata=data.frame(agemonths=0:37), level=0, abc=c(b=-1))
#[1] 50.46756 54.42044 57.63560 60.21145 62.24639 63.83883 65.08719 66.08987 66.94528
67.75150 68.57356
#[12] 69.41522 70.27362 71.14590 72.02918 72.92060 73.81730 74.71640 75.61504 76.51035
77.39947 78.27953
#[23] 79.14766 80.00099 80.83667 81.65182 82.44357 83.20906 83.94543 84.64980 85.31930
85.95108 86.54227
#[34] 87.09067 87.59658 88.06089 88.48449 88.89953
```

```
predict(e0, newdata=data.frame(agemonths=0:37), level=0, abc=c(b=1))
#[1] 40.70846 45.67855 50.46756 54.42044 57.63560 60.21145 62.24639 63.83883 65.08719
66.08987 66.94528
#[12] 67.75150 68.57356 69.41522 70.27362 71.14590 72.02918 72.92060 73.81730 74.71640
75.61504 76.51035
#[23] 77.39947 78.27953 79.14766 80.00099 80.83667 81.65182 82.44357 83.20906 83.94543
84.64980 85.31930
#[34] 85.95108 86.54227 87.09067 87.59658 88.06089

#predict velocities of those with early and late apv (-ve and +ve b)
predict(e0, deriv=1, newdata=data.frame(agemonths=0:37), level=0, abc=c(b=-1))
#[1] 4.4268309 3.5482477 2.8842912 2.2875997 1.7976716 1.4038750 1.1092390 0.9122791
0.8159046 0.8089952
#[11] 0.8329553 0.8503458 0.8658617 0.8782451 0.8878330 0.8945351 0.8983756 0.8993480
0.8974540 0.8926933
#[21] 0.8850659 0.8745717 0.8612109 0.8449833 0.8258890 0.8039280 0.7791003 0.7514058
0.7208451 0.6874155
#[31] 0.6511273 0.6119424 0.5700025 0.5267912 0.4857478 0.4408829 0.4144625 0.3933039

predict(e0, deriv=1, newdata=data.frame(agemonths=0:37), level=0, abc=c(b=1))
#[1] 5.4089428 4.9700925 4.4268309 3.5482477 2.8842912 2.2875997 1.7976716 1.4038750
1.1092390 0.9122791
#[11] 0.8159046 0.8089952 0.8329553 0.8503458 0.8658617 0.8782451 0.8878330 0.8945351
0.8983756 0.8993480
#[21] 0.8974540 0.8926933 0.8850659 0.8745717 0.8612109 0.8449833 0.8258890 0.8039280
0.7791003 0.7514058
#[31] 0.7208451 0.6874155 0.6511273 0.6119424 0.5700025 0.5267912 0.4857478 0.4408829

# predicted lenghts for less/late/slow and more/early/fast
predict(e0, newdata=data.frame(agemonths=0:37), level=0, abc=c(a=-1, b=1, c=-0.1))
predict(e0, newdata=data.frame(agemonths=0:37), level=0, abc=c(a=1, b=-1, c=0.1))

#those who grow more are longer
predict(e0, newdata=data.frame(agemonths=0:37), level=0, abc=c(a=1))
predict(e0, newdata=data.frame(agemonths=0:37), level=0, abc=c(a=-1))
#those who are later are always shorter
predict(e0, newdata=data.frame(agemonths=0:37), level=0, abc=c(b=1))
predict(e0, newdata=data.frame(agemonths=0:37), level=0, abc=c(b=-1))
#those who grow faster start short but are longer eventually
predict(e0, newdata=data.frame(agemonths=0:37), level=0, abc=c(c=0.1))
predict(e0, newdata=data.frame(agemonths=0:37), level=0, abc=c(c=-0.1))

#sex-adjusted
plot(e2, xlegend=NULL, apv=TRUE, xlab="Age (months)", ylab="Length (cm)", y2lab="Length velocity
(cm/month)", subset=sex==1, col="red", y2par=list(col="red", lwd=2), lwd=2)
lines(e2, xlegend=NULL, apv=TRUE, xlab="Age (months)", ylab="Length (cm)", y2lab="Length velocity
(cm/month)", subset=sex==2, col="blue", y2par=list(col="blue", lwd=2), lwd=2)
legend(5, 90, legend=c("Male", "Female"), col=c("red", "blue"), bty="n", cex=0.8, lwd=2)

#sex-seasonality adjusted
plot(es2, xlegend=NULL, apv=TRUE, xlab="Age (months)", ylab="Length (cm)", y2lab="Length velocity
(cm/month)", subset=sex==1, col="red", y2par=list(col="red", lwd=2), lwd=2)
lines(es2, xlegend=NULL, apv=TRUE, xlab="Age (months)", ylab="Length (cm)", y2lab="Length
velocity (cm/month)", subset=sex==2, col="blue", y2par=list(col="blue", lwd=2), lwd=2)
legend(5, 90, legend=c("Male", "Female"), col=c("red", "blue"), bty="n", cex=0.8, lwd=2)

#compare velocity curves of e2 and es2
plot(e2, 'v',  xlab="Age (months)", ylab="Length (cm)", apv=TRUE, lwd=2)
lines(es2, 'v', col="blue", apv=TRUE, y2par=list(col="blue"), lwd=2)
legend('topright', legend=c("Sex-adjusted", "Sex-seasonality adjusted"), col=c("black", "blue"), bty="n",
cex=0.8, lwd=2)

#compare distance and velocity curves of e2 and es2
plot(e2, 'dv', xlab="Age (months)", ylab="Length (cm)", apv=TRUE, lwd=2, y2par=list(lwd=2),
xlegend=NULL)
lines(es2, 'dv', col="blue", apv=TRUE, y2par=list(col="blue", lwd=2), lwd=2)
```

```
legend(19, 75, legend=c("Sex-adjusted", "Sex-seasonality adjusted"), col=c("black", "blue"), bty="n", cex=0.8, lwd=2)

#compare distance curves of 1st and 3rd quartiles of sescore
plot(es17, 'dv', xlab="Age (months)", ylab="Length (cm)", lwd=2, subset=sescore2>=0.83640, col="forestgreen", apv=TRUE, xlegend=NULL, y2par=list(col="forestgreen", lwd=2))
lines(es17, 'dv', xlab="Age (months)", ylab="Length (cm)", lwd=2, subset=sescore2<=-0.64880, col="deeppink", apv=TRUE, xlegend=NULL, y2par=list(col="deeppink", lwd=2))
legend(19, 75, legend=c("Highest asset quartile", "Lowest asset quartile"), col=c("forestgreen", "deeppink"), bty='n', cex=0.8, lwd=2)
#   apv     pv for forest green
#16.380  1.123

#    apv      pv for deeppink
#19.5800  0.9442

#compare curves by toilet facility
plot(es17, 'dv', xlab="Age (months)", ylab="Length (cm)", lwd=2, subset=stoiletshare=="Yes", col="blueviolet", apv=TRUE, xlegend=NULL, y2par=list(col="blueviolet", lwd=2))
lines(es17, 'dv', xlab="Age (months)", ylab="Length (cm)", lwd=2, subset=stoiletshare=="No", col="blue", apv=TRUE, xlegend=NULL, y2par=list(col="blue", lwd=2))
legend(25, 75, legend=c("Shared toilet", "Own toilet"), col=c("blueviolet", "blue"), bty='n', cex=0.8, lwd=2)

#    apv      pv for shared toilet
#18.5700  0.9939
#apv     pv for own toilet
#15.820  1.162

#compare curves by toilet facility (without seasons). pretty similar.
plot(e17, 'dv', xlab="Age (months)", ylab="Length (cm)", lwd=2, subset=stoiletshare=="Yes", col="blueviolet", apv=TRUE, xlegend=NULL, y2par=list(col="blueviolet"))
lines(e17, 'dv', xlab="Age (months)", ylab="Length (cm)", lwd=2, subset=stoiletshare=="No", col="orange", apv=TRUE, xlegend=NULL, y2par=list(col="orange"))
legend(25, 75, legend=c("Shared toilet", "Own toilet"), col=c("blueviolet", "orange"), bty='n', cex=0.8, lwd=1)

# by kids
plot(es17, 'dv', xlab="Age (months)", ylab="Length (cm)", lwd=2, subset=kids=="less4", col="slategray", apv=TRUE, xlegend=NULL, y2par=list(col="slategray", lwd=2))
lines(es17, 'dv', xlab="Age (months)", ylab="Length (cm)", lwd=2, subset=kids=="4plus", col="magenta", apv=TRUE, xlegend=NULL, y2par=list(col="magenta", lwd=2))
legend(25, 75, legend=c("<4 children", "4+ children"), col=c("slategray", "magenta"), bty='n', cex=0.8, lwd=1)

##### 2. PARENTAL ANTHROPOMETRY
df <- read.dta("parents.dta")
summary(df)
  ##convert to factor
df$sex <- factor(df$sex)
df$lmup2 <- factor(df$lmup2)
df$water <- factor(df$water)
df$mage25 <- factor(df$mage25)
df$fage30 <- factor(df$fage30)
df$kids <- factor(df$kids)
df$adults <- factor(df$adults)

df[,c(22, 24:29, 38)] <- lapply(df[,c(22, 24:29, 38)], as.factor)


#fouriers
#Fourier's
df$sint <- sinpi(df$agemonths/12 * 2)
df$cost <- cospi(df$agemonths/12 * 2)

u0 <- sitar(agemonths, lt, id, na.omit(df), 4)
```

```
#First set: 4 exposure models

#pow
p1 <- update(u0, a.formula=~pow+sex+sint+cost, b.formula=~pow+sex+sint+cost,
c.formula=~pow+sex+sint+cost)

#zscores
z1 <- update(u0, a.formula=~mhtz+fhtz+mwtz+fwtz+sex+sint+cost,
b.formula=~mhtz+fhtz+mwtz+fwtz+sex+sint+cost, c.formula=~mhtz+fhtz+mwtz+fwtz+sex+sint+cost)

#sum
s1 <- update(u0, a.formula=~phtz+pwtz+sex+sint+cost, b.formula=~phtz+pwtz+sex+sint+cost,
c.formula=~phtz+pwtz+sex+sint+cost)

#difference
d1 <- update(u0, a.formula=~phtzdiff+pwtzdiff+sex+sint+cost,
b.formula=~phtzdiff+pwtzdiff+sex+sint+cost, c.formula=~phtzdiff+pwtzdiff+sex+sint+cost)

#varexp for all
varexp(s1)
#88.96
varexp(p1)
#88.97
varexp(d1)
#88.97
varexp(z1)
#88.96

#which fits best?
BIC(s1, p1, d1, z1)
#df      BIC
#s1 29 42478.89
#p1 32 42562.78
#d1 29 42534.89
#z1 35 42531.90

#s1, but i wanted  to use pow anyway, so i fit both and compared them.

#Second set
pfull <- update(u0,
a.formula=~ sex+sint+cost+pow+mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+ad
ults+lmup2+fsmoke+msmoke ,
b.formula=~ sex+sint+cost+pow+mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+ad
ults+lmup2+fsmoke+msmoke ,
c.formula=~ sex+sint+cost+pow+mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+adul
ts+lmup2+fsmoke+msmoke)

sfull <- update(u0,
a.formula=~ phtz+pwtz+sex+sint+cost+mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+
adults+lmup2+fsmoke+msmoke ,
b.formula=~ phtz+pwtz+sex+sint+cost+mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+
adults+lmup2+fsmoke+msmoke ,
c.formula=~ phtz+pwtz+sex+sint+cost+mage25+fage30+sescore2+med+fed+water+stoiletshare+kids+
adults+lmup2+fsmoke+msmoke)


#save models
save(u0, file="u0")
save(s1, file="s1")
save(p1, file="p1")
save(z1, file="z1")
save(d1, file="d1")
save(pfull, file="pfull")
save(sfull, file="sfull")

#plot s1 curves by pheight and pweight zscore at IQR
```

```
summary(df$phtz) #-1.055, 0.947
summary(df$pwtz)#-1.1, 0.950

plot(s1, 'd', xlab="Age (months)", ylab="Length (cm)", lwd=2, subset=phtz<=-1.055, col='blueviolet', ylim=c(45,90))
lines(s1, 'd', lwd=2, subset=phtz>=0.947, col='blue')
lines(s1, 'd', lwd=2, subset=pwtz<=-1.1, col='magenta')
lines(s1, 'd', lwd=2, subset=pwtz>=0.950, col='forestgreen')
legend('topleft', legend=c("Parental heights 3rd Q","Parental weights 3rd Q", "Parental weights 1st Q","Parental heights 1st Q"), col=c('blue', 'forestgreen', 'magenta','blueviolet'), bty='n', cex=0.8, lwd=1)


#plot sfull curves by pweight and pheight zscore at IQR - use in thesis
plot(sfull, 'd', xlab="Age (months)", ylab="Length (cm)", lwd=2, subset=phtz<=-1.055, col='blueviolet', ylim=c(45,90))
lines(sfull, 'd', lwd=2, subset=phtz>=0.947, col='blue')
lines(sfull, 'd', lwd=2, subset=pwtz<=-1.1, col='magenta')
lines(sfull, 'd', lwd=2, subset=pwtz>=0.950, col='forestgreen')
legend('topleft', legend=c("Parental heights: 3rd Quartile","Parental weights: 3rd Quartile", "Parental weights: 1st Quartile","Parental heights: 1st Quartile"), col=c('blue', 'forestgreen', 'magenta','blueviolet'), bty='n', cex=0.8, lwd=2)


#plot sfull curves by +1 and -1 SD of pweight and pheight zscore
plot(sfull, 'd', xlab="Age (months)", ylab="Length (cm)", lwd=2, subset=phtz<=-1, col='blueviolet', ylim=c(45,90))
lines(sfull, 'd', lwd=2, subset=phtz>=1, col='blue')
lines(sfull, 'd', lwd=2, subset=pwtz<=-1, col='magenta')
lines(sfull, 'd', lwd=2, subset=pwtz>=1, col='forestgreen')
legend('topleft', legend=c("Parental heights: >= +1SD","Parental weights: >= +1SD", "Parental weights: <= -1SD","Parental heights: <= -1SD"), col=c('blue', 'forestgreen', 'magenta','blueviolet'), bty='n', cex=0.8, lwd=2)

#plot sfull curves by +2 and -2 SD of pweight and pheight zscore
plot(sfull, 'dv', xlab="Age (months)", ylab="Length (cm)", vlab="Length (cm) velocity", lwd=2, subset=phtz<=-2, col='blueviolet', ylim=c(45,90), y2par=list(col='blueviolet', lwd=2), legend=NULL)
lines(sfull, 'dv', lwd=2, subset=phtz>=2, col='blue', y2par=list(col='blue', lwd=2))
lines(sfull, 'dv', lwd=2, subset=pwtz<=-2, col='magenta', y2par=list(col='magenta', lwd=2))
lines(sfull, 'dv', lwd=2, subset=pwtz>=2, col='forestgreen', y2par=list(col='forestgreen', lwd=2))
legend(3, 90, legend=c("Parental heights: >= +2SD","Parental weights: >= +2SD", "Parental weights: <= -2SD","Parental heights: <= -2SD"), col=c('blue', 'forestgreen', 'magenta','blueviolet'), bty='n', cex=0.8, lwd=2)
```

Appendix 7

Appendix 7.1 Stata .do file for IYCF analysis

```
A/**Chapter 7: IYCF analysis */
cd "N:\Documents\IGH\Analysis\Cohort\IYCF"

**********Initiation of breastfeeding
codebook id if bfever==1 &  bl==1
codebook id if bfever==0 &  bl==1
*bf initiated within hours
sum bfstart if bl==1 & bfever==1 & bfstart<88, detail //since 88 and 99 are skip/dk
sum bfstart if bl==1 & bfever==1 & bfstart<88 //since 88 and 99 are skip/dk

	*within days
sum bfstartd if bl==1 & bfever==1, detail
sum bfstartd if bl==1 & bfever==1

*prelacteals
tab bfprelac if bl==1
	*most common
tab1 bfprelachoney bfprelacmilk bfprelacwater bfprelacsugar bfprelacgripe ///
bfprelacsugsalt bfprelacjuice bfprelacformula bfprelactea  bfprelacjanam ///
bfprelacother if bl==1
	*most commonly given together
tab2 bfprelachoney bfprelacmilk bfprelacwater bfprelacsugar bfprelacgripe ///
bfprelacsugsalt bfprelacjuice bfprelacformula bfprelactea  bfprelacjanam ///
bfprelacother if bl==1, first
	*background determinants of pre-lacteal feeding
foreach cov of varlist female mage25 fage30 med fed water stoiletshare ses2 kids adults fsmoke
msmoke lmup2{
logistic bfprelac i.`cov' if bl==1
}
	//more likely to give: top 3 asset quintiles, piped water, and older fathers.
	//less likely to give: those who shre a toilet


**** (0) Repeated cross-sectional data: descriptive only, send to appendix.
use "N:\Documents\IGH\Analysis\Cohort\cohort_anon_all.dta", clear

*gen var for each child's first obs in each month
sort id age
egen month_1st = tag(id ageint)

*proportion of bf, EBF, PBF at each month 0-5 months
foreach var of varlist bfnow ebf pbfnow {
tab ageint `var' if ageint>0 & ageint<6 & month_1st==1, row
}
	*any bf
graph bar (count) if ageint>0 & ageint<6 & month_1st==1, over(bfnow) over(ageint) asyvars stack
percent blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue)) bar(2, color(ltblue)) ytitle(%
of infants breastfed) title(Breastfeeding (1-5 months))
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_bf15.gph",
replace
	*ebf
graph bar (count) if ageint>0 & ageint<6 & month_1st==1, over(ebf) over(ageint) asyvars stack percent
blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue)) bar(2, color(ltblue)) ytitle(% of infants
exclusively breastfed) title(Exclusive breastfeeding (1-5 months))
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_ebf15.gph"
	*pbf
graph bar (count) if ageint>0 & ageint<6 & month_1st==1, over(pbfnow) over(ageint) asyvars stack
percent blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue)) bar(2, color(ltblue)) ytitle(%
of infants predominantly breastfed) title(Predominant breastfeeding (1-5 months))
```

```
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_pbf15.gph",
replace
        *combined graph
graph combine "thesis_bf15.gph" "thesis_ebf15.gph" "thesis_pbf15.gph", cols(1)
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_bf_ebf_pbf_15.gph"

*median number of times breastfed in the last 24 hours by month
forvalues i=1/5{
sum bfx bfdayx bfnightx if ageint==`i' & bfdayx!=. & bfnightx!=., detail
}
graph box bfx if ageint>0 & ageint<6 & bfdayx!=. & bfnightx!=., over(ageint) scheme(s1color)
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_bfx.gph",
replace
graph box bfdayx bfnightx if ageint>0 & ageint<6 & bfdayx!=. & bfnightx!=., over(ageint)
scheme(s1color) ytitle(No. of times breastfed) title(Day and night-time breastfeeding)
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_bfx_DayNight.gph"

graph combine "thesis_bfx.gph" "thesis_bfx_DayNight.gph", cols(1) xcommon
        //re-jig titles and size manually.
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_combi_bfx_DayNight.gph"

        *note n for each
forvalues i=1/5{
sum bfx bfdayx bfnightx if ageint==`i' & bfdayx!=. & bfnightx!=., detail
}
*Formula feeding at each age.
tab ageint bfluidformula if ageint>0 & ageint<6 & month_1st==1, row
graph bar (count) if ageint>0 & ageint<6 & month_1st==1 & bfluidformula!=99, ///
over(bfluidformula) over(ageint) asyvars stack percent ///
blabel(bar, position(center) format(%3.0f)) ///
bar(1, color(emidblue)) bar(2, color(ltblue)) ///
ytitle(% of infants given infant formula) ///
title(Infant formula feeding (1-5 months))
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_formula15.gph"

*what compromises EBF and PBF if formula feeding is reportedly uncommon
        *ebf
forvalues i=1/5{
foreach var of varlist bfluidwater bfluidformula bfluidmilk bfluidyoghurt bfluidjuice bfluidbroth bfluidtea
bfluidcolddrink bfluidother food {
tab ebf `var' if ageint==`i' & month_1st==1 & ebf==0, row
tab ageint if ageint==`i'
}
}
egen ebfcomp = rowtotal(bfluidwater bfluidformula bfluidmilk bfluidyoghurt bfluidjuice bfluidbroth
bfluidtea bfluidcolddrink ///
bfluidother food) if ageint>0 & ageint<6 & month_1st==1 & ebf==0
tab ageint ebfcomp if ageint>0 & ageint<6 & ebf!=., row
forval i=1/5{
tab ebfcomp if ageint>0 & ageint<6 & ebf!=. & ageint==`i'
}
        *pbf
forval i=1/5{
foreach var of varlist bfluidformula bfluidmilk bfluidyoghurt bfluidbroth bfluidtea bfluidcolddrink
bfluidother food{
tab pbfnow `var' if ageint==`i' & month_1st==1 & pbfnow==0, row
tab ageint if ageint==`i'
}
}
        //make graphs in Excel sheet "IYCF tables in graphs.xlsx"

*proportion that received each of the following from 6-23 months at each month:
```

```
		*!! Re-code MDD
		{
egen grains1=anymatch(foodgrain foodwhite) if food!=., values(1)
replace grains1=. if food==.
label var grains1 "FG1: Grains, roots, tubers"

egen legume1= anymatch(foodpulse foodnuts) if food!=., values(1)
replace legume1=. if food==.
label var legume1 "FG2: Legumes and nuts"
			*FG3, FG4, FG5 already exist as recoded vars for the mediation analysis
egen dairyprod= anymatch(bfluidmilk bfluidformula bfluidyoghurt fooddairy), values(1)
order dairyprod, after(legume)
label var dairyprod "FG3: Dairy products (milk, yogurt, cheese)"

egen fleshfood = anymatch(foodorgan foodpoultry foodothermeat foodfish), values(1)
order fleshfood, after(dairyprod)
label var fleshfood "FG4: Flesh foods (meat, fish, poultry and liver/organ meats)"

egen eggs= anymatch(foodegg), values(1)
order eggs, after(fleshfood)
label var eggs "FG5: Eggs"

egen vita1 = anymatch(foodorange foodglv foodredfruit) if food!=., values(1)
replace vita1=. if food==.
order vita, after(eggs)
label var vita1 "FG6: Vitamin-A rich fruits and vegetables"

egen otherfv1 = anymatch(foodotherfruitveg) if food!=., values(1)
replace otherfv1=. if food==.
order otherfv, after(vita)
label var otherfv1 "FG7: Other fruits and vegetables"

egen fg7score=rowtotal(grains1 legume1 dairyprod1 fleshfood1 eggs1 vita1 otherfv1) if food!=.
order fg7score, after(otherfv)
gen mdd1=1 if fg7score>=4 & fg7score!=.
replace mdd1=0 if fg7score<4 & fg7score!=.
order mdd, after(fg7score)

label var fg7score "7 food group score"
label var mdd1 "MDD"
label values mdd1 yesno
}
egen fv = anymatch( vita1 otherfv1) if food!=., values (1)
replace fv=. if food==.
label var fv "Fruit and Veg"
label values fv yesno
		* MDD, asf, fv, cont bf/
foreach var of varlist mdd1 asf fv bfnow{
tab ageint `var' if ageint>=6 & ageint<24 & month_1st==1, row
}
*graphs
graph bar (count) if ageint>=6 & ageint<24 & month_1st==1, over(mdd) over(ageint) asyvars stack
percent blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue)) bar(2, color(ltblue)) ytitle(%
of infants who met indicator) title(Minimum dietary diversity (6-23 months))
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_mdd623.gph",
replace

graph bar (count) if ageint>=6 & ageint<24 & month_1st==1, over(asf) over(ageint) asyvars stack
percent blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue)) bar(2, color(ltblue)) ytitle(%
of infants who met indicator) title(Animal source foods (6-23 months))
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_asf623.gph",
replace

graph bar (count) if ageint>=6 & ageint<24 & month_1st==1, over(fv) over(ageint) asyvars stack
percent blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue)) bar(2, color(ltblue)) ytitle(%
of infants who met indicator) title(Fruit and vegetables (6-23 months))
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_fv623.gph"
```

```
graph bar (count) if ageint>=6 & ageint<24 & month_1st==1, over(bfnow) over(ageint) asyvars stack
percent blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue)) bar(2, color(ltblue)) ytitle(%
of infants who met indicator) title(Continued breastfeeding (6-23 months))
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_contbf623.gph"

graph combine "thesis_mdd623.gph" "thesis_asf623.gph" "thesis_fv623.gph" "thesis_contbf623.gph",
ycommon xcommon cols(1)
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_mdd_asf_fv_contbf_623.gph"

        *components of asf
egen asftotal=rowtotal(dairyprod1 fleshfood1 eggs1) if asf!=.
tab asftotal
tab ageint asf if ageint>=6 & ageint<24 & month_1st==1 & asf==1
graph bar (count) if ageint>5 & ageint<24 & month_1st==1 & asftotal>0, over(asftotal) over(ageint)
asyvars percentages stack blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue)) bar(2,
color(ltblue)) bar(3, color(lavender)) title(Types of animal source food consumed (6-23 months))
scheme(s1color)
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_asf_comp623.gph", replace

        *snacks (any, sugary, salty, drinks)
foreach var of varlist snacks sweet salty bev bfluidtea bfluidcolddrink totalsnacks {
tab ageint `var' if ageint>=6 & ageint<24 & month_1st==1, row
}
*graphs
graph bar (count) if ageint>=6 & ageint<24 & month_1st==1, over(snacks) over(ageint) asyvars stack
percent blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue)) bar(2, color(ltblue)) ytitle(%)
title(Sugary or salty snacks consumed (6-23 months))
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_anysnack623.gph"

graph bar (count) if ageint>=6 & ageint<24 & month_1st==1, over(sweet) over(ageint) asyvars stack
percent blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue)) bar(2, color(ltblue)) ytitle(%)
title(Sugary snacks consumed (6-23 months))
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_sweet623.gph"

graph bar (count) if ageint>=6 & ageint<24 & month_1st==1, over(salty) over(ageint) asyvars stack
percent blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue)) bar(2, color(ltblue)) ytitle(%)
title(Salty snacks consumed (6-23 months))
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_salty623.gph"

graph bar (count) if ageint>=6 & ageint<24 & month_1st==1, over(bev) over(ageint) asyvars stack
percent blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue)) bar(2, color(ltblue)) ytitle(%)
title(Tea or sweet drinks consumed (6-23 months))
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_bev623.gph"

graph bar (count) if ageint>=6 & ageint<24 & month_1st==1, over(totalsnacks) over(ageint) asyvars
stack percent blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue)) bar(2, color(ltblue))
bar(3, color(lavender)) bar(4, color(gs8)) ytitle(%) title(Types of snack or drink consumed (6-23
months))
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_totalsnack.gph", replace

graph bar (count) if ageint>=6 & ageint<24 & month_1st==1 & bfluidtea!=99, over(bfluidtea)
over(ageint) asyvars stack percent blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue))
bar(2, color(ltblue)) ytitle(%) title(Tea or coffee consumed (6-23 months))
graph save Graph "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_tea623.gph"

graph bar (count) if ageint>=6 & ageint<24 & month_1st==1 & bfluidcolddrink!=99, over(bfluidcolddrink)
over(ageint) asyvars stack percent blabel(bar, position(center) format(%3.0f)) bar(1, color(emidblue))
bar(2, color(ltblue)) ytitle(%) title(Cold drinks consumed (6-23 months))
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_colddrink623.gph"
```

```
		*combine snack foods
graph combine "thesis_anysnack623.gph"	"thesis_sweet623.gph" "thesis_salty623.gph", ycommon
xcommon cols(1)
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_snack_sweet_salty623.gph"

		*combine drinks
graph combine "thesis_bev623.gph" "thesis_tea623.gph" "thesis_colddrink623.gph", ycommon
xcommon cols(1)
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_bev_tea_colddrink.gph"

*snacking before 6 months.
foreach var of varlist snacks sweet salty bev bfluidtea bfluidcolddrink totalsnacks {
tab ageint `var' if ageint>0 & ageint<6 & month_1st==1, row
}
*
clear

**********Longitudinal data analysis
cd "N:\Documents\IGH\Analysis\Cohort\IYCF"

*Create dataset

		*load parent dataset
use "N:\Documents\IGH\Analysis\Cohort\cohort_anon_all.dta", clear
		*save as another file in relevant folder
save "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\iycf_long24.dta"
clear
		*reload
use "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\iycf_long24.dta", clear

		*truncate
keep if noinfant==1 & id!=945		//61 obs deleted
codebook id //yes. includes 978
keep if age<730 //3980 obs deleted. total obs in dataset=19093


*gen var for each child's first obs in each month
sort id age
egen month_1st = tag(id ageint)

*Tag complete cases for each piece of analysis

		*Breastfeeding 0-5 months
bys id: egen obs_ebf05=seq() if ebf!=. & agemonths<6
tab obs_ebf05 // 966 have at least 1 obs, 1 infant has 9 (!)
forvalues i=0/5{
bys id ageint: egen obs_ebf`i'=seq() if ebf!=. & ageint==`i'
tab obs_ebf`i'
sort id obs_ebf`i'
bys id: replace obs_ebf`i'=obs_ebf`i'[1]
}
tab2 ebf obs_ebf*, first
egen tot_obs_ebf = rowtotal(obs_ebf0-obs_ebf5)
tab tot_obs_ebf if bl==1
		//how many have none, 1, 2... all 6 measurements?
/*
    Total |        978      100.00
*/
egen obs_ebf02 = rowtotal(obs_ebf0-obs_ebf2)
egen obs_ebf35 = rowtotal(obs_ebf3-obs_ebf5)
tab1 obs_ebf02 obs_ebf35 if bl==1
tab obs_ebf02 obs_ebf35 if bl==1
tab2 ebf obs_ebf02 obs_ebf35, first
		*how many have at least one data point in each 3-monthly period?
```

```
codebook id if obs_ebf02!=0 & obs_ebf35!=0 & bl==1
	//778

/*

Only 270/978 (28%) have BF data for all months from 0-5 months.
*/
*split into 2-monthly bands instead of monthly or 3-monthly
egen obs_ebf01 = rowtotal(obs_ebf0-obs_ebf1)
egen obs_ebf23 = rowtotal(obs_ebf2-obs_ebf3)
egen obs_ebf45 = rowtotal(obs_ebf4-obs_ebf5)
tab1 obs_ebf01 obs_ebf23 obs_ebf45 if bl==1
tab2 obs_ebf01 obs_ebf23 obs_ebf45 if bl==1
	*how many have at least one data point in each 2-monthly period?
codebook id if obs_ebf01!=0 & obs_ebf23!=0 & obs_ebf45!=0 & bl==1
	//643

	/*BF data availability:
All 6 = 270
In each of 3 2-monthly age-bands = 643
In both 3-monthly age-bands = 778
	*/

*graph of complete cases
graph bar (count) if ageint>0 & ageint<6 & month_1st==1 & tot_obs_ebf==6, over(ebf) over(ageint)
asyvars stack percent blabel(bar, position(center) format(%3.0f)) ytitle(% of infants exclusively
breastfed) title(Complete cases: exclusive breastfeeding (1-5 months))

graph bar (count) if ageint>0 & ageint<6 & month_1st==1 & tot_obs_ebf==6, over(pbfnow) over(ageint)
asyvars stack percent blabel(bar, position(center) format(%3.0f)) ytitle(% of infants predominantly
breastfed) title(Complete cases: predominant breastfeeding (1-5 months))

foreach var of varlist ebf pbfnow {
tab ageint `var' if ageint>0 & ageint<6 & month_1st==1 & tot_obs_ebf==6, row
}
*life-long definition for those with measurements in all 6 months, using 1st only
		*ebf
by id: egen ebf_life= total(ebf==1) if ageint>0 & ageint<6 & month_1st==1 & tot_obs_ebf==6
tab ebf_life	//range=0/5
forvalues i = 0/5{
codebook id if ebf_life==`i'
}
/*
0=18
1=33
2=35
3=49
4=78
5=57
*/
di 57/270 //21% still ebf at 6 months
di 270-57 //213 failures using only 1st measurement.

		*pbf
by id: egen pbf_life= total(pbfnow==1) if ageint>0 & ageint<6 & month_1st==1 & tot_obs_ebf==6
tab pbf_life	//range=0/5
forvalues i = 0/5{
codebook id if pbf_life==`i'
}
/*
0=17
1=25
2=21
3=37
4=65
5=105
*/
```

```
di 105/270 //39% still pbf at 6 months
di 270-105 //165 failures using 1st measurement only


	* using all data: age at which ebf 'fails', i.e, first age at ebf=0
by id: egen ebf_stop = min(ageint) if ebf==0 & ageint>0 & ageint<6 & tot_obs_ebf==6 & ebf!=.
tab ebf_stop //1 to 5
forvalues i = 1/5{
codebook id if ebf_stop==`i'
}
/*
1=47
2=40
3=33
4=42
5=54
*/
codebook id if ebf_stop!=. & tot_obs_ebf==6 //216 failed
di 270-(47+40+33+42+54)  //54
di 54/270 //20% still ebf at 6 months using data even if >1 obs/month
di (47+40+33+42+54) //216 failures using data even if >1 obs/month


	*age at which pbf fails, i.e., first age at pbf==0
by id: egen pbf_stop = min(ageint) if pbfnow==0 & ageint>0 & ageint<6 & tot_obs_ebf==6 & pbfnow!=.
tab pbf_stop //1 to 5
forvalues i = 1/5{
codebook id if pbf_stop==`i'
}
/*
1=39
2=28
3=23
4=32
5=41
*/
codebook id if pbf_stop!=. & tot_obs_ebf==6 //163 failed
di 270-107 //163 failures using data even if >1 obs/month
di 107/270 //40% still pbf at 6 months using data even if >1 obs/month

	*make KM curves use 1st obs/month only, because it's less chaotic!

use "\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\iycf_long24.dta", clear
gen ebfstop=ebf if age<184 & obs_ebf01!=0 & obs_ebf23!=0 & obs_ebf45!=0 & month_1st==1
recode ebfstop(0=1) (1=0)
tab ebfstop
gen ebfage=ageint if ebfstop!=.
replace ebfstop=. if ebfstop==0
tab ebfage ebfstop
codebook id if ebfstop!=.
tab ageint ebfage
replace ebfstop=. if ebfage==6
replace ebfage=. if ebfage==6

gen pbfstop=pbfnow if age<184 & obs_ebf01!=0 & obs_ebf23!=0 & obs_ebf45!=0 & month_1st==1
recode pbfstop (0=1) (1=0)
tab pbfstop
gen pbfage=ageint if pbfstop!=.
replace pbfage=. if pbfage==0
tab pbfage pbfstop
codebook id if pbfstop!=.
tab ageint pbfage
replace pbfstop=. if pbfage==6
replace pbfage=. if pbfage==6


*1 month age bands (n=270 infants)
```

```
sort id age
bys id: egen ebftime1=seq() if tot_obs_ebf==6 & ageint>0 & ageint<6 & month_1st==1
bys id: egen ebfdays1=seq() if tot_obs_ebf==6 & age<184 & month_1st==1
gen ebftimed1=ageint if ebftime1!=.
gen ebfd1=age if ebfdays1!=.
                                *fail = ebf = 0
stset ebftimed1, id(id) failure(ebf==0) //270 subjects, 212 failures.
sts list
stci

sts graph, risktable xlabel(1(1)7) xtitle(Follow-up visit) xline(7)
sts graph, xlabel(0(15)180) xline (0 30 60 90 183) risktable title("Exclusive breastfeeding")
subtitle("Kaplan-Meier survival estimate")

                        *failure = pbfnow=0
stset ebftime1, id(id) failure(pbfnow==0) //270 subjects, 161 failures.
sts list, by(childsex)
                                *number of failures per period.
tab ageint _d if _d!=.
sts test female
foreach cov of varlist bfprelac female mage25 fage30 med fed water stoiletshare ses2 kids adults
fsmoke msmoke lmup2{
sts test `cov'
}
foreach cov of varlist bfprelac female mage25 fage30 med fed water stoiletshare ses2 kids adults
fsmoke msmoke lmup2{
logistic `cov'
}
sts graph, enter xlabel(1(1)5) xtitle(Follow-up visit)
sts graph, by(childsex) xlabel(1(1)5) xtitle(Follow-up visit)

sts graph, xlabel(0(15)180) xline (0 30 60 90 183) risktable ///
title("Predominant breastfeeding") subtitle("Kaplan-Meier survival estimate")

* 2 month age bands (n=643 infants)
gen tot_obs_ebf2 = 1 if obs_ebf01>0 & obs_ebf23>0 & obs_ebf45>0
codebook id if tot_obs_ebf2==1 //643
sort id ageint
bys id: egen ebftime2 = seq() if tot_obs_ebf2==1 & ageint<6 & month_1st==1
                        *integer months
gen ebftimed2 = ageint if ebftime2!=.
tab ebftimed2 // keep as is.
tab ebftimed2 ebf
                        *cont. months
gen ebfmonths2= agemonths if ebftime2!=.

gen ebfstop=ebf
recode ebfstop(0=1) (1=0)

************************************Discrete time survival models for breastfeeding ***********
*tag 1st Bf observation per child per integer month from 1-5 months
 bys id ageint: gen bf1st = (_n==1)  if ageint>0 & ageint<6 & bfnow!=.

 gen bfn=ageint if bf1st==1
 *check for any discord between duplicates (bf1st = 0 or 1) in same month
 forvalues i = 1/5{
 duplicates tag id bf1st if ageint==`i', gen(_dup`i')
 list id age ageint ebf if _dup`i'==1
 }
 drop _dup*
            *any discordant values within id ageint for duplicates? No, just keep 1
 replace bf1st=. if bf1st==0

 *gen age interval variable to see how many BF obs in each interval
gen bfn=ageint if bf1st==1
/*
   Total |      3,206      100.00
```

```
*/
	*gen seq of measurements and compare with bfn
bys id: egen bfseq =seq() if bfn!=. & bf1st==1
tab bfseq bfn
	//719 have 1st measurement in 1st month
	// 123 of 688 1st measurements are in the 2nd month. there is delayed entry!
	//can't have delayed entry. too complex.
recode bfn (2/3 = 2) (4/5 = 3), gen(bfn2)
replace bfn2 =. if bfn2==2 & bfseq==1 & bfseq!=.
replace bfn2 =. if bfn2==3 & bfseq<3 & bfseq!=.
tab bfn2 bfseq //no 1st measure is after age=1; no 2nd measure is before/after age=2-3
                                // no 3rd measure is before or after 4-5 months

	*tag one in each 2-monthly interval, but the latest one!
bys id bfn2: gen bf1st2=(_n==_N) if bfn2!=.
tab bfn2 bf1st2
by id: egen bfall3=total(bf1st2) if bf1st2==1
codebook id if bfall3==1 //n=123
codebook id if bfall3==2 //n=184
codebook id if bfall3==3 //n=533

gen bft=bfn2 if bfall3==3

gen ebf3=ebf if bfall3==3
recode ebf3 (1=0) (0=1)
gen pbf3=pbfnow if bfall3==3
recode pbf3 (1=0) (0=1)


	*gen var for censored
by id: egen cen=max(ebf3)
replace cen=. if bft==.
by id: egen agefail=min(bft) if ebf3==1 & cen==1
replace agefail=0 if cen==0
gen lastn=bft if agefail==bft
replace lastn=3 if bft==3 & cen==0
tab lastn cen //gives n censored or failed in each interval
/*

*/
	*if last n=1, use only 1st observation, and so on...
by id: egen lastntag=max(lastn)
replace lastntag=. if bft==.
gen bftime=bft
replace bftime=. if lastntag<bftime
rename bftime ebfint
tab ebfint cen
*this works! vars ebf3==fail var, and ebfint==time var! :)


*PBF data arrangement
by id: egen cenp=max(pbf3)
replace cenp=. if bft==.
by id: egen agefailp=min(bft) if pbf3==1 & cenp==1
replace agefailp=0 if cenp==0
gen lastnp=bft if agefailp==bft
replace lastnp=3 if bft==3 & cenp==0
tab lastnp cenp //gives n censored or failed in each interval
	*if last n=1, use only 1st observation, and so on...
by id: egen lastnptag=max(lastnp)
replace lastnptag=. if bft==.
gen pbftime=bft
replace pbftime=. if lastnptag<pbftime
rename pbftime pbfint
tab pbfint cenp

	****ANALYSE DATA
```

```
*calculate discrete-time hazard function using tab
tab ebfint ebf3, row //18%, 23%, 42%
tab pbfint pbf3, row // 16%, 15%, 27%

*calculate discrete-time haz function using logit models
logit ebf3 i.ebfint
predict haz_e, pr
tab ebfint haz_e //18%, 23%, 42%
logit pbf3 i.pbfint
predict haz_p, pr
tab haz_p ebfint //15%, 15%, 27%

	*plot as step functions
label de bfint 1 "1st month" 2 "2nd to 3rd month" 3 "4th to 5th month"
label values ebfint bfint
label values pbfint bfint
twoway(line haz_e ebfint if id==1, connect(stairstep) lcolor(maroon) lwidth(medthick)) ///
(line haz_p pbfint if id==1, connect(stairstep) lcolor(green) lwidth(medthick)), ///
xtitle(Interval) ytitle(Discrete-time hazard) xlabel(#3, valuelabel) ///
title(Conditional probability of discontinuing breastfeeding practice)///
subtitle(given that it has not yet been interrupted)
	//edit graph a bit...
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_ht_ebf_pbf_3.gph", replace

	*Specifying time-dependency of the hazard to use in models.
		*Option 1: step function of time to model time-dependency of the hazard
	*EBF
tab ebfint, gen(t)
logit ebf3 t2 t3
predict haz, pr
sort ebfint
scatter haz ebfint
	*PBF
tab pbfint, gen(tp)
logit pbf3 tp2 tp3 med, or


	*Option 2: fit a quadratic function of age to specify time dependence
	*EBF
gen tsq=ebfint*ebfint
logit ebf3 ebfint tsq
predict hazquad, pr
sort bfint
scatter hazquad bfint
	*PBF
gen tsqp=pbfint*pbfint
logit pbf3 pbfint tsqp med, or

//little change in estimated effect of a covariate between step function and quadratic function
	//

	*adding covariates
			*first fit step-function and quadratic-function models with covariates
			logit ebf3 t2 t3 female med water
			logit ebf3 ebfint tsq female med water
			pred hazquadf, pr
			sort ebfint
			scatter hazquadf ebfint if (female==0 & med==0 & water==0)
	*non-proportional effects: interaction vars for female with t and tsq
		gen t_fem=ebfint*female
		gen tsq_fem=tsq*female
		logit ebf3 ebfint tsq female t_fem tsq_fem med water
		test t_fem tsq_fem
		predict hazint, pr
		sort ebfint
```

```
                scatter hazint ebfint if female==1 & med==0 & water==0, legend(label(1 "F")) ||
        scatter hazint ebfint if female==0 & med==0 & water==0, legend(label(2 "M"))

        *check to see which variables have an influence

*EBF
foreach var of varlist female mage25 fage30 med fed ses2 water stoiletshare adults kids fsmoke
msmoke lmup2{
logit ebf3 ebfint tsq i.`var', or
predict hazq_`var', pr
tab hazq_`var'
drop hazq_`var'
}
logit ebf3 ebfint tsq c.sescore2, or
        //p<0.1: med, water, adults, fsmoke, (sescore2)
*PBF
foreach var of varlist female mage25 fage30 med fed ses2 water stoiletshare adults kids fsmoke
msmoke lmup2{
logit pbf3 pbfint tsqp i.`var', or
predict hazqp_`var', xb
tab hazqp_`var'
drop hazqp_`var'
}
logit pbf3 pbfint tsqp c.sescore2, or
        //p<0.1: med, water, adults, lmup, (sescore2)
        *full model with no reduction in covs
*EBF
logit ebf3 ebfint tsq female mage25 fage30 med fed c.sescore2 water stoiletshare adults kids fsmoke
msmoke lmup2, or
predict hazq, xb


*PBF
logit pbf3 pbfint tsqp female mage25 fage30 med fed c.sescore2 water stoiletshare adults kids fsmoke
msmoke lmup2, or

        *reduced model with only 'sig' at 0.1 covars
*EBF
logit ebf3 ebfint tsq  med  water  adults  fsmoke sescore2
logit, or
        *predicted probabilities of stopping
predict haz_er, pr
                *for med=0 and med=1 holding all other covs=0
scatter haz_er ebfint if med==1 & water==0 & adults==0 & fsmoke==0, legend(label(1 "6th Standard
and above")) || scatter haz_er ebfint if  med==0 & water==0 & adults==0 & fsmoke==0 , legend(label(2
"Below 6th Standard"))
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_ht_pred_ebf_med.gph"

        *for fsmoke=1 and fsmoke=0 holding all other cov=0
scatter haz_er ebfint if fsmoke==1 & water==0 & adults==0 & med==0, legend(label(1 "Yes")) || scatter
haz_er ebfint if  fsmoke==0 & water==0 & adults==0 & med==0 , legend(label(2 "No"))
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_ht_pred_ebf_fsmoke.gph"

        *combine med and fsmoke
graph combine "thesis_ht_pred_ebf_med.gph" "thesis_ht_pred_ebf_fsmoke.gph", ycommon cols(1)
*estimated survival step graph (didn't work.)
{
predict haz_er_lo, xb
gen ln_one_e_haz = ln(1-invlogit(haz_er_lo))
by id (ebfint), sort: gen ln_surv_e = sum(ln_one_e_haz)
gen surv_e = exp(ln_surv_e)
twoway ///
(line surv_e ebfint if med==1 & water==0 & adults==0 & fsmoke==0, connect(stairstep) lcolor(green)
lpatt(dash)) ///
```

```
(line surv_e ebfint if med==0 & water==0 & adults==0 & fsmoke==0, connect(stairstep) lcolor(maroon)
lpatt(dot)), xlab(#3, valuelabel) ///
xtitle(Month) ytitle(Survival)
}
        *ORs
logit ebf3 ebfint tsq  med  water  adults  fsmoke, or


*PBF
logit pbf3 pbfint tsqp med water adults lmup2 sescore2, or
predict haz_pr, pr
        *compare med groups holding other covs constant
scatter haz_pr pbfint if med==1 & water==0 & adults==0 & lmup2==0, legend(label(1 "6th Standard
and above")) || scatter haz_pr pbfint if  med==0 & water==0 & adults==0 & lmup2==0 , legend(label(2
"Below 6th Standard"))
        //edit etc
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_ht_pred_pbf_med.gph"
        *compare lmup groups holding other covs constant
scatter haz_pr pbfint if lmup2==1 & water==0 & adults==0 & med==0, legend(label(1 "Planned")) ///
|| scatter haz_pr pbfint if  lmup2==0 & water==0 & adults==0 & med==0 , legend(label(2 "Not
planned")) ///
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_ht_pred_pbf_lmup.gph"

        *combine med and lmup
graph combine "thesis_ht_pred_pbf_med.gph" "thesis_ht_pred_pbf_lmup.gph", ycommon cols(1)

        *PH Assumption: is effect of med constant over time in the reduced model?
*EBF
        *med
gen t_med=ebfint*med
gen tsq_med=tsq*med
logit ebf3 ebfint tsq med  c.sescore2 water adults fsmoke t_med tsq_med
test t_med tsq_med //PH assumption holds. p=0.8591
logit, or
        *fsmoke
gen t_fsmoke=ebfint*fsmoke
gen tsq_fsmoke=tsq*fsmoke
logit ebf3 ebfint tsq med  c.sescore2 water adults fsmoke t_fsmoke tsq_fsmoke
test t_fsmoke tsq_fsmoke //PH assumption holds. p=0.5860

*PBF
        *med
gen tp_med=pbfint*med
gen tsqp_med=tsqp*med
logit pbf3 pbfint tsqp med c.sescore2 water adults lmup2 tp_med tsqp_med, or
test tp_med tsqp_med //PH assumption holds. p=0.5038

        *lmup
gen tp_lmup=pbfint*lmup2
gen tsqp_lmup=tsqp*lmup2
logit pbf3 pbfint tsqp med c.sescore2 water adults lmup2 tp_lmup tsqp_lmup, or
test tp_lmup tsqp_lmup      //PH assumption holds. p=0.8874

************************Introduction to foods 1 to 9 months*******************

*tag 1 obs per child in each month
bys id ageint: gen food1st = (_n==1)  if ageint>0 & ageint<12 & food!=.
gen fdn=ageint if food1st==1
        *check for discordant food=0/1 in same int month
 forvalues i = 1/5{
 duplicates tag id food1st if ageint==`i', gen(_dup`i')
 list id age ageint food if _dup`i'==1
 }
 //only 1 (id 986, ageint5 (No, then Yes). Ignore
drop _dup*
```

```
replace food1st=. if food1st==0

*how many obs in each int month?
tab fdn
/*

*/
	*gen seq of measurements and compare with bfn
bys id: egen fdseq=seq() if fdn!=. & food1st==1
tab fdseq fdn
	//727 have 1st measurement in 1st month
	// 123 of 918 1st measurements are in the 2nd month. there is delayed entry!
	//can't have delayed entry. too complex.

*create var tagging t1 to t4
	/*
permitted values:
t1-->  fdn=1/3, fdseq=1/3
t2 --> fdn=4/5, fdseq=2/5
t3 --> fdn=6/8, fdseq=3/8
t4 --> fdn=9/11, fdseq=4/11

	*/

recode fdn (1/3 = 1 "1-3months") (4/5 = 2 "4-5 months") (6/8 = 3 "6-8 months") (9/11 = 4 "9-11
months"), gen(fdn2)
tab fdn2 fdseq
replace fdn2 =. if fdn2==1 & fdseq>3 & fdseq!=.
replace fdn2 =. if fdn2==2 & fdseq==1 & fdseq!=.
replace fdn2 =. if fdn2==3 & fdseq<3 & fdseq!=.
replace fdn2 =. if fdn2==3 & fdseq>8 & fdseq!=.
replace fdn2 =. if fdn2==4 & fdseq<4 & fdseq!=.

tab fdseq fdn2 //all values within criteria


 **********ALT start-

 ******** By using 6 time bands instead of 4 (6,7,8 as separate ones)
{
recode fdn (1/3 = 1 "1-3months") (4/5 = 2 "4-5 months") (6 = 3 "6 months") (7 = 4 "7 months") (8=5 "8
months") (9/11 = 6 "9-11 months"), gen(fdn3)
tab fdn3 fdseq
replace fdn3 =. if fdn3==1 & fdseq>3 & fdseq!=.
replace fdn3 =. if fdn3==2 & fdseq==1 & fdseq!=.
replace fdn3 =. if fdn3==3 & fdseq<3 & fdseq!=.
replace fdn3 =. if fdn3==3 & fdseq>6 & fdseq!=.
replace fdn3 =. if fdn3==4 & fdseq<4 & fdseq!=.
replace fdn3 =. if fdn3==4 & fdseq>7 & fdseq!=.
replace fdn3 =. if fdn3==5 & fdseq<5 & fdseq!=.
replace fdn3 =. if fdn3==5 & fdseq>8 & fdseq!=.
replace fdn3 =. if fdn3==6 & fdseq<6 & fdseq!=.

tab fdseq fdn3

forval i=1/6{
codebook id if fdn3==`i'
}
//n= 884, 713, 545, 539, 483, 607

*tag censoring or failure within each timeband
bys id fdn3: egen cenfd6=max(food) if fdn3!=.
egen tagfd6 = tag(id fdn3)
replace tagfd6=. if tagfd6==0
gen foodfail6=tagfd6
replace foodfail6=cenfd6 if foodfail6!=.
```

```
		*how many have 1, 2, 3, ...all 6
by id: egen fd6=total(tagfd6) if tagfd6==1
forval i=1/6{
codebook id if fd6==`i'
}

/*
only 1 = 143
only 2 = 80
only 3 = 59
only 4 = 96
only 5 = 207
all 6 = 312

**/
gen fdt6=fdn3 if fd6==6

by id: egen ageintro6=min(fdt6) if foodfail6==1 & cenfd6==1
by id: egen anyfail6=max(foodfail6) if fd6==6
gen lastfdn6=fdt6 if ageintro6==fdt6
replace lastfdn6=6 if fdt6==6 & anyfail6==0
tab lastfdn6 anyfail6

by id: egen lastfdn6tag=max(lastfdn6)
replace lastfdn6tag=. if fdt6==.
gen fdtime6=fdt6
replace fdtime6=. if lastfdn6tag<fdtime6
tab fdtime6 foodfail6

*haz function
logit foodfail6 i.fdtime6
predict haz_f6, pr
tab fdtime6 haz_f6 //2%, 17%, 43%, 62%, 74%, (100% - omitted because all fail)

twoway(line haz_f6 fdtime6 if id==96, connect(stairstep) lcolor(green) lwidth(thick)), ///
xtitle(Age) ytitle(Discrete-time hazard) ///
title("Conditional probability of introduction to solid, semi-solid and soft foods") ///
subtitle((given that it has not yet occured))
graph save Graph
"\\ad.ucl.ac.uk\homea\x\Documents\IGH\Analysis\Cohort\IYCF\thesis_ht_introfood_t6.gph"


*specify time-dependency of model with covariates
			*Option 1: Step function
tab fdtime6 if fdtime6<6, gen(t6f)
logit foodfail6 t6f2 t6f3 t6f4 t6f5 med female if fdtime6<6, or

		*Option 2: fit a quadratic function of age to specify time dependence
gen ft6sq=fdtime6*fdtime6
logit foodfail6 fdtime6 ft6sq med female if fdtime6<6, or
		*very similiar co-effecient magnitudes. Use quadratic function

*check for crude influence of covariates

foreach var of varlist female mage25 fage30 med fed ses2 water stoiletshare adults kids fsmoke
msmoke lmup2{
logit foodfail6 fdtime6 ft6sq i.`var' if fdtime6<6, or
}
logit foodfail6 fdtime6 ft6sq c.sescore2 if fdtime6<6, or

logit foodfail6 fdtime6 ft6sq mage25 med fed adults kids c.sescore2 if fdtime6<6, or

logit foodfail6 fdtime6 ft6sq female mage25 fage30 med fed i.ses2 water stoiletshare adults kids
fsmoke msmoke lmup2 if fdtime6<6, or

//relationship between intro and kids holds even when finer timebands are used.
		//though ses is more apparent in this. maybe since small n due to bias in response rates?
```

```
}
************************************ ALT end-

            *how many unique id in each time band?
forval i=1/4{
codebook id if fdn2==`i'
}
//n=884, 713, 698, 652

*tag censoring or failure within each timeband
bys id fdn2: egen cenfd=max(food) if fdn2!=.
egen fd1 = tag(id fdn2)
replace fd1=. if fd1==0
gen foodfail=fd1
replace foodfail=cenfd if foodfail!=.

            *how many have 1, 2, 3, all 3
by id: egen fd4=total(fd1) if fd1==1
forval i=1/4{
codebook id if fd4==`i'
}
/*
only 1 msr=129
only 2 msr=78
only 3 msr=134
all 4 = 565 */
gen fdt=fdn2 if fd4==4
*gen var for censored
by id: egen ageintro=min(fdt) if foodfail==1 & cenfd==1
by id: egen anyfail=max(foodfail) if fd4==4
gen lastfdn=fdt if ageintro==fdt
replace lastfdn=4 if fdt==4 & anyfail==0
tab lastfdn anyfail //gives n censored or failed in each interval
/*
           |        anyfail
   lastfdn |         0          1 |     Total
-----------+----------------------+----------
         1 |         0         11 |        11
         2 |         0        108 |       108
         3 |         0        400 |       400
         4 |         2         44 |        46
-----------+----------------------+----------
     Total |         2        563 |       565
*/
            *if last n=1, use only 1st observation, and so on...
by id: egen lastfdntag=max(lastfdn)
replace lastfdntag=. if fdt==.
gen fdtime=fdt
replace fdtime=. if lastfdntag<fdtime
tab fdtime foodfail

/*

           |       foodfail
    fdtime |         0          1 |     Total
-----------+----------------------+----------
         1 |       554         11 |       565
         2 |       450        108 |       558
         3 |        50        401 |       451
         4 |         5         44 |        49
-----------+----------------------+----------
     Total |     1,059        564 |     1,623
*/
*this works! vars foodfail==fail var, and foodtime==time var! :)

save, replace
```

```
*calculate discrete-time hazard function using tab
tab fdtime foodfail, row //2%, 19%, 89% 90%
*calculate discrete-time haz function using logit models
logit foodfail i.fdtime
predict haz_f, pr
tab fdtime haz_f //2%, 19%, 89%, 90%

*plot as step function
label define fdtime 1 "1-3 mo" 2 "4-5 mo" 3 "6-8 mo" 4 "9-11 mo"
label values fdtime fdtime
        *find an id with all 4 intervals
tab id if lastfdn==4 //use id==12
twoway(line haz_f fdtime if id==12, connect(stairstep) lcolor(maroon) lwidth(thick)), ///
xtitle(Age) ytitle(Discrete-time hazard)///
title(Conditional probability of introduction to solid, semi-solid and soft foods) ///
subtitle((given that it has not yet occured))
        //re-jig: extend line for 9-11 months, change margins to Vlarge

*specify time-dependency of model with covariates
                *Option 1: Step function
tab fdtime, gen(tf)
logit foodfail tf2 tf3 tf4 med female

        *Option 2: fit a quadratic function of age to specify time dependence
gen ftsq=fdtime*fdtime
logit foodfail fdtime ftsq med female
        *very similiar co-effecient magnitudes. Use quadratic function

*check for crude influence of covariates
        *restrict to t1 to t3
foreach var of varlist female mage25 fage30 med fed ses2 water stoiletshare adults kids fsmoke
msmoke lmup2{
logit foodfail fdtime ftsq i.`var' if fdtime<4, or
}
logit foodfail fdtime ftsq c.sescore2 if fdtime<4, or

        //p<0.1: mage, med, sescore, kids, fsmoke

        *full model
logit foodfail fdtime ftsq female mage25 fage30 med fed i.ses2 water stoiletshare adults kids fsmoke
msmoke lmup2, or
                *restricted to t1-t3
logit foodfail fdtime ftsq female mage25 fage30 med fed c.sescore2 water stoiletshare adults kids
fsmoke msmoke lmup2 if fdtime<4, or
predict ff, pr
codebook id if ff!=. //550
*reduced model
logit foodfail fdtime ftsq  mage25 med i.ses2 kids fsmoke, or
                *restricted to t1-t3
logit foodfail fdtime ftsq  mage25 med c.sescore2 kids fsmoke if fdtime<4, or
predict ffr, pr
codebook id if ffr!=. //551
*only for t3
logit foodfail female mage25 fage30 med fed c.sescore2 water stoiletshare adults kids fsmoke msmoke
lmup2 if fdtime==3, or

*non-proportional hazards assumption for kids
gen tf_kids=fdtime*kids
gen tsqf_kids=ftsq*kids
logit foodfail fdtime ftsq mage25 med ses2 kids fsmoke tf_kids tsqf_kids if fdtime<4, or
test tf_kids tsqf_kids //PH assumption holds. p=0.6638

*non-proportional hazards assumption for med
gen tf_med=fdtime*med
gen tsqf_med=ftsq*med
```

```
logit foodfail fdtime ftsq mage25 med ses2 kids fsmoke tf_med tsqf_med if fdtime<4, or
test tf_med tsqf_med //PH assumption holds p=0.4003

**********Complementary feeding 6-23 months (stratified by age)**********

*Checking data availability
	*how many obs per participant with T=3 [6-11, 12-17, 18-23]
by id: egen numt1=count(mdd1) if ageint>5 & ageint<12
by id: egen numt2=count(mdd1) if ageint>11 & ageint<18
by id: egen numt3=count(mdd1) if ageint>17 & ageint<24
egen onet1 = tag(id) if numt1!=.
egen onet2 = tag(id) if numt2!=.
egen onet3 = tag(id) if numt3!=.
tab numt1 if onet1==1
tab numt2 if onet2==1
tab numt3 if onet3==1
drop numt1 numt2 numt3
drop onet1 onet2 onet3
	//n= 867, 777, 714

	*how many obs per participant with T=6 [6-8, 9-11, 12-14, 15-17, 18-20, 21-23]
		*for mdd, asf, and >1 snack
recode totalsnacks (0/1=0 "<2 snacks") (2/3=1 "2+ snacks"), gen(snk)
label var snk Snacks
foreach var of varlist mdd1 asf snk{
by id: egen n`var't1=count(`var') if ageint>5 & ageint<9
by id: egen n`var't2=count(`var') if ageint>8 & ageint<12
by id: egen n`var't3=count(`var') if ageint>11 & ageint<15
by id: egen n`var't4=count(`var') if ageint>14 & ageint<18
by id: egen n`var't5=count(`var') if ageint>17 & ageint<21
by id: egen n`var't6=count(`var') if ageint>20 & ageint<24
}
forval i=1/6{
foreach var of varlist mdd1 asf snk {
egen tag`var'_`i' = tag(id) if n`var't`i'!=.
tab n`var't`i' if tag`var'_`i'==1
}
}
		//MDD, ASF, SNACK n = 865, 798, 768, 741,713,680

	*compute scores and proportion in each interval from t1 to t6
forval i=1/6{
foreach var of varlist mdd1 asf snk{
by id: egen `var'tot_t`i' = total(`var') if n`var't`i'!=.
gen `var'prop_t`i'= `var'tot_t`i'/n`var't`i' if n`var't`i'!=.
sum `var'prop_t`i' if tag`var'_`i'==1
}
}
forval i=1/6{
foreach var of varlist mdd1 asf snk{
tab1 `var'prop_t`i' if tag`var'_`i'==1
}
}
	//very skewed distributions . but enough 1s or 0s in each age to make binary

	*make MDD, ASF, and SNACK scores binary

*MDD: 0=Never 1=Ever
label define neverever 0 "Never" 1 "Ever"
forval i=1/6{
gen mdd_t`i'=0 if mdd1prop_t`i'==0
replace mdd_t`i'=1 if mdd1prop_t`i'>0 & mdd1prop_t`i'!=.
label values mdd_t`i' neverever
tab mdd_t`i' if tagmdd1_`i'==1
}
*ASF: 0= Never/Sometimes 1= Always
label define always 0 "Never/Sometimes" 1 "Always"
```

```
forval i=1/6{
gen asf_t`i'=1 if asfprop_t`i'==1
replace asf_t`i'=0 if asfprop_t`i'<1 & asfprop_t`i'!=.
label values asf_t`i' always
tab asf_t`i' if tagasf_`i'==1
}
*SNACK: 0=Never 1=Ever
forval i=1/6{
gen snk_t`i'=0 if snkprop_t`i'==0
replace snk_t`i'=1 if snkprop_t`i'>0 & snkprop_t`i'!=.
label values snk_t`i' neverever
tab snk_t`i' if tagsnk_`i'==1
}

*make into long format variables

                        *Time variables
foreach var of varlist mdd asf snk{
gen t_`var'=1 if `var'_t1!=.
forval i=2/6{
replace t_`var'=`i' if `var'_t`i'!=.
}
}
//all three time vars are the same... but keep separate anyway

                        *Value variables
foreach var of varlist mdd asf snk{
gen ar_`var'=.
forval i=1/6{
replace ar_`var'=`var'_t`i' if `var'_t`i'!=.
}
}
label values ar_mdd neverever
label values ar_asf always
label values ar_snk neverever

*drop all the pointless vars
drop nmdd1t1- snk_t6

*are there any gaps, i.e., do any children skip timebands entirely?
tab t_mdd //unequal numbers, so yes! total n=11,462

*gen lagged variable for cf in t-1 for t2 to t6, but only when t-(t-1)=1

*tag 1 obs per child per timeband
egen tagcf=tag(id t_snk)
replace tagcf=. if tagcf==0

        *tag obs with no gap between consecutive periods
sort id tagcf t_snk
by id: gen lag1=1 if t_snk>t_snk[_n-1] & tagcf==1

        *create vars for lag_var values for mdd, asf, snack:
foreach var of varlist ar_mdd ar_asf ar_snk{
gen l_`var'=`var'[_n-1] if lag1==1
}
label values l_ar_mdd neverever
label values l_ar_asf always
label values l_ar_snk neverever

save, replace

*tagcf= tag relevant obs.

*tabulate frequencies within each timeband
foreach var of varlist mdd asf snk{
tab t_`var' ar_`var' if tagcf==1
```

```
}
**************AUTOREGRESSIVE MODELS*****************************

***STEP 1: Models without the initial condition
	/*MODEL 1: (Y at t1 is not included!)
	*Fit a random-effects xtlogit model with lagged outcome as a covariate
		*Is there evidence of
			(1)state dependence (l_`y' co-efficient)
			(2) unobserved heterogeneity (LRT of rho=0; equivalent to
sigma_u2=0)?
		*Test with no covariates, crude bi-variable, full cov set, reduced cov set*/
xtset id
	*simple
xtlogit ar_mdd l_ar_mdd t_mdd, or
xtlogit ar_asf l_ar_asf t_asf, or
xtlogit ar_snk l_ar_snk t_snk, or

		*crude associations
foreach y of varlist mdd asf snk{
foreach var of varlist  female mage25 fage30 med fed ses2 water stoiletshare adults kids fsmoke
msmoke lmup2{
xtlogit ar_`y' l_ar_`y' t_`y' i.`var', or
}
xtlogit ar_`y' l_ar_`y' t_`y' c.sescore2, or
}
	*Full models (SES both ways).
foreach y of varlist mdd asf snk{
xtlogit ar_`y' l_ar_`y' t_`y' female mage25 fage30 med fed i.ses2 water stoiletshare adults kids fsmoke
msmoke lmup2, or
xtlogit ar_`y' l_ar_`y' t_`y' female mage25 fage30 med fed c.sescore2 water stoiletshare adults kids
fsmoke msmoke lmup2, or
}
foreach y of varlist mdd asf snk{
xtlogit ar_`y' l_ar_`y' t_`y' female mage25 fage30 med fed i.ses2 water stoiletshare adults kids fsmoke
msmoke lmup2
}

	//Evidence of state dependence (sig p-value for l_ar_y): YES for all
	//Evidence of unobserved heterogeneity (rho test p-values): YES for all

	*reduced models (different covs for each IYCF)
xtlogit ar_mdd l_ar_mdd t_mdd fsmoke med fed i.ses2 adults lmup2
xtlogit, or
xtlogit ar_asf l_ar_asf t_asf med fed i.ses2 water stoiletshare adults fsmoke lmup2
xtlogit, or
xtlogit ar_snk l_ar_snk t_snk med fed stoiletshare kids
xtlogit, or

	//state dependence (sig l_ar_y): YES for all
	//unobserved heterogeneity (rho test p-values): YES for all

***STEP 2: Models with the initital condition
	*I will use the Stata command gsem

	*MODEL 2: Joint model with random effect loading =1 and MODEL 1
*dummy var for t1
by id: gen it1=(_n==1) if tagcf==1
gen itg1=1-it1
*gen 2 y vars: y=. if t>1 and y=. if t==1
foreach y of varlist mdd asf snk{
gen ar_`y'_t1= ar_`y' if tagcf==1
replace ar_`y'_t1=. if		it1==0
gen ar_`y'_tg1=ar_`y' if tagcf==1
replace ar_`y'_tg1=. if itg1==0
}
//should correspond to the values of var in that period.
	//yes. works ok!
```

```
*reduced model for MDD
gsem(ar_mdd_t1 <- t_mdd fsmoke med fed i.ses2 adults lmup2, logit) ///
(ar_mdd_tg1 <- l_ar_mdd t_mdd fsmoke med fed i.ses2 adults lmup2, logit) ///
(U[id] -> ar_mdd_t1@1) ///
(U[id] -> ar_mdd_tg1@1)


est store mdd2
estat eform ar_mdd_tg1


*reduced model for ASF
gsem(ar_asf_t1 <- t_asf med fed i.ses2 water stoiletshare adults fsmoke lmup2, logit) ///
(ar_asf_tg1 <- l_ar_asf t_asf med fed i.ses2 water stoiletshare adults fsmoke lmup2, logit) ///
(U[id] -> ar_asf_t1@1) ///
(U[id] -> ar_asf_tg1@1)

est store asf2
estat eform ar_asf_tg1


*reduced model for snacks
gsem(ar_snk_t1 <- t_snk med fed stoiletshare kids, logit) ///
(ar_snk_tg1 <- l_ar_snk t_snk med fed stoiletshare kids, logit) ///
(U[id] -> ar_snk_t1@1) ///
(U[id] -> ar_snk_tg1@1)

est store snk2
estat eform ar_snk_tg1

//fits for all 3 IYCF outcomes! phew!

        *MODEL 3: Joint model with random effect loading unconstrained and MODEL 1

/*can use with gllamm, but also possible with gsem. So I will use gsem
differs from Model 2 because we replace the ar_mdd_t1@1 with ...t1@a
indicating that we want to estimate the re for occasion 1 rather than constrain to 1 as we did for Model
2.
*/

*reduced model for MDD
gsem(ar_mdd_t1 <- t_mdd fsmoke med fed i.ses2 adults lmup2, logit) ///
(ar_mdd_tg1 <- l_ar_mdd t_mdd fsmoke med fed i.ses2 adults lmup2, logit) ///
(U[id] -> ar_mdd_t1@a) ///
(U[id] -> ar_mdd_tg1@1)

est store mdd3
estat eform ar_mdd_tg1

lrtest mdd2 mdd3
//p=0.6797

*reduced model for ASF
gsem(ar_asf_t1 <- t_asf med fed i.ses2 water stoiletshare adults fsmoke lmup2, logit) ///
(ar_asf_tg1 <- l_ar_asf t_asf med fed i.ses2 water stoiletshare adults fsmoke lmup2, logit) ///
(U[id] -> ar_asf_t1@a) ///
(U[id] -> ar_asf_tg1@1)

est store asf3
estat eform ar_asf_tg1

lrtest asf2 asf3
//p=0.8591

*reduced model for snacks
gsem(ar_snk_t1 <- t_snk med fed stoiletshare kids, logit) ///
```

```
(ar_snk_tg1 <- l_ar_snk t_snk med fed stoiletshare kids, logit) ///
(U[id] -> ar_snk_t1@a) ///
(U[id] -> ar_snk_tg1@1)

est store snk3
estat eform ar_snk_tg1

lrtest snk2 snk3
//p=0.2203

**************************

*All covariates

*MDD
        *Model 2
gsem(ar_mdd_t1 <- t_mdd female mage25 fage30 med fed i.ses2 water stoiletshare adults kids
fsmoke msmoke lmup2, logit) ///
(ar_mdd_tg1 <- l_ar_mdd t_mdd female mage25 fage30 med fed i.ses2 water stoiletshare adults kids
fsmoke msmoke lmup2, logit) ///
(U[id] -> ar_mdd_t1@1) ///
(U[id] -> ar_mdd_tg1@1)
est store mdd2f
estat eform ar_mdd_tg1

        *Model 3
gsem(ar_mdd_t1 <- t_mdd female mage25 fage30 med fed i.ses2 water stoiletshare adults kids
fsmoke msmoke lmup2, logit) ///
(ar_mdd_tg1 <- l_ar_mdd t_mdd female mage25 fage30 med fed i.ses2 water stoiletshare adults kids
fsmoke msmoke lmup2, logit) ///
(U[id] -> ar_mdd_t1@a) ///
(U[id] -> ar_mdd_tg1@1)
est store mdd3f
estat eform ar_mdd_tg1

lrtest mdd2f mdd3f
//p=0.6696
lrtest mdd3f mdd2f

*ASF
        *Model 2
gsem(ar_asf_t1 <- t_asf female mage25 fage30 med fed i.ses2 water stoiletshare adults kids fsmoke
msmoke lmup2, logit) ///
(ar_asf_tg1 <- l_ar_asf t_asf female mage25 fage30 med fed i.ses2 water stoiletshare adults kids
fsmoke msmoke lmup2, logit) ///
(U[id] -> ar_asf_t1@1) ///
(U[id] -> ar_asf_tg1@1)
est store asf2f
estat eform ar_asf_tg1

        *Model 3
gsem(ar_asf_t1 <- t_asf female mage25 fage30 med fed i.ses2 water stoiletshare adults kids fsmoke
msmoke lmup2, logit) ///
(ar_asf_tg1 <- l_ar_asf t_asf female mage25 fage30 med fed i.ses2 water stoiletshare adults kids
fsmoke msmoke lmup2, logit) ///
(U[id] -> ar_asf_t1@a) ///
(U[id] -> ar_asf_tg1@1)
est store asf3f
estat eform ar_asf_tg1


lrtest asf2f asf3f
//p=0.7721

*Snacks
        *Model 2
```

```
gsem(ar_snk_t1 <- t_snk female mage25 fage30 med fed i.ses2 water stoiletshare adults kids fsmoke
msmoke lmup2, logit) ///
(ar_snk_tg1 <- l_ar_snk t_snk female mage25 fage30 med fed i.ses2 water stoiletshare adults kids
fsmoke msmoke lmup2, logit) ///
(U[id] -> ar_snk_t1@1) ///
(U[id] -> ar_snk_tg1@1)

est store snk2f
estat eform ar_snk_tg1

        *Model 3
gsem(ar_snk_t1 <- t_snk female mage25 fage30 med fed i.ses2 water stoiletshare adults kids fsmoke
msmoke lmup2, logit) ///
(ar_snk_tg1 <- l_ar_snk t_snk female mage25 fage30 med fed i.ses2 water stoiletshare adults kids
fsmoke msmoke lmup2, logit) ///
(U[id] -> ar_snk_t1@a) ///
(U[id] -> ar_snk_tg1@1)

est store snk3f
estat eform ar_snk_tg1

lrtest snk2f snk3f
//p=0.3778

*****************************CF for thesis

********** Compare Models 1, 2, and 3 in unadjusted analysis, get ORs

        *Model 1 for all three indicators

xtlogit ar_mdd l_ar_mdd t_mdd
xtlogit, or
quadchk
xtlogit ar_asf l_ar_asf t_asf
xtlogit, or
quadchk
xtlogit ar_snk l_ar_snk t_snk
xtlogit, or
quadchk
                        //remember to square the sigma_u to report sigma_u^2 (bet ind var)
                *calculate VPC or proportion of variance attr. to bet-child diff
                        *Option 1: use the rho estimate, express as %
                        *Option 2: sigma_u^2/(sigma_u^2 +3.29), express as %,
                                *where 3.29 is the constant sigma_e parameter for a logit model
(LEMMA C7.2.1)
                                *converting sigma_u (which is the SD) and its SE (reported by
xtlogit)
                                        *to variance and SE (reported by gsem, so that bet-child
variance is comparable
                                        *--> sigma_u^2=variance
                                        *--> sigma_u/sqrt(n groups) = SE of variance

*convert sigma_u, se to sigma_u^2 and se
        *mdd
di  .9769681^2
di  .9769681/sqrt(711)
        *asf
di 1.014856^2
di 1.014856/sqrt(711)
        *snacks
di .9627165^2
di .9627165/sqrt(711)

*OR scale: exp sigma_u and its 95%CI
        *mdd
di exp(.9769681)
di exp(.7682809)
```

```
di exp(1.242341)

        *asf
di exp( 1.014856)
di exp(.7692182)
di exp(1.338935)

        *snacks
di exp(.9627165)
di exp(.7377263)
di exp(1.256324)


                        //Model 1 does not produce a coefficient of individual random effect
xtlogit ar_mdd l_ar_mdd t_mdd
estimates store mdd1
xtlogit ar_asf l_ar_asf t_asf
estimates store asf1
xtlogit ar_snk l_ar_snk t_snk
estimates store snk1
estimates table mdd1 asf1 snk1

        *Models 2 and 3 for all three indicators
                *note that var(u[id]) is sigma_u^2 (between-individual variance)
                *co-eff of lagged variable for `y't>1 output is lagged response
                *for Model 2 the co-eff of ind random is constrained to 1
                *U[id] of `y't1 output is the individual random effect for Model 3
*MDD
        *Model 2
gsem(ar_mdd_t1 <- t_mdd, logit) ///
(ar_mdd_tg1 <- l_ar_mdd t_mdd,  logit) ///
(U[id] -> ar_mdd_t1@1) ///
(U[id] -> ar_mdd_tg1@1)
est store mdd2f
estat eform ar_mdd_tg1

di exp(1.304672)
di exp(.9024205)
di exp(1.886226)


        *Model 3
gsem(ar_mdd_t1 <- t_mdd, logit) ///
(ar_mdd_tg1 <- l_ar_mdd t_mdd, logit) ///
(U[id] -> ar_mdd_t1@a) ///
(U[id] -> ar_mdd_tg1@1)
est store mdd3f
estat eform ar_mdd_tg1

di exp(1.337936)
di exp(.8917963)
di exp(2.007268)


lrtest mdd2f mdd3f
//p=0.7821

*OR, CI for occasion-level random effect
di exp(.9386846)
di exp(.5161216)
di exp(1.361248)

*ASF
        *Model 2
gsem(ar_asf_t1 <- t_asf, logit) ///
(ar_asf_tg1 <- l_ar_asf t_asf, logit) ///
(U[id] -> ar_asf_t1@1) ///
```

```
(U[id] -> ar_asf_tg1@1)
est store asf2f
estat eform ar_asf_tg1

di exp(2.093061)
di exp(1.524614)
di exp(2.873452)


        *Model 3
gsem(ar_asf_t1 <- t_asf, logit) ///
(ar_asf_tg1 <- l_ar_asf t_asf, logit) ///
(U[id] -> ar_asf_t1@a) ///
(U[id] -> ar_asf_tg1@1)
est store asf3f
estat eform ar_asf_tg1

di exp(2.152779)
di exp(1.480673)
di exp(3.129965)

lrtest asf2f asf3f
//p=0.7880

*OR, CI for occasion-level random effect
di exp(.9552352)
di exp(.6362208)
di exp(1.27425)

*Snacks
        *Model 2
gsem(ar_snk_t1 <- t_snk,  logit) ///
(ar_snk_tg1 <- l_ar_snk t_snk, logit) ///
(U[id] -> ar_snk_t1@1) ///
(U[id] -> ar_snk_tg1@1)

est store snk2f
estat eform ar_snk_tg1

di exp(1.101784)
di exp(.7252361)
di exp(1.673838)

        *Model 3
gsem(ar_snk_t1 <- t_snk, logit) ///
(ar_snk_tg1 <- l_ar_snk t_snk, logit) ///
(U[id] -> ar_snk_t1@a) ///
(U[id] -> ar_snk_tg1@1)

est store snk3f
estat eform ar_snk_tg1

di exp(1.33016)
di exp(.8442301)
di exp(2.095787)

lrtest snk2f snk3f
//p=0.1422

*OR, CI for occasion-level random effect
di exp(.6880709)
di exp(.3391894)
di exp(1.036952)


******** Crude associations of covariates with each indicator
*use Model 2
```

```
*MDD
foreach var of varlist female mage25 fage30 med fed ses2 water stoiletshare adults kids fsmoke
msmoke lmup2{
gsem(ar_mdd_t1 <- t_mdd i.`var', logit) ///
(ar_mdd_tg1 <- l_ar_mdd t_mdd i.`var',  logit) ///
(U[id] -> ar_mdd_t1@1) ///
(U[id] -> ar_mdd_tg1@1)
estat eform ar_mdd_tg1
}
gsem(ar_mdd_t1 <- t_mdd sescore2, logit) ///
(ar_mdd_tg1 <- l_ar_mdd t_mdd sescore2,  logit) ///
(U[id] -> ar_mdd_t1@1) ///
(U[id] -> ar_mdd_tg1@1)
estat eform ar_mdd_tg1
//sig at p<0.1: med, sescore, adults, fsmoke, lmup

*ASF:
foreach var of varlist female mage25 fage30 med fed ses2 water stoiletshare adults kids fsmoke
msmoke lmup2{
gsem(ar_asf_t1 <- t_asf i.`var', logit) ///
(ar_asf_tg1 <- l_ar_asf t_asf i.`var', logit) ///
(U[id] -> ar_asf_t1@1) ///
(U[id] -> ar_asf_tg1@1)
estat eform ar_asf_tg1
}
gsem(ar_asf_t1 <- t_asf sescore2, logit) ///
(ar_asf_tg1 <- l_ar_asf t_asf sescore2, logit) ///
(U[id] -> ar_asf_t1@1) ///
(U[id] -> ar_asf_tg1@1)
estat eform ar_asf_tg1
//sig at p<0.1 med+, fed+, sescore+, water+, toilet-, adults+, fsmoke-, lmup-

*SNacks
foreach var of varlist female mage25 fage30 med fed ses2 water stoiletshare adults kids fsmoke
msmoke lmup2{
gsem(ar_snk_t1 <- t_snk i.`var',  logit) ///
(ar_snk_tg1 <- l_ar_snk t_snk i.`var', logit) ///
(U[id] -> ar_snk_t1@1) ///
(U[id] -> ar_snk_tg1@1)
estat eform ar_snk_tg1
}
gsem(ar_snk_t1 <- t_snk sescore2,  logit) ///
(ar_snk_tg1 <- l_ar_snk t_snk sescore2, logit) ///
(U[id] -> ar_snk_t1@1) ///
(U[id] -> ar_snk_tg1@1)
estat eform ar_snk_tg1
//sig at p<0.1: med-, fed-, toilet-, kids+,


*FULL MODELS, use sescore


*MDD
gsem(ar_mdd_t1 <- t_mdd female mage25 fage30 med fed sescore2 water stoiletshare adults kids
fsmoke msmoke lmup2, logit) ///
(ar_mdd_tg1 <- l_ar_mdd t_mdd female mage25 fage30 med fed sescore2 water stoiletshare adults
kids fsmoke msmoke lmup2, logit) ///
(U[id] -> ar_mdd_t1@1) ///
(U[id] -> ar_mdd_tg1@1)
estat eform ar_mdd_tg1

di exp( 1.117497)
di exp(.7471904)
di exp(1.671328)
```

```
gsem(ar_mdd_t1 <- t_mdd female mage25 fage30 med fed sescore2 water stoiletshare adults kids
fsmoke msmoke, logit) ///
(ar_mdd_tg1 <- l_ar_mdd t_mdd female mage25 fage30 med fed sescore2 water stoiletshare adults
kids fsmoke msmoke, logit) ///
(U[id] -> ar_mdd_t1@1) ///
(U[id] -> ar_mdd_tg1@1)
estat eform ar_mdd_tg1

*ASF
gsem(ar_asf_t1 <- t_asf female mage25 fage30 med fed sescore2 water stoiletshare adults kids
fsmoke msmoke lmup2, logit) ///
(ar_asf_tg1 <- l_ar_asf t_asf female mage25 fage30 med fed sescore2 water stoiletshare adults kids
fsmoke msmoke lmup2, logit) ///
(U[id] -> ar_asf_t1@1) ///
(U[id] -> ar_asf_tg1@1)
estat eform ar_asf_tg1

di exp( 1.711128 )
di exp( 1.214165)
di exp(2.4115)


*snacks
gsem(ar_snk_t1 <- t_snk female mage25 fage30 med fed sescore2 water stoiletshare adults kids
fsmoke msmoke lmup2, logit) ///
(ar_snk_tg1 <- l_ar_snk t_snk female mage25 fage30 med fed sescore2 water stoiletshare adults kids
fsmoke msmoke lmup2, logit) ///
(U[id] -> ar_snk_t1@1) ///
(U[id] -> ar_snk_tg1@1)
estat eform ar_snk_tg1

di exp(.9801896)
di exp(.624391)
di exp( 1.538734)



*REDUCED MODELS
*MDD
gsem(ar_mdd_t1 <-  med  sescore2  adults  fsmoke lmup2, logit) ///
(ar_mdd_tg1 <- l_ar_mdd t_mdd med  sescore2  adults  fsmoke lmup2, logit) ///
(U[id] -> ar_mdd_t1@1) ///
(U[id] -> ar_mdd_tg1@1)
estat eform ar_mdd_tg1

di exp( 1.192802)
di exp(.8059639)
di exp( 1.76531)


*ASF
gsem(ar_asf_t1 <- t_asf  med fed sescore2 water stoiletshare adults fsmoke lmup2, logit) ///
(ar_asf_tg1 <- l_ar_asf t_asf med fed sescore2 water stoiletshare adults fsmoke lmup2, logit) ///
(U[id] -> ar_asf_t1@1) ///
(U[id] -> ar_asf_tg1@1)
estat eform ar_asf_tg1

di exp(1.685276)
di exp( 1.194685)
di exp(2.377326)


*snacks
gsem(ar_snk_t1 <- t_snk  med fed stoiletshare  kids, logit) ///
(ar_snk_tg1 <- l_ar_snk t_snk med fed stoiletshare  kids, logit) ///
(U[id] -> ar_snk_t1@1) ///
(U[id] -> ar_snk_tg1@1)
```

```
estat eform ar_snk_tg1

di exp( 1.019171)
di exp(.6597278)
di exp( 1.574452)


 a
```

Appendix 8

Appendix 8.1 Stata .do file data preparation and mediation analysis

```
*MEDIATION ANALYSIS. DATA PREPARATION.
*see DAG, and "mediation vars.xlsx" for details of
variables
*load dataset
use
"N:\Documents\IGH\Analysis\Cohort\cohort_anon_all.dta",
clear
*save in another location, and re-load
save
"N:\Documents\IGH\Analysis\Mediation\mediation_24m.dta" ,
replace
clear
use
"N:\Documents\IGH\Analysis\Mediation\mediation_24m.dta" ,
clear
//keep data on 978 infants included in cohort
keep if noinfant==1 & id!=945
codebook id //yes. includes 978
keep if age<730 //3980 obs deleted. total obs in
dataset=19093
*(M) TIME-VARYING MEDIATOR IYCF
*gen var counting pbf 0-2. Stricter criteria here than in
3-5. at
least 3 pbf
by id: egen tot_pbf02 = seq() if pbfnow==1 & agemonths<3
replace tot_pbf02=0 if pbfnow==0 & agemonths<3
tab tot_pbf02 if agemonths<3, miss
by id: egen max_pbf02 = max(tot_pbf02) if agemonths<3
recode max_pbf02 (0/2=0 "No") (3/5 = 1 "Yes"), gen(pbf02)
tab pbf02
*gen var counting pbf 3-5. Here at least 2 pbf = yes
by id: egen tot_pbf35 = seq() if pbfnow==1 & agemonths>=3
& agemonths<6
replace tot_pbf35=0 if pbfnow==0 & agemonths>=3 &
agemonths<6
tab tot_pbf35 if agemonths>=3 & agemonths<6, miss
by id: egen max_pbf35 = max(tot_pbf35) if agemonths>=3 &
agemonths<6
recode max_pbf35 (0/1= 0 "No") (2/4 = 1 "Yes"),
gen(pbf35)
tab pbf35
*gen var counting asf 6-11. At least 4 asf between 6-11
by id: egen tot_asf611 = seq() if asf==1 & agemonths>=6 &
agemonths<12
```

```
replace tot_asf611 = 0 if asf==0 & agemonths>=6 &
agemonths<12
tab tot_asf611 if agemonths>=6 & agemonths<12, miss
by id: egen max_asf611 = max(tot_asf611) if agemonths>=6
& agemonths<12
tab max_asf611
sum max_asf611, detail
forvalues i=0/7{
codebook id if max_asf611==`i'
}
recode max_asf611 (0/3 = 0 "No") (4/7 = 1 "Yes"),
gen(asf611)
tab asf611
*gen var counting asf 12-17. at least 3 between 12-17
by id: egen tot_asf1217 = seq() if asf==1 & agemonths>=12
& agemonths<18
replace tot_asf1217 = 0 if asf==0 & agemonths>=12 &
agemonths<18
tab tot_asf1217 if agemonths>=12 & agemonths<18, miss
by id: egen max_asf1217 = max(tot_asf1217) if
agemonths>=12 & agemonths
<18
tab max_asf1217
sum max_asf1217, detail
forvalues i=0/7{
codebook id if max_asf1217==`i'
}
replace tot_asf1217 = 0 if asf==0 & agemonths>=12 &
agemonths<18
tab tot_asf1217 if agemonths>=12 & agemonths<18, miss
by id: egen max_asf1217 = max(tot_asf1217) if
agemonths>=12 & agemonths
<18
tab max_asf1217
sum max_asf1217, detail
forvalues i=0/7{
codebook id if max_asf1217==`i'
}
recode max_asf1217 (0/3 = 0 "No") (3/7 = 1 "Yes"),
gen(asf1217)
tab asf1217
*gen var counting asf 18-23. at least 4 between 18-23
months
by id: egen tot_asf1823 = seq() if asf==1 & agemonths>=18
& agemonths<24
replace tot_asf1823 = 0 if asf==0 & agemonths>=18 &
agemonths<24
tab tot_asf1823 if agemonths>=18 & agemonths<24, miss
by id: egen max_asf1823 = max(tot_asf1823) if
agemonths>=18 & agemonths
<24
```

```
tab max_asf1823
sum max_asf1823, detail
forvalues i=0/7{
codebook id if max_asf1823==`i'
}
recode max_asf1823 (0/3 = 0 "No") (4/7 = 1 "Yes"),
gen(asf1823)
tab asf1823
*(L1) TIME_VARYING INTERMEDIATE CONFOUNDER DIARRHOEA
*gen var counting diarr 0-2. Any diarrhoea = 1
by id: egen tot_diarr02 = seq() if diarr==1 & agemonths<3
replace tot_diarr02=0 if diarr==0 & agemonths<3
tab tot_diarr02 if agemonths<3, miss
by id: egen max_diarr02 = max(tot_diarr02) if agemonths<3
tab max_diarr02 //range 0/1
recode max_diarr02 (0=0 "No") (1/2 = 1 "Yes"),
gen(diarr02)
tab diarr02
*gen var counting diarr 3-5. Any diarrhoea = 1
by id: egen tot_diarr35 = seq() if diarr==1 &
agemonths>=3 & agemonths<6
replace tot_diarr35=0 if diarr==0 & agemonths>=3 &
agemonths<6
tab tot_diarr35 if agemonths>=3 & agemonths<6, miss
by id: egen max_diarr35 = max(tot_diarr35) if
agemonths>=3 & agemonths<6
tab max_diarr35 //range 0/3
recode max_diarr35 (0 = 0 "No") (1/3 = 1 "Yes"),
gen(diarr35)
tab diarr35
*gen var counting diarr 6-11. Any diarrhoea = 1
by id: egen tot_diarr611 = seq() if diarr==1 &
agemonths>=6 & agemonths
<12
replace tot_diarr611 = 0 if diarr==0 & agemonths>=6 &
agemonths<12
tab tot_diarr611 if agemonths>=6 & agemonths<12, miss
by id: egen max_diarr611 = max(tot_diarr611) if
agemonths>=6 &
agemonths<12
tab max_diarr611 //range= 0/5
sum max_diarr611, detail
forvalues i=0/5{
codebook id if max_diarr611==`i'
}
recode max_diarr611 (0 = 0 "No") (1/5 = 1 "Yes"),
gen(diarr611)
tab diarr611
*gen var counting diarr 12-17. Any diarrhoea = 1
tab max_diarr611 //range= 0/5
sum max_diarr611, detail
```

```
forvalues i=0/5{
codebook id if max_diarr611==`i'
}
recode max_diarr611 (0 = 0 "No") (1/5 = 1 "Yes"),
gen(diarr611)
tab diarr611
*gen var counting diarr 12-17. Any diarrhoea = 1
by id: egen tot_diarr1217 = seq() if diarr==1 &
agemonths>=12 &
agemonths<18
replace tot_diarr1217 = 0 if diarr==0 & agemonths>=12 &
agemonths<18
tab tot_diarr1217 if agemonths>=12 & agemonths<18, miss
by id: egen max_diarr1217 = max(tot_diarr1217) if
agemonths>=12 &
agemonths<18
tab max_diarr1217 //range 0/6
forvalues i=0/6{
codebook id if max_diarr1217==`i'
}
recode max_diarr1217 (0 = 0 "No") (1/6 = 1 "Yes"),
gen(diarr1217)
tab diarr1217
*gen var counting diarr 18-23. Any diarrhoea = 1
by id: egen tot_diarr1823 = seq() if diarr==1 &
agemonths>=18 &
agemonths<24
replace tot_diarr1823 = 0 if diarr==0 & agemonths>=18 &
agemonths<24
tab tot_diarr1823 if agemonths>=18 & agemonths<24, miss
by id: egen max_diarr1823 = max(tot_diarr1823) if
agemonths>=18 &
agemonths<24
tab max_diarr1823 //range 0/5
forvalues i=0/5{
codebook id if max_diarr1823==`i'
}
recode max_diarr1823 (0 = 0 "No") (1/5 = 1 "Yes"),
gen(diarr1823)
tab diarr1823
save, replace
* (t) Create variables to mark out all the observations
in each age band
gen ageband=1 if agemonths<3
replace ageband=2 if agemonths>=3 & agemonths<6
replace ageband=3 if agemonths>=6 & agemonths<12
replace ageband=4 if agemonths>=12 & agemonths<18
replace ageband=5 if agemonths>=18 & agemonths<24
label define ageband 1 "0-2mo" 2 "3-5mo" 3 "6-11mo" 4
"12-17mo" 5
"18-23mo"
```

```
label values ageband ageband
tab1 ageband
*check that there's no missclassified age-appropriate
IYCF
tab2 ageband pbf02 pbf35 asf611 asf1217 asf1823, first
*stack M and L variables so that there is just one per M
or L
*M: IYCF
gen m=.
by id: replace m=pbf02 if ageband==1
by id: replace m=pbf35 if ageband==2
by id: replace m=asf611 if ageband==3
tab2 ageband pbf02 pbf35 asf611 asf1217 asf1823, first
*stack M and L variables so that there is just one per M
or L
*M: IYCF
gen m=.
by id: replace m=pbf02 if ageband==1
by id: replace m=pbf35 if ageband==2
by id: replace m=asf611 if ageband==3
by id: replace m=asf1217 if ageband==4
by id: replace m=asf1823 if ageband==5
label var m "Age-appropriate IYCF"
label values m yesno
tab ageband m
*L1: Diarrhoea
gen l1=.
by id: replace l1=diarr02 if ageband==1
by id: replace l1=diarr35 if ageband==2
by id: replace l1=diarr611 if ageband==3
by id: replace l1=diarr1217 if ageband==4
by id: replace l1=diarr1823 if ageband==5
label var l1 "Diarrhoea"
* (L2) INTERMEDIATE CONFOUNDER AT t02: first weight
measurement
*investigate early life weight vars
scatter waz06 age if ageint==0
scatter mean_cweight age if ageint==0
scatter whz06 age if ageint==0
scatter mean_cweight waz06 if ageint==0
scatter mean_cweight birthweight if ageint==0
hist mean_cweight if ageint==0
hist mean_cweight if ageint==0, freq
hist waz06 if ageint==0, freq
hist waz06 if ageint==0, freq norm
hist mean_cweight if ageint==0, freq norm
//--> verdict: use the raw weight measurement as it is
nearly
normally distributed
//if we need internal z-scores later: Mean = 2.924435 SD
=
```

```
0.5004883. n=876
*gen variable
*tag earliest weight obs for each child. then replicate
in all
agebands
sort id age
by id: egen wt_seq = seq() if ageint==0 & mean_cweight!=.
tab wt_seq //of 876, 20 have 2 measurements within the
first month. I
will use the first one
sum age if wt_seq==1, detail //mean=8.33 range 0/29
sum age if wt_seq==2, detail //mean=26.05 range 7/29
replace wt_seq=. if wt_seq==2
replace wt_seq=mean_cweight if wt_seq==1 //856 changes
by id: egen wt0 = max(wt_seq) if ageband==1
drop wt_seq
//NOTE: additional birthweight and LBW data:
//incorporate this as part of manipulation for X and C
vars
save, replace
clear
replace wt_seq=mean_cweight if wt_seq==1 //856 changes
by id: egen wt0 = max(wt_seq) if ageband==1
drop wt_seq
//NOTE: additional birthweight and LBW data:
//incorporate this as part of manipulation for X and C
vars
save, replace
clear
*reload, but keep only one observation per ageband
use
"mediation_24m.dta" , clear
egen ttag = tag(id ageband)
tab ttag //1= 4267 obs
keep if ttag==1 //14826 obs deleted. now= 4267 obs
*save as komal_mediation
save "N:\ komal_mediation.dta" ,
replace
*now re-arrange
clear
use "N:\ komal_mediation.dta"
tab ageband
*rectangularise dataset: fillin by id ageband.
fillin id ageband //now 4890 obs
tab ageband //978 in each ageband. Nice!
save, replace
*(Y) add y vars, to last ageband (ageband=5) only.
*use data sets from Mediation folder
//these have the correct id labels
//(pred_ht came with id number as value labels.
//have decoded to a string var, and destringed to int.
```

```
*predicted length
joinby id using
"N:\Documents\IGH\Analysis\Mediation\pred_ht.dta",
unmatched(both) _merge(merge_predht)
tab merge_predht // 15 obs in master data only. these 3
did not have
curves fitted (no anthro data)
list id if merge_predht==1 //id=251, 720, 816
drop merge_predht
replace pred_ht = . if ageband!=5 //3900 obs changed to .
sum pred_ht //n=975. mean=80cm, sd=3.12, min=64.8,
max=90.59
*a,b,c parameters (optional)
joinby id using
"N:\Documents\IGH\Analysis\Mediation\pred_abc.dta",
unmatched(both) _merge(merge_abc)
tab merge_abc // 15 obs in master data only. these 3 did
not have
curves fitted (no anthro data)
list id if merge_abc==1 //id=251, 720, 816
drop merge_abc
foreach par of varlist a b c {
replace `par' =. if ageband!=5
}
//3900 obs changed to . in each
sum a b c //975 in each.
save
"N:\Documents\IGH\Analysis\Mediation\komal_mediation.dta"
, replace
*Now arrange C vars.
foreach par of varlist a b c {
replace `par' =. if ageband!=5
}
//3900 obs changed to . in each
sum a b c //975 in each.
save
"N:\Documents\IGH\Analysis\Mediation\komal_mediation.dta"
, replace
*Now arrange C vars.
*Keep analysis vars. Save as a different file
clear
use
"N:\Documents\IGH\Analysis\Mediation\komal_mediation.dta"
keep id clusterid hhid ageband mow3 mow a b c pred_ht wt0
birthweight m
l1 water stoiletshare ses2 kids adults mage25 msmoke med
mhtz
fage30 fed fsmoke lmup2 female sescore2 _fillin
save
"\\ komal_med
_vars.dta", replace
```

```
//funny file path. hmm. ignore. but check if re-running.
clear
use
"\\ komal_med_vars.dta"
*add var for LBW
gen lbw=1 if birthweight<2.5 & birthweight!=.
replace lbw=0 if birthweight >=2.5 & birthweight!=.
label values lbw yesno
tab lbw if ageband==1 //done correctly. 128 infants are
LBW
replace birthweight=. if ageband!=1
replace lbw=. if ageband!=1
save, replace
*fill missing vars by group :
//first sort by id and var (miss at the bottom), then,
bysort id:
replace var=var[1]. where 1 is the first obs
misstable sum id-lbw
//only for C, X and ID vars which are do not vary across
agebands.
foreach var of varlist clusterid hhid fsmoke msmoke
stoiletshare
sescore2 ses2 med fed water mage25 fage30 kids adults
female lmup2 mow
mow3 mhtz{
sort id `var'
bys id: replace `var'=`var'[1]
}
misstable sum id-lbw
forvalues i=1/5 {
misstable sum id-lbw if ageband==`i'
}
//phew! numbers correspond to missing data frequencies
for each variable
sort id ageband
*order vars with optional ones at the end.
order id clusterid hhid ageband mow3 mow pred_ht m l1 wt0
water
stoiletshare ses2 kids adults mage25 msmoke med mhtz
fage30 fed
fsmoke lmup2 female sescore2 a b c birthweight lbw
_fillin
******tag complete cases
*complete (Y) (predicted height)
by id: egen yobs = seq() if pred_ht!=.
*order vars with optional ones at the end.
order id clusterid hhid ageband mow3 mow pred_ht m l1 wt0
water
stoiletshare ses2 kids adults mage25 msmoke med mhtz
fage30 fed
```

```
fsmoke lmup2 female sescore2 a b c birthweight lbw
_fillin
******tag complete cases
*complete (Y) (predicted height)
by id: egen yobs = seq() if pred_ht!=.
tab yobs
sort id yobs
bys id: replace yobs=yobs[1]
*complete (X)
by id: gen xobs = 1 if mow!=.
tab xobs
*complete (M)
by id: egen mobs = seq() if m!=.
tab mobs, miss
by id: egen mtot = max(mobs)
*how many have none, 1, 2.. all 5 timepoints
forvalues i=1/5{
codebook id if mtot==`i'
}
codebook id if mtot==.
//only 1=140, only 2=74, only 3 = 95, only 4= 101, all 5
=558,
none=10
*complete (L1)
by id: egen lobs = seq() if l1!=.
tab lobs, miss
by id: egen ltot=max(lobs)
**how many have none, 1, 2.. all 5 timepoints
forvalues i=1/5{
codebook id if ltot==`i'
}
codebook id if ltot==.
//only 1=101, only 2= 74, only 3= 96, only 4=112, all 5 =
540.
none = 55.
*complete (L2)
by id: egen l2obs = seq() if wt0!=.
tab l2obs
sort id l2obs
bys id: replace l2obs=l2obs[1]
*complete (C1-n) vars
foreach var of varlist water stoiletshare ses2 kids
adults mage25
msmoke med mhtz fage30 fed fsmoke lmup2 female {
by id: gen obs_`var' = 1 if `var'!=.
tab obs_`var'
}
*All (C)
egen cobs = rowmiss(obs_*)
tab cobs
codebook id if cobs==0 //672
```

```
*all complete cases
*how many complete cases for each component
codebook id if yobs==1 //975
codebook id if xobs==1 //690
codebook id if mtot==5 //558
*All (C)
egen cobs = rowmiss(obs_*)
tab cobs
codebook id if cobs==0 //672
*all complete cases
*how many complete cases for each component
codebook id if yobs==1 //975
codebook id if xobs==1 //690
codebook id if mtot==5 //558
codebook id if ltot==5 //540
codebook id if l2obs==1 //856
codebook id if cobs==0 //672
*var for all complete components
gen complete=1 if yobs==1 & xobs==1 & mtot==5 & ltot==5 &
cobs==0 &
l2obs==1
tab complete //2910 obs. So should be 2190/5=438 infants
codebook id if complete==1 //438 .
di 438/690 //63% of infants with exposure data are
complete cases
*without L2 data
gen complete2 = 1 if yobs==1 & xobs==1 & mtot==5 &
ltot==5 & cobs==0
tab complete2 // 2505 obs. So it should be 501 infants
codebook id if complete2==1 //501
di 501/690 //72% of infants with exposure data are
complete cases
*save this version before re-naming and dropping data for
the final
version.
save, replace
clear
*rename and label vars for analysis
*re-load and save as a different analysis file.
use
"\\ komal_med_vars.dta"
save
"\\ komal_med_analysis.dta"
clear
use
"\\ komal_med_analysis.dta"
/*Summary of changes I will make
(1) drop: hhid, clusterid (further anonymisation)
(2) drop: merging and completeness components vars
(3) change varnames; var labels;
(4) re-order for analysis
```

```
*/
*(1) drop: hhid, clusterid (further anonymisation)
drop hhid clusterid
*(2) drop: merging and completeness components vars
drop _fillin yobs xobs mobs mtot lobs ltot l2obs
obs_water
obs_stoiletshare ///
obs_ses2 obs_kids obs_adults obs_mage25 obs_msmoke
obs_med obs_mhtz ///
obs_fage30 obs_fed obs_fsmoke obs_lmup2 obs_female cobs
*(3) change varnames; var labels;
*names
*(2) drop: merging and completeness components vars
drop _fillin yobs xobs mobs mtot lobs ltot l2obs
obs_water
obs_stoiletshare ///
obs_ses2 obs_kids obs_adults obs_mage25 obs_msmoke
obs_med obs_mhtz ///
obs_fage30 obs_fed obs_fsmoke obs_lmup2 obs_female cobs
*(3) change varnames; var labels;
*names
rename ageband t
rename mow3 x
rename mow xb
rename pred_ht y
rename m m
rename l1 l
rename wt0 l2
rename water c1
rename stoiletshare c2
rename ses2 c3
rename kids c4
rename adults c5
rename mage25 c6
rename msmoke c7
rename med c8
rename mhtz c9
rename fage30 c10
rename fed c11
rename fsmoke c12
rename lmup2 c13
rename female c14
rename sescore2 c3_b
rename lbw l2_b
rename birthweight l2_c
rename a y_a
rename b y_b
rename c y_c
*labels
label var id "ID"
label var t "Age band"
```

```
label var x "Maternal BMI category"
label var xb "Maternal overweight"
label var y "Length at 24 months"
label var l "Diarrhoea"
label var l2 "First weight measurement"
label var m "Age appropriate IYCF"
label var c1 "Piped water"
label var c2 "Shared toilet"
label var c3 "Quintile of asset score"
label var c4 "4+ children in the household"
label var c5 "More than 2 adults in the house"
label var c6 "Maternal age >=25"
label var c7 "Maternal smoking"
label var c8 "Maternal education"
label var c9 "Maternal height z-score"
label var c10 "Paternal age >=30"
label var c11 "Paternal education"
label var c12 "Paternal smoking"
label var c13 "Planned pregnancy"
label var c14 "Infant sex: Female"
label var c3_b "Asset score"
label var l2_b "Low birth weight"
label var c8 "Maternal education"
label var c9 "Maternal height z-score"
label var c10 "Paternal age >=30"
label var c11 "Paternal education"
label var c12 "Paternal smoking"
label var c13 "Planned pregnancy"
label var c14 "Infant sex: Female"
label var c3_b "Asset score"
label var l2_b "Low birth weight"
label var l2_c "Birthweight"
label var y_a "Size"
label var y_b "Tempo"
label var y_c "Velocity"
label var complete "Complete cases inc. L2"
label var complete2 "Complete cases ex. L2"
foreach var of varlist l c6 c10 c14{
label values `var' yesno
}*
(4) re-order for analysis
order id t y x m l l2 c1 c2 c3 c4 c5 c6 c7 c8 c9 c10 c11
c12 c13
c14 ///
xb y_a y_b y_c l2_b l2_c c3_b complete complete2
save, replace
*checking...
*all data
tab2 t x m l, first row
//note that total n varies in each ageband for m and l
*complete cases (inc. L2) n=438
```

```
tab2 t x m l if complete==1, first row
*complete cases (exc. L2) n=501
tab2 t x m l if complete2==1, first row
************************************ .do file from
Bianca
cd "
"
adopath ++" "
set more off
cap log close
log using komal_mediation_021218b.log, replace
*-----------------------------------------*READ
DATA*-------------------------------------------------*
use komal_med_analysis,clear
count
*-----------------------------------------*DATA
MANIPULATION*-----------------------------------------*
egen first=tag(id)
ta first
*give a value to y at each visit (for later simple
regressions)
*-----------------------------------------*DATA
MANIPULATION*-----------------------------------------*
egen first=tag(id)
ta first
*give a value to y at each visit (for later simple
regressions)
sort id t
qui by id: replace y=y[_N] if y==.
qui by id: replace l2=l2[1] if l2==.
*complete cases (inc. L2) n=438
tab2 t x m l if complete==1 , first row
***Restricting to complete set:*Including L2 (First
weight measure
data). should become 2190 obs for 438 children
keep if complete==1
count
ta first
***Drop all extra vars
drop xb y_a y_b y_c l2_b l2_c c3_b
*-----------------------------------------*
*reshape long and generate new exposure var
ta first
drop first
rename l2 wt1
reshape wide m l,i(id) j(t)
*new breastfeeding var
ta m1 m2
ta m1 m2,nol
gen m_12=0 if m1==0
replace m_12=1 if m1==1 & m2==0
```

```
replace m_12=2 if m1==1 & m2==1
ta m_12 m2
ta m_12 m1
*generate new dummy vars needed for gformula
ta c3,gen(c3_)
*----------------------------------------*ANALYSIS
STEPS*----------------------------------------*
*below some preliminary examinations of associations,
then mediation
analysis proper
*----------------------------------------*
*does HT2 depend on feeding practice at waves 1 and 2
(the exposure)?
reg y i.m_12 i.c1 i.c2 b3.c3 i.c4 i.c5 i.c6 i.c7 i.c8 c9
i.
c10 i.c11 i.c12 i.c13 i.c14
reg y i.m_12 i.l1 wt1 i.c1 i.c2 b3.c3 i.c4 i.c5 i.c6 i.c7
i.c8 c9 i.
c10 i.c11 i.c12 i.c13 i.c14
* negative effect, stronger in those br.-fed for longer
* similar results when or not controlling for concurrent
size and
diarrhoea
*----------------------------------------*
*does HT2 depend on feeding practice at waves 3 to 5 (the
mediators)?
foreach t of numlist 3/5{
di
di in red "HT2 on feeding at time `t'"
reg y i.m`t' i.m_12 i.l1 wt1 i.c1 i.c2 b3.c3 i.c4 i.c5
i.c6 i.c7 i.c8
* similar results when or not controlling for concurrent
size and
diarrhoea
*----------------------------------------*
*does HT2 depend on feeding practice at waves 3 to 5 (the
mediators)?
foreach t of numlist 3/5{
di
di in red "HT2 on feeding at time `t'"
reg y i.m`t' i.m_12 i.l1 wt1 i.c1 i.c2 b3.c3 i.c4 i.c5
i.c6 i.c7 i.c8
c9 i.c10 i.c11 i.c12 i.c13 i.c14
}
* yes more so at earlier times
*----------------------------------------*
*does animal based diet depend on feeding practice at
waves 1 and 2?
foreach t of numlist 3/5{
di
di in red "later diet at time `t' on br feeding"
```

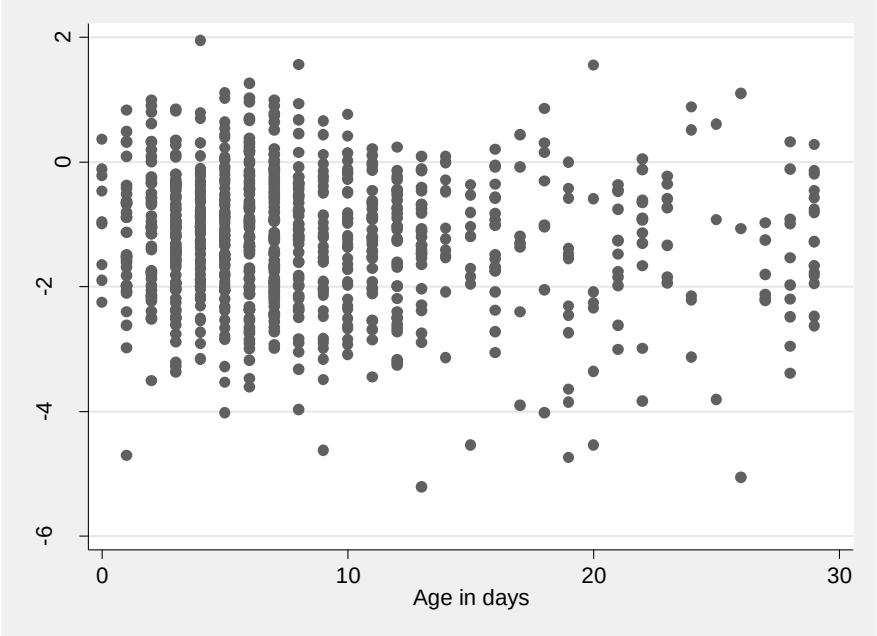
```
logistic m`t' i.m_12 l1 wt1 i.c1 i.c2 b3.c3 i.c4 i.c5
i.c6 i.c7 i.c8 c9
i.c10 i.c11 i.c12 i.c13 i.c14
}
*yes, it is a negative associaiton (ORs<1)
*----------------------------------------*
*does diarrhoea depend on diet (controlling for previous
episodes of
diarrhoea) ?
tab2 l1 l2 l3 l4 l5,firstonly row nokey
logistic l3 i.m_12 l2 l1 wt1 i.c1 i.c2 b3.c3 i.c4 i.c5
i.c6 i.c7 i.c8
c9 i.c10 i.c11 i.c12 i.c13 i.c14
foreach t of numlist 4/5{
di
di in red "diarrhea at time `t' on earlier diet "
local tau=`t'-1
logistic l`t' m`tau' l`tau' l1 wt1 i.c1 i.c2 b3.c3 i.c4
i.c5 i.c6 i.c7
i.c8 c9 i.c10 i.c11 i.c12 i.c13 i.c14
}
*odds of diarrhoea mostly depende on previous diarrhoea
and less on
previous diet
log close
ex
log using formal_mediation.log, replace
*-----------------------------------------**-------------
---------------
-------------*
*simple mediation analysis with m_12 as exposure and m3
as the only
mediator
*total effect
reg y i.m_12 i.l1 wt1 i.c1 i.c2 b3.c3 i.c4 i.c5 i.c6 i.
c7 i.c8 c9 i.c10 i.c11 i.c12 i.c13 i.c14
*direct effect
reg y i.m_12 i.m3 i.l1 wt1 i.c1 i.c2 b3.c3 i.c4 i.c5 i.c6
i.
c7 i.c8 c9 i.c10 i.c11 i.c12 i.c13 i.c14
*-----------------------------------------**-------------
---------------
-------------*
*formal mediation analysis with m_12 as exposure and m3
as the only
mediator and l3 as the interm confounder
#delimit;
*-----------------------------------------**-------------
---------------
-------------*
```

```
*formal mediation analysis with m_12 as exposure and m3
as the only
mediator and l3 as the interm confounder
#delimit;
gformula y m_12 m3 m4 m5 l5 l4 l3 wt1 l1 c1 c2 c3_1 c3_2
c3_4 c3_5 c4
c5 c6 c7 c8 c9 c10 c11 c12 c13 c14,
mediation outcome(y) exposure(m_12) mediator(m3)
post_confs(l3) base_confs(wt1 l1 c1 c2 c3_1 c3_2 c3_4
c3_5 c4 c5 c6 c7
c8 c9 c10 c11 c12 c13 c14)
oce baseline(0) control(m3:0)
commands(y:regress, m3:logit, l3:logit)
equations(
y: m3 l3 i.m_12 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5 c6
c7 c8 c9
c10 c11 c12 c13 c14,
m3: l3 i.m_12 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5 c6
c7 c8 c9
c10 c11 c12 c13 c14,
l3: i.m_12 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5 c6 c7
c8 c9
c10 c11 c12 c13 c14,
)
minsim samples(3) moreMC simulations(10000) replace
seed(1202);
#delimit cr
ex
#delimit;
gformula y m_12 m3 m4 m5 l5 l4 l3 wt1 l1 c1 c2 c3_1 c3_2
c3_4 c3_5 c4
c5 c6 c7 c8 c9 c10 c11 c12 c13 c14,
mediation outcome(y) exposure(m_12) mediator(m3)
post_confs(l3 l4 l5) base_confs(wt1 l1 c1 c2 c3_1 c3_2
c3_4 c3_5 c4 c5
c6 c7 c8 c9 c10 c11 c12 c13 c14)
obe control(m3:0, m4:0, m5:0)
commands(y:regress, m5:logit, m4:logit, m3:logit,
l5:logit, l4:logit,
l3:logit)
equations(
y: m5 m4 m3 l5 l4 l3 m1 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5
c4 c5 c6
c7 c8 c9 c10 c11 c12 c13 c14,
m5: m4 m3 l5 l4 l3 m1 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4
c5 c6
c7 c8 c9 c10 c11 c12 c13 c14,
l5: m4 m3 l4 l3 m1 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5
c6
c7 c8 c9 c10 c11 c12 c13 c14,
m4: m3 l4 l3 m1 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5 c6
```

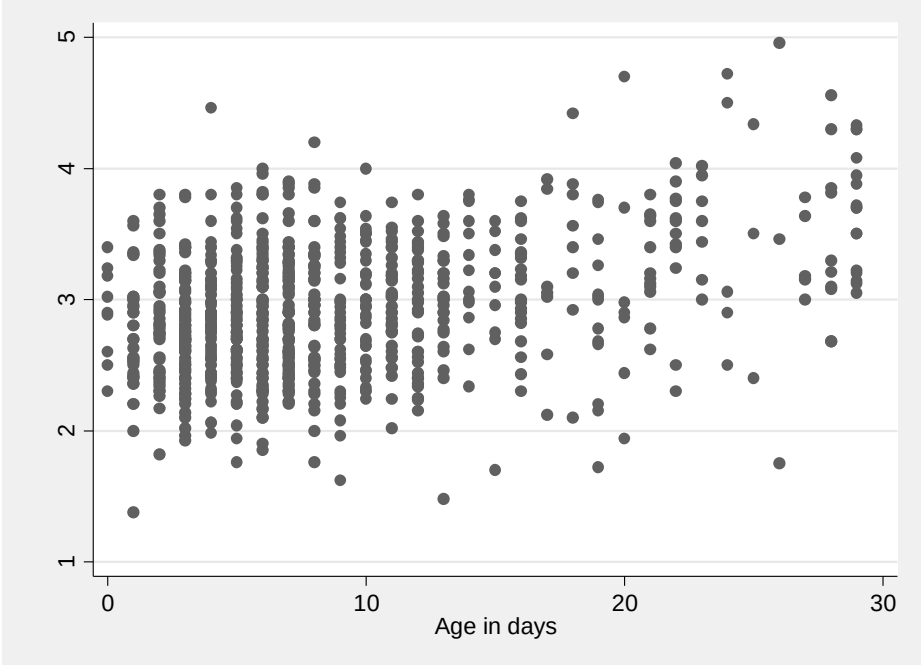
```
c7 c8 c9 c10 c11 c12 c13 c14,
l4: m3 l3 m1 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5 c6
c7 c8 c9 c10 c11 c12 c13 c14,
m3: l3 m1 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5 c6
c7 c8 c9 c10 c11 c12 c13 c14,
l3: m1 wt1 l1 c1 c2 c3_1 c3_2 c3_4 c3_5 c4 c5 c6
c7 c8 c9 c10 c11 c12 c13 c14,
)
minsim samples(100) moreMC simulations(50000) replace
seed(1202);
#delimit cr
ex
*derived(l2 xl) derrules(l2:l*l, xl:x*l)
ex
)
minsim samples(100) moreMC simulations(50000) replace
seed(1202);
#delimit cr
ex
*derived(l2 xl) derrules(l2:l*l, xl:x*l)
ex
```

Appendix 8.2 Graphs of birth weight and first weight measurement data

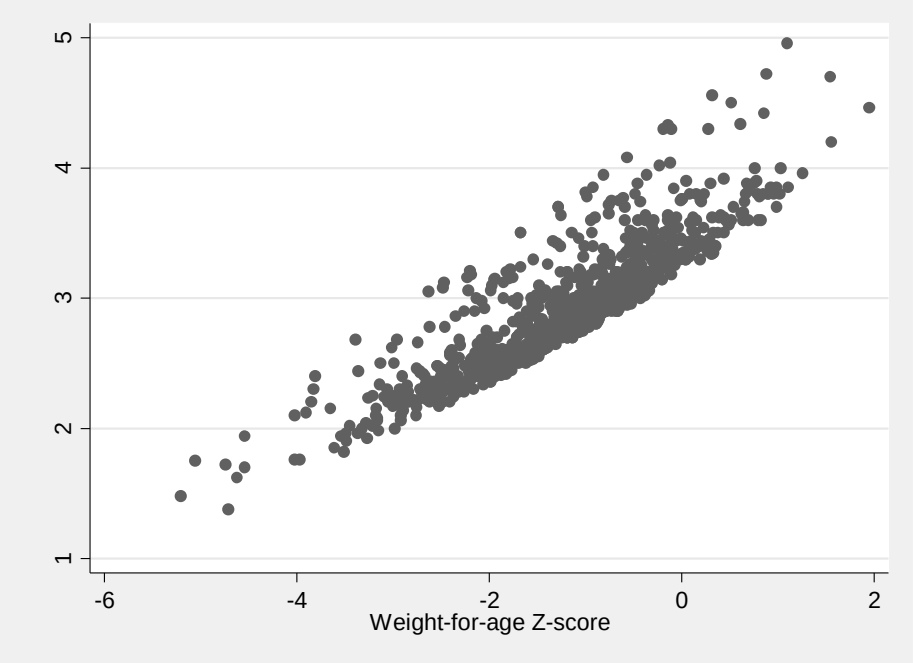
Weight-for-age z-score in first month



Weight (kg) in first month

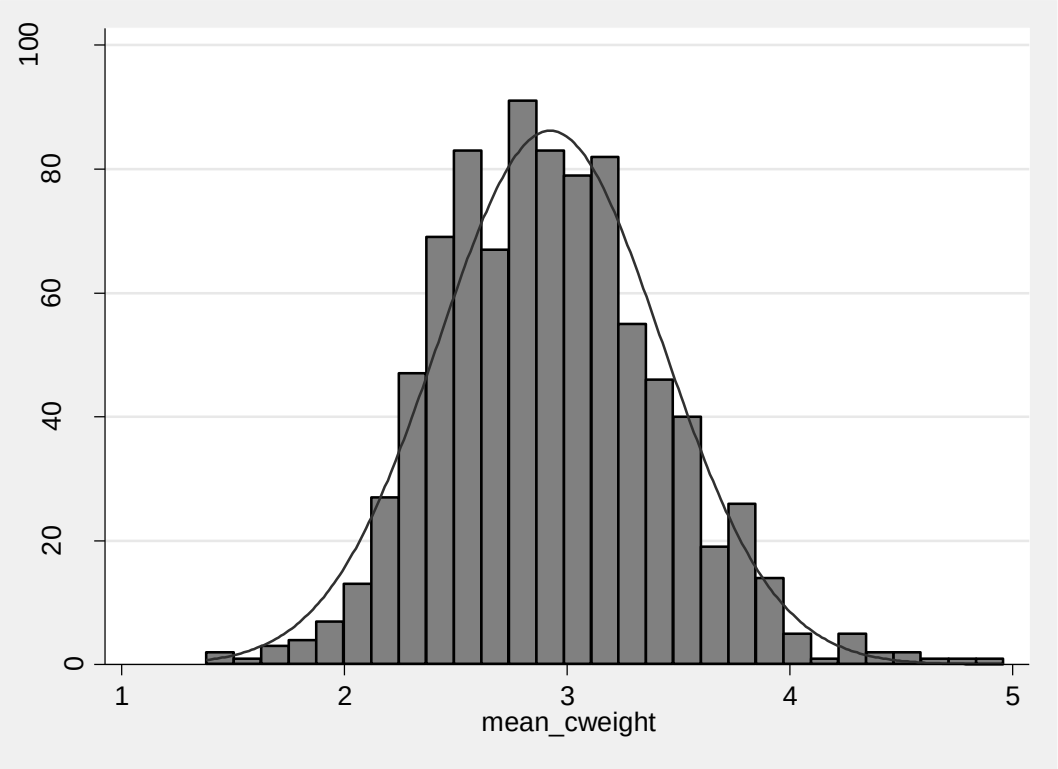


Weight vs weight-for-age z-score



Weight in first month vs birth weight

Distribution of first weight measurement



Distribution of first weight-for-age measurement