THE DETERMINANTS OF STUNTING AT AGE TWO:

A HOLISTIC MODEL FOR SOUTH AFRICA USING THE BIRTH TO TWENTY COHORT STUDY

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ABSTRACT

Despite the high prevalence of stunting among young children in South Africa, there is little work investigating the determinants of stunting using comprehensive empirical models. The objective of this paper is to empirically operationalise a holistic model of the determinants of stunting, using rich data from the Birth to Twenty cohort study. In particular, the study examines the various mechanisms, both proximate and distal, through which mother's education affects stunting among two-year olds. In multivariate analysis, a higher birth weight z-score, being in the highest socio-economic status category, the mother's positive attitude towards modern healthcare (proxied by a vaccination score), and a better-quality care environment, are protective against stunting. Higher birth order and having experienced some symptoms of illness increase the risk of stunting. Although mother's education was no longer significant in the final regression, the results show that the largest mediating effect is through socio-economic status, with other important pathways being the antenatal environment (measured by the birth weight z-score) and the mother's reproductive behaviour. Overall, many of the factors that were protective against stunting in the final analysis, whether they operated through maternal education or not, were related to the mother's involvement in the child's life. This reinforces the notion that to protect children, mothers need to be supported and empowered in providing care.

KEYWORDS: Malnutrition, children, mothers

JEL CODES: I120; J130

CONTENTS

ACRONYMS

AME	Average Marginal Effects
BF	Breastfeeding
CoE-FS	Centre of Excellence in Food Security
FAO	Food and Agriculture Organization
FFQ	Food Frequency Questionnaire
FVS	Food Variety Score
Gr	Grade
HAZ	Height-for-age Z-score
HDDS	Household Dietary Diversity Score
IFNuW	Institute for Food, Nutrition and Well-being
OLS	Ordinary Least Squares
PCA	Principal Components Analysis
T1	Tercile 1
WHO	World Health Organization
WHOCDD	WHO Child Dietary Diversity

1. INTRODUCTION

In this report, we examine the determinants of child malnutrition using the Birth to Twenty data, a cohort study of children born in Soweto-Johannesburg in 1990. In particular, we focus on the causes of low height-for-age, or stunting, at age two, the measure most commonly used to capture chronic undernutrition in children. The consequences of stunting in early childhood have been widely studied across various disciplines, and include impaired cognitive function, poor schooling outcomes, reduced earnings in adulthood and poor maternal reproductive health outcomes (Walker, Chang, Powell & Grantham-McGregor, 2005; Victora et al. 2008; Dewey & Begum, 2011).

Globally, stunting in childhood remains a substantial public health concern, with an estimated 171 million children under five years of age stunted in 2010 (de Onis, Blössner & Borghi, 2013). In South Africa, prevalence is particularly high for a country of middle-income status, with latest estimates from 2012 suggesting stunting among children younger than three years old may be as high as 27% (Shisana et al., 2013). May & Timaeus (2014) plot the prevalence of stunting against gross national income, and find that the prevalence in South Africa is well above the average of countries with a similar national income.

Given the magnitude of the problem, and a growing body of research documenting the negative consequences for cognitive function and schooling outcomes among South African children (Victora et al.; 2008; Yamauchi, 2008; Casale, Desmond & Richter, 2014; Casale & Desmond, 2016; Casale, 2016), there is surprisingly a rather limited South African literature investigating the causes of stunting in young children. As will be discussed in more detail below, data limitations constrain the ability of researchers to empirically estimate the determinants of stunting in a comprehensive model, taking into account all the main factors determining stunting that are identified in the conceptual literature.

Although the data on the early childhood period are now more than twenty years old, the Birth to Twenty cohort study probably still contains the most detailed information on young children necessary for an analysis of the causes of stunting. It collected rich longitudinal information on children, their households and care environment, their health and feeding practices and, importantly, regular anthropometric measures. The purpose of this study is to empirically estimate a more holistic model of stunting at age 2 than has currently been estimated in the South African literature, drawing on these detailed data.

Our particular focus in the empirical work is on the various mechanisms and pathways through which mother's education, a well-documented negative correlate of stunting, influences child malnutrition. More specifically, we are able to investigate the influence of, among other factors, the household's socio-economic status and access to resources and services, the care environment, breastfeeding and dietary diversity, illness, maternal reproductive behaviour, and the mother's attitude towards healthcare. Our focus on mother's education as a key factor through which other effects operate is twofold.

First, a majority of children in SA, over 70%, live with their mother resident in the household while only 30 percent of African children live with their father (Rudwick, 2013; Ward, Makusha & Bray, 2015), making the mother the most obvious channel through which child health can be accessed

and improved in the early years. Second, mother's education is amenable to policy manipulation and is receiving widespread attention in the development and public health literatures.

This research report is structured as follows. Section 2 discusses the international literature on how to model the causes of stunting conceptually and empirically, including reference to the challenges present in trying to formulate and estimate a causal model. This is followed by a more focused review of literature examining the pathways from maternal education to stunting. Section 3 contains a review of the South African research investigating the determinants of height-for-age (HAZ) or stunting, and here the work is contextualised in terms of the challenges identified in Section 2. Section 4 presents the empirical approach adopted in this research report and describes the data used from the Birth to Twenty study. Section 5 contains the bivariate and multivariate regression results. Section 6 provides a discussion of the findings, particularly with respect to the pathways through which maternal education influences child stunting. Section 7 includes brief concluding comments on the lessons learnt from using the Birth to Twenty longitudinal data to study child malnutrition, as well as the gaps that remain.

2.   INTERNATIONAL LITERATURE

2.1.   CONCEPTUAL MODELS OF STUNTING

An intuitive model of the causes of stunting would include a lack of adequate nutrition due to a poor diet and/or suboptimal breastfeeding practices, gender and genetic inheritance, and infections that limit the capacity of an infant's body to absorb nutrients. These factors are commonly called *proximal factors* in the literature.[1] While it is true that in some fundamental, medically-defined sense it is only these factors that can affect nutrient intake and uptake and stunt linear growth, to restrict the study of the determinants of stunting to these factors would provide a very limited understanding of the phenomenon.

To understand the determinants of stunting (in a more societal sense) one needs to think about the contextual factors that are associated with these proximal factors and by extension with stunting. These include socioeconomic status (SES; including wealth, parents' occupation and asset ownership), service delivery and hygiene, healthcare facilities, home and neighbourhood environment, societal beliefs about feeding, and parental education and preferences (often called *distal factors*). When one considers that the causal relationships between some of these groups of factors can be bidirectional (as between household and maternal characteristics and between infections and stunting), the difficulty in conceptually and empirically accounting for the determinants of stunting starts to become apparent.

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[1] The distinction between proximal and distal factors is often made in epidemiology in order to distinguish between individual-level behavioural factors that have direct effects on the biological phenomena causing a disease or condition (proximal), and the wider society-, community- or family-level factors that will predict a disease or condition due to their effect on the proximal factors (distal) (Krieger, 2008; Lämmle, Woll, Mensink & Bos, 2013). The extent to which proximal or distal factors should be considered in an etiological study is an area of debate (Slemming, Bello, Saloojee & Richter, 2016), with some contending that the framework itself should be abandoned (Krieger, 2008). We refer the reader to these texts for additional detail on the debate.

In order to clarify the conceptual thinking around how these causes fit together, most papers in the literature use the UNICEF (1990) framework on the causes of stunting as the foundation for their model (see Black et al., 2008; Fenske, Burns, Hothorn & Rehfuess, 2013; Stewart, Iannotti, Dewey, Michaelsen & Onyango, 2013). The models in these papers contain multiple levels (typically between two and four) on a spectrum from proximal causal factors to more distal factors, although the terminology used to describe these levels varies in the literature.[2]

For example, Black et al. (2008) place broad factor groups into three levels - immediate, underlying and basic causes - with arrows indicating casual relationships. As is customary, the proximal (immediate) causes are dietary intake and disease, while the underlying causes cover household wealth, services, the environment and the state of care and food security. Basic causes are the social, economic and political context and the resultant lack of various types of social, physical, human and financial capital (the distal factors). For illustration, we reproduce this model in Figure 1. Fenske et al. (2013) also use three levels in their model - immediate, intermediate and underlying - occupied by 16 groups of determinants (reproduced in Figure 2). Only caloric and nutrient intake and uptake and intrauterine growth occupy the immediate level, while a large range of feeding, infection and healthcare and service access groups occupy the intermediate level. Finally, regional, household and maternal characteristics constitute the underlying determinants. Stewart et al. (2013) present the WHO model (which builds on the UNICEF model) with a focus on complementary feeding, in which determinants are sorted into causes and context (replicated in Figure 3). This model contains fewer levels and no information about causal relationships between determinant groups, but within each factor group there is a lot of useful detail about which factors and variables are relevant.

This approach of extensively modelling various groups of determinants at different levels sometimes contains explicit hypotheses about which groups cause and are caused by other groups (as in Black et al., 2008). However, in many cases (such as Stewart et al., 2013), no explicit causal relationships between groups are stipulated, except for a general understanding that higher levels determine lower levels. In other words, there is not a very clear sense from these diagrams of the pathways of causation between groups at different levels, and the potential pathways among determinants at the same level. In our reading of the literature, this appears to be one of the key challenges in modelling the causes of stunting conceptually (and empirically).

2.2.   EMPIRICALLY MODELLING STUNTING

In addition to the complexities of conceptual modelling described above, operationalising the conceptual model raises challenges. These include deciding on which determinants are grouped together, and how they relate to other determinants in the empirical model. Recent research, influenced by the systems approach in epidemiology, has emphasised looking at all determinants simultaneously to avoid oversimplified regressions that are susceptible to omitted variable bias (Fenske et al., 2013).

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[2] In the review of South African studies that follows in Section 3, further examples of this proximal-distal framework can be seen.

When there are multiple variables that can be used to represent a determinant group, one has to decide on how many to include in the model and in what manner. Sometimes researchers choose not to include more than one so as to avoid collinearity, but it can be difficult to justify the grounds on which one is chosen over another (with statistical power, historical significance and theoretical support among the justifications used). Others aim for a more exhaustive utilisation of available variables and attenuate the effects of collinearity by combining variables in a determinant group into a single indicator, rather than including each variable on its own. For example, a SES index could cover the ownership of multiple assets as well as house type and parents' occupation.

Although most empirical studies we have read present sophisticated conceptual models with various levels, they tend to revert to a regression approach which basically reduces to putting in some (those significant in a bivariate analysis) or all the determinants into a single multivariate regression (see Burgard, 2002; Chopra, 2003; Fenske et al., 2013; Slemming et al., 2016). The inclusion of one or more elements from each group of determinants may be effective in isolating the effect of a single determinant while controlling for the other covariate groups. However, to attain a deeper understanding of how determinants interact with each other (say, infection and SES), or which factors are responsible for a change in a variable of interest, one has to use a more complex specification, which introduces variables in stages, for example.

A further hazard of including all groups of determinants (both proximal and distal) in the same regression is that one risks underestimating the significance and magnitude of the more distal determinants. Weitkunat and Wildner (2002) estimate the effect of treating all variables as equally proximal determinants when in fact there are sequentially causal relationships (with some of the distal variables in the model leading to an effect in the outcome through their effect on proximal variables). They find that the effect of distal variables is adjusted away by the inclusion of the proximal variables, and that the true effect of the distal variable can only be seen when the proximal variables it acts through are omitted, or when a regression strategy is used that better represents the causal process by considering sequential causation.

In our reading of the empirical literature, we found one simple approach to exploring mediating effects particularly useful, and we model our own work on this approach, as we will explain in more detail later. This involves selecting an underlying determinant of interest, such as mother's education (see Frost, Forste & Haas, 2005) or socio-economic status (see Chopra, 2003), and then adding other groups of determinants individually (for example, feeding practices, then the care environment, then health services, and so on), observing how the coefficient on the determinant of interest (mother's education or SES) changes in response and whether the added correlates are themselves significant determinants of stunting.

This indicates whether and to what extent the determinant of interest (mother's education or SES) is mediated by the added variables (i.e. whether the added variables act as a pathway) or whether the added variables are simply independent covariates. Of course, specifying which pathways are included in the empirical modelling has to be based on previous research and conceptual reasoning about the relationships between the variables.

Finally, a complete regression model is run which includes all the variables that were previously added individually. From this regression, it is evident how much of the effect of the determinant of

interest is explained by the other variables together, and which variables maintain an independent effect in the multivariate framework.[3]

2.3.   APPROACH ADOPTED IN THIS PAPER

In this study, we are also guided by the determinant groups included in the UNICEF conceptual framework, and in particular we use the model in Fenske et al. (2013) (see Figure 2) to help guide our choice of which groups of determinants to include in our model. Influenced by the systems approach to epidemiology, we attempt to incorporate as many groups as possible in the model so as to avoid omitted variable bias. Within each of these groups we generally took the approach of combining the information from multiple variables into indices to attenuate concerns with collinearity.

However, our model differs to a certain extent from that of Fenske et al. (2013) as we attempt to make some explicit hypotheses about the causal relationships between groups of determinants. Our empirical strategy follows the work of Frost et al. (2005) and others described above, where we use a more 'sequential' approach to modelling the effect of an underlying variable of interest on stunting, and the pathways through which it operates - some of which might be considered proximal pathways, such as feeding practices, and others distal, such as access to health services.

In this research report, the underlying variable of interest is maternal education, chosen due to its centrality in the care of South African children and because of its amenability to policy interventions. Over the last few decades, maternal education has come to be viewed in the global literature as one of the most influential determinants of child health outcomes (Frost et al., 2005). Significant positive relationships are commonly found in the empirical literature, although some research has questioned to what extent maternal education is significant only because it acts as a proxy for other factors, such as SES and area of residence (Desai & Alva, 1998). Below we review some key recent papers using developing country data that model the pathways from maternal education to stunting. This will act as a complement to the Fenske et al. (2013) model and will inform our hypotheses about which variables affect others.

Frost et al. (2005) is a particularly useful paper for our purposes, as it not only has a carefully crafted empirical model, but it also provides a substantial review of previous work in this area. Frost et al. focus on explaining the various causal pathways from maternal education to stunting, examining the contributions of each pathway relative to the others. In their paper, five hypothesised pathways are explored: SES, health knowledge, attitudes towards health care, female autonomy, and reproductive behaviours.

The links from education to SES are theoretically clear, as educated mothers are more likely to find gainful employment and to marry educated, employed men. SES factors consistently explain half or

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[3] One drawback of this approach is that one can only really understand the interactions between one determinant of interest and the other variables in the model. To understand the pathways, for example, from SES to stunting as well as from maternal education to stunting, would require repeating the exercise. Some papers that have sought to examine several interactions simultaneously employ a structural equation modelling (SEM) approach (see Dukhi, Sartorius & Taylor, 2016). This approach is particularly popular with those who support ecological models of behaviour (see Krieger, 2008), as it allows them to explore effects between factors of various levels and types simultaneously, enabling an understanding of the effect of a factor in its personal, community and social context (Glanz, Rimer & Viswanath, 2008). However, there are also limitations to this modelling approach which are beyond the scope of this research report to explain further (we refer the reader to Nachtigall, Kroehne, Funke & Steyer, 2003; Valluzzi, Larson & Miller, 2003; Tomarken & Waller, 2005; Bagozzi & Yi, 2012). However, future work might consider whether SEM is an appropriate approach in this context and using these data.

more of the effect of education on child malnutrition (Frost et al., 2005). The connection between education and health knowledge is conceptually clear but is less supported by the evidence, although the effect of health knowledge on child health outcomes has been found to be significant in some empirical work. The third pathway hypothesises that formal education can change mothers' attitudes towards modern medicine, making them more likely to use preventive care and take their children to medical centres, and less likely to be fatalist (Frost et al., 2005). Without qualitative attitude information, this study (like many others) uses utilisation of health care as a proxy for such attitudes.

The fourth pathway – female autonomy – relates to how increased education adjusts the power balance in favour of women. If women are more likely to be the principal caregiver and are more inclined to notice and to dedicate resources to the alleviation of child health problems, then more control over resources in the household is likely to lead to an improvement in child health outcomes. This result has been found in a number of studies that have looked specifically at the issue of female autonomy, with control over family income being a common proxy (Frost et al., 2005). Finally, education can affect the reproductive behaviour of mothers. More educated women may feel that contraceptive and reproductive outcomes are under their control, leading to a decrease in fertility rates, unwanted pregnancies and high-risk pregnancies (where the mother's age is under 18 or above 35) (Frost et al., 2005). These reproductive outcomes, as represented by parity, gravidity[4], birth spacing, birth order and maternal age, have repeatedly been found to be correlated with a variety of outcomes such as stunting and child mortality.

The previous paragraph provides some indication of the difficulty in selecting indicators or proxies for the various pathways, especially those such as attitudes that are difficult to measure in surveys. Frost et al. (2005), using data from the Bolivian DHS, tend to take the option of combining various proxies into indices to represent the pathway of interest. Two additive indices, household environment (covering type of house and services) and household wealth (an asset index), were created to represent SES along with paternal education and occupation. Similarly, an index for knowledge was created using a series of questions about awareness of programmes and remedies, and attitudes were proxied by a health utilisation index based on prenatal and other care received and ethnicity. Whether husband and wife had discussed or approved of family planning were combined into an index to act as a proxy for mother's autonomy.

Finally, the reproductive variables included were maternal age, parity and birth interval. Their regressions follow the method, discussed above, of adding each variable grouping in isolation with mother's education before running a complete model which includes all variable groupings together (Frost et al., 2005). SES and attitude are found to be the pathways that explain most of the effect of maternal education on stunting. However, maternal education remains significant even in their complete model, suggesting either unexplored pathways from education to stunting, or incomplete

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[4] Gravidity refers to the number of times a woman has been pregnant regardless of the outcome, and parity refers to the number of times a woman has given birth or been pregnant up to a viable gestational age.

capturing of the hypothesised pathways. In the complete model, the SES indices, the attitude proxies and parity and birth interval (capturing reproductive behaviours) remain significant.

Other studies examining the effect of maternal education on stunting generally use a more basic approach that fails to shed as much light on the mechanisms through which maternal education affects stunting.

Abuya, Onsomu, Kimani & Moore (2011) study the role of maternal education in determining health outcomes in Kenya, using similar groups and indicators to Frost et al. (2005), but because they do not run regressions separately for each determinant grouping, they are unable to isolate the extent to which each acts as a pathway. They find that children born to mothers with primary education are 94% less likely to be stunted than those born to mothers without primary education, but that this effect drops away once other covariates, such as attitude, autonomy and parity are included in the model.

Semba et al. (2008) examine the importance of varying levels of parental education using cross-sectional data for Indonesia and Bangladesh. They find a similar significant effect of maternal education in both countries, after controlling for paternal education, household expenditure and other maternal and child factors (Semba et al., 2008: 326). Interestingly, while higher parental education levels were correlated with higher prevalence of eight caregiving activities assessed in the study (including vaccination and family planning), these behaviours did not mediate the effect of education in the regression analysis. The effect of maternal education on stunting is assessed further in some of the South African studies reviewed in the following section.

3. SOUTH AFRICAN LITERATURE

This section reviews the South African literature on the causes and correlates of child malnutrition. Table A1 in Appendix A summarises the key studies that have estimated the determinants of stunting or low HAZ for young children in South Africa, listed in the order in which they are described below. These papers represent a good spread of the approaches, conceptual models and data sources used in the research. We organise our discussion of these studies in this section by the type of data source: The Birth to Twenty cohort, other localised panels, nationally representative surveys, and single community micro-studies. It will become apparent that no widely representative study has utilised a broad enough combination of variables to adequately populate the determinant groups in the conceptual models described above. The high degree of correlation between many of these determinants means that the results of studies that do not use a comprehensive model may be biased.

Several studies have also used data from the first two years of Birth to Twenty (1990 to 1992) to investigate the determinants of stunting (Jones et al., 2008; Willey, Cameron, Norris, Pettifor & Griffiths, 2009; Slemming et al., 2016). These studies all used multivariate logistic regressions, with a baseline model progressively expanded to incorporate all regressors, and in one case these regressions were stratified by sex (Slemming et al., 2016). The studies share a similar underlying conceptual model based on proximal and distal causes, with the latter either acting on stunting

directly or being mediated by proximal factors in the model. Where they differ is in the group of causes they choose to focus on. Willey et al. (2009) and Jones et al. (2008) focus on the relationship between stunting and SES and the variables mediating between them, while Slemming et al. (2016) look at the relationships between stunting and maternal risk exposure during pregnancy, birth characteristics, and child risk exposure in infancy. The Jones et al. paper utilised a limited range of the data available in Birth to Twenty, with most variables related to the cause of interest (SES) and a few controls, while the other two implemented more comprehensive empirical models but still omitted some important information, for example about food frequency or infections. Mixed results were found for SES (measured by an asset index in all three papers), with Jones et al. (2008) finding its effects significant, Slemming et al. (2016) finding it significant for males only (with maternal education significant for females and birth weight significant for both sexes), and Willey et al. (2009) finding some variables that are highly likely correlated with the SES asset index (parental employment and education and whether the household has a domestic worker) to be significant.

Timaeus (2012) analysed the determinants of HAZ with a focus on how the results of the regressions were affected by height measurement error for primary-school aged children. He used the KwaZulu-Natal Income Dynamics Study (KIDS), a three-wave panel survey from 1993, 1998 and 2004 of African and Indian households living in KwaZulu-Natal that were originally sampled in the 1993 national Project for Statistics on Living Standards and Development survey. The aim of the paper was not to estimate a causal model of stunting, and a very limited set of regressors was included in the regressions, namely demographic variables, an indicator for metropolitan residence, maternal education and household per capita expenditure. Race, age, gender[5], maternal education and expenditure were found to be significant.

Studies that have sought to estimate the determinants of stunting with a large sample and at a nationally representative level have generally been restricted to using the few (publicly available) national household surveys that have included a section on nutritional information and outcomes. Most prominent of these are the Project for Statistics on Living Standards and Development (PSLSD), a cross-sectional survey from 1993, and the National Income Dynamics Study (NIDS), a panel survey with four waves from 2008 to 2015.

Burgard (2002) uses the PSLSD to assess the determinants of stunting in SA, with a focus on racial inequities in health outcomes, and compares the results to estimates for Brazil (based on the Demographic and Health Survey). The paper uses a conceptual approach focused on demographic and SES factors that only act through more proximal factors. The empirical approach reflects this model by regressing stunting on mother's race before adding underlying factors (namely, child's age and sex, parental occupation and education, and household wealth) and then what Burgard refers to as more proximal factors (namely, access to services, mother's age and child's age at weaning).

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[5] The finding that males are more likely to be stunted is very common in the literature on stunting (see Aguayo et al., 2015; Slemming et al., 2016; Dukhi et al., 2016), and hypothesised reasons for the discrepancy include dietary patterns favouring females (although no evidence has been found for this in South Africa) and differences between males and female in biological responses to poor foetal growth (Slemming et al., 2016).

Race, wealth and parents' education were all predictive of stunting in South Africa, highlighting the importance of the underlying factors, while age at weaning was the only significant proximal factor.[6]

May and Timeaus (2014) use the first wave of NIDS (2008) to estimate to what extent inequalities in SES predict inequalities in nutritional outcomes. A straightforward regression including household expenditure, household size, an asset index, maternal education and some demographic and geographic controls is estimated, with the asset index found to be protective against stunting. Ardington and Gasealahwe (2012) use the first two waves of NIDS (2008 & 2010) to explore the effect of a change in household pension status on child malnutrition in Wave 2. A simple model regressing stunting on the pension and a few controls is estimated, as is a more extended model to cover a similar range of variables to those used in May and Timeaus (2014) (but using income rather than expenditure). After controlling for stunting status in the previous wave, the household's income and gaining a pension, as well as being female, are protective against stunting. The household losing a pension and being in an urban area (in wave 1) increase the risk of stunting. In Burgard (2002) and May and Timaeus (2014), the sample of children were aged 0-59 months, while Ardington and Gasealahwe (2012) included all children under the age of 16 years in their study.

Finally, there are a number of papers that are based on purpose-built cross-sectional nutrition surveys, generally conducted within single communities in rural areas (Chopra, 2003; Mamabolo et al., 2005; Dukhi et al., 2016). Sample sizes are small (ranging from 162 to 848 children in these studies) which enables a wider variety of topics (including breastfeeding and complementary feeding) to be covered in the questionnaires, and in greater depth, than is possible in national-level surveys. As a result, such studies tend to employ broad conceptual and empirical models encompassing several groups of determinants.

Chopra (2003) examined children between the ages of 3 and 59 months in the Hlabisa district in KZN, focusing on the role of SES in determining stunting and how SES interacts with almost all other determinants (directly or indirectly, according to the conceptual model). This paper employs a three-stage regression approach that is very similar to that of Burgard (2002), starting with SES and then incorporating environmental and proximal factors. Chopra's (2003) empirical model is able to include more measures of proximal factors in the regression than were available in Burgard's (2002) data, including breastfeeding, weaning and the introduction of solids. Significant results in the final multivariate model included maternal education, the presence of the father, a toilet in the household, distance to a clinic, and breastfeeding patterns.

Mamabolo et al. (2005) conducted a study on 3-year-olds in villages in the central region of Limpopo province. They argue that stunting should be understood as the product of the interplay between cultural, economic, demographic and environmental factors. Their regression contains many maternal factors (including marital status, maternal age, education and occupation), household crowding and size, presence of the father and mother, and HAZ and weight-for-age (WAZ) at earlier ages. Household crowding and maternal occupation are found to be significant determinants.

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[6] The notion of proximal used by Burgard (2002) is different to that explained earlier in this paper. For her, a factor is proximal if it is potentially endogenous to child outcomes.

Dukhi et al. (2016) explored the determinants of stunting in children between 0-59 months in the iLembe district of KwaZulu-Natal. Their conceptual model contained four levels based on Bronfenbrenner's (1994) ecological system.[7] Their empirical approach reflected this with a multivariate regression (followed by a Shapley decomposition to quantify relative contributions) and a generalised structural equation model to identify direct and indirect effects. In the multivariate model, the regressors included measures of food security and consumption (based on a series of questions about having enough food in the household), income, maternal education and access to health and other services. Significant positive determinants of stunting were food insecurity, longer distance to a clinic and lack of access to piped water, and boys were more likely than girls to be stunted.

More specific information about these studies is presented in Appendix Table A1, particularly on how the regressors were selected and which alternative models were estimated. From the summary presented here, however, it is apparent that most papers are not able to estimate a comprehensive model (mostly due to data limitations), and that their empirical approaches are often not sufficient to reveal the mediating relationships between different causes. Although some use a multi-stage regression approach, this generally involves two or three stages of adding progressively more determinants. Because factor groups are not first incorporated in isolation, one cannot attribute mediating effects to any specific determinants.

In this paper, we attempt to improve on these approaches by following the methodology employed in Frost et al. (2005) as described earlier, where we will show the various pathways (proximate and distal) through which mother's education (a distal factor) affects stunting. At the same time, we attempt to construct a more comprehensive model of the causes of stunting than has previously been implemented for South Africa. Given the extensive range of variables available in Birth to Twenty, we try to avoid the omitted variable bias problem discussed above, by populating all of the determinant groups included in the typical conceptual model on the causes of stunting. The description of the data and the variables used in the analysis as well as the empirical specifications are presented in the next section.

4. DATA DESCRIPTION AND METHODOLOGY

4.1. DATA

The Birth to Twenty cohort study (which is ongoing) is a longitudinal study of all children born in Johannesburg over a seven-week period between April and June 1990 in private and public hospitals and clinics.[8] Information was collected from mothers at antenatal clinics, in delivery centres, and

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[7] Bronfenbrenner's (1994) ecological models of human development maintain that in order to understand human growth one needs to consider an individual's entire ecological system. This system is analysed in terms of five levels or subsystems which cover a range of factors from immediate environment to institutional surroundings.

[8] It is currently housed at and conducted through the MRC/Wits Developmental Pathways for Health Research Unit (DPHRU). https://www.wits.ac.za/dphru/mrcwits-developmental-pathways-for-health-researc/

through face-to-face interviews with the caregiver and child at least once a year. The sample of eligible singleton births was 3273, but data were collected from approximately 1600 to 2200 participants at each interview point, resulting in response rates of between 50% and 68% per data collection wave. This compares favourably with birth cohort studies in other developing countries (Richter, Norris, Pettifor, Yach & Cameron, 2007). As is documented in detail elsewhere (Norris, Richter & Fleetwood, 2007), attrition seems to have been most prevalent among white South Africans, and over time the panel has become less representative of those born in private hospitals compared to public hospitals/clinics and living in suburban Johannesburg compared to other areas in the city.

For this study we draw data mostly from the first two years of the child's life. The outcome variable of interest is stunting at age two. Stunting is defined as a height-for-age z-score (HAZ) of less than two standard deviations below the mean of the reference population, using the WHO Child Growth Standards (World Health Organization, 2006). The sample size for children that have non-missing data on HAZ at age two is 1805.[9] Mean HAZ at age two (in 1992) is -1.16 (SD 1.12) and the prevalence of stunting is 20.5%.[10] This is somewhat lower than the national average of 31.5% recorded in 1993 for children under five (using the PSLSD), but this would be expected given that the population is from an urban area and rural prevalence of stunting has been found to be higher in South Africa (World Health Organization, 2012).

The Birth to Twenty study is unique, not only because it has longitudinal information on the child and its environment starting from as early as the antenatal period, but because of the wealth of information collected over the period. This includes data on child growth and development, health, the home environment, caregiver characteristics, socio-economic circumstances, access to services and, importantly for our purposes, breastfeeding and dietary diversity. This allows for a fairly comprehensive model of stunting to be estimated, which is not possible using the larger nationally-representative household surveys. We try to make use of as much of the data as possible in our modelling, described further below.

4.2. EMPIRICAL MODEL

Taking inspiration from Frost et al. (2005) we used a sequential-type approach to modelling the causal groups, although our causal groups are slightly different from theirs given the data available to us. Regression 1 is a baseline regression of stunting at age two on the child's gender. Regression 2 adds to the baseline model the main variable of interest, maternal education. Thereafter each group of factors is added in isolation to the Regression 2 specification. Regression 3 includes a birthweight variable, which could be interpreted as separating the effect of maternal education into a prenatal and postnatal component. In other words, it controls for factors in the antenatal period that may have limited or promoted intrauterine growth. Regression 4 includes an SES determinant

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[9] Of the initial sample of 3273 children, only 1873 were contacted in year 2 (Norris et al., 2007), of which 1805 children had data on HAZ. A comparison of these 1805 children with the remaining 1468 children from the initial sample shows some significant differences by race, mother's age and parity. The group of 1805 children in our sample are less likely to be African (76% vs 81%), they have younger mothers (25.8 vs 26.2 years old), and their mothers have lower parity (2.2 vs 2.3). There were no statistically significant differences between the two groups for gender, birth weight, and whether the child was born in a public or private hospital. See Norris et al. (2007) for more details on attrition.

[10] The decimal age of the children when their 2y height was collected ranged from 1.87 to 2.43, with a mean of 2.04.

group consisting of an asset index and a parental occupation index. A group capturing feeding practices is included in Regression 5, and here we include a measure of dietary diversity (the food variety score) and breastfeeding (a breastfeeding duration variable). Regression 6 attempts to capture attitudes towards healthcare, proxied by the caregiver's utilisation of available healthcare (measured by the number of vaccinations the child was taken for). Regression 7 includes information on illnesses, measured by three indices capturing different types of symptoms the child exhibited. Regression 8 attempts to capture the mother's reproductive autonomy and behaviour, and contains her age when the child was born, the birth order of the index child and a measure of birth spacing. Regression 9 includes information about the care environment, including whether the mother was the principal caregiver and an index capturing the relationship between the mother and child and their wellbeing. Regression 10 adds the services group which contains an index based on water, toilet and electricity access. Finally, Regression 11 is the complete model, estimated with all determinant groupings included in the specification.[11]

The variables are described in more detail below.

Baseline:	$y_i = f_i$	[1]
Maternal education:	$y_i = f_i + meduc_i$	[2]
Birth:	$y_i = f_i + meduc_i + bw_i$	[3]
SES:	$y_i = f_i + meduc_i + assets_i + occ_i$	[4]
Feeding:	$y_i = f_i + meduc_i + fvs_i + bf_i$	[5]
Attitudes:	$y_i = f_i + meduc_i + vac_i$	[6]
Diseases:	$y_i = f_i + meduc_i + res_i + eye_i + gast_i$	[7]
Reproductive:	$y_i = f_i + meduc_i + age_i + age2_i + bo_i + bs_i$	[8]
Care:	$y_i = f_i + meduc_i + pr_i + rel_i$	[9]
Services:	$y_i = f_i + meduc_i + serv_i$	[10]
Full model:	$y_i = f_i + meduc_i + bw_i + assets_i + occ_i + fvs_i + bf_i + vac_i + res_i$	
	$+ eye_i + gast_i + age_i + age2_i + bo_i + bs_i + pr_i + rel_i + serv_i$	[11]

where,

y_i = 1 if child i is stunted, 0 otherwise (or child i's height-for-age z-score);

f_i = female;

$meduc_i$ = completed maternal education;

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[11] These model specifications do not incorporate all of the potential determinants of stunting contained in the Birth to Twenty data. Notable omissions include access and proximity to health services, gestational age, whether the child was born in a private hospital, paternal education, marital status, parity and gravidity. These variables were generally omitted for a variety of reasons: for example, the information they contained was already described by another variable and their inclusion would have raised concerns of multicollinearity (e.g. paternal education), they were not significant even in bivariate regressions, and their inclusion did not change the effects of the other variables in multivariate analysis (ignoring effects caused by sample size changes).

bw_i = birth weight z-score;

$assets_i$ = household asset score;

occ_i = parents' occupation score;

fvs_i = food variety score;

bf_i = duration of breastfeeding;

vac_i = vaccination score;

res_i = index for respiratory symptoms;

eye_i = index for eyes and ears symptoms;

$gast_i$ = index for gastrointestinal symptoms;

age_i = maternal age at birth;

$age2_i$ = maternal age at birth squared;

bo_i = birth order;

bs_i = whether another child was born within 24 months of child i;

pr_i = whether the mother is the principal caregiver;

rel_i = index of the mother and child's relationship and wellbeing; and

$serv_i$ = index of household access to services.

While the focus of this study is on predicting stunting at age 2 (a binary variable), the same set of regressions was estimated with the continuous height-for-age Z-score (HAZ) as the dependent variable. This allows us to test whether the determinants included in the model predict height across the full distribution as opposed to only predicting the probability of falling below the stunted cutoff point. For all HAZ regressions ordinary least squares (OLS) estimation was used, while for all stunted regressions probit estimation was used and the average marginal effects (AME) were calculated to facilitate interpretation of the results.

Table 1 presents the means and standard deviations of the dependent and explanatory variables. In several cases (detailed below), the indices were calculated using principal components analysis (PCA), and sometimes scores were included in the model in categories (terciles of the score, for example). Where this is the case, the table also provides the descriptive statistics for the underlying continuous variables or count indices as they are more informative in a descriptive sense.

The summary statistics show that 51% of the children are female. Maternal education was divided into three categories to include in the regressions: Grade 5 or less, Grade 6 to Grade 10, and Grade

11 or higher (with the lowest category the omitted variable in the regressions).[12] Mothers had 9.6 years of education on average; only 8% fell into the lowest category, 50% fell into the middle category and 42% in the highest category.[13] The mean birth weight in grams was 3071g and the mean birth weight Z-score (included in the regressions) was -0.53.

The SES group contains two variables. The asset index was created by performing PCA[14] on a series of dummy variables for ownership of a TV, a fridge, a washing machine, a phone, the house they live in (all collected at year 2), and a crowding variable measured as the ratio of people to sleeping rooms (a variable derived by the Birth to Twenty data team based on information on the antenatal to 2 year period). In the table, crowding and the asset index are initially shown separately. Households have on average 2.8 of the 5 assets in question and 3.3 household members per sleeping room. The PCA index is included in terciles in the regressions.

The occupation index (also a derived variable from Birth to Twenty) allocates each parent's occupation/labour market status into one of 6 categories: 0 - unemployed, housewife, student; 1 - informal sector; 2 - unskilled manual or routine non-manual labour; 3 - semi-skilled manual, unskilled supervisor, white collar and inspectional; 4 - semi-professional, lower executive, skilled manual, semi-skilled supervisor; 5 - independent, high profession, manager and executive.[15] Then the parents' scores were added and divided by 2 to get a score ranging from 0 to 5. In cases where only one parent's score was available this was taken to be the score for both parents. The mean occupation score in the sample was 1.9.

One of the most important immediate determinants of stunting, and an obvious potential pathway from maternal education to stunting, is the child's feeding. Infants receive the nutrients they need through both breastfeeding and supplementary foods in the first two years. For this determinant group, we use information about the duration of breastfeeding and dietary diversity, the latter derived from the food frequency questionnaire at 1 year of age. The breastfeeding information was coded into a categorical variable distinguishing between those who were not breastfed at all, those who were breastfed for a short period (up to 3 months), and those who were breastfed for longer than 3 months. The mean duration children were breastfed was 10.6 months, while around 8% were not breastfed at all, and 27% were breastfed for 3 months or less.[16]

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[12] Mother's education was divided into these three categories based on the 6 categories included in the Birth to Twenty data file: no formal education, Grade 5 or less, Grades 6 to 7, Grades 8 to 10, Grades 11 to 12, and post-school education.

[13] Although father's education is likely to be an important determinant of growth, we decided against including it together with maternal education. This was to avoid problems of collinearity between the two parents' education, and to focus on the way the education level of the mother (most commonly the principal caregiver) can affect growth outcomes. The inclusion of father's occupation in the SES grouping controls for this to some extent.

[14] In all cases where an index was created, we experimented with both simple count indices and PCA indices and found little difference between the two in terms of direction and significance of the coefficients.

[15] We thank Juliana Kagura at Developmental Pathways for Health Research Unit, Wits, for providing us with these value labels.

[16] A number of other bottle and breastfeeding variables, as well as the age at which solids were introduced, are available in the Birth to Twenty dataset. We experimented with these but found that none was significant in bivariate or multivariate regressions, and so we used the duration of breastfeeding variable (although this wasn't necessarily exclusive breastfeeding). This variable was considered to be the most appropriate and reliable to include in the analysis (based on personal communication with Professor Shane Norris). We also experimented with a 6-month cut-off instead of a 3-month cut-off in defining the categories, but this did not affect our overall findings.

The food frequency questionnaire data provide valuable insight into the supplementary feeding of children in greater Johannesburg in the early 1990s. It is very rare for a large-scale survey to include such a detailed questionnaire, and the use of these data in combination with other information from Birth to Twenty should theoretically enable an understanding of how diet quality can lead to stunting independently of covariates such as SES.[17] Despite this, the data has rarely been utilised.[18] The food frequency questionnaire asked about 149 food items, with respondents reporting whether the child usually consumed each item monthly, 2/3 times monthly, weekly, 5-6 times weekly, daily, more than twice daily, or not at all. The items consumed by the largest percentages of infants at one year were eggs, mielie-meal (maize meal), gravy, brown bread, rice, biscuits, peanut butter, bananas and oranges (all above 2%) (Gitau, 2009). We coded the food frequency questionnaire data into a food variety score and used the score terciles for the regression analysis. The food variety score is a simple count of how many items the child consumed on a monthly (or more frequent) basis. The mean score of 34 means that a typical child consumed around 34 different food items on a monthly basis.[19]

The caregiver's attitude towards modern healthcare is one of the most difficult pathways to capture with the information that is available in Birth to Twenty. The only suitable proxy is a score reflecting the number of vaccination visits, of a possible 5, the child was taken to by their first birthday. The health utilisation variable is coded 0 if 2 or fewer visits were completed, 1 if 3 or 4 visits were completed, and 2 if all 5 vaccinations were administered. Around 18% of children fell into the first category, 52% in the second and 30% in the third. [20]

Infections data were collected in two distinct sections in the Birth to Twenty questionnaire, with one investigating symptoms and the other diseases.[21] The symptoms data were collected at 6 months, 1 year and 2 years, and the questions asked whether the child had experienced each of the 9 symptoms listed in the past 2 weeks. About 78% of infants exhibited one of the respiratory symptoms at one of the time points, while only 29% exhibited an eyes and ears symptom and 45% a gastro symptom. For each symptom we created a dummy variable that had a value of 1 if the symptom had been reported at one or more of the three time points. Then for the regression

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[17] See Ruel (2002) for a discussion of why SES needs to be controlled for when regressing on dietary variables. In summary, variables capturing dietary quality or diversity are likely to be highly correlated with SES and therefore finding a significant relationship between diet and stunting could merely be a reflection of SES's role as a determinant of stunting.

[18] The one significant application of the data in the estimation of the determinants of stunting was performed by Gitau (2009) in her Masters research report.

[19] A significant amount of time was spent exploring these data and attempting to create not only other common measures of dietary diversity such as the FAO's (2011: 24) household dietary diversity score (HDDS) (which groups individual food items into 12 main food groups) but also more sophisticated measures incorporating aspects of dietary quality and distribution, in line with the most recent empirical literature on the topic. Appendix B discusses the food frequency questionnaire data in much greater depth, analysing the range of variables created using the data, and offering potential explanations for their lack of explanatory power (the latter will be revisited in the conclusion).

[20] Of course, this variable may be capturing 'access' to some extent, and not just take-up or utilisation. However, in an alternative variable from Birth to Twenty measuring 'good' versus 'poor' access to health services, where good was defined as having to travel less than 45 minutes to the nearest well-baby clinic or having received antenatal care, the vast majority of this urban sample (almost 90%) reported good access.

[21] We are indebted to Rihlat Said-Mohamed (DPHRU) for her work in cleaning the infections data, creating the variables that we used for the analysis, producing a clear and informative codebook, and recommending groupings of symptoms based on her own research (see Said-Mohamed et al., forthcoming).

analysis we used 3 groupings of these symptoms: respiratory (4 symptoms), eyes and ears (2 symptoms), and gastrointestinal (3 symptoms). PCA was used to create an index for each group of symptoms. The table contains the mean number of symptoms (within each group) exhibited by the children. Despite the broad definition of the symptoms variables, the rates of reporting are very low, with the exception of the respiratory group, in which infants reported on average 2.1 of the 4 symptoms at least once in those three time points. In order to avoid problems of multicollinearity we opted to use only the symptoms data and to omit the disease data.[22]

In the reproductive behaviour group, mother's age in years and its square were included to capture possible non-linear effects of maternal age on stunting. The mean age at which mothers gave birth to the index child is approximately 26 years. Birth order ranged from 1 to 4 with the index child being on average the second child born to their mothers. Birth spacing was a dummy variable which had a value of 1 if a younger sibling was born in the 24 months following the index child's birth (which was the case for around 7% of the children).

The caregiver group contained two variables. The one was a dummy variable reflecting whether the mother was the principal caregiver, which was the case for 62% of the children. The second variable was a PCA index based on interviewer responses to a series of six questions in the year 2 questionnaire on the mother and child's relationship and wellbeing, a useful proxy for the quality of the care environment. The questions were: does the child look clean and well looked after, does the child appear happy and secure in the mother's presence, does the mother seem unhappy and worn down, does the mother demonstrate negative feelings towards the child, does the mother appear confident and assured in her care for the child, and does the mother show affection towards the child? For each question responses ranged from 1 (completely positive) to 3 (negative) and we coded each question into a dummy variable which had a value of 1 only if the most positive response was given and a value of 0 otherwise. Table 1 presents a simple count of these dummies and shows that the mean score was 4.7 (out of a maximum of 6 positive responses).

Finally, for the services group, PCA was applied to three dummy variables: whether the household had electricity, whether the household had an indoor flush toilet, and whether the household had hot and cold indoor running water. The children's households typically had access to 1.5 of these 3 essential services. Terciles of the PCA index were included in the regressions.[23]

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[22] Disease data were collected at 6 months and at 2 years. In both questionnaires, respondents were asked whether the infant had been diagnosed since birth with any of the following diseases: pneumonia, bronchitis, asthma, croup and measles. Once again, we used dummy variables that had a value of 1 if the disease was reported in either time period. Then we created a PCA index based on all 5 disease dummies. This index was not a significant determinant of stunting or HAZ in bivariate or multivariate analysis. When each disease was included individually in the regressions, only bronchitis was found to be negatively correlated with stunting. This result has been found elsewhere and could be picking up that better-off children (with more engaged mothers) are both more likely to develop bronchitis (due to their richer diet) and more likely to be taken to a doctor and diagnosed with bronchitis (personal communication with Rihlat Said-Mohamed; Said-Mohamed, Pettifor, Momberg & Norris, forthcoming).

[23] While this services group goes some way towards capturing the variation in water, sanitation and hygiene (WASH) that has increasingly been viewed as an important determinant of stunting (see Ngure et al., 2014), the lack of detailed information in our dataset about household sanitation quality and hygiene practices, for example, means the full extent of these factors is not accounted for.

5. DATA ANALYSIS

5.1. BIVARIATE REGRESSION RESULTS

Table 2 contains the results of bivariate regressions estimating the relationship between the explanatory variables described above and both stunting and HAZ. These results start to give us an impression of which variables are predictors of stunting, although at this stage we cannot say how much of the magnitude and significance of regressors is due to their correlation with other determinants of stunting. The range of sample sizes across the variables reflects the different time points at which data were collected (with different rates of unit non-response at each point), and also missing values on the individual variables (item non-response). Some variables have particularly low sample sizes and are responsible for the large reduction in sample size in the multivariate regressions; most notable of these are the food frequency questionnaire variables and the mother-child relationship variable.

The table shows that females are less likely to be stunted and have a greater HAZ on average at age 2, in line with the general finding that males are more susceptible to linear growth retardation (Burgard, 2002; Willey et al., 2010; Slemming et al., 2016). The bivariate results for maternal education show that a higher level of education is protective against stunting and increases HAZ, and that this effect is particularly strong for mothers with Grade 11 or higher. The infant's birth weight z-score is strongly negatively correlated with stunting and positively correlated with HAZ at age 2. Both SES indices, the asset index and the occupation score, are also highly significant and protective against stunting and increase HAZ.

Surprisingly, the variables in the feeding group, namely the FVS and the duration of breastfeeding, are generally not statistically significant correlates of either stunting or HAZ. The only exception is the second tercile of the FVS which is negatively correlated with stunting relative to the lowest (i.e. first) tercile, but the coefficient is only significant at the 10% level. The FVS tercile coefficients that are not significant have the expected signs, both with respect to stunting and HAZ. However, the signs on the breastfeeding coefficients are mixed. Relative to not being breastfed at all, being breastfed for less than three months and for more than three months were both positively correlated to stunting, while in the HAZ bivariate regressions, being breastfed for less than three months increased HAZ but being breastfed for more than three months reduced HAZ. The lack of significance means such interpretation is limited however. Previous research has found dietary diversity to be especially important for growth in children who are no longer breastfed (Ruel, 2002: 21), which suggests it is wise to include both in regression analysis or even to interact them. Some interactions were tested in additional regressions (i.e. between the FVS terciles and the breastfeeding categories) but none were found to be statistically significant.

The vaccination score, representing attitudes towards healthcare, is not a significant correlate of either HAZ or stunting, although the coefficient is negative in both cases. In the infections group only the eyes and ears symptoms index was significant (p<0.01) and a high score is correlated with an increased risk of stunting. The sign on gastro symptoms similarly indicates an increase in risk, while the sign on respiratory symptoms counterintuitively indicates a decrease in risk, but neither is statistically significant.

In the reproductive behaviour group, only birth order is significant (p<0.1) as a correlate of stunting and increases infant's risk. In other words, later-born siblings are more likely to be stunted, probably reflecting competition over resources (not only financial but perhaps also the caregiver's time and energy) (Monfardini & See, 2016). However, all three variables in this group - birth order, birth spacing, and mother's age - are significant determinants of HAZ, suggesting greater effects at parts of the distribution further from the stunting cut-off point. The signs on the coefficients for both the HAZ and stunting bivariate regressions suggest that higher maternal age decreases risk while a short birth space increases it.

Whether the mother is the child's principal caregiver is not a significant correlate of stunting or HAZ. The other variable in the care group which captures the interviewer's perceptions of the relationship between the mother and child and their wellbeing, is highly significant (p<0.01); a favourable evaluation is strongly negatively correlated with stunting and positively correlated with HAZ, as we would expect.

Finally, children in households which fall in the highest (third) tercile of the services index are found to have significantly higher HAZ and lower risk of stunting relative to those in the lowest (first) tercile. No statistically significant differences are found between those in the first and second terciles.

5.2. MULTIVARIATE REGRESSION RESULTS

Table 3 contains the results of the multivariate regressions, where the dependent variable is the binary stunted variable. The sample for all the regressions was restricted to that of the final and complete model with all regressors (N=691). This restriction allows us to ascertain how the magnitude of effects and levels of significance change across the regressions as different variables are added (holding sample size constant). To ascertain whether the restriction of the sample changes the effect or significance of the regressors in any of the models, we also ran the same regressions without restricting the sample. These results are presented in Appendix D, with a short discussion of how the results compare to the restricted regression results. In brief, we found only small differences in the signs and levels of significance of the effects, giving us confidence that the results were not being driven by idiosyncrasies in the smaller sample size in the full regression specification. The overall story stays much the same.

In the baseline regression (R1), gender is not a significant correlate. In R2, both maternal education categories are protective against stunting, but only the highest category of education (Grade 11 or higher) is statistically significant (p<0.05) relative to the base category of Grade 5 or less. This pattern remains in almost all further regressions (exceptions will be discussed below).

R3 includes the birth weight z-score variable. A higher birth weight z-score is found to significantly reduce the risk of stunting. When this variable is added, the effects of the maternal education categories fall (from -0.13 to -0.106 and from -0.212 to -0.169), suggesting that some of the benefit of mother's education is already reflected in higher intrauterine growth.

In R4, both SES variables are statistically significant; the third tercile of the asset index is significant relative to the first, and the third, fourth and fifth levels of the occupation score are significant relative to level 0. In all cases a higher score is found to be protective against stunting. With the

introduction of these variables, the effects on the education categories fall (from -0.13 to -0.074 and from -0.212 to -0.102), and the third category of maternal education is no longer significant. This suggests that a large part of what the mother's education 'buys' is reflected in better labour market outcomes and more resources, which in turn benefit child health outcomes.

In R5, the feeding variables are added to the model. None of the FVS terciles or breastfeeding categories are significant. Their signs remain the same as in the bivariate regressions, with the exception of being breastfed for between 0 and 3 months which now has a marginally negative effect relative to not being breastfed (which, were it significant, would suggest these infants have a lower risk of stunting, while those breastfed for longer than three months have a higher risk).

In R6, the vaccination score has a negative sign as would be expected but it is not significant. Of the symptoms indices added in R7, both the respiratory and eyes & ears symptom indices are highly significant ($p<0.01$). While eyes & ears symptoms are positively correlated with stunting, respiratory symptoms surprisingly are negatively correlated with stunting.

R8 sees the introduction of variables capturing the mother's reproductive behaviour. Mother's age is significant ($p<0.1$) and has a negative sign while the square term is not significant and has a value close to 0, implying that maternal age has a protective and linear effect on the risk of stunting. Birth order is significant ($p<0.05$) with higher birth order increasing the risk of stunting. While the effects of maternal education hardly changed in Regressions 5-7, here we see a small decline in the effect of the highest category (from -0.212 to -0.183). This is likely to be capturing that more educated mothers have their children later, and have fewer children, which improves health outcomes for each child.

In R9, of the caregiver variables added, only the index capturing the interviewer's perception of the relationship between mother and child is significant ($p<0.01$) and its sign and interpretation follow that of the bivariate case. In other words, a greater perceived quality of the care environment reduces the risk of stunting. This is the only regression in which the second category of maternal education (Grades 6 to 10) becomes statistically significant, although the size of the effect is not much different.

In R10, the terciles of the services index are added. While the signs remain the same as they were in bivariate analysis, now neither tercile effect is statistically significant. The effects of the maternal education variables are not much changed after the introduction of the services index.

In the complete model, R11, birth weight, respiratory and eyes & ears symptoms, the relationship between the mother and child, birth order, the vaccination score, the fifth level of the occupation score and the second tercile of the services index are statistically significant determinants of stunting. Of these, birth weight, parental occupation, the mother-child relationship score, and the vaccination score are protective against stunting, while higher birth order and eyes & ears symptoms increase the risk of stunting. Counter-intuitively, respiratory symptoms and the second services tercile are also positively correlated with the risk of stunting. The maternal education categories are no longer significant and the size of the effects have fallen substantially (from -0.13 to -0.051 and from -0.212 to -0.062), suggesting that in the complete model we have explained away much of the effect of mother's education (in other words, we have accounted for many of the pathways through which mother's education operates).

6. DISCUSSION

The preceding results provide some insight into the mechanisms through which maternal education affects the probability of stunting in children. Much of the effect of maternal education on stunting is due to its correlation with SES, and indeed only the addition of the variable group consisting of an asset index and a parental occupation index rendered the coefficients on maternal education insignificant. Table 4 summarises the proportion of the maternal education effect that is explained by the various determinant groups. For illustration, the first column reproduces the marginal effect on the third category of mother's education (Grade 11 or more) in all the models that were estimated. The second column contains the proportionate change in the maternal education effect for each model, relative to the effect of mother's education when only the baseline variables were included (we follow Frost et al. (2005) in this exercise).

The size of the effect of the highest maternal education category on stunting declines by 20% when birth weight is added. To the extent that birth weight and HAZ at age 2 capture the same information about the child's growth, this decline could perhaps be interpreted to mean that the effect of maternal education can be divided into a prenatal effect (20%) and a postnatal effect up to 2 years (80%). This implies that education improves the ability of the mother to foster a healthy environment for the child's growth in the womb.

By far the largest proportional decline in the maternal education effect on stunting is caused by the introduction of the SES variables, which cause a reduction of 52%. The dramatic effect of SES suggests that more than half of the maternal education effect on stunting that we observe is due to the child health and growth benefits that accompany increased wealth and occupational standing. Both the asset index (at the third tercile) and the occupation index (at the third, fourth and fifth categories) are significant simultaneously in R4 suggesting that, although highly correlated, the two factors are also influential in determining growth independently of each other. This inference is supported by the observation that only the two together (and not one in isolation) nullified the significance of maternal education (regressions not shown). That only the higher categories of each index are significant means that there is little notable difference in height between children in the lowest SES categories but that large benefits accrue at the higher end of the distribution.

The other groups of variables that cause a notable, but still relatively small, reduction in the effect of maternal education on stunting are reproductive behaviour (14%), services (6%) and feeding (5%). The effect of reproductive behaviour on maternal education can be unpacked by looking at the group's variables. Mother's age when the child was born is a crucial addition in avoiding omitted variable bias. Since a more educated mother is likely to be older when she gives birth, the coefficient and significance of maternal education could merely be a reflection of the improved growth outcomes of children born to older mothers. The other significant variable in this group, birth order, is a proxy for whether higher education leads to increased reproductive autonomy and contraceptive use by the mother, resulting in the fewer children and decreasing intra-household resource competition. The positive and significant coefficient on birth order shows that the risk of stunting increases with the number of children the mother has. The accompanying drop in the coefficient for maternal education adds some support to the hypothesis that maternal education reduces the risk of stunting by affecting the reproductive behaviours of women.

Services are more likely to be a covariate of maternal education than a pathway through which it affects stunting. While in the individual regression (R10), the principal components services index is not a significant determinant itself, it causes the education coefficient to decrease by 6%. This could be because mothers with more education are also more likely to live in wealthy areas and have access to essential services. If the SES variables are also included as regressors in the R10 specification, the addition of services has a negligible effect on the coefficients for maternal education, and the SES variables themselves do not change much either (regression not shown).

The influence of the feeding variables on the maternal education effect on stunting is small (5%), but nonetheless suggests that feeding behaviour could be a pathway if increased education leads mothers to adopt better practices in their breastfeeding and provision of complementary foods. While the multivariate regressions did not provide evidence that the feeding variables were significant in capturing differences in growth outcomes, it may be the case that they are capturing some small change in the feeding behaviour of educated mothers that is relevant to growth.

Other factors (attitude, disease and caregiver characteristics) lead to small reductions (of below 4%) in the coefficient for maternal education. However, even though they were not identified as pathways through which mother's education affects stunting, some of these same variables were significant in the final regression specification, suggesting they are important determinants of stunting independent of mother's education. The number of vaccinations the child had, our proxy for attitudes to modern healthcare, was strongly and significantly protective against stunting, potentially capturing that more engaged and proactive mothers (regardless of education) help reduce the risk of stunting in their children. Similarly, the interviewer's positive perception of the mother and child's relationship and wellbeing was negatively correlated with stunting but it did not change the effect of mother's education on stunting – in other words, the care environment independently impacts the child's growth, but it is not a pathway through which mother's education operates. Although some of the symptoms indices introduced in the diseases group were significant, the coefficient on maternal education decreased by only 3.8%. This suggests that maternal education does not necessarily affect the likelihood that a child will contract an illness, but that the illness itself can affect growth potential.

The complete model reduces the maternal education effect on stunting by 71%. While SES was responsible for most of this fall, the antenatal effects captured through birth weight and the effects of reproductive behaviour are still important in explaining the impact of maternal education on child stunting. The fact that the addition of the full set of variables in the final regression renders the maternal education variables insignificant and reduces the magnitude of the effects substantially, suggests that we are 'explaining' most of the pathways through which maternal education acts to protect stunting with our data. Similarly, while higher SES was found to be one of the main pathways through which maternal education affects stunting, it is important to note that almost none of the SES variables are significant in the complete model. This indicates that we have to a large extent also 'explained' the pathways through which SES operates, although less so for the most well-off group, given that the highest parental occupation dummy is still significant in the complete model.

Our findings with regard to the pathways from maternal education to stunting are largely similar to those of Frost et al. (2005). Their complete model contains fewer pathway groups than ours and reduces the maternal education coefficient by 59%. The pathways that explain most of the effect of

maternal education are SES, attitudes towards medicine, general health knowledge (not included in our models) and reproductive behaviour, causing the coefficient to decrease by 39.6%, 22.3%, 14.8% and 11% respectively. The magnitude of the reduction in reaction to the inclusion of SES is smaller than in our model, but the pathway shares the characteristic of explaining far more of the maternal education effect than any other group. The reproductive behaviour pathway's effect is similar to that in our model, while the attitude effect they find is far more substantial than ours. As much as there are very strong similarities between the variable specifications and methodologies of the two studies, the observed differences could reflect real differences in the pathways through which maternal education affects stunting between the South African and Bolivian contexts, and differences due to the different properties of the data collected and proxies used for a particular pathway. Having said this, the similarity across the two studies' results is notable.

This section summarised the relative strength of the various determinant groupings in explaining the maternal education effect on stunting. However, an important cautionary note is that the relative strength of the various pathways is based on the multivariate regression results, which in turn are affected by the quality of the data we have. It is possible that where variables were found to be weak predictors of stunting, or insignificant pathways through which mother's education affects stunting, we were unable to identify effects because our variables were not well-measured or were sub-optimal proxies for the constructs of interest. We will return to this point below in the concluding section.

7. CONCLUDING REMARKS

In this research report, we examined the causes of stunting at age 2 using as comprehensive an empirical model as the data from Birth to Twenty would allow. The results from the final multivariate analysis suggest that, controlling for all other factors, higher birth weight, being in the highest SES category (as reflected by the parents' joint occupational status), the mother's positive attitude towards or engagement with modern healthcare (captured by the vaccination score), and a better quality care environment, are all protective against stunting. Higher birth order and having experienced some symptoms of illness (ears and eyes) increase the risk of stunting.

While mother's education was no longer significant in the final regression when all determinant groups were included together, it is nonetheless an important distal factor in determining stunting and we were able to show in this research report the pathways through which it operates. The largest mediating effect is through SES, as measured by an asset index and parents' occupation, with other important pathways being the antenatal environment (as reflected in the child's weight at birth) and the mother's reproductive behaviour. In other words, our results suggest that mother's education affects stunting predominantly through better intrauterine growth among children with more educated mothers, the greater access to resources in these households, and the fact that more educated mothers are generally older and have fewer children.

Overall, many of the factors that were protective against stunting in the final analysis, whether they operated through maternal education or not, were related to the mother's involvement in the

child's life. This reinforces the idea that to protect children, mothers need to be supported and empowered in providing care.

Despite producing what we think are important findings, there are some clear limitations to this work. First and foremost, there were a number of variables, particularly the ones representing proximate causes, such as the duration of breastfeeding, dietary diversity and some of the symptoms of illness (especially the gastrointestinal ones), which were found not to significantly affect stunting. Given that the distal factors were expected to operate through these more proximal factors, this finding was surprising. It is hard to believe that appropriate breastfeeding and complementary feeding, and fewer cases of gastrointestinal illness, would not affect child growth. This implies that it is more likely our measures of these variables that are problematic. In discussions with members of the Birth to Twenty research unit, there appeared to be a general feeling that information on these variables is notoriously hard to collect through questionnaires conducted at discrete points in time. Further, these measures may be more prone to recall bias than, for example, questions related to the number of assets in the household or the number of vaccinations received (where there is physical evidence/record). In collecting information on dietary diversity, interviewer/interviewee fatigue could also play a role in the quality of the data collected. In future data collection efforts, alternative methods of questioning would need to be explored, and ideally information would need to be collected more frequently over the first two years of the child's life. For example, to capture dietary diversity adequately, introducing a more frequent 48-hour food diary, either in place of or in addition to a (shorter) food frequency questionnaire may prove useful.

There are also other important pieces of information that are missing from the data. A key set of variables not sufficiently covered in Birth to Twenty in the first two years is WASH. The quality of water available to the household (rather than just access), as well as sanitation and hygiene practices (such as regular handwashing by the caregiver and child, appropriate preparation and storage of food, etc) are likely to play an important role in reducing illness and promoting child growth. This group of variables would also likely act as an important pathway through which mother's education would affect stunting.

It is also useful to consider some of the areas where data collection efforts in Birth to Twenty should be replicated. Despite concerns around the quality of some of the variables, Birth to Twenty remains unique in its attempt to capture information on so many different aspects of the child's life. This is crucial to being able to estimate a comprehensive causal model of a particular phenomenon. Particularly interesting for our purposes was the attempt to collect information on the quality of the child's care environment and the caregiver's involvement in the child's life, information which is generally not collected in larger household surveys. The quality of the care received is an important determinant of child outcomes (as we also showed in this work), but in many studies it is simply omitted and treated as an 'unobservable'.

Finally, while the Birth to Twenty study is unique in providing detailed longitudinal data on the child's health and environment in an urban context, the data on the early childhood period are now over twenty years old and much has changed in South Africa since the early 1990s. One of the key policy interventions in the post-apartheid period has been the provision of the Child Support Grant (CSG), which puts resources directly in the hands of caregivers, predominantly mothers (and grandmothers). Other positive changes have included increased provision of education and

healthcare, wider access to electricity, sanitation and water, and the rollout of antiretrovirals. These policies may have helped families in caring and providing for their children. Nonetheless, further study is warranted, as these policies have clearly not been sufficient, given that a high prevalence of child stunting is recorded even in the most recent national datasets. The value of the grant may be too low to have a substantial enough impact, or it may be that simply providing cash grants without also supporting some of the other factors protective against stunting is not effective in reducing child malnutrition. Being able to repeat this analysis on the causes of stunting using more current data would tell us which of the various factors we have outlined above are still important in determining poor nutritional outcomes in young children. Furthermore, it would be useful to be able to compare causal factors in urban and rural areas, especially seeing as the prevalence of stunting is generally higher in rural areas.

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FIGURES

Figure 1: UNICEF framework on causes and consequences of maternal and child malnutrition (reproduced from Black et al. 2008)
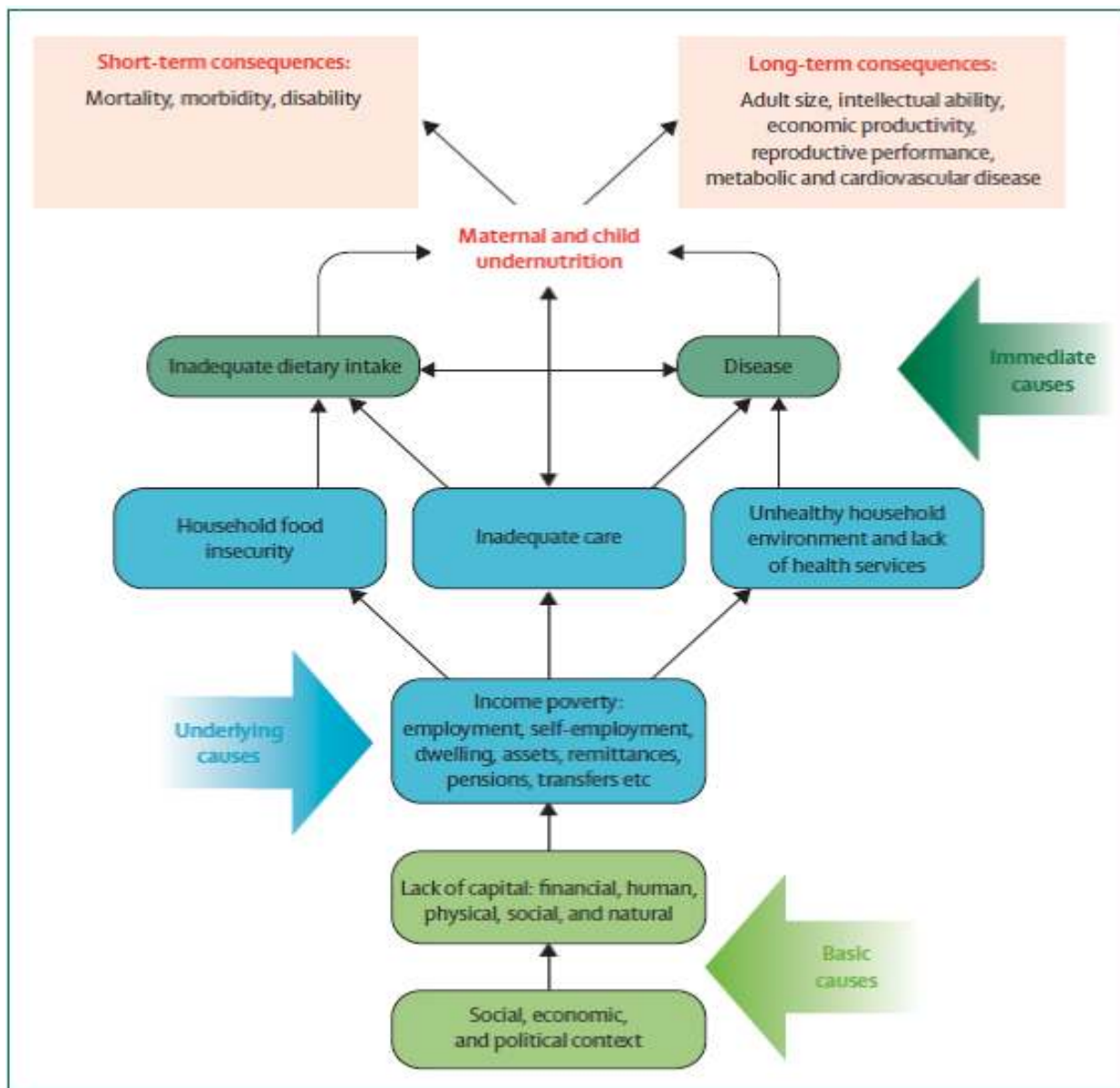
Figure 2: Multi-level conceptual model of the determinants of stunting (reproduced from Fenske et al. 2013)
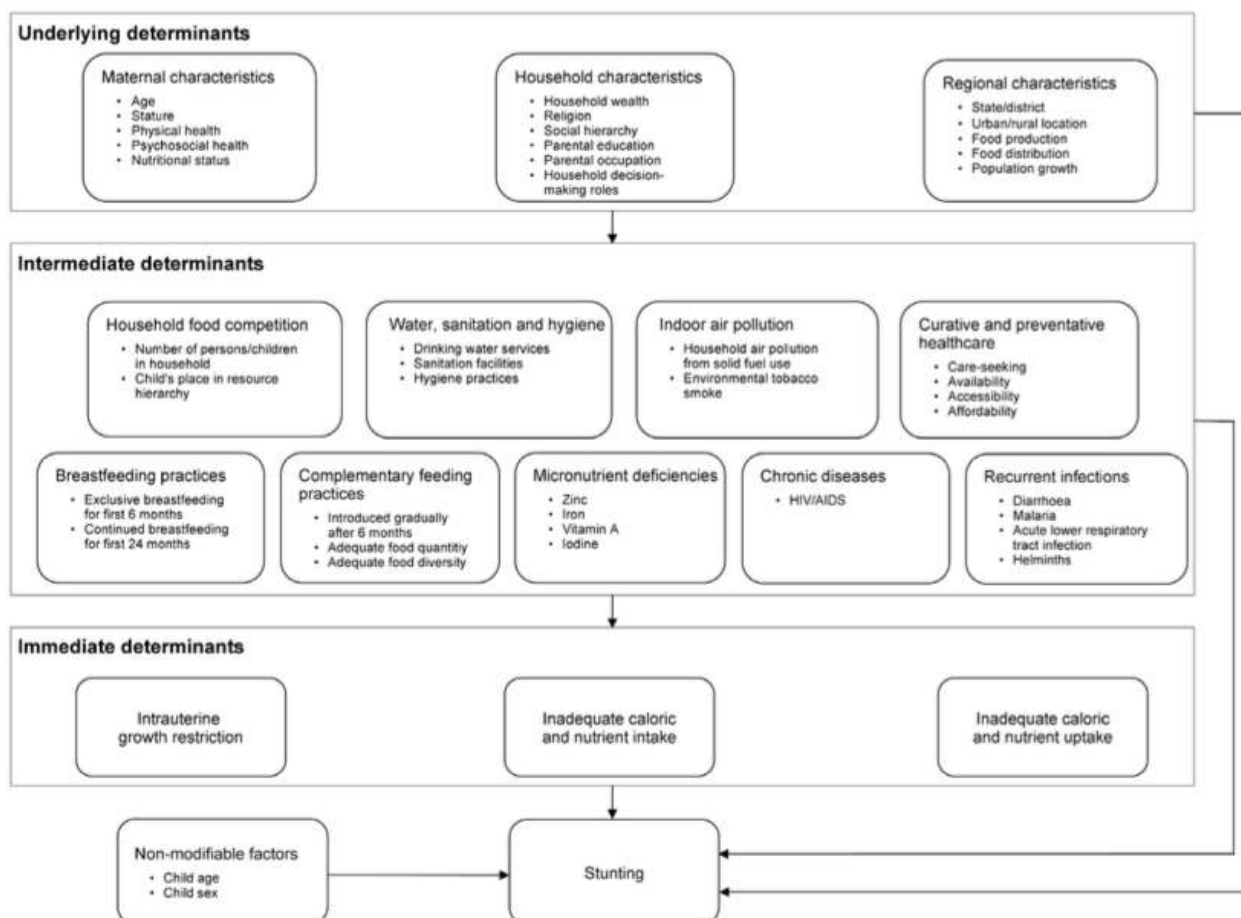
Figure 3: WHO conceptual framework on childhood stunting with an emphasis on complementary feeding (reproduced from Stewart et al. 2013)
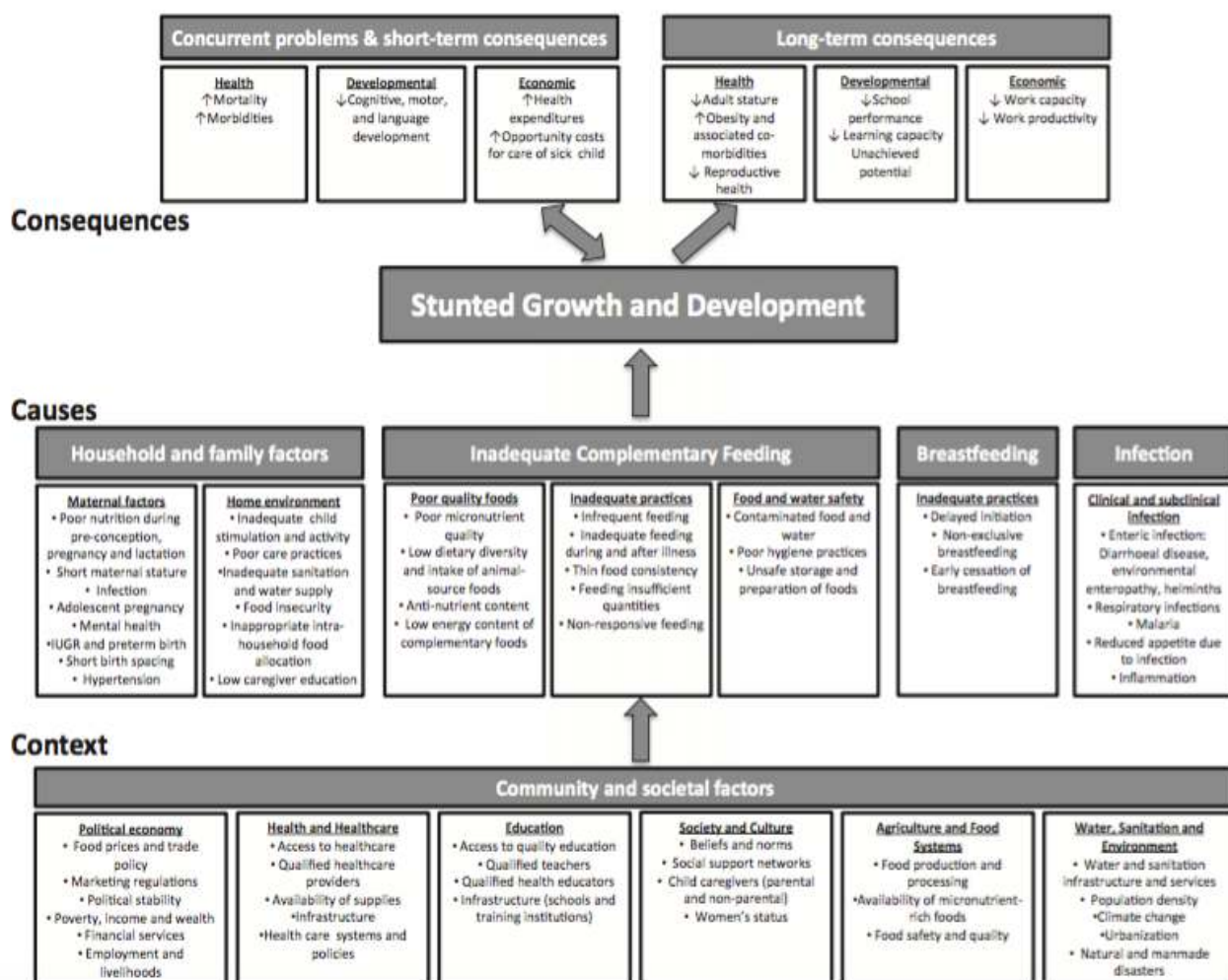
TABLES

Table 1: Descriptive statistics

	AGE COLLECTED	SAMPLE SIZE / FREQUENCY	MEAN / PROPORTION	STD DEV	RANGE
DEPENDENT VARIABLES					
Stunting (HAZ<-2)	2y	1,805	.2049	.4038	0 - 1
HAZ	2y	1,805	-1.16	1.12	-7.34 - 3.55
EXPLANATORY VARIABLES BASELINE					
Female	6m, 1y, 2y	3,273	0.5130	.4999	0 - 1
MATERNAL EDUCATION					
Years of Education	antenatal - 2y	2,932	9.56	3.00	0 - 14
Gr 5 or less	antenatal - 2y	247	.0840		
Gr 6 – 10	antenatal - 2y	1,464	.4993		
Gr 11 or more	antenatal - 2y	1,221	.4164		
BIRTH WEIGHT					
Birth Weight	0m	3,267	3070.67	512.84	1000 - 4920
Birth weight z-score	0m	3,267	-.53	1.16	-5.35 - 3.23
SES					
Asset Index	2y	1,823	2.80	1.43	0 - 5
Crowding	antenatal - 2y	2,649	3.25	1.63	0.44 - 19
PCA Asset Index	2y	1,791	0	1.56	-4.41 - 2.95
Occupation Score	antenatal - 2y	3,222	1.89	1.29	0 - 5
FEEDING					
Duration of BF	6m, 1y, 2y	2,516	10.63	9.04	0 - 37
BF 0 months	6m, 1y, 2y	203	.0807		
BF up to 3 months	6m, 1y, 2y	672	.2671		
BF >3 months	6m, 1y, 2y	1,641	.6522		
FVS	1y	1,546	33.78	11.11	3 - 104
ATTITUDES					
Vaccinations <2	1y	396	.1788		
Vaccinations 3-4	1y	1,151	.5196		
All Vaccinations	1y	668	.3016		
DISEASES					
Resp Symptoms	6m, 1y, 2y	2,848	2.08	1.44	0 - 4
PCA Resp Symptoms	6m, 1y, 2y	2,848	0	1.51	-2.20 - 1.94
Eyes & Ears Symptoms	6m, 1y, 2y	2,877	.32	.54	0 - 2
PCA Eyes & Ears Symptoms	6m, 1y, 2y	2,877	0	1.07	-0.61 - 3.63
Gastro Symptoms	6m, 1y, 2y	2,870	.68	.86	0 - 3
PCA Gastro Symptoms	6m, 1y, 2y	2,870	0	1.22	-.96 - 3.34
REPRODUCTIVE					
Mother's Age	0m	3,271	25.97	6.08	13 - 48
Birth Order	antenatal - 2y	3,273	2.12	1.07	1 - 4
Birth Spacing	2y	1,801	.0666	.2475	0 - 1
CAREGIVER					
Mother Principal Caregiver	2y	1,838	.6202	.4855	0 - 1
Mother-child relationship	2y	1,670	4.66	2.07	0 - 6
PCA Mother-child relationship	2y	1,670	0	2.06	-4.65 - 1.29
SERVICES					
Services Index	0m - 2y	2,709	1.55	.94	0 - 3
PCA Services Index	0m - 2y	2,709	0	1.33	-1.74 - 2.11

Notes: The sample size reflects the total number of Birth to Twenty respondents with data recorded for each variable.

The Mean/Proportion column shows the mean of variables for continuous variables and the proportion for binary/categorical variables

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Table 2: Bivariate regressions for HAZ and stunting

	SAMPLE SIZE	CONTINUOUS HAZ SCORE	BINARY STUNTED VARIABLE
BASELINE			
Female	1,805	.2467***	-.0747***
MATERNAL EDUCATION			
Gr 6 – 10	1,736	.3228***	-.0832*
Gr 11 or more	1,736	.6300***	-.1620***
BIRTH WEIGHT			
Birth weight z-score	1,801	.2707***	-.0674***
SES			
PCA Asset Index T2	1,457	.2110***	-.0667**
PCA Asset Index T3	1,457	.5799***	-.1420***
Occupation Score	1,781	.1496***	-.0437***
FEEDING			
FVS T2	1,036	.0022	-.0570*
FVS T3	1,036	.0144	-.0489
BF up to 3 months	1,607	.0611	.0016
BF >3 months	1,607	-.1938	.0657
ATTITUDES			
Vaccination Score	1,469	-.0278	-.0253
DISEASES			
PCA Resp Symptoms	1,711	.0059	-.0066
PCA Eyes & Ears Symptoms	1,728	-.0693***	.0234***
PCA Gastro Symptoms	1,725	-.0303	.0196
REPRODUCTIVE			
Mother's Age	1,804	.0071*	-.0021
Birth Order	1,805	-.0601**	.0153*
Birth Spacing	1,462	-.2196*	.0635
CAREGIVER			
Mother Principal Caregiver	1,497	-.0271	-.0178
PCA Mother-child relationship	1,377	.0622***	-.0188***
SERVICES			
PCA Services Index T2	1,586	.0846	.0148
PCA Services Index T3	1,586	.3289***	-.0752***

Notes: The sample size in each regression is the number of Birth to Twenty respondents who had both HAZ data and regressor data recorded. For the Continuous HAZ Score column, results are from bivariate OLS regressions. For the Binary Stunted Variable column results are AMEs (Average Marginal Effects) calculated after bivariate Probit estimation. * p<=0.1 ** p<=0.05 *** p<=0.01

Table 3: Multivariate regression results for stunting at age 2 (with restricted sample)

	BASELINE	MATERNAL EDUCATION	BIRTH WEIGHT	SES	FEEDING	ATTITUDE	DISEASE	REPRODUCTIVE	CARE	SERVICES	COMPLETE
	R1	R2	R3	R4	R5	R6	R7	R8	R9	R10	R11
Female Dummy	-0.052	-0.060*	-0.051*	-0.062**	-0.058*	-0.061*	-0.054*	-0.060*	-0.053*	-0.061*	-0.033
	(0.032)	(0.032)	(0.031)	(0.031)	(0.032)	(0.032)	(0.031)	(0.032)	(0.031)	(0.032)	(0.030)
Gr 6 – 10		-0.130	-0.106	-0.074	-0.122	-0.126	-0.123	-0.131	-0.140*	-0.128	-0.051
		(0.085)	(0.081)	(0.076)	(0.084)	(0.085)	(0.083)	(0.087)	(0.084)	(0.084)	(0.074)
Gr 11 or more		-0.212**	-0.169**	-0.102	-0.201**	-0.209**	-0.204**	-0.183**	-0.207**	-0.200**	-0.062
		(0.084)	(0.080)	(0.078)	(0.083)	(0.084)	(0.083)	(0.089)	(0.084)	(0.084)	(0.078)
Birth weight z-score			-0.083***								-0.080***
			(0.013)								(0.013)
PCA Asset T2				-0.066							-0.043
				(0.040)							(0.039)
PCA Asset T3				-0.101**							-0.072
				(0.043)							(0.044)
Occupation Sc 1				0.028							0.068
				(0.055)							(0.052)
Occupation Sc 2				-0.049							0.003
				(0.047)							(0.044)
Occupation Sc 3				-0.126**							-0.062
				(0.057)							(0.057)
Occupation Sc 4				-0.156***							-0.080
				(0.055)							(0.060)
Occupation Sc 5				-0.202**							-0.152*
				(0.081)							(0.083)
FVS T2					-0.025						-0.027
					(0.039)						(0.037)
FVS T3					-0.033						-0.031
					(0.039)						(0.037)
BF up to 3 months					-0.012						0.026

					(0.072)						(0.066)
BF >3 months					0.074						0.086
					(0.068)						(0.061)
Vaccination Score						-0.039					-0.042*
						(0.024)					(0.023)
PCA Resp Symp							-0.036***				-0.035***
							(0.012)				(0.012)
PCA Eyes & Ears							0.040***				0.038***
							(0.014)				(0.013)
PCA Gastro Symp							0.003				0.008
							(0.013)				(0.012)
Mother's age								-0.035*			-0.014
								(0.021)			(0.021)
Mother's age²								0.000			0.000
								(0.000)			(0.000)
Birth Order								0.049**			0.038*
								(0.021)			(0.021)
Birth Spacing								0.047			0.064
								(0.066)			(0.061)
Mother-child relationship									-0.023***		-0.014**
									(0.007)		(0.007)
Principal Caregiver									-0.004		0.001
									(0.033)		(0.031)
PCA Services T2										0.048	0.092*
										(0.054)	(0.051)
PCA Services T3										-0.048	0.052
										(0.036)	(0.042)
N	691	691	691	691	691	691	691	691	691	691	691

Notes: The sample size in each regression is restricted to Birth to Twenty participants who had data on HAZ and all regressors in the Complete model.
Results are AMEs (Average Marginal Effects) calculated after Probit estimation.
Omitted categories for the categorical variables are Grade 5 or less, PCA Asset tercile 1, Occupation score 0, FVS tercile 1 and Service index tercile 1.
* p<=0.1 ** p<=0.05 *** p<=0.01

Table 4: Proportion of maternal education effect explained by various pathways

	EFFECT OF GRADE 11 OR HIGHER (REPRODUCED FROM REGRESSIONS 2-11 IN TABLE 3)[1]	PROPORTION OF THE EFFECT EXPLAINED BY:[2]
Maternal education	-0.212	-
Birth Weight	-0.169	0.2028
SES	-0.102	0.5189
Feeding	-0.201	0.0519
Attitude	-0.209	0.0142
Disease	-0.204	0.0377
Reproductive Behaviour	-0.183	0.1368
Caregiver Characteristics	-0.207	0.0236
Services	-0.200	0.0566
Complete	-0.062	0.7075

Notes: This table is based on a similar one created by Frost et al. (2005: 404).

[1] The first column reproduces the average marginal effects (AMEs) from the multivariate regression results in Table 3.

[2] Figures in the second column are equal to the reduction in the effect divided by the original effect of -0.212. So, for example, the value of 0.2028 for the birth weight pathway is calculated as (0.212 - 0.169)/0.212, and indicates that 20.28% of the maternal education effect on stunting can be explained by the birth weight pathway.

APPENDICES

A: SUMMARY OF LITERATURE INVESTIGATING DETERMINANTS OF STUNTING IN SOUTH AFRICA

Table A1: An overview of papers assessing determinants of child stunting/HAZ in South Africa (in order of reference in the text)

	REGION, DATE	DATA SOURCE	CAUSES EMPHASISED	SAMPLE	CONCEPTUAL FRAMEWORK	ESTIMATION TECHNIQUE	MODEL/SELECTION OF REGRESSORS	DEPENDENT VARIABLE(S)	POTENTIAL REGRESSORS	SIGNIFICANT DETERMINANTS (<10%)
Jones et al. 2008	Soweto, Johannesburg 1990-1992	Birth to Twenty Cohort Study N=450 at 1 year or N=401 at 2 years	The role of SES in determining stunting, SA compared to the Philippines.	Children with HAZ scores recorded at 1 *or* 2 years, and other information recorded at and before this date.	The effect of SES on stunting can be understood either in itself or as influenced and/or mediated by other factors.	Baseline model had a logistic regression of stunting on the SES index, and then adjusted models added maternal height, sex and parity. Further models used separate socio-economic variables instead of an index.	Principal components analysis was used to create an SES index.	Binary stunted variable at 1 or 2 years (HAZ score < -2)	Socio-economic variables (index): maternal education, fridge and TV ownership, water and toilet facilities. Maternal height, sex, primaparous	Maternal height (year 1 & 2) SES index or individual variables not significant in baseline or adjusted model.
Willey et. al. 2009	Soweto, Johannesburg 1990-1992	Birth to Twenty Cohort Study N=1,182	Early life hh SES and social support	Children with HAZ scores recorded at 3, 6, 12 or 24 months, and other information recorded at and before this date.	Proximate influences (confounded or direct effects) and distal influences (direct, confounded or mediated by proximate effect).	Two regression models, one identifying demographic (proximate) measures and the other identifying SES and social support, before a combined model built using the framework	Forward stepwise logistical regression to identify potential regressors. Strongest association added to model first. Those not significantly adding to stepwise model not retained	Binary stunted variable (HAZ score < -2) at 3, 6, 12 or 24 months (or the latest of all available measurements)	Birth weight, gestational age, gender, maternal age at delivery, ethnicity, parental education and employment, hh water, sanitation and electricity supply, assets, cooking fuel, health insurance, employment of domestic worker, dwelling characteristics, marital status, partner support, social organisations and networks.	Positive: father has secondary school, mother is employed, hh with domestic worker Negative: Male, low birthweight Effects of SES didn't seem to be mediated through proximate causes.

Slemming et. al. 2016	Soweto, Johannesburg 1990-1992	Birth to Twenty Cohort Study N=1,098	Pregnancy events, birth characteristics, and infant risk exposure. A broad look emphasising both proximate and distal factors.	Children with HAZ scores recorded at 2 years, and other information recorded at and before this date.	Stresses the importance of both proximal and distal determinants	Sex-stratified multivariate logistic regression models.	Stepwise selection with bivariate regressions determining unadjusted association. Principal components analysis used to create SES index based on hh assets	Binary stunted variable at age 2 (HAZ score < -2)	Maternal factors: pregnancy wantedness, prenatal stress, tobacco and alcohol use, age at birth, education, marital status, depression, hh overcrowding & SES (asset index). Controls: maternal height, infant birth weight, gestational age, breastfeeding duration,	Univariate analysis: male, black or coloured, low SES, overcrowding, low maternal educ & maternal age, single motherhood, low birthweight & gestational age & breastfeeding duration all increased risk of stunting. Multivariate analysis: higher SES protective for males, maternal educ for females, birthweight for both.
Timaeus 2012	KwaZulu-Natal, South Africa 1993-2004	KIDS panel study N=4,918 (cross-section) N=1,155 (dynamic model)	The link between stunting and overweight and the effect of poor measurement of height.	Cohort of primary-school aged children with height measurements available across waves			First all children with non-missing data for waves 1998 or 2004 were treated as a cross section. Then a dynamic model was applied which included a lagged HAZ score variable.		A continuous HAZ score for 1998 and 2004.	Wave, female, age, metro, age by metro, Indian, mother's educ, pc expenditure,	In the cross-section, wave, age, Indian, upper primary dummy and expenditure. In the dynamic model, lagged HAZ, wave, age, metro, age by metro, Indian, matric, expenditure
Burgard 2002	South Africa 1993	PSLSD nationally representative hh survey. N=3,156	The association between race and stunting in SA and Brazil, and the modelling of the racial inequality in the distribution of socioeconomic resources.	Children aged 0-59 months with non-missing information for the variables of interest.	Mosley and Chen's 1984 framework. Socio-economic and demographic characteristics can only affect stunting through proximate (biological and behavioural) determinants. However, these proximate determinants are often endogenous and suffer from measurement error so both are included in models.	Generalised estimating equation logistic regressions are used. First stunting is regressed only on mother's race to find the unadjusted effect of race. Then "exogenous" background characteristics of child, hh and community added (model 2), then potentially endogenous predictors added (model 3).		Binary stunted variable (HAZ score < -2)	Model 1: mother's race Model 2: child's age, sex, parents' education, hh wealth, place of residence, province Model 3: age at weaning, mother's age, toilet and piped water in house, magisterial district female educ attainment and male employment	Mother's race, child's age and sex, parents' educ, hh wealth, some provinces (only model 2) age at weaning (only model 3). When background characteristics have already been controlled for, the only significant proximate factor was age at weaning.

May & Timaeus 2014	South Africa 2008	NIDS nationally representative panel survey N=+-2164	The connection between inequality of income and inequality of health, and specifically between income and malnutrition.	Children aged 0-59 months with non-missing information for the variables of interest.	Some evidence exists that wealth and income are no longer strong determinants of stunting. By controlling for other factors, the true determinants of stunting, and whether income is one, can be determined.	A logistic regression of stunted on a range of variables. Heckman selection probits were fitted with interviewer number as a predictor to identify potential selection bias.		Binary stunted variable (HAZ score < -2)	Male, urban, province dummies, race, mother's educ, log pc expenditure, hh size, asset index, housing score, age (months)	Eastern Cape, Free State, Gauteng, Limpopo (+ve vs KZN), Coloured (+ve), Asian (-ve vs African), asset index (-ve) No evidence of bias.
Ardington & Gasealahwe 2012	South Africa 2008-2010	NIDS nationally representative panel survey N=2,934	The effect of a change in hh pension status on children's nutritional status	Children aged 16 and under in wave 1 in hh's with at least one adult aged 50+	Nutritional outcomes depend on family income. Pensions are a significant source, but age and other possibly confounding variables must be controlled for to examine its effect.	First a simple logistic regression of stunted on a few controls and the pension variables is performed. Then the model is expanded to include various other controls. Robust standard errors allowing for correlation in unobservables for individuals from the same PSU are presented.		Binary stunted variable (HAZ score < -2) in models 1 and 2. Continuous HAZ score in model 3. All at wave 2 (2010).	All models: age, quadratic of age of oldest hh member, got pension, lost pension, stunted in wave 1, female, race Model 2 & 3 add: hh size (w1&2), urban (w1&2), assets (w1), hh pc income quintile. Model 3 uses HAZ in w1 instead of stunted.	Got pension, lost pension, previous stunting (all models), white relative to African (model 1), urban (models 2&3), hh pc income quintile 3 (model 2)
Chopra 2003	Hlabisa Health District, KwaZulu-Natal 2002	Community-based cross-sectional survey of households N=848	A broad multi-sectoral look at factors associated with stunting, with an interest in determining the effect of SES and environmental factors as well as proximate ones.	Children aged 3-59 months without severe congenital problems	Socioeconomic variables may determine, directly or indirectly, all variables being studied except for age and gender. Next level comprises environmental variables (partially determined by SES). Third level consists of immediate nutritional causes such as birth weight and feeding beliefs and practices.	First age and gender entered. Then all SES variables added (even those insignificant in bivariate), and so on for each level. Odds ratios presented in multivariate analyses are those from the equation corresponding to the level at which the risk factor was first entered.	Variables with a clear association (after comparing crude to adjusted estimates) for SES were retained, and the same process applied at each level.	Binary stunted variable (HAZ score < -2)	Age, sex Level 1: Migrant father, mother's educ & literacy, proportion income spent on food, material of housing, crowding, death of child in 2 years, distance from clinic Level 2: water source & distance, toilet, electricity Level 3: current breastfeeding & when stopped, intro solids, weaned <4 months, feeding when child sick & recovering	Father present <weekly, mother's educ, traditional material, distance to clinic, toilet presence, breastfed last child, birth weight.

Mamabolo et al. 2005	Central Region of the Limpopo Province 2005	Prospective cohort study of women from their third trimester and their infants. N=162	A broad multi-sectoral look at factors associated with stunting within a South African village.	Children who could be traced aged 3 years.	Nutrition is a product of the interplay of economic, demographic, environmental and cultural factors. Endemic poverty and lack of basic resources form the basis for poor nutritional status.	Multivariate logistic regression based on children divided into four groups: normal, stunted, overweight and both. Factors associated with various nutritional states examined with reference to the normal children.		Binary stunted variable (HAZ score < -2)	Weight, length, ponderal index, HAZ & WHZ scores at birth and 1-year; overweight at 3; maternal age, length, parity, marriage status, educ, occupation, main caregiver; house type, hh size, no. children <5 years; child sees mother daily, father in hh	1 year length, ponderal index, mother is a student, more than 9 in hh
Dukhi et al. 2016	iLembe District, KwaZulu-Natal 2014	Community-based cross-sectional weighted survey of households N=572	A broad multi-sectoral look at factors associated with stunting, and at spatial clustering	Children aged 0-59 months.	Multilevel (4 levels) determinants interact in a model based on Bronfenbrenner's Ecological systems.	Survey-weighted bivariate and multivariate logistic regressions were applied for each independent variable, with a Shapley decomposition approach to quantify relative contributions. Then a generalised structural equation model (SEM) was applied to match the conceptual framework and assess direct and indirect effects.		Binary stunted variable (HAZ score < -2) and severely stunted variable (HAZ score < -3)	Age in months, male, mother educated, hh food security, number and availability of foods, distance to clinic, caregiver income, access to piped water and electricity, regular hh income	Male gender, food insecurity, distance to clinic (unadjusted and adjusted), access to piped water (only unadjusted). SEM model shows how stunting is affected directly by different levels of determinants.

B: CREATING DIETARY QUALITY INDICATORS USING FFQ DATA IN BIRTH TO TWENTY

In exploring the Food Frequency Questionnaire (FFQ) data in Birth to Twenty, we created a range of indicators to represent the quantity, diversity, distribution and quality of the child's diet.[24] The indices most often used to capture dietary diversity are dietary diversity scores, such as the Food and Agriculture Organization's (2011) HDDS (household dietary diversity score) or the World Health Organization's (2010) infant 'Minimum dietary diversity' indicator (here abbreviated to 'WHO child dietary diversity' or WHOCDD), and food variety scores or FVS (see Ruel, 2002, Drescher et al., 2007 and Thornton, 2016). The former can be constructed from a FFQ by placing foods into groups (the HDDS has 12 and the WHOCDD has 7) and then counting how many of the groups were consumed (with a positive frequency in any food item in a group sufficient to capture that group as consumed). The mean HDDS score of 9.3 in Birth to Twenty means that an average child consumed at least one food item in about 9 of the food groups. The latter (the FVS) is simply a count of how many food *items* (as opposed to groups) were consumed of those items that were included in the FFQ. To create these indicators using the FFQ one has to create a dummy consumption variable for each child and choose a minimum frequency which qualifies an item to be classified as consumed (e.g. a child has consumed white bread if they ate it monthly or more often) (Ruel, 2002).

While these indicators capture an important element of a child's diet, diversity, they make no distinction between unhealthy and healthier food items. This failure to incorporate healthiness is especially problematic in the case of the FVS, where the scores will vary based on the idiosyncratic list of food items included in the FFQ (which could favour healthy foods or not in the questionnaire). So, for example, the FVS would increase if a child went from not consuming sweets to consuming sweets monthly. Furthermore, these indicators fail to include all of the information regarding quantity and distribution available in the FFQ. The summation (comparison) of the respective frequencies at which foods are consumed can provide more information about the quantity (distribution) of consumption than can be inferred from dummy consumption variables indicating simply whether a food item was consumed or not. So, for example, these indicators do not capture the difference between a child who eats spinach once a month and a child who eats spinach 2-3 times a week.

We experimented with two ways of incorporating diet quality into a FVS. First, we multiplied each food item by a health factor weight (see FVS health factor in Table B.1 below), reflecting its nutritional content based on the recommended consumption of the group to which it belongs (in a nutrition wheel and food pyramid). These health factor weights were taken from Drescher et al. (2007) and are based on dietary guidelines from the German Nutrition Society (DGE). It could reasonably be argued that these guidelines are not optimal for the developing country context, and that guidelines created specifically for infants would be more appropriate. However, there is a great degree of overlap in what is generally believed to be healthy across countries and ages. Second, we simply excluded from the indicator those foods that are obviously unhealthy (i.e. we weighted them

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[24] See Ruel (2002) for a review of dietary quality indicators.

by 0), leaving us with a count index of foods that are generally considered healthy (see 'FVS broad healthy' in the table).

Then, to expand the scope of our index to cover both quantity and distribution we followed Drescher et al. (2007) in creating a Healthy Food Diversity index (HFD). This involves sorting the food items into 3 main groups, plant foods, animal foods, and fats and oils, with 12 subgroups. The relative shares of each subgroup are then squared and summed to create the Berry Index (BI), an indicator of how evenly distributed an individual's consumption is across the subgroups. The BI is at its maximum for a set level of consumption if items are evenly distributed across subgroups. But to account for differential health benefits of different groups the proportional recommendations in nutrition wheels and pyramids are turned into health factors for each group. The sum of these health factors multiplied by the share of each group yields the health value, which is then multiplied by the BI to yield the final indicator: the HFD. The ability of the HFD to reflect adequate nutrition is verified by the authors who find that the indicator is highly correlated with nutrient supply and a range of biochemical parameters (to a higher degree than the simple count indices described above) (Drescher et al., 2007).

Finally, we created a variable that summed the health factors multiplied by the frequencies (rather than the share of the total consumption) that should be more sensitive to overall quantity and quality considerations (see Frequency*hf in the table). Again, the health factors were taken from Drescher et al. (2007).

Table B1: Bivariate analysis of FFQ indicators and stunting/HAZ

	HAZ Score	Stunted
FVS (broad) T2	.0023	-.0570*
FVS (broad) T3	.0144	-.0489
FVS (broad healthy) T2	.0121	-.0246
FVS (broad healthy) T3	.0386	-.0453
FVS (health factor) T2	-.0508	-.0143
FVS (health factor) T3	.0001	-.0384
HDDS T2	.0226	-.0285
HDDS T3	-.0334	-.0133
WHOCDD T2	-.2082***	.0389
WHOCDD T3	-.0644	.0644
BI	.1129	.0023
HFD T2	-.0101	.0211
HFD T3	-.0597	.0251
Frequency*health factor T2	-.0876	.0261
Frequency*health factor T3	-.1957**	.0512

Notes: For all regressions, n=1,036.
All variables, except for BI, are presented in terciles with the lowest tercile omitted and coefficients and significance presented for T2 and T3.
* p<=0.1 ** p<=0.05 *** p<=0.01

All the FFQ indicators were then divided into terciles for bivariate and multivariate analysis. Surprisingly, as can be seen from Table B.1 below, none of these indicators was consistently significantly associated with either stunting or HAZ in bivariate regressions. The direction of the effect sometimes followed the hypothesised relationship (with a higher score on the dietary indicator linked to a higher HAZ score and lower probability of stunting), and other times it was the inverse of what was expected. Furthermore, none of the coefficients was statistically significant, with the exception of the second tercile of the FVS (which decreases the risk of stunting relative to

T1, as was shown earlier in Table 2 in the main text), the second tercile of the WHOCDD and the third tercile of Frequency*hf (both of which counter-intuitively decrease the HAZ score). The weakness in correlation between any indicator created using the FFQ data and stunting/HAZ, and the contradictory signs, might suggest that the data collection process was not ideal.[25] It is hard to determine what the problems in surveying were. It could have been that the questioning was too vague, that interviewers tended to record an approximately equal number of items for each respondent before tiring of questioning, that respondents themselves suffered from interviewee fatigue, or that the FFQ needs to be performed at multiple time points to adequately capture differences between respondents. In future studies an approach that combines a FFQ with a survey of a child's exact nutritional intake over a shorter period (e.g. a food diary for the previous 24 hours) could yield superior results (for a good example of such a study see Faber, Jogessar & Benade, 2001).

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[25] This supposition is further supported by the fact that the FVS is slightly lower for the higher SES quintiles (not shown here). This disappointing lack of significance is consistent with Gitau's (2009) work. In her Masters research report, she found no significance for either a food variety score or a dietary diversity score in stunting regressions.

C: MULTIVARIATE REGRESSIONS FOR HAZ WITH RESTRICTED SAMPLE SIZE

Table C1: Multivariate regression results for HAZ, with restricted sample size

	BASELINE	MATERNAL EDUCATION	BIRTH WEIGHT	SES	FEEDING	ATTITUDE	DISEASE	REPRODUCTIVE	CARE	SERVICES	COMPLETE
	R1	R2	R3	R4	R5	R6	R7	R8	R9	R10	R11
Female Dummy	0.212** (0.087)	0.231*** (0.087)	0.198** (0.083)	0.234*** (0.086)	0.232*** (0.087)	0.231*** (0.087)	0.203** (0.086)	0.230*** (0.086)	0.208** (0.086)	0.233*** (0.086)	0.148* (0.082)
Gr 6 − 10		0.375* (0.200)	0.309 (0.191)	0.255 (0.198)	0.364* (0.199)	0.370* (0.200)	0.361* (0.198)	0.372* (0.204)	0.387* (0.199)	0.357* (0.199)	0.178 (0.195)
Gr 11 or more		0.620*** (0.200)	0.477** (0.191)	0.352* (0.204)	0.606*** (0.200)	0.618*** (0.200)	0.603*** (0.198)	0.530** (0.211)	0.584*** (0.199)	0.550*** (0.201)	0.176 (0.206)
Birth weight z-score			0.310*** (0.038)								0.302*** (0.037)
PCA Asset T2				0.177 (0.108)							0.112 (0.104)
PCA Asset T3				0.342*** (0.116)							0.180 (0.122)
Occupation Sc 1				-0.054 (0.139)							-0.153 (0.138)
Occupation Sc 2				0.159 (0.121)							0.027 (0.121)
Occupation Sc 3				0.329** (0.162)							0.108 (0.165)
Occupation Sc 4				0.332** (0.161)							0.081 (0.169)
Occupation Sc 5				0.549* (0.290)							0.267 (0.289)
FVS T2					-0.036 (0.106)						-0.058 (0.100)
FVS T3					-0.006 (0.106)						-0.020 (0.101)
BF up to 3 months					0.229 (0.214)						0.078 (0.201)
BF >3 months					-0.038 (0.199)						-0.097 (0.187)
Vaccination Score						0.057 (0.067)					0.090 (0.064)
PCA Resp Symp							0.107*** (0.035)				0.103*** (0.033)
PCA Eyes & Ears							-0.109*** (0.039)				-0.102*** (0.037)
PCA Gastro Symp							-0.054 (0.036)				-0.057* (0.034)
Mother's age								0.099* (0.060)			0.040 (0.059)

Mother's age2								-0.001			-0.000
								(0.001)			(0.001)
Birth Order								-0.151***			-0.134**
								(0.057)			(0.056)
Birth Spacing								-0.249			-0.271
								(0.191)			(0.182)
Mother-child relationship									0.078***		0.053***
									(0.020)		(0.020)
Principal Caregiver									-0.060		-0.087
									(0.089)		(0.084)
PCA Services T2										0.046	-0.079
										(0.138)	(0.131)
PCA Services T3										0.275***	0.034
										(0.101)	(0.110)
Constant	-1.604***	-2.105***	-1.816***	-2.241***	-2.100***	-2.171***	-2.063***	-3.496***	-2.014***	-2.150***	-2.177***
	(0.140)	(0.234)	(0.226)	(0.246)	(0.313)	(0.247)	(0.233)	(0.791)	(0.243)	(0.234)	(0.820)
N	691	691	691	691	691	691	691	691	691	691	691

Notes: The sample size in each regression is restricted to Birth to Twenty participants who had data on HAZ and all regressors in the Complete model.

Results are from OLS estimation.

Omitted categories for the categorical variables are Grade 5 or less, PCA Asset tercile 1, Occupation score 0, FVS tercile 1 and Service index tercile 1.

* p<=0.1 ** p<=0.05 *** p<=0.01

D: MULTIVARIATE REGRESSIONS FOR STUNTING WITHOUT RESTRICTING SAMPLE SIZE

Table D.1 shows how the sample size varies widely between the different model specifications when the sample is not restricted to that of the final, complete specification. It is apparent that the SES and Caregiver variables, in particular, contribute to the reduction in sample size.

The female dummy is now significant ($p<0.01$) in R1, although as in Table 3 earlier the effect disappears in the complete model. The second category of maternal education is significant ($p<0.1$) when introduced in R2, and in a few other regressions. The third level of education follows a similar pattern to the restricted analysis, although it now retains significance at the 10% level when SES variables are introduced (but not in the complete model). Its coefficient decreases in similar ways when other groups are introduced, with the exceptions being that it is now reduced more when Caregiver variables are added and it actually increases when Feeding variables are added. Most of the other explanatory variables exhibit the same signs and very similar levels of significance across models despite the large discrepancies in sample size. This provides some evidence that the results in our multivariate analysis are not significantly affected by selection bias due to the probability of response to any particular question being correlated with a variable of interest in the regressions. Likewise, the smaller sample size is not masking any significant determinants or interactions between variables that would substantially change our interpretation of the results.

Table D1: Multivariate regression results for stunting, with unrestricted sample size

	BASELINE	MATERNAL EDUCATION	BIRTH WEIGHT	SES	FEEDING	ATTITUDE	DISEASE	REPRODUCTIVE	CARE	SERVICES	COMPLETE
	R1	R2	R3	R4	R5	R6	R7	R8	R9	R10	R11
Female Dummy	-0.075***	-0.074***	-0.075***	-0.077***	-0.055**	-0.068***	-0.070***	-0.086***	-0.083***	-0.074***	-0.033
	(0.019)	(0.019)	(0.019)	(0.022)	(0.026)	(0.021)	(0.020)	(0.022)	(0.023)	(0.020)	(0.030)
Gr 6 − 10		-0.089*	-0.082*	-0.046	-0.114	-0.079	-0.085*	-0.087	-0.078	-0.087*	-0.051
		(0.048)	(0.046)	(0.051)	(0.069)	(0.053)	(0.048)	(0.054)	(0.055)	(0.049)	(0.074)
Gr 11 or more		-0.167***	-0.149***	-0.089*	-0.211***	-0.163***	-0.163***	-0.147***	-0.147***	-0.158***	-0.062
		(0.048)	(0.046)	(0.052)	(0.068)	(0.052)	(0.048)	(0.056)	(0.055)	(0.049)	(0.078)
Birth weight z-score			-0.066***								-0.080***
			(0.008)								(0.013)
PCA Asset T2				-0.044							-0.043
				(0.028)							(0.039)
PCA Asset T3				-0.087***							-0.072
				(0.030)							(0.044)
Occupation Sc 1				0.011							0.068
				(0.038)							(0.052)
Occupation Sc 2				-0.055*							0.003
				(0.032)							(0.044)
Occupation Sc 3				-0.125***							-0.062
				(0.040)							(0.057)
Occupation Sc 4				-0.109***							-0.080
				(0.041)							(0.060)
Occupation Sc 5				-0.109							-0.152*
				(0.079)							(0.083)
FVS T2					-0.054*						-0.027
					(0.032)						(0.037)
FVS T3					-0.039						-0.031
					(0.033)						(0.037)
BF up to 3 months					-0.026						0.026
					(0.060)						(0.066)
BF >3 months					0.065						0.086
					(0.058)						(0.061)
Vaccination Score						-0.022					-0.042*
						(0.015)					(0.023)
PCA Resp Symp							-0.011				-0.035***
							(0.007)				(0.012)
PCA Eyes & Ears							0.026***				0.038***
							(0.009)				(0.013)
PCA Gastro Symp							0.003				0.008
							(0.008)				(0.012)
Mother's age								-0.029**			-0.014
								(0.014)			(0.021)
Mother's age2								0.000			0.000
								(0.000)			(0.000)
Birth Order								0.047***			0.038*

								(0.015)			(0.021)
Birth Spacing								0.048			0.064
								(0.044)			(0.061)
Mother-child relationship									-0.015***		-0.014**
									(0.005)		(0.007)
Principal Caregiver									-0.026		0.001
									(0.023)		(0.031)
PCA Services T2										0.027	0.092*
										(0.037)	(0.051)
PCA Services T3										-0.055**	0.052
										(0.023)	(0.042)
N	1805	1736	1732	1448	938	1467	1697	1458	1374	1574	691

Notes: The sample size in each regression is the sample of Birth to Twenty participants who had HAZ data and regressor data for that particular model.

Results are AMEs (Average Marginal Effects) calculated after Probit estimation.

Omitted categories for the categorical variables are Grade 5 or less, PCA Asset tercile 1, Occupation score 0, FVS tercile 1 and Service index tercile 1.

* p<=0.1 ** p<=0.05 *** p<=0.01