Public Health Nutrition: 21(10), 1810–1818 doi:10.1017/S1368980018000125

Estimating the pathways through which maternal education affects stunting: evidence from an urban cohort in South Africa

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Submitted 6 June 2017: Final revision received 5 December 2017: Accepted 21 December 2017: First published online 19 February 2018

Abstract

Objective: To estimate the determinants of stunting using rich data from a birth cohort study from urban South Africa and to examine the various mechanisms, both proximate and distal, through which maternal education affects stunting.
Design: Multivariate regression analysis using birth cohort data, where the outcome variable was stunting at age 2 years, and multiple mediator analysis to identify pathways from maternal education to stunting.
Setting: South Africa's largest metropolitan area, Soweto-Johannesburg.
Subjects: Participants of Birth to Twenty Plus, a longitudinal cohort study of children born in 1990 (*n* 691).
Results: In multivariate analysis, the birth weight *Z*-score (−0·084; $P<0·001$; 95 % CI −0·11, −0·06), the mother's openness towards modern health care, captured by a vaccination score (−0·05; $P=0·04$; 95 % CI −0·10, −0·00), and a better-quality care environment (−0·015; $P=0·04$; 95 % CI −0·03, −0·00) were found to be negatively associated with stunting. Having experienced symptoms of illness related to ears and eyes increased the risk of stunting (0·038; $P=0·01$; 95 % CI 0·01, 0·07). Results of the mediation analysis showed that maternal education had an indirect effect on stunting largely through socio-economic status and the antenatal environment (measured by the birth weight *Z*-score).
Conclusions: 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 contribution to the child's life. This reinforces the idea that to minimise stunting, enhanced antenatal and postnatal services to better support and empower mothers may be important.

Keywords
Determinants of stunting
Maternal education
Multiple mediator analysis
South Africa

Globally, stunting in childhood remains a substantial public health concern, with an estimated 156 million children under 5 years of age stunted in 2015[1]. In South Africa, the prevalence is particularly high for a country of middle-income status[2], with the latest estimates from 2012 suggesting that 27 % of children younger than 3 years of age are stunted[3]. South Africa was recently identified as one of thirty-four countries responsible for 90 % of the global burden of child malnutrition[4]. The persistently high prevalence in South Africa is surprising given the country's level of economic development and the introduction over the last two decades of a child support grant and free health care for mothers and babies.

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[5–7]. Research in South Africa has similarly found that poor nutrition in early childhood is associated with lower levels of cognitive function and worse schooling outcomes[6,8–11].

Given the high and persistent prevalence of stunting in South Africa[12] and a growing body of research documenting the negative consequences, there is surprisingly a rather limited South African literature investigating the causes of stunting in young children. One of the main reasons for this is a lack of suitable data to empirically model the various determinants of stunting. Much of the research that has been conducted thus far has focused on one particular factor or a limited set of factors[2,13–16]. However, the high degree of correlation between many of the determinants of stunting suggests that the results from studies that do not estimate comprehensive empirical models may suffer from omitted variable bias[17]. This is a

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concern that applies not just to the South African literature on the determinants of stunting, but more generally to research in this field in other low- and middle-income countries[18,19]. Much of the work on stunting does recognise the importance of various factors, both proximate and distal, that are identified in the conceptual literature and represented in the well-known UNICEF 1990 framework on child malnutrition (updated in the second *Lancet* Maternal and Child Health series[20]). However, empirically operationalising this model is often limited by data availability.

Another limitation of the empirical work in this area is that often the regression strategy adopted is not suitable to reveal mediating relationships between the different determinants of stunting[18]. Including all groups of determinants, both proximate and distal, in the same regression may attenuate the omitted variable bias concern, but it can result in underestimating the influence of the more distal determinants[21], and these underlying causal factors may be of particular interest in social science research and policy. Although many studies have used a multistage or sequential regression approach, this has generally involved two or three stages of adding progressively more determinants, which does not allow specific mediating effects to be identified[22].

The objective of the present paper was twofold. First, a model of stunting was estimated in which all the main determinant groups were accounted for in the regression analysis, thus attenuating the omitted variable bias concern. Second, multiple mediator analysis was used to identify the various pathways through which maternal education, a key distal factor, affects stunting. The study focused on mother's education for several reasons. First, a majority of children in South Africa, over 70 %, live with their mother resident in the household (while only 30 % of children live with their father resident in the household)[23,24], 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[19,22,25,26]. Third, as will be discussed further below, almost all the determinant groups included in the fully specified model of stunting can be hypothesised to operate through maternal education[22,25].

To understand the determinants of stunting at 2 years of age, data were drawn from the Birth to Twenty Plus (Bt20) cohort study from Johannesburg, South Africa. The data set was particularly well suited to the empirical strategy in the present paper not only because it has longitudinal information on the child and his/her environment from birth, but also because of the wealth of information collected that has not yet been fully explored in the existing research. This includes data on child growth, the home environment, caregiver characteristics, socio-economic circumstances, access to services and, importantly for the present study's purposes, illness, breast-feeding and dietary diversity. This allowed a fairly comprehensive model of stunting to be estimated, as variables capturing both the proximate and distal causes of stunting are available in the data.

Methods

Data and sample

The Bt20 cohort study is a longitudinal study of children who were born in Johannesburg (South Africa's largest metropole) over a 7-week period between April and June 1990. Information was collected from mothers at antenatal clinics, from birth records and through face-to-face interviews with the caregiver and child at least once per year, and more often during infancy. The sample of eligible singleton births was 3273, with data collected from approximately 1600 to 2200 participants at each interview point. These response rates are within the range documented for other birth cohort studies in developing countries[27]. Details of the ongoing data collection process in Bt20 have been well documented elsewhere[28].

For the empirical work, data were drawn predominantly from the first two years of the study. The outcome variable of interest was stunting at 2 years, with stunting defined as a height-for-age <-2 standard deviations below the median of the healthy reference population (i.e. height-for-age *Z*-score (HAZ) <-2), using the WHO Child Growth Standards[29]. Of the initial sample of 3273 children, contact was made with 1873 in year 2, of which 1805 children had data on HAZ. A comparison of these 1805 children with the remaining 1468 children from the initial sample showed no statistically significant difference between the two groups on some key variables such as the child's sex, birth weight, and whether the child was born in a public or private hospital. A substantial part of the sample attrition appears to have been due to a large number of the mothers initially enrolled in the sample returning to rural areas within the first year of the child's birth (suggesting that they had come to the city to give birth in better health facilities)[28]. It is therefore important to note that the sample of children analysed is most closely representative of those who were born in the city and who remained urban-dwellers in the first two years of their life.

Model

The empirical approach had two main features in line with the objectives described above. First, a fully specified model of stunting was estimated which attempted to incorporate all the key determinant groups, both proximal and distal. Recent research, influenced by the systems approach in epidemiology, has emphasised examining all the determinants of a particular outcome simultaneously to avoid oversimplified regressions that are susceptible to omitted variable bias[17]. The choice of factors to include

in the empirical model was guided by the determinant groups described in the UNICEF conceptual framework on child malnutrition, where the proximate causes are dietary intake and disease, while the more distal causes are the underlying household and environmental factors, such as access to resources, the care environment, maternal factors and access to services[20]. Within each of the groups attempts were made to attenuate collinearity concerns by, for example, using principal components analysis (PCA) to combine information from multiple variables into indices, as has been done elsewhere[13].

Second, an analysis was conducted to ascertain which of these determinant groups acted as pathways, or mediators (M), through which the key independent variable of interest (X), i.e. maternal education, affected the outcome variable (Y), i.e. stunting. The multiple mediator model suggested by Preacher and Hayes[30,31] and others[32] was used. This approach considers all the mediators (M^1, M^2, …, M^j) jointly in estimating the indirect effects of X on Y, and is more appropriate than the more common single mediator analysis if there are multiple mediators in the model, some of which are thought to affect each other or may be collinear[30,32]. In other words, in this model, the specific indirect effect of a particular pathway M^1 is the unique ability of M^1 to mediate the $X \rightarrow Y$ relationship, controlling for all other mediators M^2, …, M^j in the model. The total mediated or total indirect effect of X on Y is the sum of these specific indirect effects of M^1, M^2 through to M^j.

Ordinary least-squares regression analysis was used to accommodate the multiple mediator model analysis[31]; however, when comparing the average marginal effects derived from probit models with the ordinary least-squares regression coefficients, no notable differences in size effects or *P* values were found for any of the variables. As in Preacher and Hayes[30], the product-of-coefficients approach for producing the indirect coefficients in the multiple mediator analysis was used, and bootstrap standard errors and confidence intervals were calculated. Tests for collinearity in the regression when all the variable groups were included simultaneously were conducted, and it was found that the variance inflation factors for the variables ranged between 1·03 and 2·13 (a common rule-of-thumb is that values of 10 or higher are cause for concern).

The pathways specified from maternal education to stunting in the empirical modelling were guided by the UNICEF conceptual framework as well previous research on this topic by Frost *et al.* for Bolivia[22] and later applied by Abuya *et al.*[25] in the Kenyan context. The mediator groups and the choice of variables for each group are described in more detail below.

Description of pathways and variables

The first pathway was captured by the birth weight *Z*-score (based on the WHO 2006 Child Growth Standards[29]), which could be interpreted as separating the effect of maternal education into a prenatal and postnatal component. In other words, it attempted to control for factors in the antenatal period that may have limited or promoted intra-uterine growth.

The second pathway was the child's feeding practices, one of the most important immediate determinants of stunting and an obvious potential pathway from maternal education to stunting. Infants receive the nutrients they need through both breast-feeding and supplementary foods in the first two years. Information on the duration of breast-feeding and dietary diversity when the child was aged 1 year were therefore included. Dietary diversity was captured by a food variety score (FVS), calculated from detailed information gathered in an FFQ at 1 year. The questionnaire in Bt20 was based on an FFQ validated in older ages[33] and modified to ensure the most commonly consumed local food items by infants/children were included, drawing on formative research in a multi-ethnic local population group[34]. The FFQ collected information on 149 food items, with respondents reporting whether the child usually consumed each item monthly or more frequently. The FVS, a commonly used measure of dietary diversity, is a simple count index of how many food items the child consumed[35–37]. The FVS has been found to be correlated with nutrient adequacy as well as height-for-age and weight-for-age *Z*-scores among South African children[35]. Alternative measures of dietary diversity were also calculated using the data from the Bt20 FFQ, including the FAO's twelve-food group score (the Household Dietary Diversity Score)[38], the WHO's infant 'minimum dietary diversity' indicator[39], and the more complex measure described in Drescher *et al.*[40] which takes into account the distribution and healthiness of the foods consumed. None of these measures predicted stunting in the results, so we reverted to the FVS for the final analysis.

The third mediator variable group included information on illness/disease, another proximate causal factor in stunting and a likely pathway through which mother's education might affect stunting. The incidence of disease in the first two years was proxied by symptoms of illness the child exhibited. The symptoms data were collected at 6 months, 1 year and 2 years, and the questions asked whether the child had experienced each of nine symptoms in the previous two weeks. PCA was used to group symptoms into three indices: respiratory (four symptoms), eyes and ears (two symptoms), and gastrointestinal (three symptoms).

The fourth pathway group included information about the care environment in two variables: whether the mother was the principal caregiver and an index capturing the relationship between the mother and child and their well-being. The index was created with PCA based on whether the interviewer responded positively to a series of six questions in the year 2 survey: 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? Cronbach's α for this index was 0·9018, suggesting a very high degree of internal consistency.

The fifth pathway group included proxies for the mother's reproductive autonomy and behaviour, namely maternal age, the birth order of the index child and a measure of birth spacing (equal to 1 if a younger sibling was born within 24 months of the index child's birth). More educated mothers are hypothesised to have greater reproductive autonomy, giving birth to their children later in life and having fewer children[22]. It is expected that the risk of stunting decreases the fewer children the mother has, possibly reflecting less intrahousehold competition over resources (not only financial, but also the mother's time and energy).

The sixth pathway was the mother's attitude towards modern health care, proxied by the utilisation of available health care, as has been done elsewhere[22]. A score reflecting the number of vaccination visits, out of a possible five, the child was taken to by his/her first birthday was included, as strong correlations between attitudes and immunisations have been found previously[25]. The health utilisation variable was coded 0 if two or fewer visits were completed, 1 if three or four visits were completed, and 2 if all five vaccinations were administered.

The seventh pathway was access to services, captured by an index based on the PCA of three items: whether the household had electricity, an indoor flush toilet, and hot and cold indoor running water.

The final pathway group represented socio-economic status (SES), measured by an asset index and a parental occupation score. The asset index was created through PCA of the following variables: ownership of a television, fridge, washing machine, telephone, the dwelling the household lives in and the ratio of people to sleeping rooms (crowding). The occupation index allocated each parent's labour market status into one of six 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; and 5 = independent, high profession, manager and executive. The parents' scores were averaged to create a final score ranging from 0 to 5.

Results

Summary statistics are presented in Table 1 for the analytical sample of children who had non-missing data on HAZ at age 2 years and the covariates (n 691). Mean HAZ at 2 years for this sample was −1·28 (SD 1·15) and the prevalence of stunting was 23 % (160/691).

The mean birth weight for the sample of children was 3103 (SD 491) g. Children were breast-fed on average for 13 months and the mean FVS suggests that a typical child in the sample consumed about thirty-four different food items on a monthly basis. Despite including data from three time points (6 months, 1 year and 2 years), the rates of reporting on symptoms of illness were quite low, with the exception of the respiratory group: on average 2·54 (SD 1·27) of the four respiratory symptoms were exhibited at least once in the three time periods.

Mothers had 10 years of completed education on average, and the mean maternal age at birth was 26 (SD 5·91) years. Birth order ranged from 1 to 4, with the index child being on average the second child born to their mother; and for about 5 % of children in the sample, a sibling was born within 24 months.

The children's households had a mean of 1·6 (SD 0·91) of three essential services (water, electricity, toilet) and 3·2 (SD 1·61) of the six assets for which data were available.

Table 2 contains the results of the multivariate regression analysis. Column I shows the regression of stunting *v.* mother's education only, controlling for the sex of the child; and Column II shows the fully specified regression model, where all the variable groups hypothesised to predict stunting were included simultaneously. The former shows the total effect of mother's education on stunting while the latter shows the direct effect of mother's education on stunting, once the other variable groups were included in the regression. The results in Column I suggest a protective effect of mother's education against stunting, with a total effect of −0·021 ($P < 0·001$; 95 % CI −0·03, −0·01). When all the variables were included simultaneously in Column II, the effect of mother's education fell to −0·0049 ($P = 0·45$; 95 % CI −0·02, 0·01), suggesting the other variable groups included absorbed, or mediated, most of the maternal education effect.

Variables in the fully specified regression that were negatively associated with the likelihood of stunting were the birth weight *Z*-score (−0·084; $P < 0·001$; 95 % CI −0·11, −0·06); the index capturing the relationship between mother and child (−0·015; $P = 0·04$; 95 % CI −0·03, −0·00); mother's age (−0·008; $P = 0·03$, CI −0·02, −0·00); and the vaccination score (−0·05; $P = 0·04$; 95 % CI −0·10, −0·00). The variable measuring symptoms of illness related to eyes and ears was positively associated with the likelihood of stunting (0·038; $P = 0·01$; 95 % CI 0·01, 0·07). Counter-intuitively, symptoms of respiratory illness were negatively related to stunting (−0·035; $P = 0·01$; 95 % CI −0·06, −0·01), which could be due to a greater likelihood of caregivers reporting these symptoms among better-off children.

Table 3 presents the results of the multiple mediator analysis, which estimated the indirect effects of maternal education on stunting, showing the relative strength of the various mediator pathways. The proportion of the total maternal education effect that was mediated by the various pathways is also displayed. The P values and

Table 1 Summary statistics for the analytical sample† from the Birth to Twenty Plus (Bt20) cohort study of children born in April–June 1990 in Soweto-Johannesburg, South Africa

	Age collected	Mean or %‡	SD	Range
Dependent variable				
Stunting (HAZ < −2) (%)	2 years	23·15	–	0–1
HAZ	2 years	−1·28	1·15	−6·15–2·91
Maternal education				
Years of education completed	antenatal–2 years	10·01	2·75	0–14
Baseline				
Female (%)	0 months	52·53	–	0–1
Birth weight				
Birth weight (g)	0 months	3103	491	1070–4800
Birth weight *Z*-score	0 months	−0·45	1·11	−5·66–2·63
Feeding				
Duration of breast-feeding (months)	6 months, 1 year, 2 years	13·05	8·99	0–25
Food variety score	1 year	34·07	10·77	8–75
Illness				
Respiratory symptoms	6 months, 1 year, 2 years	2·54	1·27	0–4
PCA respiratory symptoms	6 months, 1 year, 2 years	0·49	1·32	−2·20–1·94
Eyes & ears symptoms	6 months, 1 year, 2 years	0·44	0·59	0–2
PCA eyes & ears symptoms	6 months, 1 year, 2 years	0·21	1·17	−0·61–3·63
Gastrointestinal symptoms	6 months, 1 year, 2 years	0·87	0·91	0–3
PCA gastrointestinal symptoms	6 months, 1 year, 2 years	0·27	1·29	−0·96–3·34
Caregiver				
Mother principal caregiver (%)	2 years	62·23	–	0–1
Mother–child relationship	2 years	4·58	2·14	0–6
PCA mother–child relationship	2 years	−0·09	2·13	−4·65–1·29
Reproductive				
Mother's age (years)	0 months	25·52	5·91	14–43
Birth order	antenatal–2 years	2·00	1·04	1–4
Birth spacing (another child born within 24 months) (%)	2 years	5·35	–	0–1
Attitudes				
Health utilisation score (based on vaccinations received)	1 year	1·20	0·65	0–2
Services				
Services index	0 months–2 years	1·62	0·91	0–3
PCA services index	0 months–2 years	0·09	1·31	−1·74–2·11
Socio-economic status				
Asset index (simple count)	2 years	3·16	1·61	0–6
Crowding	antenatal–2 years	3·30	1·67	0·50–19
PCA asset index	2 years	0·08	1·55	−3·90–2·95
Parental occupation score	antenatal–2 years	1·88	1·34	0–5

HAZ, height-for-age *Z*-score; PCA, principal components analysis.
†The analytical sample consists of children with non-missing data on HAZ at 2 years and the covariates (*n* 691).
‡The mean/percentage column shows the mean for continuous variables and the percentage for binary variables.

bootstrap CI indicate that only the birth weight and SES pathway groups were mediators in the relationship between maternal education and stunting. The most important pathway in the relationship between maternal education and stunting was SES, with this mediator group accounting for 41% (−0·0085/−0·0209; $P=0·01$; 95% CI −0·016, −0·003) of the total maternal education effect on stunting. The birth weight pathway was found to account for 18% (−0·0037/−0·0209; $P=0·02$; 95% CI −0·0074, −0·0008) of the total maternal education effect on stunting. The final row in Table 3 shows that all the mediator groups combined accounted for 77% (−0·0161/−0·0209; $P<0·001$; 95% CI −0·025, −0·008) of the total maternal education effect on stunting.

Finally, given the relatively small sample size for the regression analysis, the group of children in the analytical sample (*n* 691) were compared with the remaining group who were contacted in year 2 but were dropped from the regression sample because of missing data on the covariates (*n* 1114 (i.e. 1805 − 691)). Based on *t* tests of differences in means, children in the analytical sample were found to be more likely to be stunted (23·2 *v.* 18·9%; $P=0·03$), their mothers were marginally more educated (10·01 *v.* 9·65 years of schooling; $P=0·01$), they were breast-fed for longer (13 *v.* 11 months; $P<0·001$), and they reported a higher number of respiratory (2·54 *v.* 2·18; $P<0·001$), eyes and ears (0·44 *v.* 0·33; $P<0·001$) and gastrointestinal symptoms (0·87 *v.* 0·67, $P<0·001$). As an informal test to try to ascertain the effect of these differences on the regression results, bivariate regressions of stunting *v.* each individual covariate were performed, first restricting the sample to the 691 children who had data on all covariates and then using the full sample available. The only noteworthy differences were that mother's age had a smaller effect on stunting for the unrestricted sample (−0·0021; $P=0·17$; 95% CI −0·0051; 0·0009; *n* 1804) compared with the smaller analytical sample (−0·0068; $P=0·01$; 95% CI −0·0122; −0·00153; *n* 691), and there was a much smaller association between respiratory symptoms and stunting for the unrestricted sample (−0·0059; $P=0·38$; 95% CI −0·0194; 0·0075; *n* 1711)

Table 2 Multivariate regression results for stunting at 2 years† from the Birth to Twenty Plus (Bt20) cohort study of children born in April–June 1990 in Soweto-Johannesburg, South Africa

	Direct effect of mother's education (I)			Complete regression (II)		
	Coefficient	*P* value	95 % CI	Coefficient	*P* value	95 % CI
Female	−0·0601	0·06	−0·12, 0·00	−0·0370	0·23	−0·10, 0·02
Mother's education	−0·0209***	<0·001	−0·03, −0·01	−0·0049	0·45	−0·02, 0·01
Birth weight						
Birth weight *Z*-score				−0·0839***	<0·001	−0·11, −0·06
Feeding						
Food variety score				−0·0007	0·65	−0·00, 0·00
Breast-feeding duration				0·0014	0·43	−0·00, 0·00
Illness						
Respiratory symptoms				−0·0354**	0·01	−0·06, −0·01
Eyes & ears symptoms				0·0383**	0·01	0·01, 0·07
Gastrointestinal symptoms				0·0047	0·71	−0·02, 0·03
Caregiver characteristics						
Mother principal caregiver				0·0117	0·71	−0·05, 0·07
Mother–child relationship				−0·0154*	0·04	−0·03, −0·00
Reproductive behaviour						
Mother's age				−0·0082*	0·03	−0·02, −0·00
Birth order				0·0355	0·09	−0·01, 0·08
Birth spacing				0·0588	0·39	−0·08, 0·19
Attitude to modern health care						
Vaccination score				−0·0500*	0·04	−0·10, −0·00
Access to services						
Services index				0·0131	0·34	−0·01, 0·04
Socio-economic status						
Asset index				−0·0225	0·07	−0·05, 0·00
Parental occupation score				−0·0250	0·07	−0·05, 0·00
Constant	0·4728***	<0·001	0·35, 0·60	0·5066***	<0·001	0·26, 0·75
R^2	0·02			0·14		
n	691			691		

*$P \leq 0{\cdot}05$, **$P \leq 0{\cdot}01$, ***$P \leq 0{\cdot}001$.
†The sample size in each regression model is restricted to Bt20 participants who had data on height-for-age *Z*-score and all variables in the Complete model. Coefficients presented are ordinary-least squares regression coefficients.

Table 3 Multiple mediation analysis of the effect of mother's education on stunting at 2 years from the Birth to Twenty Plus (Bt20) cohort study of children born in April–June 1990 in Soweto-Johannesburg, South Africa

Pathway	Observed coefficient – indirect effects	Proportion of the total education effect that is mediated by various pathways†	Bootstrap SE	*Z*	*P* > *Z*	95 % CI‡
Birth weight	−0·0037	0·1760	0·0016	−2·26	0·02	−0·0074, −0·0008
Feeding	−0·0010	0·0470	0·0012	−0·86	0·39	−0·0036, 0·0010
Illness	−0·0007	0·0328	0·0011	−0·65	0·52	−0·0029, 0·0012
Caregiver characteristics	−0·0012	0·0597	0·0008	−1·55	0·12	−0·0032, 0·0000
Reproductive behaviour	−0·0028	0·1357	0·0024	−1·17	0·24	−0·0079, 0·0017
Attitude	0·0002	−0·0115	0·0005	0·51	0·61	−0·0004, 0·0016
Services	0·0016	−0·0774	0·0018	0·92	0·36	−0·0016, 0·0052
Socio-economic status	−0·0085	0·4060	0·0032	−2·63	0·01	−0·0155, −0·0026
Total indirect	−0·0161	0·7682	0·0041	−3·91	<0·001	−0·0249, −0·0083

†The proportion of the total effect that is mediated is calculated as the indirect effect/total effect. The total effect (−0·0209) is equal to the total indirect effect (−0·0161) plus the direct effect (−0·0049), the latter being the coefficient on mother's education in the fully specified regression model with all mediators and controls (shown in Table 2, Column II).
‡Bias-corrected and accelerated bootstrap CI displayed (1000 bootstrap samples). These are non-symmetric and reflect the skewness of the sampling distribution of the product coefficients. Percentile and bias-corrected CI produced similar results.

compared with the smaller analytical sample (−0·0288; $P = 0{\cdot}02$; 95 % CI −0·0525; −0·005; *n* 691).

Discussion

In the present paper, attempts were made to estimate a comprehensive empirical model of the predictors of stunting at 2 years, focusing on the mechanisms through which maternal education affected the probability of child stunting in South Africa. The results suggested that, as has been found in other work[15,19,26,41], maternal education was indeed an important determinant of stunting. However, much of the effect of maternal education on stunting (41 %) was explained by its association with SES (measured by an asset index and a parental occupation index). This suggests

that a large part of the maternal education effect on stunting was due to the health and growth benefits that accompany increased resources and occupational standing.

Another 18 % of the maternal education effect on stunting was mediated by the birth weight pathway. To the extent that birth weight and HAZ at 2 years capture the same information about the child's growth, this effect could perhaps be interpreted to mean that the effect of maternal education on stunting could be divided into a prenatal effect (of approximately 20 %) and a postnatal effect up to 2 years (of approximately 80 %). This implies that education improves the ability of the mother to foster a healthy environment for the child's intra-uterine growth. While it is assumed that birth weight *Z*-score was a fairly good proxy for the cumulative prenatal effect, it may not capture the full effect. Unfortunately, the analysis was limited by a lack of relevant prenatal variables in the data set.

The percentages of the total maternal education effect mediated by the other hypothesised mediator groups – namely feeding, illness, the care environment, the mother's reproductive behaviour, attitudes towards health care and access to services – were much smaller (between 1 and 13 %) and these indirect effects were not found to be significantly different from zero in the multiple mediation analysis. This suggests that either these were not clear pathways through which maternal education affected stunting in the sample of children, or that other factors were playing a role, for example household or partner social support.

There are at least three key strengths to the present study's findings. First, a relatively comprehensive model of stunting was estimated and therefore most (77 %) of the maternal education effect on stunting was 'explained' by the various pathway groups. Similarly, while higher SES was found to be one of the main pathways through which maternal education affected stunting, it is interesting to note that the SES variables had a very small effect in the final fully populated model. This indicates that, to a large extent, pathways through which SES operates were also accounted for in the model.

Second, the results were consistent with other work on this topic. Frost *et al.*[22] analysed similar pathway groups using data from Bolivia, and although their complete model contained fewer pathway groups than the model in the present paper and they did not formally test the mediation effects in a multiple mediator model, the maternal education coefficient was reduced by 59 % following the joint introduction of the various pathways. The pathway that explained most of the effect of maternal education on stunting in their study was also SES (40 %), with attitudes towards medicine, general health knowledge (not included in the present study's model) and reproductive behaviour producing smaller mediated effects (22, 15 and 11 %, respectively). The differences in the relative effects of the pathways identified could reflect real differences between the South African and Bolivian contexts. Having said this, the similarity across the two country study results is notable. Abuya *et al.*[25] studied the role of maternal education in determining health outcomes in Kenya, using similar groups and indicators to Frost *et al.*[22]; however, as they did not perform regression models separately for each determinant grouping as in Frost *et al.*[22], they were unable to isolate the extent to which each acts as a pathway. Nevertheless, in line with the results of the present paper, they found that education was an important factor in predicting stunting, but that this effect dropped away once other covariates, such as SES, attitudes, autonomy and parity, were included in the model.

Third, even though some of the determinant groups in the present study's empirical analysis did not appear to be significant pathways through which mother's education operated, a number of these variables predicted stunting in the regression model with all the variables included jointly (giving us some confidence in the measures used). This implies that they were important determinants of stunting, but independent of mother's education. For example, the vaccination score, used as a proxy for the mother's attitude to modern health care, was negatively related to stunting, potentially capturing that more engaged and proactive mothers (regardless of education) helped reduce the risk of stunting in their children. Mothers might also have gained knowledge and support related to child development when visiting a clinic for the vaccination to be administered. Similarly, a more positive evaluation of the mother and child's relationship and well-being was negatively related to stunting, even though it did not mediate the maternal education effect on stunting. In other words, the quality of the care environment independently impacted the child's growth, without necessarily acting as a pathway through which mother's education operated.

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 optimise child growth, mothers need to be supported and empowered in providing care, possibly through enhanced ante- and postnatal services.

Despite producing some important findings, there were limitations to the work. The Bt20 data are now over 20 years old and there have been a number of changes since the early 1990s in maternal and child policies. Being able to repeat this analysis using more contemporary data would be useful in identifying whether the relative importance of the various factors outlined above has changed with the economic and nutritional transitions that have accompanied South Africa's democratisation. The problem of sample attrition and missing data on the covariates, which could bias and affect the precision of the results, was also noted. The resulting analytical sample was predominantly representative of children who were born in

Johannesburg during the study period and remained there for at least the first two years of their life. Therefore, the mechanisms identified in the present paper may not be applicable to rural areas, where the prevalence of stunting is generally higher in South Africa, and the causal factors may be different. Nevertheless, until new data of this nature are collected, the Bt20 study remains unique in providing detailed longitudinal data on child health and the environment in an urban context in South Africa. Given that the problem of child stunting in South Africa has not been solved, as is clear from the persistently high prevalence recorded even in the most recent national data sets, fully exploring the available information in this data set remains a useful exercise.

There are also important lessons to be learnt from the Bt20 analysis for future data collection efforts. Bt20 collected detailed information on the quality of the child's care environment and the mother's involvement in the child's life, information which is generally not collected in larger household surveys. As shown in the results, the quality of the care received was an important determinant of child outcomes, but in many studies it is simply omitted and treated as an 'unobservable'. Further, the data collected on breast-feeding, dietary diversity and illness presented the opportunity to explore proximal, in addition to distal, causes of stunting. However, the lack of predictive power of many of these variables suggests that one should consider whether there are more accurate and sensitive ways of collecting data on these aspects of the child's life. Information on these variables is difficult to collect through questionnaires conducted at discrete points in time and is potentially 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 a physical record). In future data collection, alternative methods of questioning could be explored, such as introducing a 24 h food recall questionnaire[38,42], and collecting feeding and illness data more often during infancy.

Conclusion

In conclusion, while distal factors may be key to understanding the broader environmental factors determining poor child outcomes, understanding the more proximal pathways through which they operate is also important in trying to think through the necessary interventions. These interventions may be to strengthen policy and/or services to better support women prior to, during and after pregnancy.

Acknowledgements

Acknowledgements: The authors would like to thank Rihlat Said-Mohamed for sharing her data from Bt20 on infections/illness and for advice on grouping symptoms, and Julian May, Stephen Devereux and Ian Timaeus for their comments on the working paper version of this study. *Financial support:* The University of the Witwatersrand, Johannesburg, the South African Medical Research Council, the Human Sciences Research Council of South Africa, and Wellcome Trust (UK) have supported collection of the data (Bt20). S.N. is supported by the DST-NRF Centre of Excellence in Human Development at the University of the Witwatersrand. This study was funded by the DST-NRF Centre of Excellence in Food Security at the University of the Western Cape through the Project on Food Security and the Persistence of Child Malnutrition in South Africa (project number 160501). The funders had no role in the design, analysis or writing of this article. *Conflict of interest:* None. *Authorship:* Formulating the research question: D.C., G.E. and S.A.N. Analysing the data: G.E. and D.C. Writing and/or revising the manuscript: D.C., G.E. and S.A.N. *Ethics of human subject participation:* The Birth to Twenty Study was granted approval by the Human Ethics Committee of the University of the Witwatersrand, Johannesburg.

References

1. UNICEF, World Health Organization & The World Bank (2016) *Joint Child Malnutrition Estimates*. New York/Geneva/Washington, DC: UNICEF/WHO/World Bank.
2. May J & Timæus IM (2014) Inequities in under-five child nutritional status in South Africa: what progress has been made? *Dev South Afr* **31**, 761–774.
3. Shisana O, Labadarios D, Rehle T *et al.* (2013) *South African National Health and Nutrition Examination Survey (SANHANES-1)*. Cape Town: HSRC Press.
4. Bhutta ZA, Das JK, Rizvi A *et al.* (2013) Evidence-based interventions for improvement of maternal and child nutrition: what can be done and at what cost? *Lancet* **382**, 452–477.
5. Walker SP, Chang SM, Powell CA *et al.* (2005) Effects of early childhood psychosocial stimulation and nutritional supplementation on cognition and education in growth-stunted Jamaican children: prospective cohort study. *Lancet* **366**, 1804–1807.
6. Victora CG, Adair L, Fall C *et al.* (2008) Maternal and child undernutrition: consequences for adult health and human capital. *Lancet* **371**, 340–357.
7. Dewey KG & Begum K (2011) Long-term consequences of stunting in early life. *Matern Child Nutr* **7**, Suppl. 3, 5–18.
8. Yamauchi F (2008) Early childhood nutrition, schooling, and sibling inequality in a dynamic context: evidence from South Africa. *Econ Dev Cult Change* **56**, 657–682.
9. Casale D, Desmond C & Richter L (2014) The association between stunting and psychosocial development among preschool children: a study using the South African Birth to Twenty cohort data. *Child Care Health Dev* **40**, 900–910.
10. Casale D & Desmond C (2016) Recovery from stunting and cognitive outcomes in young children: evidence from the South African Birth to Twenty Cohort Study. *J Dev Orig Health Dis* **7**, 163–171.
11. Casale D (2016) *Analysing the Links between Child Health and Education Outcomes: Evidence from NIDS Waves 1–4. SALDRU Working Paper 179/NIDS Discussion Paper 2016/6*. Cape Town: SALDRU, University of Cape Town.

12. Said-Mohamed R, Micklesfield LK, Pettifor JM *et al.* (2015) Has the prevalence of stunting in South African children changed in 40 years? A systematic review. *BMC Public Health* **15**, 534.
13. Jones LL, Griffiths PL, Adair LS *et al.* (2008) A comparison of the socio-economic determinants of growth retardation in South African and Filipino infants. *Public Health Nutr* **11**, 1220–1228.
14. Slemming W, Bello B, Saloojee H *et al.* (2016) Maternal risk exposure during pregnancy and infant birth weight. *Early Hum Dev* **99**, 31–36.
15. Timæus IM (2012) Stunting and obesity in childhood: a reassessment using longitudinal data from South Africa. *Int J Epidemiol* **41**, 764–772.
16. Ardington C & Gasealahwe B (2012) *Health: Analysis of the NIDS Wave 1 and 2 Datasets. SALDRU Working Paper 80/ NIDS Discussion Paper 2012/3*. Cape Town: SALDRU, University of Cape Town.
17. Fenske N, Burns J, Hothorn T *et al.* (2013) Understanding child stunting in India: a comprehensive analysis of socio-economic, nutritional and environmental determinants using additive quantile regression. *PLoS One* **8**, e78692.
18. Paxson C & Schady N (2007) Cognitive development among young children in Ecuador the roles of wealth, health, and parenting. *J Hum Resour* **42**, 49–84.
19. Chen Y & Li H (2009) Mother's education and child health: is there a nurturing effect? *J Health Econ* **28**, 413–426.
20. Black RE, Allen LH, Bhutta ZA *et al.* (2008) Maternal and child undernutrition: global and regional exposures and health consequences. *Lancet* **371**, 243–260.
21. Weitkunat R & Wildner M (2002) Exploratory causal modeling in epidemiology: are all factors created equal? *J Clin Epidemiol* **55**, 436–444.
22. Frost MB, Forste R & Haas DW (2005) Maternal education and child nutritional status in Bolivia: finding the links. *Soc Sci Med* **60**, 395–407.
23. Posel D & Rudwick S (2013) Changing patterns of marriage and cohabitation in South Africa. *Acta Jurid* **13**, 169–180.
24. Ward C, Makusha T & Bray R (2015) Parenting, poverty and young people in South Africa: what are the connections? In *South African Child Gauge*, pp. 69–74 [A De Lannoy, S Swartz, L Lake *et al.*, editors]. Cape Town: Children's Institute, University of Cape Town.
25. Abuya B, Onsomu E, Kimani J *et al.* (2011) Influence of maternal education on child immunization and stunting in Kenya. *Matern Child Health J* **15**, 1389–1399.
26. Semba RD, de Pee S, Sun K *et al.* (2008) Effect of parental formal education on risk of child stunting in Indonesia and Bangladesh: a cross-sectional study. *Lancet* **371**, 322–328.
27. Richter L, Norris S, Pettifor J *et al.* (2007) Cohort profile: Mandela's children: the 1990 birth to twenty study in South Africa. *Int J Epidemiol* **36**, 504–511.
28. Norris SA, Richter LM & Fleetwood SA (2007) Panel studies in developing countries: case analysis of sample attrition over the past 16 years within the birth to twenty cohort in Johannesburg, South Africa. *J Int Dev* **19**, 1143–1150.
29. World Health Organization (2006) *Child Growth Standards: Length/Height-for-Age, Weight-for-Age, Weight-for-Length, Weight-for-Height and Body Mass Index-for-Age: Methods and Development*. Geneva: WHO.
30. Preacher KJ & Hayes AF (2008) Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behav Res Methods* **40**, 879–891.
31. University of California Los Angeles, Statistical Consulting Group (2011) How can I analyse multiple mediators in STATA? http://www.stats.idre.ucla.edu/stata/faq/how-can-i-analyze-multiple-mediators-in-stata/ (accessed September 2017).
32. VanderWeele TJ & Vansteelandt S (2014) Mediation analysis with multiple mediators. *Epidemiol Method* **2**, 95–115.
33. Margetts BM, Cade JE & Osmond C (1989) Comparison of a food frequency questionnaire with a diet record. *Int J Epidemiol* **18**, 868–873.
34. MacKeown JM, Cleaton-Jones PE, Edwards AW *et al.* (1998) Energy, macro- and micronutrient intake of 5 year old urban black South African children in 1984 and 1995. *Paediatr Perinat Epidemiol* **12**, 297–312.
35. Steyn NP, Nel JH, Nantel G *et al.* (2006) Food variety and dietary diversity scores in children: are they good indicators of dietary adequacy? *Public Health Nutr* **9**, 644–650.
36. Msaki MM & Hendriks SL (2013) Do food quality and food quantity talk the same? Lesson from household food security study in Embo, South Africa. *J Am Coll Nutr* **32**, 165–176.
37. Saibul N, Shariff ZM, Lin KG *et al.* (2009) Food Variety Score is associated with double burden of malnutrition in *Orang Asli* (Malaysian indigenous peoples) households: implications for health promotion. *Asia Pac J Clin Nutr* **18**, 412–422.
38. Kennedy G, Ballard T & Dop MC (2010) *Guidelines for Measuring Household and Individual Dietary Diversity*, p. 51. Rome: FAO.
39. World Health Organization (2010) *Indicators for Assessing Infant and Young Child Feeding Practices. Part 3: Country Profiles*, p. 59. Geneva: WHO.
40. Drescher LS, Thiele S & Mensink GB (2007) A new index to measure healthy food diversity better reflects a healthy diet than traditional measures. *J Nutr* **137**, 647–651.
41. Chopra M (2003) Risk factors for undernutrition of young children in a rural area of South Africa. *Public Health Nutr* **6**, 645–652.
42. Kennedy GL, Pedro MR, Seghieri C *et al.* (2007) Dietary diversity score is a useful indicator of micronutrient intake in non-breast-feeding Filipino children. *J Nutr* **137**, 472–477.