ELSEVIER

www.elsevier.com/locate/worlddev

CrossMark

*World Development* Vol. 66, pp. 749–761, 2015

http://dx.doi.org/10.1016/j.worlddev.2014.09.022

# The Other Asian Enigma: Explaining the Rapid Reduction of Undernutrition in Bangladesh

DEREK HEADEY [a], JOHN HODDINOTT [a,b], DISHA ALI [c], ROMAN TESFAYE [a] and MEKDIM DEREJE [a,*]

[a] *The International Food Policy Research Institute (IFPRI), Washington, USA*
[b] *Cornell University, USA*
[c] *Independent Consultant, USA*

**Summary.** — Although South Asia has long been synonymous with persistent and unusually high rates of child undernutrition – the so called Asian Enigma – Bangladesh has managed to sustain a surprisingly rapid reduction in the rate of child undernutrition for at least two decades. We investigate this unheralded success through a regression and decomposition analysis of changes in child growth outcomes across five rounds of DHS surveys from 1997 to 2011. We find that rapid wealth accumulation and large gains in parental education are the two largest drivers of change, though health, sanitation, and demographic factors have played significant secondary roles.


*Key words* — undernutrition, Bangladesh, Asian Enigma, statistical decompositions

## 1. INTRODUCTION

In the nutrition literature South Asia is synonymous with unusually high rates of child undernutrition relative to its income levels, as well as sluggish reduction in undernutrition, particularly in India (Deaton & Dreze, 2008). This so called Asian Enigma (Ramalingaswami, Jonson, & Rohde, 1997) has spawned substantial research into possible explanations, including gender and intrahousehold biases (Jayachandran & Pande, 2013; Pande, 2003), unusually high rates of open defecation (Spears, 2013; Spears, Ghosh, & Cumming, 2013), genetic predispositions (Nubé, 2009), poor quality diets and food systems (Headey, Chiu, & Kadiyala, 2012), and the inefficacy of nutritional programs and strategies (Das Gupta, Lokshin, Gragnolati, & Ivaschenko, 2005; World Bank, 2005b).

Yet this literature has largely ignored Bangladesh's sustained reduction in child undernutrition for at least two decades. A recent cross-country study by Headey (2013) concluded that from 1997 to the 2007 Bangladesh recorded one of the fastest prolonged reductions in child underweight and stunting prevalence in recorded history, 1.1 and 1.3 percentage points per annum respectively, narrowly behind the much more celebrated case of Thailand in the 1980s (Heaver & Kachondam, 2002), and just ahead of several success stories identified in the nutrition literature, such as Brazil, Mexico, and Honduras (World Bank, 2006). Strikingly, Bangladesh now has lower stunting rates (41.3%) than India recorded in its 2005–06 national nutrition survey (47.5%) or Pakistan did in its 2012 survey (44.8%), despite both countries having higher mean incomes.

This puzzling neglect of Bangladesh's nutritional success is seemingly explained by the absence of any highly regarded nutrition strategy. In particular, assessments of Bangladesh's Integrated Nutrition Program 1995–2004 (BINP) and the subsequent National Nutritional Program suggest, at best, a modest impact on nutrition outcomes (Hossain, Duffield, & Taylor, 2005; Levinson & Eliot Rohde, 2005; Sack, Roy, Ahmed, & Fuchs, 2005; White, 2005; World Bank, 2005a, 2005b). The most critical of these assessments stated that "the BINP has not achieved its objective to reduce child malnutrition at a population level" (Hossain *et al.*, 2005). Moreover, even the larger National Nutritional Program only ever covered 30% of households (Ahmed *et al.*, 2012). An apparent outcome of the poor reputation of these programs is that while Bangladesh is widely recognized for its remarkable progress in poverty reduction and delivering effect health and family planning services (The Economist, 2012; World Bank, 2005a), the country's success in reducing child malnutrition remains largely overlooked, and certainly understudied. [1]

In this paper we seek to remedy this knowledge gap through an analysis of the drivers of nutritional change in Bangladesh during 1997–2011. In doing so we expand on a growing literature that seeks to explain nutritional change as a function of a wide array of nutrition-sensitive interventions. In addition to the well-documented effects of income on nutrition (Behrman & Deolalikar, 1987; Haddad, Alderman, Appleton, Song, & Yohannes, 2003; Headey, 2013; Heltberg, 2009; Smith & Haddad, 2000), there is various evidence linking nutrition outcomes to education (Burchi, 2012; Headey, 2013; Thomas, Strauss, & Henriques, 1991; Webb & Block, 2004), demography and family planning (Dewey & Cohen, 2007; Headey, 2013; Horton, 1988; Jensen, 2012; Rutstein, 2008), gender empowerment and cultural norms (Jayachandran & Pande, 2013; Pande, 2003), improved sanitation (Humphrey, 2009; Lin *et al.*, 2013; Spears, 2013), and health service utilization (Headey, 2013). Understanding the contribution of such factors to historical changes in nutrition outcomes is an important area of ongoing research, which this paper aims to make a contribution to.

The paper is structured as follows. Sections 2 jointly outlines the hypothesized drivers of nutritional change over this period and the data used to measure them. Section 3 discusses the analytical methods used in the paper. Section 4 presents our

* Final revision accepted: September 21, 2014.

core statistical models, including our decomposition approach. Section 5 presents a range of sensitivity tests. Section 6 concludes.

## 2. HYPOTHESIZED DRIVERS OF NUTRITIONAL CHANGE OVER 1997–2011

In this section we discuss the nature of nutritional change in Bangladesh over the time period in question, the hypothesized drivers of nutritional change over time, and how we measure these factors with the data in question. As noted above, we use the 1997, 2000, 2004, 2007, and 2011 rounds of the Bangladesh Demographic and Health Survey (DHS). These data are well suited to our purposes insofar as they are high quality, nationally representative, highly standardized across rounds, and cover a broad range of the hypothesized drivers of nutritional change (albeit not an exhaustive range). We principally focus on height for age (HAZ) *z*-scores for pre-school children as measured against WHO growth standards as described in de Onis, Garza, Onyango, and Borghi (2007).[2] Linear growth is now widely regarding as the single most relevant indicator of overall nutrition, and the reduction in stunting (HAZ scores of two standard deviations or less) is now the standard metric of nutritional success.[3]

Table 1 presents trends in the three undernutrition indicators analyzed in this paper – HAZ scores, stunting prevalence (HAZ < −2) and severe stunting prevalence (HAZ < −3) – for national, rural and urban samples. Mean HAZ scores and stunting prevalence improved rapidly over 1997–2011, by almost 0.6 standard deviations in the case of HAZ scores and 19 points in the case of stunting. In percentage terms the change in stunting was slightly larger than the change in mean HAZ scores. Figure 1 also shows the distribution of HAZ scores across both rounds and confirms a larger shift in the lower tail. Indeed, severe stunting fell by half from 1997 to 2011. Another finding of some note from Table 1 is that the speed of nutritional gains was somewhat larger in rural areas than in urban areas. Indeed, from 2000 to 2004 there was a slight deterioration in nutritional status in urban areas.

Figure 2 shows HAZ scores by child age. The confidence intervals for the two curves never overlap, suggesting significant improvements in HAZ scores for all ages. However, the shift is larger for some age brackets than others. At the youngest ages we observe a large shift in HAZ scores suggesting improvements in birth size. Since low birth size has been identified as one of the most important explanations of the Asian Enigma, this is potentially a finding of some importance. Among other things it suggests that improvements in maternal nutrition and healthcare may have been important sources of


Figure 1. *Distribution of HAZ scores, 1997 and 2011.*

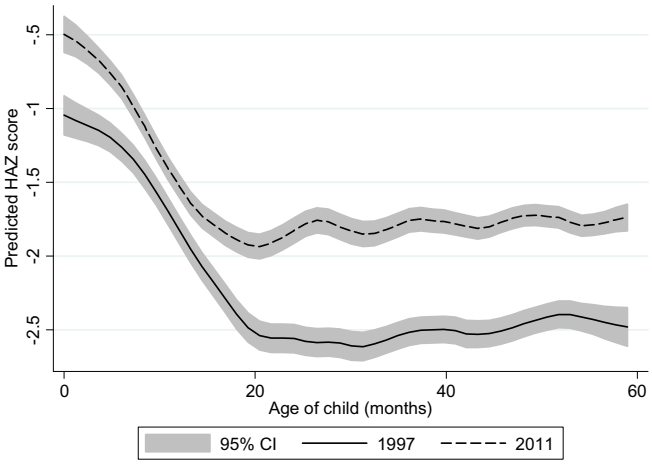


Figure 2. *HAZ scores by child age, 1997 and 2011.*

nutritional change in young children. Of secondary importance in accounting for nutritional change is some improvement in the window of steepest growth faltering. Though the 2011 round continues to show a very sharp process of growth faltering from around six months of age to 20 months of age, the gradient of this decline flattens out in 2011 compared to 1997. This suggests that improvements in child care practices and sanitation have likely improved over time.

The evidence above provides clues as to some of the potential drivers of nutritional change over time, but what specific

Table 1. *Changes in mean HAZ scores and stunting prevalence for different samples across the five BDHS rounds*

| Samples>> | Mean HAZ scores | | | Stunting | | | Severe stunting | | |
|---|---|---|---|---|---|---|---|---|---|
| | Total | Rural | Urban | Total (%) | Rural (%) | Urban (%) | Total (%) | Rural (%) | Urban (%) |
| 1997 | −2.20 | −2.25 | −1.72 | 58.5 | 60.0 | 44.1 | 31.0 | 31.9 | 20.4 |
| 2000 | −1.95 | −2.01 | −1.65 | 49.9 | 51.8 | 41.1 | 21.9 | 23.1 | 16.3 |
| 2004 | −1.92 | −1.96 | −1.72 | 49.5 | 51.0 | 44.0 | 20.5 | 21.3 | 17.0 |
| 2007 | −1.75 | −1.80 | −1.54 | 44.0 | 45.6 | 37.7 | 16.0 | 17.1 | 12.5 |
| 2011 | −1.62 | −1.67 | −1.44 | 40.2 | 41.4 | 35.8 | 14.1 | 14.6 | 12.3 |
| Change | 0.58 | 0.58 | 0.28 | −18.3 | −18.6 | −8.30 | −16.90 | −17.30 | −8.10 |
| Percent change (%) | −26.4% | −25.8% | −16.3% | −31.3 | −31.0 | −18.8 | −54.5 | −54.2 | −39.7 |

*Source:* Authors' estimates from the 1996–97 and 2011 BDHS rounds, using sampling weights.

Table 2. *Changes in the means of key variables from 1997 to 2011*

| Year | Wealth (0–10) | Maternal education (yrs) | Paternal education (yrs) | Mother can travel to health clinic alone (%) | Mothers have prenatal doctor visit (%) | Mothers have prenatal visit with other health professional (%) |
|---|---|---|---|---|---|---|
| 1997 | 3.2 | 2.4 | 3.6 | 20.1 | 19.5 | 9.5 |
| 2011 | 4.9 | 5.4 | 5.1 | 79.3 | 34.2 | 27.8 |
| Change | 1.7 | 3.0 | 1.5 | 59.2 | 0.1 | 0.2 |
| Percent change (%) | 52% | 122% | 42% | 295 | 76 | 192 |
| Year | All vaccinations (%) | Child born in a medical facility (%) | No toilet – village (%) | Water – piped (%) | Birth order (rank) | Birth interval (yrs) |
| 1997 | 44.7 | 2.9 | 25.2 | 4.4 | 3.1 | 2.4 |
| 2011 | 71.1 | 23.6 | 4.8 | 9.4 | 2.5 | 3.0 |
| Change | 26.4 | 20.7 | –20.4 | 5.0 | –0.6 | 0.7 |
| Percent change (%) | 59 | 711 | –81 | 114 | –20% | 29% |

*Notes:* "All vaccines" is only measured for children older than 6 months.
*Source:* Authors' estimates from the 1996–97 and 2011 BDHS rounds, using sampling weights.

factors are likely to be most relevant in the context of Bangladesh? In the parlance of the UNICEF (1990) framework, updated by Black *et al.* (2013), nutrition outcomes are the end result of a causal chain that starts with "basic determinants" (policies, culture, agroecology, and so on), which influence "intermediate" determinants (household food security, care practices, disease environment), which determine the "immediate determinants" of nutrition, notably food intake and utilization. In the DHS, we observe the intermediate determinants (i.e., household wealth and demography, parental education, health service utilization) rather than any basic (policy) determinants. Table 2 summarizes basic information on several broad sources of nutritional change linked to these intermediate determinants that are available to us in the DHS data.[4] Further discussion of some of the potential policy drivers of nutritional change in Bangladesh is also found in Ahmed *et al.* (2012) and The World Bank (2005a).

One candidate for sustained nutritional change is general economic progress, which facilitates larger expenditures on food, health, and other nutrition-relevant expenditures (Behrman & Deolalikar, 1987; Haddad *et al.*, 2003; Headey, 2013; Heltberg, 2009; Smith & Haddad, 2000). While Bangladesh has not achieved economic growth rates as impressive as China or Vietnam, it has achieved pro-poor economic growth on the back of rapid agricultural growth, successful nonfarm diversification, labor-intensive manufacturing and – more recently – a sharp upsurge in overseas workers' remittances.[5] These underlying drivers contributed to an 18-point decline in hunger prevalence over 1990–2010 (FAO, 2012), and a 23-point decline in the $1.25 poverty headcount over 1990–2010 (World Bank, 2012b). While the DHS does not contain information on income, it does collect on a consistent basis information on assets. These include ownership of household durables (TV, radio, motorcycle, tables and chairs, wardrobe), housing characteristics (floor, wall and roof materials, and access to electricity), house ownership, and farm land ownership. We use this information to construct an asset (or wealth) index. The weights of the different assets in this index were derived by running principal components analysis (PCA) for the pooled five rounds of data. The index was then adjusted to vary between 0 and 10, with 10 being the maximum score observed across all rounds. Consistent with this pro-poor growth, the value of the index rises by 50% during 1997–2011 (Table 2). More details of this appendix are given in Appendix A2.[6] Among other things, that appendix shows that our asset index performs just as well in predicting child growth outcomes as household expenditure in a recent non-DHS household survey from Bangladesh.

Another significant change in Bangladesh is the rapid expansion of education, which began in the early 1990s when the government and various development partners began subsidizing secondary education, particularly for girls via a stipend program designed to keep them in school.[7] According to the World Bank (2005a) the best estimates are that the stipend accelerated female secondary school enrollment 20% above other factors. As a result, growth rates of secondary school enrollment were three times as fast for females compared to males. Table 2 reflects this, with increases in grade attainment for women rising two-and-a-half times faster than men. The gender gap in grade attainment that existed in 1997 completely disappeared by 2011; in fact, the DHS data for 2011 show that among parents under the age of 25, mothers have almost one full year of education more than fathers. Given that maternal education has often been strongly linked to nutrition outcomes (Behrman & Wolfe, 1984; Burchi, 2012; Headey, 2013; Ruel & Alderman, 2013), these changes may

well have played a role in the reductions in child undernutrition reported above. Consistent with this rapid improvement in women's education, we also find substantial improvement in at least one indicator of women's empowerment collected across all DHS rounds, the percentage of women who report that they can travel to a health clinic by themselves (Table 2). In our robustness tests we also examine an alternative index of women's involvement in household decision-making.

In terms of health, Bangladesh is already well known for impressive improvements in a range of indicators, particularly child mortality. These achievements have taken place despite relatively low levels of spending on health, but with substantial innovations in community-based service delivery (Chowdhury *et al.*, 2013; El Arifeen *et al.*, 2013). Major successes – going back to the 1980s – include health extension worker programs, traditional birth attendants (more controversially), and programs to improve treatment of diarrhea. However, the limited budget on health expenditure – which is currently about half of the education budget – may also explain historically low levels of antenatal, neonatal and postnatal care in public hospitals and clinics. As a result, the last decade has seen a marked shift toward private and NGO provision of health care. A range of indicators of health service utilization were also available to us, including vaccinations, sources of antenatal care, place of delivery, and medical attendance at delivery. One issue of some concern is that some of these variables – such as antenatal care and place of birth – are likely to be highly correlated with factors that we can only imperfectly control for, such as household economic status. Hence, as a robustness test we also estimate models that drop these variables.

A more recent success story pertains to infrastructure development, particularly sanitation. Table 2 reports this measure at the village level. The existing literature provides a clear justification for this, since external bacteria are more dangerous to an individual than the bacteria of fellow household members (Spears, 2013; Spears *et al.*, 2013). The proportion of villages with no toilet (that is, engaging in open defecation) declined from 25.2% in 1997 to just 4.8% in 2011. One somewhat unique feature of sanitation improvements in Bangladesh is the adoption of community-led total sanitation (CLTS) approaches, which focus on behavioral change rather than raw infrastructural investments. The CLTS approach was first pioneered in Bangladesh in the late 1990s, seemingly to substantial effect (Kar, 2003; World Bank, 2012a). In light of recent evidence pointing to open defecation being a leading explanation of South Asia's unusually high rates of child undernutrition (Lin *et al.*, 2013; Mondal *et al.*, 2012; Spears, 2013; World Bank, 2012a) it is of interest to assess whether this decline in open defecation explains some of the reduction in child undernutrition in recent years. This large change in sanitation contrasts with the very small change in access to piped water (Table 2).

One of Bangladesh's most unambiguous policy success stories is the improvement in demographic outcomes (Kohler, 2012). At independence from Pakistan, Bangladesh had the highest population density in the developing world, and one of the highest fertility rates. The government and its development partners therefore placed high priority on family planning immediately after independence. Family Health Visitor (FHV) training schools were set up at scale in mid 1970s to promote contraception, and by 1978 some 16,700 family planning workers had been posted (White, Masset, Blondal, & Waddington, 2005). By the early 1990s several evaluations clearly established sizeable impacts of family planning on fertility outcomes (despite the inhospitable cultural and economic setting), most notably Cleland, Phillips, Amin, and Kamal (1994). The World Bank (2005a) also reviews this literature and empirically demonstrates that Bangladesh has achieved exceptionally rapid fertility reductions relative to its economic growth. Consistent with these changes, Table 2 shows a lengthening of birth intervals and a reduction in mean birth rank (in effect, fertility). An existing literature suggests that these fertility changes might have driven substantial nutritional improvements. Rutstein (2008) provides a comprehensive review of these linkages, and uses 265,000 stunting observations from 52 DHS surveys to identify a highly significant curvilinear relationship between preceding birth intervals and child growth, with stunting declining markedly as the birth interval increases after 18 months (we closely replicate this finding below). There is also a small literature – largely in economics – on birth order (in effect, fertility) and nutrition in developing countries (Behrman, 1988; Horton, 1988; Jayachandran & Pande, 2013). However, one challenge in linking these developments to policies is that fertility is very much a household choice variable, and therefore potentially correlated with household unobservables, though we do control for wealth and parental education, two variables that are indeed highly correlated with fertility outcomes. As a robustness check we also estimate models that exclude fertility and health outcomes from the right hand side of our models.

## 3. ANALYSIS

We use linear regression models and linear probability models to assess the associations between nutrition outcomes ($N$) for a child $i$ at time $t$ and a vector of time-varying intermediate determinants ($X$), and a vector of control variables (maternal height, child and maternal age dummies, location fixed effects; $\boldsymbol{\mu_i}$), trend effects represented by a vector of year dummy variables ($T$). The vector of coefficients ($\boldsymbol{\beta}$) constitutes the set of parameters of principal interest. With the addition of a standard white noise term ($\varepsilon_{i,t}$), we represent this relationship by Eqn. (1):

$$N_{i,t} = \boldsymbol{\beta}\mathbf{X}_{i,k} + \boldsymbol{\mu}_i + \boldsymbol{T} + \varepsilon_{i,t} \tag{1}$$

Apart from the standard least squares assumptions, a crucial assumption of the model is that we adequately control for all potentially confounding factors, with the most important of these being wealth and education, since these two variables can clearly drive a range of other endogenous behaviors, such as demand for health service utilization and the demand for the number of children (or contraception). Without adequate control for physical and human capital the coefficients on these other variables are less interesting, since they might substantially reflect increased household demand rather than any impact of sector-specific policies. Conversely, if a household's physical and human capital is adequately captured in the model, then statistically significant coefficients on other variables in the model are suggestive of important supply-side drivers in the domains of healthcare, family planning and infrastructure provision. Hence, as noted above, we estimate models that exclude potentially endogenous health and fertility variables.

A second important assumption in Eqn. (1) is that model is appropriately specified in other dimensions, particularly in terms of capturing various non-linearities in nutrition relationships. To that end we took three steps. First, we adopted a very flexible specification of the time-invariant determinants including monthly dummy variables to capture the progressive

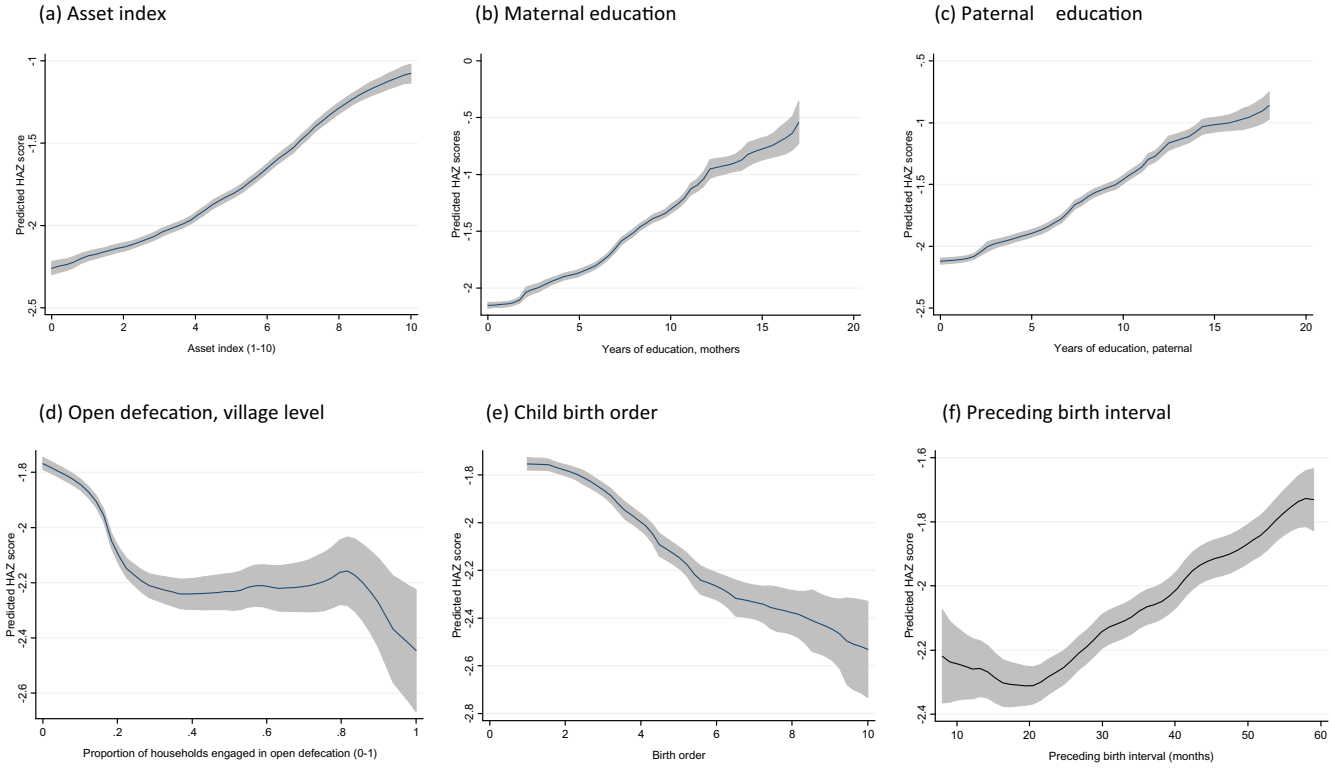


Figure 3. *Non-parametric estimates of the relationship between HAZ scores and continuous variables.*

growth faltering process that malnourished populations undergo until around two years of age (Shrimpton *et al.*, 2001; Victora, de Onis, Curi Hallal, Blössner, & Shrimpton, 2009). Second, we undertook non-parametric graphical analyses of all time-varying continuous variables to examine whether there exists non-linearities in their relationships with HAZ scores. Figure 3 shows that most of the continuous explanatory variables have approximately linear relationships with HAZ scores with two exceptions.

First, open defecation at the village level has a strikingly non-linear relationship. In the range of 0–30% open defecation (approximately) the gradient is steeply negative, but thereafter it is mostly flat, before becoming negative again for the few very high levels of open defecation. This non-linearity is quite different to Spear's (2013) findings for an Indian DHS sample, but similar to his estimates for an African DHS sample. Moreover, an earlier literature on sanitation and health outcomes had hypothesized that there were increasing health benefits as communities move toward total eradication of open defecation (Shuval, Tilden, Perry, & Grosse, 1981). To capture this non-linear relationship in our regression models we use a fractional polynomial transformation by raising this variable to the power of one-third, a transformation that performed better than alternatives that we experimented with. The second non-linear relationship pertains to birth order. Figure 3e suggests that first and second born children have similar predicted HAZ scores, but HAZ scores decline for all lower order births. Hence we interact a dummy variable for birth orders greater than two with the raw birth order variable to capture this non-linearity.

We then use the estimated parameters from Eqn. (1) to conduct a decomposition analysis, taking the first difference of Eqn. (1). Under the assumption that the $\boldsymbol{\beta}$ coefficients are time-invariant, and the error term has a mean of zero, the first difference of Eqn. (1) between time 1 and time $K$ is given by:

$$\Delta\overline{N}_{i,t} = \boldsymbol{\beta}\left(\overline{\boldsymbol{X}}_{t=K} - \overline{\boldsymbol{X}}_{t=1}\right) \quad (2)$$

where bars represent sample means.

If, however, we assume that the $\boldsymbol{\beta}$ coefficients are time varying, a different approach is needed. Specifically, we would need to use a Oaxaca–Blinder decomposition to break up the estimated change in the dependent variable into changes in endowments, changes in coefficients and interactions between the two (Jann, 2008 and Elder, Goddeeris, & Haider, 2010 provide detailed explanations of this approach). If there is a high degree of parameter stability across time, however, the two decomposition techniques are equivalent. But one limitation of the Oaxaca–Blinder analysis is that parameter instability can sometimes be an erroneous artifact of measurement issues in particular rounds. For example, our sanitation variable – open defecation – had a relatively high mean in the beginning of the sample, but was close to zero by 2011. A regression using only 2011 data therefore indicates that open defecation is not a significant determinant of nutrition precisely because there is so little variation in this indicator by the end of our period of analysis. In light of this we would prefer to avoid the Oaxaca–Blinder decomposition if the regressions show a sufficiently high degree of parameter stability across rounds. In the next section we therefore examine parameter stability using a barrage of Chow tests to test for differences in the $\boldsymbol{\beta}$ coefficients across rounds. We find little evidence of parameter instability, though we also conduct Oaxaca–Blinder decompositions as a further robustness test.

## 4. RESULTS

We now turn to formally deriving answers to two related but distinct questions: which factors explain growth outcomes across children, and which factors explain child growth trends

Table 3. *HAZ regressions pooled across years for various samples*

| Model | Full sample (baseline) | Rural only | Urban only | Boys only | Girls only | 0–6 months | 7–24 months |
|---|---|---|---|---|---|---|---|
| Asset index, 1–10 | 0.046*** | 0.045*** | 0.051*** | 0.043*** | 0.050*** | 0.019 | 0.051*** |
| | 0.004 | 0.005 | 0.008 | 0.006 | 0.006 | 0.013 | 0.008 |
| Maternal educ. (yrs) | 0.016*** | 0.014*** | 0.016*** | 0.021*** | 0.011** | 0.023** | 0.009* |
| | 0.003 | 0.004 | 0.006 | 0.005 | 0.004 | 0.009 | 0.005 |
| Paternal educ. (yrs) | 0.020*** | 0.016*** | 0.029*** | 0.018*** | 0.023*** | 0.024*** | 0.020*** |
| | 0.002 | 0.003 | 0.005 | 0.004 | 0.004 | 0.008 | 0.004 |
| All vaccines[a] | 0.037* | 0.027 | 0.051 | 0.028 | 0.046 | | 0.071** |
| | 0.02 | 0.023 | 0.04 | 0.029 | 0.028 | | 0.032 |
| Prenatal doctor visit | 0.176*** | 0.165*** | 0.189*** | 0.163*** | 0.190*** | 0.161** | 0.202*** |
| | 0.021 | 0.025 | 0.041 | 0.03 | 0.03 | 0.063 | 0.036 |
| Prenatal HP visit | 0.027 | 0.023 | 0.061 | 0.022 | 0.034 | 0.055 | 0.008 |
| | 0.022 | 0.026 | 0.043 | 0.031 | 0.031 | 0.065 | 0.038 |
| Born in med. facility | 0.115*** | 0.096*** | 0.092** | 0.121*** | 0.112*** | −0.051 | 0.134*** |
| | 0.027 | 0.037 | 0.039 | 0.037 | 0.038 | 0.08 | 0.044 |
| Open defecation[b] | −0.027*** | −0.023*** | −0.054*** | −0.041*** | −0.008 | −0.023 | −0.041*** |
| | 0.007 | 0.008 | 0.018 | 0.01 | 0.01 | 0.022 | 0.013 |
| Piped water | 0 | 0.065 | −0.019 | 0.027 | −0.026 | −0.193* | 0.006 |
| | 0.034 | 0.092 | 0.04 | 0.048 | 0.048 | 0.107 | 0.061 |
| Birth order | −0.038*** | −0.031*** | −0.065*** | −0.041*** | −0.034*** | 0.036* | −0.034*** |
| | 0.007 | 0.008 | 0.016 | 0.01 | 0.01 | 0.021 | 0.012 |
| Birth interval (yrs)# | 0.028*** | 0.028*** | 0.031*** | 0.032*** | 0.024*** | 0.034*** | 0.029*** |
| | 0.003 | 0.004 | 0.006 | 0.005 | 0.005 | 0.01 | 0.006 |
| Health clinic alone | 0.006 | 0.014 | −0.008 | 0.036 | −0.027 | 0.093* | −0.017 |
| | 0.017 | 0.021 | 0.031 | 0.024 | 0.024 | 0.053 | 0.03 |
| Maternal height | 0.051*** | 0.050*** | 0.054*** | 0.054*** | 0.049*** | 0.045*** | 0.056*** |
| | 0.002 | 0.002 | 0.003 | 0.002 | 0.002 | 0.004 | 0.003 |
| Male child | −0.009 | 0.014 | −0.062** | – | – | −0.075 | −0.094*** |
| | 0.016 | 0.019 | 0.029 | | | 0.047 | 0.027 |
| Year 2000 | 0.219*** | 0.208*** | 0.200*** | 0.205*** | 0.237*** | 0.086 | 0.136*** |
| | 0.028 | 0.03 | 0.063 | 0.041 | 0.039 | 0.083 | 0.049 |
| Year 2004 | 0.202*** | 0.216*** | 0.130** | 0.183*** | 0.223*** | −0.026 | 0.125** |
| | 0.028 | 0.03 | 0.061 | 0.04 | 0.039 | 0.083 | 0.049 |
| Year 2007 | 0.272*** | 0.298*** | 0.180*** | 0.287*** | 0.270*** | 0.059 | 0.308*** |
| | 0.031 | 0.034 | 0.062 | 0.044 | 0.043 | 0.104 | 0.054 |
| Year 2011 | 0.270*** | 0.307*** | 0.154** | 0.248*** | 0.296*** | 0.275*** | 0.03 |
| | 0.03 | 0.033 | 0.062 | 0.043 | 0.042 | 0.095 | 0.053 |
| *R*-squared | 0.252 | 0.235 | 0.273 | 0.251 | 0.263 | 0.134 | 0.233 |
| *N* | 23114 | 16651 | 6463 | 11776 | 11338 | 3124 | 7964 |

*Notes:* Village-lustered standard errors are used to estimate significance levels. *, **, and *** indicate significance at 10%, 5%, and 1% levels, respectively. The regressions above include a number of time-invariant controls, including maternal height, regional fixed effects, month-specific child age dummy variables, and dummy variables for various categories of maternal age.

[a] "All vaccines" is measured for children 6 months and older only.

[b] "Open defecation" is measured as a cubic fraction to capture its non-linear relationship to nutrition outcomes.

over 1997–2011? Our estimates of Eqn. (1) are reported in Table 3. While these are based on pooling all five rounds together, we account for trend effects through a series of year dummy variables with the 1997 round as the base, and present separate regressions for different sub-samples of the pooled data. All our regressions include time-invariant control variables (monthly child age dummies, maternal age bracket dummies, month of birth dummies, regional dummies) but these are omitted from Table 6 for the sake of brevity.

We find moderately large impacts of household wealth on HAZ scores with the impact of a 1-point increase in the asset index (measured on a 1–10 scale) usually predict around a 0.05 standard deviation increase in HAZ scores. In other words, the predicted HAZ difference between a child in the poorest household in our sample and the richest is 0.5 standard deviations (the exception is the sample of children 0–6 months of age, in which household wealth has no significant effect on HAZ scores). For both maternal and paternal education we find that an extra year typically adds 0.01–0.02 standard deviations to predicted HAZ scores, such that a household in which both parents completed high school could be expected to have a child around 0.5 standard deviations taller than a child from a household in which neither parent had attended school. In this sense the impact of household assets and household education is somewhat similar in magnitude. Also of note is that the nutritional impacts of maternal and paternal education are never statistically different from each other.

We do not find any significant association between our female empowerment variable (can walk to health clinic alone) and growth outcomes, although this variable may capture only limited dimensions of empowerment. However, when we tested a simple index of maternal involvement in four areas of household decision-making – an index only available from the 2000 round onward – we found no significant coefficient on this variable either (results available on request).

In terms of health variables we find no robust impact of vaccinations, though the coefficient on this variable is moderately significant in some samples, particularly the 7–24-month sample. Prenatal visits to doctors has a highly significant and stable coefficient across samples, predicting a relatively large impact on HAZ scores varying between 0.16 and 0.21 standard deviations. However, visits to other health professionals

Table 4. *HAZ regressions for the full sample by round with tests for coefficient differences over time*

| Model | 1997 | 2000 | 2004 | 2007 | 2011 | Significant differences? |
|---|---|---|---|---|---|---|
| Asset index, 1–10 | 0.052*** | 0.041*** | 0.066*** | 0.029*** | 0.036*** | No |
| | 0.011 | 0.01 | 0.009 | 0.01 | 0.009 | |
| Maternal educ. (yrs) | 0.022** | 0.021*** | 0.017** | 0.011 | 0.017*** | No |
| | 0.008 | 0.008 | 0.007 | 0.007 | 0.006 | |
| Paternal educ. (yrs) | 0.026*** | 0.017*** | 0.013** | 0.022*** | 0.022*** | No |
| | 0.007 | 0.006 | 0.006 | 0.005 | 0.005 | |
| All vaccines[a] | −0.071* | 0.04 | 0.065 | −0.009 | 0.096** | Yes |
| | 0.042 | 0.043 | 0.044 | 0.051 | 0.047 | |
| Prenatal doctor visit | 0.178*** | 0.221*** | 0.195*** | 0.181*** | 0.109*** | No |
| | 0.053 | 0.049 | 0.046 | 0.051 | 0.042 | |
| Prenatal HP visit | 0.085 | −0.025 | 0.067 | 0.025 | −0.025 | No |
| | 0.066 | 0.059 | 0.045 | 0.049 | 0.041 | |
| Born in med. facility | −0.039 | 0.052 | 0.175*** | 0.196*** | 0.128*** | Yes |
| | 0.114 | 0.076 | 0.066 | 0.061 | 0.04 | |
| Open defecation[b] | −0.032* | −0.031* | −0.056*** | −0.039** | −0.008 | No |
| | 0.016 | 0.018 | 0.017 | 0.019 | 0.018 | |
| Piped water | 0.063 | −0.03 | −0.059 | −0.029 | 0.04 | No |
| | 0.112 | 0.082 | 0.072 | 0.082 | 0.06 | |
| Birth order | −0.033** | −0.039*** | −0.029* | −0.041** | −0.041** | No |
| | 0.015 | 0.015 | 0.015 | 0.018 | 0.016 | |
| Birth interval (yrs) | 0.014 | 0.037*** | 0.049*** | 0.027*** | 0.016*** | Yes |
| | 0.009 | 0.008 | 0.007 | 0.008 | 0.006 | |
| Health clinic alone | 0.012 | 0.034 | −0.03 | −0.058 | 0.057* | No |
| | 0.048 | 0.042 | 0.037 | 0.04 | 0.032 | |
| Maternal height (cm) | 0.046*** | 0.049*** | 0.054*** | 0.057*** | 0.053*** | Yes |
| | 0.004 | 0.004 | 0.003 | 0.004 | 0.003 | |
| *R*-squared | 0.253 | 0.279 | 0.258 | 0.251 | 0.224 | |
| *N* | 4512 | 4019 | 4750 | 3962 | 5871 | |

*Notes:* Clustered standard errors are used to estimate significance levels. *, **, and *** indicate significance at 10%, 5%, and 1% levels, respectively. The regressions above include a number of time-invariant controls, including regional fixed effects, month-specific child age dummy variables, and dummy variables for various categories of maternal age.

[a] "All vaccines" is measured for children 6 months and older only.

[b] "Open defecation" is measured as a cubic fraction to capture its non-linear relationship to nutrition outcomes.

Table 5. *Decomposing sources of nutritional change for the full sample, 1997–2011*

| | (1) Estimated coefficient | Sample mean: 1997 | Sample mean: 2011 | (2) Change in means | Predicted change in HAZ = (1) * (2) | Share of predicted change (%) |
|---|---|---|---|---|---|---|
| HAZ score (dependent variable) | | −2.20 | −1.62 | 0.58 | 0.31 | 100.0 |
| Asset index, 1–10 | 0.046 | 3.2 | 4.9 | 1.70 | 0.08 | 25.1 |
| Maternal educ. (yrs) | 0.016 | 2.4 | 5.4 | 3.00 | 0.05 | 15.4 |
| Paternal educ. (yrs) | 0.02 | 3.6 | 5.1 | 1.50 | 0.03 | 9.6 |
| Prenatal doctor visit | 0.176 | 19.50% | 34.2% | 14.7% | 0.03 | 8.3 |
| Born in med. facility | 0.115 | 2.90% | 23.6% | 20.7% | 0.02 | 7.6 |
| Open defecation | −0.027 | 2.30 | 0.90 | −1.40 | 0.04 | 12.1 |
| Birth order | −0.038 | 3.1 | 2.5 | −0.60 | 0.02 | 7.3 |
| Birth interval (yrs) | 0.028 | 2.4 | 3.0 | 0.60 | 0.02 | 5.4 |
| Maternal height (cm) | 0.051 | 150.4 | 150.9 | 0.55 | 0.03 | 9.0 |
| Ratio of predicted HAZ change to actual (%) | | | | | *53.3%* | |

*Source:* Authors' estimates from the 1996–97 and 2011 BDHS rounds.

– such as nurses and midwives – does not significantly predict HAZ scores (though in results below we find that this variable predicts improvement in severe stunting). A child being born in any kind of medical facility (government, private, NGO) is a robust predictor of HAZ with coefficients varying between 0.09 and 0.13. It therefore appears that access to doctors and larger medical facilities before and during birth is important for child growth around the mean, although we note that the importance of these factors may be exaggerated by their associations with household economic status.

Consistent with the graphical result in Figure 3, panel 3d, open defecation has a robust but non-linear negative association linear growth outcomes. As in Spears (2013), we find that that the impact of open defecation is somewhat larger in urban areas, presumably because of greater population density strengthening the disease vectors associated with open defecation.[8] In contrast to sanitation, we find no effect of piped water supplies on growth outcomes. It is likely that water sources are a poor proxy for water quality, especially in the absence of data on whether water supplies were appropriately treated or not.

Both household demography variables – birth order and birth interval – yield statistically significant coefficients that are also quite stable across samples. Individually, each

Table 6. *Comparing the baseline model to stunting, severe stunting and models with maternal BMI included*

| Sample | Full sample | Full sample | Full sample | Full sample | 0–6 months | Full sample |
|---|---|---|---|---|---|---|
| Model | HAZ, baseline | Stunting | Severe stunting | HAZ, BMI added | HAZ, BMI added | HAZ, no health or fertility variables |
| Estimator | OLS | LPM | LPM | OLS | OLS | OLS |
| Asset index, 1–10 | 0.046*** | −0.014*** | −0.010*** | 0.040*** | 0.015 | 0.057*** |
| | 0.004 | 0.002 | 0.001 | 0.004 | 0.013 | 0.004 |
| Maternal educ. (yrs) | 0.016*** | −0.005*** | −0.004*** | 0.015*** | 0.022** | 0.023*** |
| | 0.003 | 0.001 | 0.001 | 0.003 | 0.009 | 0.003 |
| Paternal educ. (yrs) | 0.020*** | −0.007*** | −0.003*** | 0.019*** | 0.022*** | 0.024*** |
| | 0.002 | 0.001 | 0.001 | 0.002 | 0.008 | 0.002 |
| All vaccinations# | 0.037* | −0.01 | −0.018*** | 0.034* | | |
| | 0.02 | 0.008 | 0.007 | 0.02 | | |
| Prenatal doctor visit | 0.176*** | −0.059*** | −0.038*** | 0.163*** | 0.159** | |
| | 0.021 | 0.008 | 0.007 | 0.021 | 0.063 | |
| Prenatal HP visit | 0.027 | −0.012 | −0.023*** | 0.027 | 0.054 | |
| | 0.022 | 0.009 | 0.007 | 0.022 | 0.065 | |
| Born in med. Facility | 0.115*** | −0.035*** | 0.008 | 0.088*** | −0.067 | |
| | 0.027 | 0.01 | 0.007 | 0.027 | 0.08 | |
| Open defecation# | −0.027*** | 0.010*** | 0.009*** | −0.024*** | −0.02 | −0.026*** |
| | 0.007 | 0.003 | 0.002 | 0.007 | 0.022 | 0.008 |
| Piped water | 0.000 | 0.011 | 0.01 | −0.019 | −0.203* | |
| | 0.034 | 0.012 | 0.009 | 0.034 | 0.107 | |
| Birth order | −0.038*** | 0.012*** | 0.013*** | −0.036*** | 0.037* | |
| | 0.007 | 0.003 | 0.002 | 0.007 | 0.021 | |
| Birth interval (yrs)# | 0.028*** | −0.009*** | −0.008*** | 0.026*** | 0.033*** | |
| | 0.003 | 0.001 | 0.001 | 0.003 | 0.01 | |
| Health clinic alone | 0.006 | 0.001 | −0.001 | 0.007 | 0.098* | |
| | 0.017 | 0.007 | 0.005 | 0.017 | 0.053 | |
| Maternal height | 0.051*** | −0.017*** | −0.011*** | 0.052*** | 0.045*** | 0.051*** |
| | 0.002 | 0.001 | 0 | 0.002 | 0.004 | 0.002 |
| Male child | −0.009 | 0.006 | 0.006 | −0.008 | −0.076 | |
| | 0.016 | 0.006 | 0.005 | 0.016 | 0.047 | |
| Maternal BMI, log | | | | 0.625*** | 0.432** | |
| | | | | 0.062 | 0.18 | |
| *R*-squared | 0.252 | 0.181 | 0.121 | 0.256 | 0.136 | 0.239 |
| *N* | 23114 | 23114 | 23114 | 23106 | 3123 | 27130 |

*Notes:* Clustered standard errors are used to estimate significance levels. *, **, and *** indicate significance at 10%, 5%, and 1% levels, respectively. The regressions above include a number of time-invariant controls, including regional fixed effects, month-specific child age dummy variables, and dummy variables for various categories of maternal age. LPM refers to the Linear Probability Model.

variable has modest slope coefficients, but together these two variables suggest that household demography is an important predictor of HAZ scores. In the full sample, every additional child (after the second child) has a predicted HAZ score that is 0.03–0.04 standard deviations lower that the next highest order child. So relative to first and second children, a child born sixth in a family could be expected to be around 0.2 standard deviations shorter. Similarly, an extra year between births increases HAZ scores by around 0.03 standard deviations, so a five year gap yields a 0.15 standard deviation improvement in height.

The coefficient on maternal height is significant in all samples, representing an important intergenerational transmission of nutrition. Male children have some tendency to be smaller, at least in urban areas and in the samples of younger children. Indeed, graphical results reported in Appendix A (Figure A1.3) suggest that girls are slightly taller than boys until three years of age, but thereafter boys become significantly taller than girls.

Another finding of note is that many of the variables that appear to be robustly significant across different samples turn out to be insignificant in the sample of children aged 0–6 months. As noted above, wealth appears irrelevant for this sample, but so too does being born in a medical facility and village sanitation, though parental education is still important, as is prenatal doctor visits and the demographic variables.

The results above answer the question of what the general determinants of malnutrition are in Bangladesh. We now turn to the second question: which of these significant determinants of nutrition appear to have driven changes in nutritional outcomes over this period of rapid progress? In order to implement a decomposition of the sources of predicted change over time, we first need to establish whether there is substantive evidence of systematic changes in coefficients across rounds, which would render a simple linear decomposition inappropriate. Table 4 reports two types of evidence to inform this issue. First, we report regressions for the full sample (total population of children 0–59 months). The changes in point estimates and standard errors across rounds give an approximate idea of coefficient stability and any potential trends in coefficients over time that might be suggestive of meaningful change (as opposed to just random change). Second, the last column of Table 4 summarizes the results of formal tests of differences in parameter values. Specifically we conducted Chow tests for significant differences between the coefficients of each *X* variable in the 1997 round against all the other rounds. The full results of these tests are reported in Appendix A7.

There are few signs of significant changes in coefficients across rounds. Consistent with results above, the coefficient on all vaccines shows some signs of instability. In fact, this variable is only significant in the 2011 round, and its coefficient sometimes changes signs across rounds. We interpret this as further evidence that vaccinations are not a strong predictor of changes in HAZ scores, though we show some evidence below that they are a reasonably strong predictor of changes in severe stunting. Being "born in a medical facility" has insignificant coefficients in the first two rounds when medical facility births were relatively rare (with sample means of just 3.1% and 5.8%). Third, "open defecation" likewise has an insignificant coefficient in the last round when there was again very little variation (a mean of just 4.2%). Fourth, the coefficient on birth intervals shows some signs of instability. In 1997 the coefficient is positive but insignificant, but the coefficient is then relatively large in 2000 and 2004, and then more modest thereafter. Chow tests suggest that the coefficients in 2000 and 2004 are significantly different from 1997, but that the 2007 and 2011 coefficients are not significantly different from 1997. Finally, we find very small but statistically significant differences in the coefficients attached to maternal height.

Based on these tests, we conclude that there are few signs of any secular changes in the main parameters of interest, and that some of the changes we do observe are driven by lack of variation in certain variables in either the beginning or end rounds. Oaxaca–Blinder decompositions using starting and end rounds would therefore suggest that changes in these variables had no significant impact on HAZ scores, a conclusion that would appear erroneous based on our pooled regression results. We therefore prefer the simple linear decompositions described in the previous section, in which we use the coefficients from Table 3.

Table 5 reports detailed decomposition results for the full sample using only those variables which are statistically significant at the 5% level or higher in the first column of Table 3. The first column reports the estimated coefficient from that regression. The next three columns respectively report the 1997 and 2011 sample means and the change in means across time. The predicted change in HAZ scores is the product of this change in means and the estimated coefficient (for example, the predicted change in HAZ scores resulting from asset accumulation is $0.05 \times 1.65 = 0.08$). The last column reports the share of predicted change accounted for by each variable.

There are two important findings from Table 5. First, the model explains just over half (55.3%, or 0.31 standard deviations) of the actual change in HAZ scores observed over this period (0.58 standard deviations). Second, among the sources of predicted change, wealth accumulation stands out as the single largest factor, explaining 25.1% of the predicted change in HAZ scores. However, when maternal and paternal education are combined they are an equally important factor (25%), with the bulk of the change resulting from the more rapid accumulation of education among the female population. After wealth and human capital accumulation, health factors emerge as the third most important factor, with prenatal doctor visits and medical facility births jointly accounting for 16.1% of the predicted change. However, it is difficult to link this contribution to explicit policies or programs, though it is certainly possible that both public and NGO programs played a role in facilitating these improvements in health service utilization.[9] Finally, open defecation accounts for a relatively modest 12.1% of the predicted change, demographic changes (lower mean birth orders and shorter birth intervals) jointly account for 12.7% of the total change, and improvements in maternal height account for just 9%.

## 5. ROBUSTNESS TESTS

The decomposition in Table 5 – and the regression underlying it – is obviously only one of many ways of analyzing these data, and there are a number of conceptual and statistical reasons to consider a range of alternative samples, estimators, and specifications. This section therefore describes the results of the following tests. First, we decompose changes in stunting and severe stunting instead of changes in mean HAZ scores. Second, we use quantile regressions as an alternative means of exploring these distributional issues.[10] Third, we add maternal BMI to the model on the grounds that this indicator of maternal undernutrition may help explain increases in child growth in early life (0–6 months), including birth size. Fourth, we perform decompositions for rural and urban sub-samples. Fifth, we perform decompositions for under-twos instead of under-fives. Sixth, we estimate models that exclude indicators of healthcare and demographic variables, on the grounds that these might be potentially endogenous. Some of these results are reported in Table 6, while others are reported in the Appendix.

Starting with the switch from the continuous HAZ variable to stunting and severe stunting, we find that the stunting results are quite similar to the HAZ results (unsurprisingly, since the mean HAZ score in the pooled sample is close to −2). Moreover, the quantitative significance of the results also appears broadly similar (for example, the ratio of the asset index coefficient to the two educational coefficients). However, when we consider severe stunting, we find that prenatal visits with other health professionals – mostly government or NGO health workers – yield a statistically significant coefficient that is strongly associated with reducing the risk of severe stunting. By contrast, being born in a medical facility is no longer an important predictor, perhaps because only children of much wealthier households tend to be born in medical facilities. Also note that while Table 6 reports Linear Probability Model estimates, we also estimated stunting and severe stunting regressions with the LOGIT model, and found a similar pattern of results.[11]

Our quantile regressions results (reported in the Appendix) tell a similar story to the stunting and severe stunting results. Results at the 50th HAZ quantile (the median) are very similar to the OLS results, as expected, but results at the 25th HAZ quantile (which is −2.9) are similar to the regressions on severe stunting: vaccinations and prenatal visits from other health professionals now become significant. The only difference is that being born in a medical facility still significantly predicts child growth at the 25th percentile.

Table 6 also reports results from adding the log of maternal BMI to the model. We add this variable for an estimation on the full sample, and for the restricted sample of children aged 0–6 months. In the full sample we observe a large and highly significant coefficient of 0.63 on this variable. As noted above, however, this coefficient could be biased because maternal BMI and child nutrition outcomes could be jointly correlated with some unobservable third factor(s). Since we primarily expect maternal undernutrition to influence child growth through intrauterine growth and breastfeeding in the first few months of life (when breastfeeding is typically exclusive), restricting the sample to children aged 0–6 months may give a truer estimate of the relevance of maternal undernutrition. When we implement this restriction the coefficient on maternal BMI drops from 0.63 to 0.43. We use this more conservative marginal effect of 0.43 in our decomposition below.

Another endogeneity concern pertains to the health and fertility variables. The last column in Table 6 reports this result

Table 7. *Decomposition based on alternative samples and dependent variables*

| Dependent variable | HAZ | HAZ | HAZ | HAZ | Stunting | Severe stunting | HAZ |
|---|---|---|---|---|---|---|---|
| Area | All | Rural | Urban | All | All | All | All |
| Model | Full | Full | Full | BMI added | Full | Full | No health, fertility |
| *Disaggregated sources of predicted change* | | | | | | | |
| Asset index, 1–10 | 25.1% | 24.0% | 21.8% | 17.8% | 22.6% | 20.6% | 33.7% |
| Maternal educ. (yrs) | 15.4% | 15.4% | 13.3% | 12.2% | 14.9% | 14.5% | 26.4% |
| Paternal educ. (yrs) | 9.6% | 8.2% | 12.5% | 8.6% | 11.0% | 6.5% | 14.5% |
| All vaccines (0–1) | | | | | | 5.7% | |
| Prenatal doctor visit | 8.3% | 8.4% | 7.0% | 7.7% | 9.0% | 8.0% | |
| Prenatal other HP | | | | | | 5.3% | |
| Born in med. Facility | 7.6% | 8.6% | 6.5% | 5.2% | 7.2% | | |
| Open defecation | 12.1% | 12.3% | 19.7% | 9.3% | 13.3% | 16.0% | 13.9% |
| Birth order | 7.3% | 7.7% | 11.9% | 6.8% | 8.3% | 11.2% | |
| Birth interval (yrs) | 5.4% | 4.4% | 3.3% | 2.8% | 3.4% | 4.0% | |
| Maternal height (cm) | 9.0% | 10.9% | 4.1% | 20.1% | 10.2% | 8.2% | 11.5% |
| Maternal BMI | | | | 9.5% | | | |
| Sum | 100.0% | 100.0% | 100.0% | 100.0% | 100.0% | 100.0% | 100.0% |
| *Aggregated sources of predicted change* | | | | | | | |
| Wealth | 25.1% | 24.0% | 21.8% | 17.8% | 22.6% | 20.6% | 33.7% |
| Education | 25.0% | 23.6% | 25.7% | 20.8% | 25.9% | 21.0% | 40.9% |
| Health | 15.9% | 17.0% | 13.5% | 12.9% | 16.3% | 19.0% | |
| Sanitation | 12.1% | 12.3% | 19.7% | 9.3% | 13.3% | 16.0% | 13.9% |
| Demography | 12.7% | 12.1% | 15.2% | 9.6% | 11.7% | 15.2% | |
| Maternal nutrition | 9.0% | 10.9% | 4.1% | 29.6% | 10.2% | 8.2% | 11.5% |
| Sum | 100.0% | 100.0% | 100.0% | 100.0% | 100.0% | 100.0% | |
| *Predictive power of model* | | | | | | | |
| (1) Actual change in nutrition | 0.58 | 0.57 | 0.58 | 0.58 | −0.19 | −0.16 | 0.58 |
| (2) Predicted change in nutrition | 0.31 | 0.24 | 0.37 | 0.39 | −0.10 | −0.08 | 0.28 |
| Predictive power (%): (2)/(1) | 53.3% | 47.8% | 67.6% | 63.9% | 56.1% | 49.1% | 47.4% |

for HAZ scores. As might be expected, we find that excluding health and fertility variables does affect the coefficient on the asset index and the maternal education variable (since maternal education tends to affect fertility rates more than paternal education; see Schultz, 1997). However, by far the larger effect is on the asset index coefficient, which increases by 24% (a similar result holds for stunting and severe stunting – see the Appendix). This larger coefficient on the asset index will obviously mean that a larger share of the nutritional change over 1997–2011 may be attributed to wealth (see below).

Indeed, in Table 7 we examine how the different models and samples used in Tables 3 and 6 influence the decomposition results. Specifically, we compare the baseline full sample HAZ result to results for rural and urban areas, to models with stunting and severe stunting as the dependent variables, and to the model that includes maternal BMI as an explanatory variable. Along with the contributions of each variables to total predicted change, we also report more aggregated effects (e.g., maternal and paternal education are aggregated into "education") and also how well each model does in terms of explaining actual changes in the dependent variables.

Overall, the conclusions of the baseline model are fairly robust, though with some exceptions. Switching to a rural sample makes almost no difference, but switching to the urban sample unsurprisingly increases the contribution of improved sanitation to nutritional change from 12.5% in the baseline to 19.7% in urban areas. The model for urban areas also has greater explanatory power, accounting for two-thirds of the actual change in HAZ scores in urban Bangladesh. The model with maternal BMI added as an explanatory variable also explains about two-thirds of the actual change in HAZ scores for the country as a whole, and maternal height and body mass together account for almost 30% of the predicted change (even after using the more conservative marginal effect of maternal BMI), making improvements in maternal undernutrition the single largest driver of change. However, it is far from obvious what has driven this improvement in maternal BMI. Switching to stunting instead of HAZ scores makes almost no difference to the contributions of the different factors, but switching to severe stunting suggests that vaccinations and prenatal visits have contributed significantly to reducing severe undernutrition. Moreover, in this model wealth, education, and health variables each account for about 20% of the predicted change.

Finally, if we view the health and fertility variables with some suspicion and exclude them from the model, then – as expected – wealth accumulations become a much more important factor, accounting for around one third of the predicted change in mean HAZ scores. In this model, asset accumulation and parental educational gains account for almost two-thirds of the predicted change.

## 6. CONCLUSIONS

While South Asia is generally synonymous with high rates of undernutrition and poor progress against this problem, Bangladesh has managed to consistently reduce rates of stunting for at least two decades. Given that this progress was seemingly achieved without the aid of highly effective nutrition programs, this paper sought to understand which "nutrition-sensitive" factors appear to have been driving these changes.

Our principal finding is that the process of nutritional change in Bangladesh has been highly multidimensional. Bangladesh's experience shows that it is possible to achieve rapid and sustained nutritional change even in the absence of large and effective nutritional programs, provided that there is sufficient broad economic and social development.

Economic development (as reflected by wealth accumulation at the household level) and rapid gains in education (maternal and paternal) typically emerge as the two most important factors. Both factors have been heavily influenced by policies and investments, particularly the secondary school stipend for girls in the case of education. But the fact that health, sanitation, and demographic variables are highly significant after controlling for wealth and parental education might suggest that supply side factors in these sectors have played an important role too (especially if the asset index is a strong enough predictor of household economic status). From very low bases, utilization of antenatal and neonatal care has improved rapidly. Some of this change was led by the private sector, though expansion of NGO and government sector maternal healthcare seems to have played an important role in reducing severe stunting.

The government and NGO sectors likely played an even more central role in reducing fertility rates and increasing birth spacing. Indeed, the speed of fertility decline was even more rapid prior to 1997 than afterward (when economic growth and educational gains were more limited), suggesting that family planning policies have played a very important role in explaining the longer term decline in child undernutrition in Bangladesh. To investigate what role the longer term decline in fertility rates since the 1970s might have had on undernutrition rates we therefore conducted a simple "backcasting exercise. Using fertility rates and birth spacing results for 1975 (reported in Cleland *et al*., 1994) and our baseline regression results, we estimate that improvements in demographic outcomes over 1975–2011 accounted for a 0.21 standard deviation increase in child growth HAZ scores and a 6.7 point decline in stunting prevalence over this period.[12] Thus, Bangladesh's long-term emphasis on proactive family planning seems to have had sizeable benefits in reducing undernutrition, in addition to other documented benefits in terms of maternal and child mortality outcomes (Joshi & Schultz, 2013).

Finally, consistent with emerging evidence on its importance in explaining India's unusually high undernutrition rate (Spears, 2013), it appears that major efforts to reduce open defecation have played an important role in improving child growth outcomes in Bangladesh, though more so in urban areas. Also of interest is the respective role of traditional government-led investment in sanitation infrastructure, as opposed to the largely behaviorally-oriented Community-led Total Sanitation (CLTS) campaigns. Unfortunately, the DHS data are not able to shed light on this, but this seems an area for future research.

Finally, we offer some conjectures on an important limitation of our empirical models; namely that they typically account for just over half of the actual change in child nutrition outcomes. What might explain the residual changes not accounted for by our models? In a statistical sense, measurement and misspecification errors likely account for this, including omitted variables. One such set of variables may pertain to nutrition-specific interventions, which might have been more widespread and more effective than previous research has suggested. Consistent with this hypothesis, the DHS data suggest that the proportion of children aged 6–9 months who were introduced to solid foods rose from an appallingly low 21.9% in 1996–97 to around 70% by 2004. Statistically, we found that only a small portion of this change is accounted for by gains in wealth and paternal education, so it is possible that nutritional programs did play some role, especially in a country where a wide range of NGOs and international development agencies were implementing nutritional programs in a highly decentralized setting.

A second possible explanation of this residual nutritional enigma is that the asset index does not satisfactorily measure the substantial improvements in food security in Bangladesh, which are largely the result of the country's rapid agricultural development (World Bank, 2005a). Bangladesh's rapid agricultural growth was largely led by a 70% increase in rice production over 1997–2011. There are several pieces of circumstantial evidence pointing to a significant impact of rice productivity growth on maternal and child nutrition outcomes. First, nutritional improvement was much more rapid in rural (i.e., agricultural) communities than in urban communities (Section 2, Table 1). Second, a particularly important phenomenon in Bangladesh has been the rise of the once secondary dry season rice crop, the irrigated *boro* crop. In 1996–97 the *boro* crop accounted for only 40% of total rice production, but by 2011 it accounted for 58%. The rise of such a productive second season crop may have substantially reduced seasonal deprivation of nutrients, and reduced any intrahousehold rationing of food that may have adversely affected mothers and young children. Finally, the coefficients on the time dummies in Table 3 suggests that our model performs particularly poorly in the 1996–97 to 2000 period when *boro*-led growth in rice production was especially rapid, growing by 60% in just four years.

These hypotheses remain entirely conjectural, though consistent with circumstantial evidence. Exploring them further should be a priority objective of future research.

## NOTES

1. Ahmed *et al*. (2012) provide a recent study that does examine trends in a variety of maternal and child nutrition indicators for Bangladesh, up to the 2007 DHS round. While they note significant progress in reducing undernutrition in the 1990s, they argue that there was a stalling of progress in the 2000s, which would seem to contradict our more positive assessment. This discrepancy is largely due to their focus on child underweight prevalence rather than stunting, although there was indeed some slowdown in stunting from 2000 to 2004, largely in urban areas. Since underweight combines chronic with acute dimensions of undernutrition, however, it is no longer regarded by nutritionists as the preferred measure of chronic undernutrition.

2. However, several authors in the statistical epidemiology literature have persuasively argued against the use of dichotomous rather than continuous variables on the grounds that dichotomizing variables unnecessarily weakens the power of statistical tests (Royston, Altman, & Sauerbrei, 2006; Weinberg, 1995). In our case our pooled sample size is large enough to greatly reduce this concern, but we nevertheless focus on the full spectrum of HAZ scores, as well as rates of moderate (HAZ $< -2$) and severe stunting (HAZ $< -3$).

3. We also note that while it would be possible to analyze changes in other child nutrition indicators such as wasting or underweight prevalence, these indicators reflect short-run factors more than stunting or HAZ scores. Among other problems, the various DHS rounds were not conducted in the same months of the year. Given that wasting is highly seasonal in Bangladesh, it is not even obvious that wasting rates are strictly comparable over time.

4. In our introduction, we noted that Bangladesh's largest nutrition-specific intervention (the BINP) was not particularly successful. Beyond BINP, however, there is little evidence on the impacts and scale of the

myriad of other nutrition-specific interventions carried out by government and NGO bodies. Some very specific interventions have met with some success, particularly maternal Vitamin A and iron supplementation, but the general perception of the literature is that nutrition interventions have been marginalized relative to basic health and family planning objectives (Taylor, 2012). Consequently, the absence of information on standard nutrition-specific in the DHS is not a concerns for our analysis.

5. Also of note is some expansion of social protection programs over the longer run. However, social protection expenditures stayed roughly constant in real terms over the period in question, and therefore appears a more modest driver of asset accumulation relative to agricultural growth, wage income growth, and remittances. See Ahmed, Hossain, & Chowdhury (2009) for an overview and analysis of various social protection programs in Bangladesh.

6. Such indices are now standard in the analysis of DHS data and have been demonstrated to be very efficacious (Filmer & Pritchett, 2001; Filmer & Scott, 2012; Rutstein & Staveteig, 2013). We also find that our index is a strong predictor of nutrition and other welfare outcomes, including household expenditure (see Appendix A).

7. From the 1990 to 2010, public expenditures on education increase by 350%, although most of the actual service delivery in the education sector was through private institutions.

8. The regressions also suggest that sanitation is not a significant determinant of nutrition for girls, although this peculiar result appears to stem from multicollinearity, particularly sensitivity to the inclusion of the asset index in the regression for girls. Moreover, bivariate graphical results (available on request) suggest no significant difference in the sanitation–HAZ relationship between boys and girls samples.

9. The Health and Population Sector Program (1998–2003) and the Health Nutrition and Population Sector Program (2003–10) may have contributed to improved maternal health care outcomes, as could various NGO interventions, notably by BRAC.

10. See Block, Masters, and Bhagowalia (2012) and Srinivasan, Zanello, and Shankar (2013) for other examples of this approach in the nutrition literature.

11. Specifically, the LOGIT model yielded exactly the same pattern of significance variables across the model, but slightly larger marginal effects in most cases. Thus, inferences in terms of the contributions of the various indicators to predicted nutritional change remained the same, though the explanatory power of the stunting model increase from about 56% to 69%.

12. Specifically, Cleland *et al.* (1994) report an average fertility rate of 7 children in 1975 and median birth spacing of 33 months, while the 2011 BDHS reports an average fertility rate of 2.3 children and median birth spacing of 47.4 months. We multiply these changes by the relevant coefficients reported in the previous section to obtain these estimates of the long term contribution of demographic change to improved nutrition outcomes.

## REFERENCES


Ahmed, S., Hossain, M., & Chowdhury, M. (2009). Informal sector providers in Bangladesh: How equipped are they to provide rational health care? *Health Policy and Planning, 24*, 467–478.

Ahmed, T., Mahfuz, M., Ireen, S., Ahmed, A. M., Rahman, S., Islam, M. M., *et al.* (2012). Nutrition of children and women in Bangladesh: Trends and directions for the future. *Journal of Health, Population and Nutrition, 30*, 1–11.

Behrman, J. (1988). Nutrition, health, birth order and seasonality: Intrahousehold allocation among children in rural India. *Journal of Development Economics, 28*, 43–62.

Behrman, J. R., & Deolalikar, A. B. (1987). Will developing country nutrition improve with income? A case study for rural South India. *Journal of Political Economy, 95*, 492–507.

Behrman, J. R., & Wolfe, B. L. (1984). More evidence on nutrition demand: Income seems overrated and women's schooling underemphasized. *Journal of Development Economics, 14*, 105–128.

Black, R. E., Victora, C. G., Walker, S. P., Bhutta, Z. A., Christian, P., de Onis, M., *et al.* (2013). Maternal and child undernutrition and overweight in low-income and middle-income countries. *The Lancet, 382*, 427–451.

Block, S. A., Masters, W., & Bhagowalia, P. (2012). Does child undernutrition persist despite poverty reduction in developing countries? Quantile regression results. *Journal of Development Studies, 48*, 1699–1715.

Burchi, F. (2012). Whose education affects a child's nutritional status? From parents' to household's education. *Demographic Research, 27*, 681–704.

Chowdhury, A. M., Bhuiya, A., Chowdhury, M. E., Rasheed, S., Hussain, Z., & Chen, L. C. (2013). The Bangladesh paradox: Exceptional health achievement despite economic poverty. *Lancet, 382*, 1734–1745.

Cleland, J., Phillips, J. F., Amin, S., & Kamal, G. M. (1994). The determinants of reproductive change in Bangladesh: Success in a challenging environment. In *Regional and sector studies*. Washington, DC: World Bank.

Das Gupta, M., Lokshin, M., Gragnolati, M., & Ivaschenko, O. (2005). Improving child nutrition outcomes in India: Can the integrated child development services be more effective? In: *Policy research working paper series no. 3647*. Washington DC: The World Bank.

de Onis, M., Garza, C., Onyango, A. W., & Borghi, E. (2007). Comparison of the WHO child growth standards and the CDC 2000 growth charts. *The Journal of Nutrition, 137*, 144–148.

Deaton, A., & Dreze, J. (2008). Food and nutrition in India: Facts and interpretations. *Economic and Political Weekly, XLIV*, 42–65.

Dewey, K. G., & Cohen, R. J. (2007). Does birth spacing affect maternal or child nutritional status? A systematic literature review. *Maternal & Child Nutrition, 3*, 151–173.

El Arifeen, S., Christou, A., Reichenbach, L., Osman, F. A., Azad, K., Islam, K. S., *et al.* (2013). Community-based approaches and partnerships: Innovations in health-service delivery in Bangladesh. *The Lancet, 382*, 2012–2026.

Elder, T. E., Goddeeris, J. H., & Haider, S. J. (2010). Unexplained gaps and Oaxaca–Blinder decompositions. *Labour Economics, 17*, 284–290.

FAO. (2012). *AGROSTAT*. Rome: Food and Agriculture Organization.

Filmer, D., & Pritchett, L. (2001). Estimating wealth effects without expenditure data – or tears: An application to education enrollment in states of India. *Demography, 38*, 115–132.

Filmer, D., & Scott, K. (2012). Assessing asset indices. *Demography, 49*, 359–392.

Haddad, L., Alderman, H., Appleton, S., Song, L., & Yohannes, Y. (2003). Reducing child malnutrition: How far does income growth take us? *World Bank Economic Review, 17*, 107–131.

Headey, D. (2013). Developmental drivers of nutritional change: A cross-country analysis. *World Development, 42*, 76–88.

Headey, D., Chiu, A., & Kadiyala, S. (2012). Agriculture's role in the Indian enigma: Help or hindrance to the malnutrition crisis? *Food Security, 4*, 87–102.

Heaver, R., & Kachondam, Y. (2002). Thailand's national nutrition program: Lessons in management and capacity development. In: *World Bank HNP discussion paper*. Washington, DC: The World Bank.

Heltberg, R. (2009). Malnutrition, poverty, and economic growth. *Health Economics, 18*, 77–88.

Horton, S. (1988). Birth order and child nutritional status: Evidence from the Philippines. *Economic Development and Cultural Change, 36*, 341–354.

Hossain, S. M. M., Duffield, A., & Taylor, A. (2005). An evaluation of the impact of a US$60 million nutrition programme in Bangladesh. *Health Policy and Planning, 20*, 35–40.

Humphrey, J. H. (2009). Child undernutrition, tropical enteropathy, toilets, and handwashing. *The Lancet, 374*, 1032–1035.

Jann, B. (2008). The Blinder–Oaxaca decomposition for linear regression models. *Stata Journal, 8*, 453–479.

Jayachandran, S., & Pande, R. (2013). Why are Indian children shorter than African children? In: *Department of Economics working paper*. Cambridge MA: Harvard University.
Jensen, R. (2012). Another mouth to feed? The effects of (in)fertility on malnutrition. *CESifo Economic Studies*, [early online version].
Joshi, S., & Schultz, T. P. (2013). Family planning and women's and children's health: Long-term consequences of an outreach program in Matlab, Bangladesh. *Demography, 50*, 149–180.
Kar, K. (2003). Subsidy or self-respect? Participatory total community sanitation in Bangladesh. *IDS working paper 184*. Brighton: Institute for Development Studies (IDS).
Kohler, H.-P. (2012). Population growth. *PSC working paper PSC 12-03 and Copenhagen consensus 2012 challenge paper*. Population Studies Center, University of Pennsylvania, and Copenhagen Consensus Centre, Copenhagen. <http://repository.upenn.edu/psc_working_papers/34>.
Levinson, F. J., & Eliot Rohde, J. (2005). Responses to: 'An evaluation of the impact of a US$60 million nutrition programme in Bangladesh'. *Health Policy and Planning, 20*, 405–406.
Lin, A., Arnold, B. F., Afreen, S., Goto, R., Huda, T. M., Haque, R., *et al.* (2013). Household environmental conditions are associated with enteropathy and impaired growth in rural Bangladesh. *American Journal of Tropical Medicine and Hygiene, 89*, 130–137.
Mondal, D., Minak, J., Alam, M., Liu, Y., Dai, J., Korpe, P., *et al.* (2012). Contribution of enteric infection, altered intestinal barrier function, and maternal malnutrition to infant malnutrition in Bangladesh. *Clinical Infectious Diseases, 54*, 185–192.
Nubé, M. (2009). The Asian enigma: Predisposition for low adult BMI among people of South Asian descent. *Public Health Nutrition, 12*, 507–516.
Pande, R. (2003). Selective gender differences in childhood nutrition and immunization in rural India: The role of siblings. *Demography, 40*, 395–418.
Ramalingaswami, V., Jonson, U., & Rohde, J. (1997). The Asian Enigma. In *The Progress of nations*. New York: UNICEF.
Royston, P., Altman, D. G., & Sauerbrei, W. (2006). Dichotomizing continuous predictors in multiple regression: A bad idea. *Statistics in Medicine, 25*, 127–141.
Ruel, M. T., & Alderman, H. (2013). Nutrition-sensitive interventions and programmes: how can they help to accelerate progress in improving maternal and child nutrition? *The Lancet, 382*, 536–551.
Rutstein, S. O. (2008). Further evidence of the effects of preceding birth intervals on neonatal, infant, and under-five-years mortality and nutritional status in developing countries: Evidence from the demographic and health surveys. In: *DHS working paper 41*. Calverton, MD.
Rutstein, S. O., & Staveteig, S. (2013). Making the demographic and health surveys wealth index comparable. In: *27th IUSSP international population conference*. Busan, Korea.
Sack, D. A., Roy, S., Ahmed, T., & Fuchs, G. (2005). Responses to: 'An evaluation of the impact of a US$60 million nutrition programme in Bangladesh'. *Health Policy and Planning, 20*, 406–407.
Schultz, T. P. (1997). Demand for children in low income countries. In M. R. Rosenzweig, & O. Stark (Eds.). *Handbook of population and family economics* (Vol. A, pp. 349–430). Amsterdam: Elsevier Science.
Shrimpton, R., Victora, C. G., De Onis, M., Costa Lima, R., Blössner, M., & Clugston, G. (2001). The worldwide timing of growth faltering: Implications for nutritional interventions. *Pediatrics*, e75.
Shuval, H. I., Tilden, R. L., Perry, B. H., & Grosse, R. N. (1981). Effect of investments in water supply and sanitation on health status: A threshold saturation theory. *Bulletin of the WHO, 59*, 243–248.
Smith, L. C., & Haddad, L. (2000). *Explaining child malnutrition in developing countries: A cross-country analysis*. Washington, DC: International Food Policy Research Institute (IFPRI).
Spears, D. (2013). How much international variation in child height can sanitation explain? In: *Working paper*. NJ: Princeton University.
Spears, D., Ghosh, A., & Cumming, O. (2013). Open defecation and childhood stunting in India: An ecological analysis of new data from 112 districts. *PLoS One, 8*, e73784.
Srinivasan, C. S., Zanello, G., & Shankar, B. (2013). Rural–urban disparities in child nutrition in Bangladesh and Nepal. *BMC Public Health, 13*, 1471–2458.
Taylor, L. (2012). *The nutrition agenda in Bangladesh: 'Too massive to handle'? In Analysing Nutrition Governance, Bangladesh Country Report*. Sussex: Institute of Development Studies (IDS).
The Economist. (2012). *Bangladesh and development: The path through the fields*. November 3rd, 2012.
Thomas, D., Strauss, J., & Henriques, M. H. (1991). How does mother's education affect child height?. *Journal of Human Resources, 26*, 183–211.
Victora, C. G., de Onis, M., Curi Hallal, P., Blössner, M., & Shrimpton, R. (2009). Worldwide timing of growth faltering: Revisiting implications for interventions. *Pediatrics, 2010*, 473–480.
Webb, P., & Block, S. A. (2004). Nutrition information and formal schooling as inputs to child nutrition. *Economic Development and Cultural Change, 52*, 801–820.
Weinberg, C. R. (1995). How bad is categorization? *Epidemiology, 6*, 345–347.
White, H. (2005). Comment on contributions regarding the impact of the Bangladesh Integrated Nutrition Project. *Health Policy and Planning, 20*, 408–411.
White, H., Masset, E., Blondal, N., & Waddington, H. (2005). Maintaining momentum to 2015? An impact evaluation of interventions to improve maternal and child health and nutrition in Bangladesh. In: *EconWPA*.
World Bank. (2005a). *Maintaining momentum to 2015? An impact evaluation of interventions to improve maternal and child health and nutrition outcomes in Bangladesh*. Washington, DC: Operations Evaluation Department, World Bank.
World Bank. (2005b). Bangladesh integrated nutrition project: Project performance assessment report. *Report No. 32563*. Washington, DC: World Bank.
World Bank. (2006). *Repositioning nutrition as central to development: A strategy for large-scale action*. Washington, DC: The World Bank.
World Bank. (2012a). Economic impacts of inadequate sanitation in Bangladesh. In *Bank's water and sanitation program (WSP)*, East Asia and Pacific Office: World Bank.
World Bank. (2012b). *World development indicators online*. Washington, DC: The World Bank.

## APPENDIX A. SUPPLEMENTARY DATA

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.worlddev.2014.09.022.