Title:

Early-childhood growth faltering, post-infancy recovery and educational outcomes in late childhood: Evidence from Vietnam

Short title: Childhood growth, education in latter…

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Abstract:

We use longitudinal data on over 1,500 children born in 2001 in Vietnam to study the impact of early childhood stunting on height, lagging in schooling progression and cognitive outcomes in late childhood (age 8-10 years). Our preferred estimates utilize 2SLS estimators to control for the endogenous determination of early childhood stunting and also include control for child sex and birth order, mother's height and BMI, household socioeconomic status, and community characteristics. These estimates indicate that deficits in height-for-age at age 12 months have negative impacts on height in late childhood but not on schooling and cognitive outcomes in late childhood. The children who were stunted or moderately stunted at age 12 months display significant catch-up growth, recovering half of their deficits in height-for-age by age 8 years. Socioeconomic status in infancy has negative effects on both stunting in infancy and poor subsequent educational outcomes in late childhood, which result in significant *associations* between stunting in infancy and some subsequent educational outcomes in late childhood – but not causal effects once there is control for the endogenous determination of early childhood height deficits.

1. Introduction

Heckman (2006), Cunha et al (2006, 2010) and many others recently have emphasized the possible very high returns to investments in early life, particularly if there are dynamic complementarities between early-life investments and investments later in the life cycle. An important example of such a possibility is investments in early-life nutrition. A number of prominent and influential studies claim that early-life growth faltering between conception and the age of 2 years afflicts tens of millions of children primarily in the developing world and has long-run detrimental effects.[1] This literature reports that these children are likely to suffer permanent cognitive defects, unlikely to do well in school, have smaller adult stature, have lower adult cognitive skills, have lower adult wage rates, and have children with lower birth weights. Impacts on learning later in childhood are particularly emphasized in this literature as important outcomes or channels through with early growth faltering works. The majority of these studies claim that such early-life growth deficits are not likely to be reversible at reasonable costs in the environments in which these children are living, so there is a critical window of opportunity for

[1] Some examples from the economics, biomedical, biological, nutritional, and public health literatures are Adair (1999); Adair et al. (2013); Alderman, Hoddinott and Kinsey (2006); Alderman and Behrman (2006); Almond, Chay and Lee (2005); Behrman, Alderman and Hoddinott (2004); Behrman, Deolalikar and Lavy (1995);Behrman et al. (2009, 2014); Behrman and Rosenzweig (2004); Black, Devereux and Salvanes (2007); Crookston et al. (2010, 2013); Daniels and Adair (2004); de Onis, Blössner and Borghi (2010, 2011); Grantham-McGregor et al. (2007); Hoddinott and Kinsey (2001); Hoddinott et al. (2008. 2013a,b); Horton and Lo (2013); Johnson and Macvean (1995); Lutter et al. (1990); Maluccio et al. (2009); Martorell and Habichat (1986); Martorell, Khan and Schroeder (1994); Martorell, et al. (1992); Pelletier and Frognillo (2003); Prentice et al. (2013); Schott et al. (2013); Schroeder et al. (1995); Shrimpton et al. (2001); Stein et al. (2010); and Victora et al. (2008, 2010).

improving early-life nutrition up to 2 years of age.[2] For example, Victora et al (2008: 340, emphasis added)'s *Lancet* article's first "key message" is that "Poor fetal growth or stunting in the first 2 years of life leads to *irreversible* damage, including shorter adult height, lower attained schooling, reduced adult income, and decreased offspring birthweight." Likewise, Victora et al (2010: e473, emphasis added)'s *Pediatrics* article's summary of their study of child growth patterns in 54 countries is: "Children from low- and middle-income countries are born with weights and lengths below WHO growth standards, and early growth faltering is even faster than currently assumed. *The window of opportunity for preventing undernutrition ends at 2 years of age*."

However most of these studies of relations between early-life nutritional status and subsequent outcomes over the life cycle are associational using observational data and therefore have important limitations for causal interpretations. That is, most of these studies ignore that the growth measures that they consider are likely to be determined endogenously in the presence of unobservable factors for the children (e.g., their genetic endowments pertaining to health, learning and other outcomes of interest), their parents (e.g., their innate capabilities, preferences regarding types of investments in their children, and their expectations regarding returns to various types of investments) and their communities (e.g., the health environment, prices and public sector availabilities for inputs that affect child development and the incentives for child development). Effectively, thus, most of these studies implicitly assume that growth is

[2] A small subset of these studies, some fairly recent, report some subsequent "catch-up" growth, but generally still emphasize the importance of early childhood nutrition (Aldair 1999; Behrman, Deolalikar and Lavy 1995; Crookston et al. 2010, 2013; Mani 2012; Schott et al. 2013; Prentice et al. 2013).

determined randomly, not by behaviors of parents and others in the presence of unobserved factors that are not controlled in the estimates.

Indeed we are aware of only one previous study for developing countries that attempts to estimate the impact of growth faltering during the first 2 years of life on direct measures of school-age child cognitive abilities or cognitive achievement, as noted the major channels through which early-life growth faltering is posited to work, using an estimation strategy to attempt to control for endogeneity. Glewwe and King (2001) used the Cebu Longitudinal Health and Nutrition Survey (CLHNS), which was carried out in the Metropolitan Cebu area in the Philippines with a sample of 3289 children (2256 of which had data for this study) born between May 1, 1983 and April 30, 1984 in 33 randomly selected barangays (districts). They find that birth weight (kgm) and height growth (cm) between 0 and 12 months, 12 and 24 months and 2 and 8 years all are significantly *associated* with *cognitive abilities* at age 8 years measured by the Philippines Nonverbal Intelligence Tests in OLS estimates with additional controls for parental schooling attainment and household assets. But standard 2SLS/IV tests are consistent with these child anthropometric measures being treated as endogenously determined using prices, rainfall and maternal height and arm circumference as instruments. If child anthropometrics are treated as endogenous, the coefficient estimate for growth between 12 and 24 months becomes over five times larger than in the OLS estimates and is significant at the 1% level, but the coefficient estimates for the other child anthropometric measures are not significant even at the 10% level. Thus treating child anthropometrics as endogenous in this case changes our understanding of the apparent impacts of child anthropometrics on later cognitive abilities considerably with, for example, height growth before 24 months appearing much more important and height growth after 24 months no longer appearing significant. The Glewwe and King study, thus, provides support for the importance of a critical window for child growth before 24 months.

We have been able to find no other studies that examine the causal impact of growth faltering in the first 2 years of life on childhood *cognitive abilities* and no studies at all that examine the causal impact of growth faltering on *cognitive achievement* in developing countries using approaches to control for the endogenous determination of early-life growth faltering.[3] In this paper, we contribute a study to help fill the latter gap, using data from the 21st century. We use data on children in a low-income country, Vietnam, to examine whether growth retardation in the first year of life leads to poor physical and educational development in late childhood.[4] To attempt to identify causal effects we use 2SLS to control for the influence of unobserved factors. The key identifying instrument that we use is the component of birth weight that is unpredicted by major child and household and community factors, including sex, birth order, month of birth, the household socioeconomic status and some anthropometric characteristics of the mother. This

[3] We are aware of two studies that consider the impact on cognitive achievement of early-life growth, but these either use child growth when the children are older than 2 years or cognitive achievement well after school ages. Glewwe, Jacoby and King (2001) report that in the same Cebu sample that Glewwe and King (2001) use the height-for-age *Z*-score closest to the time of the school enrollment decision (and therefore well after the alleged critical window of up to 24 months) had significant impacts on child cognitive achievement measured as the sum of scores on curriculum-based English and mathematics scores at age 11 years, as well as on the age of entering school and grade repetition but not homework efforts, using older sibling height-for-age as instruments in within sibling estimates. Hoddinott et al. (2013) present significant estimates of the impacts of growth faltering as of 24 months on adult cognitive achievement for adults aged 26-42 years using the experimental allocation of nutritional supplements among the four Guatemalan INCAP villages as an instrument.

[4] Vietnam was classified by the World Bank as a low-income country in the years in which data for this study were collected.

component of the birth weight can be interpreted as a proxy for the accumulated result of all shocks to the nutritional status of the child before birth. The test statistics related to the 2SLS regressions support the use of this instrument variable. The estimates suggest that the causal effects of early-life nutritional status on nutritional status and educational development in late childhood are different in some important respects than is suggested by OLS associations, so the contributions of this study to the literature on the impact of early-life growth faltering on later child development are important.

This rest of this paper is organized as follows. Section two describes the data source and the study context. In Section three we introduce the identification strategy, and justify our selection of instruments for our instrumental variable estimates. Section four presents and discusses the regression results. Finally, Section five presents conclusions.

2. The data and the straightforward facts

2.1 The sample

The sample is a part of the Young Lives project, which is an international comparative study of child poverty. Since 2002, Young Lives has been following 12,000 children in Ethiopia, India, Peru and Vietnam. The Young Lives sample consists of two cohorts: about 8,000 children born in 2001-2 and another 4,000 children born in 1994-5. Members of the Younger cohort are sometimes called the millennium children. The Younger Cohort Vietnamese sample consists of 20 commune-based clusters in Northern Uplands, Red River Delta, Central Coast, and Mekong Delta. Each of these regions contains four rural clusters, except the Central Coast, which consists of four urban clusters in the city of Da Nang and another four in the rural province of Phu Yen.

There have been to date three rounds of child and household surveys in 2001, 2006, and 2009 (a fourth round is currently underway). Additionally a school survey was conducted in 2011 for the fifth graders in the schools with Young Lives children in Vietnam. In this study, we work with only the Vietnamese children born in 2001 because some of the educational outcomes that we use were collected in the 2011 School Survey. Only the children born in 2001 who progressed one grade every year after entering school at the appropriate age, were in the fifth grade in 2011.[5] The children in this study were on average 13.4 months old in Round 1, with the youngest being 6.6 months old and the oldest being 18 months old.[6] In Round 3, the average age of these children was 98.4 months (8.2 years).

The attrition rate has been quite low, Round 3 contains 1944 millennium children, including 1547 children born in 2001. The sample loss consists of 11 deaths, two cases of refused consent (all in urban), and the rest are not reachable. The children were unreachable because they have migrated to foreign countries (5 cases), or migrated to other communities in Vietnam and cannot be contacted. Tracking data show that the children migrated more to urban areas than to rural ones. There is no clear evidence of the about 3 percent loss in sample being poorer or wealthier than the average. With respect to their health, however, we find that the mean birth weight of the children (born in 2001, and) remaining in Round 3 (3090grams), is about the same as that for all children born in 2001 (3091grams). For height-for-age in Round 1, the corresponding figures are -0.608 and -0.606. By Round 3, there remained slightly more boys than girls (51% to 49%) in the sample. The percentage of ethnic minority children in the Young Lives sample is about the same as the population share of ethnic minorities. Anthropometric personnel have always been senior

[5] For the school calendar of Vietnam, the fifth grade is the right grade for the children born in 2001, regardless of birth month.

[6] The children older than 18 months in Round 1 are treated as outliers, and not considered in this study. The number of such children is small, however – only 45 children.

members in Young Lives fieldwork teams. In each round of surveys, the children's anthropometric data were recorded three times.

2.2 Some straightforward facts that are relevant to the framework of analysis

Shrimpton et al. (2001:4) show that there is a general decline in length-for-age z scores (or height-for-age z scores, HAZ[7]) from zero to 24 months in developing countries in which undernourishment is widespread. We find that this pattern appears in the cross-sectional data for Round 1 in the sample under study. The faltering of height-for-age in our sample happens both to poorer and richer children. Because the age of children varies over more than a year in the first round, there may be some questions about the interpretation of HAZ in this round. For instance, a child might have been non-stunted at seven months, but became stunted at 17 months, even though the general conditions were unchanged. Following Scott et al. (2013), we calculate the predicted value of HAZ for the children either older or younger than 12 months in the following way. The average HAZ for age i (in months) is defined as the mean of HAZ for all the children ages $i-1, i$ and $i+1$ months. The exception is for the ages $i=6$ and $i=17$, for which the averages are calculated for the children in two, rather than three, consecutive monthly age groups. The predicted value of HAZ at age 12 months is the HAZ in Round 1 minus the difference of the age group average for the actual age of the child in Round 1 minus the average for children 12 months of age in Round 1.[8]

From now on, we refer to the predicted value of HAZ at 12 months as a measure of the children's long-run nutritional status at age one, denoted by HAZ^1. Given the emphasis in the

[7] Z scores indicate how many standard deviations from the median in a reference population is a particular child.

For more information, see the discussion at the start of Section 3.1

[8] We do not assume linearity of the trend by age, but instead calculate the predictive values for each age group.

literature on the first 24 months noted in the introduction, we would prefer to have a measure at 2 years, HAZ^2, rather than HAZ^1. But HAZ^2 is not available in the Young Lives data. What is important for interpreting our analysis as reflecting HAZ^2 in addition to HAZ^1 is how correlated are HAZ^1 and HAZ^2 even though their levels may differ systematically due to the general decline in HAZ over this age range noted above. The more they are correlated, the more HAZ^1 is a good proxy for HAZ^2 . We do not know this correlation for our data, but we note that in one of the data sets that has been used extensively in the literature cited above, the Guatemalan INCAP data (Stein et al. 2008), the correlation between HAZ^1 and HAZ^2 is a significant 0.84 (Hoddinott et al. 2013b, Supplemental Table 1). Therefore the variance in HAZ^1 is a very good, but not perfect, proxy for the variance in HAZ^2 in the INCAP data for which we can make this comparison.

We also note that our approach differs somewhat from that in much of the literature that singles out categorical movements from being stunted in infancy to being non-stunted in childhood (for example, Crookston et al, 2010), where stunted is defined to be HAZ more than two standard deviations below the reference median. In addition to such categorical movements, we consider all changes in HAZ from Round 1 (age 1 year) to Round 3 (age 8 years).

Table 1 demonstrates that there is fair amount of change in *HAZ* between ages 1 and 8 years. Catch-up, if defined to be passing from stunting to non-stunting as is conventional, happened mostly for children with HAZ^1 that was not too far below the threshold of -2 for stunting in the conventional definition, even though the average change for the children in this interval (-2.5, -2) is smaller than that for the children with HAZ^1 in the interval (-5, -2.5), for whom growth is so important, no matter whether they become non-stunted or not. Overall, nearly half of the children, who were stunted at age one year, recovered by age 8 years. The change in the opposite direction is not so strong -- less than one of ten of the non-stunted (at age 1 year) children became stunted by age 8 years. Of the faltering cases, however, more than 70 percent are children who were moderately stunted at age one (i.e., HAZ^1in the interval (-2, -1)), and faltering is very unlikely for children with positive HAZ^1.[9] While the likelihood of faltering into stunted status is small, the average drop in HAZ for the children with positive HAZ^1 is substantial. In summary, there seems to be a fair amount of movement in both directions between ages 1 and 8 years, which suggests that at least in terms of height, the experience in infancy may not be as "irreversible" as suggested in some of the literature noted above (e.g., see quotation from Victora et al. 2010 in first paragraph).

Table 1: Growth in average HAZ and numbers of catch-ups and faltering between ages 1 and 8 years by level of HAZ at age 1 year

Interval by HAZ^1 (at age 1 year)	Average change in HAZ from 1 to 8 years□	Number of catch-ups (stunting-to-not)	Catch-up percentage
-5 to -3.5	1.11	3	10.7
-3.5 to -3.0	0.88	15	34.1
-3.0 to -2.5	0.57	39	35.8
-2.5 to -2.0	0.35	117	57.4
All the stunted	0.53	174	45.2
		Number faltering (not-to-stunting)	Faltering percentage
All the non-stunted	-0.11	139	9.6
-2.0 to -1.5	0.23	62	23.5
-1.5 to -1.0	0.23	39	11.2
-1.0 to -0.5	-0.06	17	5.3
-0.5 to 0.0	0.22	10	4.1
0.0 to 5.0	-0.70	11	4.1

Notes: □ The average change in HAZ from age 1 to 8 years applies for all the children in the range of HAZ^1

Table 1 also shows a negative association between HAZ^1 and the change in HAZ between ages 1 and 8 years. For instance, the group of stunted (at age 1) children on average gained HAZ in the post-infancy period, while the group of children with positive HAZ at age 1 on average suffered declining HAZ over the years, with a quite substantial average drop of -0.70. The question rises

[9] Given the decline on average in HAZ between age 1 and 2 years noted above, probably a number of the children who faltered between ages 1 and 8 years actually faltered by age 2 years.

to what extent the negative association presents a sort of natural bounce back and to what extent it reflects changes in related socioeconomic conditions for these children?[10] There also seems to be a positive correlation between HAZ^1 and school survey test scores. To explore the relations between HAZ^1 and school outcomes, we will apply multivariate analysis to control for other (observed) factors, and use econometric techniques to eliminate the influence of unobserved factors.

3. Empirical strategy

3.1 The variables

Descriptive statistics for the variables are presented in Table 2. We use HAZ of children to measure their state of growth and possible stunting. Supine length (Round 1) and standing height (rounds 2 and 3) were measured with length/height boards using standardized WHO methodology (WHO 2008) and measurements precise to 1mm. The length and height measurements were converted HAZ using WHO standards (WHO 2006a, b; de Onis et al. 2007). Related to HAZ is the variable growth g for the change in HAZ from age 1 to 8 years.

[10] Yet another conceptual possibility might be measurement error such that those who were reported as stunted were more likely to have negative measurement error and those with positive HAZ more likely to have positive measurement error. The multiple measurements at each round suggest that in the most direct sense such measurement error is not a big problem: the noise-to-signal ratio implied by the multiple estimates is less than 1 percent. But this refers to measurement error in the sense of obtaining the correct measure at the time that the measurement was made. There also is a more subtle possible measurement error relating to the time of measurement relative to the timing of growth spurts for a given latent growth path. If the timing of the measurement happened to be right before (after) a growth spurt, then the probability may be higher (lower) for positive growth between that time and the next measurement purely due to the random timing of the measurement relative to the timing of growth spurts.

Table 2: Descriptive statistics of core variables

Variables	Mean	Std. Dev.
If child is a boy	0.51	0.50
Birth weight (kgm)	3.08	0.43
If first child for the mother	0.38	0.48
Born in March to August	0.51	0.50
Born in December	0.09	0.29
HAZ z-score at age 1 year	-1.10	1.11
HAZ z-score at age 8 years	-1.07	1.02
Math score in cognitive test in R3	20.58	5.52
PPVT in cognitive test in R3	98.34	29.57
Grade 5 math score in school survey	31.74	13.74
Vietnamese score in school survey	35.31	13.79
If mother's age was 20-35 years	0.86	0.35
Mother's BMI	21.0	2.68
Mother's height (cm)	152.2	5.84
Mother is ethnic minority	0.12	0.32
If mother completed LSE	0.37	0.48
If father completed LSE	0.46	0.50
Wealth index R 1	0.46	0.22
Red River Delta	0.24	0.42
Northern Uplands	0.17	0.38
Mekong Delta	0.17	0.37

In addition to growth in HAZ between ages 1 and 8 years, other dependent variables for this study are whether children are under grade at age 10 (i.e., not yet in fifth grade) and measures of children's performance in the Young Lives Round 3 cognitive tests (in 2009) as well as the tests administered in 2011-2 school surveys for fifth graders.[11] The cognitive outcomes considered are test scores in math and vocabulary receptive (PPVT). The school survey tests were administered twice. The first test was administered in October-December 2011, and the second was administered in April-May 2012, in the same academic year; the values used in this study are total scores in the two tests for each subject.

[11] See Cueto and Leon (2012) for the Young Lives cognitive tests in Round 3. For the school survey, see http://www.younglives.org.uk/what-we-do/school-survey/vietnam-school-survey

The right-side variables include child, household, and community characteristics. Child characteristics include gender, birth weight, HAZ^1 (described in sub-section 2.2), a dummy variable for whether the child was the first one for his/her mother, and dummies variables on birth months. Household characteristics include: wealth index in Round 1, mother's schooling attainment, father's schooling attainment, mother's height, mother's BMI, a dummy for mother being an ethnic minority, and a dummy for whether the mother's age was between 20 and 35 years when the child was born. The variables on mother (father)'s schooling are dummy variables that equal 1 if and only if the mother (father) has completed Lower Secondary Education (LSE).

The wealth index is the simple average of three components: (1) housing quality, which is the average of scaled rooms per person, floor, roof and wall; (2) the value of consumer durables, which is the scaled sum of nine dummies for the basic consumer durables;[12] and (3) the value of services, which is the simple average of drinking water, electricity, sanitation facilities, and fuel, all of which are 0–1 variables.[13] The community characteristics are represented by dummy variables for the region or sector. We include regional dummy variables for Northern Uplands, Red River, Mekong Delta, and Urban, and therefore, the omitted category consists of the rural clusters in the Central Coast.

[12] The basic consumer durables are: radio, refrigerator, bicycle, motorcycle, car, mobile phone, land line phone, fan, and television.

[13]The list of assets in the Young Lives wealth index is similar to that in Filmer and Pritchett (2001), who use principal components to determine the weights for an index based on the individual asset variables. As the coefficients are all positive in the Young Lives wealth index and in the index in Filmer and Pritchett (2001), these two indices are expected to be fairly correlated. Filmer and Scott (2012) report that such asset indices are robust to a range of definitions.

3.2 Model specifications

There are two sets of questions regarding the long-term impact of early childhood malnutrition on children when they are in late childhood that we address in this study. The first is how the deficit in HAZ^1 affects the post-infant growth recovery/faltering (g) and therefore the height at age 8 years. The second is whether HAZ^1 affects educational outcomes – whether on track in the fifth grade and cognitive test scores -- in late childhood at ages 8 and 10 years. We apply two models to answer these questions.

Let x_{ik} represent the other (than HAZ) characteristics for child i, with $k = 1, K$. Similarly, let $y_{il}, l = 1, L$ be household characteristics; and let $z_{im}, m = 1, M$ denote community characteristics. Our first equation concerns $g_i = HAZ_i^8 - HAZ_i^1$ from age 1 year to age 8 years:

$$g_i = \alpha + \beta \times HAZ_i^1 + \sum_{k=1}^{K} \gamma_k x_{ik} + \sum_{l=1}^{L} \delta_l y_{il} + \sum_{m=1}^{M} \lambda_m z_{im} + u_i \quad (1)$$

In this equation the right-side child, household and community characteristics refer to a specific time (baseline when the child was 1 year old), and therefore, to limit clutter, time indexes are not indicated.

The next set of models is used for studying the impact of HAZ^1 on educational outcomes. The educational outcomes are: i) ug, which denotes the child having never attained grade 5 by the year 2011, ii) c, which denotes the scores in Young Lives Round cognitive tests in math, and PPVT at age 8 years, and iii) the test scores in Grade 5's math or in reading Vietnamese in the school survey in 2011-2, also denoted by c. The estimate for coefficient $\bar{\beta}$ in equation 2a below constitutes our first evidence about the impact of early childhood nutritional status on education attainment at age ten.

$$ug_i = \overline{\alpha} + \overline{\beta} \times HAZ_i^1 + \sum_{k=1}^{K} \overline{\gamma}_k x_{ik} + \sum_{l=1}^{L} \overline{\delta}_l y_{il} + \sum_{m=1}^{M} \overline{\lambda}_m z_{im} + v_i \quad (2a)$$

For the children who have progressed to grade 5, and therefore have taken the school survey tests, a further question is asked: does early childhood stunting or height-for-age deficit make any difference in late-childhood educational outcomes for the child? To answer this question, we estimate the coefficient $\hat{\beta}$ in the following equation:

$$c_i = \hat{\alpha} + \hat{\beta} \times HAZ_i^1 + \sum_{k=1}^{K} \hat{\gamma}_k x_{ik} + \sum_{l=1}^{L} \hat{\delta}_l y_{il} + \sum_{m=1}^{M} \hat{\lambda}_m z_{im} + \varepsilon_i \quad (2b)$$

In addition to school survey tests, equation 2b is also estimated with the outcome ***c*** being test scores in math and PPVT in the Young Lives Round 3 data at age 8 years. An advantage of the latter tests is that all the Young Lives children, regardless of what grade they were in, took them.

3.3 Problems: endogeneity and heterogenous partial effects

For all of the above equations, estimation is not straightforward because of possible endogeneity. As mentioned earlier, the endogeneity problem may reflect that equations do not include all the factors that directly affect the child's HAZ and educational achievement. For instance, if drinking water quality is poor for all the years under study then it may affect HAZ^1. On the other hand, as the quality of water may have an impact on growth *g* over the post-infancy years, it could be correlated with the residual terms if the quality of drinking water is not among the explanatory factors in that equation. Thus, there is a possibility of HAZ^1 being endogenous in equation (1).

The same variables can be endogenous in Models 2a-b, as well. For instance, hard work and discipline are important features of family culture. Household members often share those characteristics. These characteristics for the parents and other senior household members may matter for child care, and therefore have a positive impact on health of the infant. The same characteristics (of the child and parents) may facilitate learning and therefore contribute to educational outcomes. For this reason HAZ^1 may be correlated with the residual terms in equations 2a-b. If there is such endogeneity, application of OLS to estimate the effect of HAZ^1 may result in biases.

Separate from the question of controlling for endogeneity in the estimation of the coefficient of HAZ^1 is how to interpret this coefficient estimate in light of what Dunning calls "heterogenous partial effects" (2008:291). To illustrate, assume that HAZ^1 is determined by a linear combination of factors such as birth weight BW, mother's height MH, being a boy B, and other factors: $HAZ^1 = \mu_0 + \mu_1 BW + \mu_2 MH + \mu_3 B + \cdots$. Some 12-month old children have low HAZ^1 mainly because of low birth weight, while others may mainly because their mothers' stature is short, or being a boy or having a mother who is in an ethnic minority (see Table A2 in Appendix). If there are multiple determinants of HAZ^1, in an important sense the effect of HAZ^1 is a meaningful term only if there are "homogenous partial effects" for all the determinants of HAZ^1, which is likely to be a quite strong assumption. In the general case, in contrast, there are "heterogenous partial effects" so that the impact of HAZ^1 depends on which of the determinants of HAZ^1 is considered. That is, what is of most interest is not the effect of HAZ^1 per se, but the local average treatment effects (LATE) for each of the determinants of HAZ^1.

3.4 Identifying instrument:

For our 2SLS estimates, we need an instrument(s) that is (1) sufficiently strongly correlated with HAZ^1 and (2) exogenous and therefore uncorrelated with the disturbance terms in relations (1) and (2a-b). For this purpose we utilize a component of birth weight. Birth weight has been used as an instrument for child height by Hoddinott and Kinsey (2001) in their study of child growth in time of drought in Zimbabwe to control for possible endogeneity of initial height. Their diagnostic tests indicate that birth weight satisfies the two conditions above in their sample. On the other hand the Glewwe and King (2001) study that is summarized in the introduction above treats birth weight as endogenous and finds significant effects of parental schooling attainment and household assets in the relation determining birth weight. Also Behrman and Rosenzweig (2004) in their study of the impacts of birth weight over the life cycle find that many estimates of the impacts of birth weight over the live cycle are affected by controlling for all endowments, including family background characteristics, by using identical twins – as also is found for some estimates in subsequent studies using identical twins for controls (Almond, Chay and Lee 2005; Black, Devereux and Salvanes 2007). These twins studies, thus, reinforce that birth weight may be an important determinant of outcomes over the life cycle but also that birth weight may be affected by and therefore proxying for family background characteristics such as parental schooling attainment and household wealth.

As Almond et al. (2005, 1032) put it, ‘birth weight is an “input”—i.e., a proxy for the initial endowment of an infant’s “health human capital”.’ The biomedical literature has established that birth weight is governed by two major processes: the duration of gestation and the intrauterine growth rate. Low birth weight (LBW) is caused by short gestation periods and/or retarded intrauterine growth (Kramer 1987, 664). For developing countries, the causal effects of factors such as material height, paternal height and weight on prematurity are all ruled out with high probability (Kramer1987, 722). A big part of variation in birth weight in developing countries, however, is related to intrauterine growth retardation (IUGR). For a population comparable to that of Vietnam, the National Low Birth Weight Survey of Bangladesh 2003-2004 shows that 23% of all LBW infants were pre-term, which means that the remainder (77%) of LBW infants were growth-retarded. Some of the preterm LBW infants also may have been growth-retarded as well as preterm (BBS and UNICEF, 2005, 24). For the sample used in the present study, 191 cases reported positive numbers of weeks premature, and not all of these children are LBW. In fact, of the ones born in 2001, the number of children with birth weight under 2.5 kg is 72, and half of these LBW cases were reported to have born premature by more than one week.

Given that at least some of the previous economics literature finds that treating birth weight as endogenously determined by family background is important, that the biomedical literature suggests that IUGR is likely to be affected by family background and our sample has a number of IUBR cases, and our estimates (see Table A1 in Appendix) suggest that family background affects birth weight, for use as an instrument we purge birth weight of its components that are correlated with family background. That is, we focus only on the accumulative impact of shocks since conception to birth on birth weight. We estimate the following linear relation for birth weight:

$$BW_i = B_0 + \sum_{k=0}^{K} a_k x_{ik} + \sum_{l=1}^{L} b_l y_{il} + \sum_{m=1}^{M} d_m z_{im} + R_i \quad (3),$$

where BW is birth weight; x_0 presents the number of weeks in preterm; other explanatory factors $x_k, k = 1, K$, $y_l, l = 1, L$, and $z_m, m = 1, M$, are the same as in equations (1), (2a), and (2b). R_i is the residual for this birth weight equation, which by construction is uncorrelated with all the factors $x_k, k = 1, K$, $y_l, l = 1, L$, and $z_m, m = 1, M$. By its nature, R is independent of parental schooling attainment, household assets and mothers' height and BMI. We interpret R as reflecting accumulated shocks of all kinds on the child in the womb on the child's birth weight. For our preferred estimates we use R as the identifying instruments. We note with this approach, there is not a concern about heterogenous partial effects because there is control for other factors, such as child gender, parental schooling attainment, mothers' height and BMI, household wealth and ethnicity, so in the second stage of 2SLS the variation (in fitted values) of HAZ^1 is driven by the variation in IUGR, which is directly relevant to early childhood undernutrition.

The data on birth weight were recorded in birth documents by health clinic staff at the times of birth, while the children's heights and test scores were collected subsequently by Young Lives fieldworkers. Because the measures were taken by different teams at different times, the likelihood of correlation between measurement errors in birth weight and those for the development outcomes is not a concern. However there is another measurement issue for the birth weight data: missing values for 198 (12 percent) of the targeted children (born in 2001) for this study. There is risk of introducing biases, of course, if birth weight is missing selectively. To deal with this issue, we use both the original data on birth weight and imputed birth weight in cases in which observed birth weight is not available, but only perceived relative size at birth. The imputed value is defined as the fitted value of an OLS regression with the following right-side variables: number of weeks premature, mother's weight in Round 1, household wealth index in Round 1, four dummy variables on the sentinel sites for the isolated communes, and finally, four dummy variables on the perception of child's size at birth reported by the mother.[14] We use the OLS regression (Table A1 in Appendix) to fill in the imputed birth weight for virtually all the children with missing birth weight. These imputed values are between the lower and the upper limits for actual birth weights. The average for the 174 imputed values is

[14] For the perception of child's size at birth, the child's mother was asked "Did you think the child was large, average or small at birth" and the codes are given 1 to 5 for the answers "Very large", "Large", "Average", "Small", and "Very small" correspondingly. Table A1 in the Appendix indicates the explanatory power of these dummy variables, and the goodness of fit for the birth weight equation.

statistically insignificantly different (though slightly lower) than that for the nonmissing cases (2981 versus 3091 gms). The difference is a small part of the standard deviation in actual birth weights.

4. Empirical results

4.1 The effects of HAZ deficit in infancy on post-infancy growth

Roughly a half of the sample under study were either stunted or moderately stunted at age 1 year ($HAZ^1 < 1$). We will refer to them as "the lower half", and the other half (the non-stunted: $HAZ^1 \geq -1$) as "the upper half". The mean of HAZ^1 for the upper half is -0.17, which is close to the median for the reference population, and we do not expect undernutrition around this median. The mean of HAZ^1 for the lower half (of stunted or moderately stunted subjects) equals -1.9, which is slightly above the stunting threshold of -2. As indicated in Table 1, the changes in HAZ over the post-infancy period differ across halves, with mixed pattern of change for the non-stunted children. Thus, we choose to focus only on the lower half, for which, the notion of catch-up is more meaningful.

Table 3 presents OLS and 2SLS estimates for equation 1 for growth (g) from age 1 to 8 years. The first column present results of the OLS regressions for the group of stunted children. ($HAZ^1 < -2$), and the others refer to stunted and moderately- stunted children ($HAZ^1 < -1$), with varied methods of estimations.[15] Note, firstly that the test statistics given in the lower part of Table 3 provide support for our selection of the residual component of birth weight, hereafter referred to as R, as the identifying instrument. We can comfortably reject the hypothesis on

[15] With respect to the sample of only stunted children, we fail to find a valid identifying instrument.

Table 3: Regressions of Models 1: growth in HAZ from age 1 to 8

	Stunted only ($HAZ^1 < -2$)	Stunted and moderate stunting ($HAZ^1 < -1$)		
	OLS	OLS	2SLS- 1IV	2SLS-2IV
Residual component R of birth weight	-0.097 (0.119)	-0.010 (0.067)	Instrument	Instrument
HAZ at age 1year	-0.587*** (0.090)	-0.457*** (0.041)	-0.502* (0.263)	-0.423** (0.174)
If boy	-0.020 (0.094)	0.023 (0.051)	0.017 (0.062)	0.029 (0.055)
If mother's first child	0.008 (0.096)	0.102* (0.053)	0.105* (0.055)	0.100* (0.053)
Born in March-August	0.005 (0.132)	-0.006 (0.076)	-0.004 (0.076)	-0.008 (0.076)
Mother's BMI	-0.006 (0.018)	0.012 (0.010)	0.013 (0.013)	0.011 (0.012)
Mother's height (normalized)	0.265*** (0.058)	0.179*** (0.031)	0.188*** (0.061)	0.172*** (0.047)
Mother's age 20 to 35 when child born	0.254* (0.133)	0.160** (0.078)	0.164** (0.078)	0.159** (0.077)
Mother is ethnic minority	0.012 (0.142)	-0.032 (0.088)	-0.048 (0.127)	Instrument
Mother completed LSE	0.131 (0.127)	0.164** (0.067)	0.163** (0.067)	0.166** (0.066)
Father completed LSE	0.019 (0.118)	-0.009 (0.063)	-0.008 (0.061)	-0.009 (0.060)
Wealth index R.1 (normalized)	0.084 (0.072)	0.081* (0.042)	0.085* (0.044)	0.082* (0.044)
Number of observations	246	649	649	649
R^2/ Centered R^2	0.25	0.22	0.22	0.22
Under-ident. p-value	NA	NA	0.000	0.000
Cragg-Donald F-statistic	NA	NA	13.0	15.0
Sargan's (over-ident.) p-value	NA	NA	NA	0.71

Notes: The 2SLS regressions use residual of birth weight as an instrument variable;

The standard errors are in parentheses, underneath the estimated coefficients;

The effects of regional factors and months of birth month are not displayed;

*** p<0.01, ** p<0.05, * p<0.1

under-identification.[16] Likewise, the Cragg-Donald F-statistics show that the identifying instrument is not weak as conventionally defined.[17] Moreover, the estimates with two instrument

[16] For the under-identification test, the Anderson canonical correlation LM statistics are 13.1 and 29.5 for the regressions in columns 3 and 4 correspondingly.

variables in the last column permit Sargan's over-identification test statistic, which provides important evidence for validity of the instrument variables. In particularly, it implies that the residual component R is unlikely to be correlated with the error term.

In the OLS regressions, we include R, which is excluded in the 2SLS estimates because R is the identifying instrument. If R were correlated with the disturbance term in the 2SLS estimates, we would fail to reject the hypothesis that the coefficients for R equal zero in the OLS estimates in the first two columns of Table 3.[18] The OLS associations of HAZ^1 with growth in HAZ between ages 1 and 8 years are significantly nonzero at about -0.5 for each of the two subsamples defined by HAZ^1 categories. This magnitude indicates that the predicated growth in HAZ between ages 1 and 8 years is altered by about half of the discrepancy, between HAZ^1 and the reference median. The 2SLS estimates of causal effects indicate similar patterns. The estimated causal effect of HAZ^1 on post-infancy growth for stunted and moderately-stunted children at age 1 year is statistically significant at the 5 percent level and indicates that about half of the deficit in HAZ at age 1 year is not transmitted to HAZ at age 8 years, so for this group there is important (about half) catchup. This may reflect a channel in which, for example, there are compensating increased investments by household in children with lower values of HAZ^1 who are stunted or moderately stunted.

Our finding of partial recovery for those stunted or moderately stunted at age 1 is consistent with a small previous literature investigating catchup growth in height in developing countries using methods to control for the endogeneity of initial height, though not necessarily with the initial

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[17] For more information, see the results of the first-stage regressions in Table A2 in Appendix.

[18] The p-value for the tests for zero coefficient estimates of birth weight in the OLS regressions for the 1st and 2nd columns are 0.41 and 0.88 respectively.

height limited to the first 2 years of life. Hoddinott and Kinsey (2001, 425), for example, estimate an equation for height growth for Zimbabwe children in the aftermath of a drought and report a significant 2SLS coefficient estimate of -0.434 with a specification in which the interpretation is similar to that in our specification. Alderman, Hoddinott and Kinsey (2006, 469), using a similar specification with IV maternal fixed effects estimates and an older age range in the same sample, report a significant -0.571. Mani's (2012, 693) preferred first-difference generalized method of moments (FD-GMM) estimate for Indonesian children is for a catch-up coefficient of 0.23 in a specification in which complete catchup would be 0.0 and no catchup would be 1.0. She interprets her estimates to suggest that malnutrition during childhood causes some but not substantial growth retardation in future height. She also finds, similar to our results, that stunted children exhibit larger catch-up effects compared to children who do not suffer from growth faltering at an early age.

Some of the 2SLS estimates other than for HAZ^1 in Table A2 in Appendix also are interesting. The change in HAZ in the post-infancy years for the first child is significantly greater by 0.1 than for other children. The first child often gets more attention from household members and, at least until younger siblings are born, does not have to share household resources with siblings. That may be particularly important for households with limited food supplies. Another factor that has significant explanatory power in both HAZ^1 and the growth between ages 1 and 8 years is mother's height. This is not purely biological because it reflects the nourishment that the mother received when she was a child. An additional standard deviation of mother's height is associated with higher child HAZ at age 1 year by 0.20 and with further change about 0.15 to 0.2 in HAZ in the post-infancy period. The magnitude is even higher for the children who are stunted at age one year. Demographic factors such as sex of the child and being a member of an ethnic minority are not significantly associated with post-infancy growth. The impacts of these two factors apparently are once-off. Furthermore, mother's schooling attainment is not relevant to HAZ in early childhood, but is important for growth over the ages from 1 to 8 years. Finally, wealth in childhood is important for the growth in the first year, but in the post-infancy period, it is not significant at five percent level.

4.2 Estimates for the impact of HAZ^1 on educational outcomes in late childhood

Table 4 presents Probit and linear probability OLS and 2SLS estimates for one important educational outcome: whether the child is lagging behind in terms of grade progression and has not made it to grade 5 by year 2011. The residual component R of birth weight is used in both 2SLS regression options, of which one regression uses additionally the variable on BMI of mother as an identifying instrument. There are some interesting and a priori plausible results: those who are significantly more likely to lag behind are boys, the youngest (being born in December 2001), with mothers either too young (under 20 when the child was born) or too old (over 35), with mothers from an ethnic minority and with less household wealth. Most importantly, however, HAZ^1 does not have a statistically significant (at the conventional 5% level) impact on the likelihood of the child to attain Grade 5 at age 11 years. The OLS estimate is significant at the 10% level, but very small, indicating only a drop of -0.013 in the probability with a unit increase in HAZ.

Table 4: Regression of equation 2a: lagging behind and being under grade 5 in 2011

	Probit-mfx	OLS	2SLS-one IV	2SLS-two IVs
HAZ at age 1 year	-0.009 (0.006)	-0.013* (0.008)	-0.026 (0.040)	-0.031 (0.038)
If boy	0.027** (0.011)	0.039*** (0.015)	0.037** (0.016)	0.036** (0.016)
If mother's first child	-0.003 (0.011)	-0.002 (0.016)	-0.001 (0.016)	-0.001 (0.016)
Born in March-August	-0.042** (0.018)	-0.044* (0.023)	-0.043* (0.023)	-0.043* (0.023)
Born in December	0.121*** (0.047)	0.153*** (0.032)	0.155*** (0.032)	0.156*** (0.032)
Mother's BMI	-0.001 (0.002)	-0.002 (0.003)	-0.001 (0.003)	Instrument
Mother's height (normalized)	-0.007 (0.006)	-0.010 (0.008)	-0.006 (0.014)	-0.004 (0.014)
Mother's age 20 to 35 when child born	-0.054** (0.025)	-0.063*** (0.023)	-0.063*** (0.023)	-0.063*** (0.023)
Mother is ethnic minority	0.099** (0.041)	0.209*** (0.030)	0.204*** (0.032)	0.203*** (0.032)
Mother completed Lower Secondary Education (LSE)	0.003 (0.016)	0.016 (0.019)	0.018 (0.020)	0.018 (0.020)
Father completed LSE	-0.026* (0.014)	-0.028 (0.019)	-0.026 (0.020)	-0.025 (0.020)
Wealth index Round 1 (normalized)	-0.024*** (0.009)	-0.041*** (0.013)	-0.038** (0.015)	-0.038** (0.015)
Number of observations	1155	1155	1155	1155
Pseudo R^2 /R^2/ Centered R^2	0.26	0.18	0.18	0.17
Under-identification p-value	NA	NA	0.000	0.000
Cragg-Donald F-statistic	NA	NA	44.2	25.0
Sargan (over-ident) p-vlue	NA	NA	NA	0.71

Notes: Both of the 2SLS regressions use the residual component of birth weight as an instrument.

The standard errors are in parentheses, underneath the estimated coefficients;

The effects of regional factors are not displayed, nor the dummies on some of birth months;

*** p<0.01, ** p<0.05, * p<0.1 for 0.

Tables 5 (OLS) and 6 (2SLS) present estimates for five cognitive test scores in late childhood: Two (Math and PPVT) are from the Young Lives survey Round 3 when the children were 8 years old and the other two (Grade 5- math and Vietnamese) are from the 2011-12 school survey for those children who were on track and in Grade 5 at 10 years of age. Because not all children born in 2001 had progressed to grade 5 by the time of the school survey, there may be concern about the risk of selectivity bias for the last two of these test scores. However, we find it is hard to deal effectively with both possible endogeneity bias and selectivity bias. We chose to focus on the former because it is the focus on this study, reinforced by the evidence in Table 4 that HAZ^1 has little impact on the likelihood of reaching grade 5 by the time of school survey. Furthermore, we find that there is negligible difference in the initial health endowments between those participating and not participating in the school survey. In fact, for all the children born in 2001, the mean birth weight is 3091 grams, and for the ones taking part in the school survey the mean is 3094. The mean height-for-age for these groups of children also are virtually identical at age 12 months.[19]

In Table 5, we present two sets of results for the regressions that include either birth weight or the residual component R. For ease of interpretation, all four test scores are normalized to be in terms of standard deviations. They show that neither of the variables, birth weight and the residual component R, has significant explanatory power on any of the test score outcomes. All of the hypotheses of zero coefficients of birth weight and of R cannot be rejected at reasonable level of significance.[20] The OLS estimates indicate positive associations between HAZ^1 and test scores such that an increase of one unit of HAZ^1 is related to from 0.033 to 0.091 standard deviations in test scores. With the only exception being for PPVT, the OLS estimates for the coefficients of HAZ^1 are statistically significant. Under the assumption that the specification in

[19] The standard deviation for the sample of all the children born in 2001 is 442, and that for the subsample of all the school survey participants is 444.

[20] With respect to the residual component R, which we use as an identifying instrument in 2SLS regressions below, the p-values for its coefficients equalling zero in the OLS regressions in the right half of Table 5 are 0.57; 0.28; 0.31; and 0.59, respectively.

Table 5 is the correct specification, it is of interest to ask how the estimates are affected if one of right-side controls is not included. Table A3 in the Appendix presents OLS regressions, similar to those in Table 5, but with the variable on wealth index Round 1 excluded in the first four columns and with paternal schooling (and wealth index Round1) excluded in the last four columns. In comparison to those in Table 5, the coefficient estimates for HAZ^1 in Table A3 are greater in magnitudes, and with no less (more, in the case of PPVT) statistical significance. These results show how OLS estimates can be sensitive to omitted factors, particularly those that may have affected the right-side variable of interest. That is not a problem for 2SLS regressions with good diagnostic tests, as we show for our preferred 2SLS estimates below.

Because of the possible endogeneity of HAZ^1, once again, our preferred estimates are the 2SLS estimates in Table 6. The results in the left half of this table use one identifying instrument (R), and those in the right half use two (R, BMI of mother). The diagnostic tests, summarized at the bottom of Table 6, support the validity of the identifying instruments, including the residual component of birth weight.

The most important results, of course, pertain to the estimated impacts of HAZ^1 on test scores. Unlike most of the results of the OLS in Tables 5 and A3, none of the 2SLS estimated coefficients of HAZ^1 on test scores is statistically significant even at the 10% level. Thus, we fail to find any support for the hypothesis that HAZ^1 causes these cognitive outcomes in late childhood. This remains true, even after two important factors, the wealth index in Round 1 and father's schooling attainment removed from the specifications (Table A4 in Appendix). This latter comparison implies that 2SLS with good diagnostic tests can overcome problems of unobserved factors, unlike the findings discussed above for the OLS estimates in Table 5 and Tables A3 in the Appendix.

Table 5: OLS regressions for equations 2b

	R3-Math	R3-PPVT	G5-Math	Vietnamese	R3-Math	R3-PPVT	G5-Math	Vietnamese
Birth weight	-0.017 (0.060)	-0.049 (0.063)	-0.033 (0.080)	0.044 (0.077)	NA	NA	NA	NA
Residual comp. R of birth weight	NA	NA	NA	NA	-0.037 (0.065)	-0.075 (0.069)	-0.089 (0.087)	-0.046 (0.084)
HAZ at age 1 year	0.090*** (0.025)	0.031 (0.027)	0.069** (0.035)	0.059* (0.034)	0.091*** (0.025)	0.033 (0.027)	0.073** (0.034)	0.067** (0.033)
If boy	-0.033 (0.048)	0.011 (0.051)	-0.099 (0.065)	-0.384*** (0.063)	-0.034 (0.048)	0.006 (0.051)	-0.100 (0.064)	-0.376*** (0.062)
If mother's first child	0.071 (0.051)	0.064 (0.054)	0.123* (0.068)	0.184*** (0.065)	0.074 (0.050)	0.072 (0.053)	0.130* (0.066)	0.177*** (0.064)
Born in March-August	0.147** (0.073)	0.166** (0.078)	-0.078 (0.100)	-0.086 (0.097)	0.148** (0.073)	0.166** (0.078)	-0.077 (0.100)	-0.084 (0.097)
Mother's BMI	-0.000 (0.009)	0.012 (0.010)	0.014 (0.012)	0.007 (0.012)	-0.001 (0.009)	0.010 (0.010)	0.013 (0.012)	0.008 (0.012)
Mother's height (normalized)	0.026 (0.026)	0.024 (0.028)	0.014 (0.035)	0.014 (0.034)	0.025 (0.026)	0.021 (0.028)	0.011 (0.035)	0.014 (0.034)
Mother's age 20 to 35 when child born	0.116 (0.074)	0.020 (0.078)	0.012 (0.101)	-0.022 (0.097)	0.116 (0.073)	0.020 (0.078)	0.011 (0.101)	-0.026 (0.097)
Mother is ethnic minority	-0.291*** (0.095)	-0.203** (0.100)	-0.421*** (0.148)	-0.445*** (0.144)	-0.290*** (0.095)	-0.200** (0.100)	-0.420*** (0.148)	-0.449*** (0.144)
Mother completed LSE	0.228*** (0.061)	0.299*** (0.065)	0.259*** (0.078)	0.206*** (0.075)	0.226*** (0.061)	0.294*** (0.065)	0.255*** (0.078)	0.207*** (0.075)
Father completed LSE	0.095 (0.059)	0.116* (0.063)	0.160** (0.077)	0.222*** (0.074)	0.094 (0.059)	0.114* (0.063)	0.158** (0.077)	0.220*** (0.074)
Wealth index R1 (normalized)	0.279*** (0.041)	0.256*** (0.043)	0.342*** (0.057)	0.267*** (0.055)	0.278*** (0.041)	0.254*** (0.043)	0.340*** (0.057)	0.267*** (0.055)
Number of obs.	1,152	1,165	867	866	1,152	1,165	867	866
R-squared	0.30	0.26	0.17	0.21	0.30	0.26	0.17	0.21

Notes: All the outcomes have been normalized so they have mean zero and standard deviation 1.0;

The standard errors are in parentheses, underneath the estimated coefficients.

Not display: the regional factors and some of the dummies for month of birth;

*** p<0.01, ** p<0.05, * p<0.1

Table 6: 2SLS regressions for equations 2b

	With residual component of birth weight R as instrument				With R and BMI of mother as instruments			
	R3-Math	R3-PPVT	G5-Math	Vietnamese	R3-Math	R3-PPVT	G5-Math	Vietnamese
HAZ at age 1 year	0.016 (0.129)	-0.124 (0.142)	-0.093 (0.159)	-0.019 (0.154)	0.022 (0.122)	-0.051 (0.131)	-0.003 (0.144)	0.035 (0.139)
If boy	-0.048 (0.053)	-0.021 (0.057)	-0.132* (0.070)	-0.392*** (0.066)	-0.047 (0.052)	-0.009 (0.056)	-0.119* (0.068)	-0.384*** (0.066)
If mother's first child	0.077 (0.049)	0.079 (0.054)	0.137** (0.067)	0.182*** (0.064)	0.076 (0.049)	0.069 (0.053)	0.126* (0.066)	0.175*** (0.063)
Born in March-August	0.151** (0.073)	0.172** (0.079)	-0.076 (0.101)	-0.084 (0.096)	0.150** (0.073)	0.165** (0.078)	-0.077 (0.100)	-0.084 (0.096)
Mother's BMI	0.001 (0.010)	0.015 (0.011)	0.019 (0.013)	0.011 (0.013)	Instrument	Instrument	Instrument	Instrument
Mother's height (normalized)	0.047 (0.046)	0.067 (0.050)	0.064 (0.061)	0.042 (0.059)	0.045 (0.043)	0.043 (0.046)	0.033 (0.056)	0.023 (0.054)
Mother's age 20 to 35 when child born	0.119 (0.073)	0.026 (0.079)	0.017 (0.101)	-0.023 (0.097)	0.118 (0.073)	0.021 (0.078)	0.013 (0.100)	-0.025 (0.096)
Mother is ethnic minority	-0.315*** (0.103)	-0.254** (0.112)	-0.455*** (0.152)	-0.471*** (0.149)	-0.313*** (0.103)	-0.233** (0.110)	-0.433*** (0.150)	-0.455*** (0.147)
Mother completed LSE	0.236*** (0.062)	0.314*** (0.068)	0.274*** (0.080)	0.217*** (0.076)	0.235*** (0.062)	0.307*** (0.067)	0.268*** (0.079)	0.214*** (0.076)
Father completed LSE	0.108* (0.063)	0.142** (0.067)	0.177** (0.078)	0.230*** (0.075)	0.107* (0.062)	0.127* (0.066)	0.165** (0.077)	0.223*** (0.074)
Wealth index R.1 (normalized)	0.294*** (0.049)	0.288*** (0.053)	0.367*** (0.062)	0.282*** (0.061)	0.293*** (0.048)	0.278*** (0.052)	0.360*** (0.062)	0.277*** (0.060)
Number of obs	1,152	1,165	867	866	1,152	1,165	867	866
Centered R^2	0.29	0.24	0.15	0.20	0.29	0.25	0.17	0.21
Under-id. p-value	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Cragg-Donald F	42.8	40.5	39.7	39.3	23.9	23.4	23.9	23.9
Sargan's p-value	NA	NA	NA	NA	0.89	0.16	0.16	0.40

Notes: All the outcomes have been normalized so they have mean zero and standard deviation 1.0.

The standard errors are in parentheses, underneath the estimated coefficients.

Not display: the regional factors and some of the dummies for month of birth;

*** p<0.01, ** p<0.05, * p<0.1

Some of the other estimates in Table 6 have interesting patterns. Girls perform better than boys, but the gender gap is statistically significant (at the 5% level) only in reading Vietnamese. However the effect is fairly large, indicating that boys performed nearly -0.4 standard deviations worse in Vietnamese. The first-borns perform significantly better than higher-order births in Grade-5 mathematics and reading Vietnamese. This finding is not totally a surprise. Heiland (2009, 1815), for example, finds that 'first-borns display significantly better verbal ability compared to children later in the birth order' in the (US) National Longitudinal Survey of Youth (1979 cohort) with controls for family size and child characteristics. In the current study, we find significantly better performance in the range of 0.137 to 0.182 standard deviations by the first-born children correspondingly in the Grade 5-math and the reading Vietnamese tests.

Ethnic gaps have been always a major issue in poverty reports on Vietnam (Baulch et al., 2007). Ethnic minority groups mostly live in areas with underdeveloped infrastructure, particularly educational and healthcare services. Therefore it is no surprise that the estimated effects for the ethnic minorities are significantly negative for all test outcomes. But the magnitudes merit note, from -0.254 to -0.471 standard deviations.

Finally, parental schooling attainment and household wealth index (in early childhood) have significant and substantial coefficient estimates for these test scores. Mother having completed lower secondary education is significantly nonzero for all four test scores with estimates in the range of from 0.214 to 0.314 standard deviations, father having completed lower secondary education is significantly nonzero for three of the four test scores with estimates in the range of from 0.107 to 0.230 standard deviations, and household wealth is significantly nonzero for all four test scores with estimates in the range of from 0.277 to 0.367 standard deviations.

5. Discussion and conclusion

There is a considerable literature, summarized in the introduction, on the importance of early-life nutrition, particularly before age 2 years, on subsequent outcomes over the life cycle with substantial emphasis on impacts that work through cognitive development in late childhood. Some recent prominent studies have maintained, for example, that the life cycle stage up to age 2 years is a critical window and that growth faltering before that age is irreversible in terms of its subsequent impacts, importantly including subsequent cognitive outcomes.

However, even though growth faltering in early life reflects many parental choices related to nutrient consumption and infectious diseases, most of the literature on the impact of growth faltering before 2 years of age on subsequent outcomes does not attempt to control for the endogenous nature of such growth faltering. In fact we have been able to find only less than a handful of studies that attempt to control for the endogeneity of child growth in the first two years of life in investigations of its impact on physical growth into late childhood, only one study that attempts to control for the endogeneity of child growth in the first two years of life in investigations of its impact on late childhood cognitive abilities and no studies that attempt to control for the endogeneity of child growth in the first two years of life in investigations of its impact on late childhood educational outcomes including cognitive achievements.

Our study contributes with new estimates that control for the endogeneity of growth faltering before age 2 years on growth into late childhood and on a set of educational outcomes in late childhood. We use for an identifying instrument the estimated impact of the accumulated shocks that affect the newborn's birth weight, beyond and above those related to household socioeconomic status characteristics. The test statistics support using the residual component of birth weight as a valid identifying instrument. Moreover, our analysis shows that our 2SLS results with this instrument variable do not suffer from the problem of overestimation as a consequence of missing a relevant factor, as it is the case for the OLS estimates in this study.

We find strong evidence of growth catch-up for those children who were stunted and moderately stunted at age 1 year. Our estimates indicate that by age 8 years, children who were moderately stunted or stunted at age 1 year, recover about half of the deficit in height-for-age. This adds to a small number of results in the literature that investigate this question with approaches that treat growth measures before age 2 years as endogenous. The result of substantial catch up, at least for those who were stunted or moderately-stunted before age 2 years, contrasts sharply at least in terms of subsequent physical growth into late childhood with the prominent and influential position that growth faltering before 2 years of age is irreversible.

We find the estimated causal impacts of height-for-age at age 1 year on educational outcomes including progressing through school grades on track are negligible. These results raise questions about the claim in much of the literature summarized in the introduction that the impacts of poor child growth before age 2 years are manifested in and through poor educational performance in late childhood. Our results must be qualified, of course, because we have a measure of growth faltering at 1 year rather than at 2 years as is emphasized in this literature and the Glewwe and King (2001) results suggest that the difference may be important, even though the INCAP data that we summarize suggests that the variance in HAZ at age 1 year is a very good though not perfect proxy for the variance in HAZ at age 2.

Parental, particularly maternal, school attainment and household wealth in infancy are statistically significant and substantial factors in the relations for early-life HAZ and for late-childhood educational achievement. Our results thus are consistent with the possibility that such household socioeconomic status indicators and child characteristics drive both early-life growth faltering and poor school and cognitive outcomes in late childhood, but not the former causing the latter.

In any case our study raises the need for further research to collect better information with which to assess the challenges that our results pose to a strong and influential position on the lack of catchup growth after age 2 and on the critical role of growth faltering before age 2 on late childhood schooling and cognitive test performance.

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Appendix: Supporting Tables

Table A1: OLS Regression of birth weight (in grams)

	Coefficient	t-statistic
The baby was very large	759.71	9.79
The baby was large	411.18	17.69
The baby was small	-333.14	-14.64
The baby was very small	-622.55	-12.29
Number of weeks premature	-87.01	-10.57
Wealth index Round 1	197.56	4.97
Mother's weight	6.87	6.2
Cluster dummy No.3	-68.04	-1.64
Cluster dummy No.4	88.78	2.32
Cluster dummy No.10	68.96	1.7
Cluster dummy No.12	-123.22	-2.74
Constant	2705.02	47.65
Number of observations	1675	
F-statistic	133.3	
R-squared	0.47	

Note: For the perceived size baby, the mother/caregiver was asked "Did you think the child was large, average or small at birth". The codes are recorded for the answers "Very large", "Large", "Average", "Small", and "Very small".

Table A2: Results of the first-stage regression of Model 1

For the stunted or moderately stunted children ($HAZ^1 < -1$)

	Coef.	t-stat.
Residual component R of birth weight (kgm)	0.231***	3.60
If boy	-0.151***	-3.09
If mother's first child	0.080	1.58
Born in March-to-August	0.041	0.56
Mother's BMI	0.033***	3.36
Mother's height (normalized)	0.200***	7.05
Mother age 20 to 35 when child born	0.091	1.21
Mother is ethnic minority	-0.345***	-4.12
Mother completed LSE	-0.018	-0.27
Father completed LSE	0.011	0.18
Wealth index R1 (normalized)	0.085**	2.1
Number of observations	649	
R-square	0.24	

Note: The effects of regional factors and some of those for birth month are not displayed; *** p<0.01, ** p<0.05, * p<0.1

Table A3: OLS regressions for equations 2b,

(with variables removed: wealth index Round 1 in the first four columns; and wealth index Round 1 and father's schooling attainment for the last four columns)

	R3-Math	R3-PPVT	G5-Math	Vietnamese	R3-Math	R3-PPVT	G5-Math	Vietnamese
HAZ at age 1 year	0.110*** (0.025)	0.047* (0.026)	0.085** (0.034)	0.080** (0.033)	0.122*** (0.025)	0.062** (0.026)	0.092*** (0.034)	0.089*** (0.033)
If boy	-0.014 (0.049)	0.024 (0.052)	-0.088 (0.065)	-0.366*** (0.062)	-0.021 (0.049)	0.020 (0.051)	-0.092 (0.065)	-0.368*** (0.063)
If mother's first child	0.081 (0.051)	0.078 (0.054)	0.142** (0.068)	0.187*** (0.065)	0.079 (0.050)	0.064 (0.053)	0.124* (0.067)	0.172*** (0.065)
Born in March-August	0.156** (0.075)	0.172** (0.079)	-0.067 (0.102)	-0.075 (0.098)	0.157** (0.074)	0.169** (0.078)	-0.050 (0.100)	-0.051 (0.097)
Mother's BMI	0.006 (0.010)	0.017 (0.010)	0.020 (0.013)	0.013 (0.012)	0.004 (0.010)	0.017* (0.010)	0.021* (0.013)	0.013 (0.012)
Mother's height (normalized)	0.038 (0.027)	0.034 (0.028)	0.028 (0.036)	0.025 (0.034)	0.037 (0.027)	0.026 (0.028)	0.033 (0.035)	0.034 (0.034)
Mother's age 20 to 35 when child born	0.091 (0.075)	-0.002 (0.079)	-0.029 (0.103)	-0.057 (0.098)	0.072 (0.075)	-0.005 (0.078)	-0.038 (0.101)	-0.066 (0.098)
Mother is ethnic minority	-0.482*** (0.092)	-0.379*** (0.097)	-0.577*** (0.148)	-0.564*** (0.144)	-0.507*** (0.093)	-0.398*** (0.097)	-0.602*** (0.147)	-0.604*** (0.144)
Mother completed LSE	0.308*** (0.061)	0.370*** (0.065)	0.333*** (0.079)	0.268*** (0.075)	0.366*** (0.056)	0.458*** (0.059)	0.435*** (0.072)	0.387*** (0.070)
Father completed LSE	0.158*** (0.059)	0.176*** (0.063)	0.239*** (0.077)	0.282*** (0.074)	NA	NA	NA	NA
Number of obs.	1,152	1,165	867	866	1,152	1,165	867	866
R-squared	0.27	0.24	0.14	0.19	0.30	0.26	0.17	0.21

Notes: All the outcomes have been normalized so they have mean zero and standard deviation 1.0;

The standard errors are in parentheses, underneath the estimated coefficients.

Not display: the regional factors and some of the dummies for month of birth;

*** p<0.01, ** p<0.05, * p<0.1

Table A4: 2SLS regressions for equations 2b

(with two variables (wealth index R1 and father's schooling attainment removed, identifying instrument: residual component of birth weight)

	R3-Math	R3-PPVT	G5-Math	Vietnamese
HAZ at age 1 year	0.009	-0.136	-0.110	-0.035
	(0.133)	(0.148)	(0.166)	(0.159)
If boy	-0.029	-0.004	-0.117	-0.381***
	(0.053)	(0.057)	(0.072)	(0.068)
If mother's first child	0.078	0.078	0.140**	0.178***
	(0.051)	(0.055)	(0.069)	(0.066)
Born in March-August	0.171**	0.192**	-0.052	-0.061
	(0.076)	(0.081)	(0.104)	(0.099)
Mother's BMI	0.009	0.022**	0.028*	0.018
	(0.010)	(0.011)	(0.014)	(0.014)
Mother's height (normalized)	0.072	0.094*	0.097	0.069
	(0.049)	(0.054)	(0.065)	(0.063)
Mother's age 20 to 35 when child born	0.084	-0.012	-0.056	-0.088
	(0.075)	(0.080)	(0.104)	(0.099)
Mother is ethnic minority	-0.555***	-0.498***	-0.674***	-0.647***
	(0.115)	(0.125)	(0.159)	(0.156)
Mother completed LSE	0.401***	0.494***	0.467***	0.403***
	(0.067)	(0.073)	(0.081)	(0.077)
Number of observations	1,152	1,165	867	866
R-squared	0.25	0.20	0.09	0.16
Under-identification p-value	0.000	0.000	0.000	0.000
Cragg-Donald F	41.0	38.7	38.5	38.1

Notes: All the outcomes have been normalized to have mean zeros and standard deviations 1.0.

The standard errors are in parentheses, underneath the estimated coefficients.

Not display: the regional factors and some of the dummies for month of birth;

*** p<0.01, ** p<0.05, * p<0.1