

The Consequences of Early Childhood Growth Failure over the Life Course

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ABSTRACT

This paper examines the impact over the life course of early childhood growth failure as measured by achieved height at 36 months. It uses data collected on individuals who participated in a nutritional supplementation trial between 1969 and 1977 in rural Guatemala and who were subsequently re-interviewed between 2002 and 2004. It finds that individuals who did not suffer growth failure in the first three years of life complete more schooling, score higher on tests of cognitive skill in adulthood, have better outcomes in the marriage market, earn higher wages and are more likely to be employed in higher-paying skilled labor and white-collar jobs, are less likely to live in poor households, and, for women, fewer pregnancies and smaller risk of miscarriages and stillbirths. Growth failure has adverse impacts on body size and several dimensions of physical fitness in adulthood but does not have marked effects on risk indicators of cardiovascular and related chronic diseases. These results provide a powerful rationale for investments that reduce early-life growth failure.

Keywords: early life growth failure, undernutrition, human capital, wages, poverty, fertility, chronic disease

1. INTRODUCTION

Researchers interested in human capital have expended considerable effort to understand the determinants of schooling and the subsequent acquisition of cognitive, noncognitive, and job-specific skills. A related literature assesses the consequences of these adult forms of human capital for economic productivity, health, fertility, marriage-market outcomes, and risky behaviors. By contrast, much less is known about how human capital formed in early-life affects outcomes across the life course, and much of what is known emanates from studies on the United States and Great Britain, with uncertain applicability to poorer settings.¹ Here we examine, in the context of a poor country, the consequences of one dimension of early-life human capital formation—growth failure in the first three years of life.

Linear (height) growth failure is widespread in poor countries. An estimated 175 million or more preschool children are stunted, meaning their height given their age is more than two standard deviations below that of the international reference standard (Black et al. 2008). The physical and neurological consequences of growth failure arising from chronic undernourishment—a proxy for nutrient and energy inadequacy at the cellular level—have been studied extensively. Chronic nutrient depletion, resulting from inadequate nutrient intake, infection, or both, leads to retardation of skeletal growth in children and to a loss of, or failure to accumulate, muscle mass and fat (Morris 2001). Lost linear growth in early life typically is not fully regained (Martorell 1999; Stein et al. 2010). Chronic undernutrition also has neurological consequences. It adversely affects the hippocampus by reducing dendrite density (Blatt et al. 1994; Mazer et al. 1997; Ranade et al. 2008) and by damaging the chemical processes associated with spatial navigation, memory formation (Huang et al. 2003), and memory consolidation (Valadares and de Sousa Almeida 2005). Chronic undernutrition results in reduced myelination of axon fibers, thus reducing the speed at which signals are transmitted

¹ Shonkoff and Phillips (2000) summarize much of the early literature on this topic, which is updated in Doyle et al. (2009). The scant literature for poor countries focuses almost exclusively on schooling-related outcomes (see Victora et al. 2008 for a review). A few researchers adduce impacts on subsequent life outcomes such as lowered economic productivity (Alderman, Hoddinott, and Kinsey 2006; Behrman, Alderman, and Hoddinott 2004; Horton, Alderman, and Rivera 2008), but these estimates of indirect effects rely on strong assumptions in order to link adult outcomes to child outcomes. For example, Alderman, Hoddinott, and Kinsey (2006) link their findings on the impact of undernutrition on schooling to separate studies that assess the returns to schooling in the Zimbabwean manufacturing sector. They make the strong—and for Zimbabwe, incorrect—assumption that these returns are a good representation of what future earnings will be.

(Levitsky and Strupp 1995). It decreases the number of neurons in the locus coeruleus (Pinos et al. 2006), which plays a role in cortisol synthesis and consequently, the ability to cope with stress. Chronic undernutrition damages the occipital lobe and the motor cortex² (Benítez-Bribiesca, De la Rosa-Alvarez, and Mansilla-Olivares 1999) leading to delays in the evolution of locomotor skills (Barros et al. 2006). Brown and Pollitt (1996) note that delayed development of motor skills such as crawling and walking, together with lethargy and increased incidence of illness in undernourished infants, reduces their interactions with adults and with their environment, which in turn also slows cognitive development.

Given these physical and neurological consequences, our objective is to assess whether there are causal links between early-life growth failure and a range of life-course outcomes observed up to middle adulthood. To do so, we draw on data collected over a 35 year period in Guatemala. Between 1969 and 1977, two nutritional supplements, randomly assigned at the village level, were provided to preschool children in four villages in Guatemala. Between 2002 and 2004, we traced and interviewed individuals who had been exposed to this intervention and who were by then adults 25 to 42 years of age. These data include prospective anthropometric measures that capture early-life growth failure and outcomes across the life course, including schooling, household formation, fertility, health, wages, and consumption. Because growth failure is behaviorally determined, we need identifying variables that ensure our results are not “plagued by potential bias due to unobserved heterogeneity” (Strauss and Thomas 2008, 3382). We argue that these data contain such variables, and we carefully test the robustness of our claim. Using instrumental variable (IV) estimators, we demonstrate that individuals who did not suffer growth failure in the first three years of life have dramatically better lives. They complete more schooling, score higher on tests of cognitive skill in adulthood, have better outcomes in the marriage market, earn higher wages, and are more likely to be employed in higher-paying skilled labor and white-collar jobs, and they are less likely to live in poor households as adults, and, if they are women, they will have fewer pregnancies and smaller risk of miscarriages and stillbirths. Growth failure has adverse impacts on body size and

² Specifically, it leads to dendrites that are shorter, malformed, and less numerous.

several dimensions of physical fitness in adulthood but does not have marked effects on risk factors for cardiovascular and related chronic diseases.

The remainder of the paper is organized as follows: Section 2 sets out a simple model that illustrates the issues faced in identifying the impact of early-life growth failure. We describe our data in Section 3 and present the consequences of early-life growth failure in middle adulthood in Section 4. Section 5 reports on some robustness checks, and Section 6 concludes.

2. MODELING

To elucidate issues surrounding the identification of the causal effects of early-life growth failure, we begin with the model outlined in Behrman and Hoddinott (2005). Our representation of growth failure is an observed measure of nutritional status, individuals' height given age, which appears as an argument in the welfare function of the households in which they reside (Behrman and Deolalikar 1988; Strauss and Thomas 1995; 2008). Welfare is assumed to increase as nutritional status improves, though possibly at a diminishing rate. Decisions that parents make about devoting resources to children's nutrition are constrained in several ways. Constraints on resources reflect the limits of available income and time, as well as prices faced by households. Constraints also arise from the production process for nutritional status. These constraints link nutrient intakes—the physical consumption of macronutrients (calories and protein) and micronutrients (minerals and vitamins)—as well as time devoted to the production of health and nutrition; household sanitation, safe water, and hygienic practices; locality characteristics such as the use of preventative and curative health facilities and the prevalence of infectious diseases; and the individual's genetic make-up and knowledge and skill regarding the combination of these inputs to produce nutritional status. Maximizing the household welfare function subject to these constraints generates a set of first-order conditions that can be solved to yield a reduced-form child nutritional status demand function:

$$\mathbf{HAZ}_{i,t} = \alpha_C' \cdot \mathbf{C}_i + \alpha_M' \cdot \mathbf{M}_t + \alpha_W' \cdot \mathbf{W}_t + \alpha_P' \cdot \mathbf{P}_t + \alpha_{inv}' \cdot \mathbf{Q}_{inv} + \alpha_{vary}' \cdot \mathbf{Q}_{vary}_{i,t} + v_{i,t} \quad (1)$$

where $\mathbf{HAZ}_{i,t}$ is a measure of the extent of growth failure (height-for-age z-score in early childhood),³ \mathbf{C}_i is a vector of child characteristics such as sex and genotype, \mathbf{M}_t is a vector of characteristics of the principal caregiver, \mathbf{W}_t captures household wealth, \mathbf{P}_t is a vector of all relevant prices, \mathbf{Q} is a vector of health, sanitation, and environmental characteristics in the locality in which the child lives that are assumed to influence nutritional status (some of these,

³ Z-scores are used to normalize measured heights and weights against those found in reference (usually well-nourished) populations. They are age and sex specific; a z-score of height-for-age is defined as measured height minus median height of the reference population, all divided by the standard deviation of the reference population for that age/sex category. Therefore, a z-score of -2 for an individual child means that his or her height is two standard deviations below the median for the reference population.

Q_{inv} , are time invariant, while others, $Q_{vary}_{i,t}$, vary over time), and t refers to the early-life period. The α 's are vectors of parameters to be estimated and $v_{i,t}$ is a disturbance term that reflects, for example, shocks in nutritional status due to random shocks in the infectious disease environment for particular children.

Next, consider a vector of outcomes Y in a later life-cycle period ($t + n$), for individual i that is related to early-life nutrition in period t in the following way:

$$Y_{i,t+n} = \beta \cdot HAZ_{i,t} + \gamma' \cdot X_{i,t} + U_{i,t+n} \quad (2)$$

For example, an element of $Y_{i,t+n}$ could be $W_{i,t+n}$, the hourly wages of person i in adulthood. $X_{i,t}$ is a vector of control variables with associated parameters γ , and $U_{i,t+n}$ is a vector of disturbance terms. $X_{i,t}$ consists of individual characteristics, characteristics of the household in which the individual resided as a child and time-varying and time-invariant locational characteristics. It includes all elements of C_i , M_t , W_t , P_t , Q_{inv} , and $Q_{vary}_{i,t}$ which potentially affect $Y_{i,t+n}$ over and above their impact through HAZ in childhood. For example, macroeconomic conditions vary over time, and these may affect individuals' success in the labor market. By including birth-year dummy variables in the empirical specification of $X_{i,t}$, we thus control for macroeconomic and other shocks, common to each birth-year cohort. Some elements of C_i , and $Q_{vary}_{i,t}$ do not appear in $X_{i,t}$. In Section 4, we motivate their exclusion in the context of our identification strategy.

3. DATA: THE 1969–77 INCAP NUTRITIONAL INTERVENTION AND FOLLOW-UP STUDIES

3.1 Background

In the mid-1960s, protein deficiency was seen as the most important nutritional problem facing the poor in low-income countries, and there was concern that this deficiency affected children's ability to learn. The Institute of Nutrition of Central America and Panama (INCAP), based in Guatemala, initiated a series of studies on this subject, leading to a nutritional supplementation trial that began in 1969 (Habicht and Martorell 1992; Read and Habicht 1992; Martorell Habicht, and Rivera 1995). The main hypothesis was that improved preschool nutrition accelerates cognitive development. An examination of the effects on physical growth was included to verify that the nutritional intervention had biological potency (Martorell, Habicht, and Rivera 1995). Initially, 300 rural communities in eastern Guatemala were screened to identify villages with appropriate compactness to facilitate access to feeding centers (see below) and similarities in terms of ethnicity, diet, access to health care facilities, demographic characteristics, child nutritional status, and degree of physical isolation.

Using these criteria, two sets of village pairs (one pair of "small" villages with about 500 residents each and another pair of "large" villages with about 900 residents each) were selected. The village pairs were similar in most social and economic attributes though slightly less so in terms of schooling. The distributions of child nutritional status before the intervention, as measured by height at three years of age, did not differ significantly across villages (Habicht et al. 1995). Two of the villages, one from within each pair matched on population size, were randomly assigned to receive *atole*, a high protein-energy drink with multiple micronutrients added as a dietary supplement. In light of concern that the social stimulation for children gathering at the feeding centers also might affect child nutritional and cognitive outcomes, thus confounding efforts to isolate the nutritional effect of the *atole* supplement, an alternative supplement, *fresco*, was provided, under identical conditions in the other two villages. *Fresco* contained no protein and had about one-third of the calories of *atole* per unit volume but similar amounts of micronutrients (Habicht and Martorell 1992). The nutritional supplements were distributed in each village in centrally located feeding centers, on demand, and were available twice daily to all members of the village on a voluntary basis for

two-to-three hours in the mid-morning and two-to-three hours in the mid-afternoon. Residents were offered preventative and curative medical care free of charge throughout the intervention, including access to community health workers and trained midwives, immunization services, and deworming campaigns. To ensure that the results were not systematically influenced by the characteristics of the survey teams, all personnel were rotated periodically throughout the four villages, each of which was separated by at least 10 kilometers.

INCAP implemented the nutritional supplementation and provided medical care from 1969 to 1977. While the supplement was freely available to *all* village residents, the associated observational data collection focused on pregnant and lactating women and children between zero and seven years of age at any point during the intervention period.⁴ Thus all children under seven years of age residing in the villages at the start of the intervention, as well as those born in the villages during the intervention, were included in the survey, a total of 2,392 children. Data collected at the child level included periodic anthropometric measurements until the child reached seven years of age or until the survey data collection ended in 1977, whichever came first. Thus, the individuals in the sample were born between 1962 and 1977 with the type, timing, and length of exposure to the nutritional supplementation depending on their village and date of birth.⁵ These data were complemented by censuses in the four villages and an ethnographic study carried out prior to the intervention (Pivaral 1972).

In 2002–04, a team of investigators, including the authors of this paper, undertook a follow-up survey targeting all child participants in the 1969–77 study who ranged in age from 25 to 42 years. Of 2,392 individuals in the original sample, 1,855 (78 percent) were determined to be alive and known to be living in Guatemala: 11 percent had died—the majority from infectious diseases in early childhood, 7 percent had migrated abroad, and 4 percent were not

⁴ The intervention began in the larger villages in February 1969 and in the smaller villages in May 1969. The nutritional supplements and medical care ended in all four villages at the same time, in February 1977, and the survey data collection ended seven months later (Martorell et al., 1995).

⁵ This population has been studied extensively since the original survey, with particular emphasis on the impact of the nutritional intervention (see Stein et al. 2008). Martorell et al. (2005) gives references to many of these studies; more recent examples include Behrman et al. (2009, 2010); Hoddinott et al. (2008); Maluccio et al. (2009) and Stein et al. (2003). For part of the period covered by these surveys (particularly the 1980s and early 1990s), much of western and northern Guatemala was embroiled in civil war, though these survey villages were not directly affected.

traceable. Of the 1,855, 60 percent lived in the original villages, 8 percent lived in nearby villages, 23 percent lived in or near Guatemala City, and 9 percent lived elsewhere in Guatemala. For the 1,855 traceable sample members living in Guatemala, 1,571 (85 percent) completed at least one interview during the 2002–04 survey (Grajeda et al. 2005). Over a series of interviews, respondents reported information on schooling, marital and fertility history, income, and consumption. Participants received physical examinations and completed tests of reading and vocabulary skills, nonverbal cognitive ability, and physical fitness. A fasting capillary blood sample was obtained to determine glucose, cholesterol, triglycerides, and lipoprotein concentrations.

3.2. Descriptives: Early-Life Nutritional Status

During the supplementation trial between 1969 and 1977, children’s height was measured at age 15 days and 3, 6, 9, 12, 15, 18, 21, 24, 30, 36, 42, 48, 54, 60, 72, and 84 months, with a small range around each targeted age. The higher frequency measurements at earlier ages were designed to capture the more rapid growth that occurs during that period of life. The total number of measurements was largest for children born in 1969 and 1970 (when the supplementation trial began) and smallest for children born in 1962 and 1963 (who were therefore closer to the upper age limit at which children were measured) and for children born in 1976 and 1977, just before the intervention ended.

Using these data, we calculated HAZ scores using World Health Organization (WHO) reference standards (WHO 2006). As shown in Figure 1, average HAZ scores drop substantially in the first 15 months of life. This decline slows before leveling off and reaching a minimum at about 24 months of age. After this, the average HAZ increases slightly, approaching -2.3 at age 72 months. Correlations between HAZ scores at very early ages (for example, 1 and 6 months) and HAZ scores at 36 months and older are *relatively* low (Table 1). Correlations at older ages are fairly high; for example, the correlation of HAZ at 36 months with HAZ at 1 month is 0.39 and with HAZ at 42 months is 0.95.

Given the nonlinear trajectory of growth in early childhood, the schedule of measurements, and the pattern of correlations of HAZ measured at different ages, an issue that

arises is the choice of a suitable age at which we take the measure of HAZ as the representation of early-life growth failure. To inform our choice, we take into account both the number of observations on HAZ available at different ages as well as their correlation. As shown in Table 1, the number of observations is somewhat smaller at ages 60 and 72 months. While we have a larger number of measurements of HAZ for children less than 24 months, these are less highly correlated with HAZ at older ages. These two considerations suggest that selecting a measure between 24 and 36 months would be appropriate; further, it is within this age range that “peak” growth retardation occurred. Supplementation with *atole*, in comparison to *fresco*, increased the heights of three-year-old children by about 2.5 centimeters. It produced its biggest effects by 24 months and after 36 months did not influence child growth rates (Schroeder et al. 1995). Further, pair-wise rank correlations (not shown) after 36 months exceed 0.90, indicating that ranking on height stabilizes from that age onward. These observations suggest that using HAZ at age 36 months would be an appropriate representation of early-life growth failure. However, using data only on individuals who were measured at 36 months would be informationally inefficient, especially given that HAZ scores at certain ages are highly, though not perfectly, correlated with each other.

Instead, we estimate HAZ scores at 36 months for those individuals for whom data are missing. We start by estimating a child-level fixed effects regression where the dependent variable is HAZ using all the available HAZ information. In addition to the child fixed effects, we include dummy variables for the age categories at which the child was measured, excluding age 36 months as the reference category. The constant term from this regression is the mean estimate for 36 months. The age category dummy variables shift this mean up and down depending on the age at which the child is measured relative to the reference category, 36 months. We then generate a synthetic measure of HAZ at 36 months for all children, using the actual measurement for a child if available (880 observations). Where HAZ at 36 months is unknown, we take the closest age at which height was measured, and using the regression results above, we calculate a predicted value for HAZ at 36 months by adjusting the actual HAZ for the child at the age closest to 36 months by the age coefficient for that age in the regression

estimates. In Section 5, we assess the robustness of our results to alternative ways of selecting our representation of early-life growth failure.

3.3 Descriptives: Outcome Variables

The definitions, means, and standard deviations for our outcome variables for the full sample and disaggregated by sex are found in Table 2.

In Guatemala, the official age for starting primary school is seven years.⁶ There are six grades of primary school. Secondary school consists of five to seven grades, divided into two parts. The first three grades of lower secondary school are “basic” grades while the fourth through seventh grades are the “diversified” grades, in which students can choose from a set of academic or vocational tracks. Students planning to continue to university finish upper secondary schooling in two years; other tracks usually take three years (World Bank 2003). It also is possible to complete (primary and secondary school) grades via informal schooling, such as adult literacy programs, though few individuals in our sample did so.

We consider the following schooling-related outcomes: age at which the individual started school, whether they repeated a grade of primary school, the speed at which they progressed (number of grades passed divided by the number of years between entering and terminating school), the age at which the individual stopped attending school, and the highest grade attained. The mean age at which respondents started school was 6.8 years.⁷ Men complete slightly more grades than women (5.2 grades versus 4.5 grades) and, on average, men leave school when they are about one year older (13.0 years versus 12.1 years). That the age gap at leaving school is larger than the difference in grades attained reflects, in part, the slightly higher rate of grade repetition by men, a pattern widely observed in poorer countries (Grant and Behrman 2010).

Participants in the 2002–04 survey who passed a literacy screen pre-test or who had completed more than six grades of schooling were given the *Serie Interamericana* (SIA) vocabulary (Level 3) and reading comprehension (Level 2) test modules. The maximum possible

⁶ In our sample, many of these individuals started school in the year in which they turned seven, particularly if they were born in the latter part of the calendar year.

⁷ Nearly all individuals (86 percent) completed at least one grade of school.

score was 85 points. Those who did not pass the literacy screen (18 percent of the sample) were assigned a score of zero. All participants took Raven's Progressive Matrices test, an assessment of nonverbal cognitive ability (Raven, Court, and Raven 1984). Raven's tests are considered to be a measure of "the ability to make sense and meaning out of complex or confusing data; the ability to perceive new patterns and relationships" (Harcourt Assessment 2008). We administered the first three of five scales (A, B, and C with 12 questions each, for a maximum possible score of 36). Reading/vocabulary comprehension test (SIA) scores and Raven's test scores are expressed as z-scores standardized to have mean 0 and standard deviation 1 within the sample.

A marriage history module was administered to each individual in the sample. Here, "marriage" refers to two individuals joining in union, usually (but not always) cohabitating and not restricted to church or state-sanctioned marriages. The module consisted of questions on (1) age at first marriage, duration of first marriage, age at subsequent marriages and at marital dissolutions; (2) information on physical and financial assets brought to marriage; (3) and spouse's family background. As Table 2 shows, men tend to be older than their wives, to have more schooling, and taller. It is rare for both men and women to be living independently of their parents at the time of union formation. About one man in four brought household goods or productive assets to the union, while fewer than 10 percent of women did. Quisumbing et al. (2005) provide further background and details.

Women were asked about their fertility history, number of live births, the number of children who died, pregnancy intentions, and knowledge and use of a range of contraceptive methods. Women also were asked about their menstrual history and provided a detailed pregnancy history. Using these data, we were able to construct the following outcomes: age at menarche, whether a woman gave birth before age 18, total number of pregnancies, whether the woman had experienced a still birth or miscarriage, whether a child had died in infancy, and the number of surviving children. Just under 25 percent had given birth before age 18, 22 percent had experienced at least one still birth or miscarriage, and 15 percent had a child die in infancy. Ramakrishnan et al. (2005) provide detailed descriptive statistics on these and other fertility-related outcomes.

The field team included two physicians who collected biomedical data: measurements of body size and composition, blood pressure, tests of physical fitness, clinical histories, and a finger-stick whole-blood sample from which it was possible to measure plasma glucose and a lipid profile. Using these data, we consider the following adult anthropometric outcomes: log height, log body mass index (BMI), whether the individual is obese (defined as having a BMI >30.0), and head circumference. We also consider three measures of physical fitness: log of predicted fat free mass, isometric hand strength, and predicted maximal oxygen consumption (VO₂max). Fat-free mass is a good overall measure of physical capacity for work (McArdle, Katch, and Katch 1991), hand strength is correlated with total strength of 22 other muscles of the body (de Vries 1980), and VO₂max is the single best measure of cardiovascular fitness and maximal aerobic power (Powers and Howley 2000). We also examine outcomes related to risks associated with cardiovascular and other chronic diseases: low-density lipoprotein (LDL) cholesterol, the ratio of total cholesterol to high density lipoprotein (HDL) cholesterol, whether the individual is hypertensive or prehypertensive, plasma glucose, and whether the individual is diabetic or prediabetic. Lastly, we consider metabolic syndrome (METS), a range of risk factors associated with increased risk of stroke, diabetes, and coronary heart disease. We logarithmically transform a number of these outcomes because their distributions are closer to log normal than normal. Ramirez-Zea et al. (2005) provide a detailed description of how these data were collected and the distribution of these outcomes by individual and locality characteristics. The incidence of obesity is 25 percent for women and 9 percent for men; 20 percent of women and 43 percent of men are prehypertensive or hypertensive and the incidence of METS is 40 percent for women and 18 percent for men.

Individuals were interviewed about all of their income-generating activities, including wage labor (type of occupation, wages, deductions, fringe benefits, and bonuses); agricultural activities (amount of land cultivated, crops grown, production levels and values, and use of inputs); and nonagricultural own-business activities (type of business, value of goods or services provided, and capital stock employed). For each activity, individuals were asked the number of months in which they worked and how many days per month and hours per day they typically worked. Hoddinott, Behrman, and Martorell (2005) provide a detailed discussion of these data.

Virtually all men (98 percent) and most women (69 percent) were undertaking an income-generating activity with many undertaking more than one. In the year prior to the interview, 79 percent of men were working for wages (with more than half of these in unskilled occupations)—42 percent in own-account agriculture and 28 percent in own-account nonagricultural business. A third of women were working for wages (with the majority in unskilled occupations) a third were in own-account nonagricultural business, and a fifth were in own-account agriculture. In this sample, the wage rate for men is approximately 45 percent higher than that of women.

An expenditure module provided information on food and nonfood expenditures in the household in which the respondent was residing at the time of interview, and a community-level module provided food prices. Using these data and the method outlined in Maluccio, Martorell, and Ramírez (2005), we calculate log per capita household food consumption and log per capita total consumption. Comparing these data against a poverty line for Guatemala, we find that 35 percent of the sample lives in households with consumption levels below the poverty line. See Maluccio, Martorell and Ramírez (2005) for details.

4. RESULTS

4.1 Identification of the First Stage

The parameters of primary interest are the vector β in equation (2). If $E(\mathbf{HAZ}_{i,t} \mathbf{v}_{ijt+n}) \neq 0$ where \mathbf{v}_{ijt+n} is the element in \mathbf{U}_{it+n} for the j th element in \mathbf{Y}_i , the estimation of the j th element in β will be biased. It is not difficult to think of reasons why such a correlation could exist. For example, parents who have (unobserved) superior social networks may be wealthier, invest more in the nutrition of their children, and be better placed to help their children find high-paying jobs. If social networks are not observed in the data, then they are in \mathbf{u}_{ijt+n} , $\mathbf{HAZ}_{i,t}$ will proxy in part for the correlated unobserved social networks, and the estimate of the j th element of β will represent in part the impact of social networks, not just the impact of $\mathbf{HAZ}_{i,t}$. The critical issue, therefore, is whether we can identify the impact of $\mathbf{HAZ}_{i,t}$ by using a representation of $\mathbf{HAZ}_{i,t}$ that is uncorrelated with \mathbf{v}_{ijt+n} . This need in turn requires that we identify sources of exogenous variation that appear in (1) but do not appear in (2).

It is widely accepted that, under certain conditions, randomized control trials represent a powerful means of identifying impact. While factors that affect growth in early life have been the subject of many randomized control trials, nutritional status itself cannot be randomized for both ethical and practical reasons. Consequently, in this paper we identify impact using instrumental variables estimators. This approach has strengths but also limitations, most notably that “the estimated treatment effect is applicable to the subpopulation whose treatment was affected by the instrument” (Lee and Lemieux 2010, 292). Given Deaton’s (2010) critique of local average treatment effects (LATE), we carefully specify and justify our choice of instruments by nesting them within the model outlined in Section 2 and perform sensitivity analyses in Section 5.

Our identification strategy relies on two types of variables. The first are cohort and location-specific transitory exogenous events or shocks, assumed independent of individual unobserved characteristics, that affect $\mathbf{HAZ}_{i,t}$ in (1) but do not directly affect $\mathbf{Y}_{i,t+n}$ in (2) except through their effects on $\mathbf{HAZ}_{i,t}$.⁸ These include a subset of the elements of \mathbf{Qvary}_{it} . The second type of variable captures random variation in genotype that are found in the vector $\mathbf{C}_{i,t}$ that are

⁸ This approach is analogous to those described by Imbens and Angrist (1994); Card (2001); and Alderman, Hoddinott, and Kinsey (2006).

also assumed to affect $HAZ_{i,t}$ in (1) but do not directly affect $Y_{i,t+n}$ in (2), after controlling for other characteristics in the second stage. Cohort and location-specific transitory shocks include exposure to the INCAP intervention from the ages of 0 to 36 months; exposure to the intervention between 0 and 36 months interacted with residing in a village where *atole* was provided; whether the subject was born in 1974, 1975, or 1976 and therefore exposed in early life to the effects of a severe earthquake that shook Guatemala in February 1976;^{9, 10} and whether there was a government health post in the individual’s village of residence when they were two years of age. Measures of variation in genotype include the logarithm of maternal height (Sahn 1990) and whether the individual was a twin. While we might expect maternal height to capture aspects of parental background beyond the exogenous variation in genotype and physical attributes of the mother that are causally related to child growth, inclusion of the other parental background characteristics (education and wealth) in the second stage mitigates the possibility that the maternal height instrument will pick up these other influences.

Table 3 presents the results of estimating equation (1). In addition to the instrumental variables described above, we represent C by the individual’s sex; M_t by completed grades of the mother’s schooling; W_t by a wealth index and completed grades of the father’s schooling; and P_t by a set of year-of-birth dummy variables that capture more generally all events (including movements in prices) common to a given birth cohort. Q_{inv} is denoted by a set of location-of-birth dummy variables.

A number of our instruments are related to $HAZ_{i,t}$. Exposure to *atole* between 0 and 36 months, being exposed to the 1976 earthquake, and maternal height all increase height-for-age at 36 months, while being a twin reduces height. The coefficient estimates for these variables are all statistically significant and have the expected signs. Exposure to the intervention between birth and 36 months and having a health center in the village of birth, however, are not associated with height at 36 months. An F test of 16.5 rejects the null hypothesis that these proposed instruments are jointly zero. Stock (2010, 87) notes that, “If this F-statistic is large – a

⁹ On February 4th, 1976, an earthquake measuring 7.5 on the Richter scale, struck Guatemala killing approximately 23,000 people and leading to serious damage to housing and infrastructure in a number of the survey villages.

¹⁰ With the inclusion of this earthquake dummy spanning three years, we include year-of-birth dummy variables for 1962 through 1975, excluding 1976 and 1977.

common rule of thumb is $F > 10$ – then one can treat the instruments as sufficiently strong that the usual two-stage least squares output can be used.”

In addition to being correlated with height-for-age, an attractive feature of these variables is that they meet a key criterion specified in Deaton’s (2010) critique of IV methods; namely that they are derived from models of the determinants of the endogenous variable. However, one can construct arguments why any of these instruments could fail the uncorrelatedness assumption, *viz*:

- exposure to the intervention could have had an income effect that operated beyond the effect of the intervention on child nutritional status;¹¹
- the earthquake could have had long-lasting effects – for example on school availability and quality or on income-generating opportunities;¹²
- the establishment of a government-run health post could reflect a process of endogenous program placement;¹³
- maternal height may reflect investments made by the mother’s own parents, and dimensions (such as quality of child care in early life) might be correlated

¹¹ We think this possibility is unlikely. First, the behavior of villagers did not suggest that the supplements were of significant monetary value. Despite the fact that supplements were freely available every day to all inhabitants of the communities, few men or school-age children frequented the feeding centers, even on weekends when the opportunity cost of their time in terms of work or school presumably was lower. Second, the actual monetary value of the supplements was low. We estimate the cost of the ingredients for one cup of *atole* and one cup of *fresco* to have been US\$0.018 and 0.004, respectively. Mean household incomes were approximately US\$400 in 1975 (Bergeron 1992). Thus, one year’s worth of a daily cup of *atole* (US\$6.60) and of *fresco* (US\$1.50) was approximately 1.7 and 0.4 percent of average annual household income, and on average children 0–36 months of age consumed less than this. The medical care may have had a greater income effect for households, but this effect was equally present in *atole* and *fresco* villages.

¹² This proposition is unlikely to be a significant concern for two reasons. First, schools were rebuilt quite quickly after the earthquake. Second, as Bergeron (1992) and Estudio 1360 (2002) show, the livelihood and income trajectories of these villages were shaped and reshaped by many subsequent events both positive—such as the opening of new wage jobs in nearby towns—and negative—such as the collapse in markets for goods produced in particular villages at particular times.

¹³ Because we include village-of-birth dummy variables in all specifications, this criticism relates solely to time varying factors that might have led to differences in the timing of the establishment of these health posts while also directly affecting second-stage outcomes. While such factors cannot be ruled out, none of the three ethnographic studies conducted in these villages (Pivaral 1972; Bergeron 1992; and Estudio 1360 2002) indicate that health posts were established as a result of a location-specific, time-varying event.

intergenerationally. In addition, the genetic component of maternal height may be directly related to some of our outcomes apart from their effect on HAZ;¹⁴ and

- the proportion of the sample that is a twin is so small (less than 1 percent) that the LATE of this identifying instrument is not likely to be of much interest.

In light of these potentially legitimate concerns, we subject our instruments to a battery of tests, described below. Further, we return to these issues in our discussion of robustness tests in Section 5.

4.2 Impacts of Growth Failure into Adulthood: Overview

We now turn to the results of estimating equation (2) for outcomes that span the life course of these individuals up to middle adulthood. For each set of outcomes, we report the results of two functional form representations of preschool nutritional status: height-for-age z-scores; and a dummy variable equaling one if the individual was stunted at age 36 months, zero otherwise. When we use the z-score, we have a positive parameter estimate when an improvement in nutritional status leads to an improvement in that outcome. When we use stunting, a negative parameter estimate means that an improvement in nutritional status (such as switching an individual from being stunted to not being stunted) leads to an improvement in outcomes. We report ordinary least squares (OLS) and IV results for the full sample, then the IV height-for-age results separately for women and men.¹⁵

We apply a common set of control variables in all estimates. In addition to variables that are determinants of both height and these outcomes (an individual's sex, birth year, and place of birth; maternal and paternal schooling; household wealth as measured by the principal components of assets held in 1967), we include distance to primary school, school quality (permanent structure and student–teacher ratio) at age 7, whether a biological parent died

¹⁴ This possibility is mitigated substantially by including controls for parental grade attainment and initial household wealth in both the first- and second-stage models.

¹⁵ OLS results and results using stunting by sex are available on request.

before subject was 15 years old (15y), and access to bus service at age 15.¹⁶ By doing so, our estimates control for cohort effects, unobserved fixed effects associated with place of birth, and parental characteristics and time-varying locational characteristics that might cause outcomes across the lifecycle to be correlated with growth failure in early childhood. The full set of results is available on request.

For all outcomes, we compare results of the Kleibergen-Paap (KP) test statistic (Kleibergen and Paap 2006; Kleibergen 2007) to the critical values presented by Stock and Yogo (2005, Table 5.1) to assess whether our instruments are weak. Critical values for the KP test statistic at the 5 percent significance level are 11.29, 6.73, and 5.07 for rejecting null hypothesis of weak instruments, where weak means having bias in the IV results larger than 10, 20, and 30 percent of the bias in the OLS results, respectively. In all cases where the endogenous variable is expressed as the height-for-age z-score, we reject this null for 20 percent bias and in many instances for 10 percent bias (at a 5 percent significance level). Apart from several outcomes listed in Table 6, where we obtain test statistics of 6.2 or higher, this is also true for estimates where we represent early-life growth failure in terms of being stunted. We also report the Hansen J statistic for overidentification, where the null hypothesis is that the overidentifying restrictions are valid (that is, the model is well specified and the instruments do not belong in the second-stage equation). Failure to reject the null hypothesis for the Hansen test is evidence that if any one of the instruments is valid, so are the others. Since the instrument set includes the randomly allocated exposure to the intervention and the earthquake indicator, both of which are likely to be valid, these inclusions give us some confidence in the power of this specification test. In nearly all cases (147 out of the 156 test statistics we report), we fail to reject this null. Standard errors are robust to heteroscedasticity and clustered at the maternal level. Where the outcomes are binary, we estimate linear probability models so as to be able to compute the weak instrument and overidentification test statistics.¹⁷ We also test whether differences in the impact of early childhood growth failure differ by sex.¹⁸

¹⁶ Also included are dummy variables for the small number of cases where maternal schooling, height, or initial wealth was missing.

¹⁷ We obtain similar patterns of statistical significance if we estimate these 0/1 outcomes using an IV probit.

¹⁸ We interact all of the instrumental variables by a male dummy and include these interaction terms, along with all other instruments and control variables, in estimates where we include height-for-age z-scores and height-for-

4.3 Impacts of Growth Failure into Adulthood: Results

Table 4 reports the results of estimating the impact of nutritional status at 36 months on schooling outcomes. Table 4a reports these for the full sample, and Table 4b reports them separately for women and men. These results show that there is a direct effect of early-life growth failure on age at school entry, the age at school exit, and the number of grades completed. When nutritional status is expressed in terms of stunting, the effect on grade attainment is large—a loss of 3.6 grades of schooling compared with someone who is not stunted. There are no statistically significant differences by sex. Results for the reading/vocabulary and Raven’s tests show that growth failure in early childhood is causally related to poorer cognitive skills in adulthood. The magnitudes of these effects are large. An individual who is stunted at 36 months scores more than a full standard deviation lower on the SIA reading/vocabulary test and 0.88 standard deviations lower on the Raven’s tests.

Table 5a reports the impact of growth failure on success in the marriage market.¹⁹ Individuals who were taller at age 36 months have spouses who are taller and who have completed more grades of schooling. The magnitudes of these effects are particularly large when growth failure is represented by stunting. Individuals not stunted at 36 months are, at the time of this survey, married to someone who has nearly four more grades of schooling. The disaggregated results suggest some differences by sex (Table 5b). Impacts on the woman’s partner appear larger for his age, economic independence at marriage, possession of household goods at the time of union formation, and the age differential. By contrast, the impact on a man’s partner is larger for her grades of schooling and height. However, none of these gender-differentiated impacts are statistically significant.

The consequences of these marriage-market outcomes for women’s welfare are ambiguous. Since age and consumption levels are correlated in this sample, these results

age z-scores interacted with male as endogenous variables and test whether the endogenously determined interaction term of male and HAZ (or stunting) is statistically significant.

¹⁹ We did not find statistically significant impacts of early-life nutrition on the timing of entry into the marriage market as measured by age at first marriage, whether an individual married before 16, whether an individual married before 18, and duration of time between leaving school and forming first union. These results are available on request.

suggest that women who experience less growth failure make better matches in the marriage market. However, not only do women with better childhood nutrition marry older men, but they also marry men who are older than themselves (the estimated coefficient on the difference in ages between women and their spouses is positive). If bargaining power within the household is correlated with age differentials between spouses, while women with better early-life nutrition may marry into better-off households, they may also be somewhat more disadvantaged in terms of their ability to bargain over resources within those households.²⁰

Results for fertility outcomes are reported in Table 6. Women who did not experience growth failure in early life have fewer pregnancies. They have fewer surviving children, but this effect is smaller than the impact on pregnancies because they have a lower incidence of stillbirths or miscarriages and a lower prevalence of deaths in infancy, though the latter is not statistically significant. These effects are large. “Switching” a woman from being stunted to not being stunted would, conditional on her age, reduce the number of pregnancies she has by 1.86 and the likelihood that she experiences a stillbirth or miscarriage by 36.9 percentage points. These results are consistent with our finding that growth failure in early life causes women to complete fewer grades of school and the extensive literature showing that women with less schooling have more pregnancies.

Results on anthropometry, physical fitness, and outcomes associated with the risk of cardiovascular and other chronic diseases are reported in Tables 7a and 7b. Note that for one outcome, log height, we reject the null that the overidentifying conditions are valid. This is not surprising given that one of our instruments, maternal height, is likely to be directly correlated with height in adulthood beyond its direct effect on height-for-age in early life. This is reassuring in that it tells us that the Hansen test has power in that it detects and rejects the null regarding the overidentifying conditions for the outcome where these are most likely to be violated.

Individuals with better nutritional status at 36 months have greater hand strength and fat-free mass. However, apart from the impact of height-for-age z-score (but not stunting) on

²⁰ Other studies on marriage markets in urban Guatemala have found that, while age and schooling differentials between spouses have been narrowing, the asset differential has been rising, with husbands tending to bring more assets to the marriage than wives over time (Quisumbing and Hallman 2005).

the likelihood of being hypertensive or prehypertensive, there is little evidence that nutritional status at 36 months increases outcomes associated with the risk of chronic disease. We do not find evidence that the impacts on anthropometry and physical fitness differ by sex. Relative to men, women appear to be somewhat more likely to be hypertensive or prehypertensive if they were taller at 36 months, and men, relative to women, were more likely to be diabetic or pre-diabetic. However, differences by sex are not statistically significant.

A one-standard-deviation increase in height-for-age at 36 months increases hourly earnings in adulthood by 14.8 percent, an impact significant at the 10 percent level (Tables 8a and 8b). This effect appears larger and is more precisely measured for men (20.1 percent) than for women (7.2 percent), though we cannot reject the null hypothesis that these coefficients are equal. There is no statistically significant impact on hours worked. Individuals who did not experience growth failure in early childhood are much more likely—28 percentage points—to work in higher-paying skilled labor or white-collar work.²¹ Individuals, particularly women, who were taller at age 36 months are more likely to operate their own businesses as adults.

Tables 9a and 9b show that men and women with better nutritional status in early life live in households with higher consumption levels as adults. A one standard deviation increase in HAZ increases household per capita food consumption by 14.6 percent and total per capita consumption by 19.5 percent. An individual who was not stunted at age 36 months is 33 percentage points less likely to reside in a poor household as an adult, though we caution that the probability value of the Hansen J test is relatively low for this outcome. Since consumption is measured at the household level, all members, not just the individual who was better nourished, are better off, assuming that there are no large intrahousehold inequalities in consumption induced by better nutrition of the respondent at age 36 months. This suggests that the benefits to improving an individual's early-life nutritional status are not necessarily confined to that individual; they may spill over to other household members.

²¹ Given that many of these jobs are found in Guatemala City, we wondered if growth failure affected migration status in adulthood but found no significant effects (estimates not reported).

5. CHECKS ON ROBUSTNESS²²

5.1 Instrument Validity

In the results presented above, we used an identical set of instruments for all outcomes. While we have derived our instruments from a model of the determinants of height (equation 1) and statistical tests indicate the instruments are relevant, there are two further issues requiring consideration. First, as shown by Imbens and Angrist (1994) and reiterated by Lee and Lemieux (2010) and Deaton (2010), “the estimated treatment effect is [only] applicable to the subpopulation whose treatment was affected by the instrument” (Lee and Lemieux 2010, 292). Second, Leamer describes credible inferences in terms of the outcome of sensitivity analyses that “separate fragile inferences from sturdy ones” (Leamer 2010, 37).

Because we have several variables that we use to identify the impact of preschool nutritional status, we can assess the sensitivity of our results to the inclusion or exclusion of particular instruments. To illustrate, we consider six outcomes (reading/vocabulary scores, spouse’s grades of schooling, number of pregnancies, log of fat-free mass, log of hourly earnings (males), and log of household per capita consumption), each one taken from a different dimension of the life course outcomes (schooling, marriage markets, fertility, health, labor market, consumption) analyzed in this paper. For each outcome, we consider eight alternative instrument sets in addition to the full set of instruments used to generate the results reported in Tables 4 through 9. These are summarized in Table 10, which shows coefficient estimates for $HAZ_{i,t}$, the Kleibergen-Paap test statistic that the instruments are weak, and the p-values for the Hansen J test. (Full results are available on request.)

Alternatives 1, 2, and 3 involve dropping different combinations of the twins instrument, the negative earthquake shock, and the positive health infrastructure shock. For all six outcomes, these generate parameter estimates and confidence intervals that are virtually identical to the full set of instruments. In alternative 4, we drop exposure to the intervention between 0 and 36 months, and exposure to the intervention between 0 and 36 months and

²² In earlier papers, Hoddinott et al. (2008) and Maluccio et al. (2009), we reported the robustness checks for alternative calculations of the standard errors, such as clustering at the birth year-village level. We estimated these alternative sets of standard errors for the results presented in this paper and found that they produced smaller standard errors. To save space, we do not report them here; they are available on request.

living in an *atole* village. In alternative 5, we drop both exposure variables and also drop whether the individual is a twin. Again we obtain similar parameter estimates to those obtained with the full set of instruments for all outcomes. In these five alternative specifications, our instrument set easily meets the relevance criteria as measured by the Kleibergen-Paap statistic, and we do not reject the null hypothesis that the overidentifying restrictions are valid.

The common feature across alternatives 6, 7, and 8 is that they exclude the log of mother's height as an instrument. In alternative 6, we only drop log of mother's height as an instrument. In alternative 7, we retain only exposure to the intervention between 0 and 36 months, and exposure to the intervention between 0 and 36 months and living in an *atole* village. Under alternative 8, we consider results based on using the exposure to the intervention variables, being a twin, and being exposed to the earthquake shock as instruments but exclude log of mothers' height and access to a health post at age 24 months. We find that we cannot identify the impact of early-life growth failure solely through use of the exposure to the intervention variables as instruments; for all outcomes, the estimated models under alternative 7 have very low Kleibergen-Papp statistics and the parameter estimates have wide confidence intervals. We find that in alternatives 6 and 8, we generally obtain less precise parameter estimates as evidenced by larger standard errors. However, looking across these nine specifications for the instrument sets (the baseline and eight alternatives) for each outcome, provided we expand our instrument set beyond exposure to the intervention and the *atole* interaction term (that is alternative 7), we have instrument sets that pass the relevance and overidentification tests while producing comparable parameter estimates. Even if we drop log of mother's height as an instrument (alternatives 6 and 8), we obtain parameter estimates that are either approximately equal (reading scores, number of pregnancies, log fat-free mass) or *larger* (spouses' grade of schooling, hourly earnings for men, and log of per capita consumption) than those obtained using the full set of instruments.²³ We interpret this as particularly strong evidence since, if mother's height were an invalid instrument, it would seem most plausible that its inclusion would have biased upward the estimated impacts of HAZ on the outcomes considered because intergenerational ability bias would suggest positive

²³ When we replicate this with other outcomes that are statistically significant in Tables 4 through 9, we observe a similar pattern, though in some cases the confidence intervals become especially large.

associations between mother's height and the second-stage outcomes we measure. Based on these findings, we conclude that the estimates reported in Tables 4 to 9 are robust to concerns regarding fragile inferences and concerns regarding the generality of the LATE estimates.

5.2 Attrition

Another potential concern for our inferences is the problem of attrition across follow-ups of this cohort. Despite the considerable effort and success in tracing and re-interviewing participants from the original sample, more than one-third of the sample was not traced and re-interviewed.²⁴ Moreover, as shown in Grajeda et al. (2005), attrition in the sample is associated with a number of initial conditions, in ways that differ by the reason for attrition (for example, migration versus failure to interview someone who was located). Furthermore, there is selective nonreporting in different parts of the study, such as the collection of blood samples where attrition was higher and labor market outcomes where labor market participation was lower for women. What is of ultimate concern in this analysis is not the level of attrition, however, but whether, attrition invalidates the inferences we make using these data.

We address concerns about sample attrition bias in three ways. First, we compare nutritional outcomes measured in the 1970s for those who did and did not attrite between the end of the intervention in 1977 and the resurvey in 2002–04. Average height-for-age measured at 36 months is virtually identical between those who attrited and those who did not. Height-for-age z-scores for the two groups are within 0.01 of one another, and the p-value on a t-test of their equality has a value of 0.799. There does not appear to be any obvious selection between those interviewed or not, based on early-life nutritional status. Second, in the

²⁴ A related problem is that of mortality selection (Pitt 1997; Pitt and Rosenzweig 1989). Indirect evidence that mortality selection exists in the sample is that higher risk of death is associated with younger ages (those born later) in the original sample of 2,392. The older sample members represent the survivors of their respective birth cohorts, and hence they experienced a lower mortality rate (because most mortality was in infancy), compared with the later birth cohorts in the study who were followed from birth. Because data collection began in 1969 and included all children less than seven years of age, it excluded all children from the villages born between 1962 and 1969 who died before the start of the survey. Another facet of mortality selection, however, has to do with the intervention itself, which may have decreased mortality rates among the younger cohort in *atole* versus *fresco* villages (Rose et al. 1992). To the extent the variables included in our models are associated with these forms of selection, our estimates partly control for mortality selection, though we do not implement any special methodology to do so. To the extent that unobservable characteristics that affect the likelihood of mortality are correlated with HAZ, our identification strategy guards against biases that such a correlation might create.

specifications already shown, we include observed covariates that, in addition to playing a role in affecting outcomes, are themselves associated with attrition, including being male (+), birth year (-), and parental wealth (+). Conditional on the maintained assumptions about the correct functional form, attrition selection on right-side variables does not lead to attrition bias (Fitzgerald, Gottschalk, and Moffitt 1998b).

Lastly, we implement the correction procedure for attrition outlined in Fitzgerald, Gottschalk, and Moffitt (1998a, 1998b). We estimate an attrition probit conditioning on all the exogenous variables considered in the main models, as well as an additional set of endogenous variables potentially associated with attrition. We include a number of variables that reflect family structure in previous years, since these are likely to be associated with migration status. They include indicators of whether the parents were alive when each sample member was seven years old and whether the sample members lived with both their parents in 1975 and in 1987. During the fieldwork, locating sample members was typically facilitated by having access to other family members from whom the field team could gather information. Therefore, we also include a number of variables that capture this feature of the success of data collection. They include whether the parents were alive in 2002, whether they lived in the original village, whether a sibling of the sample member had been interviewed in the 2002–04 follow-up survey, and the logarithm of the number of siblings in the sample in each family. We emphasize that this is *not* a selection correction approach in which we must justify that these factors can be excluded from the main equations, but rather we purposively exclude them from those regressions since our purpose is to explore the determinants of the vector of outcomes found in equation (2) and not whether these are associated with the family structure and interview-related factors included in the “first-stage” attrition regression (Fitzgerald, Gottschalk, and Moffitt 1998a). While we do not formally have adjustments to correct for selection on unobservable characteristics, by including the large number of endogenous observables indicated above, which are likely to be correlated with unobservables, we expect that we are reducing the scope for attrition bias due to unobservables as well.

The factors described above are highly significant in predicting attrition, above and beyond the conditioning variables already included in the models (results available on request.)

They lead to weights between 0.27 and 2.34 for those individuals found in the 2002–04 sample. We estimate attrition-weighted IV regressions for the six outcomes used to assess robustness to instrument selection: SIA z-scores, spouse’s grades of schooling, number of pregnancies, log of fat-free mass, log of hourly earnings (for men only), and per capita household consumption. Table 11 shows that application of these weights yields only minor changes relative to the results that do not correct for attrition and all remain significant. We interpret these findings to mean that, as found in other contexts with high attrition (Fitzgerald, Gottschalk and Moffitt 1998b; Alderman et al. 2001) our results do not appear to be driven by attrition biases.

5.3 Alternative Measures of Height-for-Age

In Section 3, we described how we constructed our synthetic measure of height-for-age z-scores at 36 months. Here, we assess the robustness of our results to alternative constructions of this synthetic measure.

In the first alternative approach, we simply drop all children for whom these synthetic values are generated solely from measures when children were less than 24 months. In the second alternative approach, we only use individuals for whom we have actual measures on height z-scores between the ages of 30 and 42 months. We start with HAZ at age 36 months for which we have 880 observations. If we do not have HAZ at ages 36 or 42 months, we use HAZ at 30 months. This inclusion criterion adds an additional 109 observations. If we do not have HAZ at ages 36 or 30 months, we use HAZ at 42 months. This criterion adds an additional 81 observations. If we do not have HAZ at age 36 months, but have it for both 30 and 42 months, we use the measure that was taken closest to 36 months. This criterion adds a further 34 observations, yielding 1,104 subjects with HAZ measured between 30 and 42 months. We estimate their impact on six outcomes: SIA z-scores, the differential in ages between subjects and their spouses, number of pregnancies, log of fat-free mass, log of hourly earnings (for men only), and per capita household consumption, and we use the same specifications as those used in the results reported previously.

Results are reported in Tables 12 and 13. For both alternatives, test statistics for relevance and overidentification continue to be satisfactory. Because we have smaller sample

sizes, not surprisingly, our parameter estimates are measured with less precision. Apart from the coefficient estimates for reading/vocabulary scores that are slightly lower in these alternative specifications (but still statistically significant), results obtained in Tables 12 and 13 are similar to those found in Tables 4 to 9 for these outcomes. We conclude that our results are robust to alternative ages at which we measure height-for-age z-scores.

6. SUMMARY

This paper examines the impact of growth failure in early life as measured by height-for-age and stunting at 36 months on multiple outcomes over the life course of an individual up to middle adulthood. We overcome the formidable data requirements of this exercise by tracing individuals who participated in a nutritional supplementation trial between 1969 and 1977 in rural Guatemala who were subsequently re-interviewed between 2002 and 2004. We assess impacts across a wide range of domains: education, the marriage market, fertility, health, wages and income, and poverty and consumption in adulthood. We account for the endogeneity of height at age 36 months, using transitory shocks experienced in early life and random variation in genotype as instruments.

We find evidence of positive impacts of better early-life nutrition on a wide range of outcomes. Consistent with the literature we cite in the introduction, we find that the absence of growth failure at 36 months is causally linked to leaving school at an older age and with higher grade attainment. It increases scores on tests of reading/vocabulary skills and on nonverbal cognitive ability. The magnitudes of these impacts are large—an individual not stunted scores more than a full standard deviation higher on the SIA test—especially given that in some cases we are testing individuals nearly 35 years after these anthropometric measures were taken. Once leaving school, individuals who did not experience growth failure make better matches in the marriage market, most notably forming unions with individuals with higher schooling attainments. Women not stunted at age 36 months have 1.86 fewer pregnancies and are less likely to experience still births or miscarriages.

Growth failure in early life has adverse impacts on body size and several dimensions of physical fitness in adulthood. It does not however, have marked effects (positive or negative) on outcomes linked to greater risks of cardiovascular or other chronic diseases. Individuals who were not stunted earn higher wages and are more likely to be employed in higher paying skilled labor and white-collar jobs. These results are consistent with processes by which growth failure in early life leads to cognitive impairments, which limit schooling attainment and the acquisition of cognitive skills, both of which are rewarded in the Guatemalan labor market (see Behrman et al. 2010). For men, a one-standard deviation increase in height-for-age at 36 months raises

hourly earnings by 20 percent. For women, a similar increase raises the likelihood that they operate their own business from which they derive an independent source of income by more than 10 percentage points. Individuals who were not stunted are 33.9 percentage points less likely to live in poor households as adults. A one-standard-deviation increase in height-for-age raises the per capita consumption level of the household that they live in by nearly 20 percent.

Our study has potential weaknesses: the use of instrumental variables to identify causality, sample attrition, and the creation of a measure of anthropometric status for all individuals at a consistent age. We assess the validity of our instruments through the use of tests of instrument weakness and overidentification and find them to be satisfactory. Further, we obtain comparable parameter estimates across a range of instrument sets, which suggests that our inferences are not particularly fragile. Alternative methods that account for sample attrition do not lead to differences in estimates of impact. Our results are robust to alternative methods of constructing the measure of height-for-age.

The pattern of growth failure observed in this sample between 1969 and 1977 remains common in many poor countries. Our results imply that interventions to reduce growth failure in Guatemala and elsewhere have the potential to improve outcomes across the life course, including education, health, fertility, earnings, and consumption. Given that interventions to improve nutritional status in early life are relatively inexpensive (Behrman, Alderman, and Hoddinott 2004; Horton, Alderman, and Rivera 2008), these results provide a powerful rationale for investments that reduce growth failure in low-income countries.

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Table 1. Correlation matrix for HAZ by measured ages

| HAZ at _ months | HAZ at _ months | | | | | | | | | | | |
|--------------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|--|
| | 1 | 6 | 12 | 18 | 24 | 30 | 36 | 42 | 48 | 60 | 72 | |
| 1 | 1.0000 (861) | | | | | | | | | | | |
| 6 | 0.6319 (688) | 1.0000 (956) | | | | | | | | | | |
| 12 | 0.5293 (621) | 0.8407 (791) | 1.0000 (955) | | | | | | | | | |
| 18 | 0.4708 (568) | 0.7799 (733) | 0.8894 (795) | 1.0000 (923) | | | | | | | | |
| 24 | 0.4525 (514) | 0.7309 (676) | 0.8367 (748) | 0.9061 (790) | 1.0000 (919) | | | | | | | |
| 30 | 0.4245 (457) | 0.6573 (613) | 0.7660 (679) | 0.8385 (724) | 0.8922 (775) | 1.0000 (894) | | | | | | |
| 36 | 0.3917 (381) | 0.6743 (548) | 0.7575 (609) | 0.8224 (648) | 0.8875 (710) | 0.9241 (748) | 1.0000 (880) | | | | | |
| 42 | 0.4051 (325) | 0.6829 (485) | 0.7641 (545) | 0.8068 (581) | 0.8524 (629) | 0.9171 (675) | 0.9513 (746) | 1.0000 (863) | | | | |
| 48 | 0.4017 (284) | 0.6179 (445) | 0.7090 (509) | 0.7663 (536) | 0.7982 (593) | 0.8696 (624) | 0.9045 (703) | 0.9385 (739) | 1.0000 (864) | | | |
| 60 | 0.3971 (141) | 0.6118 (298) | 0.7066 (356) | 0.7587 (389) | 0.7847 (433) | 0.8372 (471) | 0.8754 (546) | 0.9133 (587) | 0.9366 (613) | 1.0000 (791) | | |
| 72 | 0.3285 (54) | 0.5297 (198) | 0.6659 (246) | 0.7158 (284) | 0.7391 (324) | 0.7947 (363) | 0.8343 (433) | 0.8722 (473) | 0.8911 (503) | 0.9399 (592) | 1.0000 (727) | |

Source: Authors' calculations.

Notes: Sample sizes are in parentheses.

Table 2. Outcome variables: Definitions and descriptive statistics

| Variable | Definition | Mean (Standard deviation) | | | Sample size |
|---|---|------------------------------|------------------|------------------|-------------|
| | | All | Women | Men | |
| <i>Schooling-related outcomes</i> | | | | | |
| Age started school | Age (in years) when individual commenced attending primary school | 6.80 (1.09) | 6.78 (1.00) | 6.82 (1.19) | 1,365 |
| Repeated primary grade | =1 if individual repeated a grade of primary school | 0.44 (0.50) | 0.40 (0.49) | 0.48 (0.49) | 1,365 |
| Grade progression | Number of grades passed divided by the number of years between when the individual entered and terminated school, up to and including 12th grade. | 0.84 (0.26) | 0.84 (0.27) | 0.84 (0.25) | 1,324 |
| Age left school | Age (in years) when individual stopped attending school | 12.51 (2.95) | 12.06 (2.86) | 13.02 (2.97) | 1,365 |
| Highest grade attained | Highest grade of schooling attained, maximum value is 12 | 4.70 (3.45) | 4.30 (3.31) | 5.15 (3.56) | 1,471 |
| SIA z-score | Inter-American Series test score of reading and vocabulary, standardized with mean 0 and SD 1 within the sample | 0 (1) | -0.072 (0.98) | 0.082 (1.02) | 1,453 |
| Raven's z-score | Raven's Progressive Matrices test score, standardized with mean 0 and SD 1 within the sample | 0 (1) | -0.23 (0.88) | 0.27 (1.06) | 1,452 |
| <i>Marriage market outcomes</i> | | | | | |
| Spouse's age | Age (in years) of spouse at time of current union formation | 33.30 (7.16) | 36.24 (7.31) | 30.41 (5.68) | 1,254 |
| Spouse's grades of schooling | Spouse's highest grade of schooling attained | 4.65 (3.37) | 4.94 (3.57) | 4.45 (3.20) | 1,052 |
| Spouse's height | Spouse's height (cm) | 155.66 (8.03) | 162.46 (5.72) | 150.52 (5.19) | 935 |
| Spouse economically independent at marriage | =1 if spouse lived independently of their parents at the time of union formation | 0.07 (0.25) | 0.08 (0.28) | 0.05 (0.22) | 1,209 |
| Spouse has household goods | =1 if spouse brings household goods such as consumer durables to the union | 0.19 (0.18) | 0.28 (0.16) | 0.09 (0.13) | 1,207 |
| Spouse has productive assets | =1 if spouse brings income-generating assets such as tools, working animals, vehicles to the union | 0.13 (0.22) | 0.23 (0.24) | 0.01 (0.06) | 1,207 |
| Age differential | Spouse's age - own age | 0.76 (5.96) | 3.69 (5.84) | -2.15 (4.49) | 1,254 |
| Schooling differential | Spouse's grade attained - own grade attained | -0.04 | 0.89 | -0.82 | 964 |

| | | | | | |
|------------------------------------|--|------------------|------------------|------------------|-------|
| | | (3.54) | (3.50) | (3.39) | |
| <i>Fertility- related outcomes</i> | | | | | |
| Age at menarche | Age (years) of first menstrual cycle | | 13.57 (1.38) | | 669 |
| First birth before 18 | =1 if woman gave birth before age 18 | | 0.24 (0.43) | | 592 |
| Number of pregnancies | Number of pregnancies including miscarriages and stillbirths | | 3.23 (2.16) | | 671 |
| Any still births or miscarriages | =1 if mother had stillbirth or miscarriage | | 0.22 (0.41) | | 671 |
| Any infant deaths | =1 if mother had child who died before attaining 1y | | 0.15 (0.36) | | 671 |
| Number of surviving children | Number of living children | | 2.71 (1.86) | | 671 |
| <i>Health related outcomes</i> | | | | | |
| Log Height | Log of height measured in cm | 5.05 (0.05) | 5.01 (0.04) | 5.09 (0.04) | 1,160 |
| Log Body Mass Index (BMI) | Log of body weight (kg) divided by the square of height (m ²) | 3.24 (0.17) | 3.28 (0.18) | 3.19 (0.14) | 1,160 |
| Obese | = 1 if Body Mass Index >30.0 | 0.17 (0.37) | 0.25 (0.43) | 0.09 (0.29) | 1,160 |
| Head circumference | Distance (cm) around the largest part of the head | 53.68 (1.73) | 52.81 (1.44) | 54.64 (1.55) | 1,152 |
| Log fat-free mass | Log of fat free mass (= body mass – fat mass) | 3.79 (0.17) | 3.66 (0.10) | 3.93 (0.10) | 1,142 |
| Log hand strength | Log of strength of dominant hand measured in newtons | 3.41 (0.29) | 3.22 (0.20) | 3.66 (0.19) | 1,159 |
| Log VO2 max | Log of maximal oxygen consumption (mL/kg/min) calculated from recovery rates following administration of step-test | 2.82 (0.48) | 3.03 (0.41) | 2.59 (0.45) | 1,138 |
| LDL cholesterol | Low-density lipoprotein cholesterol (mg/dL) | 90.67 (26.95) | 91.82 (26.83) | 88.84 (27.46) | 1,146 |
| Total cholesterol/HDL | Ratio of total cholesterol to LDL cholesterol | 4.61 (1.46) | 4.44 (1.34) | 4.92 (1.61) | 1,186 |
| Hypertensive or prehypertensive | =1 if ratio of systolic to diastolic blood pressure is greater than 120 / 80 | 0.31 (0.46) | 0.20 (0.40) | 0.43 (0.49) | 1,422 |
| Blood glucose level | Plasma glucose concentrations (mg/dL) | 93.83 | 94.70 | 93.53 | 1,186 |

| | | | | | |
|---------------------------------------|--|---------------------------|---------------------------|---------------------------|-------|
| Diabetic or prediabetic | = 1 if plasma glucose concentrations were between 100 and 125 mg/dL (prediabetic) or greater than 126mg/dL (diabetic) | (27.80) 0.21 (0.41) | (29.63) 0.21 (0.41) | (13.41) 0.20 (0.40) | 1,186 |
| Metabolic syndrome | =1 if blood tests show presence of diabetes mellitus, impaired glucose tolerance, impaired fasting glucose, and two of: Blood pressure: $\geq 140/90$ mmHg; BMI >30 ; waist: hip ratio >0.90 (males); waist: hip ratio >0.85 (females); triglycerides ≥ 1.695 mmol/L and HDL cholesterol < 40 mg/dL (males) and < 50 mg/dL (females) | 0.31 (0.46) | 0.40 (0.49) | 0.18 (0.39) | 1,186 |
| <i>Labor market outcomes</i> | | | | | |
| Log hourly earnings | Log of net income from wage work, own-account agriculture and own-business activities divided by hours worked conditional on earning any income in the previous 12 months | 1.95 (0.89) | 1.70 (0.99) | 2.15 (0.75) | 1,124 |
| Log hours worked | Log of hours worked in the previous 12 months | 7.20 (1.16) | 6.62 (1.45) | 7.67 (0.51) | 1,124 |
| Log earned income | Log of net income from wage work, own-account agriculture and own-business activities | 9.16 (1.54) | 8.33 (1.72) | 9.84 (0.92) | 1,124 |
| Skilled labor or white collar work | =1 if individuals currently work in clerical, administrative, technical, or professional positions | 0.22 (0.41) | 0.08 (0.27) | 0.36 (0.48) | 1,422 |
| Worked on own business, fulltime | =1 if individual operates own business for more than nine months per year | 0.23 (0.42) | 0.37 (0.48) | 0.20 (0.40) | 1,417 |
| <i>Consumption and poverty</i> | | | | | |
| Per capita household consumption | Log of per capita household consumption | 8.76 (0.65) | 8.80 (0.57) | 8.73 (0.61) | 1,524 |
| Per capita household food consumption | Log of per capita household food consumption | 7.97 (0.55) | 7.97 (0.57) | 7.97 (0.53) | 1,524 |
| Household is poor | =1 if per capita household consumption is below the poverty line | 0.29 (0.45) | 0.28 (0.45) | 0.30 (0.46) | 1,524 |

Source: Authors' calculations.

Table 3. Correlates of height-for-age z-score, 36 months

| | Covariate | Parameter estimate | Standard error | |
|------------------------------|---|--------------------------------------|----------------|---------|
| Transitory shocks | Exposure from birth to 36 months | -0.099 | (0.197) | |
| | Exposure from birth to 36 months \times <i>atole</i> | 0.279** | (0.122) | |
| | “Earthquake” (subject born in 1974, 1975 or 1976) | -0.243* | (0.142) | |
| | Ministry of Health post existed when person was 24 months | 0.117 | (0.147) | |
| Random variation in genotype | Twin (=0 if twin missing) | -0.934*** | (0.236) | |
| | Log mother’s height | 9.925*** | (1.120) | |
| Other controls, <i>C</i> | Male | -0.083 | (0.051) | |
| Other controls, <i>M</i> | Grades attained, mother | 0.004 | (0.023) | |
| Other controls, <i>W</i> | Grades attained, father | 0.002 | (0.018) | |
| | Wealth index | 0.169*** | (0.042) | |
| Prices, <i>P</i> | Birth year, 1962 | -0.711*** | (0.229) | |
| | Birth year, 1963 | -0.616*** | (0.209) | |
| | Birth year, 1964 | -0.704*** | (0.208) | |
| | Birth year, 1965 | -0.653*** | (0.208) | |
| | Birth year, 1966 | -0.737*** | (0.188) | |
| | Birth year, 1967 | -0.570*** | (0.171) | |
| | Birth year, 1968 | -0.524*** | (0.176) | |
| | Birth year, 1969 | -0.670*** | (0.236) | |
| | Birth year, 1970 | -0.725*** | (0.245) | |
| | Birth year, 1971 | -0.724*** | (0.233) | |
| | Birth year, 1972 | -0.569*** | (0.234) | |
| | Birth year, 1973 | -0.626*** | (0.223) | |
| | Birth year, 1974 | -0.436*** | (0.118) | |
| | Birth year, 1975 | -0.203 | (0.146) | |
| | Other controls, <i>Qinv_i</i> | Dummy variables for village of birth | Included | |
| | | Constant | -51.900*** | (5.620) |
| | R-squared | 0.216 | | |

Source: Authors’ calculations. Notes: Standard errors in parentheses: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. Standard errors are robust to heteroscedasticity and clustered at maternal level. In addition to these variables also included are the following variables that appear in all second stage regressions: distance to school, school quality (permanent structure and student-teacher ratio) at age 7, whether biological parent died before subject was aged 15 years, access to bus service at age 15, as well as dummy variables for the small number of cases where maternal schooling, height or initial wealth was missing. Sample size 1,267.

Table 4a. Impact of HAZ and stunting on schooling-related outcomes

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) |
|--|----------------------|------------------------|---------------------|---------------------|------------------------|---------------------|---------------------|
| Height-for-age z-score, 36 months | Age started school | Repeated primary grade | Grade progression | Age left school | Highest grade attained | SIA z-score | Raven's z score |
| OLS | -0.098*** (0.036) | -0.013 (0.016) | 0.023*** (0.008) | 0.412*** (0.083) | 0.602*** (0.091) | 0.188*** (0.029) | 0.174*** (0.028) |
| IV | -0.192* (0.116) | 0.025 (0.051) | 0.010 (0.024) | 0.639** (0.269) | 0.894** (0.323) | 0.345*** (0.099) | 0.257*** (0.087) |
| Observations | 1,201 | 1,201 | 1,164 | 1,201 | 1,285 | 1,271 | 1267 |
| R-squared | 0.105 | 0.036 | 0.065 | 0.199 | 0.251 | 0.135 | 0.173 |
| Kleibergen-Paap | 19.21 | 19.21 | 18.46 | 19.21 | 20.25 | 20.33 | 20.13 |
| Hansen J test: P-value | 0.804 | 0.625 | 0.544 | 0.277 | 0.669 | 0.702 | 0.509 |

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) |
|---------------------------------|--------------------|------------------------|----------------------|----------------------|------------------------|----------------------|----------------------|
| Stunted at age 36 months | Age started school | Repeated primary grade | Grade progression | Age left school | Highest grade attained | SIA z-score | Raven's z score |
| OLS | 0.017 (0.079) | 0.113*** (0.036) | -0.054*** (0.017) | -0.685*** (0.233) | -0.912*** (0.264) | -0.290*** (0.074) | -0.320*** (0.076) |
| IV | 0.482 (0.397) | -0.157 (0.184) | -0.049 (0.086) | -2.784** (1.095) | -3.373** (1.328) | -1.110*** (0.375) | -0.876*** (0.322) |
| R-squared | 0.079 | 0.005 | 0.067 | 0.116 | 0.143 | 0.039 | 0.121 |
| Kleibergen-Paap | 9.558 | 9.56 | 8.782 | 9.716 | 10.237 | 10.21 | 10.18 |
| Hansen J test: P-value | 0.671 | 0.702 | 0.563 | 0.557 | 0.938 | 0.589 | 0.413 |

Source: Authors' calculations. Notes: Standard errors in parentheses: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. Standard errors robust to heteroscedasticity and clustered at maternal level. Second-stage control variables are sex and birth year dummy variables; maternal schooling; paternal schooling; parental wealth index; whether either parent had died before subject was 15; school quality at age 7 (whether school building is permanent structure and student teacher ratio); distance to village center; bus access at age 15; and village of origin. Critical values for Kleibergen-Paap test statistic at the 5% significance level are 11.29, 6.73, and 5.07 for rejecting null hypothesis of weak instruments, where weak means having bias in the IV results that is larger than 10%, 20%, and 30% of the bias in the OLS results, respectively. R-squared is taken from IV results

Table 4b. IV estimates of the impact of HAZ on schooling-related outcomes by sex

| | (1) Age started school | (2) Repeated primary grade | (3) Grade progression | (4) Age left school | (5) Highest grade attained | (6) SIA z-score | (7) Raven's z score |
|--|------------------------------|----------------------------------|-----------------------------|------------------------|----------------------------------|---------------------|------------------------|
| Women | | | | | | | |
| Height- for- age z- score, 36m | -0.074 (0.141) | 0.109 (0.068) | -0.004 (0.038) | 0.954*** (0.369) | 0.941** (0.402) | 0.465*** (0.127) | 0.197* (0.104) |
| Observations | 630 | 630 | 613 | 630 | 650 | 671 | 670 |
| R-squared | 0.109 | 0.012 | 0.086 | 0.177 | 0.232 | 0.099 | 0.112 |
| Kleibergen-Paap | 8.922 | 8.922 | 8.555 | 8.848 | 9.273 | 9.768 | 9.718 |
| Hansen J test: P-value | 0.627 | 0.476 | 0.397 | 0.150 | 0.172 | 0.631 | 0.883 |
| Men | | | | | | | |
| Stunted at age 36 months | -0.232* (0.138) | -0.022 (0.064) | 0.036 (0.023) | 0.448 (0.320) | 1.036*** (0.341) | 0.224* (0.115) | 0.334*** (0.120) |
| Observations | 571 | 571 | 551 | 571 | 588 | 600 | 597 |
| R-squared | 0.176 | 0.047 | 0.070 | 0.206 | 0.268 | 0.219 | 0.165 |
| Kleibergen-Paap | 16.11 | 16.11 | 16.21 | 16.26 | 16.47 | 15.65 | 15.63 |
| Hansen J test: P-value | 0.469 | 0.137 | 0.293 | 0.228 | 0.321 | 0.564 | 0.506 |
| HAZ _F = HAZ _M : P-value of test statistic | 0.178 | 0.144 | 0.600 | 0.704 | 0.515 | 0.148 | 0.055* |

Source: Authors' calculations. Notes: Standard errors in parentheses: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. For other notes, see Table 4a.

Table 5a. Impact of HAZ and stunting on marriage market outcomes

| <i>Height- for- age z- score, 36 months</i> | (1) Spouse's age | (2) Spouse's grades of schooling | (3) Spouse's height | (4) Spouse economically independent at marriage | (5) Spouse has household goods | (6) Spouse has productive assets | (7) Age differential | (8) Schooling differential |
|---|---------------------|-------------------------------------|------------------------|--|-----------------------------------|-------------------------------------|-------------------------|-------------------------------|
| OLS | 0.610*** (0.165) | 0.344*** (0.113) | 0.580*** (0.198) | 0.008 (0.009) | 0.007 (0.005) | -0.001 (0.006) | 0.603*** (0.165) | -0.221* (0.120) |
| IV | 1.313*** (0.475) | 1.085*** (0.320) | 1.054** (0.510) | 0.032 (0.027) | 0.026* (0.014) | 0.014 (0.017) | 1.386*** (0.473) | -0.281 (0.367) |
| Observations | 1,096 | 929 | 823 | 1,056 | 1,055 | 1,055 | 1,096 | 848 |
| R-squared | 0.471 | 0.100 | 0.556 | 0.036 | 0.297 | 0.276 | 0.249 | 0.127 |
| Kleibergen-Paap | 20.41 | 25.25 | 22.21 | 19.48 | 19.54 | 19.54 | 20.41 | 22.02 |
| Hansen J test: P-value | 0.681 | 0.409 | 0.097* | 0.182 | 0.332 | 0.958 | 0.847 | 0.269 |
| <i>Stunted at age 36 months</i> | (1) Spouse's age | (2) Spouse's grades of schooling | (3) Spouse's height | (4) Spouse economically independent at marriage | (5) Spouse has household goods | (6) Spouse has productive assets | (7) Age differential | (8) Schooling differential |
| OLS | -0.772** (0.392) | -0.591* (0.303) | -0.879* (0.518) | -0.014 (0.025) | -0.005 (0.013) | -0.007 (0.016) | -0.808** (0.395) | 0.199 (0.312) |
| IV | -3.804** (1.641) | -3.990*** (1.247) | -3.242* (1.805) | -0.102 (0.091) | -0.045 (0.049) | -0.030 (0.058) | -4.237*** (1.641) | 0.828 (1.334) |
| Observations | 1,096 | 929 | 823 | 1,056 | 1,055 | 1,055 | 1,096 | 848 |
| R-squared | 0.448 | -0.009 | 0.543 | 0.027 | 0.299 | 0.279 | 0.211 | 0.120 |
| Kleibergen-Paap | 10.76 | 9.324 | 8.835 | 10.47 | 10.49 | 10.49 | 10.76 | 7.501 |
| Hansen J test: P-value | 0.453 | 0.654 | 0.078* | 0.183 | 0.202 | 0.923 | 0.696 | 0.258 |

Source: Authors' calculations. Notes: Standard errors in parentheses: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. For other notes, see Table 4a.

Table 5b. IV estimates of the impact of HAZ on marriage market outcomes by sex

| | (1) Spouse's age | (2) Spouse's grades of schooling | (3) Spouse's height | (4) Spouse economically independent at marriage | (5) Spouse has household goods | (6) Spouse has productive assets | (7) Age differential | (8) Schooling differential |
|--|------------------------|---|---------------------------|---|---|---|----------------------------|----------------------------------|
| Women | | | | | | | | |
| Height-for-age z- score, 36m | 1.903** | 0.428 | 0.408 | 0.064* | 0.047** | 0.027 | 1.927** | -0.490 |
| | (0.804) | (0.522) | (0.727) | (0.038) | (0.020) | (0.029) | (0.796) | (0.519) |
| Observations | 528 | 378 | 340 | 583 | 582 | 582 | 528 | 376 |
| R-squared | 0.355 | 0.150 | 0.131 | 0.017 | -0.010 | 0.033 | 0.009 | 0.165 |
| Kleibergen-Paap | 8.895 | 16.62 | 16.15 | 9.980 | 10.05 | 10.05 | 8.895 | 15.88 |
| Hansen J test: P-value | 0.979 | 0.213 | 0.451 | 0.765 | 0.848 | 0.907 | 0.994 | 0.783 |
| Men | | | | | | | | |
| Height-for-age z- score, 36m | 0.826* | 1.062*** | 1.397** | 0.003 | 0.001 | -0.006 | 0.901** | -0.146 |
| | (0.430) | (0.340) | (0.626) | (0.028) | (0.015) | (0.004) | (0.437) | (0.387) |
| Observations | 568 | 551 | 483 | 473 | 473 | 473 | 568 | 472 |
| R-squared | 0.431 | 0.140 | 0.046 | 0.072 | 0.075 | 0.022 | 0.086 | 0.078 |
| Kleibergen-Paap | 18.37 | 17.73 | 14.40 | 15.17 | 15.17 | 15.17 | 18.37 | 17.61 |
| Hansen J test: P-value | 0.497 | 0.634 | 0.111 | 0.0588 | 0.573 | 0.414 | 0.516 | 0.480 |
| HAZ _F = HAZ _M : P-value of test statistic | 0.305 | 0.424 | 0.732 | 0.635 | 0.068* | 0.522 | 0.274 | 0.250 |

Source: Authors' calculations. Notes: Standard errors in parentheses: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. For other notes, see Table 4a.

Table 6. Impact of HAZ and stunting on fertility-related outcomes

| | (1) | (2) | (3) | (4) | (5) | (6) |
|---|----------------------|-----------------------|-----------------------|--------------------------------|-------------------|------------------------------|
| <i>Height-for-age z-score, 36 months</i> | Age at menarche | First birth before 18 | Number of pregnancies | Any still birth or miscarriage | Any infant deaths | Number of surviving children |
| OLS | -0.148*** (0.056) | -0.018 (0.019) | -0.176** (0.084) | -0.009 (0.016) | -0.020 (0.014) | -0.144** (0.069) |
| IV | -0.082 (0.142) | -0.082 (0.050) | -0.609*** (0.232) | -0.110** (0.049) | -0.046 (0.034) | -0.458** (0.209) |
| Observations | 669 | 592 | 671 | 671 | 671 | 671 |
| R-squared | 0.071 | 0.046 | 0.158 | -0.006 | 0.077 | 0.170 |
| Kleibergen-Paap | 9.751 | 10.02 | 9.796 | 9.796 | 9.796 | 9.796 |
| Hansen J test: P-value | 0.757 | 0.516 | 0.544 | 0.289 | 0.196 | 0.536 |
| <hr/> | | | | | | |
| | (1) | (2) | (3) | (4) | (5) | (6) |
| <i>Stunted at age 36 months</i> | Age at menarche | First birth before 18 | Number of pregnancies | Any still birth or miscarriage | Any infant deaths | Number of surviving children |
| OLS | 0.349** (0.151) | 0.011 (0.054) | 0.275 (0.215) | -0.017 (0.041) | 0.030 (0.033) | 0.248 (0.180) |
| IV | 0.162 (0.498) | 0.342* (0.187) | 1.860** (0.802) | 0.369** (0.178) | 0.105 (0.111) | 1.433** (0.697) |
| R-squared | 0.069 | -0.015 | 0.118 | -0.069 | 0.073 | 0.138 |
| Kleibergen-Paap | 6.182 | 6.375 | 6.157 | 6.157 | 6.157 | 6.157 |
| Hansen J test: P-value | 0.702 | 0.672 | 0.467 | 0.313 | 0.110 | 0.515 |

Source: Authors' calculations. Notes: Standard errors in parentheses: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. For other notes, see Table 4a.

Table 7a. Impact of HAZ and stunting on health-related outcomes

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) |
|--|----------------------|---------------------|-------------------|----------------------|----------------------|----------------------|--------------------|
| <i>Height- for-age z-score, 36 months</i> | Log height | Log BMI | Obese | Head circumference | Log fat-free mass | Log hand strength | Log VO2 max |
| OLS | 0.023*** (0.001) | 0.017*** (0.005) | 0.014 (0.012) | 0.452*** (0.041) | 0.047*** (0.003) | 0.044*** (0.006) | 0.031** (0.014) |
| IV | 0.044*** (0.004) | 0.018 (0.015) | 0.038 (0.032) | 0.733*** (0.131) | 0.076*** (0.009) | 0.055*** (0.017) | 0.021 (0.037) |
| Observations | 1,160 | 1,160 | 1,160 | 1,143 | 1,142 | 1,159 | 1,138 |
| R-squared | 0.600 | 0.111 | 0.056 | 0.409 | 0.718 | 0.582 | 0.250 |
| Kleibergen-Paap | 18.12 | 18.12 | 18.12 | 18.34 | 18.37 | 17.88 | 18.61 |
| Hansen J test: P-value | 0.014** | 0.413 | 0.104 | 0.525 | 0.146 | 0.104 | 0.299 |
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) |
| <i>Stunted at age 36 months</i> | Log height | Log BMI | Obese | Head circumference | Log fat-free mass | Log hand strength | Log VO2 max |
| OLS | -0.041*** (0.003) | -0.029** (0.013) | -0.016 (0.030) | -0.713*** (0.124) | -0.077*** (0.007) | -0.073*** (0.015) | -0.043 (0.035) |
| IV | -0.159*** (0.022) | -0.076 (0.059) | -0.149 (0.121) | -2.852*** (0.609) | -0.287*** (0.049) | -0.183*** (0.071) | -0.110 (0.143) |
| R-squared | -0.034 | 0.095 | 0.042 | 0.189 | 0.496 | 0.551 | 0.245 |
| Kleibergen-Paap | 9.150 | 9.150 | 9.150 | 8.840 | 8.873 | 9.323 | 9.269 |
| Hansen J test: P-value | 0.075* | 0.475 | 0.111 | 0.761 | 0.180 | 0.088* | 0.325 |

Table 7a. Continued

| | (1) | (2) | (3) | (4) | (5) | (6) |
|---|--------------------|-----------------------|---------------------------------|---------------------|-------------------------|--------------------|
| <i>Height-for-age z-score, 36 months</i> | LDL cholesterol | Total cholesterol/HDL | Hypertensive or prehypertensive | Blood glucose level | Diabetic or prediabetic | Metabolic syndrome |
| OLS | 0.847 (0.989) | 0.005 (0.051) | 0.035*** (0.014) | 0.336 (0.778) | -0.001 (0.015) | 0.024 (0.015) |
| IV | 2.429 (3.084) | -0.017 (0.146) | 0.130*** (0.047) | 2.718 (1.688) | 0.046 (0.037) | 0.040 (0.037) |
| Observations | 999 | 1,034 | 670 | 1,034 | 1,034 | 1,034 |
| R-squared | 0.048 | 0.060 | 0.018 | 0.022 | 0.034 | 0.106 |
| Kleibergen-Paap | 15.38 | 15.10 | 9.785 | 15.10 | 15.10 | 15.10 |
| Hansen J test: P-value | 0.567 | 0.361 | 0.136 | 0.403 | 0.026** | 0.148 |
| | (1) | (2) | (3) | (4) | (5) | (6) |
| <i>Stunted at age 36 months</i> | LDL cholesterol | Total cholesterol/HDL | Hypertensive or prehypertensive | Blood glucose level | Diabetic or prediabetic | Metabolic syndrome |
| OLS | -3.551 (2.424) | 0.009 (0.128) | -0.034 (0.037) | -0.799 (1.735) | -0.040 (0.032) | -0.061* (0.036) |
| IV | -6.729 (12.055) | -0.022 (0.539) | -0.057 (0.141) | -9.580 (6.592) | -0.231* (0.138) | -0.169 (0.141) |
| R-squared | 0.050 | 0.060 | 0.090 | 0.012 | 0.016 | 0.099 |
| Kleibergen-Paap | 7.981 | 8.097 | 9.560 | 8.097 | 8.097 | 8.097 |
| Hansen J test: P-value | 0.519 | 0.360 | 0.011** | 0.362 | 0.040** | 0.170 |

Source: Authors' calculations. Notes: Standard errors in parentheses: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. For other notes, see Table 4a.

Table 7b. IV estimates of the impact of HAZ on health-related outcomes by sex

| Variables | (1) Log height | (2) Log BMI | (3) Obese | (4) Head circumference | (5) Log fat-free mass | (6) Log hand strength | (7) Log VO2 max |
|--|---------------------|------------------|------------------|------------------------------|-----------------------------|-----------------------------|--------------------|
| Women | | | | | | | |
| <i>Height-for-age z-score, 36m</i> | 0.044*** (0.005) | 0.023 (0.023) | 0.061 (0.054) | 0.705*** (0.173) | 0.074*** (0.010) | 0.031 (0.023) | 0.032 (0.045) |
| Observations | 604 | 604 | 604 | 599 | 598 | 645 | 595 |
| R-squared | 0.181 | 0.045 | 0.016 | 0.206 | 0.161 | 0.088 | 0.045 |
| Kleibergen-Paap | 8.482 | 8.482 | 8.482 | 8.305 | 8.308 | 9.720 | 8.764 |
| Hansen J test: P-value | 0.114 | 0.164 | 0.079 | 0.104 | 0.318 | 0.826 | 0.280 |
| Men | | | | | | | |
| <i>Height-for-age z-score, 36m</i> | 0.042*** (0.004) | 0.009 (0.016) | 0.025 (0.028) | 0.675*** (0.154) | 0.077*** (0.011) | 0.085*** (0.023) | 0.020 (0.049) |
| Observations | 556 | 556 | 556 | 544 | 544 | 514 | 543 |
| R-squared | 0.188 | 0.103 | 0.057 | 0.237 | 0.221 | 0.052 | 0.100 |
| Kleibergen-Paap | 14.79 | 14.79 | 14.79 | 15.96 | 16.22 | 13.44 | 15.83 |
| Hansen J test: P-value | 0.105 | 0.423 | 0.618 | 0.827 | 0.135 | 0.120 | 0.351 |
| HAZ _F = HAZ _M : P-value of test statistic | 0.993 | 0.114 | 0.295 | 0.812 | 0.814 | 0.122 | 0.102 |

Table 7b. Continued

| | (1) LDL cholesterol | (2) Total cholesterol/HDL | (3) Hypertensive or prehypertensive | (4) Blood glucose level | (5) Diabetic or prediabetic | (6) Metabolic syndrome |
|--|------------------------|---------------------------------|---|-------------------------------|-----------------------------------|------------------------------|
| Women | | | | | | |
| Height- for-age z- score, 36m | 0.137 (3.555) | -0.196 (0.174) | 0.130*** (0.047) | 3.694 (2.689) | -0.053 (0.054) | -0.034 (0.059) |
| Observations | 585 | 600 | 670 | 600 | 600 | 600 |
| R-squared | 0.056 | 0.030 | 0.018 | 0.038 | 0.063 | 0.062 |
| Kleibergen-Paap | 9.186 | 8.821 | 9.785 | 8.821 | 8.821 | 8.821 |
| Hansen J test: P-value | 0.219 | 0.758 | 0.136 | 0.436 | 0.128 | 0.572 |
| Men | | | | | | |
| Height- for-age z-score, 36m | 3.906 (3.977) | 0.317 (0.224) | -0.026 (0.052) | 1.969 (1.469) | 0.126** (0.052) | 0.092* (0.048) |
| Observations | 414 | 434 | 576 | 434 | 434 | 434 |
| R-squared | 0.092 | 0.028 | 0.039 | 0.060 | 0.036 | 0.075 |
| Kleibergen-Paap | 11.51 | 11.84 | 13.46 | 11.84 | 11.84 | 11.84 |
| Hansen J test: P-value | 0.542 | 0.151 | 0.006*** | 0.219 | 0.247 | 0.110 |
| HAZ _F = HAZ _M : P-value of test statistic | 0.769 | 0.265 | 0.569 | 0.577 | 0.127 | 0.170 |

Source: Authors' calculations. Notes: Standard errors in parentheses: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. For other notes, see Table 4a.

Table 8a. Impact of HAZ and stunting on labor market outcomes

| | (1) | (2) | (3) | (4) | (5) |
|--|---------------------|-------------------|---------------------|------------------------------------|----------------------------------|
| <i>Height-for-age z- score, 36 months</i> | Log hourly earnings | Log hours worked | Log earned income | Skilled labor or white-collar work | Worked on own business, fulltime |
| OLS | 0.116*** (0.029) | 0.020 (0.036) | 0.137*** (0.049) | 0.047*** (0.011) | 0.019 (0.012) |
| IV | 0.148* (0.078) | 0.006 (0.081) | 0.139 (0.120) | 0.079*** (0.032) | 0.062* (0.034) |
| Observations | 989 | 989 | 989 | 1,193 | 1,193 |
| R-squared | 0.137 | 0.240 | 0.295 | 0.166 | 0.046 |
| Kleibergen-Paap | 19.50 | 19.50 | 19.50 | 20.28 | 20.28 |
| Hansen J test: P-value | 0.352 | 0.820 | 0.348 | 0.178 | 0.959 |
| <hr/> | | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| <i>Stunted at age 36 months</i> | Log hourly earnings | Log hours worked | Log earned income | Skilled labor or white-collar work | Worked on own business, fulltime |
| OLS | -0.133* (0.073) | -0.008 (0.097) | -0.137 (0.120) | -0.076*** (0.032) | 0.025 (0.030) |
| IV | -0.540* (0.320) | -0.127 (0.311) | -0.646 (0.471) | -0.282*** (0.120) | -0.178 (0.129) |
| R-squared | 0.098 | 0.239 | 0.273 | 0.129 | 0.020 |
| Kleibergen-Paap | 7.660 | 7.660 | 7.660 | 10.639 | 10.639 |
| Hansen J test: P-value | 0.321 | 0.837 | 0.423 | 0.183 | 0.837 |

Source: Authors' calculations. Notes: Standard errors in parentheses: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. For other notes, see Table 4a.

Table 8b. IV estimates of the impact of HAZ on labor market outcomes by sex

| | (1) Log hourly earnings | (2) Log hours worked | (3) Log earned income | (4) Skilled labor or white-collar work | (5) Worked on own business, fulltime |
|--|-------------------------------|----------------------------|-----------------------------|--|--|
| Women | | | | | |
| <i>Height- for-age z-score, 36m</i> | 0.072 (0.130) | 0.242 (0.164) | 0.269 (0.210) | 0.072*** (0.025) | 0.108** (0.046) |
| Observations | 439 | 439 | 439 | 635 | 635 |
| R-squared | 0.083 | 0.109 | 0.118 | 0.023 | 0.034 |
| Kleibergen-Paap | 10.00 | 10.00 | 10.00 | 11.65 | 11.65 |
| Hansen J test: P-value | 0.785 | 0.783 | 0.506 | 0.743 | 0.656 |
| Men | | | | | |
| <i>Height- for-age z-score, 36m</i> | 0.201** (0.090) | -0.099 (0.063) | 0.039 (0.105) | 0.077 (0.059) | 0.022 (0.040) |
| Observations | 550 | 550 | 550 | 558 | 558 |
| R-squared | 0.108 | 0.052 | 0.094 | 0.093 | 0.062 |
| Kleibergen-Paap | 12.46 | 12.46 | 12.46 | 12.62 | 12.62 |
| Hansen J test: P-value | 0.311 | 0.038** | 0.340 | 0.045** | 0.275 |
| HAZ _F = HAZ _M : P-value of test statistic | 0.177 | 0.199 | 0.939 | 0.775 | 0.898 |

Source: Authors' calculations. Notes: Standard errors in parentheses: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. For other notes, see Table 4a.

Table 9a. Impact of HAZ and stunting on consumption and poverty

| | (1) | (2) | (3) |
|---|--------------------------------------|---|---------------------|
| Height- for-age z-score, 36 months | Log per capita household consumption | Log per capita household food consumption | Household is poor |
| OLS | 0.087*** (0.020) | 0.064 (0.016) | -0.030** (0.014) |
| IV | 0.195*** (0.057) | 0.146*** (0.048) | -0.108** (0.043) |
| Observations | 1,335 | 1,335 | 1,335 |
| R-squared | 0.078 | 0.068 | 0.043 |
| Kleibergen-Paap | 22.02 | 22.02 | 22.02 |
| Hansen J test: P-value | 0.313 | 0.141 | 0.036** |
| | (1) | (2) | (3) |
| Stunted at age 36 months | Log per capita household consumption | Log per capita household food consumption | Household is poor |
| OLS | -0.092** (0.047) | -0.066* (0.040) | 0.022 (0.033) |
| IV | -0.661*** (0.210) | -0.551*** (0.178) | 0.339** (0.152) |
| R-squared | -0.025 | -0.017 | -0.003 |
| Kleibergen-Paap | 12.21 | 12.21 | 12.21 |
| Hansen J test: P-value | 0.450 | 0.25 | 0.055** |

Source: Authors' calculations. Notes: Standard errors in parentheses: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. For other notes, see Table 4a.

Table 9b. IV estimates of the impact of HAZ on consumption and poverty

| | (1) Log per capita household consumption | (2) Log per capita household food consumption | (3) Household is poor |
|--|--|---|--------------------------|
| Women | | | |
| Height-for-age z-score, 36m | 0.182** (0.082) | 0.151** (0.070) | -0.067 (0.054) |
| Observations | 663 | 663 | 663 |
| R-squared | 0.085 | 0.063 | 0.065 |
| Kleibergen-Paap | 9.348 | 9.348 | 9.348 |
| Hansen J test: P-value | 0.146 | 0.149 | 0.402 |
| Men | | | |
| Height- for-age z-score, 36m | 0.181*** (0.058) | 0.132** (0.051) | -0.162*** (0.053) |
| Observations | 672 | 672 | 672 |
| R-squared | 0.123 | 0.122 | 0.048 |
| Kleibergen-Paap | 18.31 | 18.31 | 18.31 |
| Hansen J test: P-value | 0.760 | 0.452 | 0.129 |
| HAZ _F = HAZ _M : P-value of test statistic | 0.762 | 0.689 | 0.131 |

Source: Authors' calculations. Notes: Standard errors in parentheses: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. For other notes, see Table 4a.

Table 10. Alternative specification of instrument sets

| | Base | Alternative specifications | | | | | | | |
|--|-----------|----------------------------|----------|-----------|-----------|-----------|---------|---------|---------|
| | | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| Exposure from birth to 36 months | YES | YES | YES | YES | | | YES | YES | YES |
| Exposure from birth to 36 months \times <i>atole</i> | YES | YES | YES | YES | | | YES | YES | YES |
| Twin (=0 if twin missing) | YES | | | YES | | YES | YES | | YES |
| Log mother's height | YES | YES | YES | YES | YES | YES | | | |
| Earthquake (birth year is 1974–76) | YES | YES | | | YES | YES | YES | | YES |
| Ministry of Health post, age 24 months | YES | YES | | | YES | YES | YES | | |
| Reading scores | | | | | | | | | |
| IV estimate of β | 0.344*** | 0.363*** | 0.356*** | 0.336*** | 0.323*** | 0.306*** | 0.365* | 0.840* | 0.368* |
| Standard error | (0.098) | (0.100) | (0.104) | (0.102) | (0.105) | (0.103) | (0.221) | (0.492) | (0.222) |
| Kleibergen-Paap | 20.33 | 20.59 | 29.78 | 27.65 | 29.18 | 27.74 | 5.99 | 2.33 | 7.41 |
| Hansen J test: P-value | 0.70 | 0.74 | 0.49 | 0.49 | 0.91 | 0.84 | 0.46 | 0.53 | 0.34 |
| Spouse's grades of schooling | | | | | | | | | |
| IV estimate of β | 1.085*** | 1.058*** | 0.947*** | 1.005*** | 1.089*** | 1.111*** | 1.789** | 1.911 | 1.768** |
| Standard error | (0.320) | (0.333) | (0.339) | (0.324) | (0.336) | (0.322) | (0.890) | (4.637) | (0.884) |
| Kleibergen-Paap | 25.25 | 19.10 | 26.53 | 34.97 | 28.43 | 34.53 | 9.24 | 0.25 | 11.71 |
| Hansen J test: P-value | 0.41 | 0.30 | 0.45 | 0.57 | 0.19 | 0.31 | 0.36 | 0.17 | 0.53 |
| Number of pregnancies | | | | | | | | | |
| IV estimate of β | -0.609*** | -0.541** | -0.562** | -0.647*** | -0.678*** | -0.735*** | -0.440 | 0.806 | -0.615 |
| Standard error | (0.232) | (0.247) | (0.258) | (0.241) | (0.274) | (0.243) | (0.498) | (1.240) | (0.497) |
| Kleibergen-Paap | 9.80 | 9.97 | 14.10 | 13.08 | 14.22 | 13.67 | 2.40 | 1.31 | 3.00 |
| Hansen J test: P-value | 0.54 | 0.48 | 0.66 | 0.67 | 0.57 | 0.69 | 0.29 | 0.71 | 0.48 |
| Log fat-free mass | | | | | | | | | |
| IV estimate of β | 0.076*** | 0.083*** | 0.085*** | 0.078*** | 0.085*** | 0.077*** | 0.041** | -0.007 | 0.040** |
| Standard error | (0.009) | (0.010) | (0.013) | (0.089) | (0.010) | (0.009) | (0.015) | (0.045) | (0.016) |
| Kleibergen-Paap | 23.19 | 25.20 | 36.51 | 31.44 | 36.53 | 31.97 | 4.33 | 2.20 | 5.39 |
| Hansen J test: P-value | 0.15 | 0.18 | 0.27 | 0.20 | 0.29 | 0.21 | 0.42 | 0.98 | 0.44 |

Table 10.Continued

| | Base | Alternative specifications | | | | | | | |
|--|----------|----------------------------|----------|----------|----------|----------|----------|---------|----------|
| | | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| Exposure from birth to 36 months | YES | YES | YES | YES | | | YES | YES | YES |
| Exposure from birth to 36 months \times <i>atole</i> | YES | YES | YES | YES | | | YES | YES | YES |
| Twin (=0 if twin missing) | YES | | | YES | | YES | YES | | YES |
| Log mother's height | YES | YES | YES | YES | YES | YES | | | |
| Earthquake (birth year is 1974-76) | YES | YES | | | YES | YES | YES | | YES |
| Ministry of Health post, age 24 months | YES | YES | | | YES | YES | YES | | |
| Hourly earnings (males) | | | | | | | | | |
| IV estimate of β | 0.201** | 0.199** | 0.190* | 0.194** | 0.183* | 0.187** | 0.426* | 1.043 | 0.404* |
| Standard error | (0.089) | (0.097) | (0.098) | (0.091) | (0.098) | (0.091) | (0.232) | (1.042) | (0.236) |
| Kleibergen-Paap | 12.46 | 10.49 | 14.94 | 16.35 | 15.33 | 16.69 | 8.80 | 0.69 | 10.52 |
| Hansen J test: P-value | 0.31 | 0.21 | 0.19 | 0.32 | 0.15 | 0.25 | 0.69 | 0.75 | 0.53 |
| Consumption | | | | | | | | | |
| IV estimate of β | 0.195*** | 0.184*** | 0.171*** | 0.182*** | 0.195*** | 0.205*** | 0.357*** | -0.225 | 0.351*** |
| Standard error | (0.057) | (0.057) | (0.059) | (0.058) | (0.058) | (0.058) | (0.126) | (0.377) | (0.127) |
| Kleibergen-Paap | 22.02 | 22.07 | 32.09 | 30.00 | 32.05 | 30.42 | 6.32 | 1.52 | 7.73 |
| Hansen J test: P-value | 0.31 | 0.41 | 0.23 | 0.19 | 0.85 | 0.63 | 0.27 | 0.19 | 0.11 |

Source: Authors' calculations.

Table 11. Robustness checks: Adjusting for attrition and standard errors based on birth-year, birth-place clusters

| Outcome | (1) | (2) |
|----------------------------------|----------------------------|---------------------|
| | Not weighted for attrition | Attrition weighted |
| SIA z-score | 0.345*** (0.099) | 0.338*** (0.102) |
| Spouse's grades of schooling | 1.085*** (0.320) | 1.139*** (0.332) |
| Number of pregnancies | -0.609*** (0.232) | -0.603** (0.241) |
| Log fat-free mass | 0.076*** (0.009) | 0.078*** (0.009) |
| Log hourly earnings (males) | 0.201** (0.090) | 0.187* (0.099) |
| Per capita household consumption | 0.195*** (0.057) | 0.198*** (0.058) |

Source: Authors' calculations. Notes: Standard errors in parentheses; * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. For other notes, see Table 4a.

Table 12. Selected results using predicted values of HAZ at 36 months, excluding individuals who were only measured before 24 months

| | (1) | (2) | (3) | (4) | (5) | (6) |
|------------------------|---------------------|------------------------------|-----------------------|---------------------|-----------------------------|----------------------------------|
| | SIA z-score | Spouse's grades of schooling | Number of pregnancies | Log fat-free mass | Log hourly earnings (males) | Per capita household consumption |
| HAZ | 0.306*** (0.108) | 1.498*** (0.341) | -0.817*** (0.252) | 0.077*** (0.009) | 0.135 (0.092) | 0.196*** (0.065) |
| Observations | 1,125 | 830 | 601 | 1,017 | 483 | 1,181 |
| Kleibergen-Paap | 24.23 | 26.95 | 13.01 | 23.56 | 14.70 | 22.98 |
| Hansen J test: P-value | 0.526 | 0.097* | 0.232 | 0.30 | 0.340 | 0.225 |

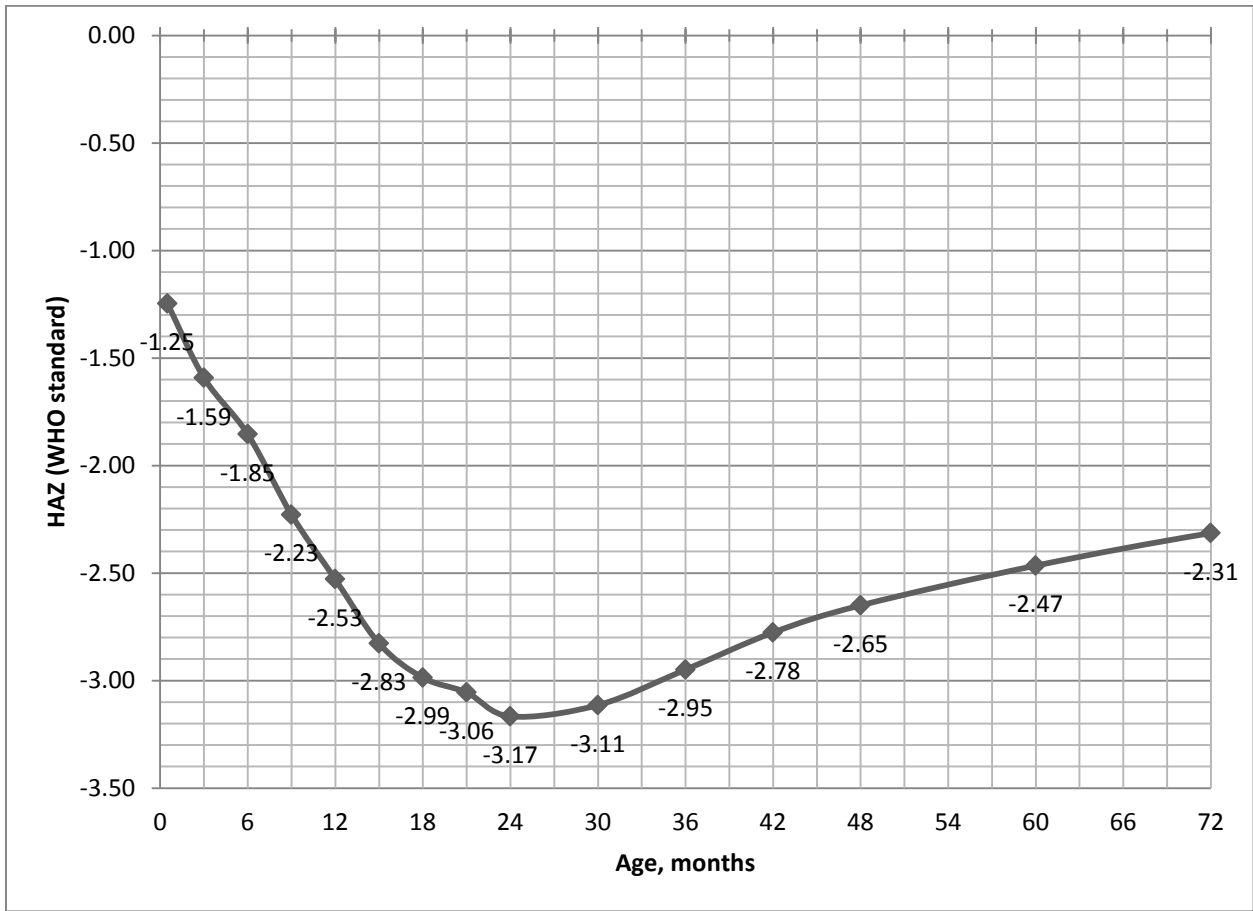
Source: Authors' calculations. Notes: Standard errors in parentheses: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. For other notes, see Table 4a.

Table 13. Selected results using actual HAZ for individuals aged 30-42 months

| | (1) | (2) | (3) | (4) | (5) | (6) |
|------------------------|--------------------|------------------------------|-----------------------|---------------------|-----------------------------|----------------------------------|
| | SIA z- score | Spouse's grades of schooling | Number of pregnancies | Log fat-free mass | Log hourly earnings (males) | Per capita household consumption |
| HAZ | 0.237** (0.120) | 1.717*** (0.362) | -0.920*** (0.283) | 0.077*** (0.009) | 0.164* (0.100) | 0.125* (0.066) |
| Observations | 796 | 590 | 417 | 716 | 348 | 836 |
| Kleibergen-Paap | 18.93 | 17.59 | 9.692 | 15.82 | 11.19 | 16.72 |
| Hansen J test: P-value | 0.313 | 0.044** | 0.617 | 0.25 | 0.241 | 0.325 |

Source: Authors' calculations. Notes: Standard errors in parentheses: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level. For other notes, see Table 4a.

Figure 1. Mean HAZ by age of children at measurement in the 1969–77 study



Source: Authors' calculations.