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The impact of malnutrition over the life course

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Abstract

This paper examines the impact of being malnourished as measured by height for age (HAZ) at 36m over the life course. It uses data collected on individuals who participated in a nutrition supplementation trial between 1969 and 1977 in rural Guatemala and who were subsequently re-interviewed between 2002 and 2004. We assess impacts across a wide range of domains: schooling; the marriage market; fertility; health; wages and income; and poverty and consumption in adulthood. We find that poor nutritional status at age 36m is linked to lower grade attainment and poorer cognitive skills in adulthood. Men earn lower wages and women are less likely to have independent sources of income from own business activities. Women who were stunted at age 36m have, on average, 1.9 more pregnancies and are more likely to give birth before age 17. Better nourished preschoolers are taller as adults and have more fat free mass and greater hand strength. Being stunted at age 36m increases the likelihood of living in a poor household in adulthood by 31 percentage points.

1. Introduction

Around the world, more than 175 million preschool children are chronically malnourished or stunted, that is their height given their age is more than two standard deviations below that of international reference standards (Black *et al*, 2008). The physiological consequences of chronic malnourishment are well understood. In early life, these young children have high nutritional requirements, but the diets typically available to them have poor energy and nutrient concentrations. They are also very susceptible to infections because their immature immune systems fail to protect them adequately. In poor countries, foods and liquids are often contaminated and are thus key sources of frequent illness. Energy that should go towards growth is instead diverted to fighting off these infections (Martorell, 1997). Over time, "prolonged or severe nutrient depletion eventually leads to retardation of linear (skeletal) growth in children and to loss of, or failure to accumulate, muscle mass and fat" (Morris, 2001, p.12). This retardation of linear growth has permanent consequences as growth in stature lost in early life is never fully regained (Martorell, 1999).

There are adverse neurological consequences of early -life chronic malnutrition.[1] Early life malnutrition damages the hippocampus by reducing dentrite[2] density (Blatt et al, 1994; Mazer et al, 1997; Ranade et al, 2008). It reduces element binding proteins responsible for the processes by which a chemical stimulus induces a cellular response. This adversely affects spatial navigation, memory formation (Huang et al, 2003) and memory consolidation (Valadares and de Sousa Almeida, 2005). In severely malnourished children, dentrites in the occipital lobe (responsible for the processing of visual information) and in the motor cortex are shorter, having fewer spines and greater numbers of abnormalities (Benítez-Bribiesca, De la Rosa-Alvarez and Mansilla-Olivares, 1999). Consistent with these findings, animal studies show that chronic malnutrition leads to delays in the evolution of locomotor skills (Barros et al, 2006). Malnutrition results in reduced myelination of axon fibers thus reducing the speed at which

[1] Scrimshaw and Gordon (1968) first posited that malnutrition might adversely affect neurological function. Levitsky and Strupp (1995) provide a review of the state of knowledge as of the mid-1990s. The literature continues to grow - a Medline search generates more than 5000 studies on the links between dimensions of malnutrition and neurological structure and function.

[2] Dentrites are branch like structures, which receive signals sent along axons

signals are transmitted (Levitsky and Strupp, 1995). Early-life undernutrition decreases the number of neurons in the locus coeruleus (Pinos, Collado, Salas and Pérez-Torrero, 2006). When activated by stress, the locus coeruleus increases the secretion of norepinephrine secretion. This stimulates cognitive function in the prefrontal cortex, while also activating the hypothalamic-pituitary-adrenal axis which release hormones that inhibit the production of cortisol. Thus early-life malnutrition diminishes the ability to exhibit down regulation and handle stressful situations.

Despite the large body of evidence demonstrating these adverse effects, the economic causes and consequences of malnutrition in early childhood have received relatively little attention from economists.[3] There are a handful of studies that have assessed the impact on dimensions of schooling (see Victora *et al*., 2008, for a review) and a few that have adduced impacts on subsequent life outcomes such as lowered economic productivity in adulthood (Alderman, Hoddinott and Kinsey, 2006; Behrman, Alderman and Hoddinott, 2004; Horton, Alderman and Rivera, 2008). However, these claims are derived indirectly by stitching together results from a variety of sources. For example, Alderman, Hoddinott and Kinsey (2006) link their findings on the impact of malnutrition on schooling outcomes to separate studies that assess the returns to education in the Zimbabwean manufacturing sector. They make the very strong– and in the case of Zimbabwe clearly an incorrect – assumption that these returns are a good representation of what future earnings will be. The strongest evidence supporting the economic case for investing in nutrition comes from Hoddinott *et al* (2008). This showed that exposure to a randomized community-level nutrition intervention from age 0-3 years increased the wages of males in adulthood by more than 40 per cent but had no effect on women's wages. A thought experiment taking this finding to its logical conclusion quickly finds itself in uncomfortable territory. Does this imply that, on economic efficiency grounds, that investments that improve pre-school nutritional status should favor boys over girls, particularly in environments where (as is the case in rural Guatemala) there are cultural factors that limit

[3] For example, typing "economics AND education" into Google generates approximately 77 million responses. Searching "economics and malnutrition" generates about 500,000 responses and "economics AND height AND stunting" generates less than 70,000 items, a response rate of less than one-tenth of one percent of "economics and education."

women's access to the labor market? Such investments would exacerbate gender disparities, the reduction of which is often seen as a desirable outcome in their own right.

In the Sherlock Holmes story "The adventure of the Gloria Scott", a murderer is discovered with a "smoking gun" in his hand (Conan Doyle, 1895). The fundamental limitation of existing studies on the long term economic consequences of undernutrition is their lack of a "smoking gun". In this paper, we seek to rectify this weakness by demonstrating a causal link between early-life malnutrition and individuals' life course outcomes. The data demands for doing so are formidable. First, we need data on the anthropometry of individuals in early-life. Second, we need outcome data for these same individuals across a range of life course characteristics such as schooling, marriage or union formation, child birth, employment, health, poverty status, as measured in adulthood. Third, because early life malnutrition 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, p. 3382).

Four decades ago, between 1969 and 1977, two nutritional supplements (a high protein-energy drink and a low-energy drink devoid of protein, randomly assigned at the village level) were provided to preschool children in four villages in eastern Guatemala. Between 2002 and 2004, we traced and interviewed individuals who were now adults and who had been exposed to this intervention, collecting data on a wide range of life-course outcomes. As we demonstrate, these data satisfy the stringent requirements described above. Using Instrumental Variables estimation, we demonstrate that individuals better nourished in the first three years of life have dramatically better lives. They complete more schooling, have more success in the marriage market, and generally are in better health. As adults, they have better cognitive skills, earn higher wages and live in households with higher consumption levels.

Section 2 describes the data we use. In Section 3, we discuss modelling and identification issues. The effects of the early childhood nutrition status on outcomes across the life course are presented in Section 4. Section 5 reports some checks on robustness while section 6 provides conclusions.

2. Data: The 1969–77 INCAP Nutritional Intervention and follow-up studies

(a) Background

In the mid-1960s, protein deficiency was seen as the most important nutritional problem facing the poor in developing countries, and there was considerable concern that this deficiency affected children's ability to learn. The Institute of Nutrition of Central America and Panama (INCAP), based in Guatemala, was the locus of a series of studies on this subject, leading to a nutritional supplementation trial begun in 1969 (Habicht and Martorell, 1992; Read and Habicht, 1992; Martorell et al., 1995a). The principal hypothesis was that improved preschool nutrition would accelerate mental development. An examination of the effects on physical growth was included to verify that the nutritional intervention had biological potency (Martorell et al., 1995a). To test the principal hypothesis, 300 rural communities with 500–1000 inhabitants in eastern Guatemala were screened in an initial study to identify villages of appropriate compactness (so as to facilitate access to feeding centres—see below), ethnicity and language, 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. Before the intervention, the village pairs were similar in terms of a variety of nutritional, social, and economic outcomes, though it turned out slightly less so in terms of educational outcomes. Child nutritional status before the intervention, as measured by length at three years of age, was similar across villages (Habicht et al., 1995), and indicated substantial undernutrition with over 50% severely stunted—height-for-age z-scores less than -3 (Martorell, 1992).[4] Two of the villages, one from within each pair matched on population size (Conacaste and San Juan), were randomly assigned to receive as a dietary supplement a high protein-energy drink, *atole*. In light of concern that the social stimulation for children might also affect child nutritional and cognitive outcomes, thus confounding efforts to isolate the nutritional

[4] Z-scores are used to normalize measured heights and weights against those found in well-nourished populations. They are age- and sex- specific; for example, 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 -3 means three standard deviations of the reference population below the reference median.

effect of the *atole* supplement. To address this concern, in the two remaining villages, Santo Domingo and Espíritu Santo, an alternative supplement, *fresco*, was provided, under identical conditions. *Fresco* was a fruit-flavoured drink, which was served cool. It contained no protein and only sufficient flavouring agents and sugar for palatability, and had about one-third of the calories of *atole* per unit volume (Habicht and Martorell, 1992). The nutritional supplements were distributed in each village in centrally-located feeding centres 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. All residents of all villages also were offered high quality curative and preventative medical care free of charge throughout the intervention. Preventative services, including immunization and antiparasites campaigns, were conducted simultaneously in all villages. To ensure that the results were not systematically influenced by the characteristics of the health, research, or survey teams, all personnel were rotated periodically throughout the four villages, each of which was separated by at least 10 kilometres.

From 1969 to 1977, INCAP implemented the nutritional supplementation and the medical care. While the supplement was freely available to *all* village residents, the associated observational data collection focused on children between zero and seven years of age at any point during the intervention period.[5] 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 2392 children. Data collected at the child level included precise measurement of actual daily supplement intakes and periodic anthropometric measurements until the child reached seven years of age or until the survey data collection ended in 1977, whichever came first. Children in the sample, then, were all born between 1962 and 1977 and the type, timing, and length of exposure for particular children depended on their village and date of birth.[6] These data were complemented by a census of all

[5] 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., 1995a).

[6] This population has been studied extensively since the original survey, with particular emphasis on the impact of the nutritional intervention. Martorell *et al.* (2005) gives references to many of these studies; more recent examples include Behrman *et al.* (2009, 2010), Hoddinott *et al.* (2008) and Maluccio *et al.* (2009). For part of the

individuals and households in the four villages, conducted in 1967-68, 1975, 1987, 1996, and 2002, as well as a series of descriptive studies conducted in 1965–68 (Pirval, 1972), 1987–88 (Bergeron, 1992) and 2002 (Estudio 1360, 2002).

In 2002–04, a team of investigators, including the authors of this paper, undertook a follow-up survey targeting all participants in the 1969–77 survey, called the Human Capital Study (HCS). At that time, sample members ranged from 25 to 42 years of age. Of 2392 individuals in the original 1969–77 sample by the time of the 2002–04 HCS: 1855 (78%) were determined to be alive and known to be living in Guatemala: 11% had died—the majority due to infectious diseases in early childhood; 7% had migrated abroad; and 4% were not traceable. Of these 1855, 60% lived in the original villages, 8% lived in nearby villages, 23% lived in or near Guatemala City, and 9% lived elsewhere in Guatemala (Grajeda *et al.* 2005). Over a series of interviews, respondents provided schooling, marital and fertility histories, took tests of reading and non-verbal cognitive ability, provided information on income and consumption, underwent physical examinations, took fitness tests and provided blood samples to measure blood glucose and cholesterol levels.

(b) Descriptives: Pre-school nutritional status

During the supplementation trial between 1969 and 1977, children's height was measured at ages 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. Finer divisions for earlier ages were used to more accurately capture the more rapid growth that occurs during those ages. Not all of these individuals, however, were measured at the same ages or at any particular given age. The number of measurements greatest for children born in 1969 and 1970 (when the supplementation trial began) and fewest for children born in 1962 and 1963 (and who were therefore closer to the upper limit in terms of age at which children were measured) and for children born in 1976 and 1977, just before the intervention closed down.

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. There was also a round of data collection on a subset of the population carried out in 2007–08 (Melgar *et al.* 2008).

Using these data, we calculated height-for age Z-scores (HAZ) using reference standards (WHO, 2006). Figure 1 shows the pattern of height-for-age z scores as given by their mean values at different ages of measurement. In the early months of life there is a sharp drop in z scores for height-for-age which slows over time before leveling off and reaching a minimum at about 30 months of age. After this, it increases slightly, approaching -2.3 at age 72m. Also of interest is a matrix of correlation coefficients of these scores at different ages (Table 1). Relative to other correlation coefficients in this table, correlations between HAZ at ages 1 and 6 with HAZ at ages 30 months and higher are *relatively* low. Correlations at older ages tend to be high; for example the correlation with HAZ at 36m with HAZ at 30m is 0.924 and with HAZ at 42m, it is 0.951. Given this, suppose we use HAZ as measured at a "young age", say 12m. This will be unsatisfactory because (as Figure 1 shows) many children will experience continued deterioration in HAZ and impaired neurological development after this age, and because HAZ at these ages is *relatively* poorly correlated with HAZ at subsequent ages. If we use an older age (say 48m or older), HAZ will generally be higher than it was for the same child at an earlier age. Thus, this HAZ risks understating the damage malnutrition may have inflicted on the child in the first few years of life. These considerations suggest that we need to pick a "middle point" during the pre-school period. However, a single age would mean throwing away observations for which we do not have a HAZ at that particular age. This is informationally inefficient especially given that HAZ at certain ages are highly correlated with each other.

Given all this, we do the following. We start by estimating a child-level fixed effects regression where the dependent variable is HAZ and, in addition to the child fixed effects, we include dummy variables for the age categories at which the child is measured. We centre this around age 36m (ie age 36m is the reference age or "omitted category" in the regression reported in Table 2. The constant term is the mean of the child fixed effects. These summarize all the observable and fixed unobservable factors that affect child HAZ; 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. The results found in Table 2 are consistent with what we observe in Figure 1; estimated coefficients at ages close to 36 months have very small coefficients while those farther away (closer to 72m and closer to 1m) are larger.

We then generate a synthetic measure of HAZ at 36m for all children. To do so, we start with 880 cases where HAZ is measured at 36m and insert these into our synthetic value. Where HAZ at 36m is not known, we take the closest age at which height was measured, and using the regression results above, we calculate a predicted value for HAZ at 36m. This has a mean value of -2.91 with standard deviation 1.03. An attractive feature of this approach is that it minimizes the use of observations found in the tails of our distribution of measures by age and where we might expect measurement error to be highest. That said, we note that the absolute value of mean prediction errors is likely to be higher for measures in early life (less than 18 months) relative to those observed late in life (at ages 60 and 72m for example) because the trend in mean HAZ once past 30m is linear while the trend in HAZ prior to 30m is curvilinear. Mindful of this concern, section 5 reports the results of robustness checks using alternative measures of HAZ at 36m.

(c) Descriptives: Outcome variables

We describe these thematically. The means and standard deviations of the outcomes we will consider, along with a brief summary of their definition, can be found in Table 3.

Schooling-related outcomes

Individuals who had participated as children in the INCAP intervention and their spouses completed a schooling history. In interpreting these data, note that the formal educational system in Guatemala is divided into primary, secondary, and post-secondary education. Primary school comprises grades one to six, and children are expected to enrol in the calendar year in which they turn seven years old. Secondary school consists of five to seven grades, divided into two parts. The first three grades of lower secondary school (to grade nine) are the “basic” grades, and instruction is expected to provide academic and technical skills necessary to join the labour force. The fourth through seventh grades of upper secondary school are the “diversified” grades; students can choose from among a set of academic or vocational tracks.

Students who plan to continue to university finish upper secondary schooling in two years; other tracks at the diversified level usually take three years (World Bank, 2003). Virtually all members of the sample had at least one grade of schooling but only a small number (less than 3%) continued beyond secondary school. Apart from formal schooling, it was also possible to complete (primary and secondary school) grades via informal schooling such as adult literacy programs. Our overall measure of grades completed (maximum value, 12 grades) includes both types of schooling, though informal schooling is relatively uncommon in this population.

Reading comprehension was measured via a two-part standardized test. Respondents who reported having passed fewer than four grades of schooling, or those who reported four to six grades of schooling but could not correctly read aloud the headline of a local newspaper article, were first given a literacy test. Individuals who passed this literacy screen, or who reported more than six grades of schooling then took the Inter-American Series test vocabulary and reading comprehension modules (*Serie Interamericana* or SIA, for its acronym in Spanish): Level 2 for comprehension and Level 3 for vocabulary (approximately 3rd and 4th grade equivalents). The reading comprehension module had 40 questions and the vocabulary module 45 questions, yielding a maximum possible score of 85 points. Questions on the test become progressively harder. Those who did not pass the literacy screen pre-test were given a zero (18% of the sample).

All individuals were administered the Raven's Progressive Matrices test (hereafter Raven's test), an assessment of nonverbal cognitive ability (Raven et al., 1984). Raven's tests are considered to be a measure of eductive ability—"the ability to make sense and meaning out of complex or confusing data; the ability to perceive new patterns and relationships" (Harcourt Assessment, 2008). The test consists of a series of pattern-matching exercises with the respondent asked to supply a "missing piece" and with the patterns getting progressively more complex. We administered the first three of five scales (A, B, and C with 12 questions each for a maximum possible score of 36). Reading comprehension test (SIA) scores and nonverbal cognitive ability test (Raven's) scores are expressed as z-scores standardized to have mean 0 and SD 1 within the sample.

Union formation and marriage market outcomes

A marriage history module was administered separately to each husband and wife in the sample. "Marriage" refers to two individuals joining in union, usually (but not always), cohabitating and is not restricted to church or state-sanctioned marriages. The module consisted of a series of questions on: (a) age at first marriage, duration of first marriage, age at subsequent marriages and at marital dissolutions; (b) information on physical and financial assets brought to marriage; (c) and spouse's family background. Quisumbing et al (2005) provides further background and details.

Fertility

Men and women were asked about their marital history, number of live births, 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 construct the following outcomes: age at menarche, whether a woman gave birth before age 17, the interval (in months) between the first and second birth, the total number of pregnancies and whether a woman had experienced an infant or child death. Ramakrishnan et al (2005) provide further details and extensive descriptive statistics.

Health related outcomes

The field study team included two physicians and four fieldworkers who collected bio-medical data: measurements of anthropometry and body 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. The full set of outcomes we use are reported in Table 3; here we highlight three measures, in particular, are noteworthy. Based on the anthropometric data we collected, it is possible to construct an estimate of fat- free mass. Fat-free is a good measure of overall health human capital that affects work productivity because it reflects muscle and skeletal mass and thus the capacity to carry out work (McArdle, Katch and Katch, 1991). We also have a measure of isometric hand strength. Using a Lafayette dynamometer, subjects were asked to exert a maximal and quick handgrip twice and maximal values of the dominant and non-dominant hands were recorded. Handgrip strength is highly correlated with total strength of 22 other muscles of the body (de Vries, 1980). Finally, another measure of physical work capacity was constructed: predicted maximum oxygen consumption (VO2max). It measures the capacity of individuals to deliver oxygen to muscle while doing physical work. Stein et al (2005) provides a detailed description of how these data were collected and how outcomes were measured.

Labor Market Activities

During HCS individuals were interviewed about all of their income-generating activities. Topics covered included: a) wage labor activities for every wage job (type of occupation; wages and fringe benefits; description of the employer; and hours, days and months worked); b) all agricultural activities (amount of land cultivated; crops grown; production levels and values; use of inputs; and hours, days and months worked); and c) non-agricultural own-business activities (type of activities; value of goods or services provided; capital stock employed; and hours, days and months worked). Virtually all men (98%) and most women (69%) were engaged in some sort of own-account income-generating activity. In the year prior to the interview, 79% of men were working for wages (with more than half of these in unskilled occupations), 42% in own-account agriculture and 28% in own-account non-agricultural business. A third of women were working for wages (with the majority in unskilled occupations) and a third in own-account non-agricultural business, and 20% were in own-account agriculture. 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. These data are used to generate annual hours worked. In the case of wage labor, individuals were asked to report gross earnings, earnings nets of taxes and social security deductions as well as additional payments such as bonuses, transport and food, for the time unit (hourly, daily, weekly and so on) most relevant to their job, for each job they had held. These data, together with net income earned from own-account agricultural activities, non-agricultural own-business activities and hours worked were used to construct a measure of income earned per hour worked. Hoddinott, Behrman and Martorell (2005) provide a detailed discussion of these data.

Consumption and poverty

The Human Capital Survey include an expenditure survey which provided information on food and nonfood expenditures in the household in which the respondent currently resided and a community level food price survey. Using these data, and the method outlined in Maluccio, Martorell and Ramírez (2005a), we construct a measure of per capita household consumption. We can compare these data against a poverty line constructed for Guatemala, again see Maluccio, Martorell and Ramírez (2005a) for details.

3. Modelling and identification

To clarify the identification issues associated with the focus of this paper, consider a vector of outcomes, Y for individual *i* which are related to early-life nutrition in the following way:

$$\mathbf{Y}_i = \beta \cdot HAZ_i + \boldsymbol{\gamma}' \cdot \mathbf{X}_i + \upsilon_i \qquad (1)$$

For example, an element of $\mathbf{Y}_i$ could be W_i, the hourly wages of person *i* in adulthood. HAZ_i is a measure of pre-school nutritional status - height-for-age z score, $\mathbf{X}_i$ is a vector of control variables with associated parameters $\boldsymbol{\gamma}$ and υ_i is a disturbance term. The parameter of primary interest to us is β. If $E(HAZ_i\ \upsilon_i) \neq 0$, β will be biased. It is not difficult to think of reasons why such a correlation could exist. For example, parents of these individuals who have superior social networks may be wealthier and may also be better placed at helping their children find high paying jobs. Given this, a critical issue for this paper is whether we can credibly identify the impact of $HAZ_{i.}$

Issues of identification have attracted considerable recent discussion and controversy in economics. One school of thought argues that a randomized control trial (RCT) design represents the most powerful and plausible way of identifying impact. In the context of this paper, an RCT would require malnourishing randomly selected pre-school children and both following them and a control group of non-malnourished children for the next 30-40 years. Such an approach is both impractical and unethical. Lee and Lemieux (2010) argue that in important ways, a Regression Discontinuity Design (RDD) can be considered as being "more similar" to a randomized design (Lee and Lemieux, 2010, p. 302). Lee and Lemieux note that in the presence of heterogenous treatment effects, RDD produces a weighted average treatment effect where the weights are proportional to the ex ante likelihood that an individual is close to the threshold. As they note, if these weights are highly varied – as is likely to be the case here – the RDD measure of impact will be very different from the treatment effect. With RCT and RDD ruled out, we adopt an Instrumental Variables approach. In doing so, we are aware of both its strengths and limitations, most notably that "the estimated treatment effect is applicable to the sub-population whose treatment was affected by the instrument" (Lee and Lemieux, 2010, p. 292). In light of Deaton's critique of local average treatment effects (LATE), we carefully specify and justify our choice of instruments. We take seriously Angrist and Pischke's (2010) and Stock's (2010) argument about the importance of establishing credible identification. Finally, following the argument laid out in Leamer (2010), we perform sensitivity analyses on our findings; these are found in section 5.

To understand the logic behind our choice of instruments, consider the model outlined in Behrman and Hoddinott (2005). A child's nutritional status (HAZ_i) is assumed to appear as an argument in the welfare function of the households in which they reside (Behrman and Deolalikar, 1988; Strauss and Thomas, 1995). Welfare is assumed to increase as nutritional status improves, though possibly at a diminishing rate. Decisions that parents make about devoting resources to the children's nutrition and health are constrained in several ways. There are resource constraints reflecting income and time available as well as prices faced by households. There is also a constraint arising from the production process for health outcomes, including nutritional status. This constraint links 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, locality characteristics such as the presence of preventative and curative health facilities and the prevalence of infectious diseases, 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 health demand function takes the general form:

$$HAZ_{it} = h_t(C_i , M_t , W_t , P_t , Z_{it}, Z_i) \quad (2)$$

where C_i is a vector of child characteristics such as sex, and genotype (for example, growth potential), M_t is a vector of characteristics of the principal care giver observed at time *t*, W_t captures household wealth, and P_t is a vector of all relevant prices. *Z* is a vector of health, sanitation and environmental characteristics in the locality in which the child lives that are assumed to influence health. Some of these, Z_i, are time invariant while others, Z_{it}, vary over time.

Given (2), our identification strategy relies on two core ideas: the existence of cohort and location specific transitory shocks that we assume are independent of individual characteristics[7] – ie elements of Z_{it} – and random variation in genotype that are found in the vector C_i. Our cohort and location specific transitory shocks include: exposure to the INCAP intervention between the ages of 0 and 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 an earthquake measuring 7.5 on the Richter scale that shook Guatemala in February 1976; and whether there was a government health post in the individual's village-of-residence when they were two years of age. Our measures of variation in genotype include the logarithm of maternal height (Sahn, 1990) and whether the individual was a twin.

Table 4 reports results of estimating a variant of (2). While not exactly the same as the "first stage regressions" that we estimate when we consider our various life-course outcomes,[8] it provides a useful assessment of the relevance of our instruments. Specifically, we represent *C* by the individual's sex, the inclusion of a dummy variable denoting that the individual is a twin and the log of mother's height; M_t by completed grades of mother's schooling; W_t by a wealth index; and P_t by a set of year-of-birth dummy variables that can be thought of more generally as

[7] This is analogous to the approaches described by Imbens and Angrist (1994), Card (2001) and Alderman, Hoddinott and Kinsey (2006).
[8] When we specify a particular outcome, we will also specify control variables relevant to that outcome but these control variables will differ across outcomes.

a set of variables that capture all events (including movements in all prices) common to a given birth cohort. Z_i is denoted by a set of location-of-birth dummy variables while Z_{it} captures the transitory shocks noted above.

There are several attractive features of the results shown in Table 2. First, a number of our proposed instruments are causally related to our endogenous variable. Exposure to the intervention between birth and 36 months and exposed to atole and maternal height both increase height-for-age at 36 months while being a twin reduces height. These variables are all statistically significant. Being exposed to the 1976 earthquake in early life reduces height but this is not precisely measured; the p-value for this coefficient is equal to 0.15. Exposure to the intervention between birth and 36 months and having a health centre in the village of birth, however, have no meaningful effect on height given age. An F test rejects the null hypothesis that these proposed instruments are jointly zero. Stock (2010) notes that, "If this F-statistic is large – a 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" (Stock, 2010, p.87). At 16.52, our F statistic comfortably passes this rule of thumb.

In addition to being correlated with height for age, an attractive feature of these variables is that they meet a key criterion laid out in Deaton's (2010) critique of IV methods; namely that they are derived from a formal model rather than being specified *ad hoc*. But we recognize that any half-decent economist sitting in a seminar room – or indeed a bar for that matter – could quibble with any of these identifying variables, *viz*:

- Exposure to the intervention could have had an income effect that persisted after the supplementation trial ended in 1977;[9]

[9] We think this is unlikely. First, the behaviour 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 centres, 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. 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% 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 both *atole* and *fresco* villages.

- The earthquake could have had long lasting effects – for example on school availability and quality or on income generating opportunities;[10]
- The establishment of a government-run health post could reflect a process of endogenous program placement;[11]
- 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 intergenerationally;
- The proportion of the sample that are twins is so small (less than five percent) that the local average treatment effect (LATE) of this identifying instrument is not likely to be of interest

In light of these legitimate concerns, we subject our instruments to a battery of tests described below. Further, we return to this issue in our discussion of robustness tests found in section 5.

4. The impact of malnutrition on schooling, marriage, fertility, health, wages, labor force participation and poverty

We now turn to the results of estimating (1) for outcomes that span the life course of these individuals. For each set of outcomes, we report the results of two functional form representations of pre-school nutritional status; height-for-age z scores; and a dummy variable equaling one if the individual was stunted at age 36 months, zero otherwise. Note that 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 (ie switching an individual from being stunted to not being stunted) means that an

[10] A priori, we are not convinced by such a claim. 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 good produced in particular villages at particular times.

[11] Because we include village-of-birth dummy variables in all specifications, this line of criticism is restricted to claiming that there were time varying factors that lead to differences in the timing of the establishment of these health posts. We note that the three separate qualitative studies carried out in these villages, all of which discussed changes in village infrastructure, reported no such evidence (Pirval, 1972; Bergeron, 1992 and Estudio 1360, 2002).

improvement in nutritional status has lead to an improvement in outcomes. We first report these results for the full sample, then separately for females and males. Throughout 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. As a rule of thumb, where our test statistic has a value of 6.3 or higher, we reject at a 5% significance level the hypothesis that the instruments are weak, where weak in this case means having bias in the IV results that is larger than 20% of the bias in the OLS results. We also report the Hansen J statistic for overidentification where the null hypothesis is that the overidentifying restrictions are valid (i.e., that 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, this gives us some confidence in the power of this specification test. Standard errors robust to heteroscedasticity and clustered at maternal level. Where outcomes are expressed as 0/1 variables, we estimate linear probability models so as to be able to compute the IV test statistics. To save space, we do not report the full set of results. The footnotes to these tables report the control variables that we include and full results are available on request.

(a) Schooling

Table 5 reports the results of estimating the impact of pre-school nutritional status on schooling outcomes. Table 5A reports these for the full sample; Tables 5B and 5C report this separately for women and men respectively. In the estimates of equation (1) for schooling related outcomes, our second stage control variables are: subject sex and birth year dummy variables; maternal age and education; paternal education; parental wealth index; school quality at age 7 and 13 (whether school building is permanent structure and student teacher ratio); distance to feeding center; electrification of village at age 2; and village of origin. In so doing, our estimates control for common cohort effects, unobserved fixed effects associated with place of birth, and parental characteristics and time-varying location characteristics that might be correlated with these schooling outcomes. Looking across the results of the Kleibergen-Paap (KP) test statistics, we see that we do well in terms of the relevance of our instruments, particularly when pre-school nutrition is represented by the height-for-age z score. In all specifications, we fail to reject the null hypothesis that the instruments are uncorrelated with the second stage schooling outcomes and in nearly all cases, the prob values of the Hansen J test are quite large.

These results show that there is a direct effect of poor pre-school nutritional status on age at entry, the age at which an individual leaves school and the number of grades completed.[12] When nutritional status is expressed in terms of stunting, the effect on grade attainment is large – a loss of nearly four grades of schooling compared to some one who was not stunted. The impact on grade attainment is the same for males and females; the impact on the age at which some one leaves school appears to be driven primarily by the female sub-sample.

The most striking results, however, are for the reading/vocabulary and Raven's tests administered in adulthood. These show that poor nutritional status in early life is casually related to poorer outcomes on these dimensions of cognitive skill. The magnitude of these effects is large. They are consistent with the literature cited in the introduction to the paper on the neurological consequences of malnutrition.

(b) Marriage market

Table 6 reports the impact of pre-school nutrition on success in the marriage market, doing so separately for women (Table 6A) and men (Table 6B). All results control for age, maternal age and education, paternal education, parental wealth index, electrification of village at age 2 and village of origin. We do well in terms of the relevance of our instruments as measured by the Kleibergen-Paap (KP) test statistics, particularly when pre-school nutrition is represented by the height-for-age z score. Out of 14 specifications, we fail to reject the null hypothesis that the

[12] Other studies include Alderman, Hoddinott and Kinsey (2006), Daniels and Adair (2004) and Mendez and Adair (1999).

instruments are uncorrelated with the second stage schooling outcomes and in 13 of them and in nearly all cases, the prob values of the Hansen J test are quite large.

Women better nourished in early-life marry men who are older. The parameter estimate reported in the bottom panel of Table 6A implies that is a woman who was not stunted at age 36m marries a man who is five years older than herself. Since age and consumption levels are correlated in this sample, this suggests that females better nourished as pre-schoolers make a better match in the marriage market. A cautionary note, however, arises from the fact that not only do women marry older men, but they also marry men 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 may marry into better off households they may also be somewhat more disadvantaged in terms of bargaining over resources within those households.

Men with better pre-school nutritional status also do better in the marriage market, but along different dimensions. They marry women with more schooling and who are slightly taller; Behrman et al (2010) show that women with more schooling earn higher wages.

Finally, in preliminary work we assessed whether early-life nutrition affected 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. We did not find statistically significant impacts.[13]

(c) Fertility

Results are reported in Table 7. We control for birth year, maternal age and education, paternal education, parental wealth index, electrification of village at age 2 and village of origin. Women were undernourished as pre-schoolers are more likely to give birth before age 17 and have more children. The latter effect is large. “Switching” a woman from being stunted to not being stunted would, conditional on her age, reduce the number of pregnancies she has by two. Our results are consistent with the finding above that better nourished females in early life

[13] These are not reported but are available on request.

complete more grades of schooling and the extensive literature that shows that better educated women have fewer pregnancies.

(d) Health

Using the extensive information we have on health outcomes, we consider the impact of early life undernutrition on the following anthropometric outcomes in adulthood: log height, log Body Mass Index (BMI), whether the individual is overweight or obese (defined as having a BMI > 25.0), whether the individual is obese (defined as having a BMI >30.0), waist-hip ratio, and head circumference. We consider three measures of physical strength: log fat free mass, hand strength and maximal oxygen consumption. We also examine outcomes related to risks associated with cardio-vascular and other chronic diseases: Low-density lipoprotein (LDL) cholesterol, the ratio of total cholesterol to High -density lipoprotein (HDL) cholesterol, blood pressure, and plasma glucose. Lastly, we consider metabolic syndrome (METS), a range of risk factors associated with increased risk of stroke, diabetes and coronary heart disease.

Results are reported in Table 8. Second stage controls are subject sex and birth year dummy variables, maternal age and education, paternal education, parental wealth index, electrification of village at age 2 and village of origin. 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. 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. Results for the full sample are found in Table 8A with results for females (Table 8B) and males (Table 8C) reported subsequently.

Individuals with better pre-school nutritional status are taller and stronger as adults, the latter as measured by hand strength and fat-free mass. While they have slightly higher body mass and are more likely to be overweight or obese, impacts on these outcomes are not precisely measured. Results on anthropometry do not differ meaningfully between men and women. Women are somewhat more likely to be hypertensive or pre-hypertensive if they had higher HAZ scores at age 36m. Males were more likely to be diabetic or pre-diabetic.

(e) Labor market outcomes

We consider five labor market outcomes: log hourly earnings; log hours worked; log earned income; years employed doing skilled labor; whether the individual is currently employed doing skilled work; and whether the individual operates their own business, fulltime.[14] Results are reported in Table 9, first for the full sample and then for females and males. Specification tests indicate that we can reject the null that our instruments are weak but not reject the null that the overidentifying restrictions are valid.

Better nutritional status in early life is causally linked to higher hourly earnings in adulthood. Being stunted at age 36m has a large adverse impact of economic productivity in adulthood, reducing earnings by 62.5 percent. Individuals who were better nourished as pre-schoolers are more likely to work in higher-paying skilled labor or white collar work and are somewhat more likely to operate their own business though this latter effect is imprecisely measured. These results are consistent with processes by which undernutrition in early life leads to cognitive impairments which limited schooling attainment and the acquisition of cognitive skills both of which are rewarded in the Guatemalan labor market, see Behrman et al (2010).

Impacts on success in the labor market differ by sex. While there are positive effects on women's economic productivity, these are imprecisely measured. Females who were better nourished at age 36m are more likely to work in skilled employment and were 33 percentage points more likely to have an independent source of income through own business activities. Productivity impacts on males are large and statistically significant. A one standard deviation increase in HAZ at age 36m increases male wages by 21.5 percent. Stunting at age 36m lowers hourly earnings by a massive 65.7 percent in adulthood.

(f) Consumption and poverty

In Table 10, we consider the impact of pre-school nutritional status, expressed both as HAZ and as whether the individual was stunted, on the consumption levels of the household in which

[14] In preliminary work, we also considered migration status as an outcome but found no significant effects.

the individuals who participated in the intervention reside as adults. We also consider the impact on the likelihood that the household has consumption levels above or below the poverty line. In these regressions, the second stage estimates control for sex, birth year, paternal education, parental wealth index, school quality at age 7 (whether school building is permanent structure and student teacher ratio), whether mother or father had died before subject turned 15, tobacco market collapse at age 15, electrification of village at age 2 and village of origin. We estimate these for the full sample and separately for men and women. The test statistics for instrument relevance are good and we do not reject the null hypothesis that the overidentification assumptions are valid.

Men and women with better nutritional status in early life live, as adults, in better-off households. A one standard deviation increase in HAZ increases household per capita consumption by 18 percent. Since consumption is measured at the household level, all members, not just those of the individual who was better nourished, are better off provided that there are not dramatic intra-household inequalities in consumption. Put another way, the benefits to improving an individual's early-life nutritional status are not necessarily confined to that individual; they spill-over to other household members. Individuals who are stunted live in households with lower consumption levels and are 30.9 percent more likely to live in a household that is poor.

5. Checks on robustness

(a) Instrument validity

A critical issue for this paper is whether we can credibly identify the impact of HAZ_i. We see the notion of "credibly" as having several inter-related meanings. First, are our instruments derived from a credible model of our endogenous variable or are they ad hoc? This notion of credibility is discussed in section 3. Second, are our instruments relevant so that we can have confidence in the inferences that we draw from our IV estimates? As shown in Table 4 and in the results presented in section 4, we satisfy concerns regarding relevance. However, there are two further meanings that we have not considered. The first issue, raised by Lee and Lemieux (2010) and by Deaton (2010), that the "the estimated treatment effect is [only] applicable to the sub-population whose treatment was affected by the instrument" (Lee and Lemieux, 2010, p. 292). A good example of why this issue is of concern here is the following. Suppose the identification of our results is driven by whether an individual is a twin. If this is the case, while our results are valid in a statistical sense (that is, they pass all the necessary specification tests), they are not especially meaningful given that twins make up such a small proportion of this (or indeed any other) population. Second, Leamer describes credible inferences in terms of the outcome of sensitivity analyses that "separate fragile inferences from sturdy ones" (Leamer, 2010, p. 37).

Because we use several variables, not just one, to identify the impact of pre-school nutritional status on later life outcomes, is possible to assess the sensitivity our results to the inclusion/exclusion of particular instruments. As described in Table 11, we consider eight alternative instrument sets. We report our results in Figures 2, 3 and 4 for three key findings: impacts on reading/vocabulary scores, hourly earnings (males) and household consumption. The bottom rows of Table 11 report three specification tests: the F statistic for the excluded instruments in the first stage regression, the Kleibergen-Paap (KP) test statistic that the instruments are weak and the prob values for the Hansen J test. In each figure, the triangle represents the point estimate while the top and bottom ends of the bars give the end points of the 95 percent confidence interval.

The first bar in each figure is the results obtained using the full set of instruments. Note that alternatives 1, 2 and 3 (dropping different combinations of the twins instrument, the negative earthquake shock and the positive health infrastructure shock) has no meaningful effect on the magnitude of the parameter estimate and only slight variations in the size of the 95 percent confidence interval. Alternatives 5 and 6, where we drop exposure to the intervention between 0 and 36 months, exposure to the intervention between 0 and 36 months and living in an *atole* village and, in alternative 6, whether the individual is a twin also produces very similar parameter estimates to those obtained with the full set of instruments and those found in alternatives 1, 2, and 3, but with slightly larger confidence intervals. In these five alternative specifications, our instrument set easily meet the relevance criteria as measured by the F statistic and we can reject the null hypothesis that the instruments are weak. In addition, we do not reject the null that the overidentifying restrictions are valid. If we drop mother's height (alternative 4) in two cases, hourly earnings (males) and household per capita consumption, we obtain *larger* estimates of impact but lose precision as seen by the wider confidence intervals. In the case of reading scores, the parameter estimate is similar to that obtained from the full set of instruments and the confidence interval is increased. Consistent with this, the "relevance statistics" are lower (but still reasonable) and we continue to not reject the null regarding the overidentifying restrictions.

Alternative 7 includes only exposure to the intervention between 0 and 36 months, exposure to the intervention between 0 and 36 months and living in an *atole* village as instruments. We get much different point estimates, much larger for reading scores and hourly earnings (males) and smaller for household per capita consumption. We also obtain, comparatively speaking, much wider confidence intervals. If we add back in exposure to the earthquake and being a twin (alternative 8), we reduce these intervals markedly, improve our relevance statistics and do not reject the null hypothesis that the overidentifying restrictions are valid.

For reading outcomes, seven of the eight alternative instrument sets produce parameter estimates very close to that obtained by our base set. In all cases, the instrument sets pass tests of relevance and do not reject the test of overidentification. The minor difference between these lies in the size of the estimated confidence intervals. If we only use exposure to the intervention, and exposure interacted with *atole*, we get a much larger estimate of impact but one that is imprecisely measured. For hourly wages (males) and household consumption, five of the eight alternative instrument sets produce parameter estimates very close to that obtained by our base set. All instrument sets pass tests of relevance and do not reject the test of overidentification. There are minor variations across these instrument sets in terms of the size of the estimated confidence intervals. Alternatives 4 and 8, both of which exclude mother's height, generate much higher estimates of impact but also wider confidence intervals. Only using exposure to the intervention, and exposure interacted with *atole* generates imprecisely measures of impact. Based on these results, we conclude that the estimates reported in Tables 5 to 10 are robust to concerns regarding fragile inferences and concerns regarding the generality of the LATE estimates.

(b) Attrition

Despite the considerable effort and success in tracing and re-interviewing participants from the original sample, attrition in this sample is substantial.[15] 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 (e.g., migration versus failure to interview someone who was located). What is of ultimate concern in this analysis is not the level of attrition, however, but whether, and to what extent, the attrition invalidates the inferences we make using these data. We address concerns about sample attrition bias in three ways. First, in the specifications already shown, we include covariates, many of which, 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 et al., 1998b).

Second, we compare nutritional outcomes measured in the 1970s for those eventually re-interviewed in HCS and those not re-interviewed. Average height-for-age measured at 48 months of age and 72 months of age are remarkably similar between those who attrited and those who did not. Height-for-age z-scores for the two groups are all within 0.006 of one another (mean for 48-month olds is -2.074, SD 1.03 and mean for 72-month olds is -1.666, SD 1.028) and the lowest p-value on a t-test with unequal variances is p=0.91. There does not appear to be any obvious selection between those interviewed or not based on early-life nutritional status.

[15] 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 2392. 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 under 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 biases that such a correlation might create.

Finally, we implement the correction procedure for attrition outlined in Fitzgerald, Gottschalk, and Moffitt (1998a, 1998b). We first 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 educational outcomes outlined in equation (1) and not whether educational outcomes 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 five outcomes where we perceive our results to be especially striking: SIA z scores, the differential (for women only) in ages between subjects and their spouses, number of pregnancies, log hourly earnings (for men only), and per capita household consumption. Table 12 shows that application of these weights generates trivial 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. 2001a) our results do not appear to be driven by attrition biases.

(c) Alternative measures of HAZ

In section 2, we described how we constructed our measure of Height-for-Age z scores at age 36m. We described this as a synthetic measure that used 880 actual values for HAZ (those available where the individual was actually measured at 36m) and estimated values where we took the closest age at which height was measured, and using results from a child fixed-effects regression, calculated a predicted value for HAZ at 36m. We noted that this minimizes the use of observations found in the tails of our distribution of measures by age where we might expect measurement error to be highest. We also noted that prediction errors were likely to be higher for measures in early life (less than 18 months) relative to those observed late in life (at ages 60 and 72m for example) because the trend in mean HAZ once past 30m is linear while the trend in HAZ prior to 30m is curvilinear.

In light of this concern, we report results based on two other approaches to calculating HAZ. In the first alternative approach, we simply drop all children from whom these synthetic values are generated solely from HAZ measures when children were less than 24m. In the second alternative approach, we only use individuals for whom we have actual measures on HAZ between the ages of 30 and 42 months. We start with HAZ at age 36m for which we have 880 observations. If we do not have HAZ at ages 36 or 42m, we use HAZ at age 30 months. This adds an additional 109 observations. If we do not have HAZ at ages 36 or 30m, we use HAZ at 42m. This adds an additional 81 observations. If we do not have HAZ at age 36m, but have it for both 30m and 42m, we use the measure that was taken closest to 36m. (Recall that each targeted age, there is a small range around this when these measurements were collected). This adds a further 34 observations, yielding a total of 1,104 subjects with HAZ measured between 30 and 42 months. We estimate their impact on five outcomes: SIA z scores, the differential (for women only) in ages between subjects and their spouses, number of pregnancies, log hourly earnings (for men only), and per capita household consumption and use the same specifications as those used in the results reported previously.

Results are reported in Tables 13 and 14. For both alternatives, test statistics for relevance and over-identification continue to be satisfactory. Because we have smaller sample sizes, not surprisingly, our parameter estimates are measured with less precision. Apart from the reading/vocabulary scores which are slightly lower in these alternative specifications (but still statistically significant), results obtained in Tables 13 and 14 are similar to those found in Tables 5 to 10 for these outcomes. We conclude that our results are robust to alternative approaches to specifying HAZ.

6. Summary

This paper examines the impact of being malnourished as measured by height for age at 36m over the life course of an individual. We overcome the formidable data demands this requires by tracing individuals who participated in a nutrition 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: schooling; the marriage market; fertility; health; wages and income; and poverty and consumption in adulthood. We account for the endogeneity of pre-school nutritional status, using transitory shocks experienced as a pre-schooler and random variation in genotype as instruments.

Our results indicate that higher height for age at age 36m is causally linked to the attainment of more schooling and on higher scores on cognitive tests in adulthood. Men earn higher wages and women are more likely to have independent sources of income from own business activities. Women stunted at age 36m have 1.9 more pregnancies and are more likely to give birth before age 17. Better nourished preschoolers are taller as adults and have more fat free mass and greater hand strength. Being stunted at age 36m increases the likelihood of living in a poor household in adulthood by 31 percentage points.

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 the same point in time, 36m. 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 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 HAZ.

Individuals better nourished in the first three years of life have dramatically better lives. They are better schooled, have greater cognitive skills, have higher wages and live in households with higher levels of 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 undernutrition in the developing world.

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Table 1: Correlation matrix for HAZ by selected ages

	HAZ at _ months								
	1	6	12	18	24	30	36	42	48
HAZ at _ months									
1	1.0000								
6	0.6319	1.0000							
12	0.5293	0.8407	1.0000						
18	0.4708	0.7799	0.8894	1.0000					
24	0.4525	0.7309	0.8367	0.9061	1.0000				
30	0.4245	0.6573	0.7660	0.8385	0.8922	1.0000			
36	0.3917	0.6743	0.7575	0.8224	0.8875	0.9241	1.0000		
42	0.4051	0.6829	0.7641	0.8068	0.8524	0.9171	0.9513	1.0000	
48	0.4017	0.6179	0.7090	0.7663	0.7982	0.8696	0.9045	0.9385	1.0000

Table 2: Child level fixed effects regression of HAZ

Dummy variable for age range within which child measured	Coefficient	T statistic
0.01-1.5m	1.2893***	36.41
1.51-2.5m	1.6528***	2.91
2.51-3.5m	1.0329***	30.48
3.51-4-5m	0.7787***	3.52
4.51-5.5m	1.3679***	4.93
5.51-6.5m	0.8790***	26.25
6.51-7.5m	0.8063**	2.65
7.51-8.5m	0.6804*	1.77
8.51-9.5m	0.5601***	16.97
9.51-10.5m	0.4994	1.62
10.51-11.5m	0.0206	0.09
11.51-13.5m	0.2695***	8.13
13.51-16.5m	0.0071	0.21
16.51-19.5m	0.0331***	-4.26
19.51-22.5m	0.0333***	-5.78
22.51-25.5m	0.0330***	-8.97
28.51-31.5m	0.0331***	-5.00
39.01-45.0m	0.1505***	4.54
45.1-51.0m	0.2920***	8.72
57.01-66.0m	0.5190***	14.72
66.01-78.0m	0.6400***	17.26
78.01-max	0.7760***	19.52
Constant	-2.6852***	-113.13

Notes: * significant at the 10% level; ** significant at the 5% level; *** significant at the 1% level.

Table 3: Outcome variables

	Variable	Definition	Mean	Standard Deviation
Schooling related outcomes	Age start school	Age (in years) when individual commenced attending primary school	6.80	1.09
	Repeated primary grade	=1 if individual repeated a grade of primary school	0.48	0.50
	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
	Age left school	Age (in years) when individual stopped attending school	12.51	2.95
	Highest grade attained	Highest grade of schooling attained, maximum value is 12	4.86	3.22
	SIA z score	Inter-American Series test score of reading and vocabulary, standardized with mean 0 and SD 1 within the sample	0	1
	Raven's z score	Raven's Progressive Matrices test score, standardized with mean 0 and SD 1 within the sample	0	1
Marriage market outcomes, females	Husband's age	Age (in years) of husband at time of union formation	36.24	7.31
	Husband's grades of schooling	Husband's highest grade of schooling attained	4.93	3.57
	Husband's Height	Husband's height (cm)	162.46	5.72
	Husband's family's land holdings	Land owned by husband's family in manzanas (1 manzana = 1.68 acres)	2.79	9.52
	Age differential	Husband's age – wife's age	3.69	5.84
	Education differential	Husband's highest grade attained – wife's highest grade attained	0.89	3.50
	Family's land holdings differential	=1 if husband's family has more land than wife's family at time of union	0.34	0.47
Marriage market outcomes, males	Wife's age	Age (in years) of wife at time of union formation	30.41	5.68
	Wife's grades of schooling	Wife's highest grade of schooling attained	4.45	3.20
	Wife's Height	Wife's height (cm)	150.52	5.19
	Wife's family's land holdings	Land owned by wife's family in manzanas (1 manzana	4.99	70.25

		= 1.68 acres)		
	Age differential	Wife's age –husband's age	-2.15	4.49
	Education differential	Wife's highest grade attained – husband's highest grade attained	-0.82	3.39
	Family's land holdings differential	=1 if wife's family has more land than husband's family at time of union	0.20	0.40
Fertility-related outcomes	Age at menarche	Age (years) of first menstrual cycle	13.60	1.39
	First birth before 17	=1 if woman gave birth before age 17	0.14	0.35
	Spacing 1st and 2nd birth	Duration of time between first and second birth (years)	3.02	1.98
	Number of pregnancies	Number of pregnancies including miscarriages and stillbirths	3.08	2.18
	Any infant deaths	=1 if mother had child who died before attaining 1y	0.15	0.36
	Any child deaths	=1 if mother had child who died before attaining 5y	0.17	0.38
Health related outcomes	Log Height	Log of: Height measured in cm	5.05	0.05
	Log BMI	Log of: Body weight (kg) divided by the square of height (m)	3.24	0.17
	Overweight or obese	= 1 if Body Mass Index >25.0	0.52	0.50
	Obese	= 1 if Body Mass Index >30.0	0.17	0.37
	Waist-Hip ratio	Ratio of circumference of the waist to the hip	0.94	0.06
	Head circumference	Distance (cm) around the largest part of the head	53.67	1.73
	Log Fat Free Mass	Log of: Fat free mass. Fat free mass = body mass – fat mass.	3.79	0.17
	Log Hand Strength	Log of: Strength of dominant hand measured in newtons	3.41	0.29
	Log VO2 max	Log of: Maximal oxygen consumption (mL/kg/min) calculated from exercise recovery rates following administration of step-test	2.82	0.48
	LDL cholesterol	Low-density lipoprotein cholesterol measured in mg/dL	90.67	26.95
	Total cholesterol/HDL	Ratio of (LDL cholesterol plus HDL cholesterol) to High -density lipoprotein (HDL) cholesterol	4.62	1.46
	Systolic blood pressure	Systolic blood pressure measured in millimeters of mercury (mm Hg) using a sphygmomanometer	112.10	12.93
	Diastolic blood pressure	Diastolic blood pressure measured in millimeters of mercury (mm Hg) using a sphygmomanometer	70.96	9.52
	Hypertensive or prehyper-	=1 if ratio of systolic to diastolic blood pressure is	0.31	0.46

	tensive	greater than 120-139 / 80-89		
	Blood glucose level	Plasma glucose concentrations measured in mg/dL	93.83	27.80
	Diabetic or pre-diabetic	= 1 if plasma glucose concentrations were between 110 and 125 mg/dL (pre-diabetic) or greater than 126mg/dL (diabetic)	0.21	0.41
	Metabolic syndrome	=1 if blood tests show presence of diabetes mellitus, impaired glucose tolerance, impaired fasting glucose, and two of: Blood pressure: ≥ 140/90 mmHg; BMI >30; waist: hip ratio >0.90 (males); waist: hip ratio >0.85 (females); triglycerides ≥ 1.695 mmol/L and low levels of HDL cholesterol (WHO, 1999)	0.31	0.46
Labor market outcomes	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)	1.93	0.89
	Log hours worked	Log of: Hours worked in the previous 12 months	7.25	1.18
	Log earned income	Log on: Net income from wage work, own-account agriculture and own-business activities (Conditional on earning any income in the previous 12 months)	9.20	1.53
	Years skilled labor	Number of years individual has worked in clerical, administrative, technical, or professional positions	1.65	3.40
	Skilled labor or white collar work	=1 if individuals currently works in clerical, administrative, technical, or professional positions, conditional on working for wages	0.37	0.48
	Worked on own business, fulltime	=1 if individual operates own business for more than nine months per year	0.23	0.42
Consumption and poverty	Per capita household consumption	Log of: Per capita household consumption	8.76	0.75
	Household is poor	=1 if per capita household consumption is below the poverty line	0.29	0.45

Table 4: Correlates of Height for Age z score, 36 months

	Covariate	Parameter estimate	Standard Error
Transitory shocks	Exposure from birth to 36 months	-0.077	(0.192)
	Exposure from birth to 36 months × *atole*	0.250**	(0.107)
	“Earthquake” (subject born in 1974, 1975 or 1976)	-0.213	(0.140)
	Ministry of Health post established when person was 2y	0.083	(0.143)
Random variation in genotype	Twin (=0 if twin missing)	-0.910***	(0.227)
	Log mother’s height	9.929***	(1.122)
Other controls, *C*	Male	-0.086*	(0.051)
Other controls, *M*	Grades attained, mother	0.004	(0.022)
Other controls, *W*	Initial wealth index	0.168***	(0.043)
Other controls, cohort effects	Birth year, 1962	-0.812***	(0.188)
	Birth year, 1963	-0.692***	(0.149)
	Birth year, 1964	-0.750***	(0.163)
	Birth year, 1965	-0.751***	(0.164)
	Birth year, 1966	-0.763***	(0.169)
	Birth year, 1967	-0.602***	(0.155)
	Birth year, 1968	-0.569***	(0.159)
	Birth year, 1969	-0.761***	(0.207)
	Birth year, 1970	-0.774***	(0.234)
	Birth year, 1971	-0.767***	(0.222)
	Birth year, 1972	-0.609***	(0.224)
	Birth year, 1973	-0.661***	(0.220)
	Birth year, 1974	-0.474***	(0.116)
	Birth year, 1975	-0.254*	(0.144)
Other controls, Z*i*	Born in San Juan	0.213	(0.143)
	Born in Conacaste	0.405***	(0.108)
	Born in Espiriru Santu	0.374***	(0.111)
	Constant	-51.679***	(5.627)
	R-squared	0.211	

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. In addition to these variables also included are dummy variables for the small number of cases where maternal education, height or initial wealth was missing. Sample size 1267

Table 5A: Impact of HAZ on schooling related outcomes

	(1) Age start 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
Height for age z score, 36m	-0.204*	-0.006	0.013	0.732***	0.985***	0.337***	0.258***
	(0.115)	(0.047)	(0.024)	(0.267)	(0.307)	(0.098)	(0.088)
Observations	1201	1285	1164	1201	1238	1271	1267
R-squared	0.106	0.038	0.066	0.188	0.239	0.137	0.178
Kleibergen-Paap	19.06	20.19	17.94	15.97	19.63	20.25	20.01
Hansen J test: P-value	0.855	0.668	0.639	0.105	0.496	0.655	0.850

	(1) Age start 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
Stunted	0.513	-0.069	-0.049	-2.361**	-3.835***	-1.106***	-0.916***
	(0.386)	(0.167)	(0.083)	(1.077)	(1.197)	(0.363)	(0.321)
Observations	1201	1285	1164	1201	1238	1271	1267
R-squared	0.078	0.026	0.067	0.140	0.115	0.041	0.121
Kleibergen-Paap	9.783	10.50	8.962	7.702	10.11	10.40	10.35
Hansen J test: P-value	0.709	0.694	0.648	0.083	0.823	0.654	0.854

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: subject sex and birth year dummy variables; maternal age and education; paternal education; parental wealth index; school quality at age 7 and 13 (whether school building is permanent structure and student teacher ratio); distance to feeding center; electrification of village at age 2; and village of origin. Critical values for Kleibergen-Paap test statistic (Kleibergen and Paap 2006; Kleibergen 2007) at the 5% significance level are: 11.29 for rejecting null hypothesis of weak instruments, where weak means having bias in the IV results that is larger than 20% of the bias in the OLS results; 6.73 for rejecting null hypothesis of weak instruments, where weak means having bias in the IV results that is larger than 20% of the bias in the OLS results; and 5.07 for rejecting null hypothesis of weak instruments, where weak means having bias in the IV results that is larger than 30% of the bias in the OLS results.

Table 5B: Impact of HAZ on schooling related outcomes, Females

VARIABLES	(1) Age start 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
Height for age z score, 36m	-0.080	0.060	-0.007	0.912**	1.107***	0.475***	0.211**
	(0.146)	(0.063)	(0.041)	(0.373)	(0.407)	(0.126)	(0.105)
Observations	630	677	613	630	650	671	670
R-squared	0.107	0.028	0.083	0.186	0.210	0.097	0.113
Kleibergen-Paap	8.840	9.615	8.248	8.579	9.281	9.716	9.649
Hansen J test: P-value	0.487	0.211	0.551	0.162	0.056	0.421	0.577

VARIABLES	(1) Age start 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
Stunted	0.341	-0.275	-0.035	-3.342**	-4.200***	-1.467***	-0.774**
	(0.454)	(0.226)	(0.134)	(1.386)	(1.448)	(0.454)	(0.374)
Observations	630	677	613	630	650	671	670
R-squared	0.085	-0.027	0.093	0.094	0.070	-0.036	0.063
Kleibergen-Paap	5.611	6.037	5.508	5.306	5.683	5.955	5.920
Hansen J test: P-value	0.539	0.283	0.551	0.274	0.230	0.495	0.644

Notes: See Table 5A.

Table 5C: Impact of HAZ on schooling related outcomes, Males

	(1) Age start 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
Height for age z score, 36m	-0.230*	-0.053	0.042*	0.421	1.108***	0.203*	0.335***
	(0.134)	(0.061)	(0.024)	(0.321)	(0.355)	(0.115)	(0.119)
Observations	571	608	551	571	588	600	597
R-squared	0.177	0.043	0.066	0.205	0.262	0.223	0.172
Kleibergen-Paap	16.34	16.26	16.21	16.12	16.59	15.75	15.66
Hansen J test: P-value	0.559	0.448	0.230	0.117	0.228	0.716	0.896

	(1) Age start 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
Stunted	0.621	0.118	-0.148	-1.712	-3.867***	-0.608	-1.122**
	(0.482)	(0.213)	(0.091)	(1.283)	(1.458)	(0.437)	(0.454)
Observations	571	608	551	571	588	600	597
R-squared	0.140	0.054	0.052	0.168	0.150	0.187	0.088
Kleibergen-Paap	5.728	5.903	4.901	5.750	5.845	5.659	5.616
Hansen J test: P-value	0.472	0.381	0.208	0.161	0.350	0.652	0.801

Notes: See Table 5A.

Table 6A: Marriage outcomes: Characteristics of spouse: Females

VARIABLES	(1) Spouse's age	(2) Husband's grades of schooling	(3) Husband's Height	(4) Husband's family's land holdings	(5) Age differential	(6) Education differential	(7) Family's land holdings differential
Height for age z score, 36m	1.832**	0.512	0.244	0.333	1.844**	-0.193	-0.053
	(0.826)	(0.534)	(0.809)	(0.377)	(0.821)	(0.567)	(0.060)
Observations	528	378	340	344	528	376	343
R-squared	0.357	0.130	0.119	0.094	0.012	0.099	0.092
Kleibergen-Paap	8.708	15.65	12.43	9.866	8.708	15.01	9.871
Hansen J test: P-value	0.957	0.265	0.505	0.436	0.985	0.945	0.901

VARIABLES	(1) Spouse's age	(2) Husband's grades of schooling	(3) Husband's Height	(4) Husband's family's land holdings	(5) Age differential	(6) Education differential	(7) Family's land holdings differential
Stunted	-5.396**	-2.844	-0.267	-0.814	-5.676**	0.344	0.158
	(2.736)	(2.142)	(2.884)	(1.602)	(2.741)	(2.010)	(0.232)
Observations	528	378	340	344	528	376	343
R-squared	0.323	0.059	0.114	0.085	-0.046	0.092	0.088
Kleibergen-Paap	5.875	3.211	3.555	3.803	5.875	3.204	3.798
Hansen J test: P-value	0.868	0.463	0.519	0.372	0.947	0.929	0.886

Notes: Second stage control variables are: birth year dummy variables; maternal age and education; paternal education; parental wealth index; electrification of village at age 2; and village of origin. For other notes, see Table 5A.

Table 6B: Marriage outcomes: Characteristics of spouse: Males

VARIABLES	(1) Wife's age	(2) Wife's grades of schooling	(3) Wife's Height	(4) Wife's family's land holdings	(5) Age differential	(6) Education differential	(7) Family's land holdings differential
Height for age z score, 36m	0.672	1.058***	1.566**	-0.629	0.734*	-0.134	0.086
	(0.431)	(0.343)	(0.640)	(3.625)	(0.433)	(0.385)	(0.053)
Observations	568	551	483	443	568	472	441
R-squared	0.425	0.136	0.040	-0.002	0.079	0.066	0.020
Kleibergen-Paap	18.01	17.96	14.51	14.67	18.01	17.78	14.69
Hansen J test: P-value	0.538	0.495	0.103	0.962	0.513	0.414	0.558

VARIABLES	(1) Wife's age	(2) Wife's grades of schooling	(3) Wife's Height	(4) Wife's family's land holdings	(5) Age differential	(6) Education differential	(7) Family's land holdings differential
Stunted	-1.773	-3.226***	-3.580*	1.227	-2.157	0.546	-0.249
	(1.464)	(1.217)	(2.165)	(12.470)	(1.470)	(1.367)	(0.188)
Observations	568	551	483	443	568	472	441
R-squared	0.413	0.070	0.042	-0.003	0.056	0.061	0.009
Kleibergen-Paap	5.192	5.979	5.752	4.764	5.192	4.748	4.598
Hansen J test: P-value	0.433	0.557	0.060	0.960	0.438	0.433	0.480

Notes: See Table 6A.

Table 7: Fertility-related outcomes: Females

	(1) Age at menarche	(2) First birth before 17	(3) Spacing 1st and 2nd birth	(4) Number of pregnancies	(5) Any infant deaths	(6) Any child deaths
Height for age z score, 36m	-0.134	-0.077*	0.190	-0.628***	-0.025	-0.053
	(0.142)	(0.040)	(0.228)	(0.240)	(0.033)	(0.037)
Observations	669	592	505	671	671	671
R-squared	0.062	0.038	0.040	0.143	0.069	0.077
Kleibergen-Paap	10.03	10.07	7.569	10.17	10.17	10.17
Hansen J test: P-value	0.491	0.354	0.186	0.126	0.300	0.640

	(1) Age at menarche	(2) First birth before 17	(3) Spacing 1st and 2nd birth	(4) Number of pregnancies	(5) Any infant deaths	(6) Any child deaths
Stunted	0.298	0.251*	0.129	1.947**	0.055	0.099
	(0.497)	(0.142)	(0.778)	(0.807)	(0.107)	(0.119)
Observations	669	592	505	671	671	671
R-squared	0.057	0.013	0.028	0.099	0.066	0.075
Kleibergen-Paap	6.426	6.542	5.417	6.393	6.393	6.393
Hansen J test: P-value	0.418	0.277	0.429	0.124	0.224	0.482

Notes: Second stage control variables are: birth year dummy variables; maternal age and education; paternal education; parental wealth index; electrification of village at age 2; and village of origin. For other notes, see Table 5A.

Table 8A: Health related outcomes, all

VARIABLES	(1) Log Height	(2) Log BMI	(3) Overweight or obese	(4) Obese	(5) Waist-Hip ratio	(6) Head circum-ference	(7) Log Fat Free Mass	(8) Log Hand Strength	(9) Log VO2 max
Height for age z score, 36m	0.041***	0.027*	0.051	0.053*	0.002	0.716***	0.073***	0.059***	0.040
	(0.003)	(0.015)	(0.043)	(0.030)	(0.005)	(0.122)	(0.008)	(0.017)	(0.037)
Observations	1160	1160	1160	1160	1112	1143	1142	1159	1138
R-squared	0.630	0.097	0.085	0.052	0.060	0.399	0.717	0.574	0.239
Kleibergen-Paap	19.81	19.81	19.81	19.81	18.84	20.47	20.41	20.66	20.86
Hansen J test: P-value	0.005	0.804	0.877	0.331	0.269	0.292	0.138	0.387	0.662

VARIABLES	(1) Log Height	(2) Log BMI	(3) Overweight or obese	(4) Obese	(5) Waist-Hip ratio	(6) Head circum-ference	(7) Log Fat Free Mass	(8) Log Hand Strength	(9) Log VO2 max
Stunted	-0.136***	-0.105**	-0.201	-0.176	-0.009	-2.510***	-0.260***	-0.189***	-0.187
	(0.019)	(0.053)	(0.159)	(0.108)	(0.019)	(0.514)	(0.042)	(0.065)	(0.135)
Observations	1160	1160	1160	1160	1112	1143	1142	1159	1138
R-squared	0.190	0.066	0.075	0.037	0.058	0.236	0.539	0.541	0.225
Kleibergen-Paap	10.91	10.91	10.91	10.91	9.953	10.57	10.57	11.51	10.99
Hansen J test: P-value	0.003	0.897	0.913	0.298	0.290	0.548	0.117	0.425	0.766

Notes: Second stage control variables are: subject sex and birth year dummy variables; maternal age and education; paternal education; parental wealth index; electrification of village at age 2; and village of origin. For other notes, see Table 5A.

Table 8A: Health related outcomes, All, cont'd

	(1) LDL cholesterol	(2) Total cholesterol/ HDL	(3) Systolic blood pressure	(4) Diastolic blood pressure	(5) Hypertensive or prehyper-tensive	(6) Blood glucose level	(7) Diabetic or prediabetic	(8) Metabolic syndrome
Height for age z score, 36m	2.910	0.047	2.298**	0.931	0.099**	2.741*	0.064*	0.066*
	(3.005)	(0.136)	(1.020)	(0.832)	(0.039)	(1.504)	(0.036)	(0.038)
Observations	999	1034	1246	1246	670	1034	1034	1034
R-squared	0.021	0.051	0.137	0.051	0.004	0.012	0.006	0.085
Kleibergen-Paap	17.82	18.30	20.19	20.19	11.70	18.30	18.30	18.30
Hansen J test: P-value	0.175	0.500	0.064	0.829	0.117	0.133	0.049	0.239

	(1) LDL cholesterol	(2) Total cholesterol/ HDL	(3) Systolic blood pressure	(4) Diastolic blood pressure	(5) Hypertensive or prehyper-tensive	(6) Blood glucose level	(7) Diabetic or prediabetic	(8) Metabolic syndrome
Stunted	-6.264	-0.275	-7.691**	-3.271	-0.021	-9.816*	-0.288**	-0.277**
	(11.148)	(0.482)	(3.854)	(3.050)	(0.131)	(5.443)	(0.129)	(0.141)
Observations	999	1034	1246	1246	1246	1034	1034	1034
R-squared	0.024	0.047	0.096	0.036	0.073	0.001	-0.023	0.062
Kleibergen-Paap	9.153	9.826	11.34	11.34	11.34	9.826	9.826	9.826
Hansen J test: P-value	0.145	0.529	0.071	0.832	0.023	0.137	0.097	0.341

Table 8B: Health related outcomes, women

VARIABLES	(1) Log Height	(2) Log BMI	(3) Overweight or obese	(4) Obese	(5) Waist-Hip ratio	(6) Head circum-ference	(7) Log Fat Free Mass	(8) Log Hand Strength	(9) Log VO2 max
Height for age z score, 36m	0.040***	0.028	0.009	0.070	0.002	0.707***	0.071***	0.041*	0.040
	(0.004)	(0.021)	(0.061)	(0.049)	(0.008)	(0.159)	(0.009)	(0.022)	(0.042)
Observations	604	604	604	604	593	599	598	645	595
R-squared	0.262	0.026	0.027	0.008	0.041	0.185	0.162	0.070	0.026
Kleibergen-Paap	10.23	10.23	10.23	10.23	9.990	10.03	10.02	11.33	10.62
Hansen J test: P-value	0.013	0.512	0.588	0.259	0.287	0.0723	0.365	0.595	0.639

VARIABLES	(1) Log Height	(2) Log BMI	(3) Overweight or obese	(4) Obese	(5) Waist-Hip ratio	(6) Head circum-ference	(7) Log Fat Free Mass	(8) Log Hand Strength	(9) Log VO2 max
Stunted	-0.103***	-0.100	-0.066	-0.218	-0.006	-2.120***	-0.199***	-0.147**	-0.135
	(0.016)	(0.063)	(0.182)	(0.157)	(0.023)	(0.508)	(0.034)	(0.070)	(0.132)
Observations	604	604	604	604	593	599	598	645	595
R-squared	-0.127	0.026	0.034	0.006	0.042	0.075	-0.030	0.041	0.028
Kleibergen-Paap	7.416	7.416	7.416	7.416	7.488	7.366	7.393	7.937	7.996
Hansen J test: P-value	0.000	0.651	0.601	0.239	0.290	0.172	0.054	0.809	0.663

Table 8B: Health related outcomes, Women , cont'd

	(1) LDL cholesterol	(2) Total cholesterol/ HDL	(3) Systolic blood pressure	(4) Diastolic blood pressure	(5) Hypertensive or prehyper-tensive	(6) Blood glucose level	(7) Diabetic or prediabetic	(8) Metabolic syndrome
Height for age z score, 36m	2.291	-0.082	4.048***	3.144***	0.099**	3.370	-0.012	0.027
	(3.379)	(0.160)	(1.364)	(1.009)	(0.039)	(2.404)	(0.051)	(0.057)
Observations	585	600	670	670	670	600	600	600
R-squared	0.021	0.019	0.007	-0.020	0.004	0.019	0.030	0.041
Kleibergen-Paap	11.52	11.46	11.70	11.70	11.70	11.46	11.46	11.46
Hansen J test: P-value	0.309	0.170	0.161	0.381	0.117	0.343	0.051	0.075

	(1) LDL cholesterol	(2) Total cholesterol/ HDL	(3) Systolic blood pressure	(4) Diastolic blood pressure	(5) Hypertensive or prehyper-tensive	(6) Blood glucose level	(7) Diabetic or prediabetic	(8) Metabolic syndrome
Stunted	-4.337	0.091	-12.215***	-10.219***	-0.234*	-10.383	-0.073	-0.201
	(11.564)	(0.547)	(4.525)	(3.589)	(0.128)	(8.465)	(0.176)	(0.193)
Observations	585	600	670	670	670	600	600	600
R-squared	0.027	0.022	-0.045	-0.095	-0.005	0.011	0.028	0.036
Kleibergen-Paap	6.778	7.088	8.185	8.185	8.185	7.088	7.088	7.088
Hansen J test: P-value	0.282	0.145	0.165	0.293	0.082	0.319	0.050	0.098

Table 8C: Health related outcomes, men

VARIABLES	(1) Log Height	(2) Log BMI	(3) Overweight or obese	(4) Obese	(5) Waist-Hip ratio	(6) Head circum-ference	(7) Log Fat Free Mass	(8) Log Hand Strength	(9) Log VO2 max
Height for age z score, 36m	0.042***	0.024	0.068	0.023	0.001	0.709***	0.083***	0.083***	0.064
	(0.004)	(0.016)	(0.056)	(0.025)	(0.006)	(0.149)	(0.011)	(0.023)	(0.049)
Observations	556	556	556	556	519	544	544	514	543
R-squared	0.181	0.073	0.058	0.040	0.105	0.178	0.159	0.019	0.070
Kleibergen-Paap	16.35	16.35	16.35	16.35	22.12	18.72	19.13	16.31	18.60
Hansen J test: P-value	0.278	0.482	0.884	0.749	0.661	0.844	0.247	0.378	0.577

VARIABLES	(1) Log Height	(2) Log BMI	(3) Overweight or obese	(4) Obese	(5) Waist-Hip ratio	(6) Head circum-ference	(7) Log Fat Free Mass	(8) Log Hand Strength	(9) Log VO2 max
Stunted	-0.166***	-0.122*	-0.275	-0.130	-0.007	-2.981***	-0.343***	-0.259**	-0.324
	(0.026)	(0.070)	(0.235)	(0.108)	(0.022)	(0.742)	(0.073)	(0.101)	(0.203)
Observations	556	556	556	556	519	544	544	514	543
R-squared	-1.416	-0.037	0.009	0.001	0.104	-0.198	-0.892	-0.155	-0.006
Kleibergen-Paap	5.885	5.885	5.885	5.885	5.191	5.711	5.725	5.711	5.755
Hansen J test: P-value	0.498	0.687	0.914	0.849	0.674	0.999	0.888	0.265	0.764

Table 8C: Health related outcomes, Men , cont'd

	(1) LDL cholesterol	(2) Total cholesterol/ HDL	(3) Systolic blood pressure	(4) Diastolic blood pressure	(5) Hypertensive or prehyper-tensive	(6) Blood glucose level	(7) Diabetic or prediabetic	(8) Metabolic syndrome
Height for age z score, 36m	3.363	0.252	1.037	-0.461	0.004	2.039	0.120***	0.120***
	(3.919)	(0.201)	(1.117)	(1.123)	(0.051)	(1.376)	(0.045)	(0.043)
Observations	414	434	576	576	576	434	434	434
R-squared	0.036	0.025	0.025	0.023	0.018	0.033	-0.006	0.015
Kleibergen-Paap	15.47	16.06	17.38	17.38	17.38	16.06	16.06	16.06
Hansen J test: P-value	0.475	0.280	0.101	0.646	0.003	0.311	0.410	0.659

	(1) LDL cholesterol	(2) Total cholesterol/ HDL	(3) Systolic blood pressure	(4) Diastolic blood pressure	(5) Hypertensive or prehyper-tensive	(6) Blood glucose level	(7) Diabetic or prediabetic	(8) Metabolic syndrome
Stunted	-13.408	-1.070	-7.011	-0.464	-0.083	-8.970*	-0.510***	-0.463**
	(13.721)	(0.722)	(5.082)	(4.335)	(0.201)	(4.997)	(0.175)	(0.189)
Observations	414	434	576	576	576	434	434	434
R-squared	0.010	-0.021	-0.046	0.027	0.010	0.020	-0.102	-0.095
Kleibergen-Paap	4.372	4.853	5.480	5.480	5.480	4.853	4.853	4.853
Hansen J test: P-value	0.524	0.395	0.187	0.626	0.003	0.431	0.713	0.751

Table 9A: Labor market outcomes, all

	(1) Log hourly earnings	(2) Log hours worked	(3) Log earned income	(4) Years skilled labor	(5) Skilled labor or white collar work	(6) Worked on own business, fulltime
Height for age z score, 36m	0.155**	0.010	0.138	0.470*	0.163***	0.059*
	(0.073)	(0.077)	(0.114)	(0.266)	(0.048)	(0.033)
Observations	989	993	989	1193	694	1193
R-squared	0.136	0.231	0.288	0.154	0.097	0.048
Kleibergen-Paap	20.71	20.71	20.62	21.30	16.44	21.30
Hansen J test: P-value	0.540	0.527	0.559	0.250	0.301	0.973

	(1) Log hourly earnings	(2) Log hours worked	(3) Log earned income	(4) Years skilled labor	(5) Skilled labor or white collar work	(6) Worked on own business, fulltime
Stunted	-0.625**	-0.170	-0.730	-1.877**	-0.599***	-0.195
	(0.304)	(0.302)	(0.462)	(0.939)	(0.197)	(0.126)
Observations	989	993	989	1193	694	1193
R-squared	0.084	0.228	0.261	0.124	-0.020	0.016
Kleibergen-Paap	8.465	8.808	8.587	11.68	6.233	11.68
Hansen J test: P-value	0.553	0.563	0.722	0.351	0.302	0.938

Notes: Second stage control variables are: subject sex and birth year dummy variables; paternal education; parental wealth index; school quality at age 7 (whether school building is permanent structure and student teacher ratio); whether mother or father had died before subject turned 15; tobacco market collapse at age 15; electrification of village at age 2; and village of origin. For other notes, see Table 5A.

Table 9B: Labor market outcomes, females

	(1) Log hourly earnings	(2) Log hours worked	(3) Log earned income	(4) Years skilled labor	(5) Skilled labor or white collar work	(6) Worked on own business, fulltime
Height for age z score, 36m	0.067	0.210	0.226	0.377**	0.149***	0.097**
	(0.122)	(0.152)	(0.198)	(0.192)	(0.052)	(0.045)
Observations	439	442	439	635	233	635
R-squared	0.077	0.098	0.102	0.038	0.088	0.044
Kleibergen-Paap	11.27	11.13	11.15	12.27	10.19	12.27
Hansen J test: P-value	0.819	0.863	0.810	0.456	0.924	0.411

	(1) Log hourly earnings	(2) Log hours worked	(3) Log earned income	(4) Years skilled labor	(5) Skilled labor or white collar work	(6) Worked on own business, fulltime
Stunted	-0.284	-0.831	-0.993	-1.113	-0.527**	-0.336*
	(0.508)	(0.657)	(0.878)	(0.720)	(0.252)	(0.173)
Observations	439	442	439	635	233	635
R-squared	0.070	0.073	0.074	0.032	0.064	-0.012
Kleibergen-Paap	4.277	4.194	4.195	6.851	2.626	6.851
Hansen J test: P-value	0.819	0.841	0.807	0.349	0.728	0.394

Notes: See Table 8A.

Table 9C: Labor market outcomes, males

	(1) Log hourly earnings	(2) Log hours worked	(3) Log earned income	(4) Years skilled labor	(5) Skilled labor or white collar work	(6) Worked on own business, fulltime
Height for age z score, 36m	0.215**	-0.083	0.104	0.530	0.167***	0.035
	(0.084)	(0.056)	(0.100)	(0.480)	(0.063)	(0.040)
Observations	550	551	550	558	461	558
R-squared	0.102	0.029	0.081	0.080	0.072	0.050
Kleibergen-Paap	12.14	11.98	11.95	12.51	10.30	12.51
Hansen J test: P-value	0.482	0.129	0.253	0.222	0.200	0.717

	(1) Log hourly earnings	(2) Log hours worked	(3) Log earned income	(4) Years skilled labor	(5) Skilled labor or white collar work	(6) Worked on own business, fulltime
Stunted	-0.657**	0.090	-0.671*	-2.227	-0.557**	-0.074
	(0.329)	(0.193)	(0.393)	(1.698)	(0.248)	(0.150)
Observations	550	551	550	558	461	558
R-squared	0.027	0.036	0.008	0.039	-0.048	0.040
Kleibergen-Paap	5.485	5.681	5.566	5.725	5.003	5.725
Hansen J test: P-value	0.295	0.0788	0.498	0.268	0.152	0.654

Notes: See Table 8A.

Table 10: Consumption and poverty

	(1) Per capita household consumption	(2) Women: Per capita household consumption	(3) Men: Per capita household consumption	(4) Household is poor	(5) Women: Household is poor	(6) Men: Household is poor
Height for age z score, 36m	0.182***	0.188**	0.159***	-0.097**	-0.061	-0.152***
	(0.057)	(0.082)	(0.057)	(0.043)	(0.054)	(0.053)
Observations	1335	663	672	1335	663	672
R-squared	0.087	0.090	0.133	0.055	0.078	0.062
Kleibergen-Paap	21.93	9.143	18.84	21.93	9.143	18.84
Hansen J test: P-value	0.273	0.267	0.323	0.106	0.590	0.109

	(1) Per capita household consumption	(2) Women: Per capita household consumption	(3) Men: Per capita household consumption	(4) Household is poor	(5) Women: Household is poor	(6) Men: Household is poor
Stunted	-0.613***	-0.509*	-0.563**	0.309**	0.133	0.552***
	(0.210)	(0.283)	(0.225)	(0.153)	(0.183)	(0.196)
Observations	1335	663	672	1335	663	672
R-squared	-0.003	0.043	0.046	0.015	0.076	-0.062
Kleibergen-Paap	12.12	5.290	7.699	12.12	5.290	7.699
Hansen J test: P-value	0.387	0.236	0.483	0.157	0.606	0.329

Notes: Second stage control variables are: subject sex and birth year dummy variables; paternal education; parental wealth index; school quality at age 7 (whether school building is permanent structure and student teacher ratio); whether mother or father had died before subject turned 15; tobacco market collapse at age 15 ; electrification of village at age 2; and village of origin. For other notes, see Table 5A.

Table 11: 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 × *atole*	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" (subject born in 1974, 1975 or 1976)	YES	YES			YES	YES	YES		YES
Ministry of Health post, age 2y	YES	YES			YES	YES	YES		
Specification tests: Reading scores									
F test on excluded instruments	16.24	15.12	21.76	21.96	6.33	21.62	20.81	3.00	7.89
Kleibergen-Paap	20.25	20.69	30.05	27.61	6.29	27.62	29.36	2.82	7.86
Hansen J test: P-value	0.65	0.69	0.56	0.56	0.43	0.67	0.71	0.58	0.41
Specification tests: Hourly earnings (Males)									
F test on excluded instruments	11.02	10.02	13.93	14.14	9.50	14.68	14.14	0.99	10.58
Kleibergen-Paap	13.93	12.75	18.16	18.19	11.59	18.80	18.43	0.94	12.80
Hansen J test: P-value	0.48	0.37	0.32	0.48	0.70	0.37	0.24	0.90	0.53
Specification tests: Consumption									
F test on excluded instruments	17.38	16.35	23.62	23.42	6.80	23.40	23.01	2.52	8.29
Kleibergen-Paap	21.93	22.28	32.52	29.89	6.71	30.16	32.10	2.27	8.26
Hansen J test: P-value	0.27	0.34	0.20	0.15	0.14	0.56	0.67	0.16	0.11

Table 12: Selected results adjusting for attrition: Parameter estimates for HAZ

Outcome	(1)	(2)
	Not weighted for attrition	Attrition weighted
SIA z score	0.337***	0.343***
	(0.098)	(0.099)
Spouse's age differential (women)	1.844**	1.812**
	(0.821)	(0.822)
Number of pregnancies	-0.628***	-0.626
	(0.240)	(0.242)
Log hourly earnings (Males)	0.215**	0.212***
	(0.084)	(0.088)
Per capita household consumption	0.182***	0.173***
	(0.057)	(0.057)

Notes: Second stage control variables are those reported in Tables 5, 6, 7, 9 and 10. For other notes, see Table 5A.

Table 13: Selected results using predicted values of HAZ at 36m, excluding individuals who were only measured before 24m

	(1) SIA z score	(2) Spouse's age differential (women)	(3) Number of pregnancies	(4) Log hourly earnings (Males)	(5) Per capita household consumption
HAZ	0.289***	1.697*	-0.679**	0.171**	0.183***
	(0.106)	(0.886)	(0.272)	(0.081)	(0.064)
Observations	1125	471	601	483	1181
Kleibergen-Paap	24.15	8.186	10.76	12.41	26.47
Hansen J test: P-value	0.644	0.644	0.230	0.802	0.225

Notes: Second stage control variables are those reported in Tables 5, 6, 7, 9 and 10. For other notes, see Table 5A.

Table 14: Selected results using actual HAZ for individuals aged 30-42m

	(1) SIA z score	(2) Spouse's age differential (women)	(3) Number of pregnancies	(4) Log hourly earnings (Males)	(5) Per capita household consumption
HAZ	0.196*	1.567	-0.820***	0.160*	0.136**
	(0.119)	(1.013)	(0.295)	(0.096)	(0.066)
Observations	796	325	417	348	836
Kleibergen-Paap	19.43	7.272	8.774	8.222	19.24
Hansen J test: P-value	0.343	0.737	0.636	0.167	0.222

Notes: Second stage control variables are those reported in Tables 5, 6, 7, 9 and 10. For other notes, see Table 5A.

Figure 1: Mean HAZ by age at measurement

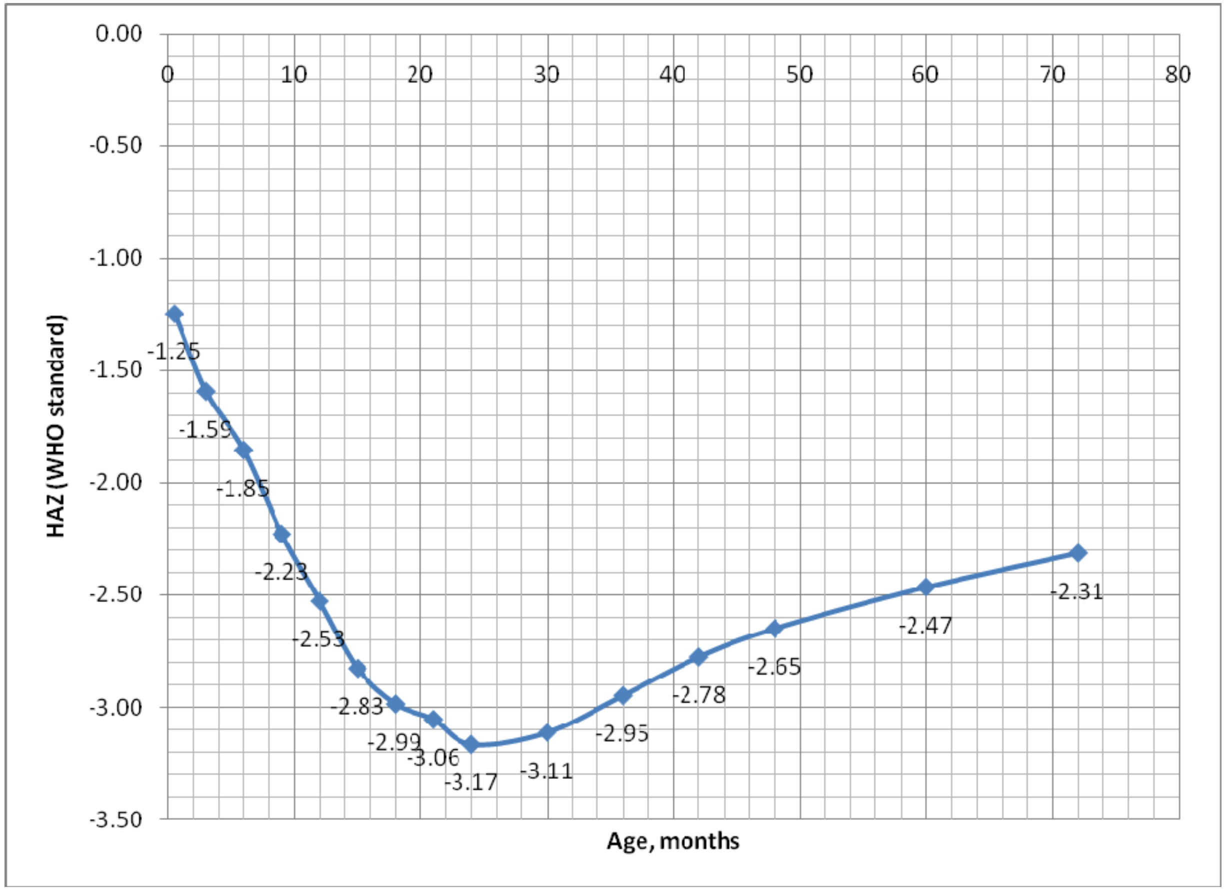

Figure 2: Impact of pre-school nutrition on reading/vocabulary scores: Parameter estimates and confidence intervals for alternative instrument sets

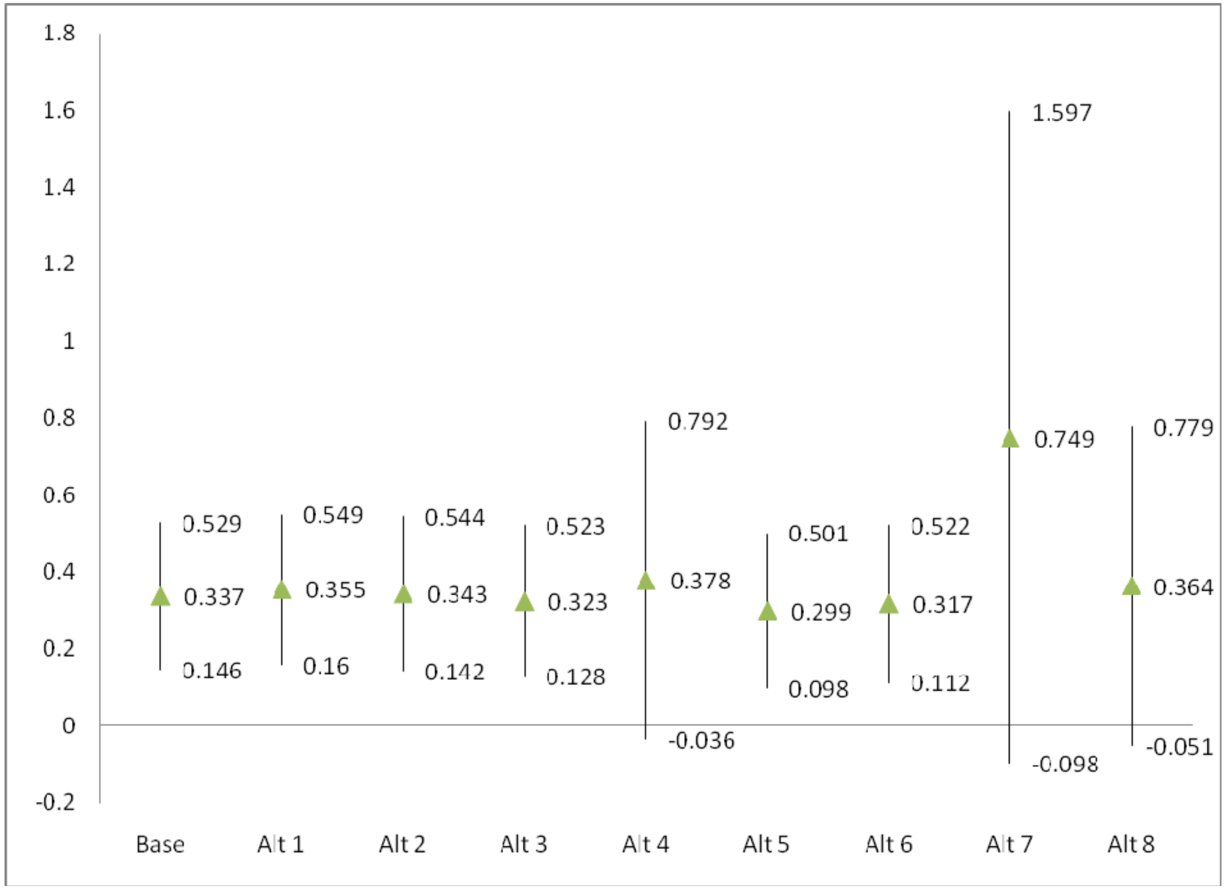

Figure 3: Impact of pre-school nutrition on hourly earnings (males): Parameter estimates and confidence intervals for alternative instrument sets

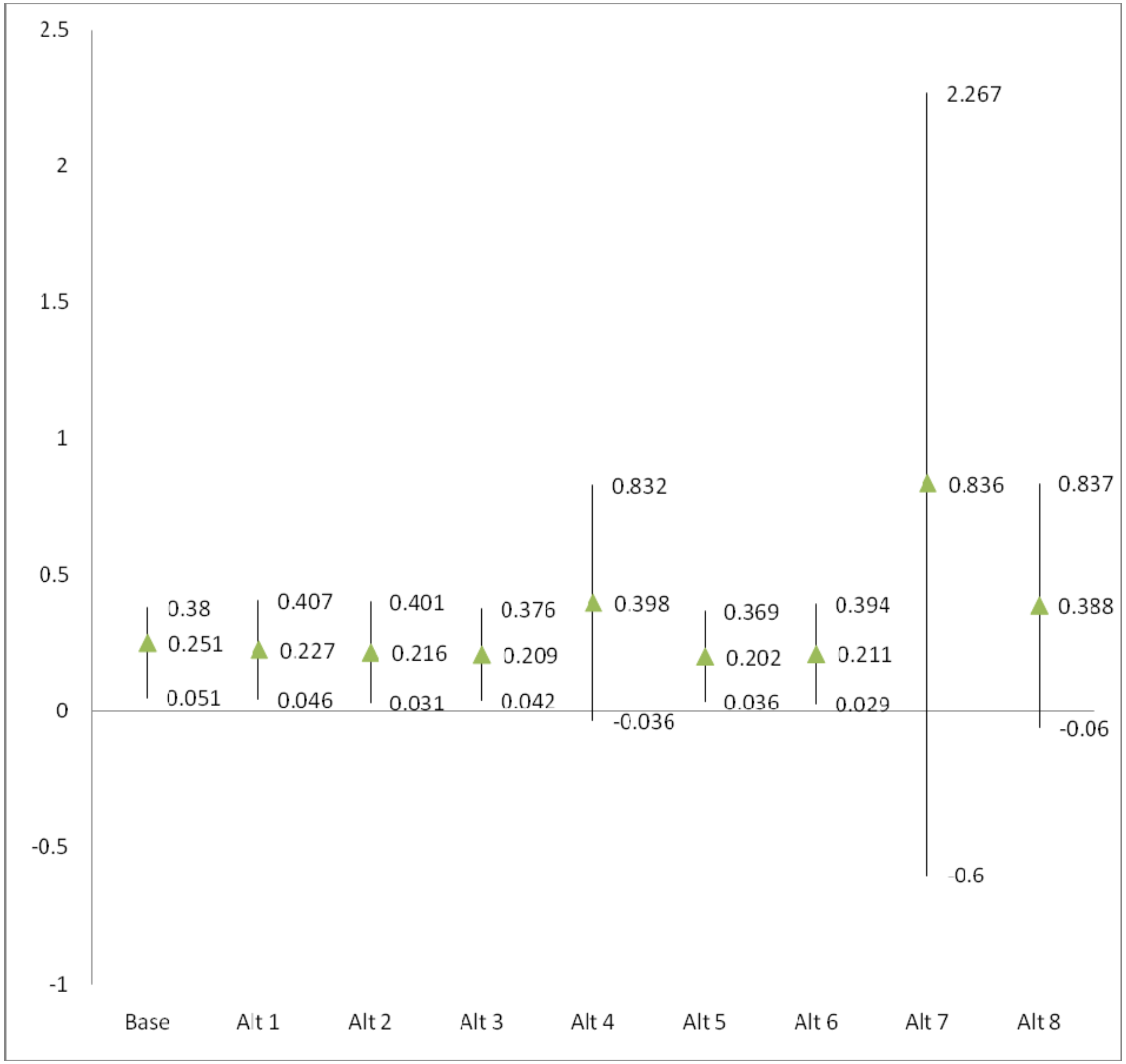

Figure 4: Impact of pre-school nutrition on household per capita consumption in adulthood: Parameter estimates and confidence intervals for alternative instrument sets