The height production function from birth to maturity

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Abstract

Height is the result of a complex process of growth that begins at birth and reaches the end in early adulthood. This paper studies the determinants of height from birth to maturity. A height production function is specified whose structure allows height to be the result of the accumulation of inputs (i.e., nutrition and diseases) over time. The empirical specification allows the causal identification of the age specific effects of both nutrition and diseases on height. Rich longitudinal data on Filipino children followed for more than 20 years are used. Considering the differences in growth patterns between boys and girls, the results show the existence of two critical periods for the formation of height: infancy and pre-puberty. In particular, diseases experienced during infancy, specially in the second year of life, and nutrition during pre-puberty play a major role.

JEL-Classification: I10; I12; O15; C13.

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1 Introduction

Starting in the 1970s, anthropometric measures have increasingly been used in the social sciences as indicators of social well-being. Since then, adult height has been considered an indicator of the general health status in life, of the relative risk of survival, and labor productivity (Fogel, 1986). In particular, Case and Paxson (2008) explain the positive correlation between adult height and labor productivity by showing that height is positively associated with cognitive ability. They show that both cognition and height are driven by early childhood investments and therefore cognitive achievements are correlated with height, and wages are affected by cognitive skills. Poor health can explain both low height and low labor productivity. This is more evident in developing countries where living conditions are poor.[1] However, adult height is just the final result of a process of growth that involves many different mechanisms and variables, and most of the common evidence looks at adult height (Steckel, 2009). It is therefore necessary to investigate the factors driving height, since understanding the determinants of height is important in order to understand health (Deaton, 2007).[2]

In this paper I study the determinants of height from birth to maturity in a developing country. To do that, I build and estimate a height production function.

To motivate the specification of a height production function, I follow Steckel's

[1]The fact that height increases wages or productivity is reported in: Behrman and Deolalikar (1989); Haddad and Bouis (1991); Steckel (1995); Thomas and Strauss (1997); Croppenstedt and Muller (2000); Schultz (2002); Dinda and Gangopadhyay (2006).

[2]Bozzoli et al. (2009) analyse the adult heights of 31 cohorts from England, the United States, and 10 European countries and show that there is a strong relationship between average heights and childhood mortality. They claim that some form of scarring in infancy negatively affects lifetime health, as marked by adult height.

(2009, pag. 7-8) reasoning:

> *. . . it is useful to think of the body as a biological machine, which consumes food as fuel - a blend of calories, protein, micronutrients and other ingredients. This machine expends fuel . . . to breathe, keep warm, circulate the blood and so forth, and in physical effort, fighting infection and physical growth. . . . The body's first priority is to survive, and growth stagnates or takes a back seat under conditions of inadequate net nutrition. . .*

Similar to the production process of a firm, the body can be considered a machine that combines different inputs through a particular technology to produce an output that in this case is height. The reason for estimating a production function is to find the *ceteris paribus* effects of each of the inputs. If I consider caloric intake and diarrhea as two of the inputs, the questions to answer are: "How does an exogenous change in caloric intake, holding all other inputs constant, affect height?" And "How does an exogenous change in diarrhea episodes, holding all other inputs constant, affect height?" And "At which age are those changes more relevant?" My intention is to find the technological parameters that answer the previous questions.

This paper uses the Cebu Longitudinal Health and Nutrition Survey (CLHNS), which is a rich longitudinal survey of a cohort of Filipino children followed from conception, in 1983-84, to 2005. The data allow the derivation of a height production function, from birth to maturity. According to Eckhardt et al. (2005a) and later to Hirvonen (2014), data to study growth that include multiple longitudinal measures spanning birth to adult stature and including adolescence are rare.

In particular, I study height as the result of the accumulation of several factors over time, identifying the direct effects of its determinants.[3] The determinants of height can be divided into non-genetic factors, genetic factors and the age when height is measured. The principal non-genetic factor is net nutrition which is the difference between food intake and the losses to activities and to diseases (Eveleth and Tanner, 1991). In developed countries there is evidence that genetic factors explain 80 percent of the variation in adult height and the rest is due to non-genetic factors. The proportion of the variation due to genetics seems to be less important when environmental stress is strong, for example in developing countries (Silventoinen, 2003). Therefore, the interplay between nutrition and diseases, and the understanding of which are the critical growth periods become crucial.[4]

There is an extensive research that demonstrates the importance of early childhood investments for child health, growth, skills development, and labor outcomes later in life (see for example, Glewwe and King, 2001; Schultz, 2002; Cunha and Heckman, 2008; Maluccio et al., 2009; Almond and Currie, 2011, and references therein). This paper extends the literature by considering also later periods of life until the body maturation.[5] In a recent paper van den Berg et al. (2014) use data on families migrating into Sweden from poorer countries and the relation between siblings' ages

[3]Todd and Wolpin (2003, 2007) consider different methods for modeling the production function for cognitive skills to account for the fact that child development is a cumulative process depending on the history of family, on school inputs and on innate ability. They consider different specifications of the skill production function that rely on different assumptions and data limitation. I follow the same approach to study the process of height formation and I clearly explain the assumptions made to identify the technological parameters of the height production function.

[4]Eckhardt et al. (2005b) have used the Cebu data from birth to age 18 to study the association between diet and height. They show that diet can continue to have independent positive effects on stature even beyond infancy. However, they do not include morbidity in their analysis.

[5]See Strauss and Thomas (2008) for the review of studies about health over the life cycle.

at migration and their adult outcomes to identify the ages that constitute critical periods in children's development towards their adult height and different economic outcomes. The main contribution of my work is the identification of critical periods that affect the entire process of height formation, emphasizing its causes, in particular, the relative importance of diseases versus nutrition. The analysis shows that the magnitude of the inputs effects during *infancy* and *pre-puberty* are the highest. The results of this paper are in line with the increasing literature on the long term effects of early childhood conditions, as well as new work on the critical influences of adolescence on health (van den Berg et al., 2014; Hirvonen, 2014).[6] This might be important to design policy interventions that target individuals in these critical periods to improve their health and potentially their socio-economic outcomes later in life.

The paper is structured as follows. In Section 2, I develop a model for studying the process of height formation, and I present the empirical specification. Section 3 presents the data and a detailed description of the variables used. Section 4 describes the empirical results. In Section 5, I present some robustness checks. Finally, Section 6 concludes.

2 The height production function

In this section I present a model for the height production function. I am interested in technological parameters such as the effect of an exogenous change in one input,

[6]Also Akachi and Canning (2007) find birth and adolescence to be critical periods, but they use cross-section data and mortality rate, GDP per capita, and protein intake as determinants of the adult height.

keeping all others constant. The technology that links inputs and output is fixed. It is created by nature and cannot be controlled. Economic agents play a negligible role in choosing some of the inputs, since the inputs they can choose are nutrition and diseases in the sense of prevention of diseases. They cannot choose either the age or the timing of children's growth.

It is widely known that height depends on the current age and on past inputs, such as health care practices, nutrient intake, disease incidence and genetic factors. A person's height is therefore a cumulative indicator because growth is a cumulative process by which past inputs and genetic endowment are combined in order to obtain height.

Let me define the height production function that relates the height measured at age t to all previous investments in the child. Suppose that for $t = 0, \ldots, T$ and $i = 1, \ldots, N$ I have:

- H_{it} the observed height for child i at age t,
- $f(t)$ an age trend,
- $\mathcal{X}_{i,t}$=$(X_{it}, X_{it-1}, \ldots, X_{i1}, X_{i0})$ the vector of inputs for child i from birth to age t,
- μ_i the child's biological endowment,
- ϵ_{it} a shock to the height production for child i at age t.

Then the **height production function** is given by:

$$H_{it} = h_t[f(t), \mathcal{X}_{i,t}, \mu_i, \epsilon_{it}]$$

where the inputs $\mathcal{X}_{i,t}$ are nutrition and diseases.

To study empirically the height production function, I make different assumptions.[7]

(A) I assume that the child's biological endowment is determined at conception and it is constant over time.[8]

(B) I assume that the height production function is linear in the inputs and in the unobserved endowment, and that the effects of the inputs depend on the child's age. The true technology that links inputs and output is unknown. This functional form implies that there is not complementarity and this means that all investments should be concentrated in one period, during the high-return period, and no investments should be made when the returns are low. This is in line with most biomedical and epidemiological studies in the "early influences" literature. They show that investments in early childhood produce effects on adult outcomes. But the effects may be bigger as individuals age, because the child's development is divided in different stages that have different influences on the adult outcomes.[9] Hence, I

[7]See Todd and Wolpin (2003, 2007) for a detailed description of different specifications of a skill production function for children and the assumptions made for the empirical specifications. The similarity of the two studies is that the processes of both height formation and achievement are cumulative processes that depend on the history of inputs chosen by the families, are due to the environment or are simply inherited genetically.

[8]Case and Paxson (2008) hypothesize an endowment determined at birth that changes according to the child's age. But their time-invariant individual effect also includes the environmental factors that in my study are observed and considered as further regressors in the model. Furthermore, I suppose that the gene-environment interactions are the same for each age of the child.

[9]It also seems plausible that there should be interactions among inputs, but their inclusion in the model is empirically intractable due to the limited number of observations.

obtain the following model:

$$H_{it} = f(t) + X_{it}\beta_t + X_{it-1}\beta_{t-1} + \cdots + X_{i1}\beta_1 + X_{i0}\beta_0 + \mu_i + \epsilon_{it}. \quad (1)$$

(C) I also assume that the time-varying coefficient β_s $(s = 0, 1, \ldots, t)$ depends only on the child's age s. For example, the impact of the infancy inputs on the final height is the same as the impact on height at age 8.[10]

2.1 Empirical specification

In order to estimate (1), I consider a within-child fixed effects specification (FE).[11] This specification is feasible because the children are observed more than once, and several outcome and input measurements are available.[12] In particular, consider differencing (1) by age:

$$\Delta_{it} = H_{it} - H_{it-1} = f(t) - f(t-1) + X_{it}\beta_t + \epsilon_{it} - \epsilon_{it-1} \quad (2)$$

[10] In a recent working paper, Griffen (2013) estimates a height production function using data from Guatemala on children up to age 7 years. He focuses on the impact of caloric intake on height and controls for the measurement error in the caloric intake. His model relies on two extra different assumptions: both contemporaneous and lagged inputs have constant effects by age.

[11] Cebu-Study-Team (1992) and Liu, Mroz, and Adair (2009) use the same data to estimate different health production functions. They adopt an another specification that includes lagged values of the outcome in the model instead of the historical inputs. In the cognitive skills literature this specification is called the "value added" specification. When data on past inputs are missing, the use of the lagged outcome is quite common. However, the lagged outcome is correlated with the shock by construction, and additional lagged outcome measures can be used as instruments to address endogeneity. Since past inputs are available, and I am specifically interested in their impact on height, I do not consider this specification. A lagged measure of height would capture almost all of the variability and it would not allow me to distinguish between the effects of nutrition and non-nutrition inputs.

[12] A within family specification would be interesting, but the data contain anthropometric measurements of some siblings but not all the information about siblings' net nutrition.

The βs parameters resulting from the above equation (2) are the specific input effects for the inputs applied between the two periods. The age trend is expressed as a linear and a quadratic term.

The within-child fixed effect estimator eliminates the endowment from equation (2), dealing with the endowment heterogeneity. However, there might be potential endogeneity of the nutrition and disease inputs. The fixed effects allow a permanent change in the inputs. On the other hand, contemporaneous inputs could respond to previous shocks causing endogeneity because correlated to unobserved parental preferences regarding their children's nutrition and preventative care. If, for example, a child is very small at a certain point in time, and the parents give him more food to help his/her growth, this is not captured by the fixed effect and produces endogeneity.

I address endogeneity of both nutrition and diseases by using variation in village-level food prices, village and household characteristics, and climatic shocks as instrumental variables (IV) to estimate the production parameters via IVFE. A second motivation for the use of IVs is that both nutrition and diseases are measured with error.

The within-child fixed effect estimator assumes that differenced omitted inputs are orthogonal to the differenced included inputs or that omitted inputs are constant over time and they are eliminated by the fixed effect estimators. In the Appendix, I report the estimates of a hybrid production function where I include family income as proxy for the time-varying omitted variables.

3 Data

The country of interest is the Philippines, and in particular the Metropolitan Cebu or Metro Cebu. Cebu is a province in the Philippines and it consists of Cebu Island and 167 surrounding islands.

The CLHNS, is a longitudinal survey of a cohort of Filipino women who gave birth between May 1, 1983 and April 30, 1984.[13] A stratified and single stage sampling procedure was used to randomly select 33 communities or barangays from the Metropolitan Cebu. Of them, 17 are urban communities and 16 are rural communities. The baseline survey includes 3327 women who were interviewed during the 6th to 7th month of pregnancy. All pregnant women of the barangay and the births were identified, and 3,080 non twin live births were consequently followed in the survey. Around 2,600 households were analyzed for the first 2 years. The children who were born during that period, their mothers, other caretakers, and selected siblings were followed through subsequent surveys conducted in 1991-2, 1994-5, 1998-9, 2002 and 2005. Apart from those last surveys, bimonthly surveys were conducted in the first 2 years of life of the children.

The initial focus of the survey was to collect information about the infants' feeding patterns. Later on, when the children were followed through adolescence and into young adulthood, the objective changed to a longitudinal intergenerational study of health. The data spans over 20 years and covers issues such as health, nutrition, water quality and sanitation. It contains detailed information about the mother's health

[13]For more information about the project and to download the data, visit `http://www.cpc.unc.edu`.

and behavior during pregnancy, such as health care practices or smoking behavior, children's education, household and individual economic situation, demographic information, family planning, intra-household relationships, and reproductive health.

Of special interest for my study is the rich collection of anthropometric measurements from birth to age 22, as well as complete disease and nutrition information. Since the data have information at the individual, household and community levels, it is possible to study the long-term effects of prenatal and early childhood nutrition and health on later adult outcomes, matching physical and socio-economic information.

The data is composed by 18 waves in total, 13 collected during infancy and 5 during pre-puberty and puberty. It is important to notice that individuals are not surveyed at the same age. The waves of the panel are not evenly spaced. Table 1 reports the children's age at the time of the different follow-ups.

Insert table 1 here.

The CLHNS is not a representative sample of the Philippines population, nor of all Cebu because of the criterion of selection based on fertility. However, Mendez and Adair (1999) find that the sample is representative of the ever married women with at least one child in the early '80s.

The *outcome* variable for this paper is raw height reported in centimeters. Height and weight were measured every two months for the first two years of life, and later during childhood and adolescence by the field staff in Cebu. The measurements were taken by specialists and this is a great advantage compared to the self-reported heights common to many datasets. Reliability checks were made to avoid heaping

and other errors in the measurements. The distribution of height by age and sex is shown in Figure 1.

Insert figure 1 here.

The inputs of the height production function can be divided into inputs during infancy, and inputs during pre-puberty and puberty. As previously specified the most relevant non-genetic inputs are nutrition and diseases.

3.1 Inputs during infancy

During the infancy period, the data are collected bimonthly. I aggregate the inputs between birth and age 1 year and between age 1 and 2.[14] In particular, I consider *caloric intake*, which is a good aggregate indicator of nutrition, even if it does not capture the role of micronutrients. The CLHNS data provide precise information about the individual's diet based on 24-hour dietary recalls or a quantitative food frequency questionnaire. Daily energy intake is calculated from 24-hour dietary recalls during the surveys from birth to age 2 years.[15] The caloric intake for infants is exclusive of breast milk. I compute the average caloric intake in the first and second year of life. Since this does not entirely capture the infant's nutrition, I also consider breast feeding.

Breast feeding has been found to improve both cognitive ability and adolescent health and therefore positively affect long-term academic achievement (Rees and

[14]Glewwe and King (2001) study the effects of malnutrition during infancy on children's cognitive development using the CLHNS data. They also aggregate the inputs over the first and second year of life, and in a second specification over 6-month periods. The shortest periods produce less precise estimates because they require an increase number of IV, and that also apply to my analysis.

[15]I am thankful to Linda Adair who provided me with the caloric intake computed by using the Food Composition Table owned by the Food and Nutrition Research Institute in the Philippines.

Sabia, 2009). Belfield and Kelly (2012) find that breast feeding for at least 6 months and not formula feeding at birth are negatively associated with obesity and positively associated with cognitive performance. In the analysis I consider the average time the child was breastfed in the first and second year.[16]

As for the diseases, I consider if the infant had feeding problems in the few hours after birth (baseline or wave 0) and diarrhea episodes later on (waves 1 to 12). In fact, some diseases reduce the absorption of nutrients, prevent food intake, produce nutrient losses or increase metabolic requirements (Stephensen, 1999). In particular, I compute the total number of times the infant had feeding problems at baseline or experienced diarrhea episodes in his/her first and second year of life. For simplicity I will refer to these infant diseases as diarrhea episodes.[17] This is likely an underreporting of the total number of diarrhea episodes experienced by the child.

3.2 Inputs during pre-puberty and puberty

The data collected for older children go from age 8 to age 22. Age 8 can be considered both pre-puberty or late childhood. For simplicity, I will refer to it as pre-puberty age.

The nutrition input considered during pre-puberty and puberty is *caloric intake*. The CLHNS data contains 24-hour dietary recalls in 1994, 1998, 2002 and 2005 for which the caloric intake has been computed. During the 1991-92 survey, the child's

[16] I combine two questions: "Was breast milk given to infant yesterday?" and "Was breast milk fed to infant seven days ago?". The child is considered breastfed if the answer is yes to at least one of the questions.

[17] During the baseline there is a question: "What are the infant's health problems affecting feeding?", and I indicate as 1 if the infant has at least one the problems. In every wave from 1 to 12 there is a yes/no question: "Has the infant had diarrhea during the past seven days?".

intake is based on a quantitative food frequency questionnaire, with items derived from a list based on 24-hour food recalls from the mother in the sample. The waves are not evenly spaced but, in the empirical specification, I control for the child's age at the interview and age squared.

I distinguish between *diseases* during infancy and later because they are age-dependent and might have a different impact on a person's growth. I consider hospitalization during pre-puberty and puberty. The diseases for which the child was hospitalized might have had a strong impact on the child's height. It could be that other reported illnesses are temporary or do not strongly affect height.[18]

Since the data contain different measures of morbidity that differ between waves, I make a distinction between pre-puberty and puberty. In particular, for pre-puberty I consider the total length of stay in the hospital between waves that is reported at age 8 and 11 (or waves 13 and 14.)[19] I assume all causes of hospitalization to have equal weight, but the length of stay in the hospital can be considered as an indicator of the intensity of the disease.

For puberty (from wave 15), since the length of stay in the hospital is not reported, I consider a dummy variable equal to 1 if the child has been hospitalized

[18]In his review, Silventoinen (2003) lists the different diseases that in the literature seem to be associated with growth. He claims that the effect of diseases on growth is strong in developing countries, where diseases are typically associated with nutritional deprivation. Silventoinen (2003) also claims that there are other diseases that might affect height, but more evidence is needed given that often the prevalence of some diseases is really low that the effects of a single disease are difficult to study. Crimmins and Finch (2006) use historical data from cohorts born before the 20th century in north Europe and find that the decline in old-age mortality and the increase in height was influenced by the reduced burden of infections and inflammation. Given that from the data I cannot exclude the possibility that any of the diseases, for which the child is hospitalized, cause inflammation of any type, I consider all causes of hospitalization.

[19]The survey questions used are: "Number of times the child was hospitalized since his/her previous interview" and "Number of days child was hospitalized for the first(second...) time."

since the previous wave, and 0 otherwise.[20] The underling assumption is that the intensity across the different diseases for which the child was hospitalized is the same. Given the context, it might be that the variables length of stay and hospitalization understate the true effect of the diseases. It can be that not all of the children were hospitalized because of lack of hospitals/clinics in the village or because parents decided not to or could not hospitalize them.

For the puberty period, I group the last three waves because similar both in terms of height and inputs and I consider the data up to when the final height is measured. These last waves correspond to the final stage of growth (see Figure 1.) In particular, I compute the average caloric intake, and the total number of hospitalizations between waves 15, 16 and 17. If, for example, the final height of a person is measured at the wave 16, then the inputs are the average caloric intake and total number of hospitalizations between waves 15 and 16, while wave 17 is discarded.[21]

Due to the different growth patterns of boys and girls, I estimate the production function by gender.[22]

[20]The survey question used are: "Was the child hospitalized of this illness/any of these illnesses, since our last visit?"

[21]The average male height at wave 15 is 158.45 cm (sd 6.79), at wave 16 is 162.59 cm (sd 5.90), and at wave 17 is 163.09 cm (sd 5.83). The average female height at waves 15, 16 and 17 is respectively 149.02 cm (sd 5.53), 150.06 (sd 5.45) and 151.17 (sd 5.46). The average male caloric intake at wave 15 is 1914.63 (sd 780.45), at 16 is 2106 kcal (sd 978.66) and 2197 kcal (sd 960.67) at wave 17. The average female caloric intake at waves 15, 16 and 17 is respectively 1285 (sd 554.57), 1510 (sd 716.11) and 1544 kcal (sd 744.03). The proportion of males hospitalized at wave 15 is 0.06 (sd .23), at 16 and 17 is 0.05 (sd 0.22) and 0.05 (sd 0.22). The proportion of females hospitalized at waves 15, 16 and 17 is respectively 0.04 (sd 0.20), 0.05 (sd 0.21), 0.04 (sd 0.19).

[22]At birth the typical boy grows faster than the typical girl, but the velocities become equal around 7 months and then girls grow faster until age 4. There are no differences until they reach adolescence. The typical girl is slightly shorter than the typical boy at all ages until adolescence. She is taller during her adolescence spurt because it takes place two years before the male spurt

Table 2 reports descriptive statistics for the main variables, by gender and age. The table reports also some other inputs that affect the child's development: genetic and environmental inputs as well as inputs from conception to birth. A proxy for the genetic inputs is *mother's height*.[23] I assume that the rest of the genetic impact is captured by the individual's biological endowment included in the model. It represents the genetic inheritance and gene-environment interactions that are unobserved factors (Case and Paxson, 2008). An extra variable that captures the environmental inputs is the *location* of the household, and in particular the percentage of time the child has lived in a urban area from conception to maturity. Moreover, table 2 shows descriptive statistics of inputs from conception to birth: the infant's *birth weight*, the *duration of the gestation* (a categorical variable indicating whether the child had normal weight and normal term, low birth weight for his gestational age or simply pre-term but with normal weight for his gestational age), and the *birth order*.[24] Both the inputs from conception to birth and the genetic and environmental inputs are time invariant variables that are not identified by the fixed effect estimators. Table 2 show that there are no relevant differences between boys and girls at birth. The

(Tanner, 1990).

[23]Many medical papers suggest that approximately about 60 to 80 percent of height variation in a population depends on genetic factors, but it is not clear what is the underlying process (see, e.g., Ginsburg et al., 1998; Silventoinen, 2003) nor is the relationship between genetics and environmental factors clear. The data do not contain father's height.

[24]Many researchers suggest that growth *in utero* may play an important role in determining health in adult life (Barker, 1998). The importance of the birth weight is well known and there is a huge literature about it in medicine as well as in economics (e.g., Rosenzweig and Schultz (1983); Behrman and Rosenzweig (2004)). The problems of prematurity are very similar to those of low birth weight. Birth order has also been found to be a significant and independent predictor of adult height (Steckel, 1995). First-born children are, during childhood, taller than children born later, since they have had a period in which they were alone. These inputs from conception to birth are not exactly inputs, but the results of pre-birth inputs that are not available (e.g., birth weight).

birthweight is on average about 3 kg, 88% of the pregnancies have normal length and the birth order is 2.5 for the girls and 2.6 for the boys. The percentage of time spent in a urban location during the entire life is about 75%. During the first year of life the children are breastfed for most of the time, about 75% for the boys, and 77% for the girls, while there is a decrease in the second year to 38% approximately. The average caloric intake is slightly higher for the boys during infancy, and it increases systematically over time reaching on average 2061 kcal for the boys at the end of their maturation period, and 1420 kcal for the girls. The number of times the infant experienced diarrhea are on average 0.65 in the first year for the boys, and 0.61 for the girls. The diarrhea episodes increase on average to 0.92 in the second year of life for the boys and 0.78 for the girls. Between age 2 and 8 the total number of days spent in hospital is on average 1.10 for boys and 0.81 for girls, while between age 8 and 11 the days in hospital reduce to an average of 0.48 for boys and 0.42 for girls. Between age 11 and the age at final height, the number of hospitalizations is on average 0.13 for boys and 0.10 for girls.

Insert table 2 here.

3.3 Age 8

It is important to note that the data contain the disease history for the child between age 2 and 8. Therefore, the hospitalizations experienced during the childhood period are not missing. The length of stay in the hospital reported at age 8 is therefore the total length of stay in the hospital experienced from age 2 to 8. However, nutrition during childhood is missing, therefore to estimate the height production function at

age 8, I impose the following assumptions. Consider the following equations:

$$H_8 = f(8) + X_8\beta_8 + X_7\beta_7 + \cdots + X_1\beta_1 + X_0\beta_0 + \mu_i + \epsilon_8 \qquad (3a)$$

$$H_2 = f(2) + X_2\beta_2 + \cdots + X_1\beta_1 + X_0\beta_0 + \mu_i + \epsilon_2 \qquad (4a)$$

Since the Xs correspond to nutrition (C as caloric intake) and diseases (D), I can rewrite (3a) and (4a) as follows:

$$H_8 = f(8) + C_8\gamma_8 + C_7\gamma_7 + \cdots + C_1\gamma_1 + C_0\gamma_0 + D_8\delta_8 + D_7\delta_7 + \cdots + D_1\delta_1 + D_0\delta_0 + \mu_i + \epsilon_8 \qquad (3b)$$

$$H_2 = f(2) + C_2\gamma_2 + C_1\gamma_1 + C_0\gamma_0 + D_2\delta_2 + \cdots + D_1\delta_1 + D_0\delta_0 + \mu_i + \epsilon_2, \qquad (4b)$$

and the difference between (3b) and (4b) is:

$$H_8 - H_2 = f(8) - f(2) + C_8\gamma_8 + C_7\gamma_7 + C_6\gamma_6 + C_5\gamma_5 + C_4\gamma_4 + C_3\gamma_3 + \\ + D_8\delta_8 + D_7\delta_7 + D_6\delta_6 + D_5\delta_5 + D_4\delta_4 + D_3\delta_3 + \epsilon_8 - \epsilon_2.$$

Unfortunately, because of the data limitation it is not possible to estimate this equation. I then consider the total days the child spent in the hospital during 6 years, between age 2 and 8, $D_3 + \cdots + D_8 = \sum_{t=3}^{8} D_t$. In order to identify the δ's I would need a large set of instruments measured between age 2 and 8 that I do not have. Therefore, I specify $D_3\delta_3 + \cdots + D_8\delta_8 = \delta \sum_{t=3}^{8} D_t$ by assuming that $\delta_3 = \delta_4 = \cdots = \delta_8 = \delta$ and I estimate $\hat{\delta}$ that represents the effect of the diseases between age 2 and 8 on height at age 8. However, to be able to identify δ I use instrumental variables that are only available at age 8, by assuming that they also explain the past diseases

$(D_3, D_4, \ldots, D_7)$.

From the data I know the caloric intake at age 8, but unfortunately I do not know the caloric intake between age 2 and 8. I assume the following $C_3 = C_4 = C_5 = C_6 = C_7 = C_8$, and I obtain

$$C_3\gamma_3 + \cdots + C_8\gamma_8 = C_8\gamma_3 + \cdots + C_8\gamma_8 = C_8 \sum_{t=3}^{8} \gamma_t.$$

I then estimate $\hat{\gamma} = \sum_{t=3}^{8} \gamma_t$. This is an aggregate estimate of γ, but it does not allow me to identify and disentangle the impacts of $\gamma_3, \ldots, \gamma_8$.

3.4 Instrumental variables

Valid instruments must be uncorrelated with height and correlated with one or more of the endogenous variables (nutrition - caloric intake and breastmilk - and diseases). Therefore, I need to find instruments that only operate through their impact on nutrition and diseases. The IVs that would seem to satisfy these requirements can be categorized in three groups: food local prices, village and household characteristics and climatic shocks.[25]

[25]Some of the IVs used in this paper have been used in other studies (see for example, Cebu-Study-Team, 1992; Glewwe and King, 2001; Liu et al., 2009; Ugaz and Zanolini, 2011). In particular, Cebu-Study-Team (1992) use the same data to estimate infants health production function and they analyze four different outcomes: gestational age, weight, diarrhea and respiratory infection. They focus only on the first year of life. They use a "value-added" specification, thus, all the past inputs collapse in the past outcome measure. An instrumental variables approach is used to find the effects of the contemporaneous endogenous inputs (e.g., birth weight, gestational age, diarrhea, and few more). Liu, Mroz, and Adair (2009) also estimate health production function using the CLHNS data and consider children in the first two years of life. They estimate an empirical model that includes parents' demand equations and the child's health production functions. Their analysis is based on a dynamic model of parental investments. They jointly estimate four health outcomes: weight, height, incidence of diarrhea and incidence of severe respiratory infections. They also use

In particular, I consider local prices of the main food items (e.g., egg, banana, cooking oil, powder milk, evaporated milk, kerosene.)[26] [27] It is difficult to imagine how these prices could be correlated with height, hence, they should be uncorrelated with the error term in the second stage regression. The food prices of the major food items are expected to be negatively correlated with food consumption. During infancy the caloric intake is exclusive of breastmilk, therefore, most of the caloric intake reported for infants in their first year correspond to breastmilk substitutes. If the price of the formula goes up then the mother might prefer to continue breasfeding.

The village or barangay characteristics considered are population density and public health facilities, both exogenous with respect to height. It is difficult to imagine that families decide where to live according to their child's height. These variables are correlated with the size of the village. Bigger villages may have better infrastructures and easier access to food and health care.

The household's characteristics are presence of an infant store in the neighborhood, availability of drinking water inside the child's house, availability of piped water as the water source for the house, possession of a refrigerator, number of houses within 50 meter to the child's house, minutes walk to the nearest infant store, distance to the nearest vehicular road. One can think that families make location decisions (where to live in the village) or what access to purchase depending on the

a "value-added" specification. The endogenous inputs are breast feeding choices, caloric intake for supplement food, prenatal care, mother's working and preventive health care. They consider diarrhea and respiratory infections as health outcomes, and not as inputs in the height production function. Since I consider only height as an outcome and diseases as relevant inputs, my model is not directly comparable with Liu, Mroz, and Adair's (2009) model.

[26] I thank the National Statistics Office of the Philippines, which provided me with the CPI and inflation rates used to deflate the prices.

[27] Note that kerosene is used for cooking.

child's height. To address this potential endogeneity of the household's characteristics variables, I average them over the same village of current residence and I use these averages as IVs. Sanitation variables (drinking and piped water) may affect the child's chances of coming into contact with pathogens; while if a household owns a refrigerator this increases the possibility of preserving food. Accessibility to stores and roads are correlated with accessibility to food. At the same time a more isolated or sparse village (number of houses within 50 meter to the child's house) may reduce the probability of person-to-person contacts and the resultant transmission of pathogens (Cebu-Study-Team, 1992).

The climatic instrumental variables considered are two. The first is season and it is a dummy variable that indicates if the survey falls in the rainy season. This variable may be important for diseases, for example, in the case of diarrhea extensive rainfall can contaminate the water supply with fecal pathogens.[28] The second variable is the effect of one of the strongest typhoon, which name is Nitang, that hit Cebu on September the 2nd in 1984 by killing about 1,500 people.[29] Extreme weather conditions, such as typhoons, are associated with problems of malnutrition, waterborne diseases and may as well disrupt income-earning activities. Ugaz and Zanolini (2011) use the CLHNS data to investigate whether the exogenous weather shock caused by this typhoon had an impact on children's anthropometric outcomes later in life. They look at infants and at the effects of the typhoon during the pregnancy and right after birth. Variation in the exposure to the typhoon comes from the

[28]When different waves are combined, such as data collected during year 1 or 2, and the final waves (15, 16 and 17), then the season variable indicates the number of rainy season months experienced in that specific time interval.

[29]See `http://www.pagasa.dost.gov.ph` for details.

date of birth of the children. This spread in the dates of birth offers an exogenous type of variation to exposure to the typhoon. They find that the likelihood of reporting diarrhea right after Nitang is greater. I consider the distance in time between the timing of the typhoon and the date of birth as an indication of the exposition time to the typhoon. This IV is used only for year 1, because the typhoon hit the island just before the baseline data collection when the children were between 4 and 16 months old. I assume that the impact of the typhoon is lower if not absent one year later to be considered as IV for year 2.

The instruments are time-variant variables computed at the time of each interview.[30] For the infants the IVs are averaged over year 1 and year 2. For the puberty period the IVs are averaged over the last three waves if final height is reported in wave 17, over waves 15 and 16 if final height is reported in wave 16 or correspond to wave 15 if final height is reported in wave 15. The set of instruments is equal for boys and girls of the same age, but it changes across ages because of price variability, date-specific events, and because the inputs are defined differently (e.g., diarrhea episodes during infancy and length of stay in the hospital during pre-puberty.) Therefore, the same IVs do not have the same predictive power across different ages. The IVs differ between year 1 and 2 because an extreme weather shock is considered at age 1 and because there is a big difference in the nutritional inputs. In year 1 most of the children are breastfed, while in year 2 the proportion is much lower. Therefore, the same IVs explain differently the endogenous inputs, since some IVs are more relevant for the weaning period, which is predominant in the second year. The IVs differ at

[30]The choice of the IVs has also been made avoiding the presence of instruments highly correlated.

ages 8 and 11 mainly because the causes of hospitalizations are different. The length of stay reported at age 8 covers 6 years of childhood where the main causes of first hospitalization are 22% diarrhea, 12% measles, 9.3% fever, 9.3% dengue and 9.3% accidents; while at age 11 the first cause of hospitalization is in 30% of the cases dengue, 16.3% accidents, 10% diarrhea and 7.7% typhoid fever.

Table 7 in the Appendix reports the descriptive statistics of the IVs used in each model.

4 Empirical results

The empirical results are shown in Tables 3-6. The tables report both the FE and IVFE estimates for each model. In the IVFE specifications I consider both nutrition and diseases as endogenous inputs. The estimation of each change in height allows to derive the effect of all the inputs. In particular, I estimate the following equations to identify all the β's contained in model (1):

$$\begin{aligned}
\Delta_{i1} &= H_{i1} - H_{i0} = \Delta f(1) + X_{i1}\beta_1 + \epsilon_{i1} - \epsilon_{i0} \\
\Delta_{i2} &= H_{i2} - H_{i1} = \Delta f(2) + X_{i2}\beta_2 + \epsilon_{i2} - \epsilon_{i1} \\
\Delta_{i8} &= H_{i8} - H_{i2} = \Delta f(8) + X_{i8}\beta_8 + \epsilon_{i8} - \epsilon_{i2} \\
\Delta_{i11} &= H_{i11} - H_{i8} = \Delta f(11) + X_{i11}\beta_{11} + \epsilon_{i11} - \epsilon_{i8} \\
\Delta_{iF} &= H_{iF} - H_{i11} = \Delta f(F) + X_{iF}\beta_F + \epsilon_{iF} - \epsilon_{i11}
\end{aligned}$$

Insert table 3 here.

Insert table 4 here.

Insert table 5 here.

Insert table 6 here.

The FE provide evidence on the effect of exogenous input variables on height. The IVFE instead allow a causal interpretation of the effects of nutrition and diseases on height. One difference between FE and IVFE results is the magnitude of the diarrhea coefficients, where the effect is negative and larger in the IVFE specification than in the FE resulting in nutrition inputs that in most of the cases lose their statistical significance. The other difference between FE and IVFE results is the magnitude of the caloric intake during the pre-puberty phase, where the nutrition effects increases when the IVFE specification is used.

Tables 3-6 report also tests on the quality of the instruments used. In most of the models the under identification test (Kleibergen-Paap test) rejects the null hypothesis indicating that the model is identified and the excluded instruments are relevant and correlated with the endogenous variables. I also check the validity of the instruments. The estimates, in fact, always satisfy the over identification test (Hansen's J statistic) where the null hypothesis is never rejected, suggesting that the instruments are not correlated with the error term. In most of the models, the instruments have strong predictive power for both nutrition and disease, as revealed by the F-test statistics (bottom of Tables 3-6), even if the F-test statistics are always nigher for the nutrition inputs. Tables 8-12 in Appendix report the first stage regression results for each endogenous variable in each model. The estimates present the expected signs as described in the Instrumental variables section.

To describe the second stage results I mainly focus on the IVFE results that address the endogeneity problem. The effects of the different inputs vary according to the age of the child, but the magnitude and timing is similar between boys and girls. The highest negative variation in height is due to diseases experienced in early life, while the highest positive variation in height is due to nutrition during pre-puberty, and this applies to both boys and girls.

In particular, once controlled for endogeneity, the inputs applied in the second year of life are significant and larger than the ones applied in the first year. This confirms the results found by Glewwe and King (2001) that using the same data underline the relevance of malnutrition on cognitive development in the second year of life. Once controlled for endogeneity, the importance of nutrition, both breast feeding and caloric intake, vanishes, except at age 2 for boys. The negative effect of diarrhea, instead, increases using the IVFE and remains statistically significant.

In particular, diarrhea has always a significant and negative impact on infants' height. If experienced in the first year of life, the results indicate that an increase by one of the diarrhea episodes decreases height of a boy by 1.376 cm, and the height of a girl by 2.113. In a 2 years old boy an increase of the caloric intake by 100 kcal results in an increase in height by 0.298 cm. Experiencing an extra episode of diarrhea in the second year of life, reduces the heigh of a boy by 2.214 cm, and the heigh of a girl by 3.171 cm.

The late childhood or early pre-puberty nutrition inputs (inputs at age 8) seem to influence the boys' height. In fact, the IVFE estimates show that if caloric intake increases by 100 kcal then height increases in boys by 0.199 cm. The IVFE estimates

for girls at age 8 indicate that the IVs are weak and have little explanatory power. If I use different sets of IVs, there are cases where the underidentification tests improve, but the IVFE second-stage estimates do not change.[31] The total length of stay in hospital included at age 8 and that covers the entire childhood period do not result to be statistically significant.

Nutrition is relevant during the pre-puberty years both for boys and girls (inputs at age 11). Once controlled for endogeneity, if caloric intake increases by 100 kcal at age 11 then height increases in boys by 0.246 cm, and in girls by 0.200 cm. Diseases captured by the total length of stay in hospital do not result significant.

As for puberty, the inputs present no impact on the final height. The IVFE specification for the final height models indicates that the instruments are not correlated with the error term, but seem weak. Even if I use different sets of IVs the results do not change and the instruments remain weak, however, in these last years of growth strong impacts of the inputs are not expected.

Overall, these results show that growth in infancy and in the pre-puberty years turn out to be critical stages in the process of height formation.

5 Robustness

5.1 Omitted variables bias

To account for the omitted variables bias, I estimate a *hybrid production function* that includes in the empirical specification (both IV and IVFE) the annual household

[31] The boys' estimates are stable even when different IVs are used, and always produce a significant effect of the caloric intake.

income in pesos.

In general, the hybrid health production functions are production functions that contain some of the health inputs and the determinants of the other non-available inputs. In this case the health outcome is height, therefore, I estimate the height production functions.

I test the possibility of omitted variables bias by looking at the coefficients of household income in the hybrid production functions. The hybrid production functions are reported in the Appendix, on Tables 13-16. Overall, a comparison of Tables 3-6 and Tables 13-16 shows that most of the estimated input effects are very similar across the nonhybrid and hybrid specifications.[32]

Once controlled for endogeneity, the effect of family income is never statistically significant except in the 1 year old boys' model (Table 13). An increase of income in the first year of life of the child by 1000 pesos increases height by 0.001 cm, while the effect of diarrhea loses its significance. This can be an indication of omitted variable bias in the 1 year old estimates for boys. However, the standard deviation of the income variable is quite high (Table 2), and it is likely that its inclusion does not address satisfactorily the omitted variables problem. Moreover, once income is included, the hybrid effect of the inputs on height is generally a biased estimate of the true technical relationship (other inputs held constant) embodied in the health production function (Rosenzweig and Schultz, 1983).

[32]Note that with the inclusion of income in the girls model at age 1, the instruments seem now weak.

5.2 Sample selection bias due to attrition

The last wave of the data contains 1888 people (993 males and 895 females, Table 1). Between the baseline and the last wave around 38.7 percent of the children are lost, 18 percent before the age of 2. This high attrition is common in long-term longitudinal studies and in data that come from developing countries. The highest attrition rate is between wave 12 and 13, that is, between infancy and pre-puberty. From wave 12 conducted in 1985-86 and wave 13 conducted in 1991-92 there are approximately 6-7 years of no data.

The two main reasons for attrition are death and migration. Two hundred and twenty-five (7.3 percent of the sample) children die: 167 (5.4 percent) in the first 2 years of life, 44 between the ages of 2 and 8, 14 children die during adolescence. In total 129 boys and 96 girls die. The remaining 31 percent are mainly lost because of migration.

It seems that the people who died tended to be shorter and in poor health. Unfortunately, there are not plausible exclusion restrictions that could be used to test and correct the selection on unobservables that determine death. Hence, given the rather low percentage of children who died, I keep them in the sample. Attrition due to mortality is claimed not to represent a big problem because only a small proportion of children in the poorest health conditions are lost (this is also claimed by Eckhardt et al., 2005a, who use the same data). If a selection mechanism is in place so that only the healthiest survive, then my estimates would be a lower bound of the true effect.

As for migration, Cebu-Study-Team (1992) tested for selectivity of infants and

the results show that the omitted variables that influence migration decisions do not coincide with those that determine child health. Older children who moved are in some years slightly healthier than the non attritors. If those who migrate are the better-off in the village, then the effects of nutrition and diseases on height are overestimated. However, the differences in terms of height, diseases and caloric intake, between the movers and the people who stay in the survey, are small and therefore I do not suspect selective migration to be a source of problem.

6 Conclusions

In this paper I study the determinants of height building a height production function from birth to maturity. I consider the cumulative nature of physical development, taking into account the biological inputs that cover the entire process of height formation. I estimate an empirical specification for the height production function where the change in height between two consecutive measurements allow the reduction of the endogenous inputs. I use both FE and IVFE. The IVFE allow the estimation of conditional demand equations for both nutrition and diseases, treated as endogenous inputs.

The results show that the diarrhea episodes experienced during infancy, and in particular in the second year of life, have the largest and negative effects on height. Diseases experienced later in life and measured with time spent in the hospital or number of hospitalizations do not seem to affect height. Pre-puberty is dominated by the effects of nutrition. Eckhardt et al. (2005a) use the same data and find that

puberty seems to be a phase of catch-up. It might be that the pre-puberty years prepare the body for the final phase of growth, and that might explain the relevance of nutrition inputs at age 11 and also age 8 for boys.

A limitation of the study is that the childhood period is missing in the data. New data that cover also this important phase of growth could potentially indicate it if is also a critical period.

The model shows the importance of including past inputs and of studying their effects according to different ages of the children. On the other hand, the results show it is important to know what factors determine a person's height at different growth periods of her life in a poor country, which periods are critical, and which inputs have the strongest impact on growth. In fact, growth during infancy and pre-puberty turn out to be critical stages in the process of height formation. The results of this paper are in line with the increasing literature on the long term effects of early life conditions (Almond and Currie, 2011) and with new studies that reveal the importance of later critical periods (van den Berg et al., 2014). Most importantly the paper shows that some critical periods are important because diseases play a major role compared to nutritional intake.

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Figures and tables

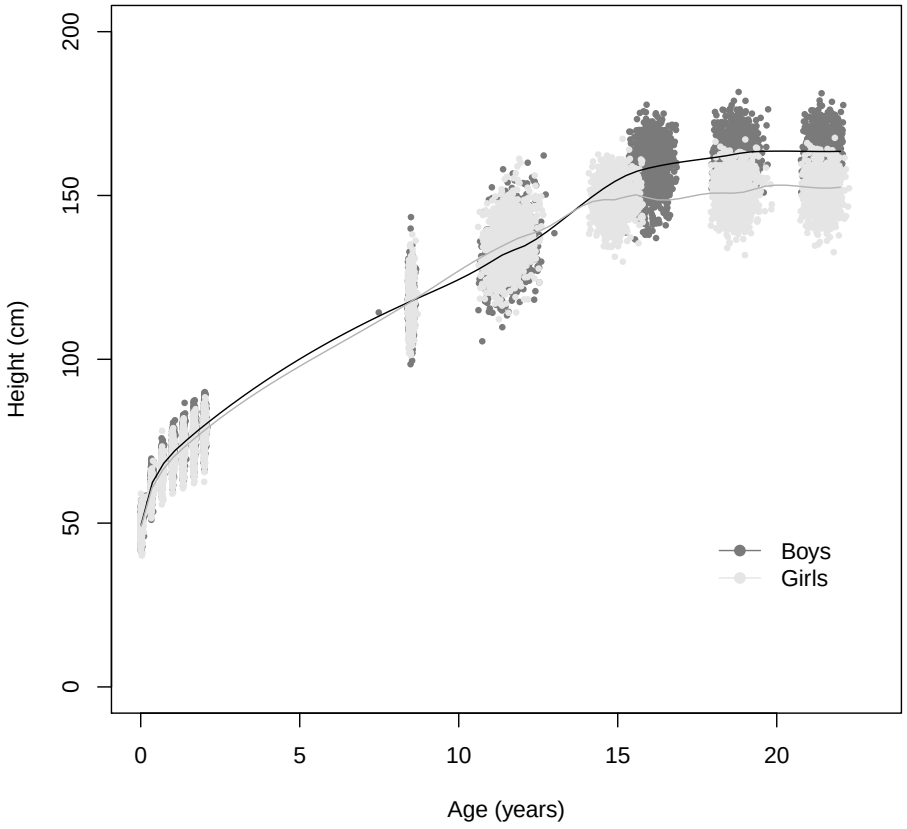

Figure 1: Height distribution by age and gender of the child. The dots correspond to the raw height measurements, while the lines correspond to spline interpolation of the mean height per gender and age of the child.

Table 1: Panel structure and range of ages by gender.

Surveys	Boys			Girls		
	N	Mean age	SD	N	Mean age	SD
Months						
Delivery 1983-4	1632	0	0	1448	0	0
Follow-up n.1	1525	2.051	.152	1353	2.051	.149
Follow-up n.2	1489	4.040	.139	1313	4.052	.170
Follow-up n.3	1439	6.051	.158	1278	6.045	.149
Follow-up n.4	1406	8.037	.126	1259	8.038	.141
Follow-up n.5	1386	10.068	.144	1239	10.068	.160
Follow-up n.6	1367	12.076	.169	1227	12.070	.164
Follow-up n.7	1342	14.072	.164	1207	14.073	.186
Follow-up n.8	1316	16.070	.178	1191	16.063	.171
Follow-up n.9	1310	18.068	.172	1197	18.054	.169
Follow-up n.10	1316	20.078	.190	1182	20.050	.161
Follow-up n.11	1302	22.047	.164	1158	22.041	.162
Follow-up n.12	1288	24.055	.153	1160	24.047	.153
Years						
1991-2 Follow-up n.13	1195	8.500	.051	1069	8.502	.044
1994-5 Follow-up n.14	1142	11.526	.405	1040	11.543	.399
1998-9 Follow-up n.15	1092	16.061	.328	997	14.905	.363
2002 Follow-up n.16	1071	18.697	.332	952	18.701	.350
2005 Follow-up n.17	993	21.467	.297	895	21.453	.312

Table 2: Descriptive statistics of the principal variables

Boys	Birth Mean	SD	Age 1 Mean	SD	Age 2 Mean	SD	Age 8 Mean	SD	Age 11 Mean	SD	Age final height† Mean	SD
N	1618		1367		1.288		1192		1138		1111	
Height (cm)	49.46	2.15	71.449	2.883	79.868	3.581	117.738	5.537	132.386	7.039	163.14	5.901
Age (years)‡	.0128	.013	1.006	.014	2.005	.013	8.500	.051	11.525	.404	20.319	1.556
Breastmilk			.753	.383	.340	.369						
Caloric intake (kcal)‡‡			331.450	283.874	670.536	328.656	1506.35	600.260	1257.38	612.153	2061.40	711.370
Diseases‡‡‡			.649	.849	.916	1.055	1.103	4.902	.476	3.019	.134	.385
Birthweight* (kg)	3.02	0.45										
Birth order*	2.59	2.46										
Normal pregnancy	88.94%											
Premature&Small*	7.53%											
Premature*	3.52%											
Mother's height*(cm)	151.58	5.02										
Prop. urban location*	.75	.42										
Family income (pesos)			93,658	153,084	84,373	130,173	123,699	163,250	141,543	174,592	222,413	1,293,341

Girls	Birth Mean	SD	Age 1 Mean	SD	Age 2 Mean	SD	Age 8 Mean	SD	Age 11 Mean	SD	Age final height† Mean	SD
N	1433		1226		1160		1067		1039		1016	
Height (cm)	49.01	2.11	69.923	2.841	78.315	3.630	117.623	5.573	135.335	7.623	151.266	5.479
Age (years)‡	.012	.013	1.005	.013	2.004	.013	8.502	.044	11.543	.399	19.906	1.965
Breastmilk			.773	.375	.343	.370						
Caloric intake (kcal)‡‡			293.536	252.486	670.093	295.044	1353.66	505.978	1140.47	514.954	1419.48	539.837
Diseases‡‡‡			.613	.793	.778	.957	.810	3.511	.417	2.767	.102	.343
Birthweight* (kg)	2.96	.43										
Birth order*	2.50	2.39										
Normal pregnancy	88.08%											
Premature&Small*	7.95%											
Premature*	3.97%											
Mother's height*(cm)	151.47	5.03										
Prop. urban location*	.74	.42										
Family income (pesos)			93,874	21,571	75,976	100,580	121,023	128,718	136,561	157,355	177,755	197,296

[a] * corresponds to time-invariant variables.
[b] † Waves 15, 16 and 17 are combined.
[c] ‡ The age at final height corresponds to the age when the maximum height is reported in the data (either at wave 15, 16 or 17).
[d] ‡‡ The caloric intake at age final height corresponds to the average kcal between waves 15, 16 and 17.
[e] ‡‡‡ The diseases correspond to the total n. of diarrhea episodes at age 1-2; the length of stay in hospital (in days) from the previous wave at age 8-11; the total n. of hospitalizations from age 11 to the age when the final height is reported in the data.

Table 3: Boys' height production function during infancy. Dependent variable: change in height

	$\Delta_{Height}1$		$\Delta_{Height}2$	
	FE	IVFE	FE	IVFE
Breastmilk age 1	0.726*	-2.594		
	[0.356]	[2.800]		
Caloric intake age 1	0.131**	-0.133		
	[0.0451]	[0.302]		
Diarrhea age 1	-0.142	-1.376*		
	[0.0882]	[0.645]		
Breastmilk age 2			0.938***	-1.174
			[0.189]	[1.984]
Caloric intake age 2			0.197***	0.298*
			[0.0210]	[0.147]
Diarrhea age 2			-0.278***	-2.214***
			[0.0540]	[0.593]
N	1359	1286	1250	1060
R-sq	0.986	0.982	0.944	0.876
Kleibergen-Paap Underif. Test		13.971*		14.713*
p-value		0.0158		0.0117
Hansen J Overid. Test		4.644		7.831
p-value		0.3258		0.098
Kleibergen-Paap Wald statistic†		1.949		2.45
F-test statistic				
Breastmilk		19.76***		5.00***
Caloric intake		38.20***		20.58***
Diarrhea		5.61***		4.91***

[a] Every model includes change in age and change in age squared between two consecutive waves.
[b] The kcal is exclusive of breast milk.
[c] A change of one unity in caloric intake corresponds to 100 kcal.
[d] Robust standard error in parenthesis.
[e] Signif. codes: (*) if p < .05, (**) if p < .01, (***) if p < .001.
[f] The Kleibergen-Paap Wald statistic is the robust version of the Cragg-Donald Wald F statistic. It is always below the Stock-Yogo critical values.

Table 4: Boys' height production function during pre-puberty and puberty. Dependent variable: change in height

	$\Delta_{Height}8$		$\Delta_{Height}11$		Δ_{Height}Final	
	FE	IVFE	FE	IVFE	FE	IVFE
Caloric intake age 8	0.0821***	0.199*				
	[0.0214]	[0.0888]				
Length of stay age 8	-0.0433*	0.162				
	[0.0210]	[0.253]				
Caloric intake age 11			0.107***	0.246***		
			[0.0162]	[0.0537]		
Length of stay age 11			-0.0356	0.776		
			[0.0236]	[0.788]		
Caloric intake age final height					-0.0688***	-0.554
					[0.0201]	[0.387]
Hospitalization age final height					-0.602	27.86
					[0.360]	[23.61]
N	1128	1123	1128	1123	1106	1099
R-sq	0.954	0.924	0.954	0.924	0.976	0.824
Kleibergen-Paap Underif. Test		10.642*		6.518*		1.71
p-value		0.0138		0.0384		0.191
Hansen J Overid. Test		1.081		0.002		
p-value		0.5825		0.9663		
Kleibergen-Paap Wald statistic		2.743		2.192		0.902
F-test statistic						
Caloric intake		23.26***		85.99***		55.33***
Length of stay/hospitalization		2.94*		2.48†		6.00**

[a] Every model includes change in age and change in age squared between two consecutive waves. In the case of final height, the age change corresponds to difference of age when the final height was measured (either wave 15 or 16 or 17) and the age at wave 14.
[b] A change of one unity in caloric intake corresponds to 100 kcal.
[c] Robust standard error in parenthesis.
[d] Signif. codes: (†) if p < .1, (*) if p < .05, (**) if p < .01, (***) if p < .001.
[e] The Kleibergen-Paap Wald statistic is the robust version of the Cragg-Donald Wald F statistic. It is always below the Stock-Yogo critical values.

Table 5: Girls' height production function during infancy. Dependent variable: change in height

	$\Delta_{Height}1$		$\Delta_{Height}2$	
	FE	IVFE	FE	IVFE
Breastmilk age 1	1.144***	3.838		
	[0.333]	[4.214]		
Caloric intake age 1	0.171***	0.363		
	[0.0470]	[0.411]		
Diarrhea age 1	-0.280**	-2.113*		
	[0.0946]	[0.905]		
Breastmilk age 2			1.066***	-0.561
			[0.206]	[2.513]
Caloric intake age 2			0.190***	0.204
			[0.0264]	[0.175]
Diarrhea age 2			-0.269***	-3.171***
			[0.0676]	[0.949]
N	1221	1156	1119	942
R-sq	0.985	0.978	0.937	0.833
Kleibergen-Paap Underif. Test		11.654*		17.96**
p-value		0.0398		0.003
Hansen J Overid. Test		0.982		5.456
p-value		0.9125		0.2436
Kleibergen-Paap Wald statistic		1.731		2.658
F-test statistic				
Breastmilk		16.25***		4.17***
Caloric intake		36.43***		19.12***
Diarrhea		3.46***		3.86***

[a] Every model includes change in age and change in age squared between two consecutive waves.
[b] The kcal is exclusive of breast milk.
[c] A change of one unity in caloric intake corresponds to 100 kcal.
[d] Robust standard error in parenthesis.
[e] Signif. codes: (*) if p < .05, (**) if p < .01, (***) if p < .001.
[f] The Kleibergen-Paap Wald statistic is the robust version of the Cragg-Donald Wald F statistic. It is always below the Stock-Yogo critical values.

Table 6: Girls' height production function during pre-puberty and puberty. Dependent variable: change in height

	$\Delta_{Height}8$		$\Delta_{Height}11$		Δ_{Height}Final	
	FE	IVFE	FE	IVFE	FE	IVFE
Caloric intake age 8	0.125***	0.0604				
	[0.0264]	[0.152]				
Length of stay age 8	-0.00995	1.173				
	[0.0486]	[1.110]				
Caloric intake age 11			0.113***	0.200*		
			[0.0235]	[0.0845]		
Length of stay age 11			-0.0325	-0.905		
			[0.0314]	[0.736]		
Caloric intake age final height					-0.0923**	-0.08
					[0.0317]	[0.278]
Hospitalizations age final height					-0.997	-9.109
					[0.554]	[30.00]
N	991	983	1020	1028	1008	1000
R-sq	0.99	0.982	0.941	0.959	0.898	0.871
Kleibergen-Paap Underif. Test		2.321		8.271*		0.316
p-value		0.5084		0.016		0.574
Hansen J Overid. Test		4.429		1.146		
p-value		0.1092		0.2844		
Kleibergen-Paap Wald statistic		0.536		2.798		0.157
F-test statistic						
Caloric intake		16.81***		78.56***		73.76***
Length of stay/hospitalization		4.18**		3.67*		1.48

[a] Every model includes change in age and change in age squared between two consecutive waves. In the case of final height, the age change corresponds to difference of age when the final height was measured (either wave 15 or 16 or 17) and the age at wave 14.
[b] A change of one unity in caloric intake corresponds to 100 kcal.
[c] Robust standard error in parenthesis.
[d] Signif. codes: (*) if p < .05, (**) if p < .01, (***) if p < .001.
[e] The Kleibergen-Paap Wald statistic is the robust version of the Cragg-Donald Wald F statistic. It is always below the Stock-Yogo critical values.

Appendix

Table 7: Descriptive statistics of the instrumental variables used.

IV variables	Definitions	Boys N	Boys Mean	Boys SD	Girls N	Girls Mean	Girls SD
Age 1							
Rainy season	rainy season	1366	0.514	0.022	1226	0.515	0.025
Evaporated price	price of 100 g evaporated milk	1324	830.214	81.098	1187	833.029	84.663
Banana price	price of 1 banana	1293	132.091	28.204	1161	130.633	29.254
Distance road †	distance (m) to nearest vehicular road	1360	263.084	493.205	1222	273.193	514.314
Refrigerator †	own a refrigerator	1366	0.066	0.042	1226	0.062	0.042
Time to infant store †	minutes walk to nearest infant store	1366	16.282	17.827	1226	17.012	18.284
Month after Nitang	distance in months between birth and the typhoon Nitang	1367	10.481	3.294	1226	10.441	3.324
Age 2							
Kerosene price	price of 1 lt of kerosene	1241	3565.118	671.498	1115	3556.290	704.747
Powder price	price of 350 g powdered milk	1084	8626.369	543.801	970	8617.647	573.267
Egg price	price of medium size egg	1196	473.714	25.502	1077	472.550	27.587
Time to infant store †	mins walk to nearest infant store	1288	16.746	18.421	1160	17.236	18.445
Distance road †	distance (m) to nearest vehicular road	1282	279.101	516.595	1156	285.455	531.207
Infant store †	presence of infant store close to home	1288	0.710	0.217	1160	0.693	0.229
Piped water †	piped water as water source for the reps house	1288	0.859	0.268	1160	0.842	0.279
Age 8							
Cooking oil price	price of 75 cl cooking oil	1189	3.041	1.368	1063	3.083	1.452
Rainy season	rainy season	1192	0.481	0.149	1067	0.488	0.151
Public health facility	presence of public health facility in the village	1189	3.172	1.655	1063	3.184	1.689
Distance road †	distance (m) to nearest vehicular road	1187	3.504	5.914	1063	3.499	5.775
Age 11							
Density	village population density	1133	13107.490	16874.120	1031	12852.760	16929.830
N. houses †	n. houses within 50 m to the resp house	1138	17.692	3.894	1039	17.575	3.995
Drinking water †	availability of drinking water inside the resp house	1138	0.849	0.293	1039	0.835	0.302
Age final height							
Density	village population density	1104	12715.640	14900.260	1008	13349.790	15409.490
N. houses †	n. houses within 50 m to the resp house	1111	17.049	4.187	1016	17.051	4.223

[a] † indicates household characteristics averaged at barangay level.

Table 8: Age 1 first-stage estimates.

	Boys			Girls		
	Breastmilk	Caloric intake	Diarrhea	Breastmilk	Caloric intake	Diarrhea
$\Delta_{age}1$	2.010**	-3.193	-0.564	1.891*	-4.712	1.411
	[0.761]	[5.048]	[1.299]	[0.751]	[5.130]	[1.403]
$\Delta_{age^2}1$	-0.993	3.231	-0.468	-1.144	5.655	-0.831
	[0.634]	[4.466]	[1.090]	[0.678]	[4.392]	[1.257]
Rainy season	-1.138	9.647*	5.133***	-0.398	7.129	1.37
	[0.694]	[4.613]	[1.444]	[0.403]	[4.189]	[0.965]
Evaporated milk price	0.000691**	-0.00372*	-0.000897	0.000477*	-0.00340*	-0.000727
	[0.000213]	[0.00156]	[0.000517]	[0.000206]	[0.00143]	[0.000420]
Banana price	-0.0003	0.0032	0.0006	-0.0003	0.0037	0.0016
	[0.000412]	[0.00277]	[0.000924]	[0.000383]	[0.00243]	[0.000882]
Distance road†	0.00004	-0.00036	-0.000272*	0.000150**	-0.000826**	-0.000251*
	[0.0000449]	[0.000286]	[0.000131]	[0.0000463]	[0.000292]	[0.000121]
Refrigerator†	-1.161***	13.72***	-0.653	-0.888*	12.18***	-0.836
	[0.330]	[2.424]	[0.760]	[0.350]	[2.215]	[0.726]
Time to infant store†	-0.0013	0.0014	0.00716*	-0.00338*	0.0155	0.00626
	[0.00130]	[0.00825]	[0.00358]	[0.00133]	[0.00879]	[0.00340]
Months after Nitang	-0.00969**	0.00812	-0.0279***	-0.0025	-0.0229	-0.0242***
	[0.00305]	[0.0227]	[0.00720]	[0.00315]	[0.0219]	[0.00684]
N	1286	1286	1286	1156	1156	1156
R-sq	0.81	0.624	0.393	0.82	0.622	0.389

[a] † indicates household characteristics averaged at village level.

Table 9: Age 2 first-stage estimates.

	Boys			Girls		
	Breastmilk	Caloric intake	Diarrhea	Breastmilk	Caloric intake	Diarrhea
$\Delta_{age}2$	2.13	-37.46*	-0.31	3.262	-52.25**	-1.738
	[1.617]	[15.12]	[4.911]	[2.086]	[16.21]	[5.782]
$\Delta_{age^2}2$	-0.717	16.47***	0.612	-1.103	20.31***	0.961
	[0.525]	[4.853]	[1.608]	[0.691]	[5.285]	[1.904]
Kerosene	0.0000258	-0.000305	0.0000854	0.0000209	-0.000627**	0.000148
	[0.0000271]	[0.000233]	[0.0000864]	[0.0000330]	[0.000223]	[0.0000770]
Powdered milk price	0.0000422	-0.00000135	-0.000237***	0.0000172	0.0000222	-0.0000883
	[0.0000259]	[0.000203]	[0.0000651]	[0.0000280]	[0.000187]	[0.0000620]
Egg price	0.000255	-0.0137***	0.00137	0.000909	-0.00654	-0.00102
	[0.000517]	[0.00389]	[0.00160]	[0.000539]	[0.00389]	[0.00149]
Time to infant store†	0.00267	0.0263	0.0225	-0.00676	0.0519	0.024
	[0.00425]	[0.0391]	[0.0155]	[0.00426]	[0.0304]	[0.0127]
Distance road†	-0.0000942	-0.00328***	-0.000516	0.0000959	-0.00365***	-0.000870***
	[0.000100]	[0.000853]	[0.000300]	[0.000122]	[0.000740]	[0.000261]
Infant food store†	-0.0385	3.280***	1.021**	0.0965	1.874**	0.827**
	[0.0977]	[0.764]	[0.317]	[0.103]	[0.698]	[0.271]
Piped water†	-0.227*	0.201	-0.424	-0.293**	1.153	-0.412
	[0.105]	[0.792]	[0.264]	[0.106]	[0.702]	[0.271]
N	1060	1060	1060	942	942	942
R-sq	0.464	0.85	0.458	0.467	0.845	0.425

[a] † indicates household characteristics averaged at village level.

Table 10: Age 8 first-stage estimates.

	Boys		Girls	
	Caloric intake	Length of stay	Caloric intake	Length of stay
$\Delta_{age}8$	8.826	-7.527	16.62**	3.807
	[6.361]	[4.302]	[5.294]	[2.926]
$\Delta_{age^2}8$	-0.615	0.719	-1.372**	-0.355
	[0.607]	[0.411]	[0.506]	[0.278]
Cooking oil price	-0.0168	-0.300**	-0.16	0.0426
	[0.240]	[0.105]	[0.162]	[0.0842]
Rainy season	-2.248	1.334	-2.632*	0.377
	[1.273]	[0.806]	[1.126]	[0.739]
Public health facility	0.458***	0.304	0.412***	0.0197
	[0.121]	[0.163]	[0.0949]	[0.0526]
Distance road†	-0.191***	0.0567*	-0.113**	-0.0326
	[0.0519]	[0.0255]	[0.0386]	[0.0201]
N	1111	1111	983	983
R-sq	0.874	0.059	0.893	0.059

[a] † indicates household characteristics averaged at village level.

Table 11: Age 11 first-stage estimates.

	Boys		Girls	
	Caloric intake	Length of stay	Caloric intake	Length of stay
$\Delta_{age}11$	9.553**	-1.153	15.09***	-0.881
	[3.357]	[1.012]	[2.850]	[1.498]
$\Delta_{age^2}11$	-0.392*	0.0554	-0.665***	0.0373
	[0.163]	[0.0489]	[0.137]	[0.0734]
Village density	0.0000597***	-0.00000692*	0.0000503***	0.00000245
	[0.0000127]	[0.00000335]	[0.0000108]	[0.00000583]
N. houses†	0.220***	0.0395	0.187***	0.0718**
	[0.0529]	[0.0290]	[0.0532]	[0.0226]
Drinking water†	3.339***	-0.00439	2.685***	-0.568*
	[0.749]	[0.400]	[0.674]	[0.285]
N	1123	1123	1020	1020
R-sq	0.834	0.028	0.856	0.027

[a] † indicates household characteristics averaged at village level.

Table 12: Age at final height first-stage estimates.

	Boys		Girls	
	Caloric intake	Hospitalization	Caloric intake	Hospitalization
$\Delta_{age}Final$	5.427***	-0.0442	3.020***	-0.0463
	[0.667]	[0.0376]	[0.527]	[0.0393]
$\Delta_{age^2}Final$	-0.127***	0.00145	-0.0675***	0.00165
	[0.0193]	[0.00110]	[0.0153]	[0.00114]
Village density	0.00000755	-0.000000967	0.0000143	0.000000581
	[0.0000170]	[0.000000747]	[0.0000118]	[0.000000815]
N. houses†	0.491***	0.00891***	0.387***	0.00262
	[0.0520]	[0.00258]	[0.0378]	[0.00240]
N	1099	1099	1000	1000
R-sq	0.893	0.137	0.885	0.091

[a] † indicates household characteristics averaged at village level.

Table 13: Boys' hybrid height production function during infancy. Dependent variable: change in height

	$\Delta_{Height}1$		$\Delta_{Height}2$	
	FE	IVFE	FE	IVFE
Family income age 1	0.00215***	0.00152*		
	[0.000523]	[0.000646]		
Breastmilk age 1	0.426	-2.417		
	[0.399]	[2.814]		
Caloric intake 1	0.0624	-0.108		
	[0.0493]	[0.300]		
Diarrhea age 1	-0.0758	-1.229		
	[0.0966]	[0.692]		
Family income age 2			0.00101*	-0.00215
			[0.000416]	[0.00147]
Breastmilk age 2			0.905***	0.534
			[0.197]	[1.891]
Caloric intake 2			0.169***	0.440*
			[0.0234]	[0.182]
Diarrhea age 2			-0.270***	-2.442**
			[0.0567]	[0.750]
N	1095	1037	1106	923
R-sq	0.986	0.983	0.947	0.865
Kleibergen-Paap Underif. Test		13.375*		15.683**
p-value		0.0201		0.0078
Hansen J Overid. Test		6.142		6.07
p-value		0.1888		0.194
Cragg-Donald Wald F statistic		2.235		1.688
Kleibergen-Paap Wald statistic		1.921		1.935
F-test statistic				
Breastmilk		13.83***		6.07***
Caloric intake		28.49***		16.57***
Diarrhea		4.84***		4.45***

[a] Every model includes change in age and change in age squared between two consecutive waves.
[b] The kcal is exclusive of breast milk.
[c] A change of one unity in caloric intake corresponds to 100 kcal.
[d] A change of one unity in income corresponds to 1000 pesos.
[e] Robust standard error in parenthesis.
[f] Signif. codes: (*) if p < .05, (**) if p < .01, (***) if p < .001.

Table 14: Boys' hybrid height production function during pre-puberty and puberty. Dependent variable: change in height

	Δ_{Height}8		Δ_{Height}11		Δ_{Height}Final	
	FE	IVFE	FE	IVFE	FE	IVFE
Family income age 8	0.000851	-0.000122				
	[0.000597]	[0.000852]				
Caloric intake age 8	0.0772***	0.199*				
	[0.0219]	[0.101]				
Length of stay age 8	-0.0433*	0.163				
	[0.0210]	[0.262]				
Family income age 11			0.00119	0.0000509		
			[0.000708]	[0.000797]		
Caloric intake age 11			0.0993***	0.245***		
			[0.0170]	[0.0588]		
Length of stay age 11			-0.0348	0.774		
			[0.0238]	[0.771]		
Family income age final height					-0.00000707	-0.000044
					[0.0000358]	[0.0000629]
Caloric intake age final height					-0.0688***	-0.554
					[0.0201]	[0.387]
Hospitalizations age final height					-0.602	27.9
					[0.360]	[23.65]
N	1118	1110	1128	1123	1106	1099
R-sq	0.99	0.989	0.955	0.924	0.976	0.824
Kleibergen-Paap Underif. Test		10.546*		6.571*		1.707
p-value		0.0144		0.0374		0.1914
Hansen J Overid. Test		1.082		0.002		
p-value		0.5821		0.9651		
Cragg-Donald Wald F statistic		2.295		0.934		0.647
Kleibergen-Paap Wald statistic		2.709		2.207		0.9
F-test statistic						
Caloric intake		18.49***		63.40***		55.16***
Length of stay/hospitalization		3.02*		2.42†		5.99**

[a] Every model includes change in age and change in age squared between two consecutive waves. In the case of final height, the age change corresponds to difference of age when the final height was measured (either wave 15 or 16 or 17) and the age at wave 14.
[b] A change of one unity in caloric intake corresponds to 100 kcal.
[c] A change of one unity in income corresponds to 1000 pesos.
[d] Robust standard error in parenthesis.
[e] Signif. codes: (†) if p < .1, (*) if p < .05, (**) if p < .01, (***) if p < .001.

Table 15: Girls' hybrid height production function during infancy. Dependent variable: change in height

	$\Delta_{Height}1$		$\Delta_{Height}2$	
	FE	IVFE	FE	IVFE
Family income age 1	0.000682*	0.00143		
	[0.000336]	[0.00146]		
Breastmilk age 1	1.037**	4.195		
	[0.365]	[5.109]		
Caloric intake age 1	0.138**	0.425		
	[0.0532]	[0.427]		
Diarrhea age 1	-0.257*	-2.393**		
	[0.102]	[0.877]		
Family income age 2			0.00023	-0.000885
			[0.000499]	[0.00170]
Breastmilk age 2			1.038***	-0.638
			[0.224]	[2.904]
Caloric intake age 2			0.177***	0.225
			[0.0294]	[0.251]
Diarrhea age 2			-0.242***	-3.438**
			[0.0727]	[1.228]
N	1003	954	989	824
R-sq	0.985	0.977	0.937	0.812
Kleibergen-Paap Underif. Test		7.256		13.362*
p-value		0.2023		0.0202
Hansen J Overid. Test		1.476		3.222
p-value		0.8309		0.5214
Cragg-Donald Wald F statistic		0.899		1.87
Kleibergen-Paap Wald statistic		1.089		2.021
F-test statistic				
Breastmilk		10.36***		3.80***
Caloric intake		27.75***		15.90***
Diarrhea		4.66***		3.60***

[a] Every model includes change in age and change in age squared between two consecutive waves.
[b] The kcal is exclusive of breast milk.
[c] A change of one unity in caloric intake corresponds to 100 kcal.
[d] A change of one unity in income corresponds to 1000 pesos.
[e] Robust standard error in parenthesis.
[f] Signif. codes: (*) if p < .05, (**) if p < .01, (***) if p < .001.

Table 16: Girls' hybrid height production function during pre-puberty and puberty. Dependent variable: change in height

	Δ_{Height}8		Δ_{Height}11		Δ_{Height}Final	
	FE	IVFE	FE	IVFE	FE	IVFE
Family income age 8	0.00318**	0.00302				
	[0.00112]	[0.00175]				
Caloric intake age 8	0.101***	0.0438				
	[0.0270]	[0.144]				
Length of stay age 8	-0.013	0.918				
	[0.0473]	[0.979]				
Family income age 11			0.00128	0.00118		
			[0.000870]	[0.00106]		
Caloric intake age 11			0.106***	0.184*		
			[0.0241]	[0.0899]		
Length of stay age 11			-0.0337	-0.913		
			[0.0318]	[0.737]		
Family income age final height					-0.00252*	-0.000706
					[0.00104]	[0.00401]
Caloric intake age final height					-0.0707*	-0.0513
					[0.0326]	[0.190]
Hospitalizations age final height					-0.867	-11.15
					[0.553]	[24.04]
N	991	983	1028	1020	1007	999
R-sq	0.99	0.985	0.96	0.941	0.899	0.856
Kleibergen-Paap Underif. Test		2.511		8.257*		0.547
p-value		0.4733		0.0161		0.4594
Hansen J Overid. Test		5.569		0.987		
p-value		0.0618		0.3204		
Cragg-Donald Wald F statistic				1.045		0.284
Kleibergen-Paap Wald statistic				2.79		0.272
F-test statistic						
Caloric intake		12.37***		61.94***		58.84***
Length of stay/hospitalization		3.29*		3.39*		0.75

[a] Every model includes change in age and change in age squared between two consecutive waves. In the case of final height, the age change corresponds to difference of age when the final height was measured (either wave 15 or 16 or 17) and the age at wave 14.
[b] A change of one unity in caloric intake corresponds to 100 kcal.
[c] A change of one unity in income corresponds to 1000 pesos.
[d] Robust standard error in parenthesis.
[e] Signif. codes: (†) if p < .1, (*) if p < .05, (**) if p < .01, (***) if p < .001.