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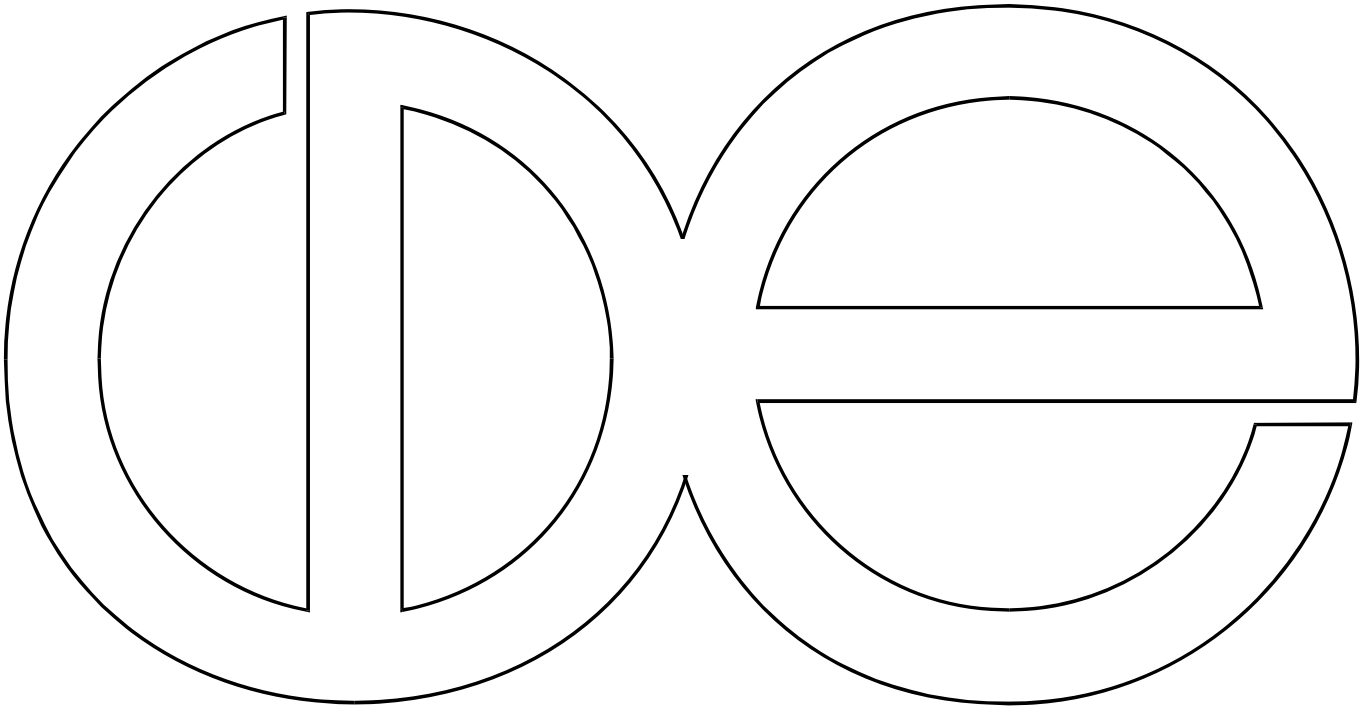

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Abstract

We know surprisingly little about the long reach of changes in education and fertility, their intergenerational implications and the long term impact on human capital formation and aggregate economic growth. The purpose of this paper is to propose a simple framework to understand how changes in education levels in one generation could promote lower fertility levels in current and in subsequent cohorts of females, increase the average health status of children, and promote increased educational attainment and higher levels of human capital. We formulate a dynamic model with feedbacks to represent the relations that link the fate of one generation to that of the next one. Our results, obtained using data drawn from the Cebu Longitudinal Health and Nutrition Survey, show important feedback effects and that any intervention that exogenously alters the birth interval distribution will have important payoffs in terms of educational attainment and that the payoffs will be spread over several generations.

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1. Introduction: education and fertility

Empirical evidence supporting the idea that increases in female education create conditions that trigger, modulate and accelerate fertility decline is massive. This evidence comes from both the historical experience of Western Europe and North America and more recent experiences in low income countries. Although there are still large patches of the observed patterns that are puzzling — e.g. the highly compressed nature of fertility changes in many countries — we know a fair amount about the linkages between individual education and couples' fertility decision-making as well as the pathways relating mass education, "Westernization," diffusion processes, and the overhaul of individual reproduction decisions.

Yet we know surprisingly little about the long reach of changes in education and fertility, their intergenerational implications and the long term impact on human capital formation and aggregate economic growth. The shift from a regime where illiteracy and high fertility are pervasive to one where virtually everybody completes elementary school, a sizeable majority makes it to high school and only a minority continues to reproduce at levels equivalent to 3 or more children per generation, contains the conditions for a reinforcing feedback mechanism whereby small shifts in the levels of women's education in one generation may trigger even larger shifts in the next via fertility reduction alone. Lower family size in one generation facilitates increased investments in children's education and this, in turn, erodes further the props of high fertility in the younger generations.

The purpose of this paper is to propose a simple framework to understand how changes in education levels in one generation could promote lower fertility levels in

current and in subsequent cohorts of females, increase the average health status of children, and promote increased educational attainment and higher levels of human capital. These changes in turn, set the stage for economic growth and self-sustained fertility reductions. We identify a handful of mechanisms through which changes in fertility may impact early environments and influence children's propensities to contract illnesses, their nutritional status, the pace of their physical growth and development, and the formation of skills and aptitudes (both cognitive and non-cognitive) thereby setting the course for scholastic achievement and ultimately educational attainment. We formulate a dynamic model with feedbacks to represent the relations that link the fate of one generation to that of the next one. We estimate parameters using an established and well-known data set from a low-income country.

2. Shifts in Education, Effects on Fertility and on Child Health: An Illustration

Assume that there is an exogenous improvement in female educational attainment in one generation that induces a permanent reduction in completed family size, and is also accompanied by shifts in the tempo of marriage and of births. The canonical model suggests that these changes will alter children's environments and early experiences because of reduced sibship size and reduction in average parity, increases in average length of birth intervals, and increases in breastfeeding uptake and average duration. Spillover effects include reduced intra-family child competition for resources (including

parental care), net increases in parental investments, and better per capita access to child health care.[1]

In turn the foregoing changes will affect (a) child nutritional status, (b) frequency of episodes of illnesses, and (c) physical and mental development. Birth order is known to have important effects on IQ (Black et al. 2011) and strategic 'soft skills' (Berglund et al. 2005) just as much as increased nutritional status is known to affect mental abilities (Brown and Pollitt 1996; Mendez and Adair 1999; Pollitt et al. 1995), psychological well-being (Pollitt et al. 1996; Walker et al. 2007), social adjustments and physical development (Habicht and Martorell 2010; Kristjansson et al. 2006). Nutritional status and illnesses exert powerful influences on school absenteeism and on the ability of children to learn and develop traits whose acquisition are relevant for educational attainment (Alderman et al. 2006; Glewwe and Jacoby 1995; Glewwe and Miguel 2008; Martorell et al. 2010; Spernak et al. 2006) and labor market experience (Case and Paxson 2008; Haas et al. 2011; Hoddinott et al. 2008).

There is uncontroversial evidence suggesting that conditions experienced in early childhood have a vast array of influences on the adult lives of individuals, affecting their adult health, educational attainment and labor market experiences (Heckman 2007; Cunha and Heckman 2009; Palloni 2006; Victoria et al. 2008). Barker's fetal programming hypothesis lays out the foundation of a growing body of work that uncovers multiple linkages between early exposure and adult conditions (Barker 1995;

[1] Eventually, decreases in fertility may also translate into higher levels of investments in infrastructure for education, better trained teachers, and reductions in the density of students per unit of school resources, and greater incentives for higher education.

Godfrey 2002; Godfrey and Barker 2000). Although in its original form the idea referred only to connections between perinatal conditions and adult health, it has been effectively generalized. There is now a large body of research that suggests the relation between early insults and stresses and the programming of brain architecture via epigenetic changes and misfiring of the HPA axis. This area has been advancing rapidly through work by Gluckman and Hanson and others (Gluckman and Hanson 2004; Hochberg et al. 2011; Shonkoff et al. 2009; Center on the Developing Child 2010). Finally, there is growing evidence that adverse early experiences disrupt the acquisition of personality traits as well as emotional and cognitive development (Shonkoff et al. 2009; Caspi et al. 2003; Danese et al. 2009; Moffitt et al. 2011). Of foremost importance are experiences of episodes of disease, poor nutrition and insufficient childhood family investments which tightly regulate the development of mental abilities, emotional and psychological well-being, and physical growth. These, in turn, are known to affect the acquisition of cognitive and non-cognitive traits that are differentially rewarded in labor markets.

One implication of these relations is that education levels and human capital formation are determined early on in the lives of individuals, well before they enter the labor market, perhaps even before they complete middle school. Another implication of these findings is that economic outcomes at any particular time are a function of investments made many years before so that weaknesses therein cannot be undone in one or even two generations. Recent empirical evidence suggests the existence of "stickiness" of poverty and poor human capital formation (Bird 2007; Magnuson and Votruba-Drzal 2009) so that if family backgrounds (partially determined by completed family size) at year *t* cannot alleviate poor childhood conditions they will also constrain the children's

own contribution to their own adult human capital formation and, indirectly, to aggregate economic growth and inequalities. The ripple effects of poor early conditions are significant.

From the above it follows that, within the arch of one generation, the initial imaginary improvement in maternal education should lead not just to lower completed family size and lower aggregate rates of population increase and density in one generation but also to improved early child health status, better child growth and development, and increased access to resources that promote acquisition of human capital relevant traits, including education attainment. From this the main conjecture follows: *by way of fertility reductions and their effects on child physical and emotional health, exogenous education improvements lay out the ground for further education improvements, increases in human capital, stronger conditions for aggregate economic growth, better adult health and decreased pressure toward aggregate inequality.*

3. Estimation of the Magnitude of Effects and the Strength of Feedback

The main goals of this paper are to estimate (a) the effects of changes in maternal fertility on selected indicators of child health, physical growth and development, (b) the strength of the relation between early health, physical growth and development and the acquisition of human capital-related traits, including cognitive skills and education, (e) evaluate the strength of the feedback mechanism whereby increased education in the children's generation promotes further fertility decline and continued progress in educational attainment. The last step is the most important in the sequence: By assembling estimates from (a), (b) and (c) and choosing parameters that describe macro relations between the

stock and growth of human capital and economic growth, one can assess the potential strength of the feedback effect: Given macro-economic and institutional conditions, how large is the return of increased education in one generation in terms of increasing educational attainment in the next?

To make the task manageable we take the relation between increased maternal education and fertility as given, known and exogenous. In particular, we assume that the effect of increased maternal education on completed family size, length of birth intervals and breastfeeding are known. We then answer the following three questions:

i. How do levels and timing of childbearing influence nutritional intake, experience with illnesses and, ultimately, nutritional status, and physical growth and development in the first two years of life?

ii. How do physical growth and development in the first two years of life translate into cognitive and non-cognitive abilities and educational attainment?

iii. What is the effect of an initial increase in maternal education on levels of education in the next generation?[2]

In the following sections we describe the data set, propose a simple model and then estimate parameters. In the last section the estimated parameters are used as inputs in a simple simulation to assess the strength of the feedback mechanism.

4. Relations and simple models

[2] Of relevance, but beyond the scope of this paper, is an assessment of feedback effects expressed in a different currency: How large are the payoffs to initial educational improvements in terms of aggregate income inequality, better adult health, and reduced health disparities in succeeding generations?

In what follows we identify a set of relations linking maternal education and fertility, on one hand, and child health, child cognition and educational attainment, on the other. We also describe a simple model to estimate some of these relations and the feedback mechanisms. Finally, we describe the data set we use to generate estimates.

4.1 Relations between fertility, birth intervals and child health and development

The relations between maternal education and fertility dynamics (levels and patterns) have been empirically assessed in detail since the 1980's and are reasonably well understood. Thus, we know that short previous and following birth interval have a very large effect on mortality (Hobcraft et al. 1983; Palloni and Millman 1986; Rutstein 2005) so that shorter birth intervals significantly increase mortality risks of exposed children. The mechanisms that explain these relations are: maternal depletion, increased child competition for scarce resources, interrupted breastfeeding, diminished parental investments and child neglect. The same mechanisms should erode nutritional and health status thereby hindering children's physical and emotional development.

We also know that longer and sustained breastfeeding provides a strong source of nutritional intake, promotes neurological and brain development and strengthens the formation of the immune system. Absence of or short breastfeeding increases children' propensity to become malnourished, incidence of childhood infectious diseases, and mortality risks (Palloni and Millman 1986; Schack-Nielsen and Michaelsen 2007). When breastfeeding is disrupted or altogether halted as a consequence of new conceptions, children become exposed to higher risks of malnutrition, more susceptible to infectious diseases and less able to recover from episodes of disease. These, in turn, are impacts that will translate into effects on physical, mental and emotional growth and development of

both children whose births bound a birth interval (Knudsen 2004; Pollitt et al. 1995, 1996, 1998; Power et al. 2006; Roncagliolo et al. 1998; Goosby and Cheadle 2009)

4.2. Models

Figure 1 displays the main relations of interest. These relations can be expressed as a simple system of equations. We first estimate an equation for initial health outcomes (at birth) and subsequently throughout the first two years of life. The following is a most basic representation:

$$b(B_i(0)) = \alpha + \beta X_i(0) + \varepsilon(o) \quad (1)$$

$$s(S_i(t)) = \gamma_s + \varphi_s Z_{si}(t-k) + \varepsilon_s(t) \quad (2)$$

$$w(W_i(t)) = \gamma_w + \varphi_w Z_{wi}(t-k) + \varepsilon_w(t) \quad (3)$$

$$c(C_i(t)) = \gamma_c + \varphi_c Z_{ci}(t-k) + \varepsilon_c(t) \quad (4)$$

$$e(E_i(t)) = \gamma_e + \varphi_e Z_{ei}(t-k) + \varepsilon_e(t) \quad (5)$$

where $B_i(0)$ is a four-category discrete variable combining low/normal birthweight with preterm/term birth for child i, $X_i(0)$ is a vector of characteristics defined before birth; they include maternal education, age at birth, two indicators of maternal poverty (availability of water and dwelling's materials), and a dichotomous variable for length of previous birth interval. $S_i(t)$ and $W_i(t)$ are dichotomous variables for stunting and wasting at various ages t. $Z_{si}(t-k)$ and $Z_{w}j(t-k)$ are vectors of characteristics that include $X_i(0)$, $B_i(0)$, dichotomous variables for a following conception in the time interval (t-k,t), a dichotomous variable for breastfeeding continuation at age t-k, a dichotomous variable for illness in the interval (t-k, t) and lagged values of wasting and stunting. $C_i(t)$ is an

indicator for cognitive scores evaluated at time t and $E_i(T)$ an indicator for educational attainment evaluated at a terminal time T. The vectors $Z_{ci}(t\text{-}k)$ and $Z_{ej}(t\text{-}k)$ are as before but they also include lagged values of wasting and stunting. The symbols β and φ·s are for effects (regression coefficients), ε for errors and b through e for functional forms.

[Figure 1 about here]

5. Data and measures

Data for this project were drawn from the 1983-84, 1994-95, 2002, and 2005 waves of the Cebu Longitudinal Health and Nutrition Survey (CLHNS) study. The CLHNS study is part of an ongoing study of a cohort of Filipino women who gave birth between May 1, 1983, and April 30, 1984. In order to identify all births, including preterm births, 3327 women were interviewed during the 6th to 7th month of pregnancy. Additional waves took place immediately after birth and then at bimonthly intervals for 24 months. There are a total of 3,080 non-twin live births (index children). Detailed health, nutrition, demographic, and socioeconomic data were collected in each survey.

For each available index child, the 1994-95 follow-up gathered information on diet, health history (major illnesses, immunizations, and hospitalizations), nutritional status (based on anthropometry), schooling, and IQ, as well as tests of mathematics, and Cebuano and English reading skills. The 1998 follow-up study collected anthropometric measures, self-assessed pubertal development, diet, major illnesses, physical activity, school attainment, IQ, parent–child communication, and reproductive health. In 2002 and 2005, the information gathered for the index children focused on their schooling

outcomes and entry into the labor force while continuing to monitor their health and anthropometry.[3,4] Measures and indicators are described in detail in the Appendix.

6. Results

6.1. Descriptive Analysis

Our sample only includes children for whom there is information on educational attainment in 2005, when index children were around 20 years old. As a result the sample for estimation consists of 1,984 index children out of the original 3,080 live births. All estimates are obtained in this sample, without correction for attrition.[5] Table 1 displays the most important statistics of the sample.

[Table 1 around here]

6.2. Estimates

[3] For detailed information on the CLHNS study refer to Adair and Popkin (2001) and Adair et al. (2011).

[4] The Philippine Nonverbal Intelligence Test (PNIT) was used for measuring IQ. The PNIT is a 100-item test for children aged 5 to 14 and was designed to assess analytical skills in the Filipino cultural context (Guthrie et al. 1977). The Cebuano and English reading comprehension and the mathematics tests (29, 59, and 58 items respectively) were based on official school curricula at various grades in 1994-95 and a limited survey in 1996-97 (Bacolod and Ranjan 2008; Glewwe et al. 2001).

[5] It should be noted, however, that we are in no way different from the vast majority of studies that use the CEBU data as we are not aware of a single one that pays due attention to selective attrition.

Selected results are displayed in Table 2.[6] The most important results can be summarized as follows: (a) there are strong effects of preceding interval on LBW/PREM and on stunting at virtually all ages; (b) these effects are remarkably persistent throughout the life of a child as they affect not only stunting and wasting (results for wasting are not shown) but also cognition as well; the effects of stunting and wasting on cognition and high school graduation are large and statistically significant, (c) the effects of maternal education on child health status indicators is diluted once all the other variables are controlled for but (d) consistent and pervasive effects of maternal education on cognitive traits and child's educational achievement remain across all outcomes throughout the period of observation.

[Table 2 around here]

These results lend themselves to a simple interpretation: a child in generation G+1 starts out with two signatures: length of preceding interval and birth weight and prematurity. This defines its health status at time *0*. Both these characteristics depend on maternal education at generation G. Length of preceding interval as well as birth weight and prematurity determine risks of stunting and wasting until the second year of life. In turn, stunting and wasting at any time t are also dependent on preceding stunting and

[6] To abbreviate presentation of results and to simplify the simulation exercise required for the estimation of a feedback effects, we only show estimated effects and corresponding standard error for some key variables used later to estimate feedback effects. In particular, we do not show (and do not use in our simulation) estimates of the effects of breastfeeding and following conception. By the same token, we ignore wasting and consider only role of stunting.

wasting in addition to direct effects of length of preceding birth interval and maternal education. As shown before, both maternal education and length of preceding interval have persistent and enduring effects. In addition, cognition at two points in time is influenced by length of birth interval, lagged stunting and wasting as well as by maternal education. Cognition at a second point in time is strongly determined by its lagged values as well. Finally, and as expected, the likelihood of graduating from HS depends on past cognitive scores, preceding growth faltering, maternal education and by length of preceding birth interval.

These relations suggest a system of relations with a potentially powerful feedback mechanism leading to one or more steady states in the distribution of population by educational attainment (see Figure 1). Let's suppose that through the implementation of family planning we are able to introduce an exogenous shock among mothers in generation G that redistributes the duration of birth intervals among those in generation G+1 in such a ways that the proportion of short and very short birth intervals is reduced. This will change the distribution of birth by birth weight and prematurity. Both changes will have ripple effects as follows: (a) there will be a reduction of growth faltering at times t, t+1, t+3,…T; (b) cognitive performance will be improved for ages above 7 or 8 and (c) educational attainment will be increased. Thus the original behavioral shock in generation G translates into higher educational attainment in generation G+1 and this, in turn, should be experienced as a behavioral shock that redistributes births by length of preceding birth intervals and LBW/Prematurity in generation G+2. The consequence will be an improvement in cognitive scores and an additional upward bump to the high school graduation rate. The system will be ratcheted upwards until prevalence of levels of

educational attainment reaches a ceiling or saturates whereupon only stochastic variability prevail.

6.3. Feedback effects

To test the conjecture of feedback effects and to assess the magnitude of the effects of changes in distribution of birth intervals on educational attainment we designed a simple simulation using the estimates from the model. Throughout we ignore differential fertility by generation and the effects from breastfeeding, following birth intervals and of wasting. Ignoring these determinants will produce estimates of lower bounds for the effects we are trying to assess. The simulation proceeds in two stages, calibration and assessment.

i. Calibration: we start out with a population (generation G) where only 35% of individuals have attained high school. We then simulate what the high school graduation rate will be in generations *G*+1, *G*+2, etc…. until the prevalence of high school graduation stops changing. This prevalence is consistent with the estimates of the model (is calibrated to be consistent with estimates).

ii. Assessment: In a second step we alter the distribution of birth intervals so that the prevalence of short preceding birth intervals drops from a high value of .35 to a low value of .10 in steps of .05 and assess the high school graduation rate of generation *G*+1[7]. Since a single simulation uses the estimates from the models as well as the distribution of errors

[7] This is where the assumption about an exogenous relation between education and fertility enters. We do not estimate the effects that fertility changes have on the distribution of birth intervals (or breastfeeding) from the CEBU data but instead we assume that the relation is similar to that found in other data sets (from WFS and DHS).

from each equation, all results are subject to Monte Carlo variation. We then repeat the simulation 50 times.

The prevalence of high school graduates associated with each scenario (including the one with 35% of short birth intervals) is plotted in Figure 2. Note that the x axis corresponds to scenarios with proportion of short birth intervals going from .4 to .10 in jumps of .05.

[Figure 2 around here]

The impact of an exogenous shock on the distribution of length of birth intervals is initially quite large but, as one should expect, improvements are non-linear and tend to decelerate with the passage of generations.

7. Discussion

Our results show that the system of relations estimated from the data contains important feedback effects and that any intervention that exogenously alters the birth interval distribution, i.e. an efficient family planning program, will have important payoffs in terms of educational attainment and that the payoffs will not be concentrated solely in one generation but will be spread over several of them.

Our study has several limitations. First, our models are too simple. For example, we ignored maternal depletion, general child health, measures of child competition for scarce resources, the effects of changes in parental investments and child preferences and neglect. Second, we assume somewhat naively (and only for simplification purposes) that

nothing other than health and cognitive inputs matter for educational progress. What we ignore is quite vast as it includes investments in infrastructure, changing distribution of quality of teachers, geographic and regional constraints, etc. Third, we do not consider explicitly the effects of parental investments either exogenous or prompted as reaction to a child's health status or school progress. Fourth, we ignore the effects of breastfeeding and following conception and only focus on measures of extreme health failure (stunting and wasting). As a consequence of these decisions our estimates of the importance of fertility dynamics for cognition and educational achievement and the consequent feedback effects are surely underestimated. We only claim to produce a lower bound estimate of both parameters.

A final shortcoming is that, like all other studies of the CEBU data, we ignore selective attrition and missingness. Therefore, we should be cautious with our inferences since selective attrition may be consequential for the magnitude, direction and statistical significance of estimates.

Despite these shortcomings our results remain relevant. They show that the relations involving education, fertility, health and some aspects of human capital contain an important amount of inertia that can lead to favorable developments: once the distribution of children by health and nutritional status is improved as a response to an initial shift in mothers' education, there will be a push toward further improvements that takes place by boosting cognition and educational attainment among the children of mothers who benefited from the initial improvement.

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Figure 1:

Potential Educational Attainment: Social Context

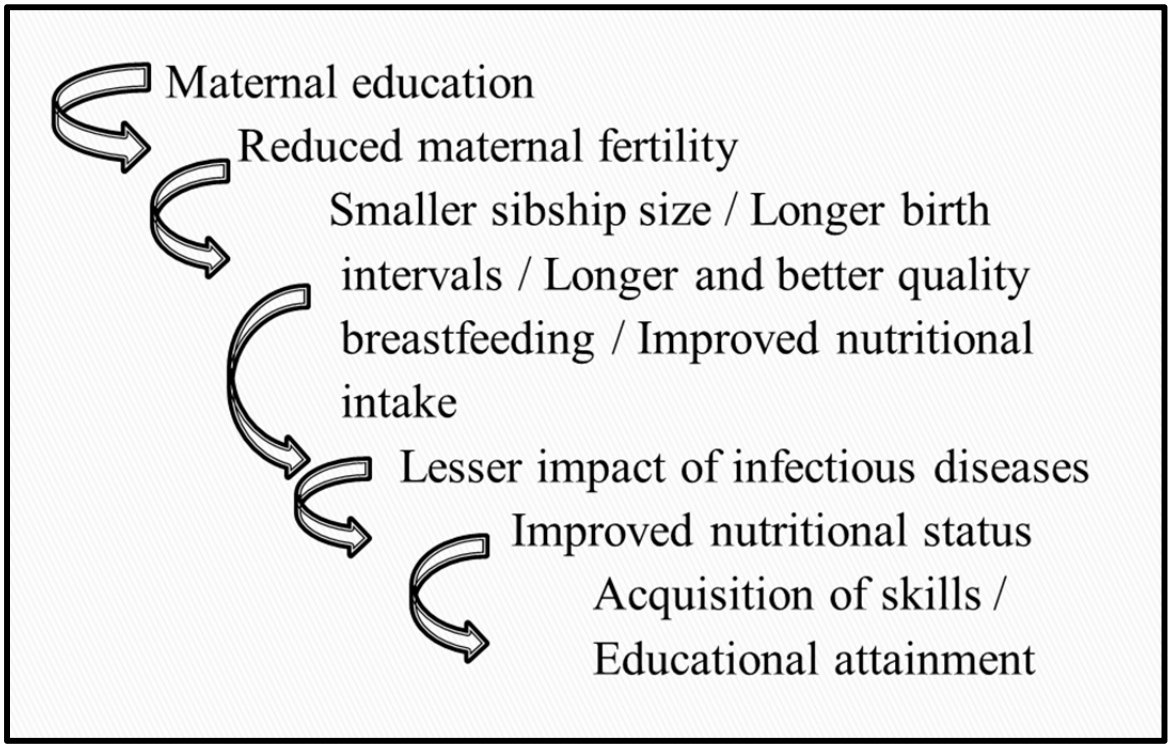

Figure 2:

Increases in high school rates after exogenous shock

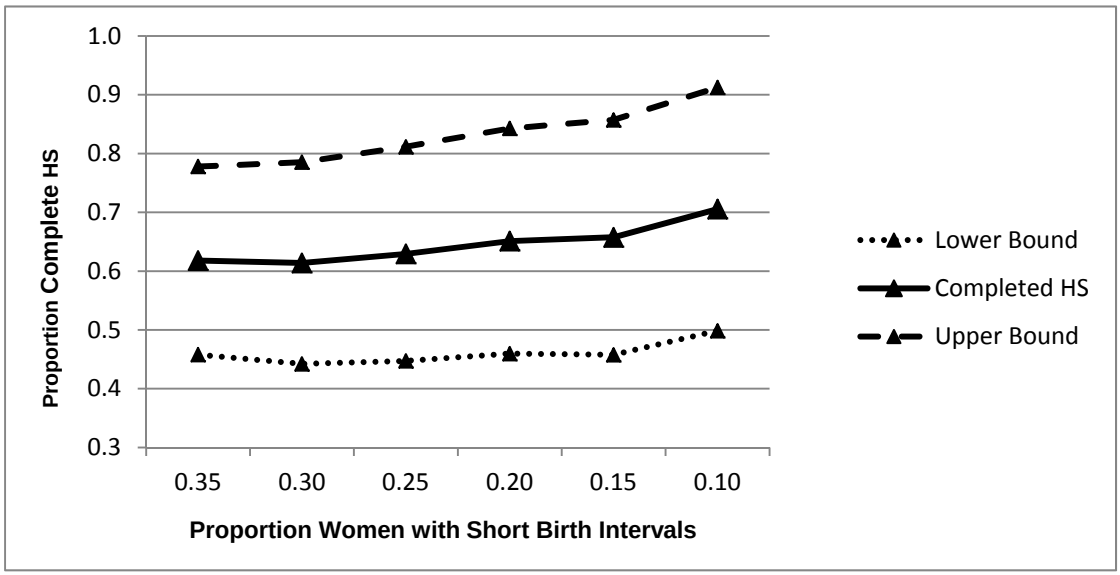

Table 1:

Summary statistics (percentage)

Variable	%	St. Dev.
Birth weight and prematurity		
Normal birth weight and not premature	81.49	38.85
Low birth weight and not premature	3.87	19.30
Normal birth weight and premature	10.20	30.28
Low birth weight and premature	4.44	20.60
Mother's education		
Some primary education	33.71	47.29
Some High School education	40.32	49.08
Some College education	25.97	43.87
Mother's age at birth		
Between 0 and 20 years old	8.40	27.76
Between 21 and 39 years old	88.20	32.28
At least, 40 years old	3.40	18.13
Birth order		
First or second child	29.27	45.52
Previous birth interval		
Less than 2.5 years	61.19	48.75
2.5 and 4 years	24.93	43.28
At least, 4 years	13.88	34.59
Who were stunted at the		
2^{nd} bimester	15.49	36.19
4^{th} bimester	25.31	43.50
6^{th} bimester	38.90	48.78
8^{th} bimester	54.58	49.81
12^{th} bimester	63.93	48.04
Who were wasted at the		
2^{nd} bimester	5.48	22.76
4^{th} bimester	6.80	25.18
6^{th} bimester	10.76	31.01
8^{th} bimester	9.92	29.90
12^{th} bimester	5.95	23.67
N	1059	

Source: Cebu Longitudinal Health and Nutrition Survey (CLHNS)

Table 2:

Effects estimates – Logistic models selected outcomes

Predictor	Outcome				
	LBW/ Premature	**Stunted 2nd Bimester**	**Stunted 4th Bimester**	**Stunted 6th Bimester**	**Stunted 8th Bimester**
Mother's Educational Attainment (ref. Some Elementary School)					
Some High School	-0.21(0.15)	0.22(0.20)	-0.02(0.18)	0.12(0.20)	-0.06(0.21)
Some College	-0.56(0.19)	-0.25(0.25)	-0.24(0.22)	-0.34(0.23)	0.06(0.23)
Previous Birth Interval (ref. 4 Years or more)					
Less than 2.5 Years	0.43(0.22)	0.60(0.30)	0.11(0.25)	0.43(0.26)	0.07(0.26)
2.5 to 4 Years	0.22(0.25)	0.37(0.32)	-0.35(0.28)	0.15(0.30)	0.44(0.30)
LBW/Prematurity (ref. Normal Birth Weight and not Premature)					
LBW/Premature		0.81(0.37)	0.04(0.45)	0.51(0.50)	0.66(0.61)
LBW/Not Premature		-0.13(0.29)	0.07(0.26)	-0.11(0.28)	0.08(0.30)
Normal W /Premature		1.77(0.32)	-0.54(0.40)	0.75(0.50)	0.16(0.53)
Stunted 2nd Bimester (1=Yes; 0=No)		2.43(0.20)	1.65(0.23)	0.80(0.31)	1.43(0.42)
Stunted 4th Bimester (1=Yes; 0=No)				1.11(0.25)	-0.38(0.33)
Stunted 6th Bimester (1=Yes; 0=No)					1.77(0.27)
Stunted 8th Bimester (1=Yes; 0=No)					
Stunted 12th Bimester (1=Yes; 0=No)					
IQ: Age 7 years (1=Yes; 0=No)					
IQ: Age 11 years (1=Yes; 0=No)					
N	1449	1398	1348	1312	1276
D(f)	9	13	15	17	19
LL	-953	-476	-522	-475	-435

Note: Estimated effects on log odds. All models control for an indicator of poverty (housing material quality), and indicator of hygiene (availability of potable water), birth order and age of mother at the time of the birth. SE in parentheses

Table 2- Continuation:

Effects estimates – Logistic models selected outcomes

Predictor	Outcome			
	Stunted 12th Bimester	**IQ Age 7 Years**	**IQ Age 11 Years**	**High School**
Mother's Educational Attainment (ref. Some Elementary School)				
Some High School	0.11(0.25)	-0.62(0.15)	-0.64(0.17)	0.26(0.16)
Some College	-0.51(0.28)	-0.97(0.20)	-0.95(0.23)	0.93(0.22)
Previous Birth Interval (ref. 4 Years or more)				
Less than 2.5 Years	0.64(0.32)	0.48(0.23)	-0.09(0.25)	0.08(0.23)
2.5 to 4 Years	0.71(0.35)	0.13(0.25)	0.01(0.27)	0.14(0.25)
LBW/Prematurity (ref. Normal Birth Weight and not Premature)				
LBW/Premature	0.99(0.76)	0.51(0.34)	-1.03(0.45)	0.23(0.39)
LBW/Not Premature	0.62(0.38)	0.31(0.21)	0.38(0.23)	0.29(0.25)
Normal W /Premature	-0.84(0.58)	-0.05(0.34)	-0.25(0.37)	-0.01(0.35)
Stunted 2nd Bimester (1=Yes; 0=No)	0.13(0.47)	0.04(0.22)	0.10(0.25)	-0.09(0.24)
Stunted 4th Bimester (1=Yes; 0=No)	0.21(0.40)	0.10(0.22)	0.09(0.24)	0.20(0.24)
Stunted 6th Bimester (1=Yes; 0=No)	0.77(0.36)	0.53(0.23)	-0.05(0.25)	0.14(0.24)
Stunted 8th Bimester (1=Yes; 0=No)	0.35(0.29)	-0.13(0.23)	0.05(0.26)	-0.48(0.25)
Stunted 12th Bimester (1=Yes; 0=No)		-0.09(0.25)	-0.16(0.28)	-0.37(0.27)
IQ: Age 7 years (1=Yes; 0=No)			1.92(0.16)	-0.79(0.17)
IQ: Age 11 years (1=Yes; 0=No)				-0.86(0.17)
N	1226	1211	1209	1068
D(f)	23	24	25	26
LL	-311	-659	-551	-584

Note: Estimated effects on log odds. All models control for an indicator of poverty (housing material quality), and indicator of hygiene (availability of potable water), birth order and age of mother at the time of the birth. SE in parentheses

Appendix: Measures and Indicators

Mother's Educational Attainment: We defined two dichotomous variables for maternal education, one for having some high school (HS) education and the other for having some college education. Mother Some HS=1 if mother's highest educational attainment was some HS education; 0 otherwise. Mother Some College=1 if mother's highest educational attainment was some college education; 0 otherwise. Therefore, the reference category is less than some HS education.

Age of the Mother: We defined two dichotomous variables for mother's age, one for less than 20 years and the other for over 39. Mother's Age<20=1 if mother's age < 20 years; 0 otherwise. Mother's Age 40+=1 if mother's age ≥40 years; 0 otherwise. Therefore, the reference category is mother's age between 20 and 39.

Poverty Indices: Two indices of poverty were defined: One for water availability and the other for quality of the materials of the house. Water Availability=1 if the child's home has water supply; and 0 otherwise. Strong Home =1 if the child's home was a concrete or wooden one with galvanized iron roofing; and 0 otherwise.

Birth Weight and Prematurity: We defined two dichotomous variables, one for low birth weight (LBW) and other for prematurity. LBW=1 if the index child weighed less than 2.5 kg at birth; and 0 otherwise. Prematurity=1 if the index child was born prematurely (less than 37 weeks of gestation). We also defined a categorical variable combining all for possible states for being born prematurely or not and having LBW or

not. The reference category is the best possible one, that is to say not being premature and not having low weight at birth. Therefore, LBW/Premature = 0 if baby had normal birth weight and was not premature; = 1 if baby had LBW and was not premature; = 2 baby had normal birth weight and was premature; and = 3 if baby had LBW and was premature.

Length of Previous Birth Interval: The interval in months between the index child birth and the previous birth was estimated as the difference between the index child birth date and the previous pregnancy date of birth. For stillbirths, miscarriages and abortions the date in which any of these events took place was used. If the index child was the first born then length of the previous birth in months was zero. Using the estimation of previous birth interval in months two dichotomous variables were defined: Previous Birth Interval ≤2.5 Years=1 if the length of the previous birth interval is between 0 and 30 months; and 0 other wise. Previous Birth Interval 2.5 to 4 Years= if the length of the previous birth interval is between 31 and 48 months; and 0 otherwise. Therefore, the default category is previous birth interval greater than 4 years or zero (the index child is the first born).

Birth Order: Birth order was taken into account by means of a dichotomous variable. Birth Order=1 if the index child was the first or second born; and 0 otherwise.

Nutritional Status: We characterize nutritional status by means of two measures of growth retardation, stunting and wasting. The assessment of growth retardation requires

the comparison with a reference group. For this purpose we used the Child Growth Standards statistical package provided by the World Health Organization (WHO). This package uses as a standard for comparison across countries the National Center for Health Statistics (NCHS) growth reference population. The WHO Global Database on Child Growth and Malnutrition[8] utilizes a Z-score system to evaluate the anthropometric measure as a number of standard deviations (SD) below or above the reference mean. We use the cut-off point of <-2 SD to classify low height-for-age (stunting) and low weight-for-height (wasting) as moderate undernutrition following the WHO definitions. We evaluated stunting and wasting every two months at all ages between 2 and 24 months with 2 months interval. At each age a dichotomous variable Stunting at Month X was set to 1 if height for that particular age was below 2SD of the reference mean, and 0 otherwise. A similar procedure was used to define the set of variables Wasting at Month X.

Cognition: As mentioned earlier, the PNIT for measuring IQ was administered twice, in 1994 and in 1997. We defined two dichotomous variables, one for each measurement, indicating low IQ. Low IQ 94=1 if the score obtained in the PNIT is in the first quartile of PNIT distribution for 1994; and 0 otherwise. Low IQ 97=1 if the score obtained in the PNIT is in the first quartile of PNIT distribution for 1997; and 0 otherwise.

Child's Educational Attainment: A dichotomous variable High School was defined=1 if the index child completed HS; 0 otherwise.

[8] World Health Organization, Department of Nutrition for Health and Development (2010) WHO Global Database on Child Growth and Malnutrition. http://www.who.int/nutgrowthdb/en/

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