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Parental Education and Child Human Capital: Evidence from Indonesia

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Key words: parental education, human capital, child health, child skills, child education, development economics, Indonesia, IFLS, school construction

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Contents

List of Tables

List of Figures

1 Introduction

It is widely accepted that human capital, economic growth, and economic well-being are closely related. At the macroeconomic level, human capital fosters growth, for instance, by reducing fertility, encouraging technological innovations, and improving institutions (see e.g. Barro 1991, Mankiw, Romer & Weil 1992, Chakraborty 2004, Hazan & Zoabi 2006, Glaeser, La Porta, Lopez-de Silanes & Shleifer 2004, Galor 2011). Moreover, at the individual level, human capital, primarily shaped in childhood and youth, is found to increase earnings on the labor market, affect health in later life, increase the propensity to adopt new agricultural technologies in developing countries, among others (see e.g. Duflo 2001, Case & Paxson 2008, Almond 2006, Foster & Rosenzweig 2010, Glewwe & Kremer 2006).

Therefore, it is vital to understand the determinants of an individual's formation of human capital during childhood and adolescence. An important factor is believed to be parental education, as several studies have found that better educated parents tend to have children with better human capital outcomes measured in various ways such as school attainment and health (for broad reviews see e.g. Björklund & Salvanes 2011, Black & Devereux 2011, Glewwe & Kremer 2006, Strauss & Thomas 1995). Many of these studies find that maternal schooling is stronger correlated with child human capital outcomes than paternal education. Based on this, policymakers have favored educational expenditures targeting girls (Breierova & Duflo 2004).

It is, nevertheless, not clear whether these correlations reflect any causal impact of parental education or if they rather represent unobserved characteristics between parents and their children such as genetics. Furthermore, the difference between the estimated effects of maternal and paternal education might be upward biased. This might be the case if family background more strongly determines female than male education as women tend to be less educated than men. Additionally, another source of bias might stem from the marriage market if the education of the woman is correlated with unobserved traits of her husband. For instance, better educated women might be able to attract more caring and better educated men. As a result, in order to get a better picture of the paths for economic development and poverty eradication, the impact of parents' education on the human capital formation of their children is a vital question in development economics as well as for policymakers.

On this base, this study aims to answer the essential question: *Does parental schooling causally affect child human capital?* where human capital is understood

as a multidimensional vector in which health and skills are central factors.

In order to investigate whether such a causal link exists, this paper exploits a large primary school construction program taking place in Indonesia during the 1970s to instrument parental length of schooling. In 1973, the Indonesian government released its second five-year development plan emphasizing the importance of primary education. The Sekolah Dasar INPRES program, an immense school construction project, was part of this plan. Consequently, between 1973-74 and 1978-79, nearly 62,000 primary schools were constructed corresponding to more than two schools per 1,000 school age (5-14 years) children in 1971. This political focus was in great contrast to the preceding development plan that had frozen capital expenditures. As a result, parental program exposure is determined by the number of schools built in the district of birth the year prior to school start –two dimensions (district and year of birth) plausibly exogenous to the individual– and is used as instrument for parental education. This approach is similar to the one performed by Duflo (2001), in which educational effects on wages are studied. However, in addition to the variation between regions (which Duflo uses), I also use the variation in intensity across time.

Consequently, my study differs in important ways from the existing literature by providing new evidence of causal effects on the child human capital formation in a developing country. Our previous knowledge of such impacts is very limited within the field of development economics as it has proved difficult to find settings where the source of parental education is presumably exogenous. Compared to previous, similar studies on developed countries, this study focuses on notably low levels of education as the group of compliers in my sample has six or fewer years of schooling.

Surprisingly, the findings show no causal effects of maternal or paternal education on a broad range of child human capital outcomes, covering health (height, weight, BMI, micronutrient status, being iron deficient) and skills (years of schooling, cognitive achievement, school exam score). However, there are some indications –although not very robust– of a positive impact of parental schooling on children's cognitive skills. Therefore, in line with some existing literature on the topic in developed countries (e.g. Black, Devreux & Salvanes 2005, Lindeboom, Llena-Nozal & van Der Klaauw 2009, McCrary & Royer 2011), the results suggest that at the lower end of the educational distribution, the strong associations between parental education and child human capital are predominantly due to family background and inherited ability and not the education per se, i.e. not educational spillovers across generations. On the other hand, the results differ from for example Lund-

borg, Nilsson & Rooth (2012) and Carneiro, Meghir & Parey (2013) as both studies find evidence for causal effects of maternal education on several child human capital indicators.

Furthermore, there are indications of a negative impact of paternal education on family size. This gives some support for the quantity-quality hypothesis as a potential pathway through which paternal schooling might affect sons' cognitive skills. Moreover, there is strong evidence that positive assortative mating is taking place for both mothers and fathers. However, the IV estimates do not differ significantly from the OLS estimates, which suggests that assortative mating is not due to unobserved heterogeneity but primarily spillover effects. Thus, if this analysis should point to one single mechanism of the indicated causal impact of parental education on child skills, assortative mating is a clear candidate.

The rest of this paper is organized as follows. Section 2 presents the conceptional framework and the channels through which parental education might affect child health and skills, followed by a review, first of the development literature and second of evidence on natural experiments mainly in developed countries in section 3. Section 4 outlines the method by providing details on the school construction program and some evidence of the effect on parental education. Thereafter, section 5 describes the data and section 6 presents the empirical strategy. Finally, section 7 is devoted to the results on child human capital and potential mediators for parental education, while section 8 discusses and concludes.

2 Conceptual Framework

In this section, the formal framework of human capital production functions is outlined with emphasis on the role of parental education. Thereafter, the two key channels, stressed in the literature for the impact of parental education on child human capital, are presented.

2.1 Human Capital Production Functions

Theoretically, human capital is defined as the stock of human factors in a person that increases productivity (e.g. Becker 1964); examples of these are skills, health, social ability, and creativity. In this study, the focus is directed to the two former, i.e. skills and health. First, the production functions of each of these are presented, after which they are generalized. For this, focus is directed to the impact of parental

education on child human capital and the mechanisms through which it might work.

The health status of an individual is important across the entire life course as it affects his (her) economic, mental and physical well-being in both the present and future (see e.g. Luo, Mu & Zhang 2006, Almond 2006). However, physical and social exposure taking place in utero and during childhood is of particular importance for the individual's health and economic well-being the rest of his (her) life as deficiencies of specific health inputs (e.g. nutrients and nutrition) during certain critical periods of growth and development of the human body program immediate as well as long term health outcomes which again have consequences for labor productivity among others (see e.g. Barker 1995, Doblhammer & Vaupel 2001, Barker, Eriksson, Forsen & Osmond 2005, Almond & Currie 2011, Almond, Mazumder & Van Ewijk 2011, Almond 2006, Doyle, Harmon, Heckman & Tremblay 2009). Therefore, in order to narrow the time span in question, this study emphasizes health in childhood. For an individual, assume[1] that the static[2] health production function is:

$$H = h\left\{N(E_p); X, \mu\right\}, \tag{1}$$

where H represents a wide range of health outcomes (such as height, body mass, and morbidity) and is a function of –for the individual– endogenous as well as exogenous factors. The endogenous part of these is composed by a vector of health inputs and behaviors, N, which for instance might include diet, exercise, and the use of preventive or curative health care. This part is the focal point of this analysis as these inputs and behaviors are thought of as being under the control of the parents during childhood. The exact aspect of interest is how parental education affects child health, reason for which N here depends on parental education, E_p. At the same time, the underlying health production function (the exogenous part, X) is likely to vary over the life time (age), gender, and other exogenous factors affecting health such as genetic endowment and local environmental factors. Finally, μ represents unobserved characteristics which can be divided into two; first, factors unobserved by the researcher but known to the individual (e.g. innate healthiness) and second, measurement errors assumed to be classical.

[1]This health framework draws on Strauss & Thomas (2007), while the one of skills is inspired by Glewwe & Kremer (2006)

[2]See Strauss & Thomas (2007) for dynamic models of health investments and outcomes. The main difference from the static model is that the dynamic model discretely depends on time and therefore, health behaviors, inputs, and outcomes at one point in time affect health later in life. Thus, the important difference is that the variables included in the production function are assigned a time subscript.

In terms of skills, this area of human capital is broad and difficult to define exactly. However, it can for example be measured by a cognitive test score, quality of education etc. and a general production function of learning can for an individual be assumed to be:

$$L = l\left\{M(E_p); W, \eta\right\}. \tag{2}$$

where L represents skills learned and M constitutes inputs and behaviors important for the formation of learning such as years of schooling, school and teacher characteristics, cognitive stimulation, school attendance, purchases of school supplies, and health. All these inputs and behaviors are thought, at least partly, to be under the control of the parents and to depend on their education. Therefore, as N in the health production function, M is also a function of parental education, E_p. Moreover, the channels through which health might affect learning are many; for instance, nutrition and nutrients in early life are important for the cognitive development (Bryan, Osendarp, Hughes, Calvaresi, Baghurst & Klinken 2004) and being ill often will necessarily lead to greater school absenteeism. Therefore, the exogenous part of the learning production function, W, is composed by similar factors as X, and η is the error term.

Thus, as health and skills are thought to be two important aspects of *child quality*, Q, we here generalize the two production functions (1) and (2) to:

$$Q = q\left\{K(E_p); W, \zeta\right\}, \tag{3}$$

where $K(E_p)$ is parental inputs and behaviors associated with child quality being dependent on parental education and where ζ is the error term. It is difficult to assess the direct effect of parental education, E_p, on child quality, Q. Assume that parental demand for own length of education follows (2):

$$E_p = e_p\left\{M_p(E_{pp}); W_p, \eta_p\right\}, \tag{4}$$

where η_p represents unobservable characteristics such as parental "aptitude" and innate "willingness". Then, if

$$Cov(\zeta, \eta_p) \neq 0 \tag{5}$$

–for example if the unobserved parental characteristics that affect the parent's education also affect his (her) child's education– equations (3) and (4) would be simultaneously determined. An obvious example of this is genetics as the parent and his

(her) child share genes which is only possible to control for when having data on monozygotic twins. An alternative example could be where the parent possesses a special "passion for knowledge" which in turn influences his (her) child's desire of seeking knowledge. In such case, the effect of parental education on child quality would be impossible to asses directly without confusing it with other potential determinants. Consequently, the theoretical considerations on the formation of child human capital is complex and causes several empirical challenges discussed further in section 4.

2.2 Channels for Parental Education

Overall, income and learning are the two ways through which parental education might affect child quality (Michael 1973). First considering the former channel, it is well-documented that education increases labor earnings and hence income (see e.g. Duflo 2001, Angrist & Keueger 1991). Moreover, conventional theory on parents' fertility decision suggests a trade-off between quantity and quality of children (see e.g. Becker 1965, Becker & Lewis 1973, Galor 2011). In this framework, it is assumed that parental utility depends on quantity and quality of children as well as other consumption goods. Thus, parents choose the number of children and their quality under a time constraint, allocating time between child-rearing and labor market participation. As the relative price of child quality inputs (compared to the time needed for child-raising) differ across income levels while the real price is constant, parents will reach different optimal levels. Therefore, it is predicted that parents with more education will spend more on child quality inputs and choose a lower fertility level than less educated parents in order to optimize utility.

A further enhancement of the income channel is positive assortative mating as men and women with similar education tend to pair (see e.g. Kalmijn 1998, Behrman & Rosenzweig 2002). This implies that not only the education of one parent would affect child quality through own income but that the effect might be multiplied due to the parent's choice of mate and implicitly income. In other words, income might link parental education and child quality by changing parental health inputs or behaviors represented by $K(E_p)$ in relation to the production function above.

The second channel –learning– is in the literature thought to be an important factor for parental health knowledge (McCrary & Royer 2011). This can both be through the curriculum and the information acquisition skills taught in school. Glewwe (1999) argues in favor of the latter in a development setting, i.e. that the

most essential skill obtained in school is the ability to search for new knowledge of, for instance, how to treat an illness or what to eat during pregnancy. However, this might, to a great extend, depend on the level of education and the group of persons of interest. Formally, Grossman (1972) presents a model on demand for health emphasizing that education makes the health production more efficient. Thus, in an allocative way, with more education, the parent might be able to better combine the inputs into the child health (quality) production function and consequently shift it. Relating this to equation 3, education might shift the whole production function, q. Another argument of a productivity shifter is presented in Becker & Mulligan (1997), where the authors argue that people have endogenous time preferences and that education changes these, reason for which it shifts the production technology.

Consequently, following McCrary & Royer's (2011) distinction between the two major channels through which parental education might affect child human capital, learning represents a direct effect –also referred to as the Grossman effect– as it directly shifts the production technology. This shift is thought of as being in the direction of a better outcome. On the other hand, the income channel constitutes an indirect effect as more education indirectly augments the parent's financial resources through labor productivity and hence earnings. Additionally, it makes him (her) more attractive on the marriage market, increasing the likelihood of pairing with a partner with a similar level of education. Thus, the indirect impact of parental education is thought to increase or improve the inputs into the production function.

3 Literature Background

3.1 Parental Education and Child Human Capital

Many studies on developing countries have found strong positive associations between parental education and the formation of child human capital (for a review of the early literature see e.g. Strauss & Thomas 1995)[3]. In the attempt to estimate the impact of parental education, two approaches have been applied. One is to control for as many variables correlated with parental education as possible, including income and community or family fixed effects, in order to avoid the omitted variables problem. The other is to include explicit measures of school quality –such as ability test scores– as well as other indicators of parental human capital accu-

[3]For more recent reviews of mainly developed countries, see e.g. Björklund & Salvanes (2011) and Black & Devereux (2011).

mulation in childhood in the search for possible mediators through which parental education might affect child outcomes. However, as argued in the previous section, it is reasonable to presume that parental education is not exogenous to human capital investments in the child. For this reason, some studies –mainly the most recent ones within development economics– have used *Instrumental Variables* (IV) approaches. Nevertheless, a general concern about existing IV estimates is that they may not fulfill the exogeneity criterion (not being *valid*) as they are composed by variables likely to be correlated with parental innate ability such as grandfather's occupation and schooling.

As an example of a fixed effects approach, Behrman & Wolfe (1987) argue that maternal education simply reflects the mother's unobserved characteristics such as inherited values or beliefs from her family and that these might be correlated with the health status of her children. Thus, with Nicaraguan data, they show that once controlling for maternal and community endowments, no significant association between maternal education and child health is found in contrast to the case when leaving these controls out. On the other hand, Strauss (1990) uses a similar approach with data from Cote d'Ivoire and finds stronger positive correlations once controlling for household fixed effects.

In terms of possible pathways, several studies have found that when controlling for these, the association between parental education and child health disappears or at least diminishes in strength. For instance, Thomas, Strauss & Henriques (1991) find with Brazilian data that when controlling for access to media (reading newspaper, listening to radio, watching TV), maternal education is not significantly associated with child health while the media controls point to a potential mechanism through which education might work. Furthermore, more recent studies find evidence for positive correlations between parental nutrition or health knowledge and child health (e.g. Glewwe 1999, Block 2007, Aslam & Kingdon 2012).

For instance, Glewwe (1999) separates expected learning outcomes of schooling into three (literacy, numeracy, health knowledge) and finds from Moroccan data that the only type of maternal skills associated with child health is health knowledge. In the search for causal estimates, the author uses a set of instruments for maternal education including the educational level of both her parents as well as the number of married sisters. However, the problem with these instruments is, as explained in subsections 2.1 and 4.1, that if for instance innate ability is inherited across generations, they would not fulfill the exogeneity condition.

With data from Pakistan, Aslam & Kingdon (2012) try to get around the en-

dogeneity problem by instrumenting pathways through which parental education might affect child health[4]. Thus, they instrument both parents' health knowledge as well as maternal labor force participation, media exposure, and empowerment within the household. As instruments, they use their respective length of education and the score from a cognitive test as well as the school attainment of the mother's siblings and grandfather. However, these instruments are not necessarily exogenous for the same reasons as in the study done by Glewwe (1999).

Nevertheless, under the assumptions of this identification strategy, the authors find that only paternal health knowledge is an important mediator for health inputs into the child's production function measured by immunization decisions. At the same time, the results suggest that the mother's empowerment and health knowledge are the mechanisms through which her education affects the child's short term health given by weight and long term health measured by height. Hence, despite of worries in terms of a causal interpretation, they interestingly find that the father's role for child health is associated with health decisions (immunization) while the mothers health knowledge is correlated with everyday inputs reflected by height and weight.

While most studies use the anthropometric measures height, weight, or *Body Mass Index* (BMI) as health indicators (of nutritional intake in particular), Block (2007) uses a biomaker represented by hemoglobin concentration as an indicator for the child's micronutrient status. The results show that maternal education is correlated with child micronutrient status and that its effects partially go through maternal nutrition knowledge and household expenditures. Moreover, nutrient knowledge and maternal education substitute each other. However, a potential threat to the analysis is that Block assumes that maternal education is exogenous, while he instruments nutrition knowledge and expenditures with maternal schooling and mean distance to health center among other variables. Therefore, it does not appear convincing that he in fact estimates the causal effect as most newer studies agree on the necessity to instrument education.

3.2 Evidence from Natural Experiments

So far, in general, a well-established positive correlation between parental education and child human capital (inputs as well as outcomes) has been documented

[4]However, their empirical approach seems somewhat questionable as they start by OLS regressions adding as many explanatory variables as possible and then instrument the variables that appeared significant in the OLS version.

in the literature. However, a causal interpretation of this relationship is not as clear. Nevertheless, some studies have been able to find settings similar to natural experiments in terms of the length of parental education. The only such paper performed in a development context on the topic of interest is Breierova & Duflo (2004). This study examines the impact of parental education on fertility and child mortality using as instrument the large scale school construction program taking place in Indonesia during the 1970s –which is also the approach used in this paper and is discussed further in section 4.

Breierova & Duflo (2004) find that neither female nor male education reduces fertility. Moreover, they do not find that the average years of education of the spouses or the difference in years of education between them reduces fertility. Remarkably, the two latter results are in strong contradiction to the very significant OLS estimates. Though, they do find that the probability of giving birth before turning 15 years is negatively affected by average years of education and positively by educational difference. Furthermore, they find that child mortality is reduced by an increase in average education of the parents, while the difference in education is not an important determinant. Also, in contrast to the OLS specifications, they do not find a stronger causal impact of maternal than paternal education on child mortality. Thus, in conclusion, Breierova & Duflo (2004) find support for earlier findings that parental education has a strong causal effect on the reduction of child mortality while not on fertility, where the former does not differ between men and women.

Turning the attention to a developed country context, Black et al. (2005) use a Norwegian school reform increasing the required school attendance by two years (from seven to nine years of schooling) implemented at different times across the country during the 1960s as an instrument for parental education. Despite high correlations between parental and child education, they do not find strong evidence of a causal impact of parental education on the education of their children. In fact, only the effect of maternal education on son's education is significant when employing the instrument. Therefore, their findings indicate that the close associations between parental and child education are predominantly due to selection (family characteristics and inherited ability) and not causation (educational spillovers).

Similarly, Lindeboom et al. (2009) do not find clear evidence of a causal impact of parental education on child health using a school reform in the UK in 1947 raising the minimum school leaving age. Moreover, McCrary & Royer (2011) use a discontinuity design arising from school entrance policies and date of birth for which

they thoroughly document its validity as an instrument for length of schooling for mothers below 24 years. Likewise they do not, in general, find significant effects of maternal schooling on fertility, infant mortality, or other infant health measures from American data. Nevertheless, they do find that mothers with less education on average have younger and less educated partners.

This is, however, in contrast to the findings in Currie & Moretti (2003) that show evidence of improvements in infant health from maternal college education instrumented by the availability of colleges in the mother's county at the age of 17 years in the U.S. Moreover, the study finds that higher maternal education increases the use of prenatal care and reduces smoking, suggesting some important channels through which maternal education might work. These pathways are contrary to the findings in Lindeboom et al. (2009) and McCrary & Royer (2011) as these latter studies do not find any impact of parental schooling on child care or prenatal behavior. Furthermore, Carneiro et al. (2013) use a similar IV to Currie & Moretti (2003) and find causal effects of maternal education on child test cores and measures of behavioral problems.

As a consequence of these contradicting results, Lindeboom et al. (2009) suggest that it might be of importance which end of the educational distribution the policy change affects as the type of skills attained due to an extra year of education is different at the secondary compared to the tertiary level. An additional explanation emphasized in McCrary & Royer (2011) is that the subpopulation affected by the specific instrument at work varies and therefore, the findings in one study cannot necessarily be transferred to other settings. In this case, the results in Lindeboom et al. (2009) and McCrary & Royer (2011) would be more relevant for the present study than the ones in Currie & Moretti (2003) and Carneiro et al. (2013) as the school attainment is generally low in Indonesia compared to an already developed country. However, Lundborg et al. (2012) exploit a very similar school reform setting as in Black et al. (2005) as a natural experiment and do in fact find positive effects of parental education on human capital outcomes of 18 years old Swedish men attending the military enlistment. Their results suggest a positive causal impact of maternal education on cognitive as well as non-cognitive skills, height, and overall health, while paternal education only indicates an improvement in physical capacity. Despite of these results, the large difference in significance levels between the OLS and IV regressions is generally repeated.

To sum up, it is clear from the literature that in most settings –within developing countries as well as across developing and developed countries– strong positive

correlations between parental (in particular maternal) education and child human capital are found. However, when it comes to causal relations, even when employing policy changes as natural experiments, conclusive evidence for whether parental education has a causal impact on child human capital does not exist. The different results in the literature on causal relations are not astonishing as different outcomes are examined as well as different identification strategies are employed with very different groups of compliers. Therefore, further analysis of the effects of parental education on child human capital is not trivial as one could initially believe and especially not in a development context where studies of pure causal effects are scarce.

4 Method

4.1 Empirical Implications of Theory

As seen in section 2, it is clear that the variables of interest are likely to be interdependent, making it difficult to isolate the direct impact of parental education on child quality. If equation (5) holds, the empirical estimation of (3) would suffer from an *Omitted Variables Bias* (OVB). This would be driven by the impossibility for the researcher to control for unobservable characteristics of the parents that are likely to determine both their own education and their children's human capital formation. A straightforward reason might simply be due to genetics, although other reasons are of course also possible. Therefore, the estimate of parental education would not yield any causal impact precisely because parents' length of schooling is not randomly allocated but rather endogenously. This makes it an argument for the necessity of *instrumenting* parental education in order to estimate the *causal* effect.

Assuming linearity in the parameters and rewriting equation (3) for child i with parent p yields:

$$Q_{ip} = \alpha_0 + \alpha_1 E_p + W_i^{'} \alpha_2 + \zeta_{ip}. \tag{6}$$

Thus, if the *Zero Mean Condition* (ZMC) is not fulfilled, i.e. $E[\zeta_{ip}|E_p] \neq 0$, because the true relationship is

$$\zeta_{ip} = \beta B_p + \vartheta_{ip}, \tag{7}$$

where B_p is parental innate ability, will, motivation etc. and $E[\vartheta_{ip}|B_p] = 0$, then if

$$Cov(E_p, B_p) \neq 0, \tag{8}$$

the estimate of parental education, $\widehat{\alpha}_1$, would be biased. In such case, the estimate of $\widehat{\alpha}_1$ would be:

$$\widehat{\alpha}_1 = \alpha_1 + \beta \frac{Cov(E_p, B_p)}{Var(E_p)}. \tag{9}$$

As this parental innate ability, B_p, is not observable, another strategy than simple OLS is needed, for instance IV. For an IV approach, we need an instrument[5], Z_p, that is correlated with parental education, E_p, (being *relevant*) and uncorrelated with the problematic part of parental education (being *valid*). With a relevant and valid instrument, parental education can be instrumented and estimated by using *Two Stage Least Squares* (2SLS), in which case the estimated relation from the second stage is:

$$Q_{ip} = \varphi_0 + \varphi_1 \widehat{E}_p + W_i^{'} \varphi_2 + \psi_{ip}, \tag{10}$$

where $\widehat{E}_p$ is obtained from the first stage

$$E_p = \pi_0 + \pi_1 Z_p + W_i^{'} \pi_2 + \psi_{ip}^{Z}, \tag{11}$$

and where ψ_{ip}^{Z} is the error term. Consequently, as long as the requirements for the instrument are met –i.e. $\pi_1 \neq 0$, $E[\psi_{ip}|\widehat{E}_p]=0$ and $E[\psi_{ip}^{Z}|Z_p]=0$–, $\widehat{\varphi}_1$ is the estimated causal impact of parental education on child quality.

Thus, due to the self-selection into education by parents and hence the omitted variables bias, an IV approach is needed in order to obtain a causal estimate of parental education on child quality. Therefore, the rest of this section is dedicated to a presentation and discussion of the instrument used for this study.

4.2 The School Construction Program

The Indonesian Sekolah Dasar INPRES program is one of the largest primary school construction programs to date. Nearly 62,000 new primary schools were constructed between 1973-74 and 1978-79 at a cost of 1.5 percent of the Indonesian GDP in 1973 (Duflo 2001). The magnitude of this number equals one school constructed per every 430 children in the age between 5 to 14 years in 1971. This corresponds to a doubling of the school stock prior to the program launch, although the pre-program

[5]For a more detailed description of IVs see e.g. Angrist & Pischke (2008).

schools tended to be of larger capacity than the INPRES schools. The program was one of the first Indonesian "presidential instructions", i.e. centrally administered development programs, established in 1973 (for more details see Duflo 2001, Duflo 2000), and whose goal was to increase equity across the Indonesian regions. It was financed through increased oil revenues due to the boost in oil prices, which allowed real expenditures on regional development to more than double in the period between 1973 and 1980.

The program was in clear contrast to the previous (first) five-year governmental development plan which had focused on basic infrastructure and sectoral development and had frozen capital expenditure as well as teacher recruiting. Therefore, due to the change in priorities as well as the large increase in oil revenues and consequently in the development budget, the Sekolah Dasar INPRES program became exceedingly important. Thus, in the second five-year plan, 15 percent of the national budget was allocated to regional development of which the school construction program represented 12 percent in 1973 and 28 percent in 1979 in comparison to health expenditures representing 3.4 percent in the former year and 5.5 percent in the latter (Duflo 2000).

Each INPRES school was designed for three teachers and 120 pupils and was equipped with furniture and textbooks. As soon as a school was constructed, the government hired the teachers and paid their wages. Alongside the construction of schools, an effort was put into the training of more teachers leading to a growth in the stock of teachers by 43 percent between 1971 and 1978 such that the student-teacher ratio remained virtually constant. Moreover, the share of teachers meeting the minimum qualification requirements did not worsen considerably during the period.

The program aimed to construct new schools as a way to supplement existing schools rather than replacing them. The objective was explicitly to provide schools for children that had not previously been able to attend school, and low-income urban as well as remote areas should be prioritized (Duflo 2000). All this was in order for each region to achieve the target primary school enrollment rate of 85 percent in 1978. Thus, the policy rule of school construction in each district[6] was that the number of schools to be constructed should be proportional to the number of primary school age children *not* enrolled in school in 1972. However, Duflo (2001) shows that the actual rule put into practice was less re-distributive than intended

[6]Indonesia is divided into provinces that are again divided into districts corresponding to municipalities.

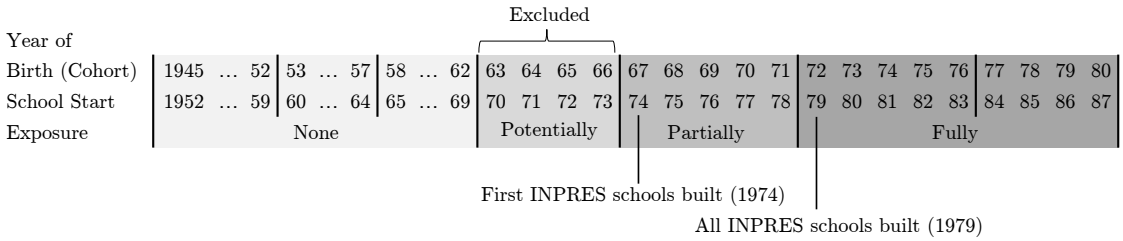
although generally complying the presented policy objective.

Consequently, the treatment of the school program should be interpreted as an exposure to an increased stock of schools. In particular, Duflo (2001) finds support for that the reduction of the traveling time was the most important effect of the construction of these new schools. In this way the alternative cost of going to school decreased due to the shorter distance to school and might have been the trigger for the increase in educational attainment. Furthermore, she finds that the impact of the program does not differ significantly between high and low poverty regions or between regions with high and low pre-program educational level.

4.3 Identification Strategy

The individual's exposure to the INPRES program was determined by his (her) year and district of birth; being two aspects exogenous to the individual. In Indonesia, children normally attend primary school –which spans over six grades– between the ages of 7 to 12 years. Therefore, a person aged 12 years or more in 1974, when the first INPRES schools were completed, was too old to benefit from the program. Thus, individuals born in or before 1962 are considered *not* exposed to the program. However, due to the possibility of grade repetition and delayed school entry, some people might of course have been able to benefit from the program during their last year in school. Nevertheless, this case is neglected in the proceeding analysis since according to Duflo (2001), the vast majority of people born in 1962 or earlier (97 percent[7]) had already left primary school in 1974. Also, note that in case they are mistakenly considered not exposed when in fact being so, the bias will be downward in the estimation of the impact of the program.

Figure 1: Illustration of Program Exposure



Note: Individuals are grouped into birth cohorts according to the vertical lines for year of birth. The year of school start is assumed to be at age 7 years.

[7]This estimate is calculated on the basis of the first IFLS wave, i.e. the same data source as the one used in the current study as described in section 5.

Moreover, persons born in 1967 were 7 years in 1974 and thus form the first group exposed to the program from their first grade. This cohort started school in the school year 1974-75 and was therefore fully exposed to the INPRES schools built in 1973-74. Further, the first cohort being fully exposed to the entire stock of INPRES schools was born in 1972 since schools were constructed over the entire period between 1973-74 and 1978-79. As the persons born between 1963 and 1966 were potentially partially exposed to the program, making it difficult to assess their exposure, they are excluded from the analysis[8]. Figure 1 illustrates the different intensities of program exposure by birth year.

The program consisted of three waves, each of a duration of two years, generating further variation in the individual's exposure additional to the intensity across districts. During the first two years (1973-74 and 1974-75), 6,000 schools were constructed annually, increasing to nearly 15,000 schools annually in the second period, and 10,000 annually in the third. The measure of program exposure for this study is constructed from this variation in school construction intensity both across time and space. In this way, program exposure is the cumulative number of INPRES schools constructed per 1,000 children aged 5 to 14 years in 1971 in a given district in the year prior to the one an individual started primary school[9]. As a result, persons born in 1962 or before have an observed exposure value of zero. Thus, under the assumption of school start in the year of an individual's 7th birthday, program exposure is found from the number of INPRES schools completed before his (her) school start.

Considering potential endogenous migration, an issue might arise if some parents care particularly much about their children and consequently move their family to another district in order to be able to benefit from a higher program intensity. In this case of systematic migration, an upward bias would be introduced for two reasons. First, the measured exposure of these children would be lower than their actual and therefore, the estimated impact of the program would be overstated. Second, children in such families would probably attain more education not as a

[8]This is in line with other studies (e.g. Pettersson 2012, Hertz & Jayasundera 2007) using the same instrument but with much fewer observations than Duflo (2001) and Breierova & Duflo (2004).

[9]This measure is similar to the one in Pettersson (2012), although her instrumental approach differs from the one used in this study. (I do not follow Pettersson's (2012) IV approach as she also controls for the father's education in the first stage which strongly contradicts what I argue in subsections 2.1 and 4.1.) This way of measuring program exposure differs from the one used in Duflo (2001) and is partly used due to the smaller sample size, but also as it is considered a more precise measure of actual exposure to the program.

causal consequence of the program but because of their unusual parents. As a result, one approach might be only to consider individuals who lived in their district of birth at age 12 years –which is done in the sensitivity analysis, reaching similar results. For the individuals studied in the analysis below, more than 90 percent lived in their district of birth at the time they were supposed to finish primary school.

Another concern might be that extraordinary caring parents might have moved to a high intensity region before the birth of their child such that he (she) could benefit from the INPRES program. Nonetheless, this cannot have been the case before 1973 since the program was not known by the public before then. However, as mentioned in the previous section, the districts with high school construction intensity were the ones with the lowest enrollment rates previously to the program start and were hence in this respect not particularly attractive compared to places with lower intensity. Therefore, district of birth is seen as exogenous to the program at least in terms of persons born before 1973 and most likely also for the ones born thereafter; this issue is looked into in the following section.

4.4 The Program Effect on Parental Education

For the purpose of presenting the basic approach behind the identification strategy, districts are divided into respectively high and low intensity regions.[10] On average, 2.79 INPRES schools were constructed per 1,000 children in the school age in high intensity regions in contrast to 1.66 in districts with low intensity (note that data is described in section 5 and that descriptive statistics of the instrumental variables are to be found in appendix A).

However, before looking at effects of the program, it is of relevance to consider characteristics of movers[11] and non-movers due to the potential issue of endogenous migration as discussed in the previous subsection. Panel A in table 1 compares program details between district of birth and district at age 12 years for those moving parents that report their district at age 12 years such that it could be identified (which also explains the difference between panel A and B). From this, it is seen that mothers as well as fathers did not predominantly move to high

[10]It is done by regressing the total number of schools constructed by district on the number of school age children (age 5 to 14 years in 1971) in that district. A district is defined as high intensity for positive residuals and low intensity for negative residuals. This definition is similar to the one used by Duflo (2001).

[11]A person is defined as a "mover" if he (she) reports a different district or province of residence at age 12 years from the one of birth.

Table 1: Differences between Movers and Non-Movers

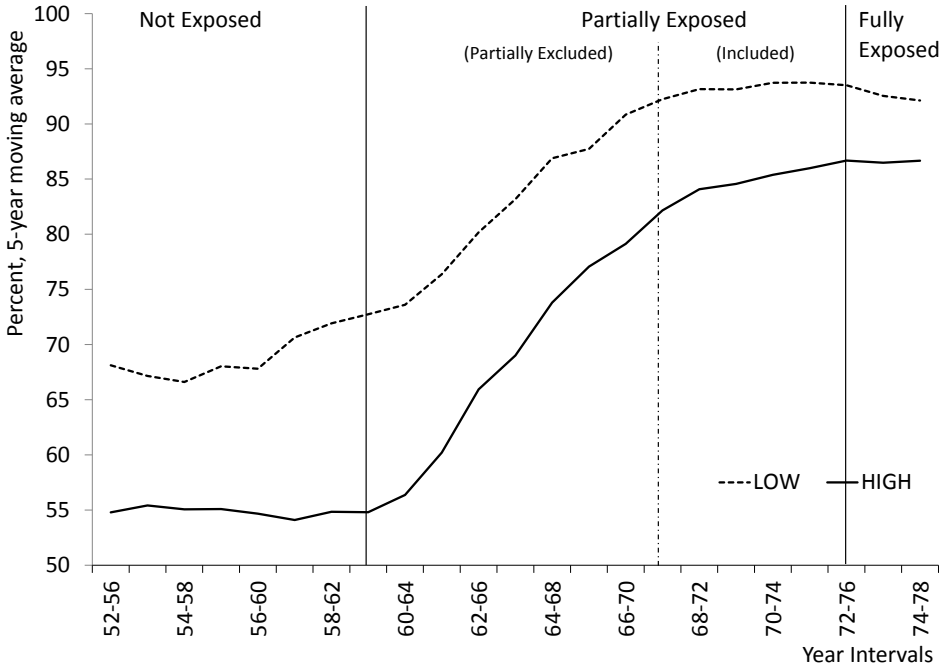
	Mothers		**Fathers**	
	Mean	S.D.	Mean	S.D.
Panel A: Movers (District of Birth vs. at Age 12)				
High - Birth (share)	0.44	0.50	0.41	0.50
High - age 12 (share)	0.45	0.50	0.50	0.50
Program Exposure - Birth	0.80	1.12	0.36	0.73
Program Exposure - age 12	0.81	1.14	0.42	0.89
Schools Constructed - Birth	2.05	1.07	1.86	0.80
Schools Constructed - age 12	2.05	1.02	2.24	1.28
Length of Schooling	8.59	4.43	8.38	4.47
Year of Birth	1965	9.78	1959	9.72
N	370		78	
Panel B: All Movers (District of Birth)				
High	0.44	0.50	0.45	0.50
Program Exposure	0.95	1.18	0.66	0.97
Schools Constructed	2.04	1.04	1.95	0.89
Length of Schooling	8.57	4.30	9.46	4.27
Year of Birth	1966	9.66	1963	10.02
N	534		520	
Panel C: All Non-Movers (District of Birth)				
High	0.50	0.50	0.51	0.50
Program Exposure	1.06	1.20	0.88	1.14
Schools Constructed	2.09	0.99	2.10	0.97
Length of Schooling	7.42	4.05	8.10	4.04
Year of Birth	1967	9.33	1965	9.71
N	4,980		4,912	

intensity districts, implying that their computed program exposure does not differ remarkably between their district of birth and the one at age 12 years. Similarly, the INPRES schools constructed in these districts did not differ for mothers. However, the difference is slightly larger for fathers, but they are also more likely to be born before the program start and is a considerably smaller group. Therefore, it is not reasonable to believe that they moved due to the school program.

From comparing panel B and C, it is apparent that, in general, movers are more likely than non-movers to be born too early to have benefited from the program and in low intensity regions. Hence, there is no clear indication in table 1 that movers or their parents should have self-selected them(selves) into high intensity regions either by moving there before their birth or before turning 12 years. Therefore, the proceeding analysis will examine non-moving and moving parents together; though, the sensitivity analysis excludes non-movers, cf. subsection 7.3.5.

A first simple indication of the impact of the INPRES program on education is

Figure 2: Percentage of Parents with at leat Primary School by Intensity



Note: Five year moving average by year of birth for all non-moving parents.

seen in figure 2. It is evident that parents not exposed to the program had a relatively constant probability of having finished primary school within both regions (high vs. low intensity). This fact supports the important identification assumption of a non-systematic change in educational attainment across the two types of regions in absence of the school construction program when considering it a natural experiment. Furthermore, the first cohorts partly exposed (born after 1963) in each group experiences a large increase in this probability, while it is seen that the difference between the regions narrows over time. All this might indicate a relevant instrument.

A further illustration of the basic idea behind the identification strategy is a simple two-by-two table (table 2). First, it is seen that the persons fully exposed to the program in high intensity districts are, on average, exposed to 1.11 more INPRES schools pr. 1,000 school age children than the ones from low intensity districts. Panel B shows that both cohorts from high intensity regions have lower educational attainment than their peers in low intensity regions which reflects the policy rule of constructing new schools in areas where the initial school attainment was low. Moreover, it is seen that in both types of regions, the length of schooling

Table 2: Simple Difference-in-Differences by Program Intensity and Exposure Status, All Parents

	High	**Low**	**Difference**
Panel A: Program Exposure for Exposed			
Exposed (1972-80)	2.67	1.56	1.11
	(1.03)	(0.58)	[0.00]
N	1,942	1,703	3,645
Panel B: Experiment - Length of Education			
Exposed (1972-80)	8.07	9.14	-1.07
	(3.38)	(3.33)	[0.00]
Non-Exposed (1945-62)	5.89	7.07	-1.18
	(4.22)	(4.20)	[0.00]
Difference	2.19	2.07	0.11
	[0.00]	[0.00]	[0.52]
N	3,836	3,809	7,645

Note: Numbers in parentheses are standard derivations and numbers in brackets are p-values obtained from separate regressions. Movers and partially exposed (born 1967-71) are excluded. Only parents with data on control variables used in the multivariate analysis are included.

enhances over time, although the increase is larger in high intensity districts. This difference in differences over time can be interpreted as the causal impact of the program. However, this is only true under the the assumption that in absence of the program, the increase in educational attainment would not have been systematically different between low and high program regions –which was visually shown in figure 2.

Consequently, under this assumption, each school constructed contributed, on average, to an increase in length of schooling of 0.18 years[12], although being insignificant. In other words, exposed parents from high intensity regions increased their length of schooling with a fifth of a year more, on average, compared to the ones from low intensity regions due the the larger increase in the school stock in high intensity regions. Nevertheless, note that this estimate is inaccurate since for instance the partially exposed group has been excluded and the policy rule has not been controlled for.

[12]This increase of 0.18 years is calculated as 0.11/0.59, where 0.11 is the DiD estimate in panel B and 0.59 is the ratio between the high and low intensity regions (1.66/2.79).

5 Data

5.1 Data

The data used for this study comes from the Indonesian Family Life Survey (IFLS), an ongoing longitudinal survey run by the RAND Corporation[13]. Currently, four waves have been conducted, in 1993-94, 1997-98, 2000, and 2007-08. The survey is not nationally representative but covers 83 percent of the population living in 13 of the country's 27 provinces. In the first wave, 7,224 households were interviewed and in the most recent more than 30,000 individuals were surveyed. Moreover, the recontact rate has been high both for original households and their members as well as their split offs; for instance, 93.6 percent of the original households were recontacted in the fourth wave (for more details on the survey design etc. see e.g. Strauss, Witoelar, Sikoki & Wattie 2009). The survey contains detailed questions on a very wide range of topics such as health, education, migration, fertility, income, consumption, etc.

For the analysis, data from all four waves is used in order to get as precise information as possible on important variables such as year and place of birth as well as length of education for the parents. As non-exposed parents in general have older children than exposed parents, the earliest available observations are used for children of the former group while the most recent have been used for children of the latter in order to compare outcomes of individuals from more similar ages[14]. Children are restricted to be born between 1980 and 2004 in order to only observe those born after the school construction program. Moreover, children with inconsistent birth year have been excluded and children without enough information for the IV on at least one parent are not used for the analysis[15]. Furthermore, parents are restricted to be born between 1945 and 1980 in order to observe those born before and after the school construction program; however, the ones born between 1963 and 1966 are excluded as explained in section 4.3.

[13]In collaboration with different partners: Lembaga Demografi of the University of Indonesia, University of California-LA, Center for Population and Policy Studies (CPPS) of the University of Gadjah Mada, and Survey Meter.

[14]However, performing the main analysis with the child outcome variables for the most recent observation for all children gives very similar results.

[15]There is one case where one of the parents has been proxied when the other parent is known. This is when all children of a household have the same mother (and vice versa for the father) and some of the children miss information on the father but all the rest share the same father. Then, the father has been proxied to be the same person as for the rest of the children. In practice, it concerns 77 mothers and 206 fathers. If just one child in the household has a different father or mother, this procedure has not been used.

5.2 Child Human Capital Outcome Variables

The two aspects of child human capital examined in this study are, as mentioned in section 2, health and skills. However, as these are very broadly defined, there is no single measure for them. Therefore, several key variables have been selected in order to get a more complete picture of each of these aspects. Some simple descriptive statistics of these are displayed in table 3.

Table 3: Descriptive Statistics on Child Human Capital

	N	Mean	SD	N	Mean	SD
	Mother Not Exposed			**Mother Exposed**		
Age (years)	4,844	8.23	4.87	6,732	8.76	4.73
Male (share)	4,844	0.51	0.50	6,732	0.51	0.50
Year of Birth	4,844	1987	5.23	6,732	1997	4.94
IFLS Survey Round	4,844	1.52	0.79	6,732	3.77	0.50
Height (cm)	4,844	117.52	25.89	6,732	121.81	23.49
Maternal Height NA (share)	4,844	0.04	0.20	6,732	0.08	0.28
Paternal Height NA (share)	4,844	0.09	0.29	6,732	0.14	0.35
Weight (kg)	4,837	23.77	12.44	6,740	26.07	12.61
BMI (kg/m^2)	4,832	16.03	2.40	6,684	16.20	2.62
Hemoglobin (Hb g/dL)	4,675	12.48	1.64	6,399	12.48	1.46
Anemia (share)	4,675	0.30	0.46	6,399	0.22	0.41
Schooling (years)	4,854	3.60	2.60	4,840	4.52	3.17
Ln Cognitive Test Score	4,475	3.80	0.53	4,603	3.99	0.43
Ln School Exam Score	2,925	3.50	0.25	784	3.58	0.37
	Father Not Exposed			**Father Exposed**		
Age	6,676	7.08	4.78	4,574	7.24	3.90
Male	6,676	0.51	0.50	4,574	0.51	0.50
Year of Birth	6,676	1989	6.07	4,574	1999	3.97
IFLS Survey Round	6,676	1.73	0.94	4,574	3.83	0.42
Height	6,676	111.51	26.05	4,574	114.97	21.43
Maternal Height NA	6,676	0.04	0.20	4,574	0.11	0.31
Paternal Height NA	6,676	0.08	0.27	4,574	0.15	0.36
Weight	6,663	21.14	11.84	4,575	22.45	10.54
BMI	6,651	15.82	2.32	4,523	15.75	2.32
Hemoglobin	6,449	12.24	1.61	4,290	12.27	1.34
Anemia	6,449	0.32	0.46	4,290	0.23	0.42
Schooling	6,423	3.37	2.46	2,722	3.59	2.82
Ln Cognitive Test Score	6,011	3.84	0.52	2,607	4.03	0.40
Ln School Exam Score	3,486	3.51	0.27	210	3.65	0.40

Note: Only children who are part of the main analysis are included. All the outcome variables are the ones used for their respective regressions, while the statistics for the remaining variables (age, male, year of birth, and IFLS survey round) correspond to the ones used for the height-regression only.

Concerning health, five outcome variables are studied: height, weight, *Body*

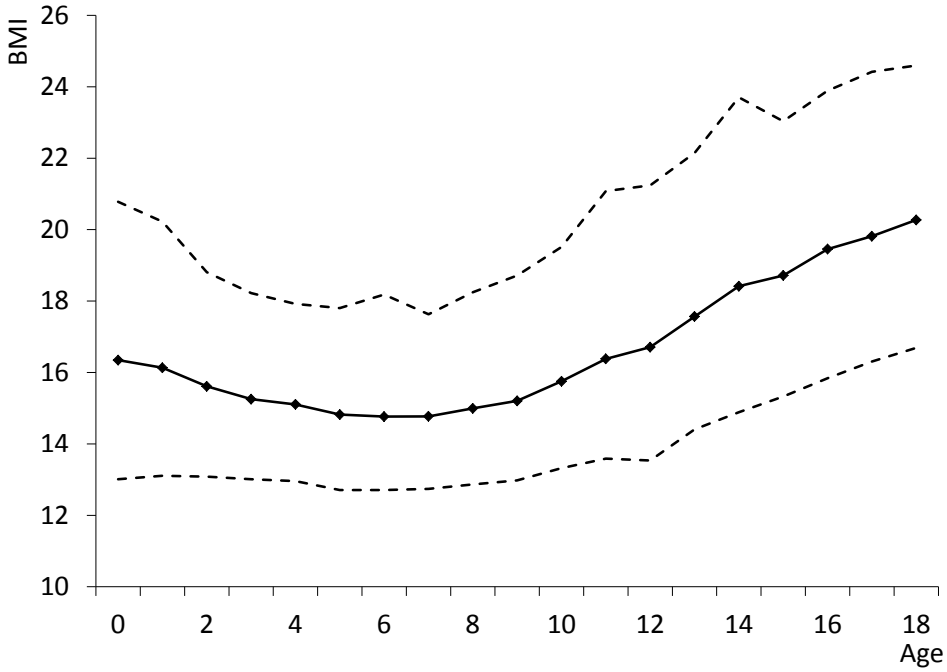
Mass Index (BMI), hemoglobin concentration in blood (henceforth *hemoglobin*), and the probability of being iron deficient, i.e. suffer from anemia. The first three are so called anthropometric measures and are measured in the field. Height is a common measure of long term overall health in the literature as growth is a cumulative process. In particular, it is affected by health inputs such as quantity (calories) and quality (nutrients) of food and by prior health status during childhood such as infectious diseases (see e.g. Strauss & Thomas 2007, Bozzoli, Deaton & Quintana-Domeque 2009). However, the child's genetic endowment is also important when considering the stature of a person, reason for which all regressions in the later analysis of anthropometric measures control for the height of both parents and in case such information is not available, it is indicated by a dummy. In this way, parental height should be interpreted as a proxy for genetics[16].

Weight is also a normal measure of health though less clean-cut as it is easier to change one's weight during a relatively short period of time. Also, while taller is always better, heavier is not necessarily better. Nonetheless, it is so for the vast majority in the current sample. Therefore, weight should be seen as a somewhat shorter term health measure. Furthermore, BMI is a summary measure of the relationship between weight and height (kg/m^2) as for instance shorter persons are normally also lighter. Healthy BMI-values are age dependent and lowest for the ages 6-8 years where the minimum value for healthy American boys is about 13.6; this is compared to the maximal healthy value of 27 for 20 years old men[17]. Extremely high values of BMI are associated with increased risk of cardiovascular diseases and diabetes in adulthood among other risk factors, while extreme values in general –both in the low and high ranges– are related to increased morbidity and mortality (Strauss & Thomas 2007). In the current sample, overweight is not a real problem, whereas underweight is more predominant. Figure 3 shows the average BMI (solid line) by age for both males and females as it only varies very little across the genders, where the dashed lines represent respectively the 5th and 95th percentiles.

[16]Note, that this control strategy is only valid when assuming that height is not an outcome of own education, cf. section 6.3, which seems reasonable as people normally begin school relatively late compared to important periods of growth. For the initial analysis, the models with hemoglobin and anemia also included parental height, but as the preciseness of the estimates did not improve, they have been left out. Moreover, using other parental health measures might cause problems as they are relatively easy to affect later in life (e.g. weight and hemoglobin) which might be correlated to parental length of education. For that reason, such controls have not been used.

[17]For more details see e.g. the homepage for Centers for Disease Control and Prevention, an American governmental organization: http://www.cdc.gov/.

Figure 3: Child BMI by Age



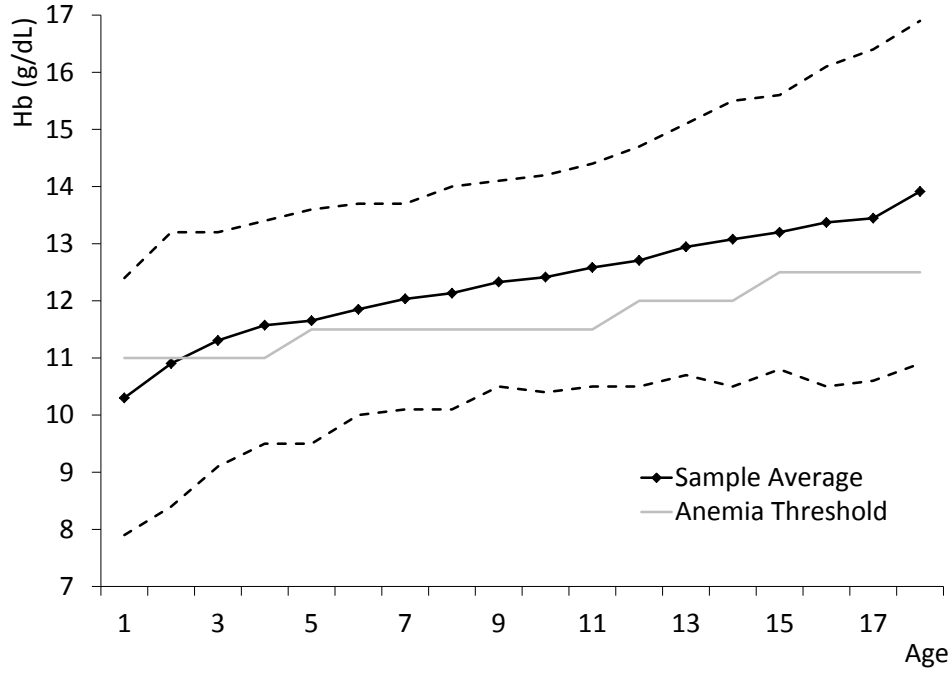
Note: Only individuals used for the main analysis are included. Age 18 represents age 18-27 years (corresponding to 2 percent of the sample for the figure). The dashed lines represent respectively the 5th and 95th percentiles.

Moreover, hemoglobin concentration in blood is one important indicator of micronutrients (iron) that the human body cannot produce itself but are crucial in small quantities; other such micronutrients are vitamin A, iodine, and various minerals among others (Block 2007). This biometric measure is the most reliable indicator of anemia and is a symptom of iron deficiency and an indicator of poor nutrition and health (De Benoist, McLean, Egli & Cogswell 2008). The health consequences of anemia can be serious as it has been documented that iron deficiency increases maternal and child morbidity and mortality. Furthermore, iron deficiency has negative effects on cognitive, mental, and physical development of children and on physical activity for adults (see e.g. De Benoist et al. 2008, Demment, Young & Sensenig 2003, Strauss & Thomas 2007). As longer term consequences, several studies have suggested that iron deficiency anemia in turn also reduces labor earnings (for further references see Strauss & Thomas 2007).

The data on hemoglobin was collected as part of the IFLS survey as small blood samples from the fingertip and analyzed in the IFLS headquarter. From the hemoglobin values and thresholds for anemia by age and gender given in De Benoist

et al. (2008), the dummy variable *anemia* is defined with the value one if the individual's hemoglobin concentration was below the threshold and zero otherwise. From table 3 and figure 4, it is seen that a relatively large proportion of the children is iron deficient. All in all, general health and anemia are negatively correlated which is in contrast to general health and hemoglobin.

Figure 4: Child Hemoglobin Concentration by Age



Note: Only individuals used for the main analysis are included. Age 18 represents age 18-28 years (corresponding to 3 percent of the sample for the figure). From age 15 years, the anemia threshold is divided by gender (12 for women and 13 for men), however, this figure does not distinguish by gender. The dashed lines represent respectively the 5th and 95th percentiles.

In terms of the skills aspect of human capital, three variables are analyzed: years of schooling, a cognitive test score, and a primary school exam score. The first measure is a typical measure of quantity of schooling as an indicator of skills since many academic abilities are taught in school (for a review of schooling in developing countries, see e.g. Glewwe & Kremer 2006). Nonetheless, it is associated with inaccuracy as it does not tell anything about the particular skills attained. For this measure, only children in the school age (7 years or older) are included.

The second measure of skills is the score from a test, performed by children aged 7 years or more and introduced in the second survey round. During this round, the test was divided into two comprehensive sections (Indonesian and mathematics)

and performed by four different age groups (7-9, 10-12, 13-15, and 16-24 years). Unfortunately, this type of test was not repeated in the subsequent rounds but instead replaced by one more focused on cognitive skills and divided into only two age groups (7-14 and 15-24 years). Its major section is comprised by non-verbal items, for each of which the test taker is asked to identify the missing element that completes a pattern[18], but it also contains a section on mathematics. In order to get enough observations, all tests have been used and constitute together the cognitive test score. Therefore, the multivariate analysis controls for type of test dummies. Moreover, in order to streamline the test scores, they have been standardized such that the maximal score within each type of test is 100 and zero corresponds to no correct answer.

Finally, the respondents are also asked whether they have taken the national exam at the end of primary, junior high, and senior high school and in such case what the total score was. These might also be good indicators of skills, although they suffer from attrition as one would expect the least able children not to have taken the exam or being able to remember or show the score. This is, on the other hand, the advantage of the cognitive test as it has been performed by nearly all children and consequently also by the ones not enrolled in school. Therefore, the results from the national exam should be interpreted with caution. Moreover, as not all children are old enough to have taken these three exams, it has only been possible to analyze the exam performed at the end of primary school. As seen from table 3 only few children with parents exposed to the school program have yet finished primary school and taken the exam, which might cause some empirical problems in terms of the instrument.

6 Empirical Strategy

6.1 Econometric Model

Based on the theoretical framework and the school construction program, the empirical strategy is based on the following two equations:

$$H_{ipdcs} = \alpha_0 + \alpha_1 E_p + X_{dc}^{'}\alpha_2 + Y_{dc}^{'}\alpha_3 + V_{is}^{'}\alpha_4 + \varrho_{ipdcs}, \tag{12}$$

[18]This is a version of a Raven's Colored Progressive Matrices.

$$E_{pdct} = \beta_0 + \beta_1 P_{dt} + X_{dc}^{'}\beta_2 + Y_{dc}^{'}\beta_3 + V_{is}^{'}\beta_4 + \varepsilon_{pdct}, \quad (13)$$

where i denotes the child and p[19] one of his (her) parents, who was born in district d in the birth cohort c[20], in year t[21], and is observed in survey round s. Moreover, H_{idcs} is the outcome of interest and E_p is parental length of education. V is a vector of child controls –such as age, gender, and survey round– and ϱ and ε are the error terms. P_{dt} is the program exposure measured as the cumulative number of INPRES schools constructed per 1,000 school children in the district of birth the year before starting in primary school. Y_{dc} is a vector of controls for the allocation rule of the program; these are district of birth dummies, birth cohort dummies, interactions between these cohort dummies and the number of children in the region of birth in 1971, and place of birth dummies[22]. Finally, X_{dc} is a vector of two groups of controls; firstly, interactions between the birth cohort dummies and the enrollment rate in the region of birth in 1971, and secondly, interactions between the birth cohort dummies and the allocation of a contemporaneous water and sanitation program.

The water and sanitation program was the second largest INPRES program at the time of the school construction program and was also centrally administered[23]. The reason behind controlling for this program is that as the programs are simultaneous, an increase in school attendance could be due to healthier children and not exclusively to the availability of more schools. Consequently, if these two INPRES programs are correlated and both affect parental schooling, not controlling for the health program would introduce a bias. This bias is most likely to be upward as both improved water and sanitation facilities as well as more schools are thought of as positive factors for education. However, a downward bias might also be a

[19]Note, that I only consider the effect of one parent's education at a time. The reason is that the parents usually share district of birth and therefore, it is not possible to perform the specification with both parents simultaneously due to multicollinearity. However, not controlling for both parents' education in the specification has some implications. First, the estimates covers implicitly the effects of assortative mating. Second, the effect of assortative mating is contained in the error term which could cause problems as the potential bias most likely is upward. Nonetheless, this is a potential threat to all studies examining the impact of one parent's education.

[20]These birth cohorts are divided into the following periods of birth: 1945-52, 1953-57, 1958-62, 1967-71, 1972-76, and 1977-80. This is also visually seen from figure 1.

[21]The birth year is used for constructing the measure of program exposure.

[22]These place of birth dummies indicate whether the person was born in a village, small town, or big city. The reason for controlling for this is that the access to schools might depend on the location in the district where the person lived (proxied by the place of birth), i.e. whether the person lived in a rural or urban area within the district. The findings in the econometric analysis are, however, not sensitive to the inclusion or exclusion of these place of birth dummies, but their inclusion improves the efficiency of the estimate of parental program exposure.

[23]For an evaluation of the program, see e.g. Sejahtera et al. (1987)

possible scenario if the health program is an indicator of initially poor health. In this case, poor health would probably negatively influence both school attainment and the initial enrollment rate which would be low and thus, these districts would also attract more INPRES schools according to the allocation rule.

To have a baseline to compare the IV results with, equation (12) is first estimated using OLS estimation. However, as argued above, such relations suffer most likely by heterogeneity between parental education and the different child outcomes and therefore, the estimates of parental education cannot be interpreted as causal effects. For that reason, 2SLS estimations are applied, where equation (13) form the first stage with the program exposure as the instrument for schooling. Hereby, the identification of α_1 relies on the variation in parental schooling which is generated by the INPRES school program. Thus, the estimation strategy is based on a difference-in-differences (DiD) approach with a continuous treatment variable in the first stage as the intensity of program exposure varies over time within the same district as well as across districts. This approach is similar in concept to previous studies on school reforms except that they use binary treatment indicators (e.g. Black et al. 2005, Lindeboom et al. 2009, Lundborg et al. 2012). However, four conditions must to be met in order to interpret α_1 as weighted averages of the causal response of those individuals whose treatment status was changed by the program exposure, i.e. the compliers. The discussion of the likelihood of these conditions is discussed in the next subsection.

6.2 Necessary Conditions

Since the school construction program only *intended* to get children to attend school (they were not forced to do so by law), we allow for treatment effect heterogeneity[24], and therefore, the *independence assumption* has to be fulfilled. It requires that program exposure is as good as random conditioned on the controls included in the first stage. Thus, the interpretation of program exposure depends on the identification assumption that there are no omitted time-varying or district-specific effects correlated with the school construction program.

However, as different time trends might be prevalent across regions not because of the program but due to other regional characteristics, a potential problem with these assumptions could exist. Since older parents had their children at earlier dates than younger parents, the differences in child outcomes by different cohorts

[24]For further clarifications of the IV approach with heterogeneous treatment effects and the necessary assumptions, see e.g. Angrist & Pischke (2008).

might simply represent evolution over time. For instance, mean reversion could have taken place since an increase in child health might have been faster in high intensity regions even in absence of the program if these regions started with a lower baseline. Nevertheless, this would be likely to affect cohorts evenly over time and not just the exposed groups of parents. Therefore, the specifications 12 and 13 control for district specific variables in interaction with parental birth cohort dummies represented by X and Y. This should capture time-varying factors correlated with pre-program characteristics potentially related to educational achievement.

This control approach closely follows the one carried out by Duflo (2001), although she has not access to data on place of birth and instead of birth cohort dummies, she uses year of birth, but her sample is also about 30 times larger than the present. Moreover, Breierova & Duflo (2004) solely uses the interactions with the enrollment rate as a robustness check of the specifications and hence, they do not use the water and sanitation program at all. The latter might be explained by the fact that the results in Duflo (2001) show that adding the controls makes the estimates of the school construction program on length of schooling slightly larger. This indicates that omitting the water and sanitation program does not introduce an upward bias. Thus, all in all, Duflo (2001) does not find evidence that the water and sanitation program cause an omitted bias when leaving it out. However, for the present sample, this will be examined closer in section 7. Finally, another good reason for not controlling for the water and sanitation program is that it restricts the data available for performing the estimations.

Also important for the independence assumption is that individuals or their parents do not choose their exposure status. As discussed in section 4.3, this could happen if they systematically moved to or from high intensity districts either before starting in school or before giving birth to the child. However, as shown in section 4.4, this is not likely to be the case. Moreover it is further checked for in subsection 7.2 in table 6 by excluding and including movers in the estimation of equation (13). From this, there is no evidence of endogenous migration being an issue.

The second assumption required for a causal interpretation of the estimate of program exposure is the *exclusion restriction*. This condition requires that program exposure should only affect children through its effect on parental length of schooling. Thus, it indirectly implies that in absence of the program, the child human capital formation across parental cohorts would not have been different in high and low intensity regions. This could be violated if the quality of schools changed over time. However, as explained in subsection 4.2, Duflo (2001) documents that

the qualifications of teachers did not worsen considerably due to the government's large effort to train new teachers and because of the previous five-year plan's recruitment freeze of civil servants which had put many newly educated teachers into unemployment.

Another potential issue might be that the school construction affected class composition as school availability was improved for groups that had not earlier had access to primary school. This might affect social interchange between groups that had not met before with consequences for patterns of assortative mating as an example (Lundborg et al. 2012). The ability composition in the classroom could also have changed if the group of compliers predominantly belonged to the lower end of the ability distribution, which in turn could have had negative consequences for the learning outcome of high ability pupils. However, as mentioned earlier, Duflo (2001) finds that the most important effect of the program was the reduction of the distance to school. From this, it is therefore not clear that a change in the ability composition should have taken place or at least not that it should have affected the compliers as long as the ability distribution is evenly distributed across space. Furthermore, program exposure could have indirect impacts on the local society through a generally better educated population through the labor market as an example. This would, however, be a more general threat to all studies of school reforms or programs as noted by Lundborg et al. (2012).

The third assumption to be fulfilled is that program exposure has to be *relevant* –i.e. it has to affect the educational achievement by parents– such that it can be used as an exogenous source of variation in length of schooling. The results in subsection 7.2 show that this condition is met and that the relation is fairly strong.

Finally, the *monotonicity assumption* requires that the sign of response in the study population is homogeneous. So while program exposure may not have had any effect on some persons, the ones who are affected must have been affected in the same direction. In other words, in order to obtain an estimate of the weighted average of the individual causal effects, the affected persons must have got *more* education as an effect of the school construction program and hence no person may have reduced his (her) investment in schooling as a response to the program. While such possibilities can never be completely ruled out, it seems particularly unlikely that individuals would reduce their length of education *because* more schools became available.

Thus, given these four assumptions –which are all likely to be fulfilled as argued above–, the IV estimate can be interpreted as the effect of years of schooling on those

individuals (the compliers) whose length of education was changed by the INPRES school construction program. This estimate reflects the *local average treatment effects* (LATE). As documented in section 7.3.4 and in Duflo (2001), the program fundamentally increased the average length of education by augmenting primary schooling. Therefore, the group of compliers clearly belongs to the lower end of the educational distribution with considerably less education than examined in the papers referred to in subsection 3.2 in developed countries.

6.3 Second Stage Controls

It would be interesting to explore the impact of parental education besides, for instance, the direct wage effect; corresponding to the estimate of parental education conditioned on parental wage. This is, nevertheless, only possible if this extra control is not endogenous to the system. Otherwise, it is necessary to instrument it for exactly the same reasons as parental education. Moreover, even if this extra control is exogenous (which is very unlikely) or we had an instrument for it (which is not the case), another problem arises as it is generally found in the literature that the wage is an outcome of education (see e.g. Duflo 2001, Angrist & Keueger 1991). Therefore, controlling for the household income, consumption per capita, parental wage, or similar variables would lead to a bias in the estimators –typically referred to as the *bad control problem*[25].

The bad control problem stems from adding a control variable that in itself is an outcome of the *treatment* variable –in this case parental education– which leads to a subtler version of selection bias than the omitted variable bias discussed previously. To see this, suppose we want to evaluate the impacts of higher education while conditioning on income. In this case, even if higher education is randomly (or as good as randomly) assigned, individuals with low and high educational backgrounds who have the same income can be expected to differ systematically, because it takes special qualities to attain a high income despite not being well educated or vice versa. Then, as the treatment (highly educated) group cannot be expected to have had the same outcome as the control (less educated) group in absence of the treatment, the difference between their outcomes can no longer be interpreted as the *causal* effect of having better education.

Consequently, I will not follow much of the past development literature that condition on different possible pathways such as the more recent studies done by

[25]For a more thorough, econometric explanation see Angrist & Pischke (2008).

Block (2007) and Aslam & Kingdon (2012). Instead, I only control for variables that are not outcomes of parental education and that I consider exogenous such as age, gender, and parental height. However, as it is still of great interest to get some hints of potential mediators of the effect of parental education, subsection 7.3.3 tries to shed some light on this.

7 Results

7.1 OLS Relationships

Table 4 and 5 present OLS results[26] on the relationships between parental years of education and the different outcome variables, described in section 5, based on the estimation specification in equation (12). In general, when considering all children, the magnitude of the maternal estimates is slightly larger than the paternal ones[27]. Moreover, in overall, the size of the estimates points to stronger relationships between parental education and child human capital outcomes for boys than for girls.

In particular, table 4 displays strong associations between parental education and child anthropometric measures (height, weight, BMI), except for girls' BMI. Moreover, as documented in Block (2007), strong, positive associations are found between parental education and the child's biometric measure of hemoglobin concentration. Further, the child's probability of suffering from anemia is negatively related to parental education, while not significant for girls and mothers or boys and fathers separately. Thus, all these results on child health outcomes support the conventional findings in the literature that there are strong positive relations between parental education and child health measured in different ways.

Table 5 presents strong, positive relationships between parental education and child education and skills of nearly same size for mothers and fathers. One more year of parental education is for instance associated with around 0.08 more years of schooling for the child conditioned on age among others. Also, one more year of parental education is associated with a two (one) percent higher score in the

[26]Note that only the observations used for the main analysis with data on the water and sanitation program are included in these two tables, although including all observations does not change the results substantially.

[27]For the three binary outcome variables used in this study (anemia, immunization, iodized salt), the significance level is the same when performing probit estimations instead of OLS. Due to the triviality, no probit estimation is reported.

Table 4: OLS Relationships on Child Health

	Independent Variable: **Parental Length of Schooling**					
	Maternal	Paternal	Maternal	Maternal	Paternal	Paternal
Dependent	**All Children**		**Girls**	**Boys**	**Girls**	**Boys**
Variable	(1)	(2)	(3)	(4)	(5)	(6)
Height	0.226***	0.169***	0.222***	0.238***	0.153***	0.186***
	(0.022)	(0.021)	(0.028)	(0.029)	(0.027)	(0.027)
N	11,576	11,250	5,674	5,902	5,543	5,707
Weight	0.121***	0.083***	0.104***	0.142***	0.073***	0.093***
	(0.017)	(0.015)	(0.023)	(0.025)	(0.018)	(0.022)
N	11,577	11,238	5,664	5,913	5,526	5,712
BMI	0.026***	0.013**	0.013	0.039***	0.012	0.014*
	(0.007)	(0.006)	(0.009)	(0.009)	(0.008)	(0.008)
N	11,516	11,174	5,638	5,878	5,498	5,676
Hemoglobin	0.016***	0.016***	0.012**	0.020***	0.014***	0.017***
	(0.005)	(0.004)	(0.006)	(0.006)	(0.005)	(0.006)
N	11,074	10,739	5,391	5,683	5,274	5,465
Anemia	-0.003**	-0.004***	-0.003	-0.004†	-0.004**	-0.003
	(0.001)	(0.001)	(0.002)	(0.002)	(0.002)	(0.002)
N	11,074	10,739	5,391	5,683	5,274	5,465

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. All regressions control for parental district of birth, parental place of birth, cohort intervals indicating year of parental birth, interactions between parental birth cohort dummies and the number of children in the parental district of birth in 1971, province of residence dummies, survey round dummies, age, age squared, and a constant term. Model (1) and (2) also controls for gender. The height, weight, and BMI regressions control additionally for maternal and paternal height and a dummy indicating whether these data are missing. BMI regressions further control for age cubed. Only observations used for the main analysis are used. Each estimate is obtained from separate regressions.

cognitive test (primary school exam) which is very similar both between mothers and fathers as well as girls and boys.

Consequently, both tables show strong associations between parental education and a wide range of child human capital outcomes similar to the conclusions reached in the existing literature. However, what is not clear is whether this reflects causal relationships or rather unobserved characteristics.

Table 5: OLS Relationships on Child Education and Skills

	Independent Variable: **Parental Length of Schooling**					
	Maternal	Paternal	Maternal	Maternal	Paternal	Paternal
Dependent	**All Children**		**Girls**	**Boys**	**Girls**	**Boys**
Variable	(1)	(2)	(3)	(4)	(5)	(6)
Length of Schooling	0.089***	0.063***	0.084***	0.096***	0.054***	0.073***
	(0.006)	(0.004)	(0.007)	(0.008)	(0.005)	(0.006)
N	9,694	9,145	4,738	4,956	4,506	4,639
Ln Cognitive Test Score	0.023***	0.021***	0.025***	0.022***	0.023***	0.018***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
N	9,078	8,618	4,431	4,647	4,241	4,377
Ln School Exam Score	0.011***	0.010***	0.010***	0.011***	0.009***	0.011***
	(0.001)	(0.001)	(0.002)	(0.002)	(0.002)	(0.002)
N	3,709	3,696	1,839	1,870	1,834	1,862

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. For details on controls, see the table note in table 4. Regressions for cognitive test score additionally control for type of test, while the ones for school exam score control for age at the time of the exam and year of exam dummies. Only observations used for the main analysis are used. Each estimate is obtained from separate regressions.

7.2 First Stage Results

In this subsection, the focus is directed to the instrumental variables. For the INPRES school construction program to be a relevant instrument, exposure to the program needs to have a strong effect on parental length of schooling. Table 6 presents the regression results for equation (13) when adding more controls for the group of non-movers and all, respectively; however, this is without controlling for V.

It is seen both when excluding –model (1) to (3)– as well as including movers –model (4) to (6)– that there is a strong relationship between program exposure and parental years of schooling. On average, each additional INPRES school per 1,000 school age children (5-14 years) that had been built the year prior to a person's school start increased the educational attainment by approximately a third of a year for mothers and a fourth for fathers, although the estimates are not significantly different. Hence, the school construction did in fact have a large impact on parental school achievement as the youngest cohort fully exposed was, on average, exposed to more than two INPRES schools per 1,000 children and around 1.5 schools when including the partially exposed.

Table 6: The Impact of the School Construction Program on Parental Schooling

Independent Variable	Dependent variable: **Length of Schooling** – – **Non-Movers** – – (1)	(2)	(3)	– – – **All** – – – (4)	(5)	(6)
Maternal Exposure	0.340***	0.338***	0.332***	0.308***	0.306***	0.287**
	(0.105)	(0.095)	(0.116)	(0.109)	(0.092)	(0.112)
N	4,980	4,980	4,980	5,514	5,514	5,514
Paternal Exposure	0.246**	0.236**	0.261**	0.250**	0.239**	0.222†
	(0.100)	(0.104)	(0.113)	(0.101)	(0.103)	(0.115)
N	4,912	4,912	4,912	5,432	5,432	5,432
Controls by District Interacted with Cohort Dummies:						
Enrollment rate		Yes	Yes		Yes	Yes
W. & S. Program			Yes			Yes
Excluding Movers	Yes	Yes	Yes			

Note: Robust standard errors are clustered at the district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. All regressions control additionally for district of birth, place of birth, birth cohort dummies, interactions between these and the number of children in the district of birth in 1971, and a constant term. Each estimate is obtained from separate regressions. F-values are not reported due to reason explained in the text.

The estimates change somewhat –though not significantly– when adding controls for the enrollment rate among the population in 1971 in the district of birth and for the water and sanitation program, both in interaction with birth cohort dummies. Furthermore, including movers makes especially a difference for the magnitude of the estimate of maternal program exposure. However, the estimates do not, in particular, suggest that they should be biased due to endogenous migration meaning that the validity of the procedure does not seem to worsen. Moreover, note that only parents with data on all controls have been included in table 6 in order not to confuse changes in the estimates from the addition of more controls with different subgroups of compliers.

As a rule of thumb, the F-value should be at least 10 in order to have a valid IV estimate (see e.g. Staiger & Stock 1997). It is, however, not possible to obtain the F-statistic for the OLS regressions in table 6 because the estimated covariance matrix of moment conditions is not of full rank since there are some singleton dummy variables as some districts of birth have only one corresponding parent. Though, it is possible for the 2SLS regressions when partialling out some dummy variables. Therefore, the F-values reported for the first stages in the appendices are obtained

when partialling out the dummy variables of province of residence, survey round, and parental district of birth. This procedure generally lowers the F-values to a large degree as variables that are partialled out do not contribute to the calculation of the F-statistics and these excluded dummies are, in general, highly significant. Thus, the reported F-values should only be interpreted as minimum values and hence not the actual value as they for all regressions are much larger. Nonetheless, generally, the F-values are sufficiently above 10.

As a result of this examination, the approach chosen for the main IV analysis is to use all parents (i.e. non-movers and movers) that have data on the water and sanitation program and present the results with the three control versions corresponding to model (4) to (6). From the first stage of these results[28], it will be seen that the estimate of paternal exposure is not particularly affected by the addition of controls. However, in most cases, adding the controls for the water and sanitation program indicates a downward bias when omitting it which has as its consequence that it introduces more noise in the estimation. But as long as we believe that these two INPRES programs are positively correlated in relation to their effect on paternal schooling, not controlling for the water and sanitation program would not be a threat to the validity of the identification strategy. Consequently, in addition to this main approach, estimations of paternal schooling will be presented without controlling for the water and sanitation program including the group of fathers without data on this program.

On the other hand, the first stage results for maternal exposure point to the opposite effect than for fathers, meaning that omitting the water and sanitation program would possibly cause an upward bias and that the variation in the maternal instrument might not solely reflect the effect of the school program on length of schooling but also the health impact that the water and sanitation program had on mothers. Finally, as a robustness check of the results, section 7.3.5 investigate whether the results are sensitive to the exclusion of the parents that had moved from their district of birth at age 12.

7.3 IV Results

Before turning to the instrumental variables estimates, some comments on the first stage are appropriate. All first stage results from these estimations are shown in appendix B. As not all specifications for the fathers have a relevant first stage, since

[28]See Appendix B.

the estimate of program exposure is not significant, the second stage estimates are reported in italic when the first stage is not valid, while all first stage results are presented. However, in case none of the first stages for an outcome variable is valid, the second stage is not reported. In the sample, more mothers are identified than fathers which might explain why the maternal instrument is stronger.

Finally, due to the triviality, results on the effect of maternal education on child human capital is not reported separately by the child's gender, although the instrument is always valid. The reason is that *no* significant impact is found for *any* of the specifications. Therefore, it is considered sufficient only to report the results for the mothers for all children once. But since the results on sons' human capital for paternal education differ from the specifications with all children, these are reported separately. On the other hand, the separate effect is not reported for girls because the paternal instrument does not work for daughters solely.

7.3.1 Child Health Outcomes

Having commented on the first stage and established that it is strong in general, focus is now directed to the actual results of interest. Table 7 and 8 show the 2SLS results on child health with the three different versions of controls. Surprisingly, hardly any estimate is significantly different from zero indicating that parental education does *not* affect child health, which is in great contrast to the OLS results. However, it is even more unexpected that the sign of all estimates in the height and weight regressions are negative. Moreover, the only borderline significant results –not significant at a five percent level– even suggest a negative causal effect of paternal education on child BMI.

In a developed country setting, one would normally argue that it is a good thing that paternal education affects child BMI negatively. This is, however, not a valid argument in the Indonesian context as nearly everybody in the sample has a BMI-value way below 25 (the rule of thumb for not being overweight), cf. figure 3. Though, when adding the controls for the water and sanitation program, it is no longer significant and the magnitude is reduced. Moreover, only in model (1) in table 8 when including the sons with fathers lacking the water and sanitation controls, the negative effect remains borderline significant, while not when controlling for the enrollment rate interacted with paternal birth cohort dummies in model (2).

In contrast to Block (2007), which examines a different Indonesian data set, the results do not support a causal effect of maternal schooling on child hemoglobin concentration; a measure used as an indicator of general micronutrient status. No-

Table 7: IV Results on Child Health Outcomes, All Children

Dependent Variable	Independent Variable: **Parental Length of Schooling** – – **Mother** – – (1)	(2)	(3)	– – **Father** – – (4)	(5)	(6)
Height	-0.764	-0.746	-0.901	-0.077	-0.050	0.347
	(0.620)	(0.558)	(0.693)	(0.833)	(0.723)	(0.694)
N	11,576	11,576	11,576	11,250	11,250	11,250
Weight	-0.208	-0.181	-0.388	-0.590	-0.601	-0.387
	(0.399)	(0.395)	(0.511)	(0.498)	(0.474)	(0.494)
N	11,577	11,577	11,577	11,238	11,238	11,238
BMI	0.024	0.028	-0.024	**-0.304†**	**-0.326†**	-0.226
	(0.140)	(0.139)	(0.169)	(0.166)	(0.178)	(0.185)
N	11,516	11,516	11,516	11,174	11,174	11,174
Hemoglobin	0.061	0.062	0.119	-0.008	0.005	*0.015*
	(0.107)	(0.104)	(0.141)	(0.133)	(0.139)	*(0.148)*
N	11,074	11,074	11,074	10,739	10,739	10,739
Anemia	-0.010	-0.011	-0.038	-0.029	-0.034	*-0.028*
	(0.027)	(0.026)	(0.035)	(0.041)	(0.043)	*(0.047)*
N	11,074	11,074	11,074	10,739	10,739	10,739
Controls by District Interacted with Cohort Dummies:						
Enrollment rate		Yes	Yes		Yes	Yes
W. & S. Program			Yes			Yes

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. All 2SLS-regressions are run in STATA 12 using the ivreg29 command (Baum, Schaffer & Stillman 2011). Movers are included. For further details on controls, see the table note in table 4. Each estimate is obtained from separate regressions.

tice, that this absence of causal support is found for all three specifications for both parents' education on the group of all children, maternal schooling on respectively boys and girls, and paternal education on boys. Hence, it seems to be robust that parental education does *not* affect the child's micronutrient status.

This is important since Block (2007) does not instrument maternal education as he argues that the assumption of exogenous education made in the early literature is good[29]. Nonetheless, this present study clearly documents that the assumption of exogenous maternal education is not reasonable. Moreover, the number of ob-

[29]More precisely, he argues that this assumption of maternal education being exogenous is empirically plausible because 55 percent of his sample of mothers have six years of education, i.e. primary school completed. However, this fact might exactly be the reason why it is *not* exogenous as the mothers with either more or less education probably are different from the rest.

Table 8: IV Results on Son's Health Outcomes

Dependent Variable	Independent Variable: **Paternal Length of Schooling** (1)	(2)	(3)	(4)	(5)
Height	-0.467	-0.463	-0.372	-0.343	-0.013
	(0.871)	(0.811)	(0.761)	(0.683)	(0.631)
N	6,076	6,076	5,707	5,707	5,707
Weight	-0.751	-0.804	-0.614	-0.653	-0.466
	(0.516)	(0.564)	(0.427)	(0.456)	(0.461)
N	6,082	6,082	5,712	5,712	5,712
BMI	**-0.360***	-0.370	-0.242	-0.251	-0.212
	(0.213)	(0.225)	(0.162)	(0.167)	(0.184)
N	6,043	6,043	5,676	5,676	5,676
Hemoglobin	0.096	0.080	0.057	0.037	0.026
	(0.136)	(0.142)	(0.122)	(0.123)	(0.154)
N	5,798	5,798	5,465	5,465	5,465
Anemia	-0.091	-0.090	-0.069	-0.066	-0.063
	(0.056)	(0.059)	(0.045)	(0.048)	(0.056)
N	5,798	5,798	5,465	5,465	5,465
Controls by District Interacted with Cohort Dummies:					
Enrollment rate		Yes		Yes	Yes
W. & S. Program					Yes
Restricted Sample			Yes	Yes	Yes

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. All 2SLS-regressions are run in STATA 12 using the ivreg29 command (Baum et al. 2011). Movers are included. For further details on controls, see the table note in table 4. Each estimate is obtained from separate regressions.

servations should not be a problem compared to the analysis done by Block (2007) as that study uses a sample of one third the size of the current. Though, still, it cannot be ruled out that the lack of significance might be due to low statistical power due to the relatively small sample size.

Consequently, the IV results on child health are somewhat surprising in light of the existing literature on child health in a development context. Contrary to what was anticipated, child BMI was found to be borderline significant and negatively affected by paternal education; although not very robust across specifications. Nevertheless, certainly, the most important result is that *no* impact of parental schooling on child health is found. However, this study is not the first that cannot reject

the null-hypothesis of *no* causal impact of parental education on child health. For instance, Lindeboom et al. (2009) do not find any impact of increasing the school leaving age in the UK by one year on child health (height and BMI among other indicators). Also, McCrary & Royer (2011) do not find significant effects of exogenous variation of maternal education from school entry policies in the U.S. on infant health outcomes. Finally, the results from this analysis do not support the assumption of maternal education being exogenous in Indonesia implying that the findings in Block (2007) might only reflect correlations between maternal schooling and child micronutrient status and hence not any causal effect.

7.3.2 Child Education and Skills

Table 9: IV Results on Child Education and Skills, All Children

Dependent Variable	Independent Variable: **Parental Length of Schooling** – – **Mother** – – (1)	(2)	(3)	– – **Father** – – (4)	(5)	(6)
Length of Schooling	0.034 (0.104)	0.047 (0.107)	-0.018 (0.143)	0.023 (0.129)	0.051 (0.124)	*-0.158 (0.200)*
N	9,694	9,694	9,694	9,145	9,145	9,145
Ln Cognitive Test Score	**0.054†** (0.029)	**0.058†** (0.030)	0.012 (0.039)	0.140 (0.093)	0.141 (0.094)	*0.094 (0.124)*
N	9,078	9,078	9,078	8,618	8,618	8,618
Ln School Exam Score	-0.027 (0.036)	-0.028 (0.034)	-0.019 (0.039)			
N	3,709	3,709	3,709			
Controls by District Interacted with Cohort Dummies:						
Enrollment rate		Yes	Yes		Yes	Yes
W. & S. Program			Yes			Yes

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. All 2SLS-regressions are run in STATA 12 using the ivreg29 command (Baum et al. 2011). Movers are included. For further details on controls, see the table notes in table 4 and 5. Each estimate is obtained from separate regressions.

Directing the attention to the aspect of child skills, there is no evidence of any causal impact of parental education on child length of schooling, cf. tables 9 and 10. Moreover, remember that despite it is not reported here, there is neither any impact of maternal education on the education of girls nor boys separately. It is remarkable not to find any educational spillover effect from parents to their children. Therefore,

Table 10: IV Results on Son's Education and Skills

Dependent Variable	Independent Variable: **Paternal Length of Schooling** (1)	(2)	(3)	(4)	(5)
Length of Schooling	0.002	0.021	-0.011	0.012	-0.123
	(0.168)	(0.162)	(0.140)	(0.133)	(0.206)
N	4,930	4,930	4,639	4,639	4,639
Ln Cognitive Test Score	**0.170†**	**0.174†**	**0.135****	**0.135****	*0.097*
	(0.094)	(0.094)	(0.067)	(0.066)	*(0.088)*
N	4,646	4,646	4,377	4,377	4,377
Controls by District Interacted with Cohort Dummies:					
Enrollment rate		Yes	Yes		
W. & S. Program			Yes		
Restricted Sample			Yes	Yes	Yes

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. All 2SLS-regressions are run in STATA 12 using the ivreg29 command (Baum et al. 2011). Movers are included. For further details on controls, see the table notes in table 4 and 5. Each estimate is obtained from separate regressions.

as a robustness check, similar regressions using the most recent observation for all children instead have also been performed as one could imagine that the lack of findings might simply be that most children still attend school due to the relatively low average age in the sample (10.7 years). However, that exercise (in which the average age is 12.2 years) did not produce different results.

In contrast, at the same time, the results point to a positive impact of maternal education on children's cognitive skills. This finding is, however, not very robust as it turns insignificant when adding the controls for the water and sanitation program as well as when dividing the sample by gender. Moreover, when only considering paternal education on sons' cognitive skills (table 10), there are indications of a positive effect –both for the broader sample of fathers and the one only containing fathers with data on the water and sanitation program. However, the first stage is not relevant for the specification controlling for the water and sanitation program, questioning the robustness of these results.

Neglecting the weak robustness of the results on cognitive score, the estimates of the impact of paternal schooling on son's cognitive ability are large in economic magnitude. One additional year of paternal education might result in an increase in son's cognitive test score of 14-17 percent, ceteris paribus. These estimates

are much larger than the corresponding OLS estimates, although they are only borderline significantly different from each other due to the inefficiency of the IV estimates. On the other hand, the IV estimates of maternal education on child cognitive test score are considerably smaller in magnitude than the ones of paternal education on sons' test score and not significantly different from the OLS estimate. Furthermore, this difference in size is opposite to what was anticipated as one would expect cognitive skills to be positively correlated with unobserved ability, in which case the OLS estimate would be larger than the IV estimate.

Finally, the primary school exam score is not found to be affected by maternal education and the estimated sign is even negative, while the instrument for fathers is not relevant. It is interesting to note that contrary to the expectation, on average, the mother's length of schooling does not have an impact on the exam score of her child. One explanation might be that, when taking the national exam at the end of primary school, the child has already on average achieved more education than his (her) mother.

All in all, evaluating the findings on child education and skills displayed in tables 9 and 10, the findings indicate positive impacts of parental education on child cognitive ability. However, the estimated effects do not strongly differ from the OLS estimates which points to no unobserved ability —or other variables that might bias the estimates– in the formation of children's skills but instead complete spillover effects. Moreover, importantly, the exam score is not affected by maternal education and no causal effect of parental schooling on child education is found; the latter is in line with the conclusion reached in Black et al. (2005). In other words, the results suggest that the strong associations between parental and child education are predominantly due to heterogeneity such as family background and inherited ability and are not to a large degree explained by educational spillovers. However, this lack of evidence of educational spillovers could possibly be explained by the fact that most children are simply too young to have finished their schooling and therefore the potential spillover effects from parental education might not have had time to express themselves.

7.3.3 Possible Pathways

In the previous subsections, only little evidence of a causal impact of parental education on child human capital was found. However, some indications were found in terms of cognitive skills and BMI. Therefore, this subsection is devoted to the analysis of whether the findings might be driven by mediators as child health inputs,

household size, assortative mating, and per capita expenditure. It is not possible to perform a complete analysis of the potential mediating effect of these variables as it would require one instrument for each of them. It is, meanwhile, still possible to examine how parental education affects the outcome of them –a strategy which might provide some suggestions for the channels through which parental education might work[30].

Table 11: IV Results on Potential Mediators

	Independent Variable: **Parental Length of Schooling**					
Dependent	– – **Mother** – –			– – **Father** – –		
Variable	(1)	(2)	(3)	(4)	(5)	(6)
Immunization	0.043	0.040	0.048			
	(0.062)	(0.059)	(0.053)			
N	3,965	3,965	3,965			
Iodized Salt	0.005	0.004	-0.001	0.039	*0.033*	*0.009*
	(0.041)	(0.036)	(0.051)	(0.071)	(0.076)	(0.093)
N	5,432	5,432	5,432	5,357	5,357	5,357
Household	0.028	0.031	-0.122	**-0.840***	**-0.834***	**-1.023***
Size	(0.298)	(0.291)	(0.311)	(0.479)	(0.503)	(0.604)
N	5,462	5,462	5,462	5,384	5,384	5,384
Schooling	**1.276*****	**1.283*****	**1.426*****	**0.609***	0.593	0.511
of Spouse	(0.332)	(0.315)	(0.411)	(0.357)	(0.373)	(0.359)
N	5,449	5,449	5,449	5,376	5,376	5,376
Ln pc	0.008	0.006	0.036	0.050	0.045	0.089
Exp.	(0.052)	(0.049)	(0.059)	(0.069)	(0.072)	(0.079)
N	5,432	5,432	5,432	5,354	5,354	5,354
Controls by District Interacted with Cohort Dummies:						
Enrollment rate		Yes	Yes		Yes	Yes
W. & S. Program			Yes			Yes

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. All 2SLS-regressions are run in STATA 12 using the ivreg29 command (Baum et al. 2011). Movers are included. All regressions control for parental district of birth, parental place of birth, cohort intervals indicating year of parental birth, interactions between parental birth cohort dummies and the number of children in the parental district of birth in 1971, province of residence dummies, survey round dummies, and a constant term. The immunization regressions also control for age, age squared, and gender of the child. Each estimate is obtained from separate regressions.

First, according to the theory presented in section 2, an indirect effect of more

[30]The corresponding OLS relationships are presented in appendix C.

parental education would be better health inputs into the production of child health and cognitive ability. Therefore, in theory more parental education would induce parents to prevent the child from serious childhood diseases through various harmless vaccinations. Hence, we would expect maternal education to increase the child's probability of having received the five common immunizations in Indonesia. These vaccinations are for polio, measles, hepatitis B, BCG (tuberculosis), and DPT (the three infectious diseases diphtheria, pertussis (whooping cough), and tetanus)[31]. However, the results in table 11 do not support this expectation as the estimates are clearly not significantly different from zero, which is opposite to the OLS relations.

Moreover, another very cheap but extremely important health input for the cognitive development particularly during gestation but also in general is iodine (for an economic paper on the topic, see e.g. Field, Robles & Torero 2009). The most normal way to make sure to intake enough of this essential micronutrient is through salt. It is not a difficult or expensive process to enrich salt with iodine but it is necessary to be aware of the importance of its intake in the daily food. Therefore, it is anticipated that parents with more education would be more likely to ensure that the salt used for cooking in the household contains iodine. The data on this variable is obtained from a simple test done in the field[32]. However, no such evidence is found on the probability that the salt is iodized. Again, this is in large contrast to the very significant OLS relations.

Second, as argued in the theory section, it is expected that longer education introduces a higher alternative cost of having children due to a higher shadow price of the parent's time. Therefore, it would be reasonable to expect that the number of children, and implicitly the size of the household, would be negatively affected by parental education. However, using the same instrumental approach as in this paper, Breierova & Duflo (2004) do not find any impact of parental schooling on fertility before age 25 years. Their finding might also be the reason why maternal education is not found to impact household size in table 11.

On the other hand, paternal education proves to reduce the household size with as much as one person for each additional year of schooling for the fathers in the

[31]Descriptive statistics on the outcome variables analyzed in this section is to be found in appendix A.

[32]The test turns blue if it contains iodine and remains white otherwise. Therefore, "white" and "blueish" have been coded as 0 and "blue" as 1. For the analysis, the earliest observation has been selected for each parent as it has become more normal to use iodized salt over time. However, it was not observed in the first survey round.

sample. This large effect on fathers' family size suggest that the quantity-quality hypothesis of children might contribute to an explanation behind the large and positive effects of paternal education on sons' test score in cognitive skills. However, as these human capital outcomes could not be investigated separately for girls and the same positive effects on cognitive ability were not found for all children together, the quantity-quality theory might only be the case for sons. Moreover, it is difficult to explain the negative impact of paternal education on BMI which in the Indonesian context rather is a deterioration of child health than an improvement.

Comparing these IV findings with the OLS relations is interesting as maternal education and household size in the latter are strongly and negatively correlated. This suggests that mothers with more education do not reduce their family size per se although smaller families often have better educated mothers. On the contrary, the OLS relationship did not show any association between paternal education and household size different from zero.

Moreover, another way parental education might affect the child's human capital outcome is through assortative mating as discussed in section 2.2. The only very robust significant finding in this analysis is that there is strong evidence of positive assortative mating for mothers. In other words, a woman's education makes her more attractive on the marriage market and as a result, it enables her to choose a husband with longer education than what she otherwise could have expected. This effect of women's education on the choice of partner and consequently the father of their children is quite large such that each extra year of schooling increases the father's length of education with around 1.3 years. Furthermore, the estimate is significantly different from the OLS estimate at the 10 percent significance level, pointing to heterogeneous effects although they are not strongly different from each other.

The effect for fathers is not as strong since the estimate in model (6) is only significant at the 10 percent level in a one-sided test with the alternative hypothesis that it is larger than zero. However, as seen later from the sensitivity analysis (subsection 7.3.5), the results suggest a robust estimate of positive assortative mating for fathers. Thus, it is found that for each extra year of education that the father has, the mother's length of schooling is increased by around 0.6 years. This estimate is, nonetheless, not much different from its corresponding OLS estimate. This implies that the man's choice of wife and thus her embodied educational level is *not* affected by some omitted variables biasing the OLS estimate.

As both parents' length of schooling were suggested to influence child cognitive

skills, the findings of assortative mating might hint to a channel through which parental education might influence child human capital. Hence, assortative mating might possess the property as a multiplier of parental education. Furthermore, if we only have confidence in the findings for sons, one way could still be that men with longer education would marry women with more ability and better genes if we reasonably assume that these (ability and "good" genes) are positively correlated with maternal length of education. Thus, even if maternal education itself does not affect the child's (son's) human capital, her good genes would be transmitted to the child and then indirectly affect his outcome.

Finally, one would expect the per capita expenditures[33] to increase in the education of the parent. Despite the OLS estimates showing that each additional year of parental schooling is associated with seven percent larger per capita expenditures[34], the instrumental variables results do not show any significant impact. This is contrary to what was anticipated as Duflo (2001) found that each additional year of education increased men's wages by around nine percent in Indonesia, which would loosen the budget constraints and thus increase per capita expenditures. Using levels instead of the logarithm yields similar results.

Consequently, the most robust potential mediator of parental education found for child outcomes is assortative mating, while there is also some evidence pointing to smaller households for fathers. Remarkably, parental education is, meanwhile, not found to increase the likelihood of better health inputs or augment per capita expenditures.

7.3.4 The Group of Compliers

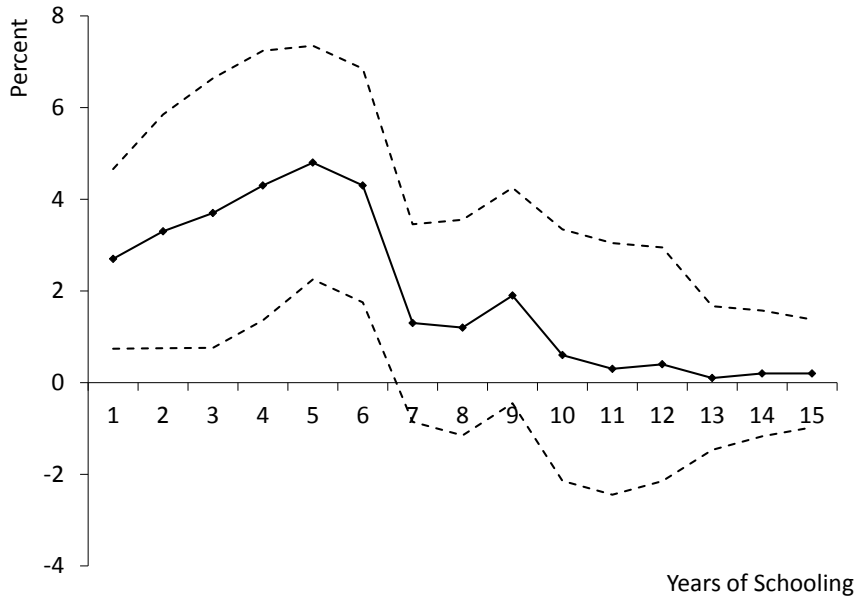
The interpretation of the results of parental education on the various outcome variables studied in the analysis depends on the group of compliers; i.e. the group of parents whose length of education was affected by the school construction program. This is especially useful information to possess when comparing the results with other studies as one more year of schooling might have quite different (beneficial) effects for the outcome of interest depending on the level of education. For instance, the essential skill learned during the first years of education is to read and write while at higher levels, the effect of education might rather be a shifter of preferences

[33]These are given in Indonesian Rupiah in 2010 prices deflated by data from the World Bank. The individual's most recent observation has been selected for the analysis in order to avoid to much noise due to different time periods.

[34]Very similar results are found both for per capita food and educational expenditures and are therefore left out of the analysis due to their triviality.

or the ability to understand the more scientific aspects of the beneficial or damaging effect of various inputs to the child quality production function.

Figure 5: Difference in Differences in Percentage in CDF for Mothers



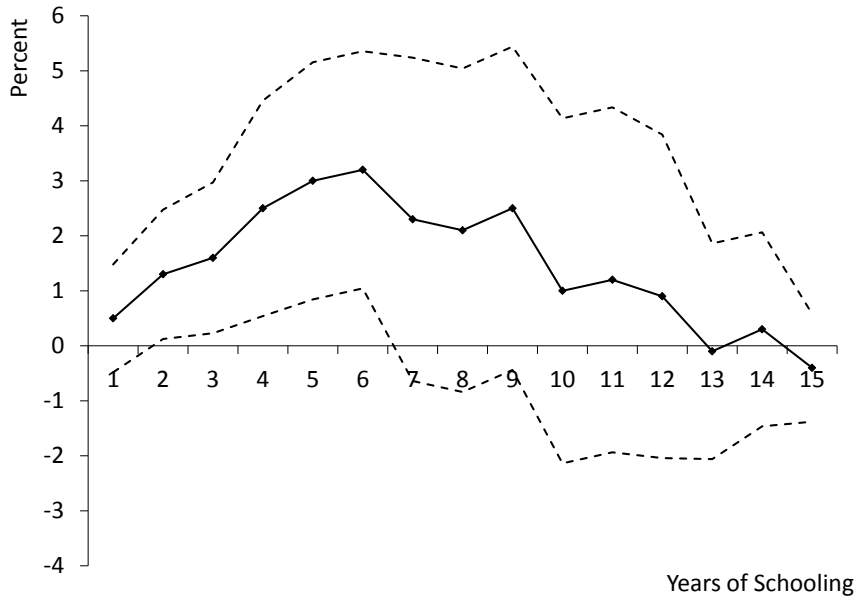
Note: Estimated from linear probability model for mothers used in the main analysis. The dashed lines represent the 95 percent confidence interval.

To investigate this, I estimate regressions similar to model (4) in table 6 but instead of length of education, the dependent variable is a dummy indicating whether the individual has at least a certain number of years of schooling[35]. From this approach, estimates of the DiD in the cumulative distribution function (CDF) of schooling are obtained. These estimates provide information on the impacts of the program at each level of education and are plotted in figure 5 and 6 for respectively mothers and fathers. More precisely, each estimate gives the change in probability of having finished that certain level of education for each school per 1,000 school children in the district of birth that had been built the year prior to the parent's school start compared to the non-exposed parents.

For mothers, the shape of the plot indicates that the impact of the program is increasing until the two last years of primary school after which the effect is decreasing although not significantly different from zero. As the average program exposure for mothers exposed to the program was 1.6 schools per 1,000 children, these women

[35]This is similar but not identical to Duflo (2001) as the author only distinguishes between high and low intensity regions and thus not the actual program exposure.

Figure 6: Difference in Differences in Percentage in CDF for Fathers



Note: Estimated from linear probability model for fathers used in the main analysis. The dashed lines represent the 95 percent confidence interval.

had, on average, a seven percent higher probability of finishing primary school than their older peers caused by the school program. Likewise, fathers had an increased likelihood of finishing primary school by nearly five percent. These findings are slightly different from what Duflo (2001) finds as she additionally observe an effect, though smaller, at the junior high school level (until nine years of education).

It is remarkable to observe such large difference in differences at the primary school level –which was the exact, political target– and that at the same time, there were no significant spillover effects on post-primary schooling. This offer further evidence that the underlying assumption of the identification strategy is reasonable. A reason why spillover effects are absent could be that both the direct and indirect costs of junior high school were much higher than the costs of primary schooling additionally to the fact that the political target only concerned the latter level of schooling (Duflo 2001).

Consequently, it is clear to see that the group of compliers in this Indonesian context is composed by parents that otherwise would have gotten even fewer years of primary schooling. Thus, it is noteworthy to keep in mind that, when interpreting the impacts of parental education on child human capital, it is the effect of attaining one more year of primary school education, adding a considerable percentage to the

length of schooling while not a high level of knowledge. This could also be a reason for the lack of evidence of parental education as the acquired skills and curriculum taught might not be of much relevance for child rearing in practice.

7.3.5 Sensitivity Analysis

As a way to check the robustness of the findings in the previous subsections, similar specifications to the ones in table 7 to 11 are explored but while *excluding* the parents that had moved to another district at age 12 years. In this manner, any potential issues with endogenous migration and measurement errors can be assessed, despite the fact that section 4.4 showed that the former is unlikely to be an actual problem. At the same time, what should be taken into account is that since the sample size is relatively small, leaving out some observations might naturally change the estimates slightly. All the results are to be found in appendix D.

First, the general findings of absence of impact of parental education on child health including movers are also found when excluding them (table 18). There is no longer significant indications of a negative impact of paternal schooling on child (sons') BMI. Moreover, there is still no sign of an effect of parental education on child micronutrient status (hemoglobin concentration or probability of suffering from anemia). Consequently, the conclusion that child health is *not* affected by parental education seems robust.

Second, when excluding movers, the positive estimates of maternal schooling on the cognitive test score remain significant at the five percent level; however, not when dividing the sample by gender which was also the case before. Also, as when including movers, adding the water and sanitation program to the controls still makes the estimate insignificant. In terms of the impact of paternal education on sons' cognitive test score, it is still large in magnitude and significant at the 5 percent level with the two first control versions, but when adding the water and sanitation controls, the first stage turns invalid. Therefore, from this robustness check, we still observe indications of positive effects of parental education on child cognitive skills, nonetheless, no clear and unambiguous evidence. At the same time, there is still no evidence that parental education should impact the child's length of education or exam score.

Finally, the results on potential mediators still do not show any impact of parental education on per capita expenditures or health inputs. Meanwhile, there is support for a strong and robust impact of maternal education on the education of her spouse, and the size of this estimate stays by and large unchanged. More-

over, the corresponding estimate of paternal education is now robust across all three specifications and is also of same magnitude as found earlier. Consequently, it is fairly robust to claim that positive assortative mating is prevalent in this Indonesian subpopulation and that it is not caused by unobserved heterogeneity as the IV estimates are not significantly different from the OLS estimates. In contrast, the negative effect of paternal schooling on household size is smaller in magnitude and no longer significant. Consequently, the suggestive support of the quantity-quality hypothesis of children between fathers and sons is not to be found among only non-moving fathers.

All in all, this sensitivity analysis strongly supports the findings of no evidence of parental education on child human capital. However, there are still some indications of a positive impact of parental schooling on the cognitive test score. Lastly, the finding of assortative mating is very robust, while no other potential mediator is significant.

8 Discussion and Conclusion

This paper has examined the intergenerational effects of parental education on child human capital in a developing country setting. Similar to the general literature, positive relationships are found between parental schooling and a wide range of child human capital outcomes. In order to evaluate whether these associations in fact represent causal effects, an arguably exogenous increase in the primary school supply in the 1970s in Indonesia is used as an instrument for parental school attainment. This governmental school construction program is shown to have increased schooling significantly both for mothers and fathers. In particular, the group of parents affected by the large increase in the primary school stock is composed of parents belonging to the lowest end of the educational distribution corresponding to six or fewer years of schooling.

For this subpopulation of parents affected by the school construction program, the results provide little evidence of a causal impact of parental education on child health and skills. Some indications of positive effects on cognitive skills are however found, though not robust. Put differently, the results suggest that the strong associations between parental education and child human capital are predominantly due to heterogeneity such as family background and inherited ability and are not to a great extend explained by educational spillovers. These findings are in line with similar conclusions reached in, for instance, Black et al. (2005), Lindeboom

et al. (2009), and McCrary & Royer (2011); all studying developed countries, in which different policy measures are used as exogenous ways of manipulating length of schooling.

Moreover, there are some indications of a negative impact of paternal education on family size; despite, it is not a very robust finding. This might indicate some support for the quantity-quality hypothesis as a potential pathway through which paternal schooling might affect sons' cognitive skills. Furthermore, there is strong evidence that positive assortative mating is taking place for both mothers and fathers. Though, the IV estimates are not greatly different from the OLS estimates which suggests that assortative mating is not due to unobserved heterogeneity but predominantly spillover effects. Therefore, if this analysis should point to one single mediator of the indicated causal impact of parental education on child skills, assortative mating is a clear candidate.

Consequently, this analysis finds no evidence of a causal impact of maternal education on child hemoglobin concentration –a measure of micronutrient status– in great contrast to Block (2007). Therefore, I argue that Block's (2007) conclusions most likely suffer from biased estimates because he assumes that maternal education is exogenous which, the current study has shown, is most likely not the case. Thus, the findings in Block (2007) might only reflect correlations between maternal schooling and child micronutrient status and hence not any causal effect. Though, this study has not been able to examine the interesting aspect of specific nutrition knowledge due to data limitations in contrast to Block (2007).

Contrary to Lundborg et al.'s (2012) findings in the case of Sweden, this evidence from Indonesia does not find that increasing maternal education would be an efficient, political tool to improve child quality. Although it might still be beneficial to augment parental education in Indonesia due to other policy objectives, it is not found to be important for the purpose of enhancing child human capital. On the other hand, as suggested by Glewwe (1999), it would maybe be more relevant and effective to inform parents about healthy inputs and behaviors through other means than expanding primary education such as through health clinics and health campaigns etc. However, this is left for future research to clarify as it is outside the limitations of this study.

A possible explanation of the absence of evidence of any educational spillover of parental education on child human capital might be that at the primary school level, one additional year of schooling might simply not matter for the formation of child human capital. In other words, the acquired skills in primary school might not be

the trigger for a change in preferences, behaviors, and other important inputs in the production of child quality. This is, for instance, found in Lindeboom et al. (2009) and McCrary & Royer (2011) in terms of prenatal care in the context of developed countries. Also, in this study, parental education was not found to increase the child's likelihood of having received immunization or affect the probability that the salt used for the household's cooking contains iodine –an important mirconutrient for human development. Thus, it might be that in that end of the educational distribution, parents continue normal practice concerning child care, perception, and attitude in spite of their additional years of schooling. Or alternatively, the findings might simply reflect that important knowledge for the production of child quality is not part of the curriculum and the skills taught in primary school do not encourage people to seek new information on their own. This might for example explain why Currie & Moretti (2003) *do* find an effect of maternal education on infant health as they study exogenous shocks to maternal college education contrary to primary education.

Moreover, although Duflo (2001) finds that each additional year of education enhances Indonesian male wages by 7-11 percent, I did not find an impact of parental education on per capita expenditures in the household. This absence of finding could be due to lack of statistical power, while another plausible explanation might be that the absolute increase in real wages due to education is relatively small and therefore is mainly allocated to more expensive rent, insurance, or the like. At least, it is worth keeping in mind that, as the average per capita expenditure is around 2 U.S. dollars a day, an economic return to education of around ten percent for each working (male) member of the household can necessarily not make a large difference in terms of a potential income effect. In other words, a possible increase in income due to increased education cannot enable people to go much more to the doctor, pay for much better schooling, eat much better and healthier food etc. All this might explain the absence of the findings of causal effects.

Finally, it is worth noting that compared to most other studies of developing countries, my sample size is large, however, compared to register-based data, it is small. This latter fact might cause a lack of statistical power which in turn might be a reason for the non-rejection of the null-hypothesis that parental schooling does not impact child human capital. Moreover, household surveys are always subject to measurement errors, a trait that is more pronounced than when working with register data; but this is always the case when working with data from developing countries. However, the advantage of the data used in this paper is that four waves

have been carried out making the data source rich in order to compare data for most individuals across the years and thus find the most consistent observations.

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Appendices

A Descriptive Statistics

Table 12: Descriptive Statistics on Parents' Instrumental Variables

	N	Mean	SD	N	Mean	SD
	Mother Not Exposed			**Mother Exposed**		
Potential Mediators:						
Immunization (share)	631	0.51	0.50	3,334	0.62	0.49
Iodized Salt (share)	1,959	0.60	0.49	3,473	0.63	0.48
Household Size	1,971	7.29	2.75	3,491	6.00	2.78
Schooling of Spouse	1,969	7.14	4.33	3,480	8.79	4.04
Ln pc Total Exp. (IDR 2010 prices)	1,969	8.48	0.72	3,463	8.34	0.64
Instrumental Variables:						
Year of Birth	2,002	1956	4.49	3,176	1973	3.99
INPRES Schools per 1,000 children	2,002	2.01	0.92	3,176	2.13	1.03
Program Exposure	2,002	0.00	0.00	3,176	1.63	1.12
Children 1971	2,002	171113	108744	3,176	169604	112280
Enrollment rate 1971	2,002	0.17	0.06	3,176	0.16	0.06
W. & S. Program	2,002	0.48	0.19	3,176	0.48	0.19
Place: Small Town (share)	2,002	0.19	0.39	3,176	0.20	0.40
Place: Big City (share)	2,002	0.10	0.30	3,176	0.06	0.24
	Father Not Exposed			**Father Exposed**		
Potential Mediators:						
Immunization	1,335	0.54	0.50	2,587	0.66	0.47
Iodized Salt	2,449	0.60	0.49	2,908	0.66	0.47
Household Size	2,461	6.89	2.61	2,923	5.70	2.74
Schooling of Spouse	2,460	6.30	4.10	2,916	8.71	3.50
Ln pc Total Exp.	2,456	8.48	0.70	2,898	8.31	0.62
Instrumental Variables:						
Year of Birth	2,505	1955	4.83	2,927	1973	3.77
INPRES Schools per 1,000 children	2,505	2.04	0.95	2,927	2.13	0.98
Program Exposure	2,505	0.00	0.00	2,927	1.59	1.09
Children 1971	2,505	172218	109912	2,927	166416	110611
Enrollment rate 1971	2,505	0.17	0.06	2,927	0.16	0.06
W. & S. Program	2,505	0.48	0.21	2,927	0.48	0.20
Place: Small Town	2,505	0.19	0.39	2,927	0.19	0.39
Place: Big City	2,505	0.09	0.28	2,927	0.07	0.25

Note: Only parents who are part of the main analysis are included, although the number of observations in the regressions can be slightly smaller due to missing observations on province of residence.

B First Stage Results for All Parents

Table 13: First Stage Results for *Child Health Outcomes, All Children*

	First Stage Dependent Variable: Parental Length of Schooling					
	First Stage Independent Variable: Parental Program Exposure					
Second Stage	– – **Mother** – –			– – **Father** – –		
Dependent Var.	(1)	(2)	(3)	(4)	(5)	(6)
Height	0.308***	0.311***	0.286**	0.288**	0.283**	0.305**
	(0.092)	(0.092)	(0.112)	(0.117)	(0.119)	(0.137)
F	30.5	25.8	22.3	14.4	14.1	12.9
N	11,576	11,576	11,576	11,250	11,250	11,250
Weight	0.305***	0.308***	0.283**	0.268**	0.263**	0.276**
	(0.092)	(0.092)	(0.111)	(0.113)	(0.115)	(0.133)
F	31.8	26.7	23.0	13.6	13.1	12.0
N	11,577	11,577	11,577	11,238	11,238	11,238
BMI	0.307***	0.311***	0.286**	0.287**	0.283**	0.302**
	(0.091)	(0.091)	(0.110)	(0.116)	(0.118)	(0.136)
F	30.2	25.8	22.5	14.6	14.7	13.5
N	11,516	11,516	11,516	11,174	11,174	11,174
Hemoglobin	0.329***	0.336***	0.297**	0.226†	0.224†	0.222
	(0.099)	(0.100)	(0.119)	(0.117)	(0.122)	(0.137)
F	26.4	21.9	19.2	23.4”	21.4”	20.1”
N	11,074	11,074	11,074	10,739	10,739	10,739
Anemia	0.329***	0.336***	0.297**	0.226†	0.224†	0.222
	(0.099)	(0.100)	(0.119)	(0.117)	(0.122)	(0.137)
F	26.4	21.9	19.2	23.4”	21.4”	20.1”
N	11,074	11,074	11,074	10,739	10,739	10,739
Controls by District Interacted with Cohort Dummies:						
Enrollment rate		Yes	Yes		Yes	Yes
W. & S. Program			Yes			Yes

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. F-values marked with ” do not partial survey round dummies out. All 2SLS-regressions are run in STATA 12 using the ivreg29 command (Baum et al. 2011). Movers are included. For further details on controls, see the table note in table 4 and 5. Each estimate is obtained from separate regressions.

Table 14: First Stage Results for *Child Education and Skills, All Children*

Second Stage Dependent Var.	First Stage Dependent Variable: Parental Length of Schooling First Stage Independent Variable: Parental Program Exposure – – **Mother** – –			– – **Father** – –		
	(1)	(2)	(3)	(4)	(5)	(6)
Length of Schooling	0.341***	0.352***	0.300***	0.260**	0.265**	0.203
	(0.095)	(0.096)	(0.114)	(0.109)	(0.114)	(0.135)
F	29.3	26.6	22.5	17.3"	16.3"	14.9"
N	9,694	9,694	9,694	9,145	9,145	9,145
Ln Cognitive Test Score	0.288***	0.303***	0.252**	0.211†	0.218*	0.140
	(0.099)	(0.100)	(0.118)	(0.116)	(0.122)	(0.142)
F	39.8	35.9	35.3	15.1	14.4	13.9
N	9,078	9,078	9,078	8,618	8,618	8,618
Ln School Exam Score	0.733***	0.758***	0.693***	0.402	0.434	0.441
	(0.235)	(0.231)	(0.239)	(0.473)	(0.531)	(0.532)
F	13.5	12.7	12.3	10.2	9.4	9.0
N	3,709	3,709	3,709	3,696	3,696	3,696
Controls by District Interacted with Cohort Dummies:						
Enrollment rate		Yes	Yes		Yes	Yes
W. & S. Program			Yes			Yes

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. F-values marked with " do not partial survey round dummies out. All 2SLS-regressions are run in STATA 12 using the ivreg29 command (Baum et al. 2011). Movers are included. For further details on controls, see the table notes in table 4 and 5. Each estimate is obtained from separate regressions.

Table 15: First Stage Results for *Son's Health and Skills Outcomes*

Second Stage Dependent Var.	First Stage Dependent Var.: Paternal Schooling First Stage Independent Var.: Paternal Exposure (1)	(2)	(3)	(4)	(5)
Height	0.352***	0.331***	0.405***	0.394***	0.411***
	(0.118)	(0.126)	(0.120)	(0.129)	(0.147)
F	15.3	15.1	14.8	14.5	12.7
N	6,076	6,076	5,707	5,707	5,707
Weight	0.319***	0.299**	0.370***	0.357***	0.360**
	(0.114)	(0.120)	(0.115)	(0.123)	(0.140)
F	15.4	14.6	14.9	14.0	12.4
N	6,082	6,082	5,712	5,712	5,712
BMI	0.343***	0.322**	0.394***	0.382***	0.394***
	(0.118)	(0.125)	(0.119)	(0.128)	(0.147)
F	14.4	14.1	14.1	13.5	12.0
N	6,043	6,043	5,676	5,676	5,676
Hemoglobin	0.303**	0.290**	0.338***	0.333**	0.324**
	(0.122)	(0.131)	(0.124)	(0.135)	(0.152)
F	16.4"	14.8"	16.1"	14.3"	220.8"
N	5,798	5,798	5,465	5,465	5,465
Anemia	0.303**	0.290**	0.338***	0.333**	0.324**
	(0.122)	(0.131)	(0.124)	(0.135)	(0.152)
F	16.4"	14.8"	16.1"	14.3"	220.8"
N	5,798	5,798	5,465	5,465	5,465
Length of Schooling	0.327**	0.339**	0.403***	0.424***	0.338†
	(0.133)	(0.147)	(0.131)	(0.147)	(0.178)
F	12.9"	11.2"	12.3"	10.7"	9.9"
N	4,930	4,930	4,639	4,639	4,639
Ln Cognitive Test Score	0.302**	0.307†	0.362**	0.374**	0.265
	(0.153)	(0.167)	(0.153)	(0.171)	(0.196)
F	13.4	11.4	11.8	10.2	9.7
N	4,646	4,646	4,377	4,377	4,377
Controls by District Interacted with Cohort Dummies:					
Enrollment rate		Yes		Yes	Yes
W. & S. Program					Yes
Restricted Sample			Yes	Yes	Yes

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. F-values marked with " do not partial survey round dummies out. All 2SLS-regressions are run in STATA 12 using the ivreg29 command (Baum et al. 2011). Movers are included. For further details on controls, see the table notes in table 4 and 5. Each estimate is obtained from separate regressions.

Table 16: First Stage Results for *Potential Mediators*

Second Stage Dependent Var.	First Stage Dependent Variable: Parental Length of Schooling First Stage Independent Variable: Parental Program Exposure – – **Mother** – –			– – **Father** – –		
	(1)	(2)	(3)	(4)	(5)	(6)
Immunization	0.262†	0.267†	0.308†	0.099	0.101	0.142
	(0.141)	(0.140)	(0.169)	(0.149)	(0.150)	(0.167)
F	14.5	12.0	11.2	7.7	6.8	6.4
N	3,965	3,965	3,965	3,922	3,922	3,922
Iodized Salt	0.277***	0.276***	0.242**	0.179†	0.168	0.150
	(0.105)	(0.093)	(0.111)	(0.100)	(0.102)	(0.117)
F	37.1	33.4	30.6	19.0	18.5	18.5
N	5,432	5,432	5,432	5,357	5,357	5,357
Household Size	0.302***	0.301***	0.294**	0.241**	0.229**	0.238**
	(0.108)	(0.094)	(0.114)	(0.100)	(0.101)	(0.114)
F	61.2	54.3	50.4	28.9	27.1	23.2
N	5,462	5,462	5,462	5,384	5,384	5,384
Schooling of Spouse	0.302***	0.301***	0.294**	0.237**	0.226**	0.239**
	(0.110)	(0.096)	(0.115)	(0.101)	(0.102)	(0.114)
F	61.6	54.9	51.1	28.8	27.0	23.0
N	5,449	5,449	5,449	5,376	5,376	5,376
Ln pc Exp.	0.297***	0.295***	0.288***	0.232**	0.220**	0.239**
	(0.111)	(0.095)	(0.114)	(0.101)	(0.102)	(0.115)
F	63.4	57.5	52.1	29.2	27.7	23.1
N	5,432	5,432	5,432	5,354	5,354	5,354
Controls by District Interacted with Cohort Dummies:						
Enrollment rate		Yes	Yes		Yes	Yes
W. & S. Program			Yes			Yes

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. All 2SLS-regressions are run in STATA 12 using the ivreg29 command (Baum et al. 2011). Movers are included. For further details on controls, see the table note in table 11. Each estimate is obtained from separate regressions.

C OLS Relationships

Table 17: OLS Relationships on Potential Mediators

Dependent Variable	Independent Variable: Parental Length of Scholling	
	Maternal	Paternal
Immunization	0.024***	0.021***
	(0.003)	(0.002)
N	3,965	3,922
Iodized Salt	0.018***	0.017***
	(0.002)	(0.002)
N	5,432	5,357
Household Size	-0.023†	-0.006
	(0.012)	(0.010)
N	5,462	5,384
Schooling of Spouse	0.685***	0.575***
	(0.014)	(0.012)
N	5,449	5,376
Ln pc Exp.	0.073***	0.063***
	(0.003)	(0.003)
N	5,432	5,354

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. For details on controls, see the table note in table 11. Only observations used for the main analysis are used. Each estimate is obtained from separate regressions.

D First and Second Stage Results for Non-Movers

Table 18: IV Results on Child Human Capital, Only Non-Movers

Dependent Variable	Independent Variable: **Parental Length of Schooling** – – **Mother** – – (1)	(2)	(3)	– – **Father** – – (4)	(5)	(6)
Height	-0.764	-0.756	-0.766	-0.264	-0.162	0.190
	(0.529)	(0.487)	(0.557)	(1.012)	(0.828)	(0.738)
N	10,429	10,429	10,429	10,135	10,135	10,135
Weight	-0.212	-0.192	-0.243	-0.784	-0.762	-0.549
	(0.316)	(0.308)	(0.351)	(0.642)	(0.593)	(0.565)
N	10,434	10,434	10,434	10,121	10,121	10,121
BMI	-0.000	0.005	-0.004	-0.303	-0.325	-0.221
	(0.123)	(0.124)	(0.134)	(0.193)	(0.207)	(0.195)
N	10,376	10,376	10,376	10,064	10,064	10,064
Hemoglobin	0.077	0.077	0.147	-0.111	*-0.103*	-0.074
	(0.093)	(0.091)	(0.122)	(0.170)	*(0.180)*	(0.157)
N	9,991	9,991	9,991	9,693	9,693	9,693
Anemia	-0.017	-0.018	-0.040	-0.014	*-0.018*	-0.015
	(0.025)	(0.024)	(0.031)	(0.047)	*(0.050)*	(0.047)
N	9,991	9,991	9,991	9,693	9,693	9,693
Length of Schooling	0.039	0.045	-0.004	-0.009	0.030	*-0.162*
	(0.088)	(0.093)	(0.116)	(0.147)	(0.142)	*(0.209)*
N	8,723	8,723	8,723	8,224	8,224	8,224
Ln Cognitive Test Score	**0.045****	**0.048****	0.020	0.165	0.171	0.103
	(0.022)	(0.022)	(0.027)	(0.118)	(0.128)	(0.128)
N	8,158	8,158	8,158	7,748	7,748	7,748
Ln School Exam Score	-0.023	-0.026	-0.018			
	(0.033)	(0.032)	(0.034)			
N	3,317	3,317	3,317			
Controls by District Interacted with Cohort Dummies:						
Enrollment rate		Yes	Yes		Yes	Yes
W. & S. Program			Yes			Yes

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. All 2SLS-regressions are run in STATA 12 using the ivreg29 command (Baum et al. 2011). Movers are excluded. For further details on controls, see the table notes in table 4 and 5. Each estimate is obtained from separate regressions.

Table 19: First Stage Results for *Child Human Capital*, Only Non-Movers

	First Stage Dependent Variable: Parental Length of Schooling First Stage Independent Variable: Parental Program Exposure					
Second Stage	– – **Mother** – –			– – **Father** – –		
Dependent Var.	(1)	(2)	(3)	(4)	(5)	(6)
Height	0.361***	0.362***	0.346***	0.257**	0.252**	0.306**
	(0.100)	(0.106)	(0.125)	(0.112)	(0.114)	(0.130)
F	27.3	24.2	20.5	13.4	12.5	11.3
N	10,429	10,429	10,429	10,135	10,135	10,135
Weight	0.359***	0.361***	0.347***	0.235**	0.229**	0.275**
	(0.099)	(0.105)	(0.125)	(0.109)	(0.112)	(0.129)
F	28.9	25.4	21.5	12.5	11.6	10.3
N	10,434	10,434	10,434	10,121	10,121	10,121
BMI	0.361***	0.363***	0.347***	0.255**	0.250**	0.302**
	(0.099)	(0.105)	(0.124)	(0.110)	(0.113)	(0.130)
F	27.1	24.5	21.1	12.9	12.4	11.0
N	10,376	10,376	10,376	10,064	10,064	10,064
Hemoglobin	0.388***	0.394***	0.367***	0.199*	0.196	0.233*
	(0.106)	(0.113)	(0.133)	(0.114)	(0.120)	(0.134)
F	28.1	22.5	19.3	22.2”	19.9”	17.5”
N	9,991	9,991	9,991	9,693	9,693	9,693
Anemia	0.388***	0.394***	0.367***	0.199*	0.196	0.233*
	(0.106)	(0.113)	(0.133)	(0.114)	(0.120)	(0.134)
F	28.1	22.5	19.3	22.2”	19.9”	17.5”
N	9,991	9,991	9,991	9,693	9,693	9,693
Length of	0.433***	0.441***	0.398***	0.246**	0.247**	0.220
Schooling	(0.105)	(0.113)	(0.129)	(0.110)	(0.116)	(0.137)
F	29.6	28.8	25.3	16.9”	15.2”	13.2”
N	8,723	8,723	8,723	8,224	8,224	8,224
Ln Cognitive	0.383***	0.394***	0.347***	0.184	0.184	0.140
Test Score	(0.109)	(0.118)	(0.133)	(0.115)	(0.122)	(0.144)
F	39.4	35.5	35.4	14.8	13.8	12.8
N	8,158	8,158	8,158	7,748	7,748	7,748
Ln School	0.855***	0.880***	0.852***			
Exam Score	(0.232)	(0.230)	(0.243)			
F	11.0	10.8	10.4			
N	3,317	3,317	3,317			
Controls by District Interacted with Cohort Dummies:						
Enrollment rate		Yes	Yes		Yes	Yes
W. & S. Program			Yes			Yes

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. F-values marked with ” do not partial survey round dummies out. All 2SLS-regressions are run in STATA 12 using the ivreg29 command (Baum et al. 2011). Movers are excluded. For further details on controls, see the table notes in table 4 and 5. Each estimate is obtained from separate regressions.

Table 20: First and Second Stage Results for Son's, Only Non-Moving Fathers

Second Stage Dependent Var.	First St. Dep. and Second St. Indep. Var.: Paternal Schooling First Stage Independent Variable: Paternal Program Exposure – – **Second Stage** – –			– – **First Stage** – –		
	(1)	(2)	(3)	(4)	(5)	(6)
Height	-0.467	-0.339	0.002	0.363***	0.342***	0.400***
	(0.912)	(0.807)	(0.686)	(0.117)	(0.126)	(0.147)
F				15.2	14.4	12.0
N	5,111	5,111	5,111	5,111	5,111	5,111
Weight	-0.702	-0.741	-0.528	0.326***	0.304**	0.349**
	(0.503)	(0.546)	(0.517)	(0.114)	(0.122)	(0.142)
F				15.0	13.7	11.4
N	5,115	5,115	5,115	5,115	5,115	5,115
BMI	-0.218	-0.240	-0.193	0.352***	0.330***	0.386***
	(0.180)	(0.192)	(0.192)	(0.116)	(0.124)	(0.146)
F				14.3	13.5	11.3
N	5,084	5,084	5,084	5,084	5,084	5,084
Hemoglobin	-0.036	-0.076	-0.061	0.307**	0.293**	0.336**
	(0.146)	(0.154)	(0.161)	(0.126)	(0.135)	(0.156)
F				16.6"	14.6"	11.8"
N	4,911	4,911	4,911	4,911	4,911	4,911
Anemia	-0.055	-0.050	-0.052	0.307**	0.293**	0.336**
	(0.050)	(0.054)	(0.057)	(0.126)	(0.135)	(0.156)
F				16.6"	14.6"	11.8"
N	4,911	4,911	4,911	4,911	4,911	4,911
Length of Schooling	0.001	0.025	-0.101	0.417***	0.432***	0.365**
	(0.147)	(0.140)	(0.202)	(0.135)	(0.151)	(0.185)
F				11.9"	10.3"	9.5"
N	4,151	4,151	4,151	4,151	4,151	4,151
Ln Cognitive Test Score	**0.152****	**0.155****	*0.119*	0.369**	0.371**	0.279
	(0.076)	(0.076)	*(0.097)*	(0.161)	(0.175)	(0.206)
F				12.0	10.5	9.3
N	3,912	3,912	3,912	3,912	3,912	3,912
Controls by District Interacted with Cohort Dummies:						
Enrollment rate		Yes	Yes		Yes	Yes
W. & S. Program			Yes			Yes

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. F-values marked with " do not partial survey round dummies out. All 2SLS-regressions are run in STATA 12 using the ivreg29 command (Baum et al. 2011). Movers are excluded. For further details on controls, see the table notes in table 4 and 5. Each estimate is obtained from separate regressions.

Table 21: IV Results on Potential Mediators, Only Non-Movers

Dependent Variable	Independent Variable: **Parental Length of Schooling** – – **Mother** – – (1)	(2)	(3)	– – **Father** – – (4)	(5)	(6)
Immunization	0.026	0.023	0.032			
	(0.055)	(0.053)	(0.047)			
N	3,587	3,587	3,587			
Iodized Salt	0.018	0.015	0.027	0.038	*0.036*	*0.024*
	(0.037)	(0.033)	(0.043)	(0.077)	(0.082)	(0.081)
N	4,911	4,911	4,911	4,845	4,845	4,845
Household Size	0.013	0.026	-0.095	-0.699	-0.674	-0.798
	(0.262)	(0.263)	(0.268)	(0.488)	(0.502)	(0.502)
N	4,939	4,939	4,939	4,870	4,870	4,870
Schooling of Spouse	**1.120*** **	**1.117*** **	**1.297*** **	**0.662***	**0.672***	**0.572***
	(0.288)	(0.276)	(0.343)	(0.383)	(0.401)	(0.339)
N	4,927	4,927	4,927	4,863	4,863	4,863
Ln pc Exp.	0.034	0.032	0.077	0.055	0.053	0.087
	(0.050)	(0.048)	(0.056)	(0.074)	(0.076)	(0.077)
N	4,912	4,912	4,912	4,844	4,844	4,844
Controls by District Interacted with Cohort Dummies:						
Enrollment rate		Yes	Yes		Yes	Yes
W. & S. Program			Yes			Yes

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. All 2SLS-regressions are run in STATA 12 using the ivreg29 command (Baum et al. 2011). Movers are excluded. For further details on controls, see the table note in table 11. Each estimate is obtained from separate regressions.

Table 22: First Stage Results for *Potential Mediators*, Only Non-Movers

	First Stage Dependent Variable: Parental Length of Schooling First Stage Independent Variable: Parental Program Exposure					
Second Stage	– – **Mother** – –			– – **Father** – –		
Dependent Var.	(1)	(2)	(3)	(4)	(5)	(6)
Immunization	0.294**	0.305**	0.352**	0.114	0.120	0.194
	(0.136)	(0.136)	(0.167)	(0.152)	(0.152)	(0.168)
F	12.0	11.1	10.5	6.5	5.8	5.4
N	3,587	3,587	3,587	3,525	3,525	3,525
Iodized Salt	0.306***	0.304***	0.288**	0.170*	0.161	0.178
	(0.101)	(0.096)	(0.113)	(0.097)	(0.101)	(0.113)
F	35.7	32.0	29.2	16.2	16.1	14.4
N	4,911	4,911	4,911	4,845	4,845	4,845
Household	0.334***	0.333***	0.340***	0.234**	0.224**	0.267**
Size	(0.105)	(0.097)	(0.115)	(0.100)	(0.102)	(0.112)
F	65.0	56.1	53.2	27.8	24.8	21.3
N	4,939	4,939	4,939	4,870	4,870	4,870
Schooling	0.336***	0.334***	0.347***	0.231**	0.222**	0.268**
of Spouse	(0.107)	(0.099)	(0.116)	(0.101)	(0.103)	(0.112)
F	65.2	56.3	53.5	27.8	24.8	21.3
N	4,927	4,927	4,927	4,863	4,863	4,863
Ln pc	0.335***	0.332***	0.341***	0.232**	0.223**	0.268**
Exp.	(0.109)	(0.099)	(0.115)	(0.101)	(0.103)	(0.113)
F	69.0	60.6	56.1	28.4	25.2	21.8
N	4,912	4,912	4,912	4,844	4,844	4,844
Controls by District Interacted with Cohort Dummies:						
Enrollment rate		Yes	Yes		Yes	Yes
W. & S. Program			Yes			Yes

Note: Robust standard errors are clustered at the parental district of birth level and are shown in parentheses: *** $p < 0.01$, ** $p < 0.05$, † $p < 0.075$, * $p < 0.1$. F-values marked with " do not partial survey round dummies out. All 2SLS-regressions are run in STATA 12 using the ivreg29 command (Baum et al. 2011). Movers are excluded. For further details on controls, see the table note in table 11. Each estimate is obtained from separate regressions.