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IZA DP No. 4353

Gradients of the Intergenerational Transmission of Health in Developing Countries

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August 2009

Forschungsinstitut
zur Zukunft der Arbeit
Institute for the Study
of Labor

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ABSTRACT

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This paper investigates the sensitivity of the intergenerational transmission of health to exogenous changes in income, education and public health, changes that are often delivered by economic growth. It uses individual survey data on 2.24 million children born to 600000 mothers during 1970-2000 in 38 developing countries. These data are merged with macroeconomic data by country and birth cohort to create an unprecedentedly large sample of comparable data that exhibit massive variation in maternal and child health as well as in aggregate economic conditions. The country-level panel is exploited to control for aggregate shocks and trends in unobservables within countries, while a panel of children within mother is exploited to control for family-specific endowments and neighbourhood characteristics. Child health is indicated by infant survival and maternal health by (relative) height. We find that improvements in maternal education, income and public health provision that occur in the year of birth and the year before birth limit the degree to which child health is tied to family circumstance. The interaction (gradient) effects are, in general, most marked for shorter women suggesting that children are more likely to bear the penalty exerted by poor maternal health if they are conceived or born in adverse socio-economic conditions.

JEL Classification: O12, I12

Keywords: intergenerational transmission, early life conditions, health, infant mortality, height, growth, income, education, public health, gene, environment, *in utero*

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1. Introduction

The persistence of disadvantage within families has attracted interest across the social sciences. Even in the USA, the land of opportunity, the incomes of sons exhibit a strong correlation, estimated at 0.4, with the incomes of fathers (Solon 1992, Corak 2006). Some of this correlation can plausibly be put down to the heritability of income-enhancing traits such as ability or patience, but some of it will reflect differences in human capital investment between families. What is of interest for public policy is the extent to which these differences can be narrowed, so that children from unequal families start life with more equal opportunities.

We assemble a dataset of unprecedented scope in this literature, with comparable data on 2.24 million children born to 600,000 mothers in 38 developing countries in the period 1970-2000. Focusing upon health human capital, we investigate the extent to which changes in economic growth, maternal education and public health services (indicated by immunization rates) that occur around the birth of the child weaken the intergenerational transmission of health. It is of particular interest for the purposes of public policy to consider the further question of whether any weakening is asymmetric, being greater for children who start life with a weaker health endowment. We therefore allow the influence of the early life environment on child health to depend upon the mother's (relative) health. Although evidence in support of gene-environment interactions is accumulating (Ridley 2003, Cunha and Heckman 2007), few empirical studies explicitly allow for these interactions; exceptions include Björklund, Lindahl and Plug (2005), Currie and Moretti (2007), van den Berg et al. (2008).

As we are able to identify siblings, we also estimate specifications that include mother fixed effects. These sweep out the additive effects of not only genetic frailty but also non-genetic endowments shared by siblings, and permanent income. This is an improvement upon previous studies that, using data on sisters or twin sisters of the *mother* or else on adopted children, can only purge genetic endowments passed on by the mother (Behrman and Rosenzweig 2002, Plug 2004, Royer 2005, Currie and Moretti 2007).[1] The reason that previous studies have not exploited data on siblings or twin *children* is no doubt that this involves throwing away the baby with the bathwater. In other words, the indicator of mother's education or health is typically a time-invariant mother-specific variable (like years of schooling or birth weight) that would be absorbed by the mother fixed effects. This is the case in our specification as well. The difference is that the parameter of interest here is not the intergenerational correlation itself but its gradient, and this is still identified.

This is the first study to nest micro in macro data to study country-cohort variation in intergenerational persistence. We exploit the panel of countries over time to purge country-specific unobservables and aggregate shocks. We also include country-specific trends, so that economic growth, public services and maternal education are effectively measured as within country deviations from trend, or shocks. Identification of the gradient rests upon children (and, in the mother fixed effects models, siblings) being conceived or born under different conditions. This is assisted by the massive variation in our sample. Income volatility in developing countries is endemic, as indicated by the standard deviation of annual growth rates, which is much higher than in OECD member countries (Pritchett 2000, Malik and Temple 2009). There are corresponding sharp fluctuations in public health provision. While public spending in richer countries tends to

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[1] In section 2 we discuss the limitations of previous fixed effects -and instrumental variables- approaches in more detail.

rise in bad times (Lane 2003, Cutler et al. 2007), in poorer countries it tends to decline when national income falls (Woo 2005, Paxson and Schady 2005). In addition, war and natural disasters often directly destroy health infrastructure. While maternal education cannot fluctuate over time for a given cohort of women, the average education of mothers in a birth cohort does because of variation in the composition of women giving birth. This creates exogenous (aggregate) variation in mother's education that we exploit to identify its effects.

Although identification that rests upon shocks is cleaner, short run elasticities will tend to differ from longer run elasticities. For example, short run income shocks will include substitution effects that may even out over a longer run that allows for institutional change (e.g. Ruhm 2000). We therefore also explore an alternative specification that tests the hypothesis that the decline in the intergenerational health coefficient between cohorts separated by two decades is faster in countries that recorded positive growth than in countries that experienced stagnation or negative growth. This "experiment" is possible on our data because of the diversity of positive and negative growth experiences in developing countries across the last three decades. Also, while immunization rates exhibit a strong trend on average, the trend varies considerably between countries and cohorts. These stylized facts enhance the weight of the questions we pose.

Health is a multidimensional latent variable that is notoriously difficult to measure; see, for example, Ahlburg (1998), Currie (2007). Our indicator for mother's health is height measured relative to a country-cohort mean. Height is an index of permanent health which reflects the cumulative impact of health shocks from conception to adulthood (Martorell and Habicht 1986, Cole 2000, Strauss and Thomas 2008). While height is clearly not equivalent to health, if the children of shorter women are systematically more likely to die in early childhood (across several countries), as we find

(see Figures 1 and 2), then it would seem that height does proxy health. Our measure of child health is infant survival, the most widely used measure of population health in developing countries (Cutler et al. 2006). Infant mortality rates proxy the health of the entire cohort, that is, of survivors and not only of succumbents. For instance, van den Berg et al. (2006) show that individuals who survive negative income shocks in infancy die earlier in their adult lives. Further discussion of our health indicators is in section 4.

The main findings are summarised here. There is considerable and widespread intergenerational persistence in health. A one standard deviation (7.13 cm) gain in height is associated with close to a 0.66%-point reduction in infant mortality risk, which is about 6.8% of the average mortality rate in the sample. The penalty associated with poor maternal health is greater than the gain associated with good maternal health, and the penalty is increasing in the deviation of maternal health from its mean.

Improvements in mother's education, economic growth and public health timed in the year of birth of the child and in the year before birth attenuate intergenerational persistence. Overall, the estimated effects for the year of birth and the year *in utero* are similar. Here we summarise the magnitude of the effects for the year of birth. An extra year of education amongst mothers of children in the country and cohort of the index child lowers the intergenerational correlation of health by 17%. Attenuation of the correlation associated with one standard deviation increases in log p.c. GDP and immunization rates respectively is about 29% and 18.5% respectively. The interaction coefficients are enlarged rather than eliminated upon controlling for mother level unobserved heterogeneity. The gains from improvements in income and maternal education are greatest for children who are initially most disadvantaged by being born of relatively unhealthy mothers. This defends the increasing prevalence of the view that government policies to promote early accumulation of human capital should be targeted to the children of disadvantaged families; see Cunha and Heckman 2007.

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We studied changes in the intergenerational correlation over a twenty year period which, we expect, encompass the effects of changes in income, education, public services and other secular change. We find that intergenerational persistence in health declines more rapidly in countries with positive growth. A plot of the country-specific intergenerational correlation coefficient against average GDP over the entire period shows a negative relationship.

Our results provide new evidence to support interventions that promote public health and women's education. As infant mortality in poor countries is driven by infectious disease, public health policies such as improving immunization coverage have a public goods element. Policies that improve education and, especially female education, similarly have a public goods component; see Basu and Foster (1998), who argue that that the literacy of an individual creates externalities for members of her household. Their logic is easily extended to the community level. Our findings also suggest that income improvements in very poor countries are beneficial for health[2] and, by highlighting the irreversible damage caused by income volatility, provide a motivation for economic stabilization policies and for social protection in times of crisis. For example, micro-credit (Armendariz and Morduch 2004) and cash transfers (Fiszbeing and Schady 2009) are now widespread in anti-poverty programmes and they could be timed to cushion health against shocks. Further discussion of the policy implications of our analysis is in section 6.2.4.

1.1. Relation to Previous Literature

A recent literature on the consequences of child health for adult socio-economic status (SES) motivates our analysis, in which child health is the outcome. Using US data, Currie and Madrian 1999, Currie and Hyson 1999, Case et al. 2005, Oreopolous 2006,

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[2] For a wide-ranging discussion that leaves open the question of how much income matters, especially as compared with education and public health is in Cutler et al. 2006. For a discussion focused on current developing countries, see Bhalotra 2009a.

Royer 2009, Smith 2009, for example, show that childhood health predicts education and earnings in adulthood. To some extent, this may be because childhood health predicts adult health (e.g., Barker 1997, Almond 2007, Smith 2009) and adult health is correlated with adult SES (e.g. Smith 1999). Adult SES, in turn, appears to influence child health, e.g., Currie and Hyson 1999, Case et al. 2002, Currie, Price, and Shields 2007. Linking these studies, it is clear that child health may be an important conduit for the intergenerational transmission of health. In this case, "health dynasties" may be an important if neglected aspect of persistent inequality in living standards (see Case et al. 2005 and Currie 2007).

The wider literature has focused upon education as the element of human capital that drives earnings; see the reviews by Ahlburg (1998), Grawe and Mulligan (2002). A couple of recent studies underline the relevance of the intergenerational correlation of health. Currie and Moretti (2007) demonstrate a positive association of maternal and child birth weight in California and further show, on the same data, that birth weight predicts future socio-economic status (SES). Eriksson et al. (2005) show that conditioning upon health status lowers estimates of the intergenerational earnings elasticity in Denmark by a quarter.

Numerous studies have investigated the effects of either family socio-economic status (SES) *or* maternal health on child health, rather than an *interaction* of the two, which is the focus here. The only previous studies that directly investigate the gradient of the intergenerational correlation of health are Conley and Bennett (2001) and Currie and Moretti (2007). Both specify models of the intergenerational correlation of birth weight in which mother's birth weight is interacted with a measure of family SES. In the first study this is family income (a likely issue with which is that it will tend to be correlated with mother's birth weight) and in the second it is mother's education or local poverty in the birth year of, alternatively, the child and the mother. There is a further related study.

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Almond and Chay (2006) analyse changes in the black-white differential in a set of health outcomes for women born in the Civil Rights Era, and their children. An interaction between maternal health and public services is implicit in that the Civil Rights Era was associated with improved access for black women. These studies find that intergenerational health transmission is weaker amongst more educated and less poor mothers (Conley-Bennett, Currie-Moretti), and that it is weakened by the more even spread of public services (Almond-Chay). They also show that these effects are strongest amongst less healthy women, indicated in the first study as low birth weight and in the second as black. A recent paper using data from pre-industrial Denmark is related in that it shows that the long run health (susceptibility to cardio-vascular disease) of an individual is more strongly determined by genetic and household-level environmental factors if early-life conditions are poor.

In the wider domain of studies concerned with direct or additive effects, not many studies are able to identify causal effects as distinct from correlations of family income, mother's education or mother's health with child health that arise on account of unobservables. For a survey of studies set in richer countries, see Currie (2007). We address this problem here by exploiting shocks at the country-cohort level while also controlling for mother-level unobserved heterogeneity. Amongst studies set in poorer countries (including those in nineteenth century Europe), a few previous studies, like us, use the state of the business cycle to proxy exogenous changes in income; see van den Berg 2006, Baird et al. 2007, Bhalotra 2009.[3] We know of no previous work that uses variation in the composition of women giving birth to create exogenous variation in education, or that exploits annual country-cohort variation in an indicator of public health provision. The literature attempting to estimate causal effects of maternal on child

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[3] Like them and other previous work, we use the terms recession/downturn, boom/upturn to refer to annual deviations of GDP from trend.

health is small; in section 2.4 we consider why. A discussion of the relationship between the results in this paper and the (tangentially related) literature on the persistent health effects of *in utero* conditions is in section 6.2.4 below.

Evidence of intergenerational persistence for developing countries is particularly scarce (Solon 2002), and there are no studies of gradients, whether for health, education or earnings. Yet it is in poorer countries that family background effects on poor health are most likely to persist given weak health infrastructure, that health is most likely to be tied to low wealth, and that the consequences of poor health and low wealth are most severe. In extensions of the original theoretical models of mobility (Becker and Tomes 1979, 1986), Grawe (2001) incorporates credit constraints and Solon (2004) incorporates progressivity of public expenditure. These papers suggest a role for economic growth and public services in determining the intergenerational transmission of human capital but there is no previous systematic empirical analysis of the role of either. Growth and development economists are increasingly interested in poverty traps but evidence of underlying mechanisms and, especially, of the role of health transmission is scarce (e.g. Steckel 1986, Dasgupta and Ray 1997, Schultz 2005).

The rest of this paper unfolds as follows. Section 2 sets out the methodology and section 3 details potential mechanisms. Section 4 describes the micro- and macro-data and the indicators of health. Descriptive statistics and non-parametric estimates are in section 5. Results are in section 6 and section 7 concludes.

2. Methodology

This section sets out the estimated specifications and the rationale behind them.

2.1. The baseline model

This is the "levels" model that estimates the intergenerational correlation of health :

$$C_{imjt} = \alpha + \beta H_{mjt} + X'_{imjt}\lambda + \gamma_{jt} + \varepsilon_{imjt} \qquad (1)$$

The dependent variable (C) is a binary variable indicating whether child i born to mother m in country j in year t died by the age of 12 months (infancy). To allow for (observed) age-heaping at the twelfth month, we define infant mortality to include the twelfth month. To ensure that every child in the sample had full exposure to infant mortality risk, children born less than 12 months before the date of interview (which is specific to each of the 77 surveys) are excluded. γ_{jt} comprehensively capture country-year unobservables including shocks associated with, for example, oil prices, war, famine or medical technological progress and potential confounders such as income, and the spread of public services. The regressor of interest is H, mother's height. We expect $\beta<0$ since our dependent variable is inverse to child health. β reflects the average effect on child health of within-country variation in maternal height, holding constant (observed) characteristics of the mother and child (X). These include indicators for the gender, birth order and birth month of the child, maternal age at index birth, the years of education of the mother and father, urban *vs* rural residence and religion.[4] Education and age at birth (lifecycle) control for family SES.

The country-year fixed effects absorb country-specific mean height, taking care of the fact that being 1.6m tall in Kenya (where women are tall) may not confer the advantage it does in Nepal (where women are short). In our initial specification, we use height in metres. We estimate an alternative specification that allows for non-linear effects, consistent with an upper bound to survival chances and diminishing returns in the health production function. For this, we define six dummy variables, indicating whether the mother is half, one, or two standard deviations above or below the mean height in her country.[5] This specification naturally allows asymmetric effects.

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[4] Although predetermined, maternal age, birth month and birth order are not strictly exogenous to child health. We confirm that the results are not sensitive to excluding them.
[5] Our results were not sensitive to replacing the mean with the median.

The estimator for equation (1) is probit and, given the long time dimension to the country panel, (country) fixed effects estimates are expected to be consistent. Reported standard errors are robust to arbitrary forms of heteroskedasticity and clustered by country to allow for autocorrelation within country (Bertrand et al. 2004). Since mothers are plausibly assumed not to migrate across countries between births this naturally also allows for sibling correlations.

2.2. Interactions with early life conditions

We extend the baseline model by adding interaction terms that provide estimates of the gradient of the intergenerational correlation of health-

$$C_{imjt} = \tilde{\alpha} + \tilde{\beta} H_{mjt} + X'_{imjt} \tilde{\lambda} + \tilde{\delta} y_{jt} + \tilde{\tau}(H_{mjt} \cdot y_{jt}) + \theta_j + \theta_t + \zeta_j t + \tilde{\varepsilon}_{imjt} \quad (2)$$

y_{jt} refers, in turn, to the cohort-average of mother's education, GDP and immunization rates in the child's birth year. In an alternative specification, we replace these variables with their first lag, y_{jt-1}, which represents conditions *in utero*. These variables are all effectively measured as (plausibly exogenous) within-country deviations or shocks. To accommodate country-cohort varying y_{jt} in the model, the country-year fixed effects γ_{jt}, are replaced with country and year fixed effects and country-specific trends. To avoid clutter this is not displayed but we also explore interactions with individual family SES measures, y_{mjt}. The gradients that we are after are denoted τ. We expect $\beta<0$, $\delta<0$ and $\tau>0$ for reasons set out in section 3.

These models are estimated using the linear probability model as this gave us similar results in the baseline model and the coefficients are easier to interpret when the model includes interactions (Ai and Norton, 2003). Also, once we replace country fixed effects with mother fixed effects (equation 3), the relevant panel is of births within mother. As this is short, the probit estimator is not consistent. Equation (2) and indeed, all equations displayed henceforth, are re-estimated replacing height in metres with the

dummies for standardised height that allow for nonlinearity and asymmetry in the intergenerational correlation. This allows us to test the hypothesis that improvements in the health environment around birth are more effective in lowering intergenerational persistence for children who start life with a relative disadvantage.

2.3. Mother fixed effects estimates

We have sibling linked data with a mean of five siblings per mother which can be exploited to control for endowments, fixed local area characteristics and possibly endogenous selectivity of the sample of births (see section 3), using mother fixed effects (α_m). The fixed effects absorb mother's height, which is time-invariant (i.e. the same across siblings), so we cannot identify β in equation (2). However, the coefficient τ on the *interaction* term is identified:

$$C_{imjt} = \ddot{\alpha}_m + X'_{imjt}\ddot{\lambda} + \ddot{\delta}y_{jt} + \ddot{\tau}(H_{mjt} \cdot y_{jt}) + \theta_j + \theta_t + \zeta_j t + \ddot{\varepsilon}_{imjt} \qquad (3)$$

This model is estimated using the 95% of women in the sample who have at least two births (and the 96% of children who have at least one sibling). The parameter δ provides an estimate of the direct influence of the external health environment on child health obtained by comparing siblings born under different economic conditions (as in Bhalotra 2009a). The parameter τ now indicates whether this effect is stronger (or weaker) amongst shorter women given their endowments. These include not only genetically transmitted frailty but any other fixed traits of the mother or her environment that are common across siblings, for example, the permanent health of the mother (and father if he is common across siblings), permanent income, local climate, the mother's efficiency in producing child health, her tastes for health, and physiological characteristics such as pelvic size that may constrain a small woman to produce a small child. The permanent health of the mother will include any aspect of frailty that is acquired over her lifetime – for example by exposure to adverse environmental conditions- and that may be passed

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on to her offspring by biological mechanisms that may not involve genes (e.g. Barker 1998). The mother fixed effects also control for heterogeneity in fertility responses and in foetal mortality across time. As explained in section 3, this will generate compositional effects that, if not purged, will tend to confound the causal effects.

2.4. Digression on grandmother fixed effects & instrumental variables approaches

Recent studies of the intergenerational correlation of education have taken one of two approaches to isolating behavioural effects of parental schooling from endowment effects. They either (a) exploit data on twin/sibling mothers (grandmother fixed effects) or on adoptee children to purge the influence of persistent genes, or (b) use instrumental variables. Both of these approaches only achieve their objective under the strong assumption that behavioural and endowment effects appear additively in the outcome equation, but there is now considerable evidence that they interact (Turkheimer et al. 2003, Ridley 2003, Moffitt et al. 2005, Currie and Moretti 2007, Cunha and Heckman 2007, van den Berg et al. 2008).

A further limitation specific to the studies that use grandmother fixed effects (e.g. Behrman and Rosenzweig 2002) is that while they do remove the mother's biological endowments, the father's endowments remain. This problem is resolved by using data on adoptee children (Sacerdote 2002, Plug 2004). However, even with this approach, the potentially confounding influence of non-biological endowments remains (see Plug 2004). For instance, adoptee mothers schooling may be correlated with their child rearing abilities or tastes for education. Or, if mothering ability and tastes for human capital are acquired by mothers as a function of their environment then it is plausible that this sort of ability is different amongst twin mothers and so not differenced out. As explained above, our approach addresses *both* of these problems: an interaction between an index of maternal health and shocks to the health environment is explicitly modelled and, in an interactions model, we are able to difference across siblings rather than across mothers.

The other approach in the antecedent literature involves using school reforms or exogenously determined school expansion to instrument mother's school years (Chevalier 2003, Currie and Moretti 2003, Black et al. 2005). A natural analogue in our context (if one is willing to assume that endowments and investments have separable effects) is to consider as potential instruments for maternal height events like famines or epidemics in the mother's childhood. But while such events are external in that they occur independently of mother's height they are not exogenous in that they may influence child health conditional upon mother's height (Heckman 2000, Deaton 2009). The problem is a reflection of the impossibility of finding a comprehensive index of (mother's) health.[6]

2.5. Longer run economic development

In the specifications set out so far, the longer run effects of economic development are not identified as they are absorbed by time dummies and country-trends. To gain an indication of whether the intergenerational correlation is diminishing in economic growth, we exploit the enormous diversity of growth experiences across the 38 countries and 31 birth cohorts in our sample (see section 5.1) as follows. We estimate the baseline model for birth cohorts 1970-75 and 1990-95, separately for countries with positive growth of at least 1.5% p.a., countries with negative growth of at least 1.5%, and countries with insignificant growth, where growth is the average linear rate over the entire period. Our hypothesis is that the intergenerational correlation will have declined faster in countries with positive economic growth than in other countries.

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[6] The difference with education is not that years of schooling are a perfect proxy for cognition (they are not: e.g. Behrman et al. 2009) but rather that the instrument is narrowly tailored to influence schooling and nothing else, so that the exclusion restriction holds. In contrast, it cannot be argued that mother's height fully captures the influence of her childhood health environment. Indeed, we find, on our sample, that events in the mother's childhood impact on child survival conditional upon mother's height. Bhalotra (2009b) studies the implied reduced form.

3. Mechanisms

This section sets out mechanisms that may drive the influence on child health of each, parental health and characteristics of the early life environment, and their potential interaction.

3.1. The intergenerational correlation of health

Poor parental health may lead to lower investments in child health by creating any of *income, time/energy or biological constraints*. Although the tendency for poor adult health to lower earnings is well established, there is relatively little discussion in the literature of the latter two mechanisms. Consider the evidence for each. Poor health tends to lower *earnings* (e.g. Smith 1999) and, if there are diminishing productivity returns to health, this tendency is stronger at the lower levels of health evident in poorer countries (Steckel 1986, Fogel 1994, Schultz 1997, Dasgupta and Ray 1997). So parents in poor health are more likely to be credit constrained and this may impact child health *via* any of maternal nutrition and stress, antenatal care, disease exposure, delivery conditions, or postnatal investments.[7,8]

Second, poor maternal health may limit maternal *time* inputs to child health or lower the productivity of other inputs. For instance, poor health may make mothers less responsive to symptoms of infection or less able to breastfeed. Poor health may cause time constraints to bind if it challenges energies available for routine tasks (e.g. Basu 1997, Ray 1998), including accessing public services relevant to child health. Mothers, who are the primary caregivers, may have to travel long distances to access a health centre or make several visits because of staff absenteeism; see Chaudhury et al. 2006. A recent literature establishes that business cycle variation in mother's disposable time

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[7] The 1995 World Health Report (WHO, 1995) states that extreme poverty is the world's most ruthless killer and the greatest cause of suffering on earth, listed in the International Classification of Diseases, an A to Z of all ailments known to medical science, as code Z59.5; Gordon 2004.
[8] Causal effects of income on child health in developing countries are identified, for example, in Duflo 2000, Hoddinott and Kinsey 2001, Bhalotra 2009a.

influences health seeking behaviours and infant mortality (Dehejia and Lleras-Muney 2005, Miller and Urdinola 2007, Bhalotra 2009a).

Third, less healthy mothers tend to give birth to less healthy children; Kramer 1987. This is partly but not only because of genetic transmission of *frailty*. Recent evidence suggests that maternal health is a bundle not only of genes and the influences of contemporary environment but that it also carries *shadows* of the environment the mother was exposed to in her childhood (e.g. Barker 1997, 1998) and, in many developing countries, boys are prioritised over girls in the allocation of scarce nutritional resources (e.g. Behrman and Deolalikar 1989, Rose 1999). The fetal origins literature suggests that an adverse nutritional/health environment in early childhood can result in biological constraints upon the health of the next generation. Mothers growing up in adversity may be less able, for example, to nourish their births *in utero* (Osmani and Sen 2003, Drake and Walker 2004, Gluckman and Hanson 2005).

3.2. The influence of the early life environment on health

We analyse three indicators of the health environment at birth: maternal education, immunization rates and aggregate income. Consider the mechanisms associated with each. Maternal education is expected to capture both permanent income and "technical efficiency" (or information) in combining inputs to produce child health (Currie and Moretti 2003, Cutler and Lleras-Muney 2006). Child morbidity and mortality in developing countries are predominantly associated with infectious disease, and immunization will tend to improve child health at any given level of maternal health (Unicef 2009). A recent literature demonstrates variation of (child) mortality across the business cycle: see Ruhm 2000, Dehejia and Lleras-Muney 2004, van den Berg et al. 2006, Baird et al. 2007, Bhalotra 2007b, Ferreira and Schady 2008. The state of the business cycle at birth may influence child health through any of three channels. These are changes in (a) the level of public spending and hence the supply of health services, (b)

household income and therefore the demand for purchased inputs, and (c) the opportunity cost of mothers time, and so in maternal inputs to child health. Each of these channels is demonstrated in Bhalotra 2009a. Business cycle variation may further induce changes in the composition of mothers giving birth and hence in average infant survival chances. Consider the evidence on each of these channels.

Public spending tends to be pro-cyclical in developing countries (Woo 2005, Bhalotra 2007a) and there are several accounts of public services collapsing during economic crises (Lustig 1999, Frankenberg et al. 2003, Paxson and Schady 2005). Recessions are typically associated with a rise in the fraction of credit constrained households (Agenor et al. 2000, Koren and Tenreyo 2007). Women's labour supply is sensitive to the business cycle. While in richer countries, the average woman reduces her participation during recessions as wages sink below reservation levels, there is a tendency in developing countries for (informal) work to increase during recessions, presumably to maintain consumption (Bhalotra and Umana-Aponte 2009). Endogeneity in sample composition may arise because of mother-level heterogeneity in fertility timing (Dehejia and Lleras-Muney 2004) or foetal death (Bhalotra 2009a). *Overall*, child health may be worse in recessions because *public services are weaker, households are poorer and mothers are busier.* These causal effects may be hard to recover if there is an opposing tendency for the composition of births in a recession to be selectively low risk. We use mother fixed effects to control for endogenous selection into the sample.

It is plausible that early life conditions have larger effects for children born to mothers in relatively poor health as these mothers are more likely to be constrained by money, time and energy in the investments that they can make in their children. Interactions may be critical to the case of maternal health imposing biological constraints on child health. Negative shocks that strike when the mother is *in utero* induce endocrine

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or physiological adaptations that are "thrifty" at the time but that tend to create problems later in life, especially if environmental conditions change (Barker 1998).

4. Data

This section describes the four main data sources and the way in which they are merged. It also discusses our choice of indicators of health for the mother and child. Many previous studies focus on narrow measures like chronic conditions that either have low prevalence or relatively small effects (Currie 2007, p.4).

4.1. Microdata

The microdata are compiled from 77 Demographic and Health Surveys (DHS) for 38 developing countries; see Appendix Tables 1 and 2. Women aged 15 to 49 years at the time of the survey record their complete fertility histories so that we have information on the birth of every child of the sampled women, and any deaths. This means that we naturally have linked siblings. Although interview dates span 1986-2006, births occur across 1952-2006. We drop births before 1970 because the data are thinner and less representative there. We drop mothers younger than 20 years to allow for continued height growth to that age, and to limit selectivity in height for the surveys that interview only married women. These and other data issues are discussed in more detail in Bhalotra and Rawlings (2009). The DHS data contain relevant information on education and demographics. They do not contain information on household income or consumption. A great advantage of the data is their breadth. The sample analysed has *comparable* information on about 2.24 million children born to 600,000 mothers during 1970-2000.

4.2. Measure of child health

We use infant mortality or death in the first year of life, which is the most widely used indicator of child and, indeed, population health in developing countries; Jones et al. 2003. While in richer countries early childhood deaths account for 1% of all deaths, in

developing countries, they account for 30%, as a result of which infant mortality rates are strongly correlated with life expectancy; Cutler et al. 2006. The proximate cause of infant mortality is infectious disease combined with poor nutrition (of the mother as much as the child); e.g. Black et al. 2003. For this reason, infant mortality rates are often used to proxy the broader health environment (e.g. Deaton 2007). Survivors of cohorts that experience high rates of infant mortality tend to be scarred for life, experiencing higher adult morbidity and earlier mortality (Elo and Preston 1992, van der Berg et al. 2006). Overall, infant mortality rates do not just capture health at the tail of a distribution but, rather, are indicative of a wider health environment that affects the entire population.

Mortality rates are available from the fertility histories in the DHS for all births which, as noted earlier, constitute three decades of birth cohorts. Previous studies of the intergenerational correlation of health (mostly set in the USA) tend to use birth weight; see Royer 2005, Currie and Moretti 2007 and citations therein. Birth weight is recorded in the DHS data for children born up to five years before the date of the survey, so potentially for 14.9% of all births. However, measured birth weight (in grams) is missing for about 63% of this (five year) sample.[9]

4.3. Measure of mother's health

Our measure of maternal health is height. Height has been shown to predict life expectancy (Galton 1886, Waaler 1984). It is thought to be a particularly good signal of health at lower standards of living where nutrition is relatively scarce and the extortions of infectious disease are greater (Costa 1998, Fogel 1994, Steckel 1995). Adult height is produced as a combination of genetic and environmental influences, both of which can be transmitted to children. Cohort differences in adult height have been used to proxy trends and fluctuations in net nutrition in historical data (Steckel 1995) and a recent study

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[9] In a companion paper that focuses upon the levels (baseline) model, we estimate the intergenerational correlation of health using subjective reports (provided by the mother) of birth weight. We also consider child height and neonatal and under-5 mortality; see Bhalotra and Rawlings 2009.

set in Guatemala uses height, like us, to proxy mother's biological human capital (Behrman et al. 2009). Two features of adult height are relevant. First, once fully attained, it does not change. This has the implication that although mother's heights are measured at the time of the survey while her births occur across retrospective time, we have an indicator of her permanent health for all of her births. Second, height is a stock that reflects the cumulative impact of net nutrition over the entire growth period, with early childhood being especially significant (Schmidt et al. 1995, Cole 2000, Bozzoli et al. 2009). Indeed, adult stature is correlated with birth weight (Emmanuel et al. 1992, Costa 1998), which is what previous studies have used to indicate maternal health (e.g. Currie and Moretti 2007). Height in the DHS is measured by trained surveyors who use similar measurement devices across the 77 surveys that we exploit. This is relevant since measures of self-reported height have been shown to bias downwards estimates of the heritability of height (Macgregor et al, 2006). For details of how we impute and check missing data and remove outliers, see Bhalotra and Rawlings (2009).

Like other studies of the intergenerational correlation of health, we focus on mothers. This may be justified by paternity uncertainty or by the greater significance of maternal health in relation to fetal growth, breastfeeding and early childhood care. Fertility histories recorded in the DHS provide a clear link between mothers and children. Fathers are only identified as (changeable) partners of mothers and, with a few exceptions, the DHS surveys do not record father's height. Our estimates condition upon the father's education, which contributes to controlling for SES. Previous studies tend to ignore fathers altogether although, as argued in Currie and Moretti (2007), under assortative mating, missing information on the father's health will not add too much noise.

4.4. Country-level panel data

We merge macro-data with the DHS microdata by country and birth year of the child. The years of education of mothers in a country-cohort are obtained from the DHS by taking averages using sample weights. Data on GDP per capita in constant 2000 prices (chain series; henceforth *GDP*) for 1970-2000 is obtained from the Penn World Tables (Summers, Heston and Aten, 2006). There are no publicly available data on health expenditure or total social spending for the full range of our sample. Instead we use immunization rates for measles and DPT (diphtheria and tetanus toxoid with pertussis) which are available for 1985-2000 from the World Development Indicators for all countries in our sample other than Namibia. These data measure the percentage of children aged 12-23 months who have been vaccinated. In the infectious disease environment that characterises developing countries, immunization is a significant input to child survival and in contrast to other health inputs like nutrition or hygiene it is publicly provided. Indeed, immunization rates are regarded as indicating the quality of the health care system across countries (UNICEF, 2009). These data are probably more useful than data on health expenditure given the generally weak record of its effectiveness; e.g. Filmer and Pritchett 1999, though see Bhalotra 2007a. We also investigated data on sanitation, income inequality and poverty. However, these data are too scarce for most countries for the range of years in our sample.[10]

5. Descriptive Statistics

This section presents descriptive statistics that demonstrate poor levels of health and substantial dispersion within and across countries. It then presents a compelling non-parametric description of the relationship of interest.

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[10] The WDI data with immunization rates are at http://go.worldbank.org/ 6HAYAHG8H0. Inequality data are at www.wider.unu and poverty data at PovcalNet, http://go.worldbank.org/NT2A1XUWP0. Oliver Morrissey kindly sent us the sanitation data used in Morrissey et. al. 2005.

5.1. The key variables

Across the 38 countries and the 31 years in the sample, the mean rate of infant mortality is 9.8%.[11] This fell from 15.0% in 1970 to 6.9% in 2000, at a linear rate of 0.2% a year. The mean height of mothers is 1.554 metres (standard deviation 0.068m). The average mother in our sample has 4.074 years of education. Infant mortality and mother's height range from 3.4% in Colombia to 16.3% in Mali and 1.505 in Peru to 1.627 in Senegal, respectively. Mother's education varies from 0.601 years in Niger to 7.524 years in Peru. Every country shows a decline in infant mortality while height trends are varied but unremarkable. The average education of mothers rises from 1.972 years for the 1970 birth cohort to 3.829 years for the 2000 birth cohort. Immunization rates show an impressive trend, rising from just under 30% in 1985 to almost 70% in 2000. Average immunization rates over the period vary, for DPT, from 15.2% in Chad to 80.5% in Kenya and, for measles, from 22.8% in Chad to 79.2% in Zimbabwe. Differences across countries showed some tendency to narrow over the period. On average across countries GDP trends upwards until 1975, after which there is no secular trend and many instances of negative growth during the 1980s. Annual GDP growth varies from -2.98% in Nicaragua to 3.13% in Egypt. Of the 38 in the sample, 9 countries experience significant negative growth, 14 experience significant positive growth, and 15 experience insignificantly small growth over the period (Table 2, Appendix B). Our econometric model will exploit this dramatic country and cohort variation.

5.2. Non-parametric estimates of the intergenerational correlation

There is raw evidence of a positive intergenerational correlation in health in that shorter women have children who are less likely to survive. Since infant mortality is recorded as a discrete variable (0/1), we obtained a continuous prediction of individual

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[11] All descriptive statistics are obtained using sample weights provided in the surveys to make the data representative of the country population.

risk from a non-parametric (lowess) regression and plotted this against mother's height. Infant mortality risk declines in maternal height until about the mean height in the sample, after which it levels out (Figures 1, 2). The graphs exhibit two striking features. First, there is evidence of asymmetry whereby maternal height and child survival are more closely tied for shorter than for taller women. Second, there is greater cross-country variation in health amongst children born to shorter women. Since socio-economic conditions clearly vary across countries but there is no *a priori* reason for endowment effects to vary systematically across countries, this is suggestive of larger SES effects on the gradient for shorter women. The following section investigates this more carefully, conditioning upon level effects of SES (broadly defined to indicate the health environment) and on country-year unobservables.

6. Results

6.1. Baseline Estimates

Maternal stature has a substantial negative influence on infant mortality (Table 1). A one standard deviation (7.13 cm) gain in height is associated with close to a 0.66%-point reduction in infant mortality risk, which is about 6.8% of the average mortality rate in the sample. If we separate boys and girls we find that the intergenerational correlation of health is slightly larger (by 1 s.d.) for boys but that the difference is not significant. Conditional upon country-year effects, the coefficient on mother's height is -0.111. It is 32% smaller once we condition upon all available indicators of family SES including education and demographics. This provides an indication of the extent to which persistence is correlated with SES. Below we investigate the more pertinent question of an SES *gradient* to the correlation. This will indicate the extent to which improvements in socio-economic conditions can diminish persistence.

Asymmetry and nonlinearity in the levels effect

We replaced height in metres with six dummies indicating positive and negative standard deviations of height relative to the country-mean (Table 2). This reveals that (a) the intergenerational correlation of health is strongest at the tails of the height distribution and (b) the disadvantage associated with having a short mother is much greater than the advantage of having a tall mother. These results are consistent with constraints binding for women in poor health; see section 3 above. Below we further identify asymmetry and nonlinearity in the interaction effects.

6.2. Gradients of the Intergenerational Correlation

In this section we report estimates of equation (2) in which maternal height is interacted with indicators of the health or "SES" environment around the birth of the child. These estimates are presented with and without conditioning upon mother fixed effects. For the country-cohort measures, they are also represented for conditions in the child's birth year (y_{jt}) and in the foetal period (y_{jt-1}).

6.2.1.   Interactions with Early Life Conditions

We first describe estimates of effects of birth year conditions on the intergenerational correlation. We then specify, in separate sub-sections, how the estimates change with mother effects, and when conditions *in utero* are modelled.

Socioeconomic status of the individual mother

This is measured using, one at a time, her years of education, an indicator for whether she has at least secondary education and lives in an urban location, and the years of education of her partner. To the extent that the mother's education does not change once she initiates fertility, her education (recorded at the time of the survey) will reflect her SES at the birth of the child. However, her partnership and location may have changed since the index birth. We therefore focus on interactions with maternal education, casting the other two specifications as providing indicative checks.

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Results are in Table 3. The interaction terms are all positive, upholding the hypothesis that the intergenerational correlation of health is attenuated in educated and urban families. An increase of 1 year in mother's education reduces the average correlation by 0.005, which is 5.4% of the total effect. If the mother has at least secondary education and is urban (12.8% of women), the correlation is reduced by 0.046, which is 47.9% of the total effect. The attenuation associated with father's education is insignificantly small.

Since the education and height of mothers is positively correlated, interactions of her height with her education may simply reflect non-linearity in the effect of her height. However, we continue to find positively-signed interactions when we allow for this nonlinearity; see Table 1, Appendix A. The results are well-determined, suggesting there is sufficient variation in education within height group, even if the two are correlated. The nonlinear specification is revealing. It shows that the average interaction effects rely almost entirely upon interaction effects below the mean. For women between 1 and 2 standard deviations (*henceforth s.d.*) below mean height, an additional year of education reduces the intergenerational correlation by 9.1% of the estimated total effect, while being urban and educated to at least the secondary level completely nullifies the infant mortality risk associated with being short.

Average education of mothers

Since short women are on average less educated, presumably because deficits in health and education are both determined by a disadvantaged family background, a possible concern with the previous specification is that short women who are highly educated are selected on unobservables (e.g. high ability). We therefore consider now how the intergenerational correlation would change if the *average* (short) woman had an

additional year of education.[12] We find that it would fall by 16.8% (Table 4). There is, again, asymmetry and also non-linearity in the interactions effects (Table 2, Appendix A). For women 1-2 s.d. below mean height, an additional year of average education reduces the intergenerational correlation by 18%.

Aggregate income shocks

The interaction term between income and maternal height is positive and indicates that a one s.d. increase in log p.c. GDP produces a significant reduction of intergenerational persistence of 28.7% (Table 4). Using indicators of the mother's place in the height distribution of her country, we find that income is most effective in weakening the intergenerational coefficient for short women (Table 2, Appendix A). For example, for women whose height is 1-2 standard deviations below the country mean, a one s.d. increase in log real p.c. GDP creates a 24.6% reduction in the size of the intergenerational coefficient. While the implications of this volatility for growth have been analysed, analysis of its effects on welfare has been more limited (Pallage and Robe 2003). Our findings are consistent with pro-cyclicality in public services relevant to child survival and with business cycle fluctuations in the fraction of constrained households. This may also reflect endogenous fertility and labour supply responses to business cycle variation.

Public health

Family background tends to play a greater role in determining children's human capital in poorer countries, primarily because of the limited spread of public health services; for evidence of the latter see Fan and Rao 2003, World Bank 2004. Using immunization rates for the sub-sample 1985-2000, we again find significant interaction effects, weak for DPT but well determined for measles. A 1 s.d. increase (21.9 %-points

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[12] As explained in section 1, the average education of a cohort of mothers does not change over time but the average education of mothers of a cohort of babies does, and this is the variation we exploit here. Some variation in composition arises naturally (i.e. is random), while some is likely to be a response to the macro-environment (see section 3).

for DPT and 23.2 %-points for measles) in immunization rates results in a 16.1% reduction in the intergenerational correlation of health when we use DPT rates and a 18.5% reduction when we use measles (Table 4). In contrast to the results for education and income, attenuation is not significantly greater for shorter women (Appendix A Table 2). This is probably a reflection of the public good nature of immunization.

We repeated estimation of equation (2) without country-specific trends, and found similar results (see Appendix B, Table 3). *Overall*, the results in this section show that every measure of SES that we consider attenuates the intergenerational correlation. For education and income improvements, this attenuation is clearly stronger amongst shorter (less healthy) women. Currie and Moretti (2007) similarly report attenuation with mother's education and local poverty rates at the birth of each of the mother and child, and show that this is greater amongst women of lower birth weight. They do not investigate father's education, aggregate growth or indicators of public health.

6.2.2. Mother Fixed Effects Estimates

For reasons discussed in sections 1 and 2, we re-estimated the models in which mother's height is interacted with indicators of the early life environment using mother fixed effects to remove all time-invariant mother-level characteristics. We gain estimates of the interaction term that are purged of potential confounders although the additive term in mother's height is now absorbed by the fixed effect. Results using height in metres are in Table 4 and results with height dummies are in Table 2, Appendix A. The results resoundingly reject the concern that unobservable endowments drive our results. The interaction terms are all considerably larger, being larger by a factor of four, three and two for education, income and immunization respectively. They remain significant for education and immunization. The interaction with income loses significance at the conventional level in the linear model although, in the nonlinear model, it is significant

for women of height 1-2 s.d. below the mean. The nonlinear models continue to display the non-linearity and asymmetry observed earlier.

6.2.3. Conditions in the Foetal Period

The specifications reported in Table 4 which show the gradient effects of cohort-country shocks with and without mother fixed effects were re-estimated replacing birth year with foetal year shocks (see Table 5). We also estimated these lagged effects using the non-linear specification of height (see Table 3, Appendix A). When mortality risk is linear in height, absent mother fixed effects, the lagged interaction terms are of very similar magnitude and insignificantly different from the birth year interaction terms. Once we condition upon mother fixed effects, the interaction and direct effects of lagged mother's education are about a third as small as the birth year effects. The interaction and direct effects of income (GDP) are also about a third as small, but they are now significant. The interaction and direct effects of current and lagged immunization rates are similar in size to those for birth year shocks. Full details are in Appendix A but once we replace height in metres with a set of dummies indicating height deviations, we again see estimates that are, overall, of similar size and significance in the foetal period as for birth year.

6.2.4. Discussion

A recent literature documents the importance of environmental conditions at the time children are *in utero* for their longer term health. We extend this literature, looking for how *in utero* conditions influence the gradient of the intergenerational correlation of health, allowing for the possibility that mothers of different health status experience different effects.

In the existing literature, the mother's *consumption* during pregnancy has been shown to matter. For example, alcohol consumption and fasting during pregnancy appear to have adverse effects on the child (Nilsson 2008, Almond and Mazumder 2008).

However, there is limited if any evidence that prenatal *income* shocks have an impact; see Cutler, Miller and Norton 2007, Banerjee et al. 2007 and van den Berg et al. 2009 for early twentieth century America and nineteenth century France and Denmark respectively. We similarly find, on average, no significant direct effect of income shocks on child health. However, once interactions of income with an index of mother's health (height) are allowed, the direct and interaction effects of income are significant and this is the case for income in the birth year and income in the year before birth. The interaction effects are stronger for children born to relatively short women.[13] Our analysis suggests that allowing the effects of prenatal or birth year income to depend upon the initial endowment of the child (proxied by her mother's permanent health) may be crucial to realising its potential to improve health for children who start life with relatively poor endowments.

We find that a proxy for public health provision in the *in utero* period has significant effects. This underlines the importance not only for women but also for children (and their children) of policies targeted at pregnant mothers. This includes tetanus immunization for pregnant women but, in fact, immunization rates will proxy wider public health services. The finding of weak income effects in 19th century France is argued by Banerjee et al. 2007 to possibly reflect the positive countervailing influence of public health infrastructure. The only other similar evidence of the ameliorating effects of public health infrastructure in this domain is in Almond and Chay (2005), a study we described in section 1. We also find that maternal education has pre-natal effects. This is

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[13] There is a fair overview but there is some variation across model specifications. The direct effect of income is negative but insignificant in Tables 1 and 2. Table 4 shows specifications that include interactions of income and mother's height with income measured in the birth year. The direct and interaction effects are significant in the absence of mother fixed effects. With mother fixed effects, both direct and interaction terms are larger, but poorly determined. In specifications that incorporate nonlinear height, some interaction terms are significant even in the fixed effects model (Appendix A). Table 5 reports estimates of the gradient models using income in the year before birth. Now direct and interaction terms are significant with and without mother fixed effects. The Tables in Appendix A show that the interaction terms are larger amongst shorter women.

consistent with education working through pre- and post-natal investments in children. For example, Currie and Moretti (2003) show that exogenously generated improvements in mother's education result in improved child health at birth because educated women are more likely to use prenatal care, less likely to smoke, more likely to be married and likely to have lower fertility. Our results show that maternal education is especially beneficial for offspring health amongst mothers who are of relatively poor health status. This is important because genetic and possibly other aspects of permanent health are carried through generations, making it difficult today to alter the fact that some children are born in weaker health because they are born to mothers of lower health status. However, if we know that education works through, say, the use of antenatal care or the avoidance of smoking, then policy efforts to encourage these healthy behaviours should be focused upon less educated women.

The timing of in-utero and birth year effects: While it is interesting that there are significant effects of lagged conditions, we are conscious that changes in conditions cannot be accurately timed with annual data. For children born in January, lagged conditions provide a better approximation of *in utero* conditions but for children born in December, current conditions provide the better approximation.[14] One way of interpreting the similarity of our findings for current and lagged conditions is that it indicates robustness of the main result (that early life conditions attenuate the intergenerational persistence of health) to the timing problem.

6.2.5. Longer Run Economic Development

The preceding specifications capture the effects of annual changes, but it is also of interest to consider the effects of longer run economic growth, which "wraps"

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[14] We used information on quarter of birth (as in Cutler et al. 2007, Bhalotra 2009a) to make this adjustment and there was no qualitative change in the results. The timing problem arises in many similar studies. For example, amongst those cited here, Banerjee et al. (2007) interpret effects for children born <u>during</u> the phylloxera crisis as *in utero* impacts while van den Berg et al. (2009) interpret effects of <u>lagged</u> conditions as *in utero* effects.

together education, income and public health. We initially investigated whether the intergenerational correlation of health has weakened over time, estimating decade-specific regressions, with observations pooled over countries (see Bhalotra and Rawlings 2009). We found no systematic variation over time, possibly because our sample (which includes Africa) defies the common presumption that living standards improve systematically with time. Using the strategy set out in section 2.5, we find, as predicted, that the intergenerational coefficient decreases more rapidly between cohorts 1970-75 and 1990-95 in countries with positive growth than in countries that failed to grow (Table 6). Indeed, in the stagnating group, the intergenerational correlation coefficient rises over time. We also see that the intergenerational correlation is more similar across the country-groups at the end of the period than it was at the start, consistent with diffusion of technology and with aggregate improvements in public health that diminish the weight of family background.

Long run growth is also captured in cross-country averages. Country-specific estimates of equation (1) show that the intergenerational coefficient is significant in 28 of the 38 countries, with marginal effects ranging between -0.158 in Rwanda and -0.044 in Turkey (Bhalotra and Rawlings 2009). A plot of these against the average of GDP during 1970-2000 shows a significantly negative relation; see Figure 3 where the slope of the fitted lines is -0.027 (t= -3.34).

7. Conclusions

We have analysed an unprecedentedly large and diverse sample of some 2.24 million children of 31 birth cohorts from 38 developing countries for whom comparable data on their own and their mother's health are available. The analysis has focused upon estimating the gradient of the intergenerational correlation of health, allowing the level and gradient effects to vary across the distribution of maternal health.

We find that maternal stature has a substantial negative influence on infant mortality conditional upon family and country living standards. This result supports the proposition that adult height is an indicator of health (Steckel 1995, Costa 1998, Deaton 2007, Bozzoli et al. 2009). The increase in mortality risk associated with being born to a short mother is larger than the decrease in mortality risk associated with being born to a tall mother. The policy question then concerns what can be done to release children born to shorter women from being condemned to poorer health and thereby poorer living standards in the long run. Natural questions pertain to the role of economic growth and public health provision. We show that the correlation declined faster in countries that experienced significant growth over the period 1970-1995 than in those that experienced slower or negative growth. However, long range growth is potentially confounded with other changes such as medical technological progress that may be correlated with child health (Deaton and Paxson 2004). So we estimated a model that controls for growth and other unobservable trends, exploiting more plausibly exogenous annual variation in income, education and public health provision (indicated by immunization rates).

We find that mother's education, immunization rates and economic upturns (including cyclical and episodic income variation captured in annual data) in the year of and in the year before birth weaken the intergenerational transmission of health. These gradients are all enlarged once we condition upon mother fixed effects. Attenuation of the intergenerational correlation induced by income and education is stronger amongst shorter women. This suggests that improvements in the health environment around a child's birth help to override the effects of weak endowments (genetic and other aspects of family background- all of which are reflected in mother's height), limiting the degree to which child health is tied to family circumstance. Overall, this paper contributes unique evidence on an important and under-studied aspect of persistent inequality in

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developing countries, where underdeveloped markets and states result in children being

unable to escape from the family circumstances that they are born into.

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Figures and Tables

Figure 1: Infant mortality against mother's height: lowess predictions
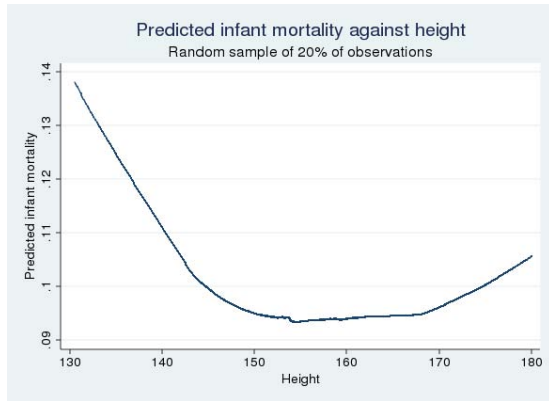
Notes: As lowess on the full sample is computationally intensive, this graph was estimated using a random sample of 20% of observations. The density of observations in the lowess graph is as follows: the 10th, 25th, 50th, 75th and 95th percentiles of height are 1.468m, 1.510m, 1.558m, 1.606m, and 1.679m, respectively.

Figure 2: Country-specific plots of infant mortality against mother's height: lowess predictions
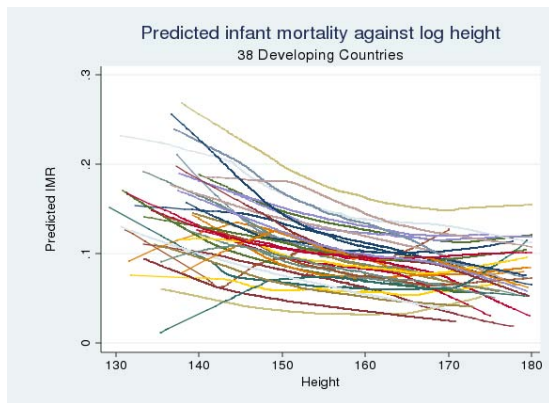
Figure 3: Country-specific marginal effects against average Log GDP
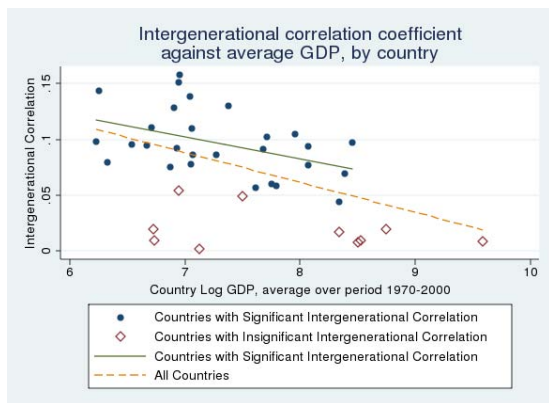
Table 1: Baseline model: The effect of mother's height on infant mortality risk

	(1)	(2)	(3)	(4)	(5)	(6) Baseline
	country*year dummies	Add individual characteristics	Add further characteristics	Add mother's education	Add father's education	Add log p.c. GDP
Height	-0.135**	-0.121**	-0.111**	-0.095**	-0.092**	-0.092**
	[0.014]	[0.013]	[0.011]	[0.010]	[0.010]	[0.010]
Mother's Education						
Primary				-0.017**	-0.013**	-0.013**
				[0.001]	[0.001]	[0.001]
Secondary				-0.037**	-0.030**	-0.030**
				[0.001]	[0.001]	[0.001]
Higher				-0.048**	-0.040**	-0.040**
				[0.001]	[0.002]	[0.002]
Father's Education						
Primary					-0.006**	-0.006**
					[0.001]	[0.001]
Secondary					-0.016**	-0.016**
					[0.001]	[0.001]
Higher					-0.021**	-0.022**
					[0.002]	[0.002]
Log p.c. GDP						-0.012
						[0.009]
Observations	2237491	2237491	2237491	2237491	2235090	2235094
Mean Height	1.559	1.559	1.559	1.559	1.559	1.559
IMR	0.097	0.097	0.097	0.097	0.097	0.097
s.d. Log p.c. GDP						0.744

Robust standard errors clustered by country are in brackets. * significant at 5%; ** significant at 1%. The dependent variable is 1 if the index child dies in infancy i.e. by their first birthday. Sample is restricted to children born at least 12 months before the date of the survey. Mother's height is in metres. The estimator is probit and these are marginal effects. Incremental changes to the specification are noted in the column head and are cumulative in moving from left to right except that when we include GDP then we replace country-time dummies with country and time dummies and country-specific trends. The controls in col. 2 are gender, religion and rural/urban residence and in col.3 they are birth month, birth order, mother's age at birth. Religion is controlled for by dummy variables for Christian, Muslim, other, no religion, and religion missing. The number of observations changes slightly as we add more controls due to missing data present in these controls. Column (6) shows us that GDP is insignificant in the pooled sample. However, when we split by continent (see Bhalotra and Rawlings, 2009), we see that the effect of GDP is negative and significant in Africa and Latin America. IMR denotes infant mortality rate, the mean of the dependent variable.

Table 2: Baseline model generalized to allow non-linear (& asymmetric) effects of height

	(1) Linear	*(2)* Non-linear
Height in metres	**-0.092****	
	[0.010]	
Height in deviations from country mean		
Std deviations *above* mean		
Tall (0.5-1 s.d.)		**-0.004****
		[0.001]
Tall (1-2 s.d.)		**-0.007****
		[0.001]
Tall (> 2 s.d.)		**-0.006***
		[0.003]
Std deviations *below* mean		
Short (0.5-1 s.d.)		**0.005****
		[0.001]
Short (1-2 s.d.)		**0.012****
		[0.001]
Short (> 2 s.d.)		**0.023****
		[0.005]
Log p.c. GDP	-0.012	-0.012
	[0.009]	[0.009]
Observations	2235094	2235094
Mean Height	1.559	
IMR	0.097	0.097
s.d. Log p.c. GDP	0.744	0.722
% mothers tall (1-2 s.d)		10.9
% mothers tall (>2 s.d.)		1.4
% mothers tall (0.5-1 s.d.)		14.9
% mothers short (1-2 s.d.)		11.6
% mothers short (>2 s.d.)		1.3
% mothers short (0.5-1 s.d.)		16.0

Robust standard errors clustered by country in brackets. * significant at 5% level; ** significant at 1% level. See notes to Table 1. Controls include family demographics, education of both parents and country*year dummies. Column (1) reproduces column (6) of Table 1 for comparison. In Column (2) we replace height in metres with a set of dummies indicating the mother's place in the height distribution of her country. Tall (0.5-1 s.d.), Tall (1-2 s.d.) and Tall (>2 s.d.) refer to being between 0.5 and 1, between 1 and 2, and greater than 2 standard deviations above mean height, respectively. Short (0.5-1 s.d.), Short (1-2 s.d.) and Short (>2 s.d.) refer to being between 0.5 and 1, between 1 and 2, and greater than 2 s.d. below mean height, respectively.

Table 3: Gradients: Interactions of mother's height with mother's socioeconomic status

	(1) Education of mother	(2) Mother is educated and urban	(3) Education of father
	Marginal Effects		
Mother's Height	-0.110**	-0.102**	-0.106**
	[0.011]	[0.010]	[0.010]
Mother's height*SES	**0.005****	**0.043****	**0.002**
	[0.001]	**[0.013]**	**[0.001]**
SES	-0.011**	-0.064**	-0.005*
	[0.002]	[0.021]	[0.002]
Observations	2234269	2235094	2141890
Mean Mother's years of education	3.135		
% sample educated and urban		12.40%	
Mean Father's years of education			4.612
s.d. Log p.c. GDP	0.744	0.744	0.744
	Total Effects		
Mother's Height	-0.093**	-0.096**	-0.096**
	[0.010]	[0.010]	[0.010]
SES	-0.003**	0.007**	-0.002**
	[0.000]	[0.002]	[0.000]

Robust standard errors clustered by country in brackets. * significant at 5%; ** significant at 1%. Controls include family demographics, education of both parents and country and year dummies and country*year dummies. See notes to Table 1. Socioeconomic status or 'SES' refers to (1) years of education of mother, (2) an indicator for whether the mother has secondary or higher education and lives in an urban area, and (3) years of education of father. Refer equations (2) and (3) in the text. The 'total effect' of height is calculated as the partial derivative of child health with respect to mother height, and we evaluate the effect of the interaction term at the mean of the SES variable. Similarly for the total effect of SES, we calculate the partial derivative of child health with respect to that variable and evaluate it at the mean of mother height. Estimates that cast height as indicators for deviations from the mean are in Appendix A Table 1. See Table 1 for means of IMR and height in the sample.

Table 4: Gradients: Interactions of maternal height with country-cohort level indicators of conditions in the child's birth year:

Estimates with & without mother fixed effects

	Log p.c. GDP		Avg Education Mothers		DPT immunization		Measles immunization	
	No FE	FE	No FE	FE	No FE	FE	No FE	FE
Mother's Height	-0.351**		-0.145**		-0.136**		-0.139**	
	[0.076]		[0.018]		[0.025]		[0.023]	
Mother's height*Log p.c. GDP	**0.034****	**0.117**						
	[0.010]	**[0.077]**						
Mother's height*average education of mothers			**0.016****	**0.063****				
			[0.005]	**[0.015]**				
Mother's height*immunization					**0.069**	**0.130***	**0.075***	**0.123***
					[0.037]	**[0.057]**	**[0.036]**	**[0.054]**
Log p.c. GDP	-0.069**	-0.202						
	[0.018]	[0.121]						
Average years of mother's education			-0.019*	-0.093**				
			[0.008]	[0.023]				
Immunization rates					-0.120*	-0.221*	-0.127*	-0.206*
					[0.058]	[0.084]	[0.057]	[0.080]
Observations	2235094	2137324	2235094	2137324	1432036	1344815	1432036	1344815
Mean Height	1.559	1.559						
IMR	0.097	0.099						
Mean Mother's Education			3.093	3.051				
DPT Immunization Rate					0.600	0.591		
Measles Immunization Rate							0.593	0.588

Robust standard errors clustered by country in brackets. * significant at 5%; ** significant at 1%. FE denotes mother fixed effects. Controls include family demographics, education of both parents and country and year dummies and country-specific trends. See notes to Table 1. The 'SES' variables are measured at the country-cohort level. They are (1) average years of education of mothers in the index child's country and birth cohort, (2) GDP (3)/(4) DPT and measles immunization rates, % of 12-23 month old children immunized. (1) & (2) use data on 38 countries in 1970-2000 while (3) & (4) use data on 37 countries in 1985-2000. See Table 1 for s.d. of Log GDP for columns (1) and (2).

Table 5: Gradients: Interactions of maternal height with country-cohort indicators of conditions in the child's foetal year:

Estimates with & without mother fixed effects

	Log p.c. GDP		Avg Education Mothers		DPT immunization		Measles immunization	
	No FE	FE	No FE	FE	No FE	FE	No FE	FE
Mother's Height	-0.362**		-0.144**		-0.133**		-0.133**	
	[0.075]		[0.018]		[0.022]		[0.019]	
Mother's height*Log p.c. GDP, lagged	**0.036****	**0.129**						
	[0.010]	**[0.075]**						
Mother's height*average education of mothers, lagged			**0.016****	**0.068****				
			[0.005]	**[0.017]**				
Mother's height*immunization, lagged					**0.067**	**0.149****	**0.066***	**0.111****
					[0.035]	**[0.039]**	**[0.032]**	**[0.036]**
Log p.c. GDP, lagged	-0.064**	-0.214						
	[0.017]	[0.119]						
Average years of mother's education, lagged			-0.021*	-0.105**				
			[0.008]	[0.026]				
Immunization rates, lagged					-0.11	-0.241**	-0.110*	-0.180**
					[0.056]	[0.061]	[0.051]	[0.055]
Observations	2235094	2137234	2216133	2118829	1334874	1249171	1334874	1249171
Mean Height	1.559	1.559	1.559	1.559	1.559	1.559	1.559	1.559
IMR	0.096	0.098	0.097	0.099	0.086	0.089	0.086	0.089
Mean Mother's Education			3.02	2.981				
DPT Immunization Rate					0.587	0.581		
Measles Immunization Rate							0.584	0.579

Robust standard errors clustered by country in brackets. * significant at 5%; ** significant at 1%. FE denotes mother fixed effects. Controls include family demographics, education of both parents and country and year dummies and country-specific trends. See notes to Table 1. The 'SES' variables are measured at the country-cohort level. They are (1) average years of education of mothers in the index child's country and birth cohort, (2) GDP (3)/(4) DPT and measles immunization rates, % of 12-23 month old children immunized. (1) & (2) use data on 38 countries in 1970-2000 while (3) & (4) use data on 37 countries in 1985-2000. See Table 1 for s.d. of Log GDP for columns (1) and (2).

Table 6: Estimates for polarised cohorts in growing, regressing and stagnating countries

| | A: Growth > 1.5% p.a. | | B: Growth < -1.5% p.a. | | C: Insignificant Growth p.a. | |
	1970-1975	1990-1995	1970-1975	1990-1995	1970-1975	1990-1995
Height	-0.110**	-0.090**	-0.058	-0.091**	-0.029	-0.077**
	[0.020]	[0.014]	[0.058]	[0.014]	[0.025]	[0.021]
Observations	65765	256646	17488	73247	37376	162619
IMR	0.140	0.073	0.110	0.085	0.131	0.103
Mean Height	1.547	0.155	1.554	1.552	1.584	1.588
s.d. Log GDP	0.637	0.632	1.102	1.148	0.529	0.590

Robust standard errors clustered by country in brackets. * significant at 5%; ** significant at 1%. Controls include family demographics, education of both parents and country and year dummies and country-specific trends. See notes to Table 1. The baseline model in the last column of Table 1 is estimated on three sub—samples, created to reflect different average levels of growth over the period 1970-2000. The growing countries in Panel A are Egypt, Lesotho, Morocco, Mali, Colombia, Dominican Republic, India and Turkey. The regressing countries in Panel B are Gabon, Madagascar, Togo, Zambia, Nicaragua, and Cambodia. The stagnating countries in Panel C are Benin, Cameroon, Chad, Comoros, Cote d'Ivoire, Ghana, Guinea, Mozambique, Namibia, Rwanda, Senegal, Tanzania, Uganda, Zimbabwe, Haiti. Countries that are in none of these samples are: Brazil, Burkina Faso, CAR, Ethiopia, Honduras, Kenya, Malawi, Niger, Peru. As a check on the construction of these three groups, we repeated the estimation grouping all countries into those with positive growth, negative growth and insignificant growth. The results were similar. See Notes to Table A2 for an explanation of how growth rates are calculated.

Appendix A: Main Results with a Nonlinear Specification of Height

Table 1: Interactions of Height Dummies with Mother's Socioeconomic Status

	(1) Education of mother	(2) Mother is educated and urban	(3) Education of father
		Marginal Effects	
Tall (0.5-1 s.d.)	-0.005**	-0.004**	-0.005**
	[0.001]	[0.001]	[0.001]
Tall (1-2 s.d.)	-0.008**	-0.007**	-0.008**
	[0.001]	[0.001]	[0.001]
Tall (> 2 s.d.)	-0.008*	-0.006*	-0.005
	[0.003]	[0.003]	[0.004]
Tall (0.5-1 s.d.)*SES	0.000	0.003	0.000
	[0.000]	[0.001]	[0.000]
Tall (1-2 s.d.)*SES	0.000*	0.002	0.000
	[0.000]	[0.002]	[0.000]
Tall (> 2 s.d.)*SES	0.001	0.001	0.000
	[0.000]	[0.003]	[0.000]
Short (0.5-1 s.d.)	0.007**	0.006**	0.007**
	[0.001]	[0.001]	[0.001]
Short (1-2 s.d.)	0.015**	0.013**	0.014**
	[0.001]	[0.001]	[0.001]
Short (> 2 s.d.)	0.026**	0.024**	0.026**
	[0.005]	[0.005]	[0.005]
Short (0.5-1 s.d.)*SES	-0.000**	-0.002	-0.000*
	[0.000]	[0.001]	[0.000]
Short (1-2 s.d.)*SES	-0.001**	-0.012**	0.000
	[0.000]	[0.001]	[0.000]
Short (> 2 s.d.)*SES	-0.001	-0.010	0.000
	[0.001]	[0.009]	[0.001]
SES	-0.271**	0.816**	-0.003**
	[0.022]	[0.196]	[0.000]
Observations	2234269	2235094	2141890
		Total Effects	
Tall (0.5-1 s.d.)	-0.004**	-0.004**	-0.004**
	[0.001]	[0.001]	[0.001]
Tall (1-2 s.d.)	-0.007**	-0.007**	-0.007**
	[0.001]	[0.001]	[0.001]
Tall (> 2 s.d.)	-0.006*	-0.006*	-0.006*
	[0.003]	[0.003]	[0.003]
Short (0.5-1 s.d.)	0.005**	0.005**	0.005**
	[0.001]	[0.001]	[0.001]
Short (1-2 s.d.)	0.011**	0.012**	0.013**
	[0.001]	[0.001]	[0.001]
Short (> 2 s.d.)	0.023**	0.023**	0.024**
	[0.005]	[0.005]	[0.005]
SES	-0.003**	0.007**	-0.003**
	[0.000]	[0.002]	[0.000]

Interactions as in Table 3 but with a non-linear specification of height. Robust standard errors clustered by country. * significant at 5%; ** significant at 1%. See notes to Table 2, 3.

Table 2: Interactions of Height Dummies with Country-Cohort Indicators of Conditions in the Child's Birth Year, with and without Mother Fixed Effects

	(1) Average Education of Mothers in child birth cohort		(2) Log p.c. GDP		(3) DPT immunization		(4) Measles immunization	
	No FE	FE	No FE	FE	No FE	FE	No FE	FE
Tall (0.5-1 s.d.)	-0.008**		-0.030**		-0.009**		-0.008**	
	[0.002]		[0.008]		[0.002]		[0.002]	
Tall (1-2 s.d.)	-0.011**		-0.026**		-0.009**		-0.009**	
	[0.002]		[0.009]		[0.002]		[0.002]	
Tall (> 2 s.d.)	-0.011		-0.056		-0.015*		-0.018*	
	[0.007]		[0.028]		[0.008]		[0.007]	
Tall (0.5-1 s.d.)*SES	0.001**	0.002	0.004**	0.009	0.009**	0.015*	0.008**	0.009
	[0.000]	[0.001]	[0.001]	[0.005]	[0.003]	[0.006]	[0.003]	[0.005]
Tall (1-2 s.d.)*SES	0.001*	0.006**	0.003*	0.012	0.006	0.016**	0.005	0.012*
	[0.001]	[0.002]	[0.001]	[0.007]	[0.004]	[0.005]	[0.003]	[0.005]
Tall (> 2 s.d.)*SES	0.002	0.007	0.007	0.025	0.017	0.011	0.021*	0.015
	[0.002]	[0.006]	[0.004]	[0.018]	[0.010]	[0.024]	[0.009]	[0.020]
Short (0.5-1 s.d.)	0.008**		0.023**		0.013**		0.014**	
	[0.001]		[0.005]		[0.003]		[0.003]	
Short (1-2 s.d.)	0.019**		0.041**		0.009**		0.009**	
	[0.002]		[0.010]		[0.002]		[0.002]	
Short (> 2 s.d.)	0.038**		0.108**		0.035**		0.034**	
	[0.010]		[0.037]		[0.010]		[0.009]	
Short (0.5-1 s.d.)*SES	-0.001*	-0.004*	-0.002**	-0.009	-0.006	-0.005	-0.006*	-0.007
	[0.000]	[0.002]	[0.001]	[0.009]	[0.003]	[0.005]	[0.003]	[0.005]
Short (1-2 s.d.)*SES	-0.002**	-0.007**	-0.004**	-0.015*	-0.002	-0.016*	-0.002	-0.019*
	[0.001]	[0.001]	[0.001]	[0.007]	[0.006]	[0.008]	[0.005]	[0.007]
Short (> 2 s.d.)*SES	-0.004*	-0.022**	-0.011*	-0.048	-0.017	-0.02	-0.015	-0.02
	[0.002]	[0.003]	[0.005]	[0.025]	[0.014]	[0.017]	[0.012]	[0.017]
SES	0.006*	0.005	-0.015	-0.018	-0.013	-0.019	-0.011	-0.014
	[0.002]	[0.003]	[0.010]	[0.010]	[0.009]	[0.011]	[0.008]	[0.010]
Observations	2235094	2235094	2235094	2235094	1432036	1432036	1432036	1432036

Total Effects

	(1)	(2)	(3)	(4)
Tall (0.5-1 s.d.)	-0.004**	-0.004**	-0.003**	-0.003**
	[0.001]	[0.001]	[0.001]	[0.001]
Tall (1-2 s.d.)	-0.007**	-0.007**	-0.006**	-0.006**
	[0.001]	[0.001]	[0.001]	[0.001]
Tall (> 2 s.d.)	-0.006*	-0.006*	-0.005	-0.005
	[0.003]	[0.003]	[0.003]	[0.002]
Short (0.5-1 s.d.)	0.005**	0.005**	0.006**	0.006**
	[0.001]	[0.001]	[0.001]	[0.001]
Short (1-2 s.d.)	0.012**	0.012**	0.012**	0.012**
	[0.001]	[0.001]	[0.001]	[0.001]
Short (> 2 s.d.)	0.025**	0.025**	0.025**	0.025**
	[0.005]	[0.005]	[0.004]	[0.004]
SES	0.006*	-0.015	-0.012	-0.010
	[0.002]	[0.010]	[0.009]	[0.008]

Robust standard errors in brackets, allowing for clustering within country. * significant at 5%; ** significant at 1%. See notes to Table 1, 2 and 4. See Tables 2, 4 and Appendix B Table 2 for sample means of variables of interest.

Table 3: Interactions of Height Dummies with Country-Cohort Indicators of Conditions when the Child is *in Utero*, with and without Mother Fixed Effects

	(1) Average education of mothers in index child's birth cohort		*(2)* Log p.c. GDP		*(3)* DPT immunization		*(4)* Measles immunization	
	No FE	FE	No FE	FE	No FE	FE	No FE	FE
Tall (0.5-1 s.d.)	-0.008**		-0.030**		-0.007**		-0.006**	
	[0.002]		[0.008]		[0.002]		[0.002]	
Tall (1-2 s.d.)	-0.011**		-0.026**		-0.009**		-0.009**	
	[0.002]		[0.009]		[0.003]		[0.002]	
Tall (> 2 s.d.)	-0.011		-0.057*		-0.015		-0.016*	
	[0.007]		[0.028]		[0.007]		[0.007]	
Tall (0.5-1 s.d.)*SES, lagged	0.001**	0.001	0.003**	0.008	0.007	0.009		0.002
	[0.000]	[0.001]	[0.001]	[0.005]	[0.004]	[0.006]		[0.006]
Tall (1-2 s.d.)*SES, lagged	0.001*	0.006**	0.003*	0.012	0.006	0.019**		0.013*
	[0.001]	[0.002]	[0.001]	[0.008]	[0.004]	[0.007]		[0.006]
Tall (> 2 s.d.)*SES, lagged	0.002	0.009	0.007	0.031	0.016	0.004		0.007
	[0.002]	[0.005]	[0.004]	[0.019]	[0.010]	[0.023]		[0.021]
Short (0.5-1 s.d.)	0.008**		0.024**		0.010**		0.010**	
	[0.001]		[0.005]		[0.003]		[0.003]	
Short (1-2 s.d.)	0.019**		0.042**		0.014**		0.012**	
	[0.002]		[0.009]		[0.003]		[0.003]	
Short (> 2 s.d.)	0.037**		0.116**		0.033**		0.033**	
	[0.010]		[0.037]		[0.010]		[0.009]	
Short (0.5-1 s.d.)*SES, lagged	-0.001*	-0.004*	-0.002**	-0.013	-0.007	-0.011*		-0.013*
	[0.000]	[0.002]	[0.001]	[0.008]	[0.004]	[0.005]		[0.006]
Short (1-2 s.d.)*SES, lagged	-0.002**	-0.007**	-0.004**	-0.015	-0.002	-0.017*		-0.008
	[0.001]	[0.001]	[0.001]	[0.007]	[0.005]	[0.007]		[0.007]
Short (> 2 s.d.)*SES, lagged	-0.004*	-0.022**	-0.012*	-0.05	-0.014	-0.026		-0.025
	[0.002]	[0.004]	[0.005]	[0.025]	[0.014]	[0.017]		[0.017]
SES, lagged	0.005	0.002	-0.008	-0.01	-0.006	-0.007		-0.005
	[0.003]	[0.004]	[0.008]	[0.007]	[0.006]	[0.006]		[0.005]
Observations	2235094	2137234	2216133	2118829	1334874	1249171	1334874	1249171

Interactions as in Table 4 but with a non-linear specification of height. Robust standard errors clustered by country. * significant at 5%; ** significant at 1%. See notes to Tables 2, 5.

Table 1 – Countries in our sample by continent

Country	Country Code	Country	Country Code
African Countries			
Benin	BE	Madagascar	MD
Burkina Faso	BF	Malawi	MW
CAR	CR	Mali	ML
Cameroon	CM	Morocco	MO
Chad	CH	Mozambique	MZ
Comoros	CO	Namibia	NB
Cote d'Ivoire	CI	Niger	NG
Egypt	EG	Rwanda	RW
Ethiopia	ET	Senegal	SE
Gabon	GB	Tanzania	TZ
Ghana	GH	Togo	TO
Guinea	GU	Uganda	UG
Kenya	KE	Zambia	ZB
Lesotho	LE	Zimbabwe	ZW
Latin American Countries			
Brazil	BR	Honduras	HO
Colombia	CB	Nicaragua	NC
Dominican Republic	DR	Peru	PE
Haiti	HA		
Asian Countries			
Cambodia	CD	Turkey	TK
India	IN		

Table 2 – Country Details

Country	GDP growth, 1970-2000	IMR decline, 1970-2000	Possible child birth-year range	Interview Dates
Benin	0.103	-2.067**	1960-2001	1996, 2001
Brazil	1.367*	-4.220**	1959-1996	1996
Burkina Faso	0.955*	-2.257**	1955-2003	1992-1993, 1998-1999, 2003
CAR	-0.927*	-1.427*	1958-1995	1994-1995
Cambodia	-2.275*	-1.911**	1965-2000	2000
Cameroon	0.682	-1.311**	1960-2004	1998, 2004
Chad	-0.044	-1.113**	1960-2004	1996-1997, 2004
Colombia	1.781*	-4.327**	1958-2005	1995, 2000, 2004-2005
Comoros	-0.312	-3.389**	1961-1996	1996
Cote d'Ivoire	0.123	-2.056**	1956-1999	1994, 1998-1999
Dominican Republic	2.277*	-3.281**	1952-1996	1991, 1996
Egypt	3.126*	-5.316**	1954-2005	1992-1993, 1995-1996, 2000, 2005
Ethiopia	1.018*	-3.554**	1955-1997	2000, 2005
Gabon	-1.722*	-2.634**	1962-2000	2000-2001
Ghana	0.188	-2.000**	1957-2003	1993-1994, 1998-1999
Guinea	-0.098	-2.223**	1961-2005	1999, 2005
Haiti	-0.12	-3.752**	1957-2000	1994-1995, 2000
Honduras	0.471*	-3.783**	1969-2006	2005-2006
India	2.831*	-3.070**	1961-2000	1998-2000, 2005-2006
Kenya	0.338*	-0.980*	1954-2003	1993, 2003
Lesotho	2.889*	-1.366*	1967-2005	2004-2005
Madagascar	-1.860*	-1.027	1962-2004	1997, 2003-2004
Malawi	1.210*	-1.913**	1954-2005	1992, 2000, 2004-2005
Mali	1.557*	-2.226**	1960-2001	1995-1996, 2001
Morocco	1.659*	-4.319**	1953-2004	1992, 2003-2004
Mozambique	-0.27	-1.437**	1960-2004	1997, 2003-2004
Namibia	-0.139	-1.791*	1957-1992	1992
Nicaragua	-2.984*	-5.453**	1961-2001	1997-1998, 2001
Niger	-0.931*	-2.299**	1954-1998	1992, 1998
Peru	-0.964*	-4.473**	1956-2000	1991-1992, 2000
Rwanda	-0.688	0.052	1963-2005	2000, 2005
Senegal	-0.091	-1.805**	1954-2005	1992-1993, 2005
Tanzania	0.353	-0.856**	1953-2005	1991-1992, 1996, 2004-2005
Togo	-1.965*	-2.516**	1958-1998	1998
Turkey	2.068*	-5.598**	1957-1998	1993, 1998
Uganda	-0.492	-1.095**	1959-2001	1995, 2000-2001
Zambia	-1.861*	0.372	1954-2002	1992,1996-1997, 2001-2002
Zimbabwe	-0.057	-0.311	1957-1999	1994, 1999

Notes: Mothers are 15-49 years old at the time of interview and they record their complete fertility histories. For two countries, the lower age limit is 13, for one it is 12 and for 3 it is 16. The growth rate of x is the estimated coefficient from a regression of x on a linear trend. Growth rates are reported in percentages.

Table 3: Interactions of Height Dummies with Country-Cohort SES without country-specific trends

	(1) Education of Mother	*(2)* Log p.c. GDP	*(3)* DPT Immunization	*(4)* Measles Immunization
		Marginal Effects		
Tall (0.5-1 s.d.)	-0.008**	-0.029**	-0.009**	-0.009**
	[0.002]	[0.008]	[0.002]	[0.002]
Tall (1-2 s.d.)	-0.011**	-0.027**	-0.009**	-0.009**
	[0.002]	[0.010]	[0.002]	[0.002]
Tall (> 2 s.d.)	-0.01	-0.060*	-0.015	-0.018*
	[0.007]	[0.029]	[0.008]	[0.007]
Tall (0.5-1 s.d.)*SES	0.001**	0.003**	0.009**	0.009**
	[0.000]	[0.001]	[0.003]	[0.003]
Tall (1-2 s.d.)*SES	0.001*	0.003*	0.006	0.005
	[0.001]	[0.001]	[0.004]	[0.004]
Tall (> 2 s.d.)*SES	0.002	0.007	0.017	0.021*
	[0.002]	[0.004]	[0.010]	[0.009]
Short (0.5-1 s.d.)	0.008**	0.022**	0.009**	0.009**
	[0.001]	[0.005]	[0.002]	[0.002]
Short (1-2 s.d.)	0.020**	0.038**	0.013**	0.013**
	[0.002]	[0.010]	[0.003]	[0.003]
Short (> 2 s.d.)	0.040**	0.102**	0.035**	0.033**
	[0.010]	[0.037]	[0.010]	[0.009]
Short (0.5-1 s.d.)*SES	-0.001*	-0.002**	-0.006	-0.006*
	[0.000]	[0.001]	[0.003]	[0.003]
Short (1-2 s.d.)*SES	-0.002**	-0.003*	-0.001	-0.002
	[0.000]	[0.001]	[0.006]	[0.005]
Short (> 2 s.d.)*SES	-0.005*	-0.010*	-0.016	-0.014
	[0.002]	[0.005]	[0.013]	[0.012]
SES	0	-0.028*	-0.030*	-0.025*
	[0.007]	[0.013]	[0.012]	[0.010]
Observations	2235094	2235094	1432036	1432036
		Total Effects		
Tall (0.5-1 s.d.)	-0.004**	-0.004**	-0.003**	-0.003**
	[0.001]	[0.001]	[0.001]	[0.001]
Tall (1-2 s.d.)	-0.007**	-0.007**	-0.006**	-0.006**
	[0.001]	[0.001]	[0.001]	[0.001]
Tall (> 2 s.d.)	-0.006*	-0.006*	-0.005	-0.005
	[0.003]	[0.003]	[0.003]	[0.002]
Short (0.5-1 s.d.)	0.005**	0.005**	0.006**	0.006**
	[0.001]	[0.001]	[0.001]	[0.001]
Short (1-2 s.d.)	0.013**	0.013**	0.012**	0.012**
	[0.001]	[0.001]	[0.001]	[0.001]
Short (> 2 s.d.)	0.025**	0.025**	0.025**	0.025**
	[0.005]	[0.005]	[0.004]	[0.004]
SES	-0.000	-0.028*	-0.028*	-0.024*
	[0.007]	[0.010]	[0.012]	[0.009]

Robust standard errors in brackets, allowing for clustering within country. * significant at 5%; ** significant at 1%. See notes to Table 1, 3 and Table 6. See Tables 3 and 6 for sample means of variables of interest for columns (1), (2), (3) and (4). For columns (3) and (4), percentages of women falling in height categories are as follows: tall (1-2 s.d.) 11.0%, tall (>2 s.d.) 1.3%, tall (0.5-1 s.d.) 15.1%, short (1-2 s.d.) 11.1%, short (>2 s.d.) 1.2%, short (0.5-1 s.d.) 15.9%.
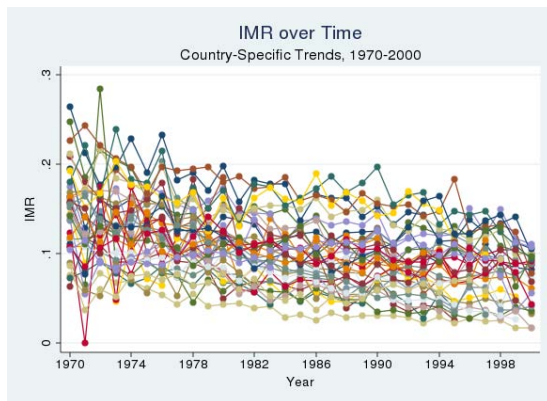
Figure A1: Trends in the Infant Mortality Rate

Pooled Sample

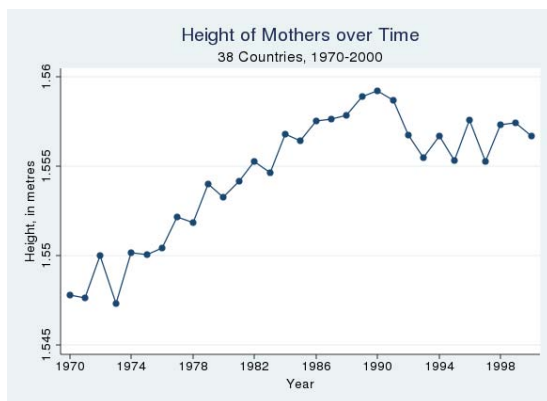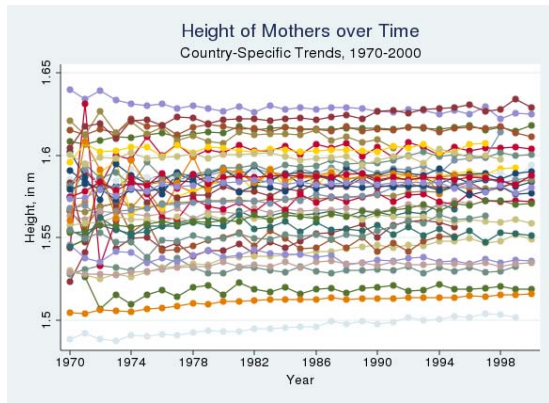Country-specific Trends

Figure A2: Country-Specific Trends in Mother's Height
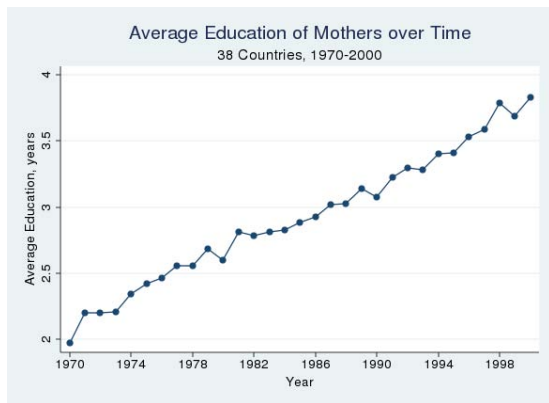
Pooled Sample

Country-specific Trends

Height of Mothers over Time
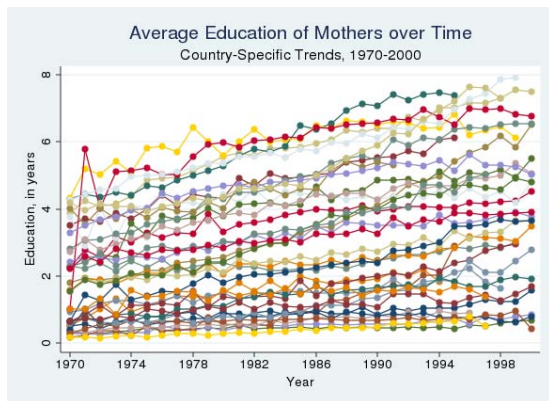Country-Specific Trends, 1970-2000

Figure A3: Country-Specific Trends in Mother's Education

Pooled Sample

Average Education of Mothers over Time
38 Countries, 1970-2000

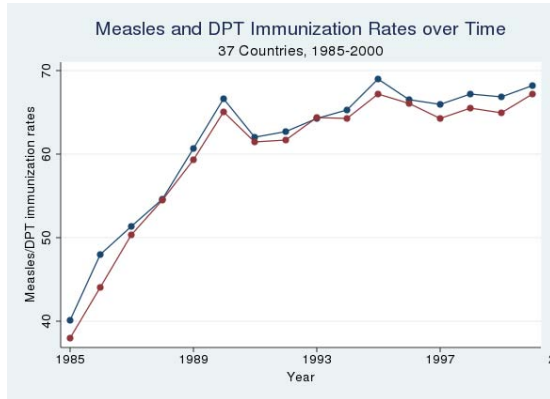Country-specific Trends

Average Education of Mothers over Time
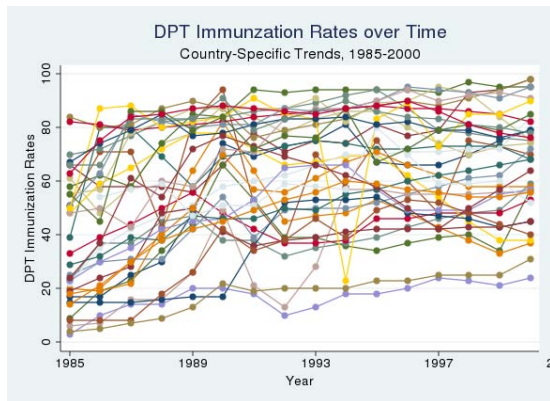Country-Specific Trends, 1970-2000

Figure A4: Country-Specific Trends in Immunization Rates

Pooled Sample, Measles/DPT trend over time

DPT immunization rates over time, country-specific trend

Measles immunization rates over time, all countries