Intergenerational Transmission of Capabilities: Mothers and Children in Sub-Saharan Africa

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Abstract

Childhood conditions are important for adult capabilities (health and education). Child health and educational attainment are to some extent determined by capabilities of parents to insure adequate care and investments. Even in developed countries with low disease exposures and good health service there has been observed intergenerational transmission of health and socioeconomic status from parents to children through childhood conditions. In sub-Saharan Africa (SSA) transmission is expected to be greater due to poverty, lack of health care, harmful disease environment and vulnerable food security. Many mothers have not lived up to their genetic potentials in capabilities which may negatively influence children's capabilities. In this paper intergenerational transmission of capabilities from mother to child in SSA will be studied. The focus will be on maternal capabilities determined by mother's childhood conditions. The main results are that maternal health has an influence on child capabilities but education is an important pathway.

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First draft

Introduction

People in sub-Saharan Africa face harsh living conditions due to rampant diseases and vulnerable food security. Infant and child mortality is the highest in the world and many children are underweight and stunted (Horton, Alderman, & Rivera, 2008; UNICEF, 2007; WHO, 2013). Low levels of education and poor adult health play an important role for determining child health in the developing world (Caldwell, 1979; Schell, Reilly, Rosling, Peterson, & Ekstrom, 2007). Many women in sub-Saharan Africa do not live up to their full potentials in physical health and cognition as reflected in their short stature[1] (Moradi, 2010) and the generally low levels of education (United Nations, 2012). Due to limited publicly provided services and inefficient national governance, households and communities are to a large extent left on their own in terms of securing the health and education of their children (Deaton A. , 2013, p. 121). Hence, the risk that poor health is transferred between generations is greater in sub-Saharan Africa than in more developed countries. This provides a setting for research on intergenerational transmission of capabilities and for a better understanding of the role of preventive mechanisms. Since women in sub-Saharan Africa generally hold a greater responsibility for childrearing and providing sufficient nutrition, as well as preventing and responding to diseases (FAO, 2014), their capabilities for child rearing are of great importance for improving child health. Healthier and more educated mothers are more likely to effectively use information and have more energy for successful child rearing (Caldwell, 1979; Bishai, Cohen, Alfonso, Adam, Kuruvilla, & Schweitzer, 2014).

Health is determined by a number of factors throughout the life course, and conditions already in utero and first year of life can have permanent effects on health, abilities and physical

[1] African women are shorter than women in developed countries although they are relatively tall compared to other developing regions and much taller than their living conditions would predict (Deaton A. , 2007).

conditions (Barker, 2004; Gluckman, Hanson, & Buklijas, 2010). Malnutrition and disease exposure during early life can negatively affect health as well as cognitive functions, which may further affect adult health, education and income (Case & Paxson, 2008). Since early life is the period when environmental factors have the greatest permanent effects on individuals' health and cognitive ability, young children need to be given adequate care and sufficient investments in favorable conditions in order to live up to their full potentials. Such investments concern for example nutrition, immunization, and education. Adequate care relates to parental capabilities and knowledge of childrearing practices such as feeding and ability to prevent and respond to diseases. Living conditions should be hygienic and disease free, and the ability of parents to provide favorable conditions is partly determined by their own health, abilities and socioeconomic status (SES). In turn, these are partly determined by the conditions they faced in their own childhood. In this way capability formation (e.g. health and education) of children is a function of capabilities of their parents, which is intimately connected to parental early-life conditions.

In this paper we study the role of mother's capabilities, as measured by her height, in determining child capabilities in sub-Saharan Africa, using individual-, household-, and community-level data from multiple Demographic and Health Surveys (DHS). The main research questions are: Do maternal capabilities as determined in early life, affect health and educational outcomes of children? If so, which socioeconomic pathways are important, and for which social group is the intergenerational transmission of capabilities strongest? Child health is measured by HAZ for children under 5, which represents deviations from the growth trajectory of healthy children, and the probability of dying before age 5. For child education we use two outcomes; school attendance at the time of survey, and school enrolment before age 10.

Previous studies of intergenerational transmission of health have found maternal height to be associated with both the probability of dying in infancy and anthropometric measures in childhood (Özaltin, Hil, & Subramanian, 2010). Demographic and socioeconomic pathways appear to be important, and maternal education at the community level seems to reduce the intergenerational persistence in health (Bhalotra & Rawlings, 2013). Palloni et al. (2009) find that a part of the relationship between intergenerational transmissions of social class can be attributed to childhood health and these effects run through effects of childhood health on human capital formation and cognitive skills. Mother's height determined by conditions in her childhood seems to be more important in determining child's HAZ than height in general (Venkataraman, 2011). Most of these studies are either focusing on more developed countries, using adult outcomes, or use large samples for the whole developing world, while micro-level studies of developing contexts looking at these long-term processes are rare.

We study the least developed region in the world focusing on childhood outcomes, and investigating the pathway of household SES factors, especially mother's education. In the analysis we employ community fixed-effects models controlling for unobserved heterogeneity across relatively small geographic areas. We also use husband fixed-effects in polygynous households and explore the possibility of using IV estimation to identify the causal effect from maternal early-life conditions to child outcomes. The study is limited to the transmission of mothers' capabilities since there is limited information on fathers in the surveys. Outcomes observable in childhood are only considered and not the consequences for adult health and capabilities, which pervious research has indicated (Case & Paxson, 2008; Moradi, 2010).

Theory and previous research

Mosley & Chen (1984) suggested to model child health using five broad categories of proximate determinants; maternal factors (e.g. mother's age, parity and birth interval); injuries; nutrition; environmental contamination (e.g. disease environment, poor hygiene); and illness control (e.g. preventive measures such as vaccination). Socioeconomic determinants can be used as proxies operating through proximate determinants, for example mother's education and household living standards. They capture skills for child rearing, e.g. regarding knowledge of adequate feeding practices, ability to prevent infections and respond to symptoms in children, the resources available for health related investments, and the ability to provide sanitary living conditions. Heckman (2007) developed a production function based on previous work by Grossman (1972), which complements the Mosley and Chen framework. In this model child capability formation (health and education) θ at each stage *t+1* is determined by investments I at previous stages and parents' capabilities *p.*

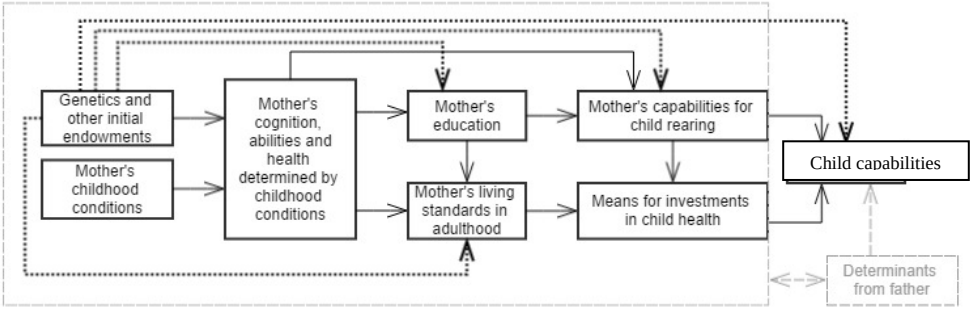
$$\theta_{t+1} = f_t(p, \theta_1, I_1, \dots, I_t)$$

Capabilities at each stage are also determined by capabilities at previous stage θ_t. θ_1 represents capabilities at the first stage or initial endowments, which is the capability stock the child starts out with at birth (or even at conception), determined mostly by genetic factors and some maternal factors, but also possibly early life conditions of the parents, especially the mother.[2] This framework implies that good conditions and sufficient care and investments should be insured already in early life, since children are the most vulnerable to environmental impact, and since later capabilities are determined by earlier investments and capabilities at earlier stages. As can be seen from figure 1, the pathways of intergenerational transmission of capabilities are

[2] Smaller physique of stunted mothers could play a role (Barker D. , 1997) as well as epigenetic inheritance (Low, Gluckman, & Hanson, 2012).

interwoven with genetic determinants, early-life determinants and SES. What is of primary interest in this paper is how capabilities are transmitted between generations through mother's early-life conditions and the resulting SES and capabilities in adulthood.

Figure 1. Pathways of intergenerational transmission of health



As discussed before, this can have a direct effect on maternal capabilities for child rearing, as well as for living standards in adulthood, which determines the resources available for health investments. However, it may also determine maternal education which affects both mother's capabilities for child rearing and her living standards in adulthood. Maternal capabilities could also influence how effectively means for investments in child health are utilized. Mother's cognition, abilities and health determined by early life conditions are not completely independent of genetic factors and other initial endowments, since it may influence how an individual responds to these conditions. The flow chart could be mirrored at child capabilities to demonstrate determinants stemming from the father. Assortative mating would bias estimates upwards, while marriage complementarity might bias it downwards.

The causes of low educational attainment, malnutrition and morbidity are interrelated, diverse and multi-factorial, and poverty is one of the underlying determinants of all of them. Poverty is widespread in the region, and low socioeconomic status of individuals, families and

communities are associated with food insecurity, lack of health care and education opportunities, poor housing and large family size (Collier, 2008). Thus place of residence can have important implications for population health and education because their influence can operate at various levels. A growing number of studies concerned with child health show that contextual factors are independent of individual factors in improving the chances of survival (Alderman, Hentschel, & Sabates, 2003; Kravdal, 2004; Fotso & Kuate-Defo, 2005; Andrzejewski, Reed, & White, 2009; Gessner, Chimonas, & Grady, 2010). Along this line, most studies have relied on maternal education as the main vehicle for explaining contextual differences in child health. One reason is that education is a powerful indicator of the social and psychosocial transformation of a society at the individual, household and community level (Caldwell, 1979; Gakidou, Cowling, & Murray, 2010), and is therefore central to understand the context people live in. Another reason is that it is less likely to change with time as most other measures of socioeconomic status.

Educated mothers are most likely better able to use health knowledge and technology, more likely to work, and have fewer children. Taken together this makes them more likely to be successful in improving the lives of their children by increasing their capabilities. There is also the competing view that education is a strong proxy for socioeconomic origin, which implies that educated mothers tend to come from wealthy families in good neighborhoods with access to water and other public utilities, as well as access to health care and good schooling (Desai & Alva 1998). In other words, the association between maternal education and child health is to some degree spurious. In addition, the educational level in the community could affect child health regardless of mother's own education (Parashar, 2005).

In a study of a UK cohort born in 1958, Palloni et al. (2009) find that a part of the intergenerational transmission of social class can be attributed to health in childhood. These effects run through effects of childhood health on human capital formation and cognitive skills.

They further find early life health to relate to the SES gradient observed in adulthood. The effects are small but the authors argue that they are lower bounds. Environmental conditions in the U.K. are likely to be far superior to most of sub-Saharan Africa, so the intergenerational transmission is likely to be much more persistent in our case.

Bhalotra & Rawlings (2013) find a considerable intergenerational persistence in health using a large sample from 38 developing countries for children born 1970 - 2000. They use mother's height to indicate her health and infant mortality to measure child health. They firstly find that a one standard deviation increase (7.3 cm) in mother's height decreases the probability of dying before the age of one by around 0.7% (or 8% of the average mortality rate in their sample), while controlling for country and household living standards. They find the effects to be asymmetric and gains from being born to tall mothers lower than the penalty for being born to shorter mothers. The effects increase by the deviation in height from the country specific mean. Favorable environmental factors, related to community education, and country level immunization status and income, were estimated to benefit children of mothers who were at the lower end of the health distribution more than those at the higher end. Some of these effects are large, e.g. a one year increase in mother's education lowers the persistence of intergenerational transmission of health by 17%, and a one standard deviation increase in log of GDP and immunization rates was associated with a 20% and 18.5% decreases in the persistence. When controls for parental SES and demographics were removed the intergenerational correlation increased by 45%, which indicates that much of the effects run through SES.

In a study of Vietnam, Venkataramani (2011) attempts to sort out environmentally determined height using an instrumental variable approach with several instruments relating to both mother's and father's early life environment, such as region and year of birth, rainfall, population density and education of grandparents to obtain a local treatment effect of height

determined by early life environmental conditions of parents. This approach yields a 2-3 times higher estimate than the OLS estimate, about 0.4 - 0.6 z-scores for mothers. This would indicate that environmental mechanisms are more important in determining the child's health.

Studies have also been done on the effects of mother's education on child health showing a strong correlation as indicated by height, mortality and immunization status. Some of the association between education and health could undoubtedly be accounted for by higher socioeconomic status, but some is likely due to increased knowledge and capabilities. When controls for household wealth and husband's education are included, the coefficient for mother's education is reduced but remains statistically significant (Desai & Alva, 1998). Community level education is also likely to be important since information and knowledge may be diffused between mothers in a community (Mosley & Chen, 1984).

Based on the preceding discussion we expect maternal health to have an impact on child capabilities, measured as child health and educational outcomes. This intergenerational transmission is mediated by maternal education and socioeconomic status. However, the mediation is not complete, so we expect a direct effect of maternal health on child outcomes as well.

Data and methods

We use data from Demographic and Health Surveys (DHS) for 35 sub-Saharan African countries, covering children born from the late 1970s until late 2000s. For child education outcomes 28 of these countries could be used. The DHS aims to provide nationally representative and reliable data (DHS, 2014b). The sampling procedure is a stratified two-stage sampling process; most commonly by geographic or administrative regions crossed with type of residence (urban/rural). Clusters (census enumeration areas) are randomly selected from each strata, and then households

are sampled from these clusters. Oversampling and probability weights are used to obtain reliable and representative estimates. The main outcome variables are child's HAZ, a binary variable for whether the child died before the age of 5 (U5 mortality), a binary variable for whether a child of compulsory school age (7-12) is attending school (Attending school), and whether a child started primary school before the age of 10 (School by 10). The main indicator for maternal health is mother's HAZ.

Although individual height is mostly determined by genetic factors it is also influenced by environmental factors (Silventoinen, 2003). Height has become a widely used indicator of accumulated health and net-nutritional history, i.e. the nutrition that has been available for growth after claims for maintenance, physical activity, and fighting diseases are accounted for. It is affected by nutrition and disease exposure in early life, which influences growth of children and finally adult height.[3] It has been shown to relate to characteristics such as health, cognition, education and income. Case & Paxson (2008) find a relationship between height of children and their cognitive test scores and that this difference in cognition also explains the higher wages of taller people. Although height is mostly determined by genetic factors, observed differences over time and space are mostly determined by changes in early-life conditions (Steckel, 1995; Martorell & Habicht, 1986; Malcolm, 1974). This assumes that height determines child health only through this environmentally determined component. Whether the component of height determined by genetic factors is related to health and capabilities is unclear but there are indications that it is not, or at least to a much lower extent (Case & Paxson, 2008; Sundet, Tambs, Harris, Magnus, & Torjussen, 2005). However, other initial endowments, such as grandmother's

[3] It may also be influenced by living conditions during late childhood and adolescence but not as strongly as during early life (Steckel, 1995).

age at mother's birth and her parity influence both her height and health (Bhargava, 2003; Curtis, 1993).

The height-for-age z-score (HAZ) is a standardized distribution of height which represents deviations from the median height of a healthy population by age and gender. It is measured at the time of the survey, generally for children under 5 (in few cases 3). Children younger than 3 months old are excluded due to reports of measurement error (Montgomery, Lloyd, Hewett, & Heuveline, 1997). For the binary outcome of whether the child survived until age 5, children born 5-20 years prior to the survey are included - lower bounds to avoid complications with censoring and upper bounds to reduce recall bias. Both these outcomes have the advantage of indicating a process with chronic conditions or multiple exposures rather than an acute phenomenon (Mosley & Chen, 1984). For the binary outcome of whether a child in a compulsory schooling age range during the time of the survey attended school during the year of the survey includes children born 7-12 years prior to the survey. Compulsory age range varies slightly in sub-Saharan Africa but this age range is always compulsory (UNESCO, 2011). For the binary outcome indicating whether a child started education by age 10 children born 10-15 years prior to the survey are included. The lower bounds avoid complications with censoring and upper bounds reduce potential bias due to children leaving home, since this variable is only recorded for children currently living in the household. So in reality the study consists of 4 samples which only partially overlap. Having different outcomes is good for comparing results, and comparing consistency between indicators.

Other independent variables of main interest are maternal education which is important for capabilities of mothers for child rearing, and can be expected to be a mediating factor for the effect of early life conditions on child outcomes. A relative index of household living standards and father's education will also be controlled for. This may relate to parental capabilities, as well

as hygiene and means for investing in children. Controls for maternal factors such as mother's age at birth, mother's age at first birth, birth interval, parity and number of siblings, and gender are included. Standard controls are mother's year of birth, child's year of birth in 5 year categories. Other specifications of birth years, such as single years, continuous and squared were also tested but did not change the results. Child's age in months and age in months squared were also included where the outcome is child's HAZ and whether the child is attending school to control for exposure time.

The average of each variable is displayed in the first row of Table 1. They are much in accordance with other sources. Although the samples do not overlap much the descriptive statistics look similar. The proportion of mothers with primary education in the U5 mortality sample is slightly lower. This could be an indication of selective mortality in the other groups, although other averages do not support this.

Table 1 here

We first estimate OLS and linear probability models for the four child outcomes *y* for child *i* including controls for *country* as well as a vector of *l* controls *x*.

$$y_i = \alpha + mHAZ_i\beta^{ols} + mEdu_i\,\rho^{ols} + x_{il}'\gamma_l^{ols} + country_{ik}\delta_k + \varepsilon_i$$

The main coefficients of interest are β for mother's HAZ but also ρ for mother's education. There is the concern that unobserved contextual factors which relate both to mother's HAZ and child outcomes could bias the results. To account for unobserved factors at community level we add cluster fixed effects. A cluster is a confined geographic area used in the first stage of the sampling procedure and can for example be a village. A cluster-specific average of all variables is subtracted from each variable of child *i* in community *c*.

$$y_{ic} - \bar{y}_c = \alpha + (mHAZ_{ic} - \overline{mHAZ}_c)\,\beta^{FE} + \left(mEdu_{ic} - \overline{mEdu}_c\right)\rho^{FE} + (com_{ic} - \overline{com}_c) + (x_{icl} - \bar{x}_{cl})'\gamma_l^{FE} + (\varepsilon_{ic} - \bar{\varepsilon}_c)$$

com indicates all unobserved heterogeneity at the community level which is shared by all *i* within *c* so $com_{ic} = \overline{com}_c$ and is cancelled out. This leaves the transformed model

$$y_{ic}^* = \alpha + mHAZ_{ic}^*\,\beta^{FE} + mEdu_{ic}^*\,\rho^{FE} + x_{icl}^{*\prime}\gamma_l^{FE} + \varepsilon_{ic}^*$$

where community level unobserved heterogeneity has implicitly been eliminated from the error term ε_{ic}^*. To further control for unobservable factors a husband fixed effects are used for polygynous households. There children of different wives of the same husband, which is likely to be the father in most cases, are compared, implicitly controlling for father, and household specific heterogeneity in the same way as for community before. These properties are very desirable especially since limited control for father's characteristics are employed as indicated in figure 1. It should be kept in mind that this analysis is limited to a very specific (although quite common) household structure which greatly limits generalizability of results. Therefore main focus will be on community fixed effects.

Interactions of several characteristics with mother's HAZ using community fixed effects are used to test for difference in strength of the association between mother's HAZ and child outcomes. Dummy variables for characteristics z_{ic} are added as

$$y_{ic}^* = \alpha + z_{ick}^*\delta_k^{FE} + mHAZ_{ic}^*\,\beta^{FE} + (z_{ick}^* * mHAZ_{ic}^*)\vartheta_k^{FE} + x_{icl}^{*\prime}\gamma_l^{FE} + \varepsilon_{ic}^*$$

With this specification δ_k^{FE} are the net associations of *k* dummy variables coded from *k+1* characteristic in *z* and β^{FE} is the slope parameter for the association of mother's HAZ in the reference category, the association of the omitted category from z. ϑ_k^{FE} are *k* coefficient for additional association of mother's HAZ for each *k* dummy variables of *z* above the reference

category. For characteristics *z* which are fixed within clusters only the slope parameter is identified.

In addition we explore the possibility of using an instrumental variable approach (IV) to obtain a local average treatment effect (Imbens & Angrist, 1994) of maternal height as determined by her early-life conditions. The instrument used in this study is the under 5 mortality rate during the first 5 years of mother's life (U5MR) in the region of interview. It is calculated from birth histories provided by the DHS as

$$U5MR_{mr} = \frac{\sum_{t=m}^{m+60} deaths_{tr}}{\sum_{t=m}^{m+60} births_{tr}}$$

where *m* stands for year and month of birth and *r* is the region of interview. The mother is born at *t* = *m* but the U5MR is calculated using counts of births and deaths from the month of birth and 60 months onwards. Because the instrument used is calculated from birth histories the sample used for the IV estimation is restricted and mostly includes mothers for more recent surveys. Minimum number of deaths allowed over this 60 month period is 50. Any periods with fewer deaths are coded as missing. If these U5MR series are made at the country level they show the same patterns as U5MR series provided by the World Bank, although ours are consistently slightly lower (World Bank, 2014).

The estimation is done using two stages least squares estimation (2SLS) where maternal height is first predicted by the instruments relating to her early-life conditions.

$$mHAZ_{mri} = \pi_1 + U5MR_{mr}\,\pi_2 + country_{ik}\,\pi_k + x'_{il}\,\pi_l + \omega_{ic}$$

These predicted values $\widehat{mHAZ}_{ic}$ are then incorporated into the main equation.

$$y_i = \alpha + \widehat{mHAZ}_i\,\beta^{2SLS} + x'_{ik}\,\gamma_k + \varepsilon_i$$

This instrument is chosen as it is likely to capture the early life environment and reflect both disease and nutritional environment. Although infant mortality rates in infancy has more

traditionally been used to indicate disease exposure (Bengtsson & Lindström, 2003) and its influence on adult height (Hatton, 2014) it is not feasible with our limited information on births and deaths obtained from the birth histories. But since under 5 mortality is mostly determined by a combination of disease exposures and malnutrition (UNICEF, 2013; Jones, et al., 2003) this gives an estimate of maternal height which relates to child capabilities mostly through its early life determinants. This would mean that the influences of the genetic pathways to child outcomes as indicated by the dotted lines in figure 1 are reduced, but genetic factors and initial endowments could still influence how mothers respond to early-life conditions. Unfortunately the instrument was not robust to community fixed effects which is most likely due to omitted variables driving the relationship, since it is likely to capture many aspects of development. Therefore the IV model is estimated without controlling for community fixed effects, which increases the likelihood of omitted variables bias and violation of the exclusion restriction. One such factor could be poverty, because regions that are poor in mother's childhood are more likely to be poor also when the child is born. Poverty is likely to be positively associated with greater U5MR, as well as all the child outcomes, which would bias the estimates upwards. The instrument U5MR is significant in all first stage regressions without community fixed effects, although it may be too weak in the child education samples, indicated by an f-statistic lower than 10 which could bias the estimates (Verbeek, 2012, pp. 137-171).

Results

Results from the OLS regressions are presented in the first two columns of Table 2. The first column shows that mother's HAZ has a strong association with all child outcomes. When education is added to the models in the second column this relationship gets much weaker for the educational outcomes. While this is what could be expected if part of the effect of maternal health

is indirect, channeled through mother's education, it is important to note that there is also a direct effect from maternal health to educational outcomes of the children. The association between maternal height and the two health related outcomes, child HAZ and U5 mortality, is not reduced muchby adding mother's education to the model, which indicates that the direct effect is much more important here. There is also a strong association between mother's education and all outcomes, especially the educational ones, which is also what could be expected.

Table 2 here

Estimates from community fixed effects models are shown in columns 3-6. The first models (3) include only basic controls and could be compared to the OLS estimates in column 1. The effects of maternal health on all child outcomes are smaller than in the OLS model. In the fixed-effects model a single z-score increase in mother's height is associated with a 0.7 percentage point higher probability of starting school before age 10, and a 1 percentage point increase in the probability of being enrolled in education at the time of the survey. The probability of dying before age 5 is reduced by 1 percentage point, and the child HAZ is increased by 0.25 by a 1 z-score increase in mother's height. As in the OLS models adding mother's education as a control (column 4) further reduces the association between mother's HAZ and the educational outcomes, but much less so for the health outcomes.

Adding household living standards and father's education as control variables (column 5) further reduces the association between mother's HAZ and the child educational outcomes considerably, while the association with child HAZ and U5 mortality does not change that much. The association between mother's education and child outcomes is reduced considerably for all outcomes. In column 6 controls for maternal- and individual factors are added. These do not have much impact on the magnitude of the effects of maternal height on the education outcomes or child height, but further reduces the effect on U5 mortality. In this model a one unit increase in

maternal HAZ is associated with a 0.3 percentage point increase in the probability of starting school before 10, 0.6 percentage point increase in the probability of school attendance, a 0.5 percentage point lower probability of dying before age 5, and a 0.24 increase in the HAZ of the child.

Considering averages of 0.7 for the child schooling variables, 0.17 for U5 mortality and -1.557 for child HAZ, these effects are not very large. Still they indicate that there is a connection between mother's capabilities determined in early life and child capabilities.

Comparing the husband fixed effects models (column 7) to the community fixed effects models in column 3 and 5 there are some differences. The association of mother's HAZ with child education variables is estimated slightly higher than in model 3 and considerably higher than model 5, although it is not significant for school by 10. The association with child HAZ and U5 mortality is slightly lower. When all controls are added (column 8) the association of mother's HAZ with child education does not change much, unlike in the community fixed effects models. Child HAZ and U5 mortality show a similar pattern between models. OLS regressions on the polygyny sample (not shown) corresponding to model in columns 1 and 2 show slightly lower association with mother's HAZ on all outcomes except U5 mortality where the association is roughly the same. Association with mother's education and all child outcomes is roughly the same for the polygyny sample using OLS while in the husband fixed effects models (model 8) they are lower and often not significant for the child education variables, especially attending school. Mother's education has roughly the same size coefficient for U5 mortality, although not statistically significant, while for child's HAZ it is considerably larger.

Estimating separate community fixed effects models corresponding to the most basic model (column 3) for each country shows a consistent relationship, even though many samples are too small to yield statistical significance (results not shown). The association with mother's

HAZ is significant in 9 countries for school by 10 and 11 countries for attending school, out of 28 countries. The association with mother's HAZ is significant in all countries for child HAZ and 29 out of 35 countries for U5 mortality.

In Table 3 interaction terms are included to the community fixed effects specification from column 3, Table 2. The first part looks at interactions of mother's HAZ and mother's age at the birth of the child. The only statistically significant interactions are found for school enrolment before age 10 and U5 mortality. For school enrolment by age 10 the effect of maternal height is limited to women younger than 35, while there is no positive association at all for women over 35. For U5 mortality the association with mother's HAZ has a clear age gradient which is weaker for older mother's but is always significantly negative.

Table 3 here

The interaction of mother's HAZ and mother's education shows that it is only children to mothers with no or primary education that show an association between maternal HAZ and school enrolment at age 10. When mothers have at least secondary education there is no relationship (the negative interaction effect is actually somewhat larger than the positive base effect). Also for child mortality there is less of an intergenerational effect for highly educated mothers, but all groups show a negative association between maternal height and U5 mortality. For child HAZ having a mother with at least some education strengthens the intergenerational association in height, but there is no apparent gradient in this.

Turning to interaction with household wealth, the effect of mother's height on schooling is strongest in the poorest households, but there is a positive effect in all wealth quintiles. For child HAZ on the other hand, the effect of maternal height is stronger the richer the households. For U5 mortality children in the 4^{th} quintile show weaker association than the reference category while there are no statistically significant differences between other quintiles.

Finally, the interaction between maternal HAZ and quintiles of maternal HAZ shows that the association is lowest and statistically insignificant for the tallest groups for the educational outcomes and U5 mortality. For child HAZ the association with mother's HAZ is much lower for the tallest quintile of mother's HAZ although there is still a statistically significant association. Interactions with gender and urban/rural residence did not show statistically significant differences (results not shown).

Turning to the IV estimates in Table 4, the effect of maternal HAZ is much stronger than in the OLS models. For the educational outcomes it is extremely large and above 1, which indicates a more than 100 percentage points increase in the probability. The effects for the health outcomes are also much bigger. A one unit increase in maternal HAZ increases child HAZ by around 2.6 and lowers the probability of dying in childhood by almost 43 percentage points. The Angrist-Pischke multivariate F test (AP F test) of excluded instruments indicates that the instruments might be too weakly identified for the education outcome which is indicated by an f-statistic of less than 10 which might cause biased estimates.

Table 4 here

Discussion

Our results of the effect of maternal height on educational and health outcomes of the children are quite consistent across the different outcomes. Results are also similar across different countries in sub-Saharan Africa. The height of the mother has a clear impact on the educational outcomes of her children as well as on their height and mortality. The effect is somewhat mediated by maternal education, especially in the case of the educational outcomes. Nonetheless, there is a direct effect as well of maternal health, as measured by her height.

Although our IV estimates also indicate importance of early life disease exposure and nutrition of mother's for child capability formation there are still many issues that need to be solved, and this part of the analysis should be considered as work in process. Better instruments should be obtained which more clearly predict mother's height, are robust to controlling for community level heterogeneity and are less likely to violate the exclusion restriction. One way would be to utilize information from other household surveys to calculate regional level indicators, or obtain them from other sources. But the intuition would be the same - to isolate the component of mother's height determined in her early life to obtain a local average treatment effect of early life determined mother's height on child potentials.

Although there are some differences between the husband fixed effects and community fixed effects this could solely be due to the husband fixed effects being limited to a specific part of the population which is likely to differ in many ways. It is still possible that husband fixed effects may reduce positive assortative mating bias in the case of child HAZ and under 5 mortality outcomes.

The interactions between maternal height and a number of variables related to the living conditions of the mother also show interesting results. Effects of older mothers are smaller than for younger mothers in terms of educational outcomes and mortality. Education and to some extent survival of children in better-off households, with better educated mothers are less affected by the height of their mothers, while the opposite is true when looking at child height. This is likely to be explained by the different components determining mother's height driving the association. Better-off mothers are more likely to have lived up to their genetic potential in terms of height, and their height is therefore largely genetically determined. Hence, the association between maternal and child height will then not be an indication of health and cognition, but simply of shared genes. The opposite is true for mothers in poor households, whose height is to a

greater extent is determined by environmental factors, such as disease exposure and nutrition in early life. The only result opposing this is that mother's HAZ has a stronger association with under 5 mortality for the fourth quintile of wealth. This inconsistency is hard to explain but it only concerns one coefficient while other quintiles do not have significantly different association from the reference category.

Even though the associations found are not large they still indicate intergenerational transmission of capabilities, which are likely to be through early life conditions of mother. Considering that early life conditions determine only a component of height the link may be stronger than indicated by these estimates. Poor health of mothers affects both schooling and health of her children, which in turn has great implications for both productivity and later life mortality of these individuals. Poor capability formation during childhood traps families in a vicious circle of underinvestment in children, leading to low adult capabilities. It is important to break this circle by making sure that children in disadvantaged household get external support and monitoring for adequate care and investments. It not only concerns individual wellbeing but also future human capital formation, which is of crucial importance if these countries are to reap the full benefits of the demographic dividend, stimulating economic growth and societal development.

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Table 1: Descriptive statistics

	Samples by outcome			
	Mean (SD)			
Variables:	School by 10	Attending school	Child HAZ	U5 mortality
Outcome	0.699	0.705	-1.557	0.169
	(0.459)	(0.456)	(1.624)	(0.375)
Mother HAZ	-0.856	-0.876	-0.859	-0.819
	(1.11)	(1.112)	(1.113)	(1.111)
Rural	0.742	0.751	0.757	0.752
	(0.437)	(0.433)	(0.429)	(0.432)
Female	0.488	0.49	0.496	0.491
	(0.5)	(0.5)	(0.5)	(0.5)
Mother primary education	0.558	0.556	0.522	0.458
	(0.497)	(0.497)	(0.499)	(0.498)
Mother year of birth	1967.79	1970.366	1974.867	1969.644
	(6.916)	(7.422)	(8.343)	(8.234)
Child year of birth	1993.36	1996.787	2001.995	1994.709
	(3.932)	(3.824)	(4.994)	(6.586)
Number of observations	148,014	164,386	287,122	958,051
Number of communities	18,704	18,948	30,475	31,331

Data source: DHS, 2014

Table 2. OLS, community fixed effects and husband fixed effects

	(1) OLS	(2) OLS	(3) FE	(4) FE	(5) FE	(6) FE	(7) FE[1]	(8) FE[1]
Outcome: School by 10								
Mother's HAZ	0.0172***	0.00714***	0.00698***	0.00471***	0.00295**	0.00303**	0.00908	0.00893
	(0.00156)	(0.00145)	(0.00138	(0.00136)	(0.00135)	(0.00135)	(0.00674)	(0.00688)
Mother's education[2]: Primary		0.227***		0.0879***	0.0614***	0.0623***		0.0561*
		(0.00497)		(0.00472)	(0.00466)	(0.00466)		(0.0326)
More than primary		0.358***		0.153***	0.0957***	0.0946***		0.0664*
		(0.00534)		(0.00521)	(0.00527)	(0.00536)		(0.0357)
Outcome: Attending school								
Mother's HAZ	0.0194***	0.0103***	0.00989***	0.00803***	0.00634***	0.00633***	0.0142***	0.0147***
	(0.00145)	(0.00134)	(0.00123	(0.00122)	(0.00121)	(0.00121)	(0.00535)	(0.00530)
Mother's education [2]: Primary		0.224***		0.0782***	0.0549***	0.0535***		-0.0296
		(0.00487)		(0.00407)	(0.00407)	(0.00408)		(0.0244)
More than primary		0.336***		0.131***	0.0799***	0.0761***		0.00245
		(0.00542)		(0.00471)	(0.00483)	(0.00492)		(0.0307)
Outcome: Child's HAZ								
Mother's HAZ	0.293***	0.273***	0.254***	0.250***	0.246***	0.245***	0.236***	0.233***
	(0.00391)	(0.00388)	(0.00396	(0.00396)	(0.00396)	(0.00396)	(0.0215)	(0.0217)
Mother's education[2]: Primary		0.243***		0.0663***	0.0364***	0.0333***		0.119**
		(0.0105)		(0.0109)	(0.0111)	(0.0111)		(0.0584)
More than primary		0.656***		0.247***	0.155***	0.137***		0.306**
		(0.0139)		(0.0145)	(0.0155)	(0.0156)		(0.119)
Outcome: U5 mortality								
Mother's HAZ	-0.0155***	-0.0132***	-0.0103***	-0.00979***	-0.00944***	-0.00537***	-0.00796***	-0.00494**
	(0.000541)	(0.000533)	(0.000544	(0.000545)	(0.000545)	(0.000448)	(0.00292)	(0.00235)
Mother's education[2]: Primary		-0.0400***		-0.0162***	-0.0112***	-0.00944***		-0.00916
		(0.00159)		(0.00165)	(0.00168)	(0.00138)		(0.00881)
More than primary		-0.0851***		-0.0398***	-0.0263***	-0.0378***		-0.0252
		(0.00187)		(0.00199)	(0.00209)	(0.00185)		(0.0160)

continued	*(1)*	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*
Community fixed effects			Yes	Yes	Yes	Yes		
Husband fixed effects							Yes	Yes
Controls:								
Constant	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Country [3]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Child's year of birth	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Mother's year of birth	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Child's age in months [4]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Child age in months squared [4]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Father's education [5]					Yes	Yes		Yes
Wealth index quintile [5]					Yes	Yes		Yes
Sex of child						Yes		Yes
Mother's age at first birth						Yes		Yes
Mother's age at birth						Yes		Yes
Birth interval * order						Yes		Yes
Alive siblings						Yes		Yes

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

[1] Husband fixed effects for polygamous households. Sample sized are reduced to 29,013 ; 31,911; 56,144; and 236, 890

[2] No education is reference category

[3] Country is implicitly controlled for with community and husband fixed effects

[4] Only included where outcomes are attending school and child HAZ

[5] Household wealth index and father's education are implicitly controlled for in husband fixed effects

Data source: DHS, 2014

Table 3: Community fixed effects: Interactions of maternal characteristics and SES with mother's HAZ

Interaction term:	Outcome:			
	School by 10	**Attending school**	**Child HAZ**	**U5 mortality**
Mother's HAZ * mother's age at birth[1]				
+ Under 20	-0.00308	0.00126	0.00658	-0.00546***
	(0.00348)	(0.00317)	(0.0105)	(0.00128)
+ 20-25	0.00512*	-9.25e-05	-0.00546	-0.00254**
	(0.00279)	(0.00261)	(0.00797)	(0.00106)
25-35 years old (reference category)	0.00690***	0.00990***	0.254***	-0.00837***
	(0.00186)	(0.00182)	(0.00538)	(0.000743)
+ over 35	-0.0123***	-0.00210	-0.00125	0.00356**
	(0.00436)	(0.00345)	(0.00983)	(0.00169)
Mother's HAZ * mother's education				
No education (reference category)	0.00430*	0.00856***	0.222***	-0.00970***
	(0.00225)	(0.00200)	(0.00556)	(0.000782)
+ Some primary	0.00462	0.000450	0.0557***	-0.000465
	(0.00342)	(0.00292)	(0.00883)	(0.00129)
+ Completed primary	0.000665	-0.000147	0.0639***	-0.000393
	(0.00380)	(0.00331)	(0.0122)	(0.00159)
+ Some secondary	-0.00613*	-0.00463	0.0465***	0.000433
	(0.00343)	(0.00298)	(0.0114)	(0.00144)
+ Secondary or more	-0.00803**	-0.00615*	0.0389**	0.00456**
	(0.00344)	(0.00324)	(0.0181)	(0.00201)
Mother's HAZ * household wealth index				
1st quintile (reference category)	0.00967***	0.0123***	0.227***	-0.00878***
	(0.00291)	(0.00250)	(0.00763)	(0.00111)
+ 2nd quintile	-0.00209	-0.00274	0.0241**	-0.00185
	(0.00412)	(0.00353)	(0.0105)	(0.00155)
+ 3rd quintile	-0.00522	-0.00802**	0.0226**	-4.35e-05
	(0.00425)	(0.00362)	(0.0106)	(0.00153)
+ 4th quintile	-0.0104**	-0.00809**	0.0305***	-0.00350**
	(0.00407)	(0.00380)	(0.0110)	(0.00157)
+ 5th quintile	-0.00789**	-0.00404	0.0423***	1.32e-05
	(0.00375)	(0.00340)	(0.0118)	(0.00156)
Mother's HAZ quintiles * Mother's HAZ				
1st quintile (reference category)	0.0172**	0.0198***	0.239***	-0.0160***
	(0.00670)	(0.00553)	(0.0167)	(0.00259)
+ 2nd quintile	0.0213	-0.00421	0.0413	0.00214
	(0.0199)	(0.0169)	(0.0481)	(0.00751)
+ 3rd quintile	0.0134	-0.0311*	0.0628	-0.00354
	(0.0206)	(0.0180)	(0.0526)	(0.00773)
+ 4th quintile	0.00738	-0.0195	0.0401	-0.000232
	(0.0174)	(0.0157)	(0.0487)	(0.00661)

continued	*School by 10*	*Attending school*	*Child HAZ*	*U5 mortality*
+ 5th quintile	-0.0223***	-0.0173**	-0.0885***	0.0137***
	(0.00831)	(0.00725)	(0.0226)	(0.00320)
Constant	Yes	Yes	Yes	Yes
Community fixed effects	Yes	Yes	Yes	Yes
Child's year of birth	Yes	Yes	Yes	Yes
Mother's year of birth	Yes	Yes	Yes	Yes
Child's age in months	Yes	Yes	Yes	Yes
Child age in months squared		Yes	Yes	
Observations	148,014	164,386	287,122	958,051
Number of communities	18,704	18,948	30,475	31,331

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

[1] Also includes controls for birth order

Data source: DHS, 2014

Table 4: OLS and 2SLS models

	Outcome:			
	School by 10	**Attending school**	**Child HAZ**	**U5 mortality**
OLS				
Mother's HAZ	0.0139***	0.0159***	0.285***	-0.0159***
	(0.00241)	(0.002)	(0.00445)	(0.000791)
2SLS				
Mother's HAZ	1.376***	1.926***	2.632***	-0.427***
	(0.372)	(0.406)	(0.153)	(0.0355)
First stage (AP F test)[1]:				
Mother's HAZ	3.57	5.6	74.91	40.86
Controls (exogenous):				
Mother's year of birth	Yes	Yes	Yes	Yes
Child's year of birth	Yes	Yes	Yes	Yes
Country	Yes	Yes	Yes	Yes
Child's age in months		Yes	Yes	
Child's age in months squared		Yes	Yes	
Observations	64,229	76,785	204,065	444,751

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

[1] Excluded instruments are 60 month U5MR.

Angrist-Pischke multivariate F test (AP F test) of excluded instruments test weather endogenous regressors are weakly identified. An F-statistic of less than 10 is often used as a reference for indicating weak instruments.

Data source: DHS, 2014

Table 5: Proportion of sampled for each country

	Proportion of each sample by outcome			
Countries:	**School by 10**	**Attending school**	**Child HAZ**	**Under 5 death**
Benin	0.0663	0.0690	0.0712	0.0772
Burkina Faso	0.0398	0.0394	0.0569	0.0529
Burundi	0.0136	0.0135	0.0099	0.0076
Cameroon	0.0321	0.0331	0.0262	0.0246
Central African Republic	0	0	0.0064	0.0047
Chad	0	0	0.0324	0.0214
Comoros	0	0	0.0062	0.0091
Congo (Brazzaville)	0.0265	0.0266	0.0185	0.0170
Congo (DR)	0.0440	0.0450	0.0150	0.0254
Ethiopia	0.0811	0.0784	0.0463	0.0419
Gabon	0.0109	0.0101	0.0043	0.0065
Ghana	0.0253	0.0241	0.0164	0.0151
Guinea	0.0121	0.0124	0.0212	0.0266
Ivory Coast	0	0	0.0047	0.0071
Kenya	0.0494	0.0461	0.0569	0.0416
Lesotho	0.0171	0.0142	0.0083	0.0082
Liberia	0.0147	0.0174	0.0145	0.0195
Madagascar	0.0555	0.0547	0.0390	0.0347
Malawi	0.0598	0.0593	0.0487	0.0404
Mali	0.0431	0.0454	0.0771	0.0735
Mozambique	0.0341	0.0356	0.0528	0.0517
Namibia	0.0118	0.0112	0.0128	0.0150
Niger	0.0167	0.0183	0.0334	0.0360
Nigeria	0.1066	0.1121	0.0989	0.1423
Rwanda	0.0340	0.0345	0.0224	0.0192
Sao Tome and Principe	0.0068	0.0062	0.0040	0.0036
Senegal	0.0115	0.0111	0.0169	0.0156
Sierra Leone	0.0088	0.0091	0.0095	0.0210
Swaziland	0.0096	0.0077	0.0045	0.0053
Tanzania	0.0286	0.0281	0.0434	0.0341
Togo	0	0	0.0097	0.0079
Uganda	0.0410	0.0412	0.0442	0.0346
Zambia	0.0440	0.0451	0.0317	0.0255
Zimbabwe	0.0550	0.0510	0.0360	0.0333
Number of observations	148,014	164,386	287,122	958,051

Data source: DHS, 2014